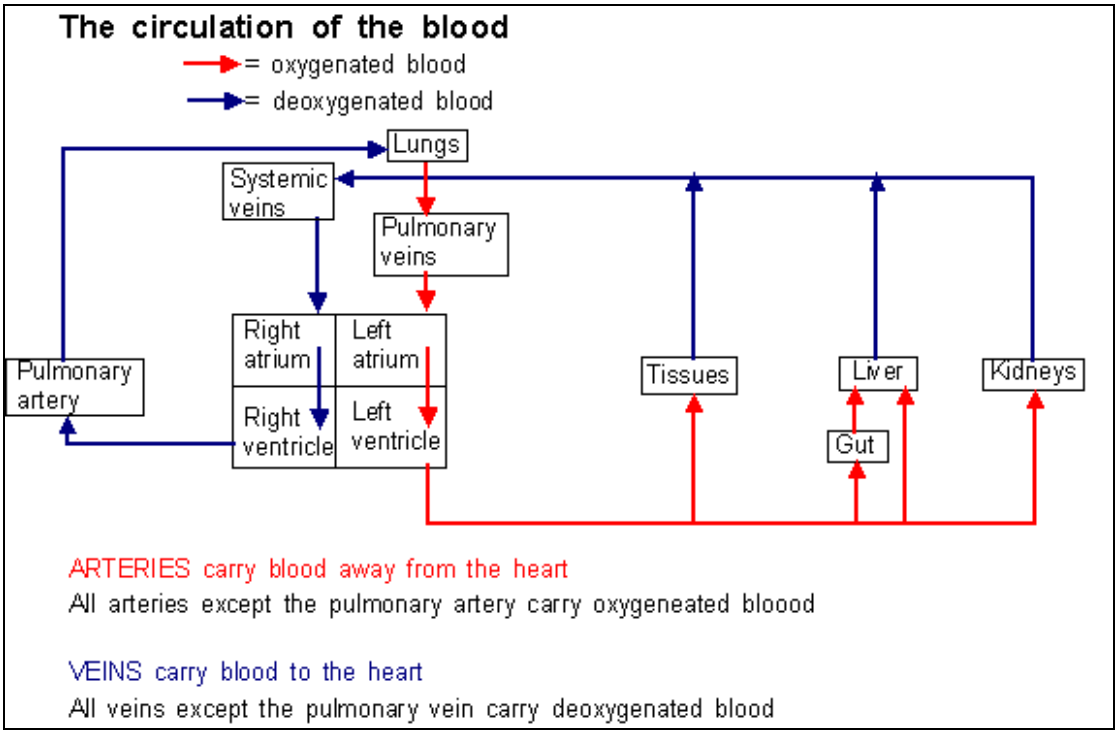
THE CIRCULATORY SYSTEMS

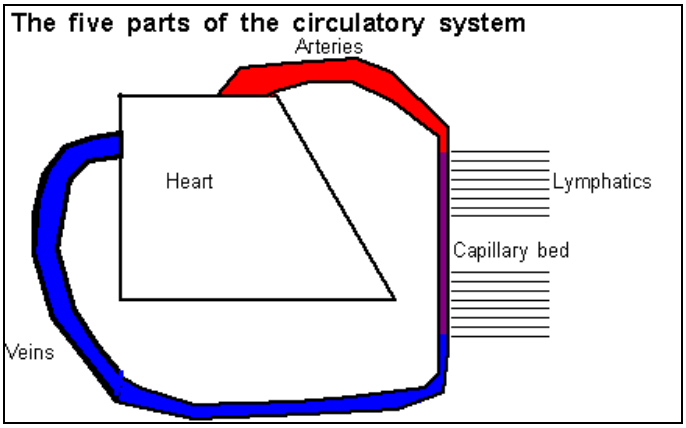
The circulatory systems of the body

- Transport fluids, respiratory gases, nutrients, wastes, hormones, parts of the immune system, and heat
- Help maintain the internal environment
- Can repair leaks in themselves
- Can divert blood to areas of need

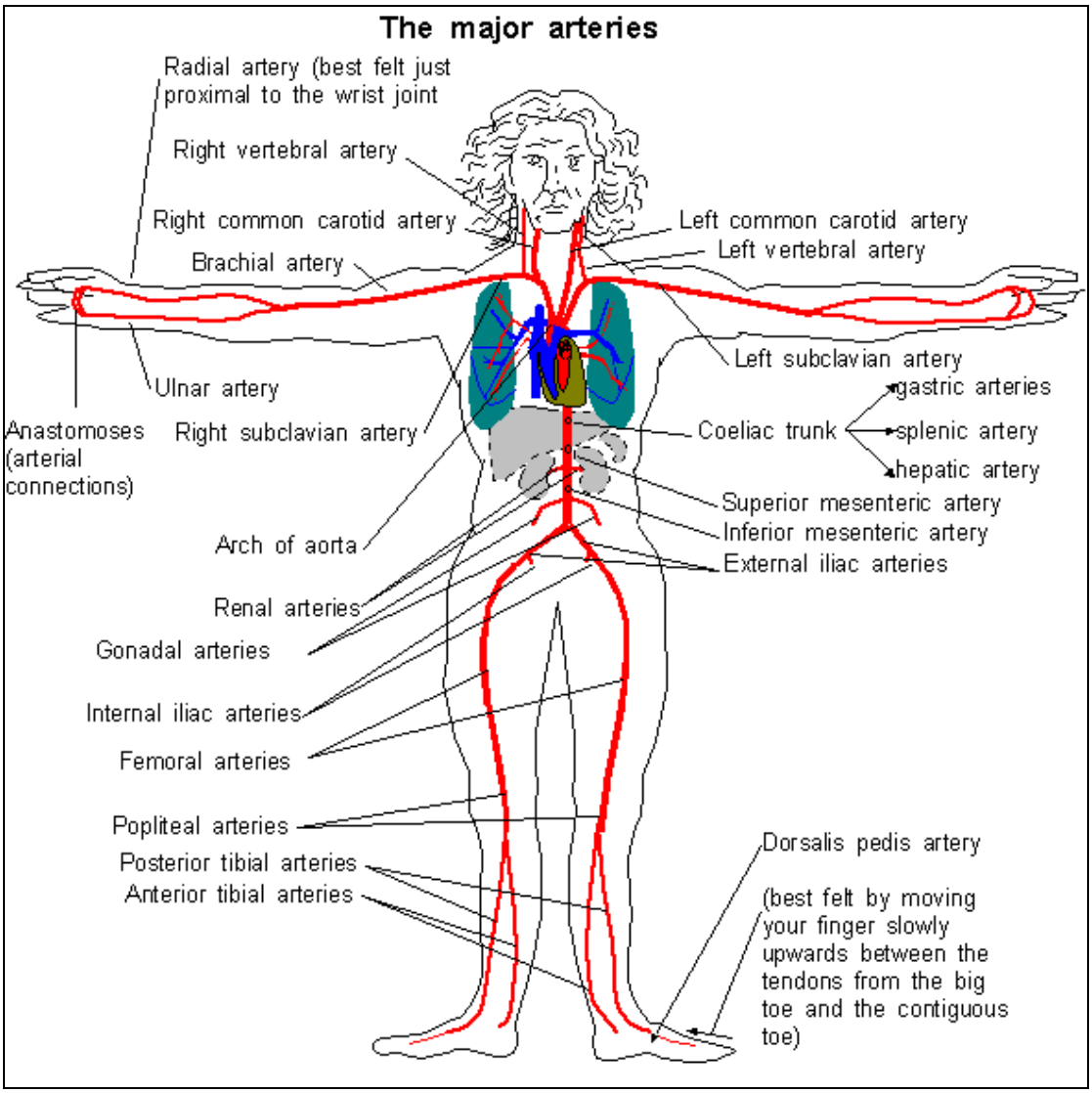


The circulatory system has five components.

1. Arteries, which apart from coronary arteries, transmit blood away from the heart) contain about 1,000mls of blood, 2. veins (which transmit blood to the heart) contain a variable amount of blood, 3. capillaries contain about 4,000mls of blood and 4. the heart contains about 200mls of blood. The normal total blood volume is about 4-6 litres.

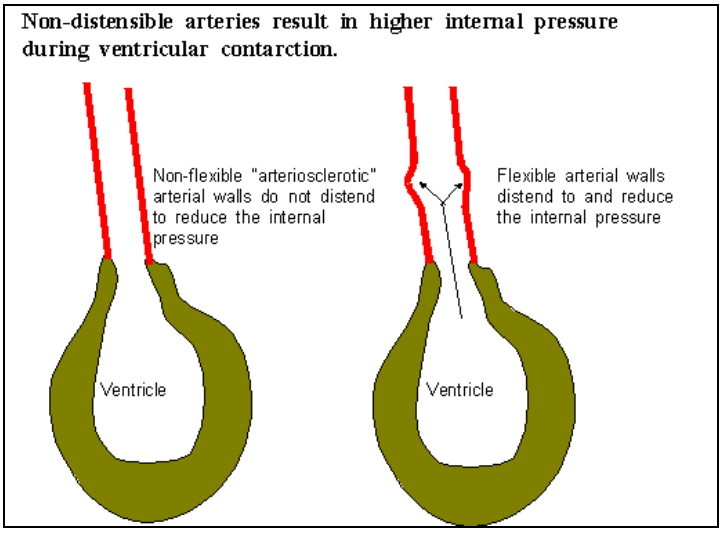


THE ARTERIES



Arteries are usually relatively thick walled, elastic, and muscular (the coronary arteries are an exception to this definition as they carry blood to the heart). The large systemic arteries are distensible and thus can smoothen out pressure changes delivered by the left ventricle and ensure constant, albeit jerky, blood flow. The pulsation that can be felt by palpation of arteries is a pressure wave and (in theory) would be felt even if there were no blood flow through the palpated artery. Pressure waves travel faster along wide arteries, explaining why the femoral pulses precedes the radial pulses although they are approximately equidistant from the heart.

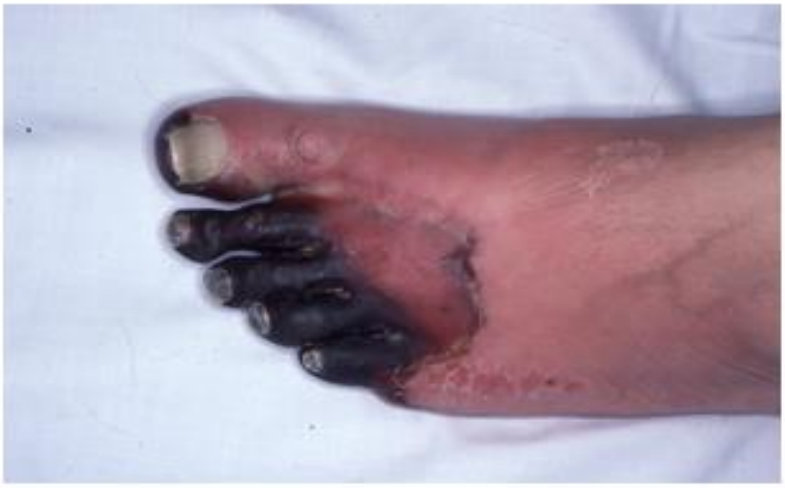
If the major arteries are stiff (arteriosclerotic) the blood pressure after ventricular contraction (systole) tends to be higher because of the lack of distension.



Peripheral resistance

Peripheral resistance mostly depends on the radius of the arterioles (=minute branches of the arteries). Sympathetic vasoconstrictor tone is derived from the vasomotor centre in the brain medulla. Increase in vasomotor tone leads to arterial vasoconstriction and causes a rise in arterial blood pressure (if cardiac output remains constant).

If there is rapid onset of arterial blockage the areas supplied become painful, pale, cold, anaesthetic (lost sensation) and gangrenous if the obstruction is not rapidly relieved.



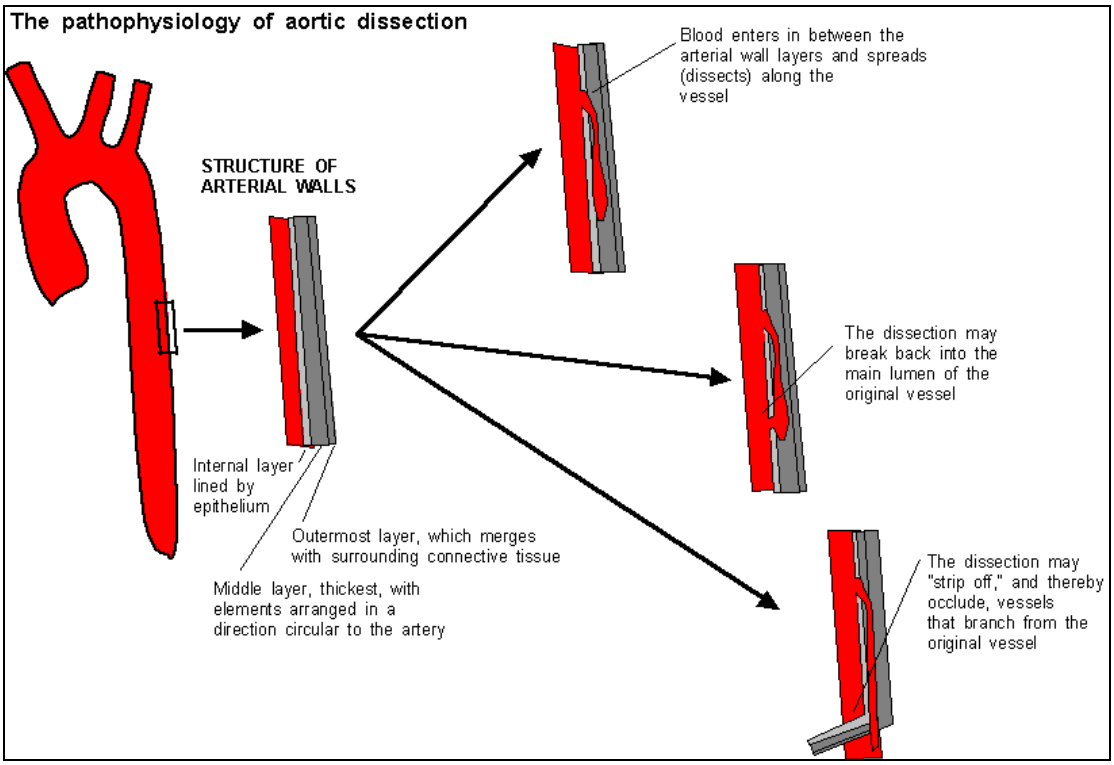
Arterial disease may be associated with smoking, diabetes mellitus, hypertension, some inherited conditions including high blood fat levels and inflammatory diseases of arteries (arteritis)

Atheroma (derived from a Greek word meaning porridge) furs up arteries, usually at sites of vessel wall stress, particularly in the abdominal aorta and its medium sized branches. Atheromatous plaques form which comprise a fat rich necrotic central area, surrounded by muscle cells and fibrous tissue on which platelet thrombi (clots) can develop.

