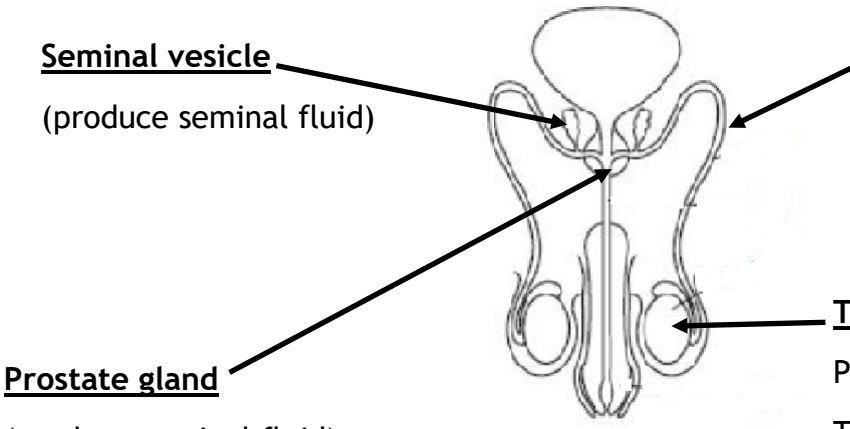
Unit 2 Revision Notes

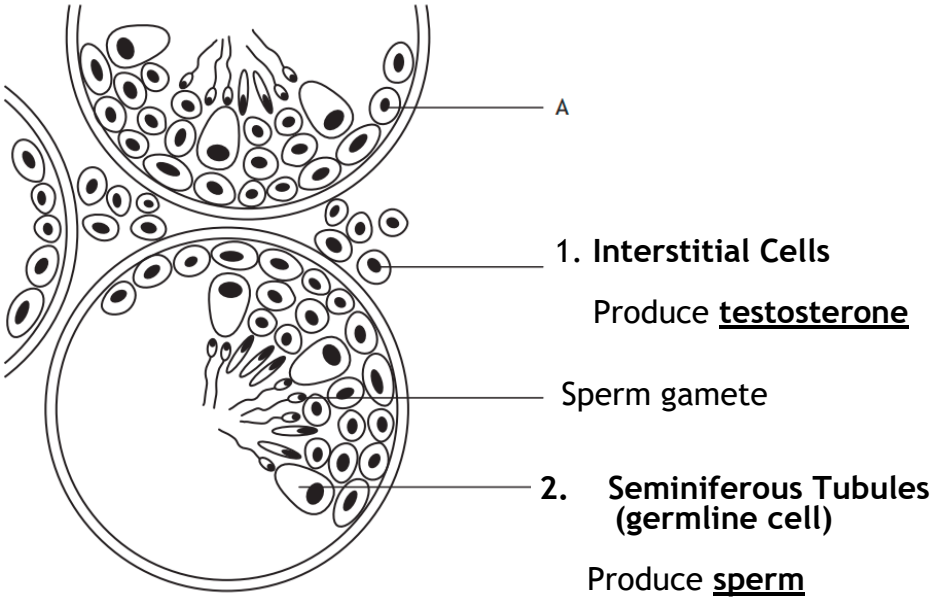
BRIGHT MINDS
MENTORING

Gamete Production in the Testes

Male Reproductive System



Parts of the Testes



Other Male reproductive parts

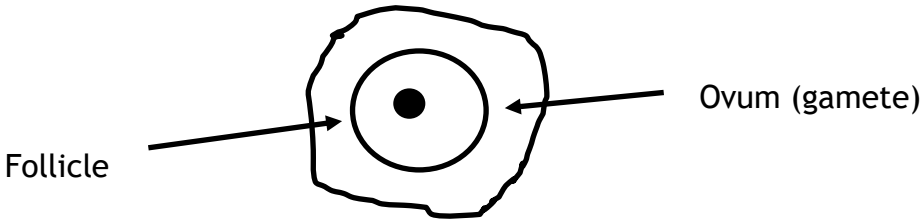
Prostate gland/Seminal vesicles

Secrete **Seminal fluid t**hat **maintains** the **mobility** & **viability** of the sperm.

Gamete Production in the Ovaries

Ovaries (germline cell)

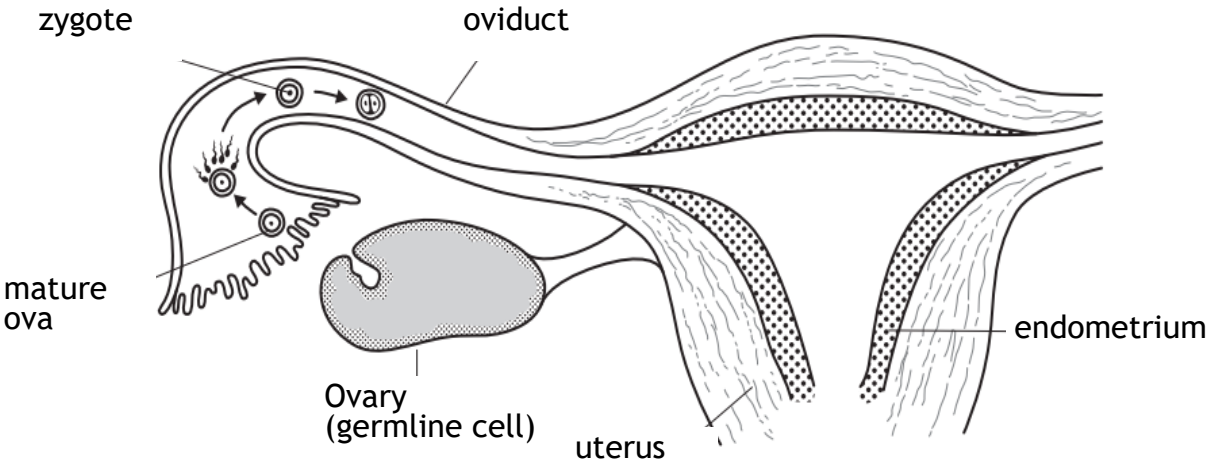
Contain **immature** ova in various stages of development.



1. **Protects** the developing ovum
2. **secretes hormones (oestrogen)**

Fertilisation

Mature ova are released into the **oviduct** where they may be fertilised by sperm, forming a **zygote**.



Hormonal influence on Puberty

1. Hormonal Influence on Puberty

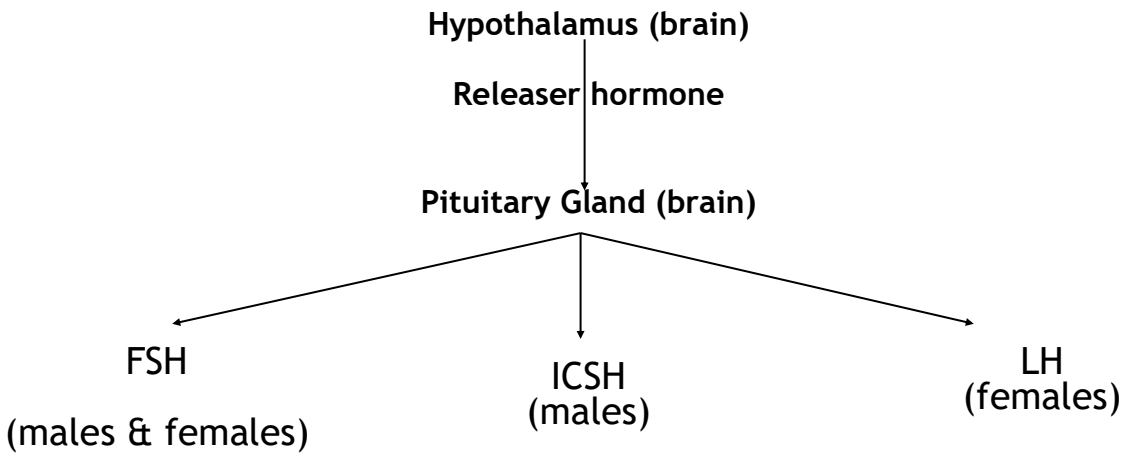
Hypothalamus (brain)

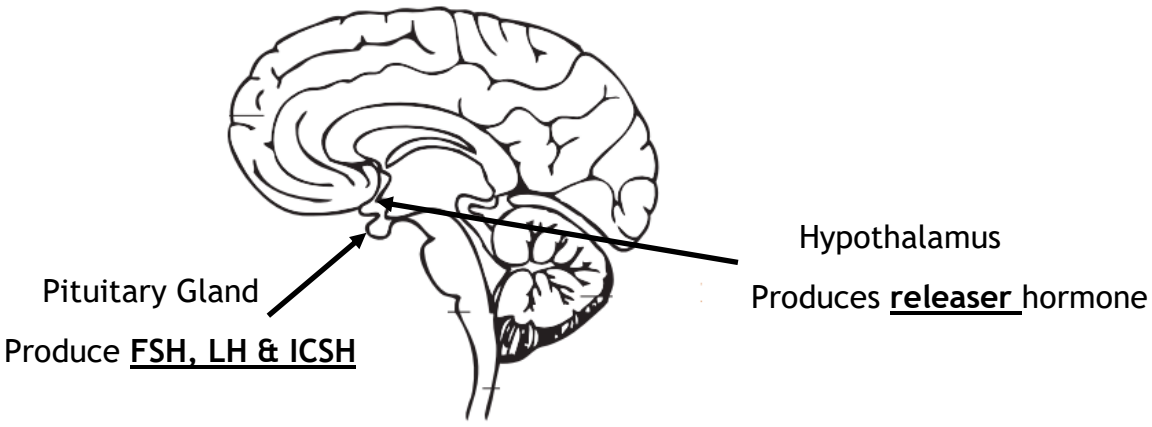
Produces a **releaser** hormone that acts on the **pituitary gland**.

Pituitary gland (brain)

Releases **FSH, LH & ICSH** in response to the releaser hormone.

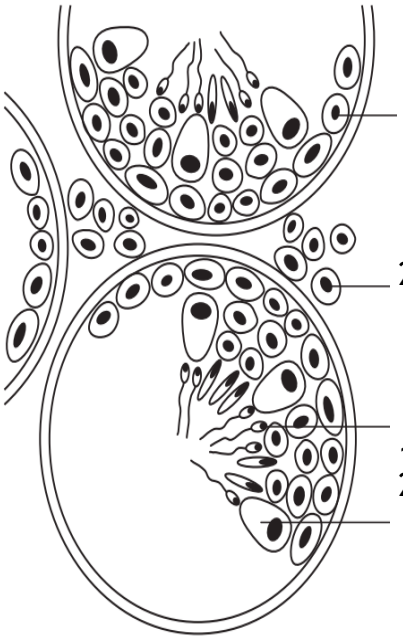
This triggers the onset of **puberty**.





Hormonal influence on Sperm production

Parts of the Testes



1. **Seminiferous Tubules (germline cell)**

   FSH stimulates **sperm production**.