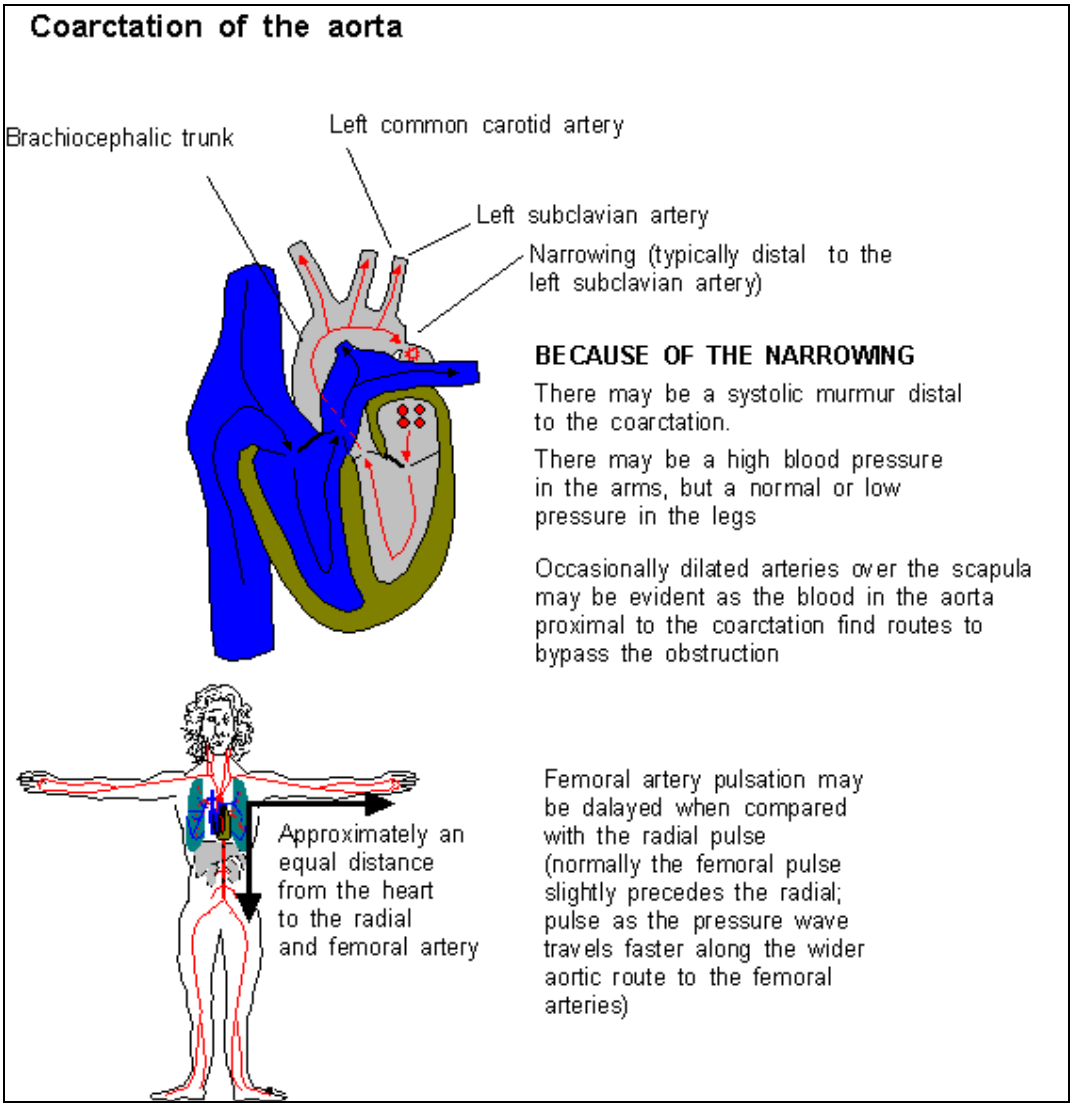
Ischaemic (=locally and temporarily suffering from a lack of blood) tissues are often painful because of accumulation of anoxic metabolites. Death of heart muscle (known variously as myocardial infarction, heart attack, coronary thrombosis) occurs when there is severe local restriction in blood flow through the coronary arteries.

Principles underlying some arterial problems

Aortic dissection occurs when blood forces its way between the layers of the arterial wall.



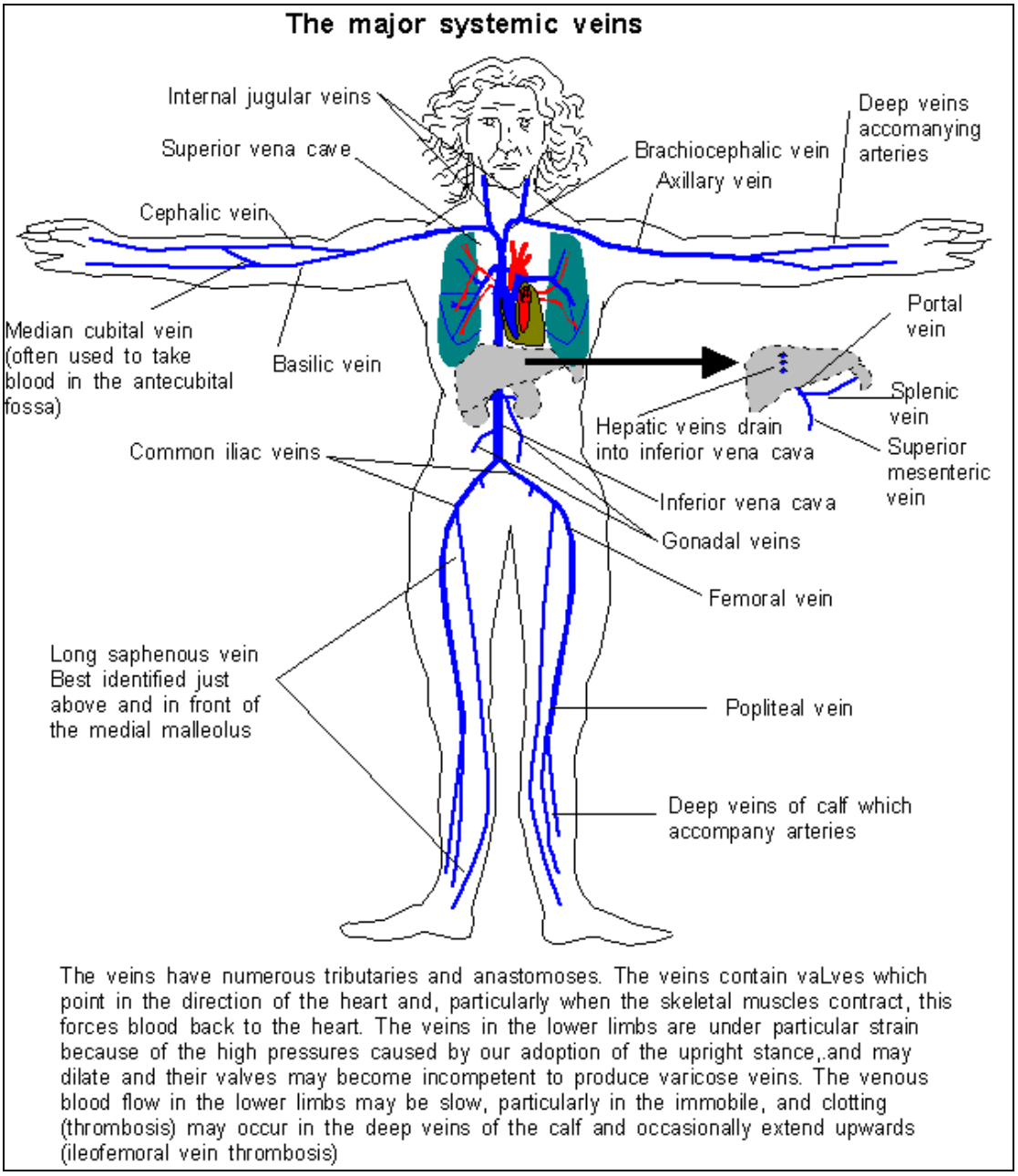
Coarctation of the aorta is a focal narrowing of the aorta which tends to occur between the left subclavian artery and the ductus arteriosus. Coarctation may be associated with aortic valve abnormalities and/or weakness of the aortic wall. The pulse amplitude beyond the narrowing may be diminished, the blood pressure may be lower in the lower limbs, and the femoral pulse may be delayed when compared with the radial pulse (normally the pressure wave travels faster along the wider aortic route to the femoral arteries) and is usually felt first. There may be a murmur, caused by abnormal blood flow, distal to the coarctation,



Raynaud's disease is a specific type of vasospasm that occurs in relation to cold or emotion. The extremities (usually the hands) become white (lack of blood caused by vasospasm), then cyanosed (because of deoxygenation of what blood there is) and then a bright red flush develops once vasospasm remits.

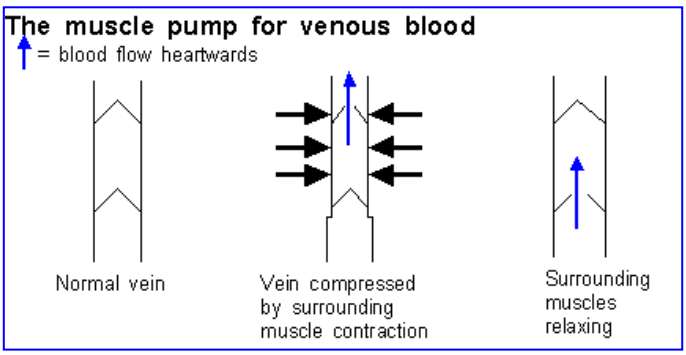
Thrombosis (=a clot developing in situ) may occur in veins or, more rarely on atheromatous arteries.
An embolism (=a wedge) is the transfer of an intravascular mass (usually a blood clot) from its point of origin to a distant site where it causes obstruction. A pulmonary embolism is impaction of a blood clot (formed as a thrombus, usually in the leg veins) in the lungs. Although the clot originates and travels to the right side of the heart in systemic **veins**, it then travels to its site of impaction in the lungs in the pulmonary **arteries**.

THE VEINS



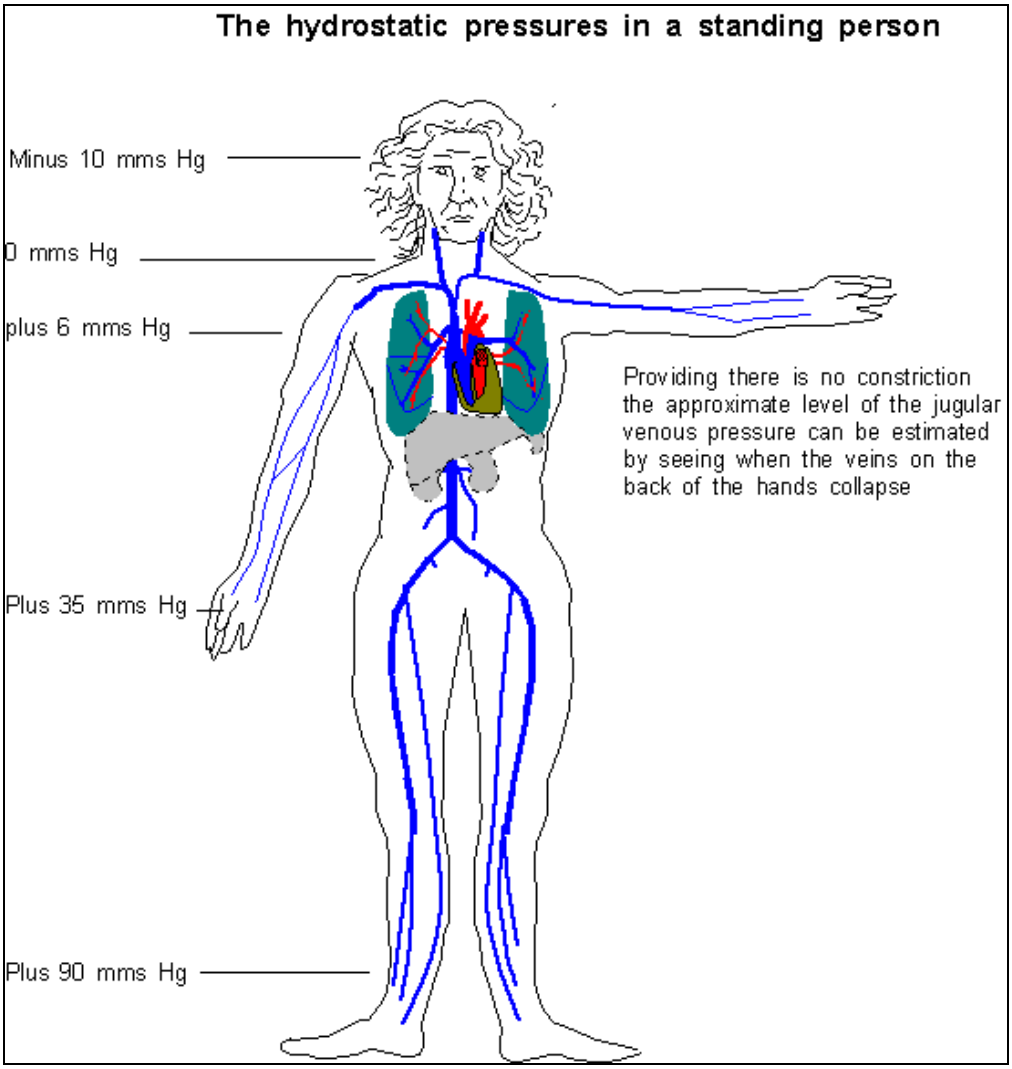
The veins have numerous tributaries and anastomoses. The veins contain vaLves which point in the direction of the heart and, particularly when the skeletal muscles contract, this forces blood back to the heart. The veins in the lower limbs are under particular strain because of the high pressures caused by our adoption of the upright stance,.and may dilate and their valves may become incompetent to produce varicose veins. The venous blood flow in the lower limbs may be slow, particularly in the immobile, and clotting (thrombosis) may occur in the deep veins of the calf and occasionally extend upwards (ileofemoral vein thrombosis)

Nearly all veins contain non-pulsatile blood and venous return mostly relies upon body movement (including that of the lungs which, when expanded in inspiration, tend to suck blood into the chest) and heartward pointing valves in some leg veins. Major veins close to the heart show some pulsation caused by some reflux of returning blood from the right atrium when it is contracting. Muscle contraction can assist venous blood to return heartwards.



Veins which lie between two capillary networks constitute a portal system, for example the veins which deliver blood from the gut capillaries to the liver capillaries.

The following diagram shows the venous pressures at various sites in the circulation. The pressure in the systemic veins just above the heart is usually 0 mm Hg. Distension of the neck veins does not usually occur because there is insufficient pressure inside the veins to produce distension.

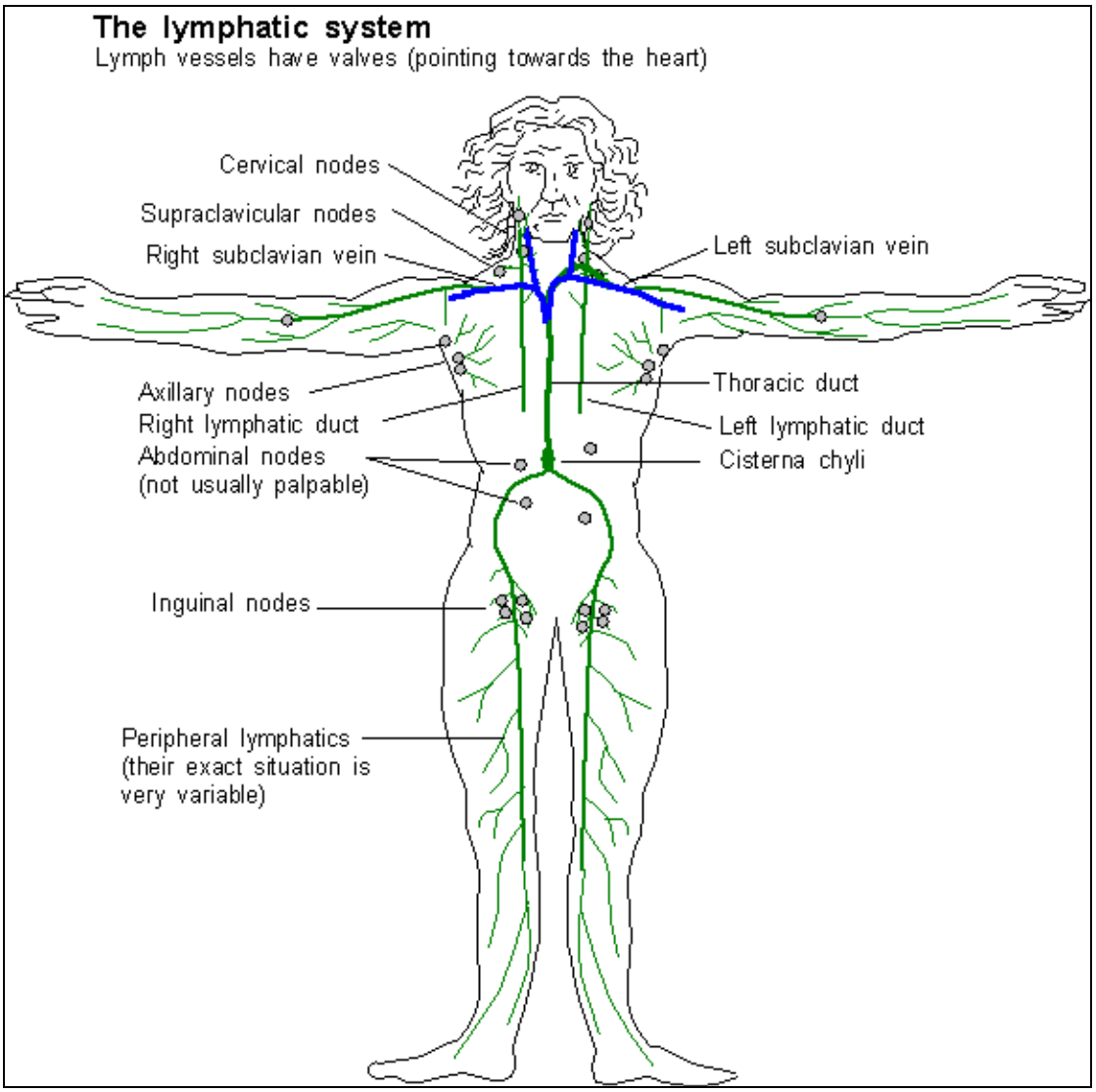


Veins can contract without affecting arterial peripheral resistance. The capacity of the blood vessels and the blood volume must be kept equal if there is to be sufficient venous blood to fill each ventricle for each contraction.. The state of the venous system thus is an important determinant of heart function. For example after a brisk haemorrhage *both* veins and arterioles constrict (the veins to allow filling of ventricles and arterioles to maintain perfusion pressure). If this mechanism fails then shock results with an inadequate circulation and a low arterial blood pressure.

THE CAPILLARIES (=small hairs)

By the time systemic arterial blood reaches the capillaries it is not pulsatile. Capillaries, the fine network of vessels between the arterioles and venules, are permeable to most plasma constituents (except the plasma proteins) and most of the movement of fluids into and out of the vascular system occurs through the thin capillary walls.

THE LYMPHATIC SYSTEM



Exudation of extra fluid into the tissues occurs if arteriolar and capillary blood pressures tend to rise and the lymph collection system caters for this excess. The lymphatics, some of which possess valves, are a necessary alternative to the veins for returning fluid from the tissues back into the bloodstream. The final common pathway for lymph drainage is the thoracic duct which enters the systemic venous system at the junction of the left subclavian vein and the left internal jugular vein. If lymphatics are blocked then tissue swelling will be high in protein, relatively stiff, and less likely than venous oedema to pit when pressed with a finger.

THE HEART

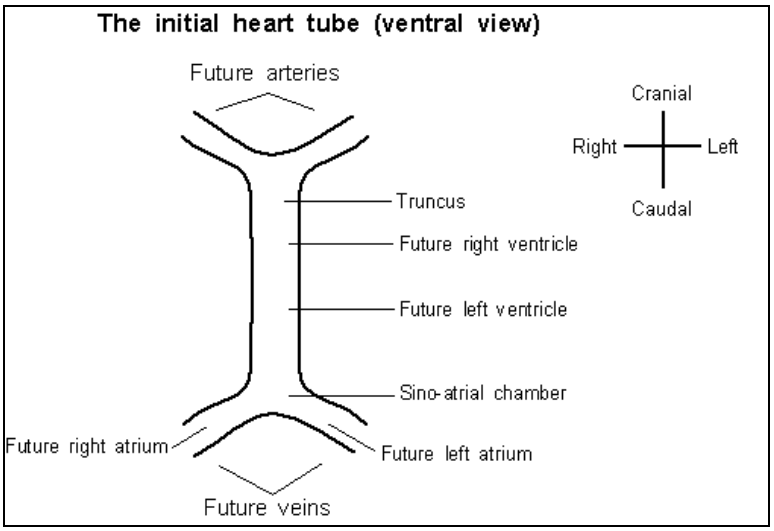
Formation of the heart in the embryo

The finished heart has two separate major pumping systems - the left and right ventricles (ventricle = small cavity), two minor pumping systems (the left and right atria.The right atrium (=entrance) delivers blood from the body into the right ventricle which then pumps blood to the lungs. Blood from the lungs then returns to the left atrium which delivers blood to the left ventricle which then pumps blood to the body. Each ventricle will require two valves, one at the entrance and one at the exit.

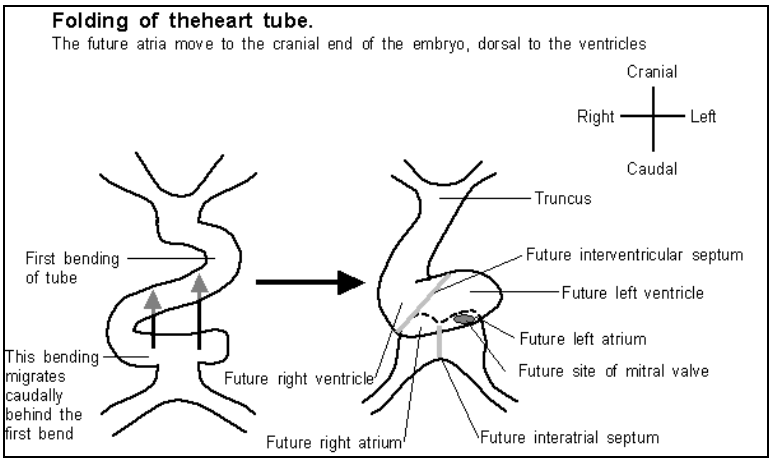
The heart begins embryonic life as a tube and thus major remodelling is required but during the change from the primitive tube to a four-chambered heart blood must continue to flow at all times. Knowledge of the embryological changes are necessary to understand the malformations which may occur.

An impulse conducting system also has to develop to co-ordinate atrial and ventricular contractions (so that the atrial muscles contract when the ventricular muscle is relaxing) and *vice versa.*

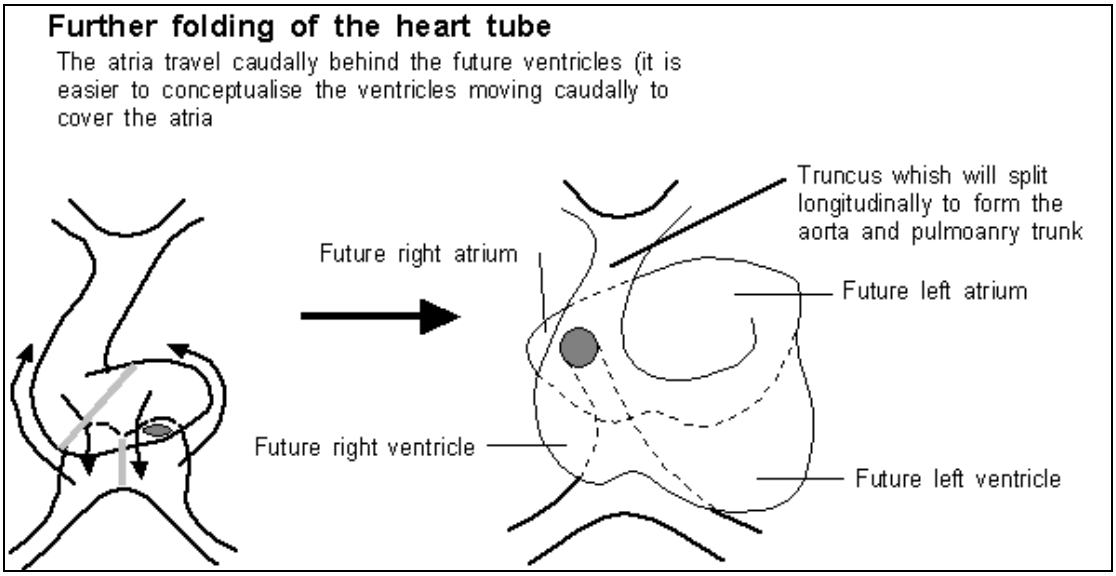
In the embryo a contracting heart tube forms, into which veins enter and arteries exit.



However a contracting tube with no valves is no use as a pump. Accordingly this primitive tube receives blood from the great veins and empties into an atrium, then via atrioventricular valves into a ventricle then into the arteries via semilunar valves.



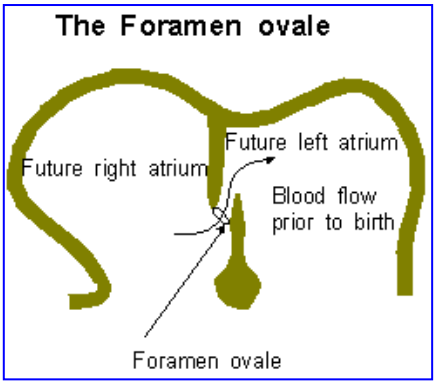
The heart tube is attached at its ends and is free to grow and fold. The caudally situated Y junction (the sino-atrial chamber where venous blood enters the primitive heart tube) develops two swellings which will later form the right and left atria. The atria move cranially, passing dorsal to the ventricles.



Veins (which become the superior and inferior vena cavae which return deoxygenated blood from the body) later enter directly into the right atrial cavity wherea four pulmonary veins carrying oxygenated blood enter the left atrium.

The truncus, the artery that will form the outflow tracts of the ventricles, indents the groove separating the atria. The truncus splits longitudinally into two tubes which provide separate outlets for the left and right ventricles (these outlets will become the aortic and pulmonary arteries respectively), and each of the two tubes develops one-way valves. Three valves (the tricuspid, aortic, and pulmonary) have three cusps and one (the mitral) has two cusps. The ventricular walls grow thicker, become contractile, and the communication between the two ventricles becomes smaller as an interventricular septum develops.