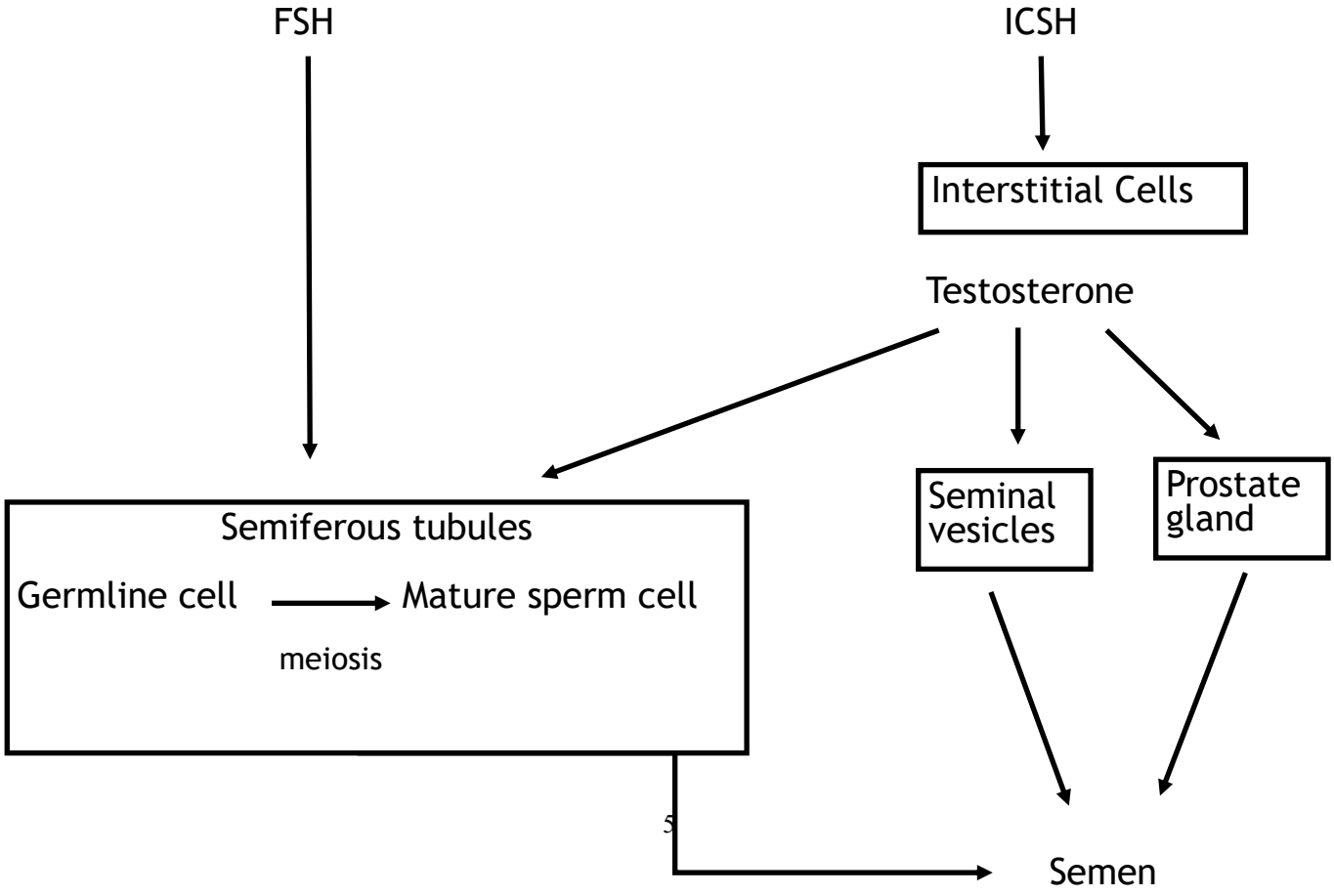
2. **Interstitial Cells**

   ICSH stimulates **testosterone production**

   Testosterone

   1. Stimulates sperm production
   2. Activates **prostate gland** & **seminal vesicles**

Summary Diagram of male hormones



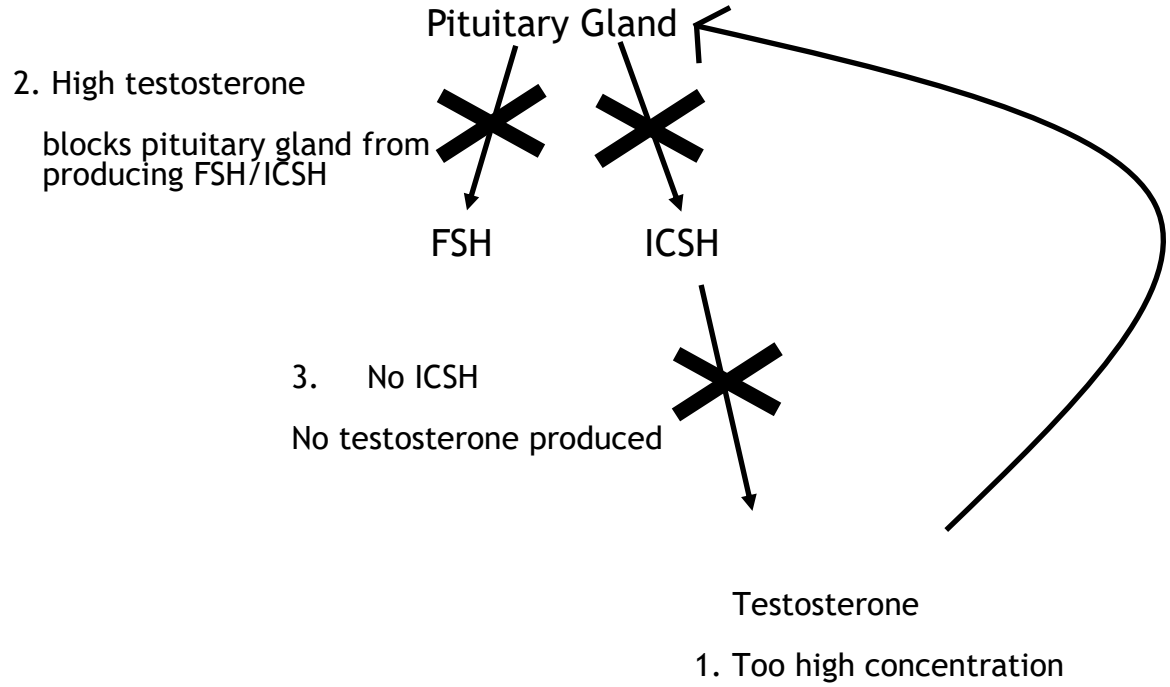
Hormonal influence on Sperm production

Negative Feedback by Testosterone

1. Testosterone reaches a **critically <u>high concentration</u>**

2. It feeds back to the **<u>pituitary gland</u>** and **<u>inhibits</u>** the secretion of **<u>FSH</u>** & **<u>ICSH</u>**

3. Preventing further testosterone being producing, **<u>decreasing testosterone</u>** concentration



Hormonal influence

Four hormones control the menstrual cycle

Two Pituitary Gland hormones

1. FSH — Stimulates development of follicle around ova in ovaries
2. LH — Causes ovulation in ovaries

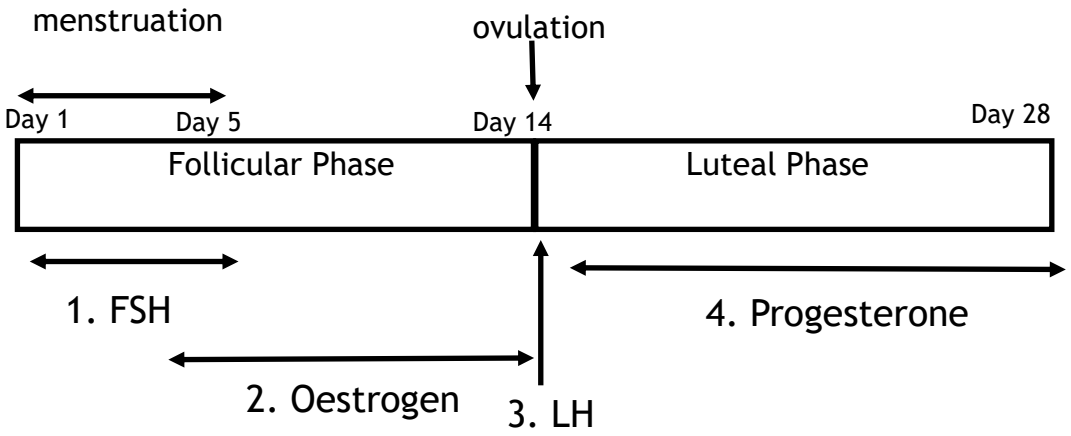
Two Ovarian Hormones

1. Oestrogen — Proliferation of endometrium
   Thins cervical mucus
   Causes LH surge
2. Progesterone — Further development/vascularisation of endometrium

Hormone	Phase of Menstruation	Produced	Target
FSH	Follicular	Pituitary gland	Ovaries
Oestrogen	Follicular	Follicle of ovum in ovaries	Endometrium Cervix Pituitary gland
LH	Ovulation	Pituitary gland	Ovaries
Progesterone	Luteal	Corpus luteum	Endometrium

Hormonal influence

Hormones in the menstrual cycle (~28 days)



Follicular Phase

1. FSH

   Stimulate the development of a follicle. Causes oestrogen to be produced from follicle.

2. Oestrogen

1. Stimulates proliferation of the endometrium in preparation for implantation of fertilised egg.

2. Affects the consistency of cervical mucus making it more easily penetrated by sperm.

3. Peak oestrogen levels stimulate surge in LH secretion by pituitary.

3. LH

   Triggers ovulation; the release of an egg (ovum) from a follicle in the ovary.

   Causes corpus luteum to start producing progesterone.

Luteal Phase

4. Progesterone

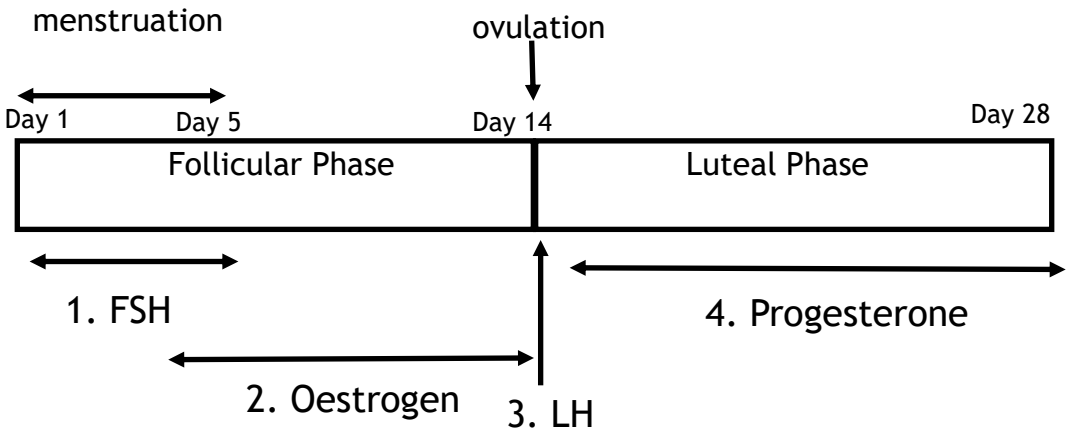
   Further development/vascularisation of the endometrium in preparation for implantation of fertilised egg.

   High levels of oestrogen/progesterone in luteal phase inhibit the pituitary gland from producing FSH and LH by negative feedback.

   This prevent further follicles from developing

Hormonal influence

Hormones in the menstrual cycle (~28 days)



- **Day 1-5:**

  The menstrual period of the follicular phase where part of the endometrium is shed from the uterus.

  Primary follicles begin to develop by release of FSH from pituitary

- **Day 5-13:**

  Follicular stage—where the follicle matures.

  Increase in oestrogen causes proliferation of endometrium and thinning of cervical mucus leading to an LH surge.

  Day 14: Ovulation; release of an egg (ovum) from a follicle in the ovary.

- **Day 15-28**:

  Luteal phase.

  The follicle develops into a corpus luteum which secretes progesterone.

  Increase in progesterone results in further vasularisation of the endometrium. ready for implantation.

  No fertilisation

  Corpus luteum degenerates and progesterone levels fall.

  Fertilisation

  The corpus luteum does not degenerate & progesterone levels remain high.

Female Hormone summary

Following ovulation and release on an egg by LH the follicle is now a corpus luteum releasing progresterone

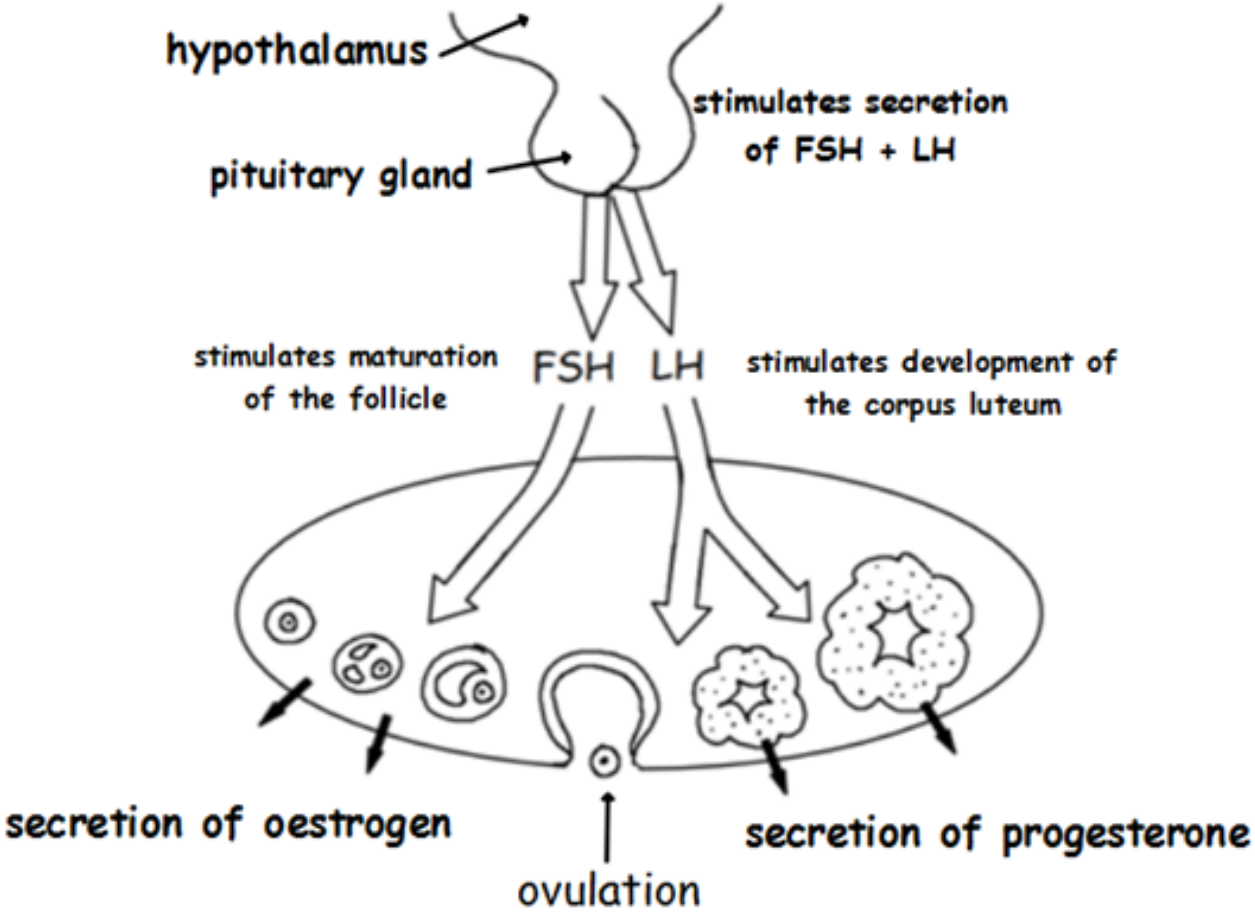
No pregnancy

At the end of the luteal phase, the corpus luteum breaks down and progesterone levels

Fall triggering menstruation and the start of a new cycle.