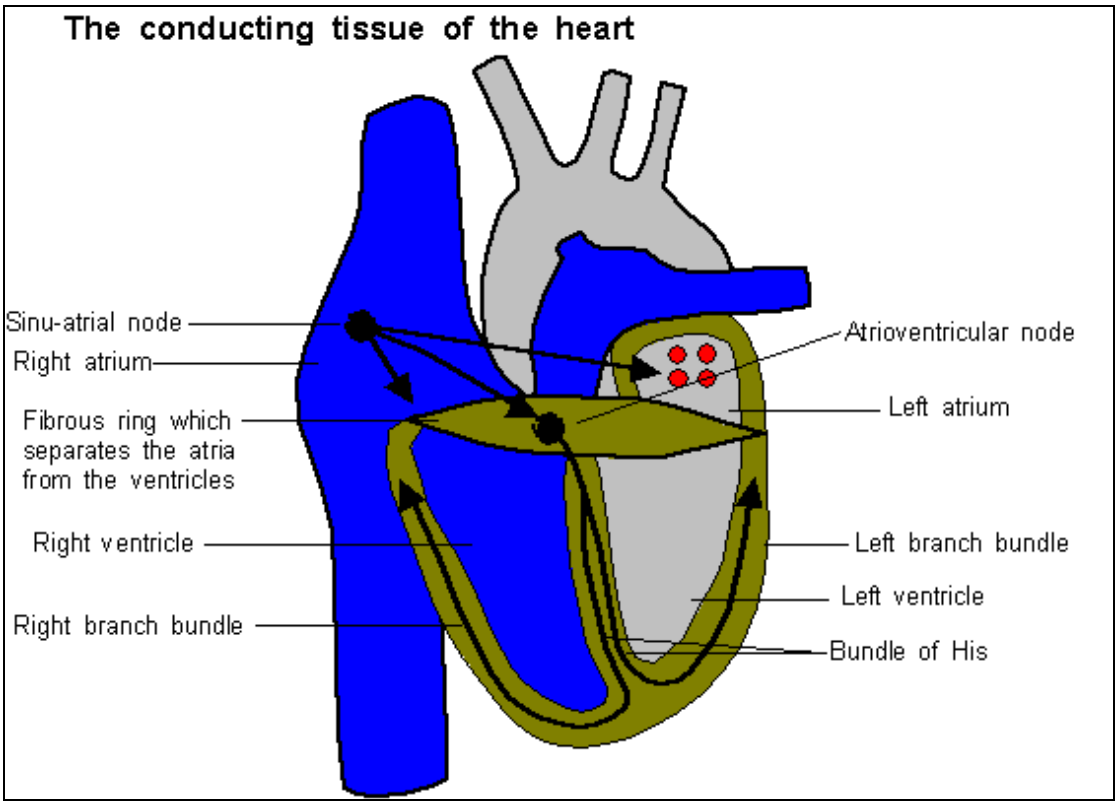
The right and left atria are separated by a thin sheet of tissue (the septum primum) but, prior to birth, blood has to continue to flow from the right to the left atrium through an opening, the ostium primum in order to bypass the "useless" lungs.



The ostium primum subsequently closes but, because blood must still bypass the lungs,a second opening develops (the ostium secundum), which is oval in shape (the foramen ovale).

The ostium secundum is surrounded by tissue which provides a flap-like valve which remains open only if the pressure in the right atrium exceeds that in the left atrium (at birth the pressure in the left atrium will rise as blood returns to it from the lungs and this communicating hole will close).

Initially the outer muscle wall which surrounded the primitive heart tube contracted as a whole, but with subsequent convolutions of the tube and the need for reciprocal contractions of the atria and ventricles, modified heart muscle fibres that transmit depolarisation waves (faster than would occur between normal heart muscle cells) were developed.



An electrical initiating point, the sinoatrial node, forms in the right atrium and sends out a depolarisation wave that causes contraction to spread radially over the atria. A fibrous ring develops to separate the atria from the ventricles and a "focal penetrating area," the atria-ventricular node, develops and connects with a short stem of conducting tissue (the Bundle of His) which then divides into right and left branches to transmit depolarisation waves to the right and left ventricles.

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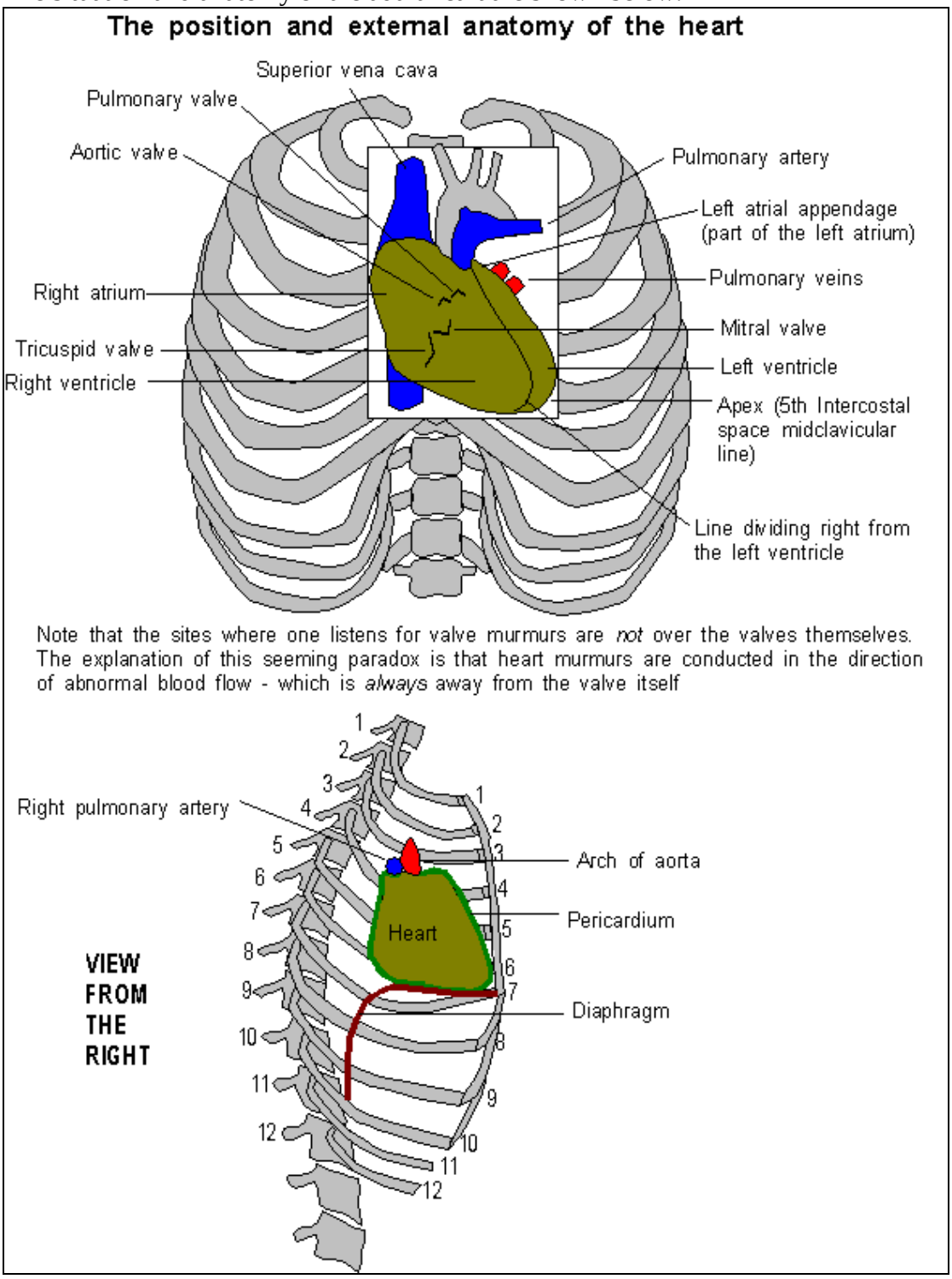
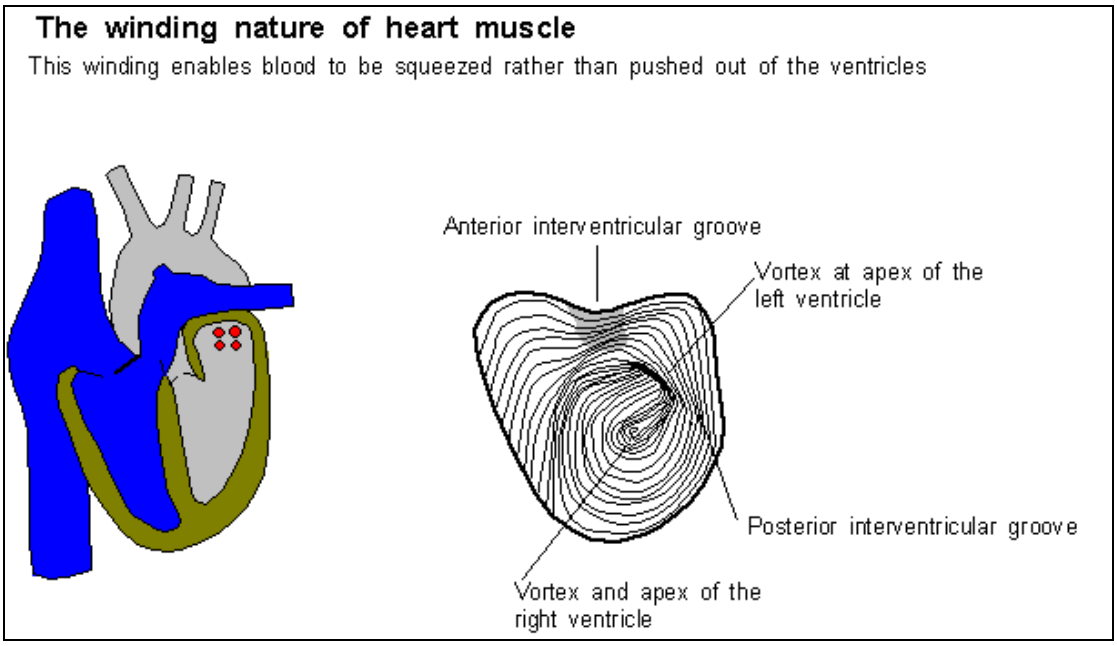
All the above begs the question "Why do we need a two-sided heart?" There are two answers. *Firstly*, a high pressure system is required to perfuse the head in an upright posture (otherwise we would faint when we stood up). *Secondly*, a lower pressure system is required to supply the lungs - if blood pressure in the pulmonary arteries were at aortic pressure then the pulmonary capillaries would have to have walls that were too thick to allow gas exchange (fish which, by and large, do not stand up thus only need one ventricle to pump blood through their gills to the body).

The situation and anatomy of the adult heart are shown below.



The heart weighs 300 grams and is much smaller than is commonly supposed (the size is exaggerated by chest X-rays). Until the advent of cardiac transplantation the heart was only guaranteed to last for a lifetime "three score years and ten" and thus a heart had to contract about 2,500 million times to pump about 170 million litres of blood.

Cardiac muscle is a specialised form of striated muscle. As muscles can only contract in a linear fashion the heart muscle has to be wound so they can compress the blood within the heart chambers.



Venous blood returns to the right atrium via the superior and inferior vena cavae. This blood then flows (with the assistance of right atrial contraction) through the tricuspid valve into the right ventricle during ventricular relaxation. At the start of ventricular contraction, the tricuspid valve shuts and the venous blood derived from the tissues is ejected into the lungs, passing through the pulmonary valve into the pulmonary arteries.

HEART FUNCTION

The function of the heart is to pump blood to the lungs for oxygenation and then to the tissues for utilisation. To achieve this, a two-sided pump (the heart), a distribution system (the systemic and pulmonary arteries) and return system (the systemic and pulmonary veins) are required. Cardiac contraction (systole) or relaxation (diastole) refers to *ventricular* events. Both atria contract together (during ventricular diastole) and, later, both ventricles contract together during ventricular systole. When both ventricles are contracting, both atria are relaxing and *vice versa*. Heart valves ensure one-way progress of blood flow.

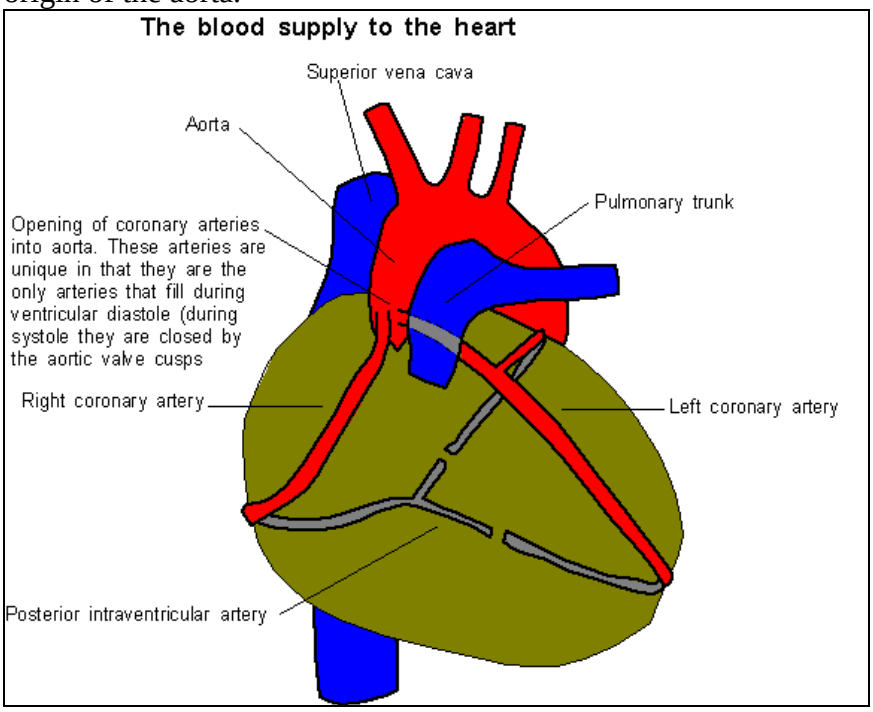
With increasing heart rates the ventricular relaxation (filling) time becomes shorter, rather than the contraction time becoming shorter. Fast heart rates, above about 120 per minute, do not allow sufficient time for ventricular filling and thus predispose to heart failure. With atrial fibrillation, in which the atria contract haphazardly "like a bag of worms," the lack of atrial assistance for ventricular filling means that heart failure can occur at lower heart rates than this.