Pregnancy

At the end of the luteal phase, the corpus luteum DOES NOT break down and progesterone levels remain high and menstruation DOES NOT occur.



Hormone Summary

Female Hormone Summary

Hormone	Production Gland	Target Gland	Effect
Releaser	Hypothalamus	Pituitary	Releases FSH & ICSH (triggers puberty)
FSH	Pituitary	Ovaries	Promotes development & maturation of follicle. Stimulates ovary to release oestrogen
LH	Pituitary	Ovaries	Triggers ovulation Stimulates development of corpus luteum & release of progesterone
Oestrogen	Ovary (follicle)	Uterus & Pituitary	Stimulates cell division of endometrium Regulates cervical mucus viscosity Stimulates LH secretion by pituitary
Progesterone	Ovary (corpus luteum)	Uterus & Pituitary	Promotes development of endometrium (preparing it for blastocyst implantation) Inhibits secretion of FSH & LH.

Male Hormone Summary

Hormone	Production Gland	Target Gland	Effect
Releaser	Hypothalamus	Pituitary	Releases FSH & ICSH (triggers puberty)
FSH	Pituitary	Testicle	Promotes sperm production in seminiferous tubules
ICSH	Pituitary	Testicle	Promotes testosterone production in interstitial cells
Testosterone	Interstitial Cells	Testicle & Pituitary	Promotes sperm production in seminiferous tubules. Activates prostate gland & seminal vesicles to produce secretions. Has inhibitory effect in release of FSH & ICSH from the pituitary gland.

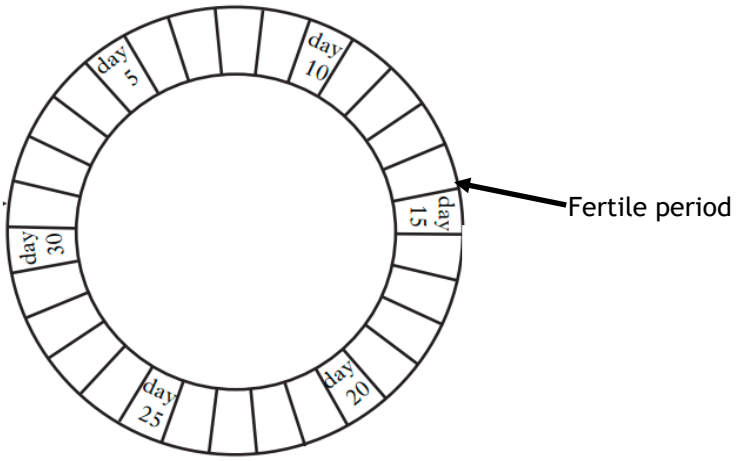
Fertility

Continuous fertility.

Men always produce sperm in their testes throughout the month.

Cyclical fertility

Women are only fertile for a **few days around day 14 (ovulation)** each menstrual Cycle. This is termed the fertile period.



Signs of Fertile period

1. A woman's body temperature rises by around 0·5°C after ovulation

2. Cervical mucus becomes thin and watery

Treating Infertility

1. **Artificial insemination**

   Artificial injection of sperm into the vagina/uterus.

   Several semen samples are collected **over time**.

1. Male has a **low** sperm count.
2. Use of a **semen donor** if male sterile

2. Ovulation Drugs

Stimulate ovulation if a woman is not ovulating/ovulating irregularly.

1. Ovulation drugs Type 1

   Prevent the **negative feedback** effect of **oestrogen** on **FSH** secretion.

2. Ovulation drugs Type 2

   Mimic the action of **FSH and LH**.

   Risk of Super ovulation

   When **multiple mature ova** are released at the same time.

   This is desirable during **IVF programmes** to collect multiple eggs but NOT when couples are trying to conceive naturally using ovulation drugs.

   Risk of Ovulation drugs

   Increased likelihood of **super ovulation** resulting in **multiple births.**

ICSI used when

1. **Defective** Mature sperm
2. **Low** sperm count

Treating Infertility

In vitro fertilisation (IVF) Stages

1. **Surgical removal of eggs** from ovaries after **hormone stimulation**.
2. Eggs are **mixed** with sperm in a **culture dish**.
3. The **fertilised eggs** (zygotes) are incubated until they have formed at least **eight** cells.
4. **Uterine implantation** of zygotes.

Intra-cytoplasmic sperm injection (ICSI)

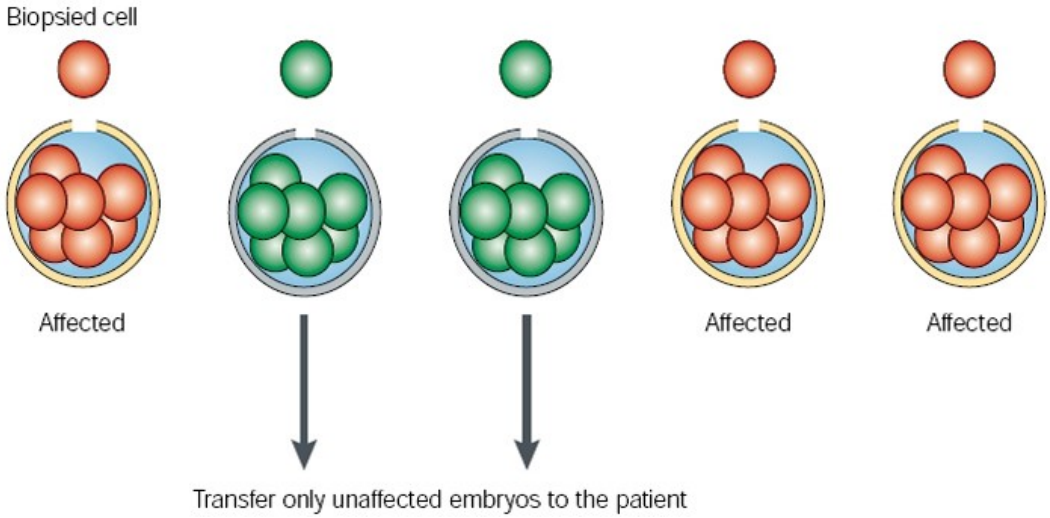
The **head** of the **sperm** is drawn into a needle and **injected directly** into the **egg** to achieve **fertilisation**.

Genetic Disease & IVF

Individuals with family histories of genetic disease may use **IVF to enable PGD** following incubation of zygotes **prior** to uterine implantation.

Pre implantation Genetic diagnosis (PGD)

Screening of zygotes prior to uterine implantation to detect **single gene disorders/ chromosome abnormalities**



Physical/Chemical methods of Contraception

Physical methods of contraception.

Prevent sperm entering uterus,

1. **Barriers**: to prevent sperm meeting egg and resulting in fertilisation.

   Males: condoms

   Females: femidoms OR cap/diaphragm

2. **Intra-uterine devices** (coil)

   Placed in the uterus and **reduce the motility** of the sperm.

3. **Sterilisation procedures**

   Males: **Vasectomy** by cutting/closing **Vas Deferens** in men preventing sperm leaving male body.

   Females: **Tubal ligation** by cutting/closing **oviducts** prevent egg implanting in uterus.

Chemical methods of Contraception

1. Oral contraceptive pill/mini pill

2. Emergency hormone contraceptive pill (morning after pill)

(i) **Oral contraceptive pill**

Contains **synthetic oestrogen & progesterone**

Mimics the natural negative feedback response preventing the release of FSH/LH f from the pituitary gland.

(ii) **Mini Pill**

Contains **progesterone only**

Causes **thickening of the cervical mucus** to prevent sperm entry.

(iii) **Emergency hormonal contraceptive pills** (morning after pill)

These drugs deliberately prevent or delay ovulation after unprotected sex.

Effective up to **72 hours** OR **120 hours** after dependent on type.

Screening

A variety of techniques can be used to monitor the health of the mother, developing fetus and baby

1. Post Natal Screening PKU

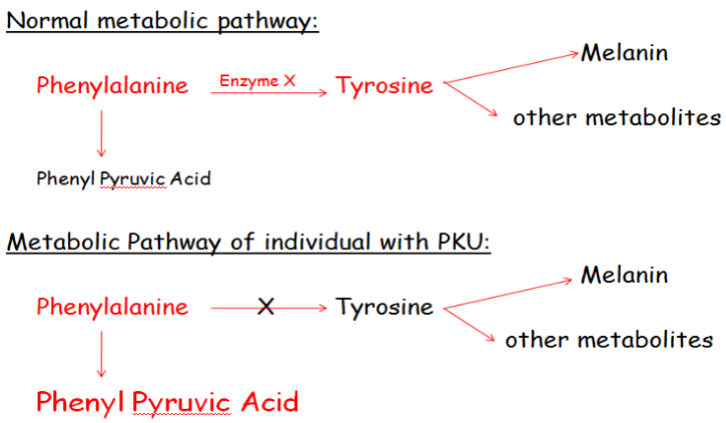
PKU is detected after birth by testing the baby's blood for high phenylalanine.

Cause

A **substitution mutation** means that the enzyme which converts phenylalanine to tyrosine is non-functional.

Phenyl alanine builds up which is toxic to the child.

Treatment
Low phenyl alanine diet



2. Antenatal screening

Antenatal screening identifies the risk of a disorder so that further tests and a prenatal diagnosis can be offered.

Types

1. **Ultrasound imaging**

   Pregnant women are given two ultrasound scans.

2. **Routine blood and urine tests**

   Carried out throughout pregnancy at specific times to monitor the concentrations of marker chemicals

Antenatal Screening

Ultrasound Scanning

1. Dating scan (8-14 weeks gestation)

   Used to determine pregnancy stage and due date

2. Anomaly scan (18—20 weeks)

   May detect serious physical abnormalities in the fetus

Blood/Urine Test Screening

Measuring a chemical at the wrong time could lead to a **false positive result**.

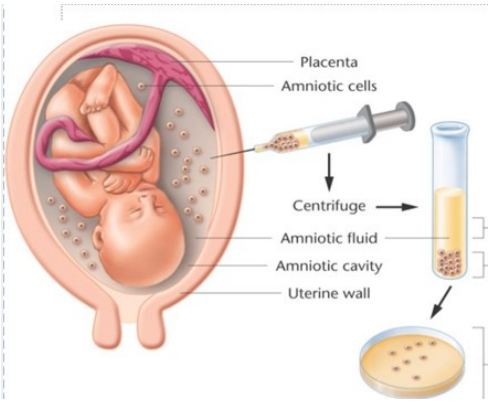
Diagnostic Testing

An **atypical** chemical concentration/scan can lead to diagnostic testing to determine if the fetus has a medical condition.

Considerations

When deciding to proceed with diagnostic tests consideration must be made of

1. The risk of miscarriage
2. Likely decision if test is positive.



Types of Diagnostic test

1. Amniocentesis
   Used at later stage of pregnancy
   Lower risk of miscarriage

2. CVS
   Used at earlier stage of pregnancy
   Higher risk of miscarriage

Karyotype

Cells from samples can be cultured to obtain sufficient cells to produce a karyotype to **diagnose** a range of conditions

Screening tests detect signs and symptoms of conditions/disorders.

Diagnostic testing is a definitive test.

Karyotype

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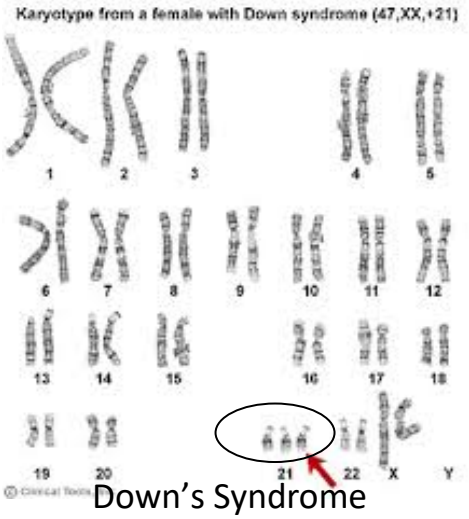
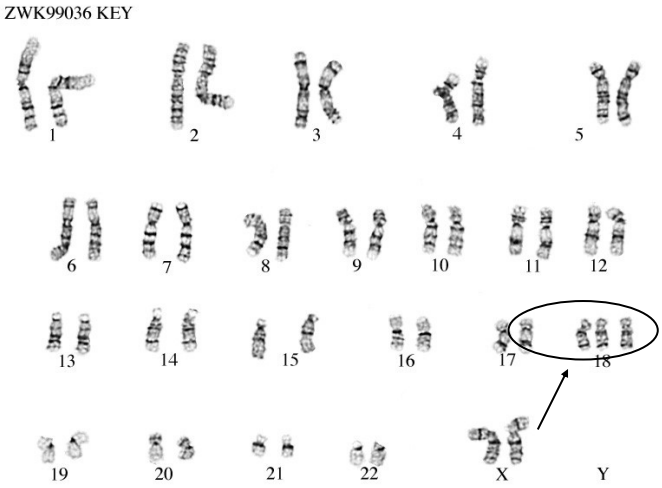
karyotype shows an individuals chromosomes arranged as **homologous pairs.**

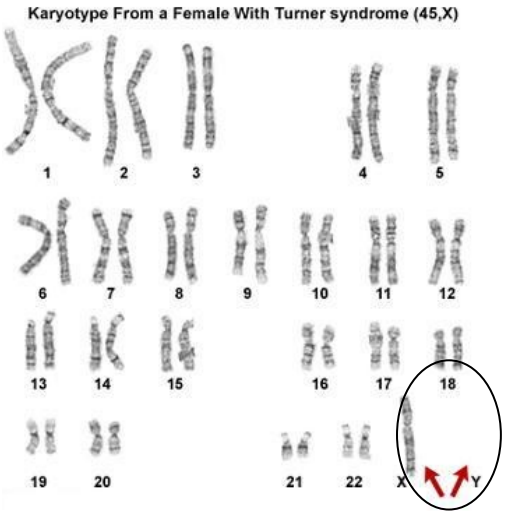
A



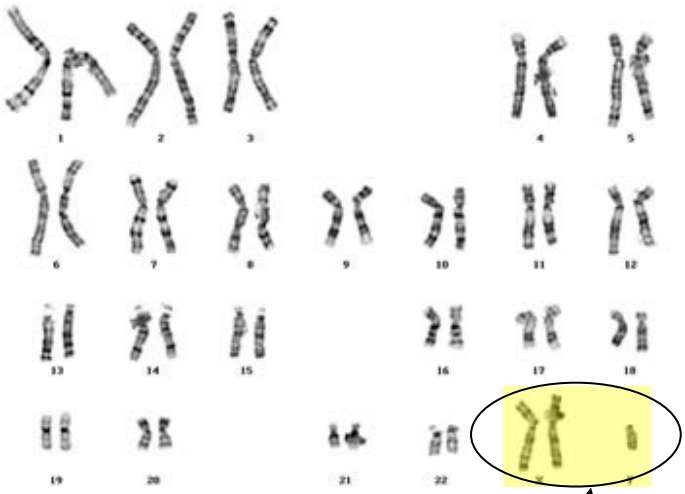
Down's Syndrome



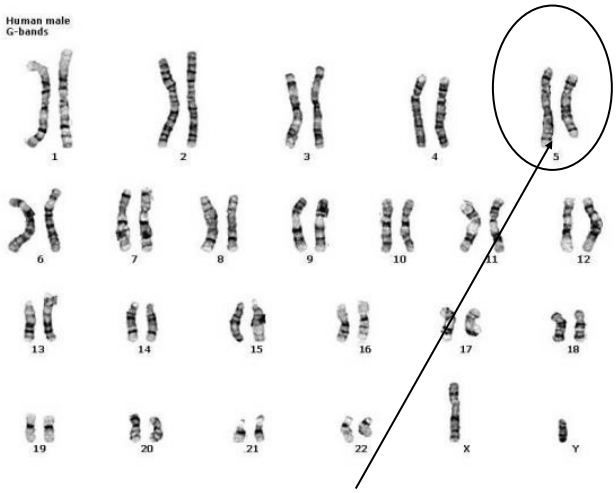
Edwards Syndrome



Turners Syndrome



Klinefelter's Syndrome



Cri-du-chat

For more information see Chapter 12.

Pedigree Charts (family trees)

Used to analyse patterns of inheritance in genetic screening.

Once the phenotype for a characteristic is known and a family tree is constructed, most of the genotypes can be determined.

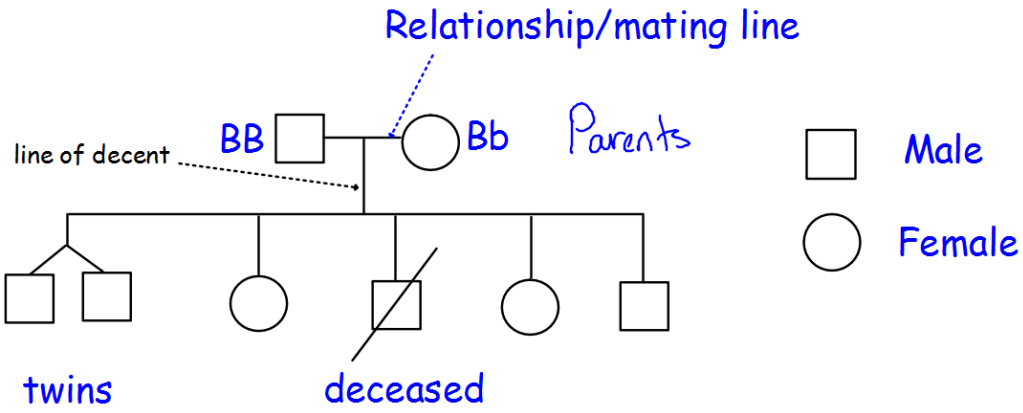
Genetic Screening

This information is used by genetic counsellors to advise parents of the possibility of passing on a genetic condition to their child (assessing the risk).

The four patterns of inheritance we will look at include:

1. *Autosomal recessive*
2. *Autosomal dominant*
3. *Autosomal incomplete dominance*
4. Sex linked recessive trait.

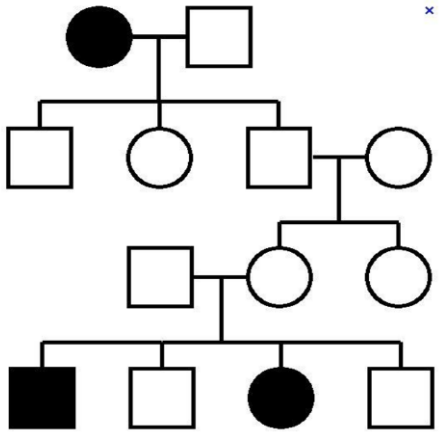
Pedigree Chart no-menclature and symbols



Pedigree Charts (family trees)

Autosomal Recessive

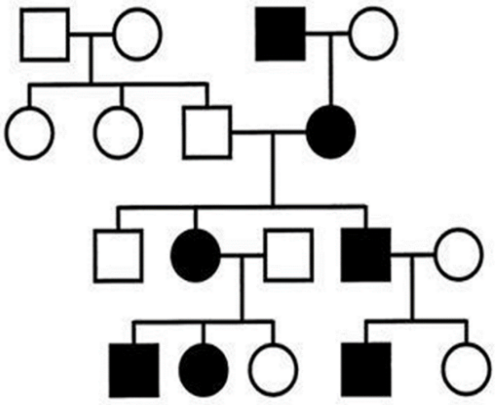
- Expressed relatively rarely & may skip generations.
- Males & females equal chance of being affected.
- All sufferers are homozygous recessive, so non-sufferers will be homozygous dominant or heterozygous.



e.g. cystic fibrosis

Autosomal Dominant

- Appears in every generation.
- Each sufferer has affected parent.
- Males & females equal chance of being affected.
- All non-sufferers are homozygous recessive

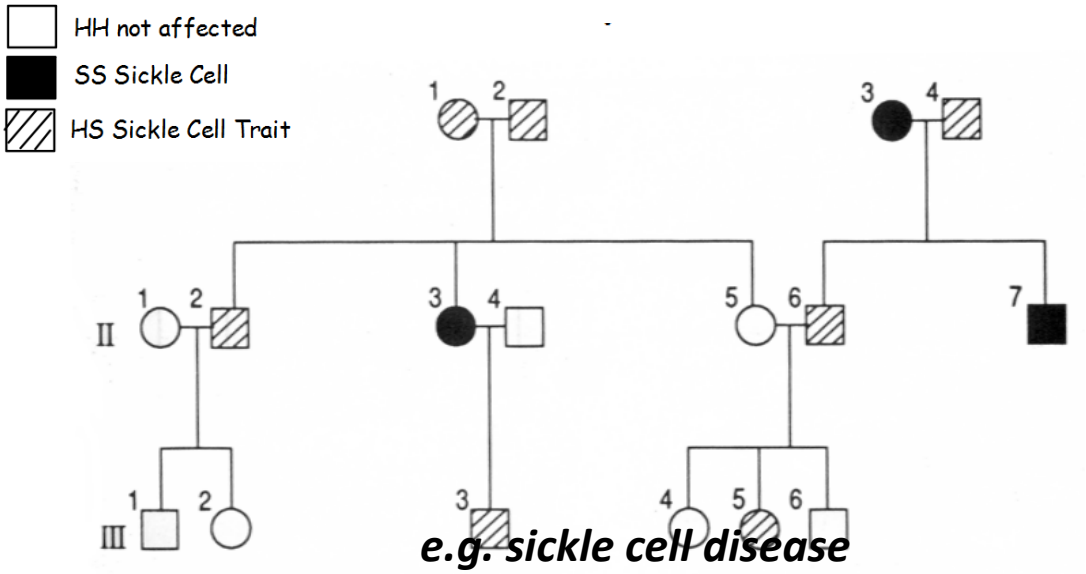


e.g. Huntington's chorea

Pedigree Charts (family trees)

- Incomplete Dominance

- The fully expressed form of the disorder occurs relatively rarely
- Partially expressed form occurs more frequently
- Males & females equal chance of being affected.
- Non sufferers homozygous for one incompletely dominant allele
- Sufferers of the fully expressed form of the disorder are homozygous for the other incompletely dominant allele
- Sufferers of the partly expressed form are heterozygous for the two alleles

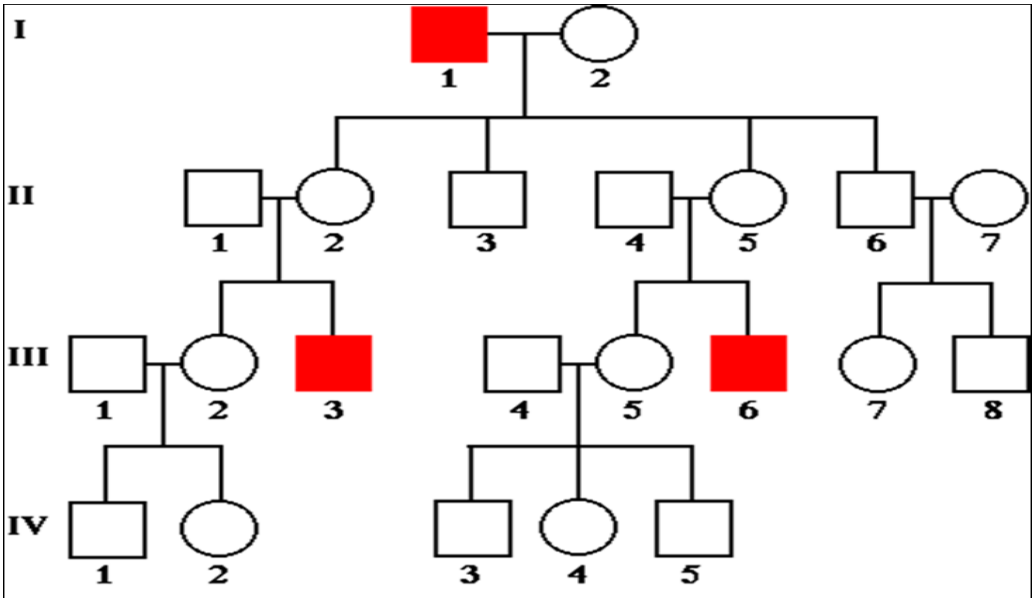


e.g. sickle cell disease

Pedigree Charts (family trees)