The power of heart contraction -inotropy- is related to metabolic factors and workload. *Preload* is heart work required to cope with the venous blood returning to the heart and is related to the length of cardiac muscle fibres when contraction commences. *Afterload* is the heart work required when the heart muscle contracts to

push blood around the systemic circulation and this is in effect the resistance to blood flow in the arteries.

In general the heart tries to function as a constant flow pump rather than a constant pressure pump (variation of arteriolar constriction provide most of the pressure regulation). If peripheral resistance increases then arterial blood pressure hase to rise to maintain perfusion, which requires more work by the heart. As an approximation, the more the stretching of heart muscle in ventricular relaxation the more strong the ventricular muscle contraction and the greater the resulting stroke volume (Starling's Law of the Heart).

Blood is supplied to the heart by two coronary arteries which arise from near the origin of the aorta.



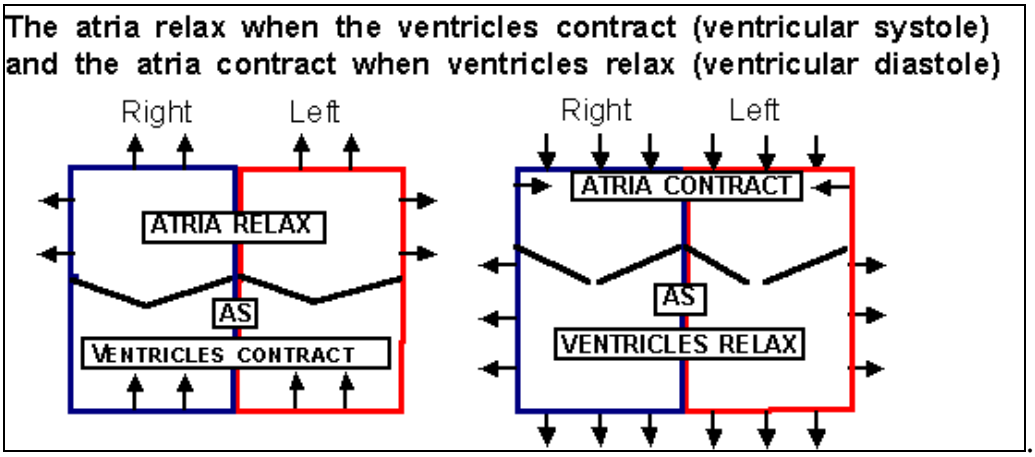
These coronary arteries are functionally end arteries, and partial or complete blockage is likely to result in heart muscle death distal to the blockage. The left coronary artery supplies most of the left ventricle and the right coronary artery supplies most of the right ventricle but also supplies the conducting tissue comprising the sino-atrial node and the interventricular node. Thus dysrythmias (abnormal heart rhythms caused by abnormal conduction) are particularly likely to occur if there is right coronary artery narrowing or obstruction.

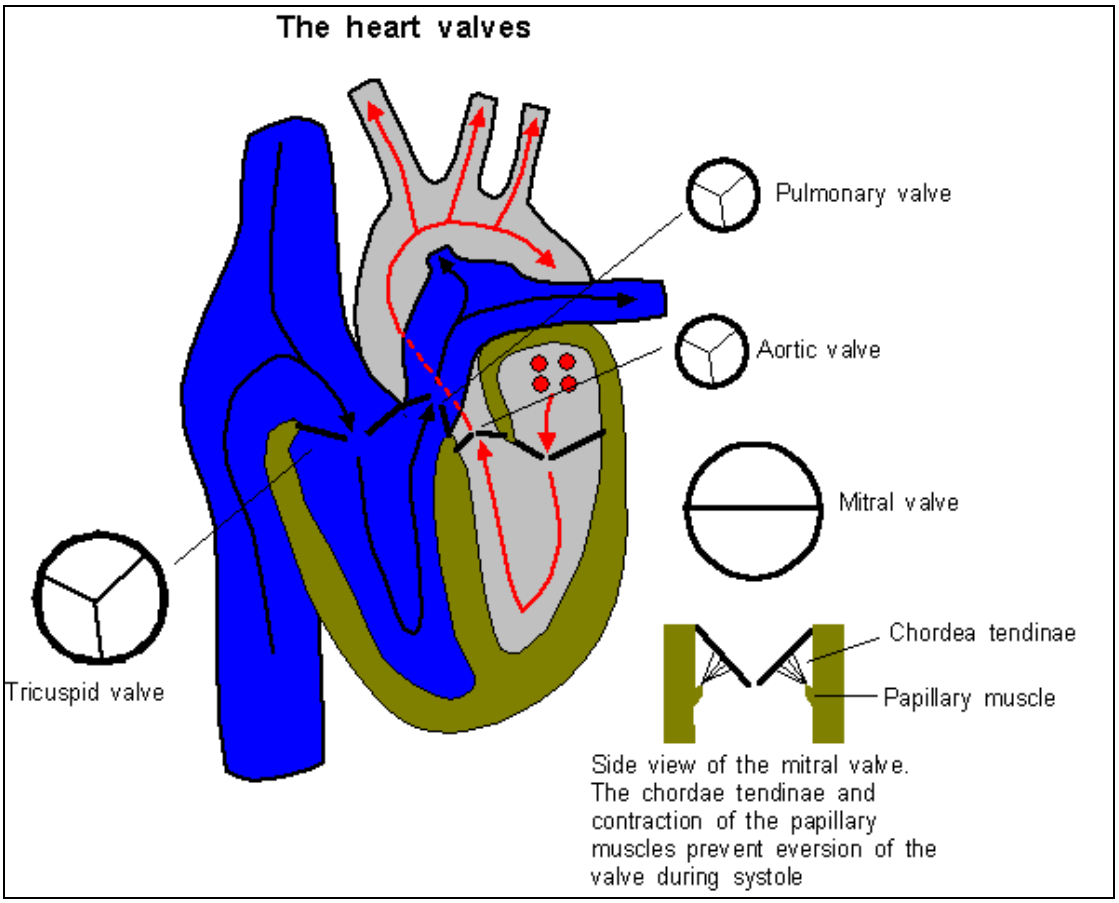
Ventricular muscle

Ventricular dilatation is usually caused by an abnormal volume load or by poor muscle function (cardiomyopathy). Ventricular dilatation allows stroke *volume* to increase but ventricular wall tension is increased, leading to a need for extra oxygen, and if this cannot be provided then the dysfunction may get progressively worse. Hypertrophy (=abnormal increase in size) of ventricular muscle usually occurs when there is an increase in ventricular wall stress, particularly if the stress is caused by the need for increased *pressure*. Often the hypertrophy is concentric and pushes into the

lumen of the ventricle. There is a risk that this "overgrown" muscle may outgrow its blood supply and thus become less efficient.

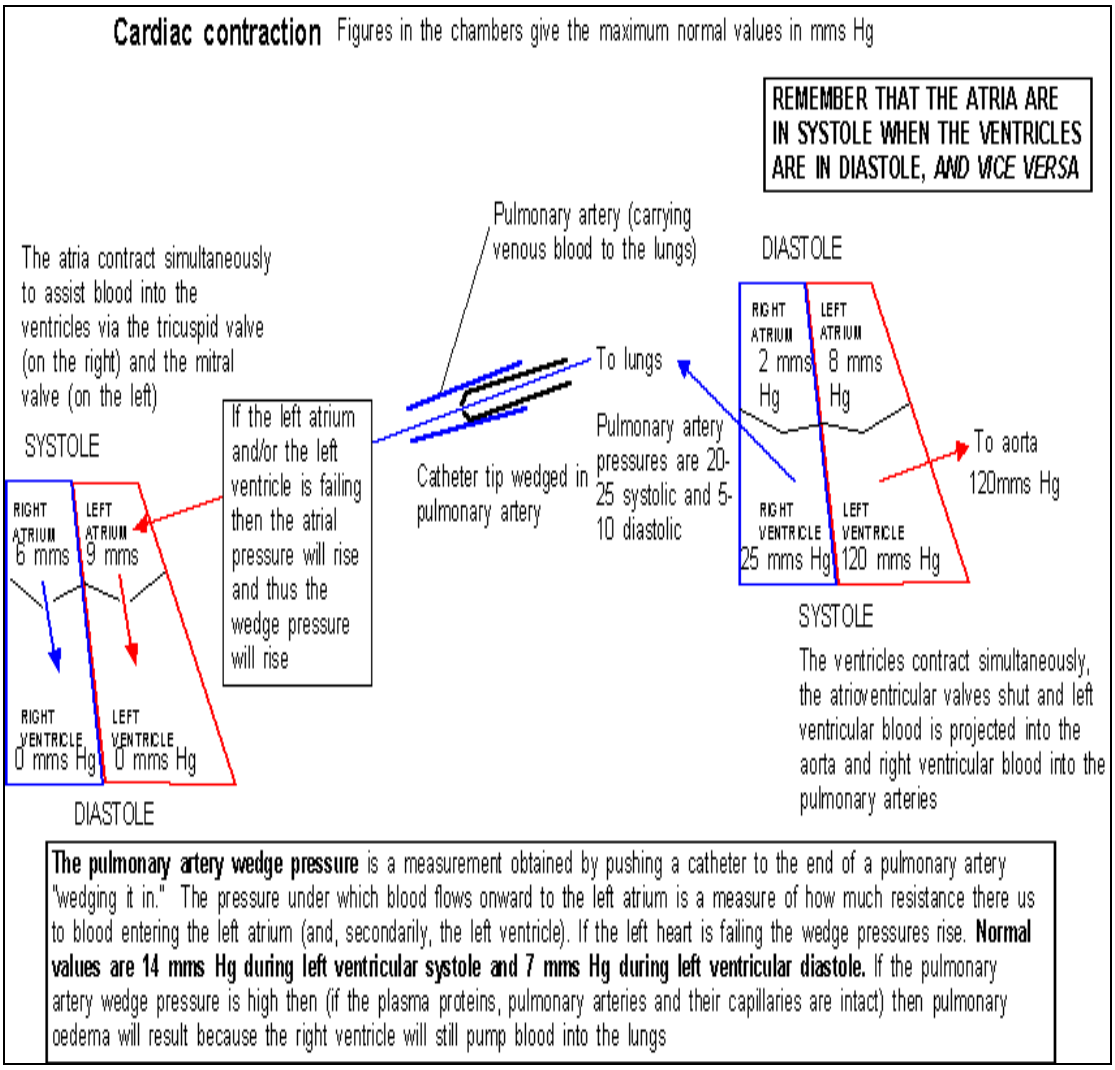
about 1 litre of blood. Normally the pulmonary artery pressure does not exceed plasma protein pressure to avoid formation of excess tissue fluid in the lungs (pulmonary oedema). But if the pulmonary artery pressure rises (as it might have to do as the right ventricle attempts to push blood through the lungs when the left ventricle is failing) then pulmonary oedema will form.





Although there are no true valves, there are muscle rings around the entry sites of the superior and inferior vena cavae into the right atrium and around the entry sites of

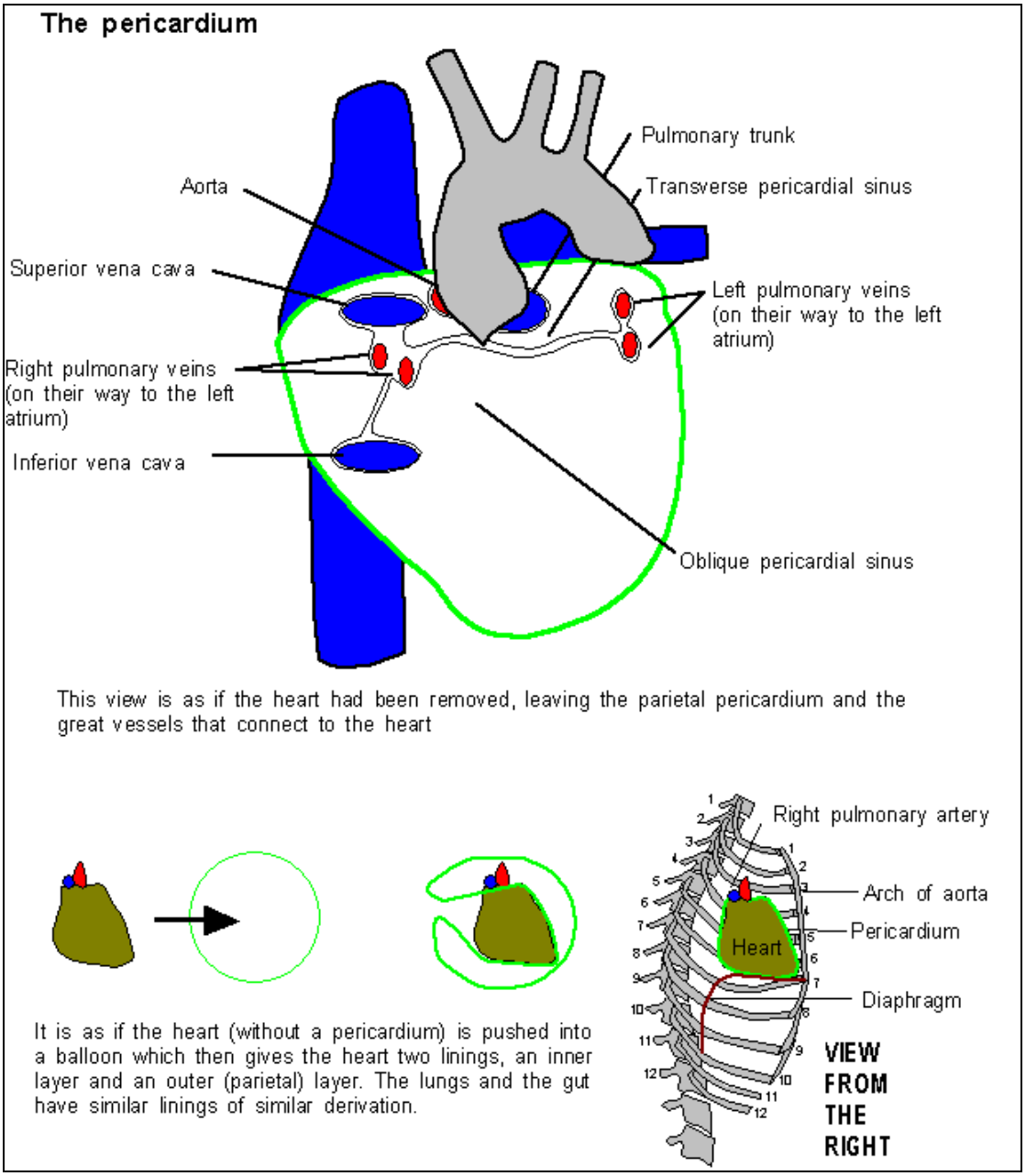
pulmonary veins into the left atrium (both to minimise retrograde blood flow during atrial contraction). There are valves "pointing" heartward" in the jugular veins



After passing through the lungs the blood, now oxygenated, returns to the left atrium via the pulmonary veins. During ventricular relaxation blood flows (with the assistance of atrial contraction) from the left atrium through the mitral valve into the left ventricle. During ventricular contraction the mitral valve shuts and blood is ejected through the aortic valve into the aorta and, thence, to the body tissues.

All four heart valves lie at much the same level in the area which separates the atria from the ventricles.

The pericardium, the sac that contains the heart, consists of two layers, an outer fibrous and inner serous layer.

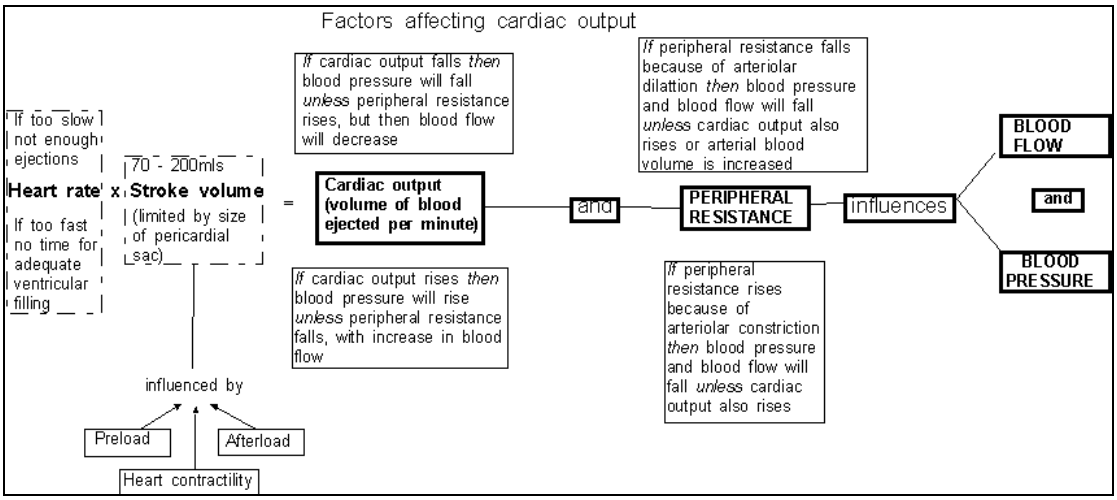


The fibrous layer act as a limiting enclosure for the heart, preventing overstretching of the heart muscle. A small amount of fluid between the two layers serves to lubricate movement.

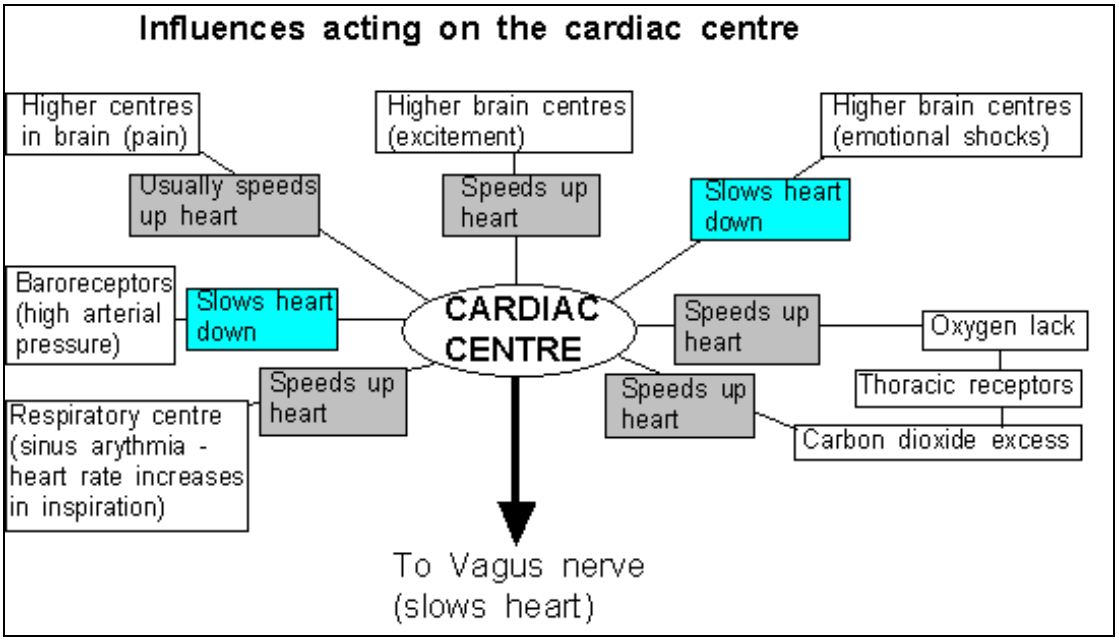
Blood pressure

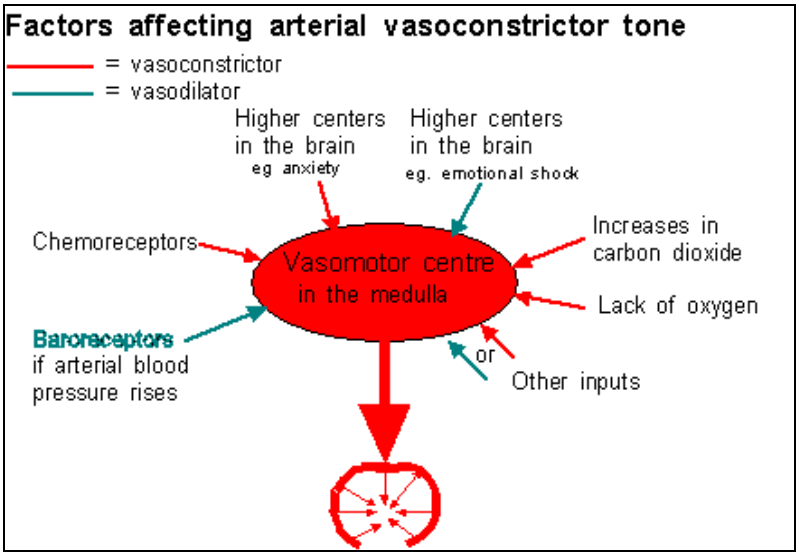
The blood pressure is the result of the force of cardiac contraction, the volume of blood ejected from the heart with each contraction and the resistance provided by the arterial system. With increased sympathetic activity the heart rate rises, the heart contraction strength rises, and the vascular smooth muscle tension rises to cause vasoconstriction. The parasympathetic system, largely via the vagus nerve, slows the heart and causes a mild vasodilation of the coronary arteries. The endocrine system, notably the adrenals and renin-angiotensin system (page 00) are also important. Pressure receptors (baroreceptors), particularly those in the aortic arch, common carotid arteries and their bifurcation, provide sensory inputs so that the peripheral resistance, heart rate, and force of heart contraction can be modified to maintain

moment to moment control of blood pressure, especially during rapid changes in posture such as standing up.



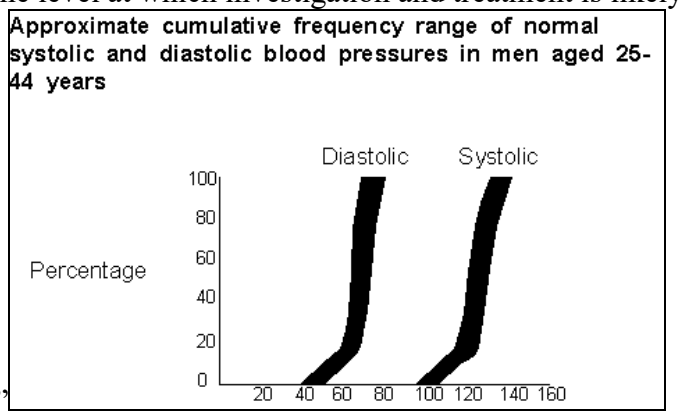
The various factors that influence vagus nerve activity (emanating from the brain cardiac centre) are shown below, followed by factors affecting vasoconstrictor tone..



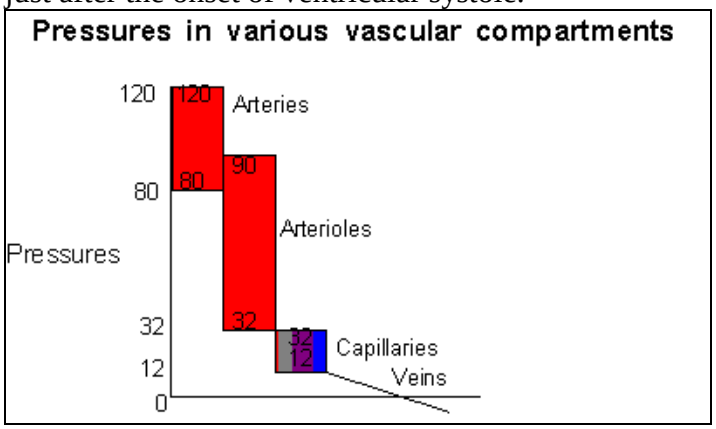


The systolic blood pressure in the aorta is just below that in the left ventricle but in ventricular diastole the pressure in the left ventricle is at a minimum about 0mms Hg. The pressure does not fall to 12mms Hg in the aorta because closure of the aortic valve prevents return of blood into the heart. However if the aortic valve is incompetent then blood rushes back into the heart from the aorta and thus the aortic blood pressure is low during ventricular diastole. It might be expected that aortic systolic blood pressure would also be low but extra left ventricular filling during ventricular diastole leads to an ejection of an extra volume of blood and thus the systolic blood pressure may not be reduced.

The systolic blood pressure is the maximum pressure achieved in large arteries during ventricular systole and the diastolic blood pressure is the minimum pressure during ventricular diastole (the difference is the pulse pressure). Opinions have varied about which is more important and which should be used predict future problems. Systolic hypertension is obviously more important if there are weak arteries which might burst during the *continual* surges of high pressure, but the diastolic hypertension could be more important as it reflects the *continuous* stress to which arteries are exposed. Both systolic and diastolic blood pressures form a continuous spectrum and it is thus difficult to define a cutoff point between normal and pathological blood pressure. I like "the level at which investigation and treatment is likely to do more good than harm."



Blood flows from areas of high pressure to areas of low pressure. The pressure is highest in the left ventricle and aorta (the arterial blood pressure is about 120mms Hg) just after the onset of ventricular systole.

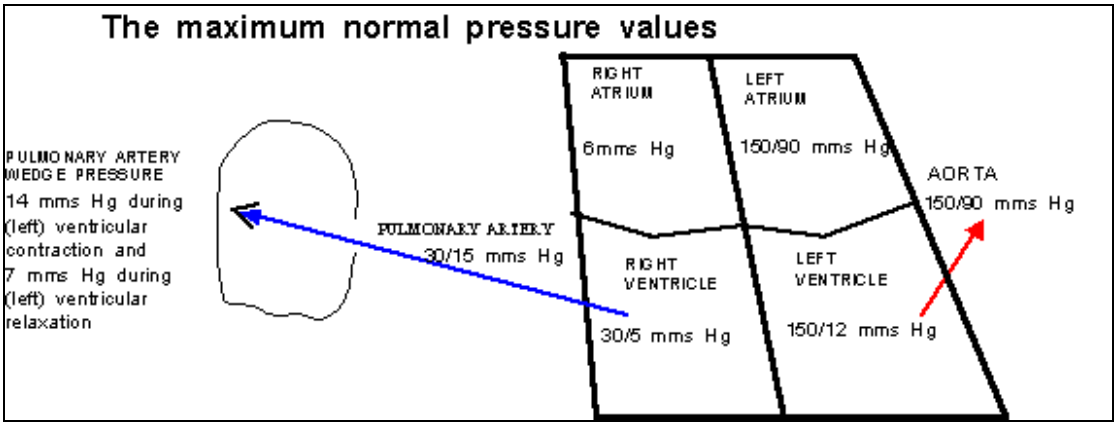


Maintaining blood pressure and blood flow is important especially for brain and kidney perfusion. If the kidneys are poorly perfused then renin is released which acts on angiotensin to cause a raise in arterial blood pressure by arterial and venous vasoconstriction, aldosterone release, and fluid retention.