- Sex-linked Recessive

- Many more males are affected than females
- None of the sons of an affected male show the trait
- Some grandsons of an affected male show the trait
- Sufferers of the trait are homozygous recessive (normally male X^hY rarely female X^hX^h)
- Non sufferers are homozygous dominant X^HY X^HX^H or heterozygous carrier
- females X^HX^h



Male

Fe-

e.g. Duchenne muscular dystrophy

Blood Vessels

There are 3 main blood vessels:

1. Arteries — take blood away from heart
2. Veins — take blood Into heart
3. Capillaries — Site of gas exchange with tissues

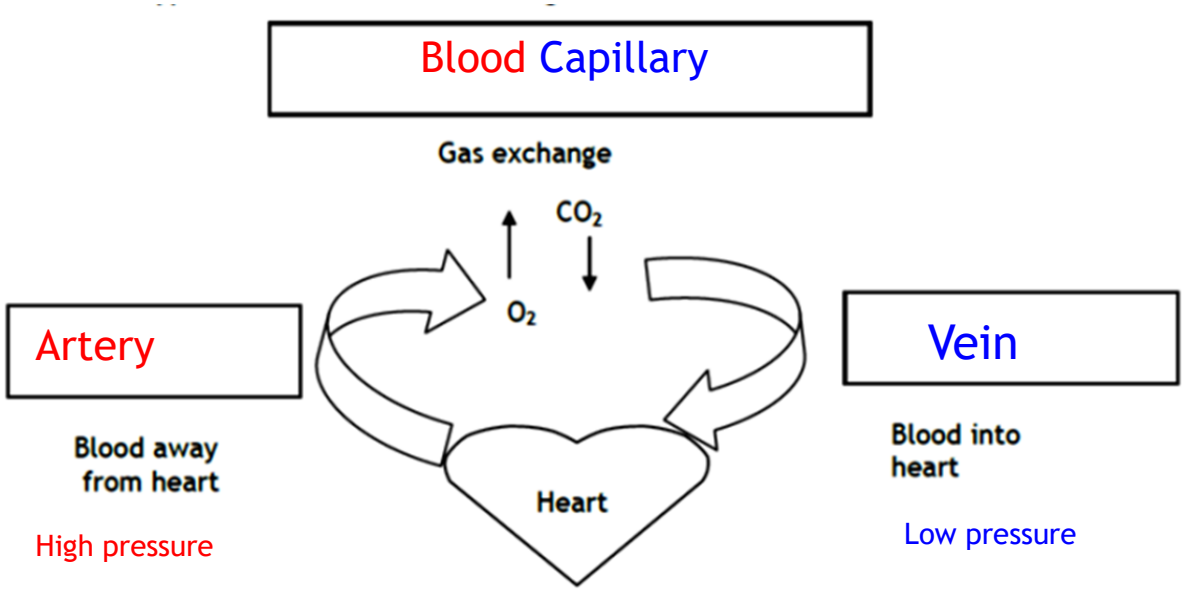
Direction of blood flow

Blood circulates from the heart through the arteries to the capillaries then to the veins and back to the heart.

arteries → capillaries → veins.

High pressure — Low pressure

There is a **decrease in blood pressure** as blood moves away from the heart.



Blood Vessel Structure

Arteries

A) Outer layer of connective tissue containing **elastic fibres**

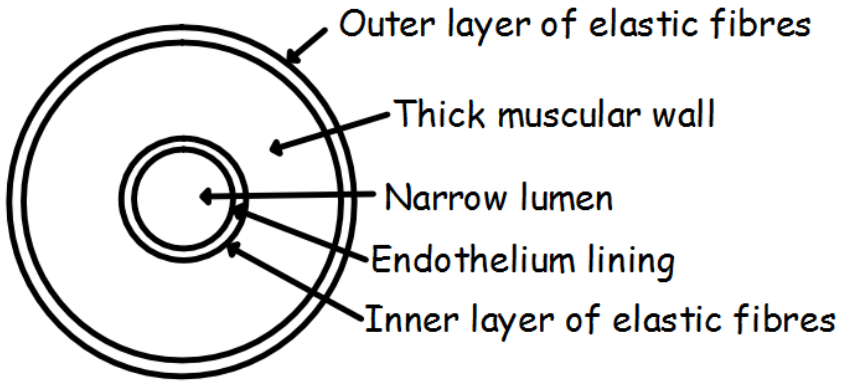
B) Middle layer containing smooth muscle with **more elastic fibres.**

C) Inner endothelial lining

This can become damaged by chronically high blood glucose

high levels of blood cholesterol can cause atheromas to deposit under the lining.

D) Narrower central lumen due to thicker smooth muscle (increased pressure)



Elastic nature of artery walls
Accommodates the **stretching** r& recoil required when blood surges through from the heart.

Smooth muscle of arteries cause

1. **Vasoconstriction**
   Smooth muscle contracts
   Lumen narrower
   Decreasedblood flow

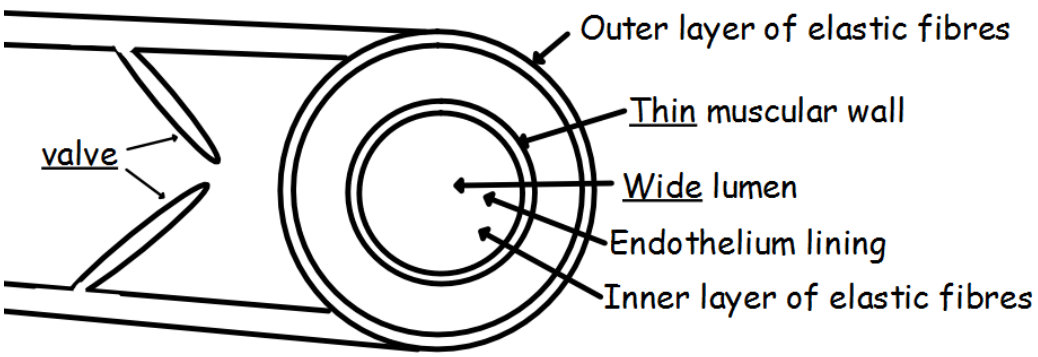
2. **Vasodilation**
   Smooth muscle relaxes
   Lumen wider
   Increased blood flow

2.Veins

Outer layer of connective tissue containing elastic fibres'.

Much **thinner smooth muscle** wall than arteries as blood flows at much lower pressure.

Valves prevents backflow. of blood

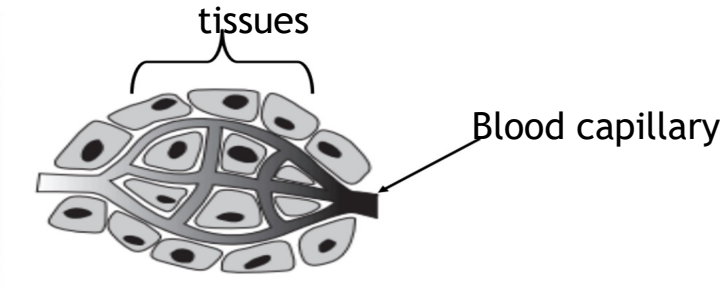


Blood Vessel Structure

Capillaries

Capillaries allow exchange of materials with tissues.

They are composed of epithelial cells that are one cell thick to allow easy diffusion of substances.



Tissue Fluid

Blood plasma enters the blood capillary from an artery containing **glucose, oxygen and plasma proteins.**

Pressure filtration

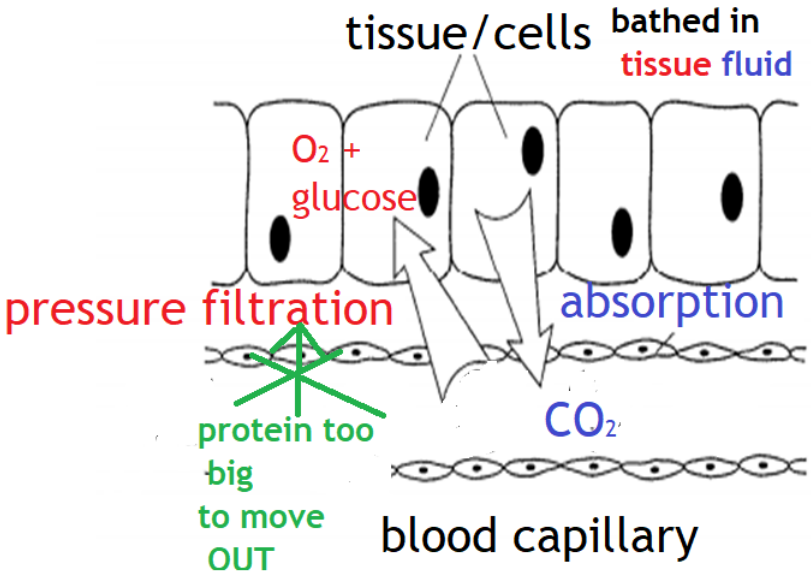
Blood plasma is **forced out** the blood capillary through the permeable walls of the blood capillary forming **tissue fluid**

All substances move out to bathe the tissues in tissue fluid **except plasma proteins** which are **too large** to fit through the gaps and are retained in the blood capillary

O_2 and glucose move from tissue fluid into tissues/cells for respiration

CO_2 the waste product of respiration is

1. Absorbed via the tissue fluid into blood capillary
2. **Excess tissue fluid** is absorbed into the **lymph**



Cardiac Output & Heart Structure

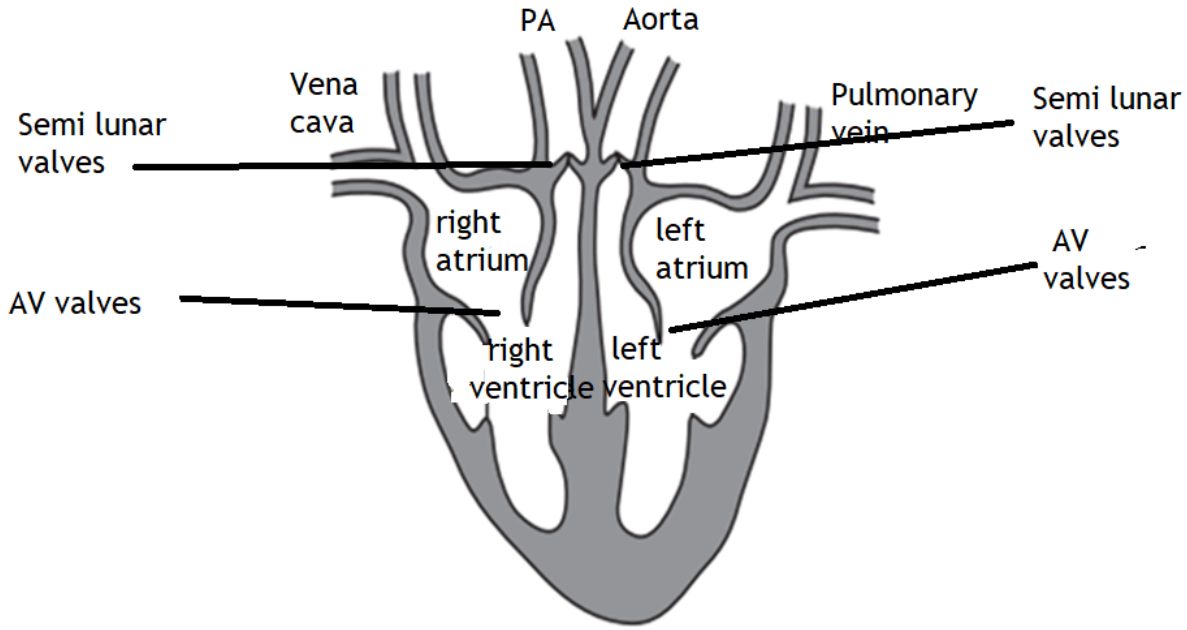
Cardiac Output

The volume of blood pumped through each ventricle per minute

CO= HR x SV

HR= number of heart beats per minute (pulse)
SV= volume of blood expelled by each ventricle contraction

Flow of Blood Through the Heart



1. Vena cava bring deoxygenated blood
2. Deoxygenated blood enters the right atrium
3. Deoxygenates blood enters the right ventricle
4. Pulmonary artery takes blood to the lungs
5. Pulmonary veins returns blood to the left atrium
6. Oxygenated blood enters the left ventricle
7. Oxygenated blood exits heart through the aorta (artery)

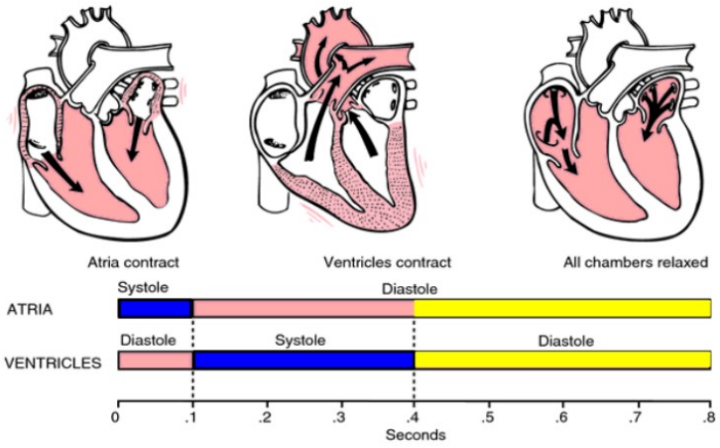
Cardiac Cycle

Cardiac cycle.