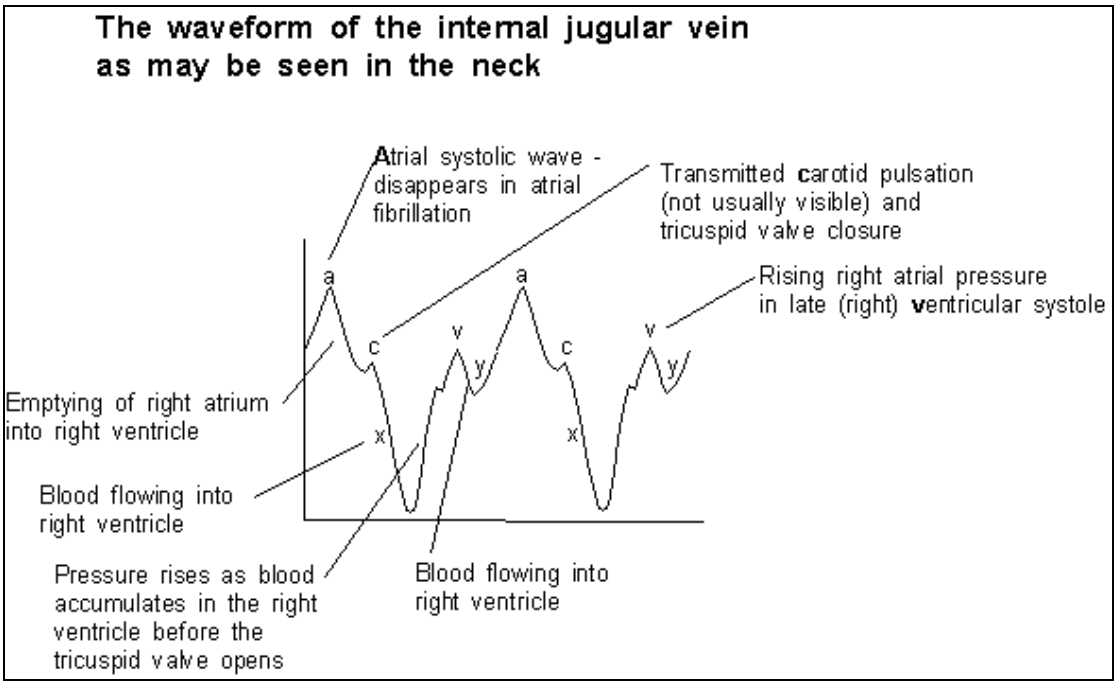
Blood pressure rises with age. The retinal blood vessels, the only blood vessels that can be seen, will show hypertension-related changes. Malignant or accelerated hypertension occurs when the blood pressure rises rapidly to high levels causing the systemic arterial vessels to sustain small areas of necrosis and/or become obstructed and /or rupture. Headache (an unusual symptom in uncomplicated mild hypertension) may occur and cerebral function may be impaired.

Hypertension may occur secondary to diseases of the kidneys, the endocrine system, therapy with certain drugs, blood vessel malformation (coarctation), or secondary to raised intracranial pressure (which requires that systemic arterial pressure is high enough to drive blood into the high pressure area within the cranium).

Complication of raised blood pressure include strokes (caused by arterial narrowing), myocardial infarction, heart failure, renal failure or blood vessel disease (notably atheroma or rupture).



Jugular venous pressure waves



The jugular venous pressure is the height of a column of systemic venous blood awaiting entry into the right ventricle when the patient is reclined at an angle of 45 degrees. The jugular venous pressure is normally less than 4 cms above the sternomanubrial joint.

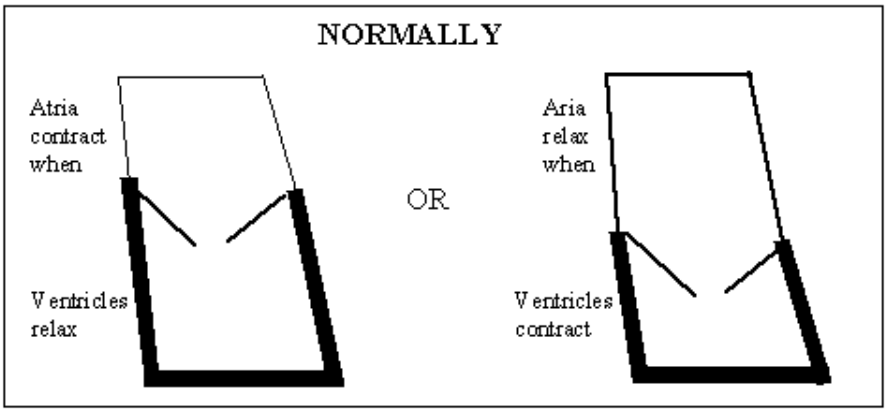
- **a** waves represents **a**trial systole if atria are contracting in a controlled fashion (in atrial fibrillation the a waves will be lost)
- a waves are followed by x descents
- **c** waves occur simultaneously with **c**arotid pulsation and are not normally visible
- **v** waves represent build up of venous blood waiting to return to the almost full right atrium which occurs during late **v**entricular contraction

The jugular venous pressure may be raised in:

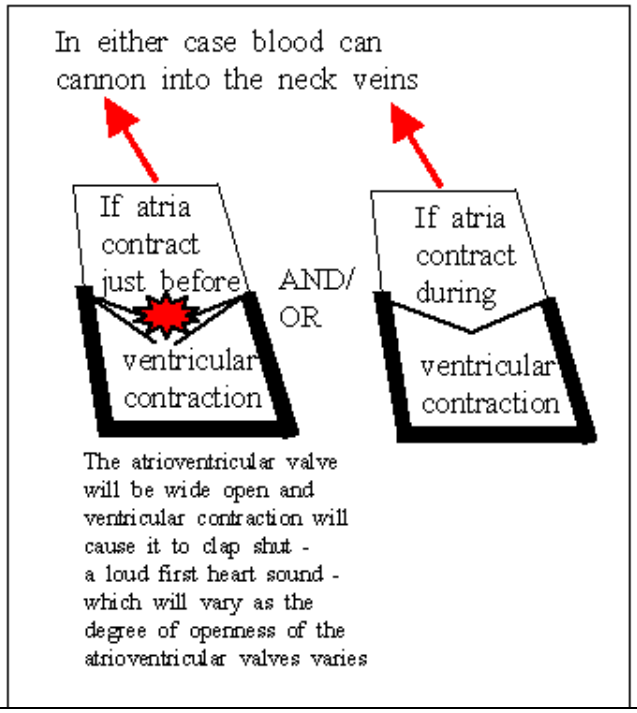
- Obstruction of the superior vena cava
- Right heart failure
- Slow heart rates which delay the processing of returning venous blood
- Tricuspid valve incompetence (in which there will be a large v wave)
- Restriction of heart movement by a tight pericardium, or excessive pericardial fluid with cardiac tamponade
- A hyperdynamic circulation
- A generalised increase in blood volume

If the atria contract independently of the ventricles, the right atrium will occasionally contract when the tricuspid valve is closed and then blood “cannons” back into the veins to produce “cannon waves.” Similarly the loudness of the first heart sound may vary because ventricular contraction may occur when the mitral valve is in varying degrees of closure.

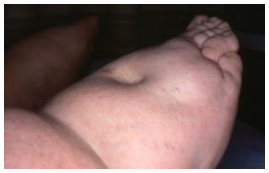
The production of varying first heart sounds and cannon waves when the contractions of the atria are independent from the contractions of the ventricles in complete heart block



BUT IN HEART BLOCK THE ATRIA CONTRACT INDEPENDENTLY OF THE VENTRICLES



Oedema



There is a dynamic leakage of water out of and into capillaries at the arterial and venous ends respectively. If the hydrostatic pressure *at the venous end* of the capillary rises (as it may do if the heart fails to deal with blood returning to it then there is a small net outflow of water and pitting oedema results. Such swelling tends, for hydrostatic reasons, to be in the lowermost parts of the body.

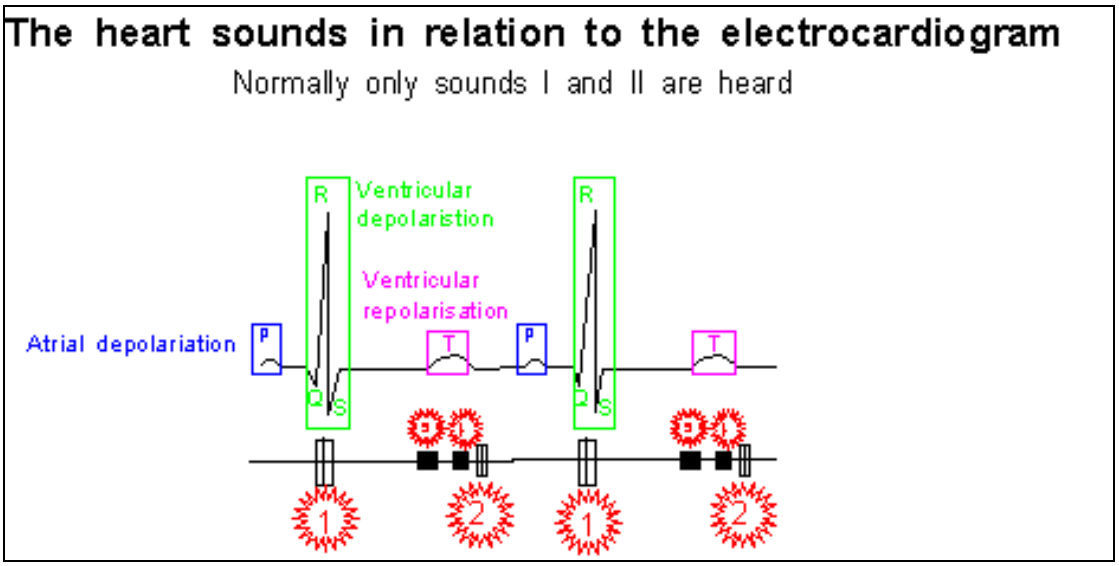
In babies, who lie flat for most of the time, oedema is more generalised (and therefore much less obvious).

The concentration of proteins in the plasma ensures that fluid enters the plasma. If osmolality rises in the extracellular fluid then water is "osmotically sucked" into the extracellular fluid. If the extracellular fluid osmolality falls (for example severe leakage of protein into the urine, or failure of protein synthesis by the liver) then water leaves the extracellular fluid. Water retained in the tissues because of low plasma proteins is mostly interstitial (between cells) rather than intracellular. Such interstitial oedema is pitting in type in that it can be indented by pressure with a finger.

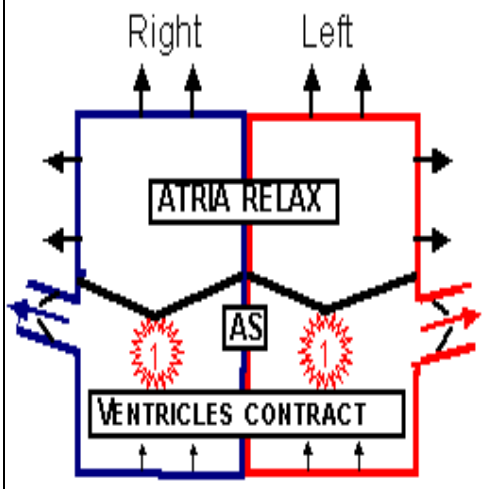
Lymphatics gather up and returns interstitial fluid (mostly protein and interstitial water) and returns them to the venous circulation via the lymphatic duct. If lymphatics are blocked oedema will be high in protein, relatively stiff, and thus less likely to pit. Forward and backward flow across the capillary bed must be *almost* the same. The volumes and pressures involved are different with the arterial side being a high pressure lower volume system which contains about a tenth of the total plasma volume and the venous side being a low pressure high volume system which contains just above a half of the total plasma volume.

If plasma volume decreases because of low osmolality, the kidney retains water in an attempt to increase the plasma volume but water still leaks out into the interstitial fluid and thus oedema develops. This mechanism is particularly mediated by an increase in antidiuretic hormone with water being reabsorbed by the distal convoluted tubules. If plasma volume falls the kidneys retain sodium because there is an increased release of aldosterone (a sodium retaining hormone) which acts on distal kidney tubules.

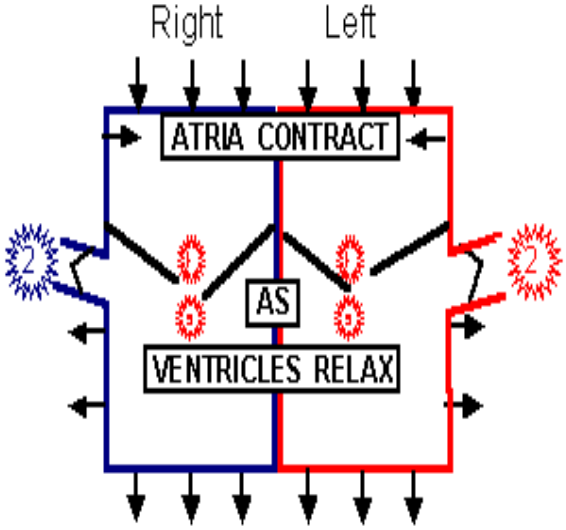
Below is a diagrammatic representation of the heart sounds in relation to the electrocardiogram.