This consists of a pattern of relaxation (diastole) and contraction (systole) during one heart beat

Systole: contraction of the heart (Atrial first, then ventricular), blood forced out of chambers.

Diastole: relaxation of the heart, chambers fill with blood.



Diastole blood returning to the atria flows in to the ventricles.

AV valve open **SL valve shut**

↓

Atrial systole transfers blood through the atrio-ventricular AV valves to the ventricles.

↓

Ventricular systole pumps blood out the ventricle through the aorta and pulmonary artery.

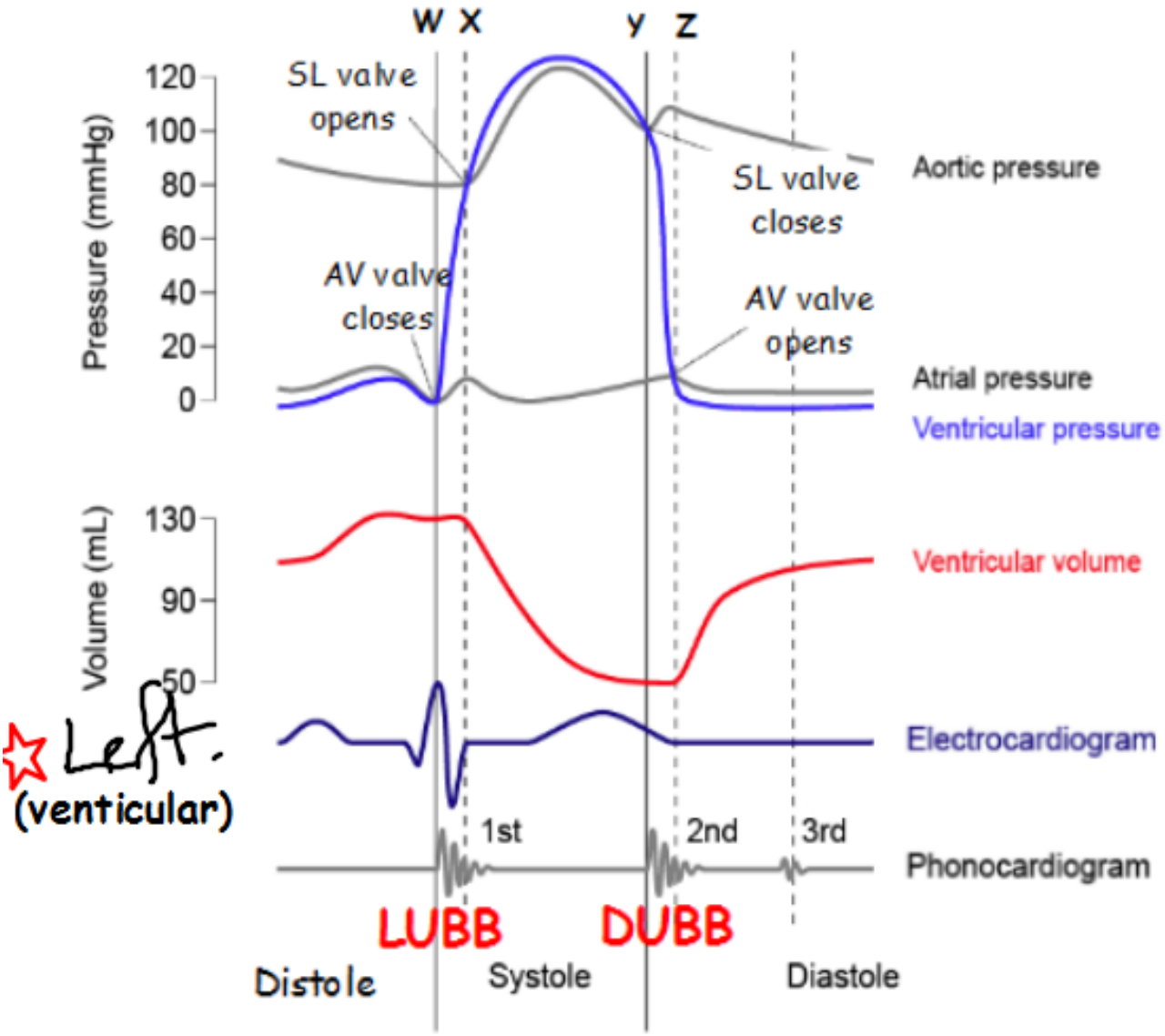
AV valve shut **SL valve open**

Valves

The opening and closing of the valves give the heart its familiar beating noise 'lub dub'with a stethoscope.

The higher blood pressure in arteries during diastole of heart (120mmHg vs 80mmHg) closes SL valve

Cardiac Cycle



W - AV valve closes as ventricular pressure is greater than atrial pres sure. This creates the 'LUBB' noise. This is the beginning of atrial diastole and ventricular systole.

X - SL valve is forced open as ventricular pressure is greater than atrial pressure forcing the blood through the artery.

Y - As the pressure falls the SL valve closes. Results in the 'DUPP' noise. Ventricle diastole begins.

Z - Ventricular pressure falls below the atrial pressure and the AV valve opens.

Cardiac Conducting System

Heart beat

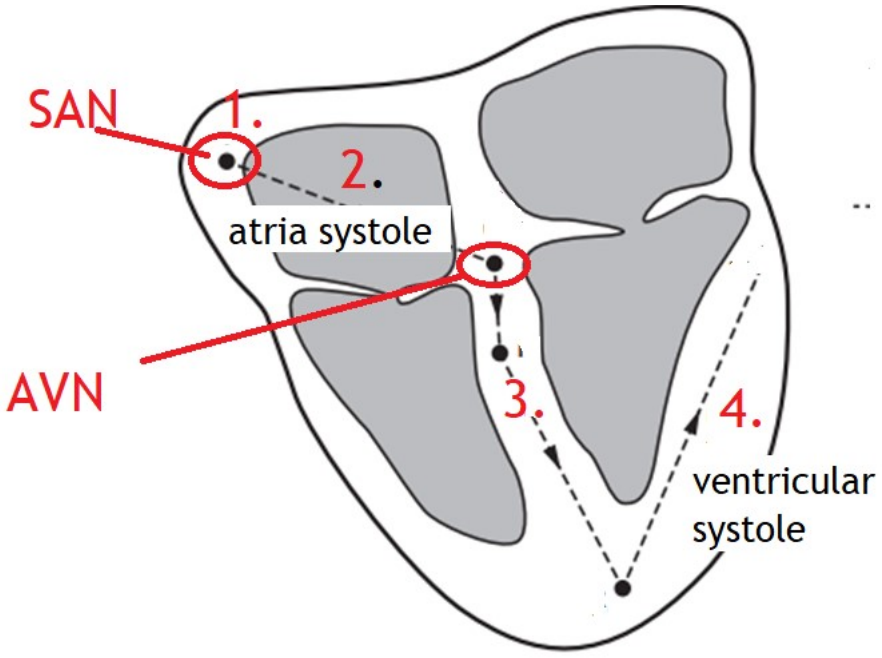
1.Originates in the heart itself via the **auto-rhythmic SAN (pacemaker) & the conducting system.**

2. Heart rate also controlled by ANS & hormones (adrenaline)

Cardiac Conducting System

The SAN is located in the wall of the **right atruim.**

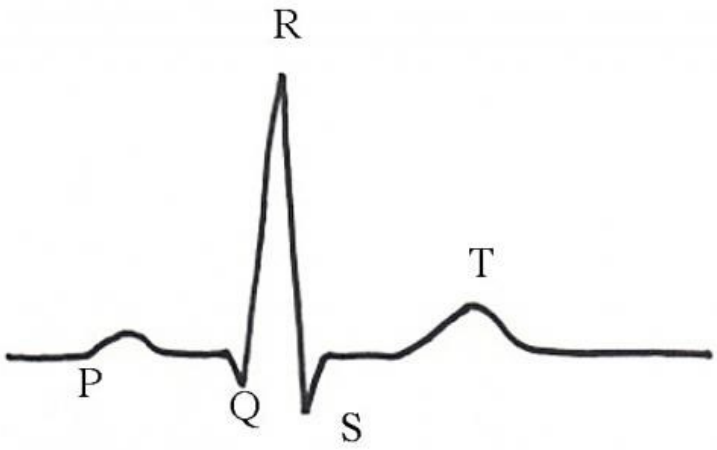
1. SAN **initiates the electrical impulse** which makes the **atria contract** at a certain rate (**atrial systole**).
2. This wave of excitement spreads to the **AVN** at the base of the atria.
3. Electrical impulses **pass down the bundle of conducting fibres** in the central wall of the heart.
4. The fibres then divide into the right and left ventricles to make the ventricles contract from bottom up (ventricular systole).



ECG

The electrical impulses generated by SAN/AVN are seen as electrical currents using an ECG.

Normal ECG pattern
Consists of **3 distinct waves** referred to as **P,Q,R,S and T.**

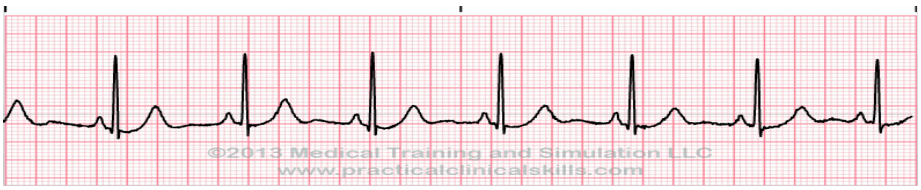


P - Atrial systole **QRS -** Ventricular systole **T - Diastole**

ECG

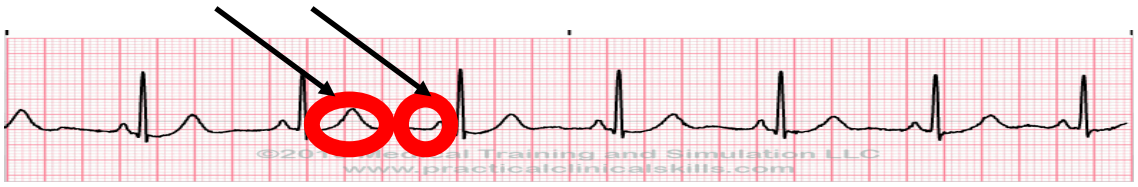
Normal ECG

Shows sinus rhythm. This is a repeat of the PQRS and T waves in a coordinated healthy way.



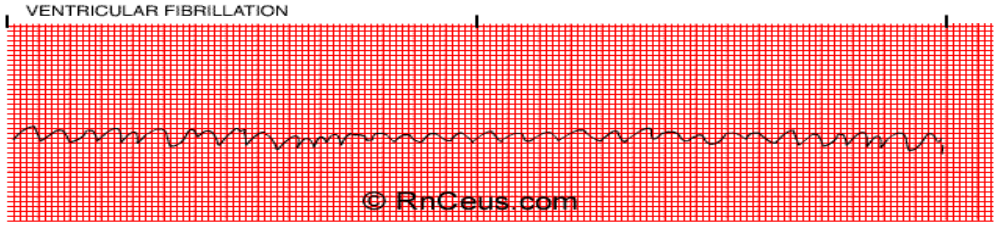
Abnormal ECG's Atrial Flutter -

Excited heart, rapid contraction in the atria .
Lots of P waves.in one heart beat.



Ventricular fibrillation

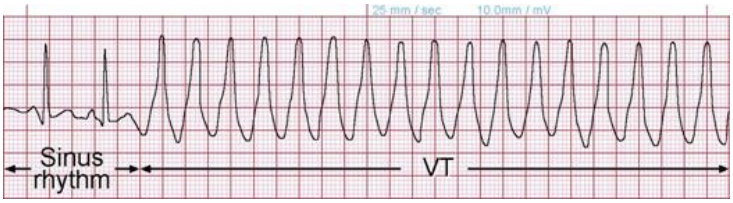
Different groups of muscle contracting at the same time,
loss of coordination. **Lethal** if not corrected. **Missing QRS waves.**



Ventricular tachycardia

Ventricles are contracting independently.
A pacemaker is required to return the heart beat to normal**. No P waves.**

Very fast heart beat, more than 100 beats per minute.



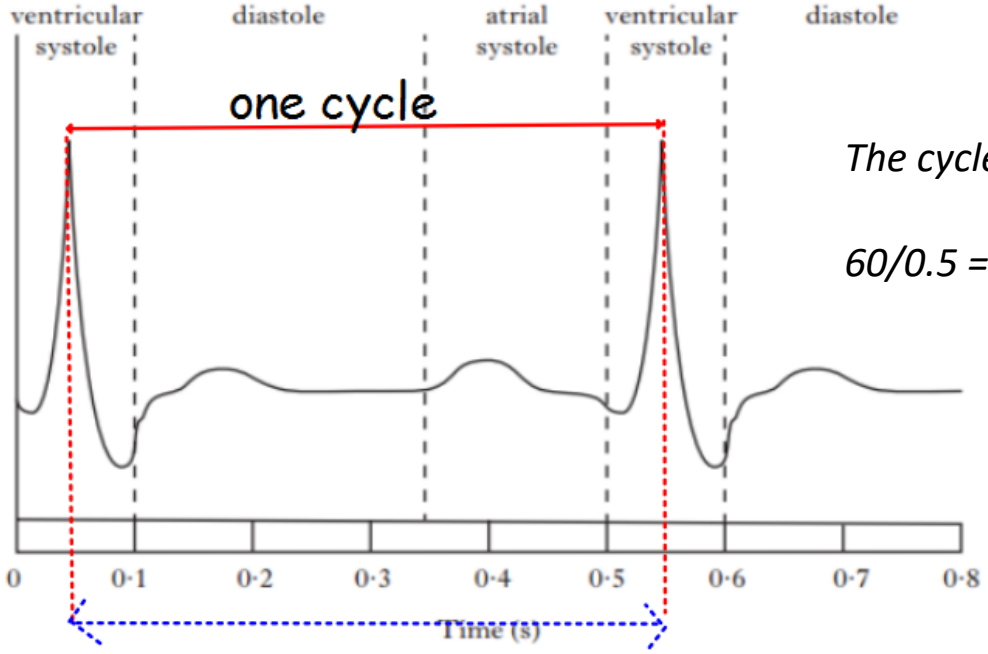
Calculating Heart rate

Calculating the Heart Rate

A person's heart rate can be calculated by counting the time between two separate peaks of ventricle systole then applying formula

60 seconds/ time for 1 cycle (time between peaks)

The diagram below shows an electrocardiogram (ECG) trace of an individual's heartbeat.



The cycle takes 0.5 secs.

60/0.5 = 120beats/min.

(*a*) Calculate the heart rate of this individual.

Example-

11. The average duration of diastole and systole in a hospital patient over a period of time were measured and are shown below.

diastole = 0.3 seconds
atrial systole = 0.1s
ventricular systole = 0.2 seconds

What was the average heart rate of this individual over the period of time?

A 60 beats per minute
B 72 beats per minute
C 100 beats per minute
D 120 beats per minute

Answer—one cardiac cycle is 0.3+0.1+0.2 = 0.6.
60secs/0.6 = 100 beats per mins

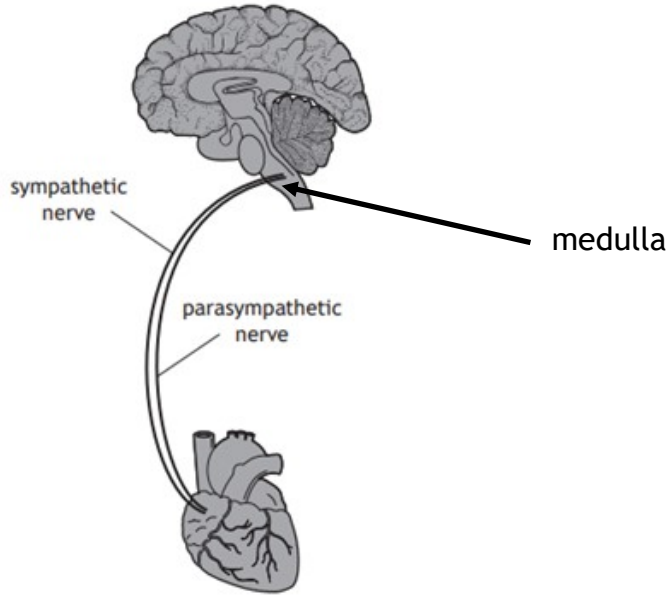
Control of cardiac system

Nervous Control of heart rate

The **medulla (brain**) regulates the rate of auto rhythmic **SAN** through the **antagonistic action** of the two branches of the autonomic nervous system (ANS).

- Sympathetic nerves (fight or flight response)
  Releases noradrenaline and increases heart rate
- Parasympathetic nerves (rest or digest)
  Releases acetylcholine and decrease heart rate

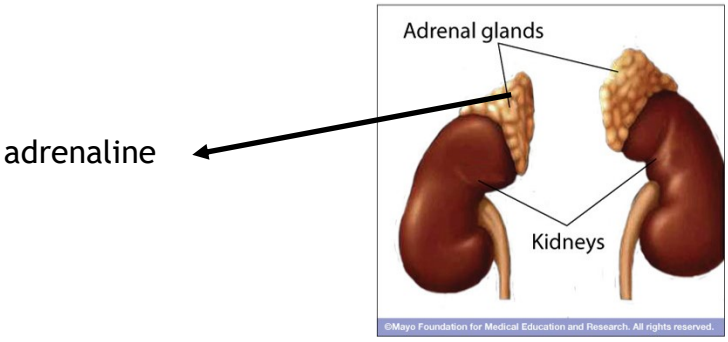
Heat rate at any one point in time is determined by which of the above nerves exerts the greatest control at that point.



Hormonal Control of Heart rate

Hormones released during **fight or flight** response during **stress /physical activity** can act on the **adrenal glands** to cause **adrenaline** to be released into blood.

This stimulates the SAN to **increase heart rate**.



Blood Pressure

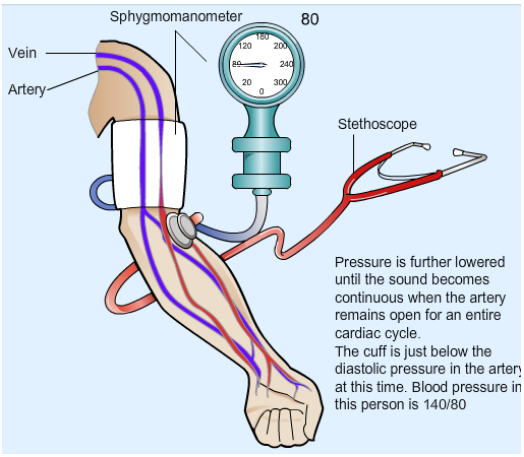
A healthy young adult should have a typical reading of **120/80mmHg.**

Sphygmomanometer.

An inflatable cuff **stops the flow of blood** and deflates gradually.

Systolic Pressure (120mmHg)

The blood pressure starts to flow (pulse)



Diastolic Pressure (80 mmHg)

The blood flows freely through the artery

(pulse not detected)

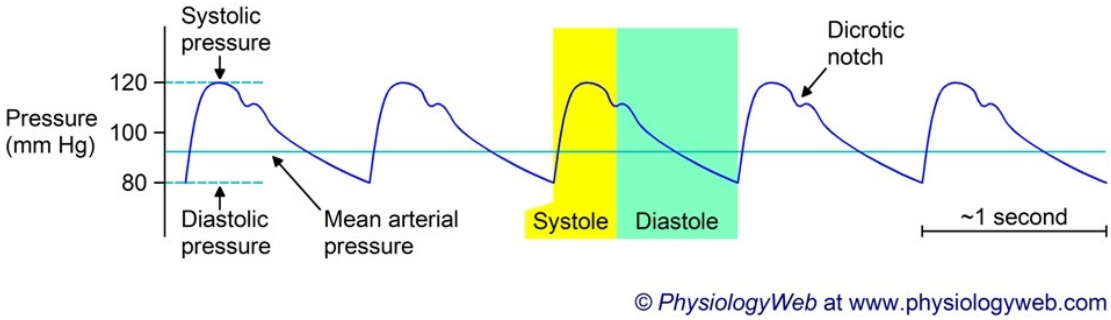
Hypertension

Hypertension is high BP.

This is any value over **140/90mm Hg.**

This is a major risk factor in many diseases such as stroke and coronary heart disease

Blood Pressure Graph.



Blood pressure #changes

Cardio vascular Disease (CVD)

Any disease affecting the heart, blood vessels or blood pressure.

Examples

1. Angina 2. Heart attack 3. Stroke 4.PVD

Cause: atherosclerosis.

<u>Atherosclerosis</u>

1. Atheroma forms

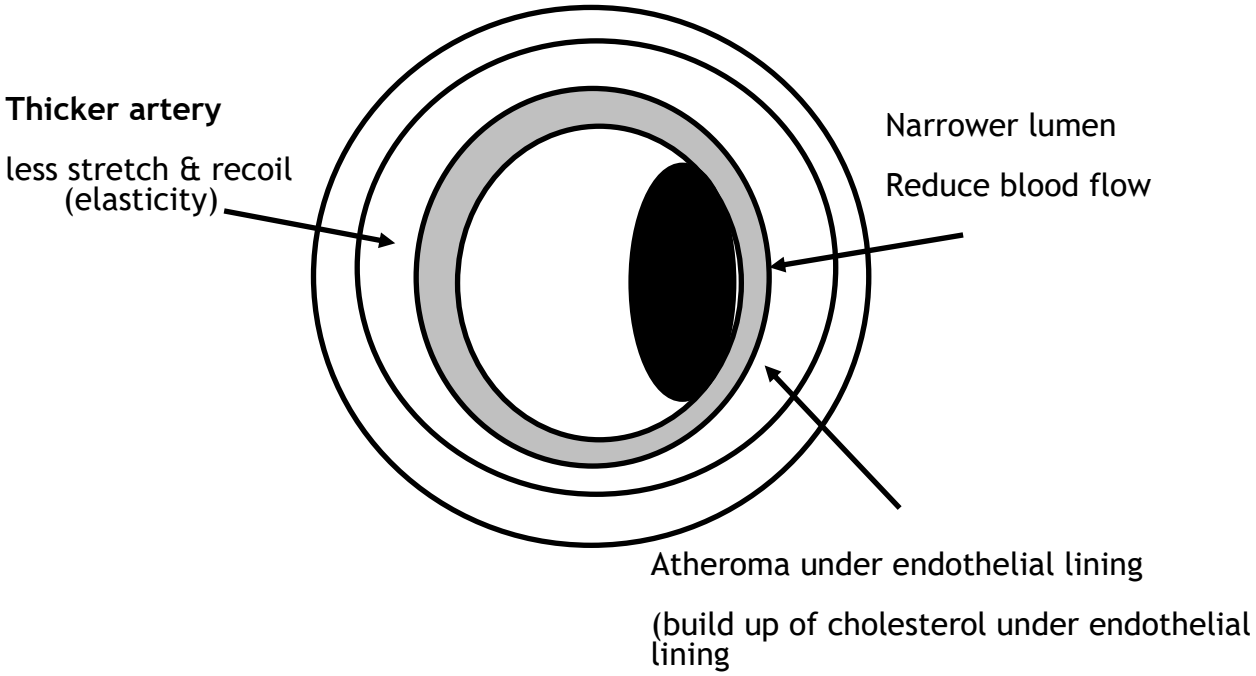
   Accumulation of fatty material (cholesterol, fibrous material and calcium) beneath the **endothelium** of the artery

2. Atheroma grows

   Thickening of the artery & narrowing lumen diameter

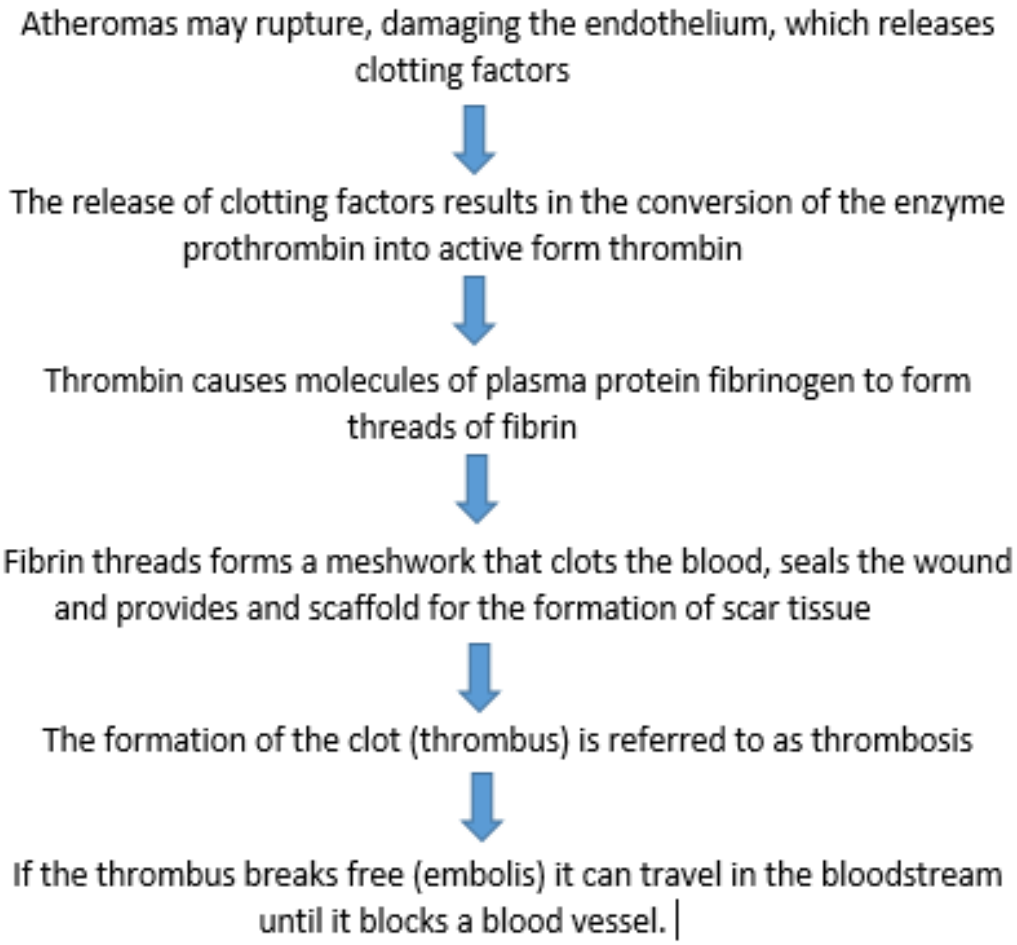
- Thicke walls = loss of elasticity (no stretch/recoil)
- Narrower lumen = Reduced blood flow

3. High blood pressure due to loss of elasticity/narrower lumen.



Thrombosis

The steps involved in the formation of a thrombus (clot) are:



Consequences of thrombosis

1. **Myocardial Infarction/heart attack**

   Blockage of coronary artery by thrombus/clot

   Lack of oxygen delivered to heart cells causing **death of heart tissue**

2. **Stroke**

   Blockage by arteries in the brain caused by a thrombus/embolus

   Lack of oxygen delivered to brain cells causing **death of heart tissue**

Peripheral Vascular Disease

PVD

Narrowing of the arteries due to **atherosclerosis**.

Most common in the **leg arteries** but anywhere apart from brain/heart.

Pain is experienced in the (leg) **muscles** due to a **limited supply of oxygen**.

DVT

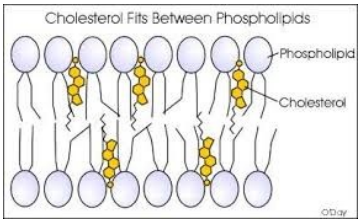
A blood clot/thrombus that forms in a deep vein, most commonly in the leg.

Pulmonary embolism

Thrombus in deep vein of leg breaks off and result in a pulmonary embolism in the lungs.

Cholesterol

Function of cholesterol



1. Make sex hormones
   (testosterone & oestrogen/progesterone)
2. Type of lipid in cell membrane

Production of Cholesterol

All cells produce cholesterol but **25%** of all production takes place in the **liver.**

Lipoproteins

Proteins in blood that transport cholesterol to/from liver.

1. HDL

Transports **excess** cholesterol from the body cells ⟶ liver for **elimination.**

This **prevents accumulation** of cholesterol in the blood.

2. LDL

Transports cholesterol from liver ⟶ body cells.

Most cells have LDL receptors that take LDL into the cell where it **releases cholesterol.**

Negative Feedback

1.Once a cell has sufficient cholesterol, it inhibits the synthesis of new LDL receptors

2. LDL cannot enter cell & stays in the blood

3. It may deposit **cholesterol** under the **endothelial** lining of **arteries** forming **atheromas**

High Blood Cholesterol

A diet **high** in **saturated fats/ cholesterol,** increases **risk of atherosclerosis**

Treatment

1. **Regular physical activity**

   Increases HDL: LDL ratio Or HDL levels
   This **lowers** blood cholesterol thereby **reducing atherosclerosis**

2. **Dietary changes**
   Reduce the levels of total fat in the diet and to replace saturated with unsaturated fats

3. **Statin drugs**
   Reduce blood cholesterol by inhibting the synthesis of cholesterol by liver cells

Familial Hypercholesterolemia (FH)

Familial Hypercholesterolemia

Inherited **dominant allele** that **increases** an individuals chance of having **high blood cholesterol**

Cause of high cholesterol

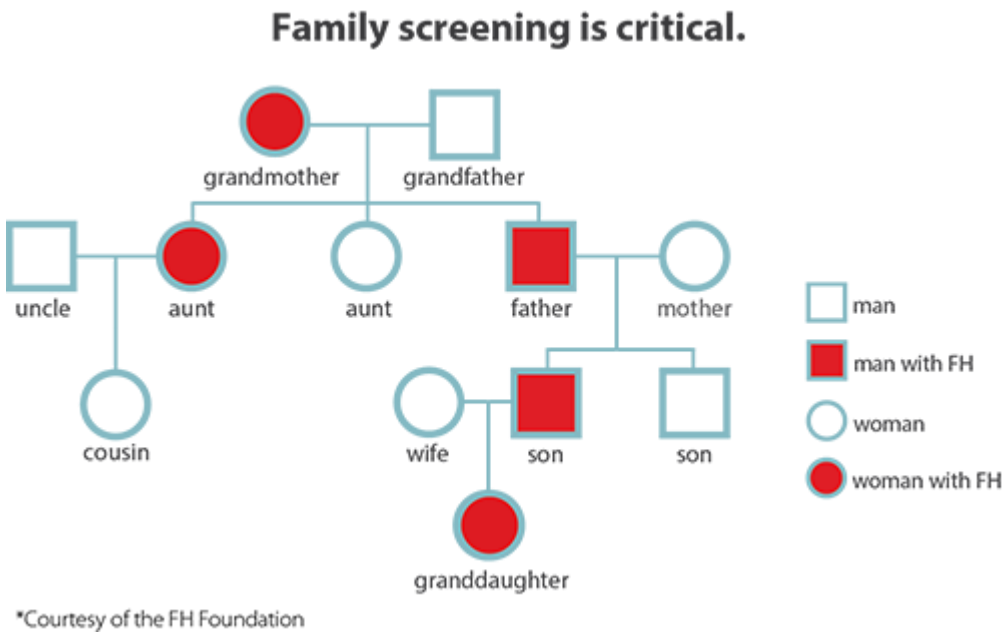
1. **Reduction** in the number of **LDL receptors**
2. **Altered structure of** LDL receptor

Diagnosis

Genetic testing can be done to determine if the dominant FH gene has been inherited.

Treatment

1. Lifestyle modification (reduced fat intake & regular physical activity
2. Statin drugs (prevent cholesterol production)



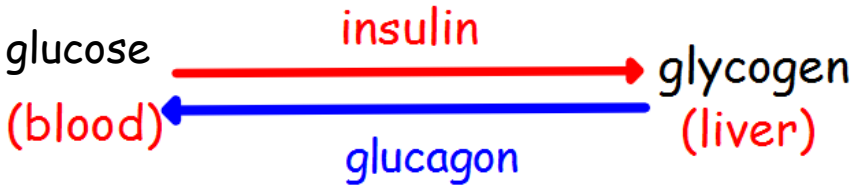
Blood Glucose

A **constant supply of glucose** is needed by ALL cells for **respiration.**

However **chronic high levels of glucose** can **damage** blood vessels

It is important that the concentration of glucose in the blood is maintained at a constant level via two hormones released by pancreas

Blood Glucose too High	Blood Glucose too Low
Detected by pancreatic receptors	Detected by pancreatic receptors
Insulin produced	Glucagon produced
Converts glucose into glycogen in liver	Converts glycogen in liver back into glucose
Lowers blood glucose	Lowers blood glucose



In cases of fight/flight/exercise

The body needs additional supplies of glucose to provide energy quickly.

The **adrenal glands** secrete **Adrenaline** into the bloodstream.

Chronic Elevation of glucose

Leads to the endothelium cells **taking in more glucose** than normal, damaging the blood vessels.

Atherosclerosis may develop leading to

1. cardiovascular disease
2. Stroke
3. peripheral vascular disease.

Small blood vessels damaged by elevated glucose levels may ALSO result in micro vascular disease

1. haemorrhage of blood vessels in the retina
2. renal failure
3. peripheral nerve dysfunction

Diabetes

Types of Diabetes

Type 1 Diabetes

Unable to produce insulin which usually begins in **childhood**

Treatment: regular insulin injections/tablets

Type 2 Diabetes

Cells are **less sensitive/resistant to insulin** due to a **decrease** in the number of insulin receptors at the liver which tends to **develops later in life.**

Obesity is a risk factor for type 2 diabetes**.**

Diabetes Testing

1. **Urine test**

   Glucose present in urine often indicates diabetes. Further testing needed.

   The kidneys remove some of the excess blood glucose that normally is stored as glycogen in the liver by insulin production.

   This removal by kidneys results in glucose appearing in the urine of diabetics

2. **Glucose Tolerance Test**

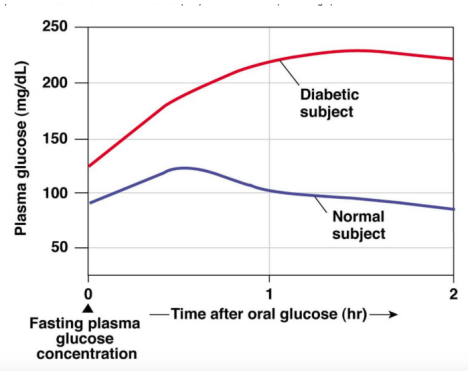
   This test formally diagnoses diabetes

Stage

1. Initial blood glucose concentrations measured after **fasting.**
2. The individual drinks a glucose solution
3. Changes in blood glucose concentration are measured for **≥ 2 hours**

The glucose concentration of the diabetic person:

1. Starts at a higher concentration
2. Rises more quickly
3. Reaches a higher maximum level.



Obesity

Characterised by **excess body fa**t: lean body tissue (muscle).

Causes

1. **high fat diets**
2. **decrease** in physical **activity.**

Body Mass Index (BMI)

BMI ≥ 30 indicates obesity.

$$BMI = \frac{\text{body mass (kg)}}{\text{height}^2 \text{ (m)}}$$

Problem with BMI Measurement

Individuals with **low body fat** but a large bone mass or muscle bulk can be **wrongly identified** as obese. *e.g. – body builders/rugby players.*

Obesity & Health

Obesity may **impair health** as major **risk factor** for **CVD/ type 2 diabetes.**

Treatment

1. **Diet (energy intake)**

- Limit fats as they a high calorific value per gram
- Limit free sugars as no metabolic energy expended in their digestion.

2. **Exercise**

- Preserve lean muscle tissue
- increases energy expenditure

Exercise lowers risk of CVD by

1. keeping weight under control
2. minimising stress
3. reducing hypertension
4. increasing HDL: LDL ratio