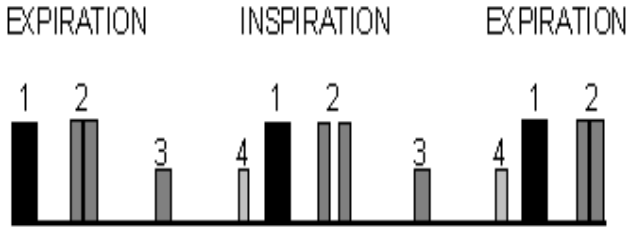
The generation of the heart sounds. Normally only the first and second are audible

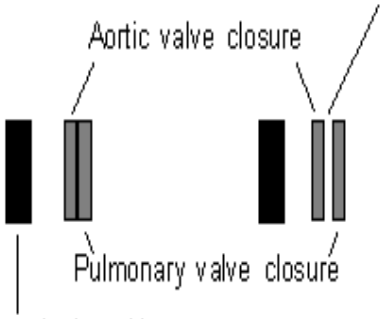


The first heart sound is made by the mitral and tricuspid valve closures



The second heart sound made by the aortic and pulmonary valve closures.
The third heart sound is made by ventricular filling, and the fourth heart sound occurs in association with atrial contraction





The split in the second heart sound

Normally the aortic valve closes sooner than the pulmonary valve because the pressure at the start of ventricular diastole is higher (maximum normals being 150 and 30 mms Hg respectively). During inspiration the lung volume is higher, more blood can pass through the pulmonary valve which delays its closure and thus the pulmanary second sound is normally delayed in inspiration

Heart sounds are abrupt brief sounds. *First heart sounds* "Lup" are associated with mitral and tricuspid valve closure. *Second heart sounds* "Dup" are associated with aortic and pulmonary valve closure. The first and second heart sounds are predominantly created by the left-sided valves because of the higher pressure gradients in the left heart. Heart sounds are louder if the causative valve shuts more quickly or forcibly than normal (because of hypertension or if valves are thickened as in mitral stenosis).

Third and fourth heart sounds

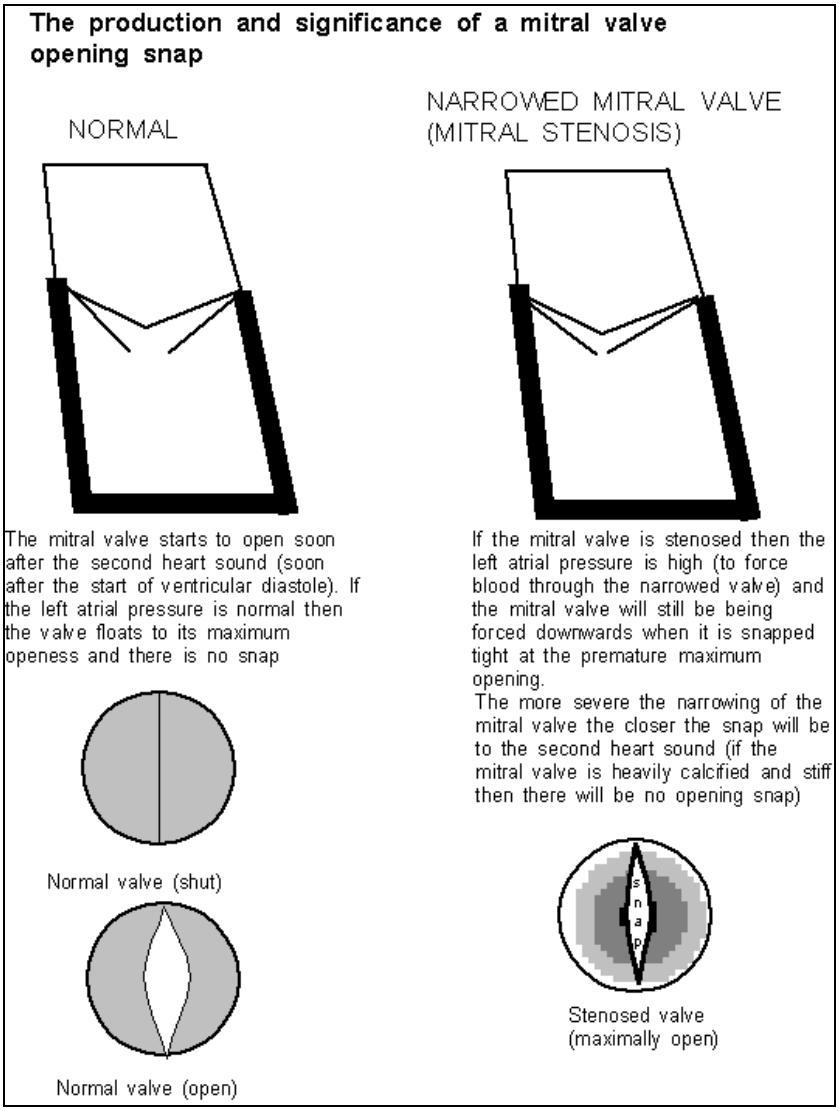
Third and fourth heart sounds are usually, but not always, abnormal findings. *Third* heart sounds are associated with rapid ventricular filling during ventricular relaxation, whereas *fourth* heart sounds are associated with atrial contraction at the end of atrial emptying during ventricular relaxation - it is impossible to have a fourth heart sound in the presence of atrial fibrillation because fourth heart sounds require co-ordinated atrial contraction.

A third heart sound can be normal in those who have slow pulse rates (including athletes), in febrile conditions and during pregnancy (in each case rates of blood flow into the ventricles are faster than normal).

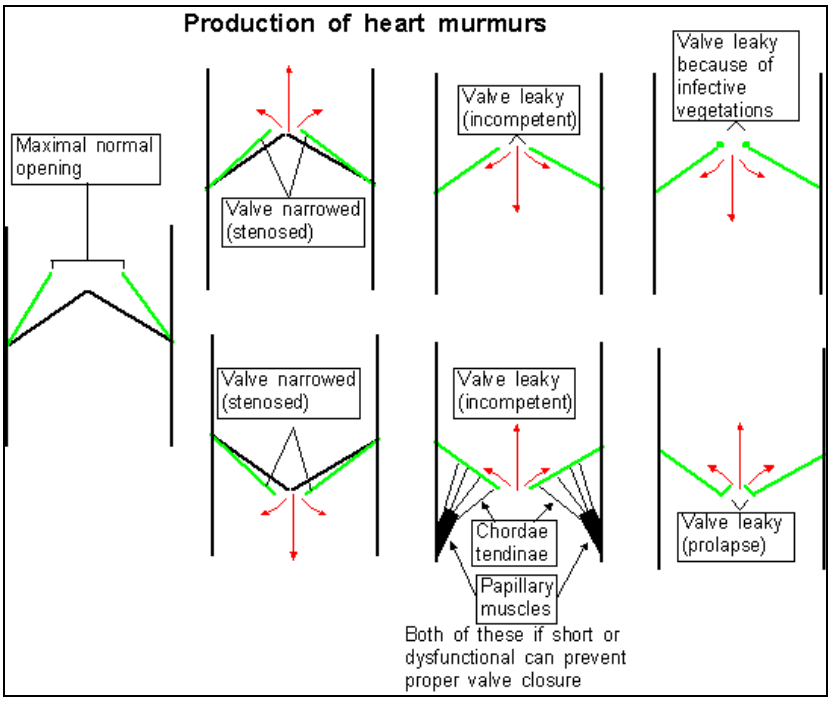
An abnormal (left ventricular) third heart sound may be found in severe hypertension, myocardial infarction (which usually affects the left side of the heart), left heart failure or mitral incompetence. In mitral incompetence blood regurgitates into the left atrium during ventricular contraction and, during ventricular relaxation, the unusual amount of blood in the left atrium rapidly re-enters the left ventricle thereby causing a third heart sound.

Either a third or a fourth heart sound may give rise to a triple or a 'gallop' rhythm and, if both third and fourth heart sounds are audible, a quadruple 'summation gallop' rhythm results.

Other added sounds include clicks (very brief sounds notably occurring if the mitral valve prolapse into the left atrium) and snaps (notably in mitral stenosis when narrowed valves snaps open). The closer the snap to the second heart sound the more premature the snap and thus the more severe is the stenosis.



Heart murmurs



Heart murmurs are:

- Smooth (rather than abrupt) musical-like sounds
- Caused by turbulent blood flow
- Heard at discrete, usually regular, points in the heart cycle
- Transmitted in the direction of abnormal blood flow
- Worsen when blood flow (and thus turbulence) across valves increases, during exertion for example

Rheumatic fever used to be caused by Streptococcal sore throats with heart valves damaged resulting from immunological cross-reactivity between streptococci and heart muscle and valves. In the United Kingdom rheumatic fever (and thus rheumatic heart valve disease) has become uncommon and degenerative and congenital disease are now more common causes of heart valve disease.

The aortic and pulmonary valves and their lesions are small and, like small musical instruments, tend to produce high-pitched sounds. The mitral and tricuspid valves and their lesions are larger and, like large musical instruments, tend to produce lower pitched murmurs.

If the aortic or pulmonary valves are stenosed or have extra blood flowing through them, the murmurs produce crescendo and diminuendo during ventricular contraction, leading to the so-called diamond-shaped ejection systolic murmurs, the murmur tending to be loudest during maximum flow.

Valves can become:

- Narrowed (stenosed)
- Leaky (incompetent, regurgitant, insufficient)
- Both the above

In general heart valve abnormalities tend to become worse as time passes. Rapid onset valvular dysfunction is often poorly tolerated whereas a slow onset of valve dysfunction gives time for heart muscle to adjust. Abnormal valves may be more vulnerable to infection - endocarditis - which makes valves incompetent.

Right heart murmurs, unlike left sided murmurs, may be increased when inspiration expands the lungs which can then accommodate extra blood from the right ventricle. This allows extra blood to pass through the right side of the heart, thereby accentuating murmurs.

Narrowed heart valves

When a normal valve is open the pressures either side of the valve are different so that flow occurs but if there is significant stenosis the pressure in the pushing chamber will be much higher (unless the pushing chamber is unable to provide the necessary pressure (in which case the heart is failing).

If stenosis of a ventricular *outflow* valve (aortic valve for the left ventricle and pulmonary valve for the right ventricle) is significant the muscle of the heart chamber which pushes blood through the valve may increase in bulk to maintain flow across the valve by increasing ventricular pressure. In general if there is a *pressure* overload in a ventricle (as in the left ventricle in aortic stenosis) the "pushing" muscle enlarges.

If there is narrowing of ventricular *entry* valves (mitral for the left ventricle, tricuspid for the right ventricle) then heart failure may result because of inadequate blood flow. The ventricle concerned will be protected from both pressure and volume overload and will not enlarge (unless its exit valve is also abnormal).

Incompetent heart valves

Incompetent heart valves leads to *volume* overload in a ventricle when (particularly with aortic incompetence) the requirement is not for increased ventricular pressure but rather a requirement for increased ventricular volume to accommodate more blood than usual and thus the relevant heart chamber tends to dilate.

The treatments of valvular heart disease include:

- Treatment of associated heart failure
- Valve repair
- Balloon valvuloplasty - a balloon is inflated in narrowed valves
- Valve replacement - non-biological Starr-Edwards type ("ball in cage") or biological (pig valve xenograft. Xeno =foreign = from another species)
- Cardiac bypass techniques to pump blood whilst the heart is undergoing surgery

The pathophysiology of various stenoses and incompetences of the heart valves are detailed below. Mitral stenosis, pulmonary stenosis, pulmonary incompetence and tricuspid stenosis are relatively uncommon. Currently the majority of murmurs originate from the left side of the heart because the pressures are higher and this stresses left heart valves and, with the decline in rheumatic fever and thus rheumatic heart valve disease, left-sided murmurs are usually caused by degenerative processes, particularly to cause aortic stenosis/sclerosis and mitral incompetence.

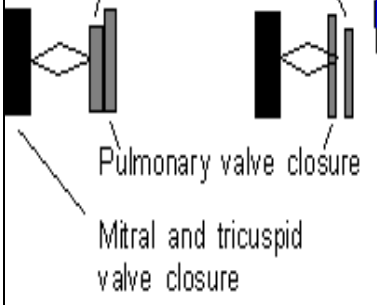
Heart murmurs that occupy all of ventricular contraction (pansystolic murmurs) occur when flow through leaky valves tends to be constant and occur in mitral incompetence, tricuspid incompetence and ventricular septal defects.

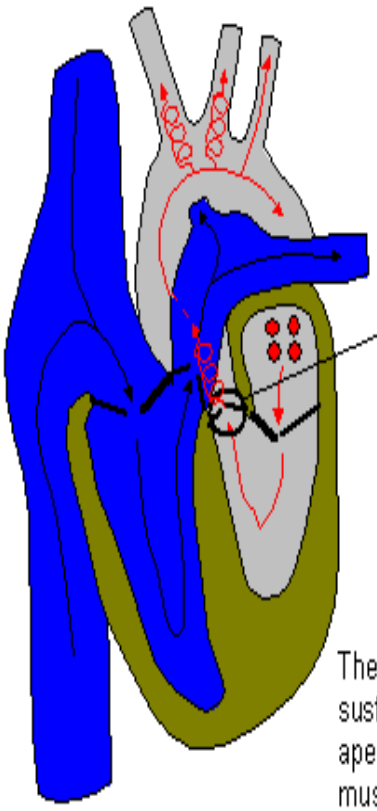
The pathophysiology of aortic stenosis

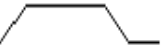
Because the **aortic valve** is small and has a high pressure gradient, the murmur will be high pitched and loud, and will be maximal during mid-systole (when the maximum rate of blood ejection from the left ventricle occurs)

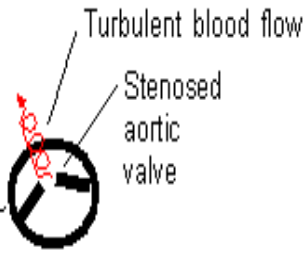
There may be a **thrill** in the carotid arteries in the neck

There may be a plateau pulse









The **aortic second sound** is usually soft as the stenosed valve only has a small distance to travel before it shuts

A diamond-shaped, high-pitched systolic murmur results. The second heart sound may be delayed and the aortic valve closure may be delayed until after pulmonary valve closure.

The **apex beat** is forceful and sustained. Displacement of the apex beat may be absent as muscle hypertrophy is mostly into the lumen of the ventricle (typical of pressure overload hypertrophy)

Aortic stenosis may be congenital (usually associated with bicuspid valves, rheumatic damage, or senile valve calcification)

Fainting on exertion may result if the heart cannot cope with increased demand by increasing its output

Angina may result because of inadequate coronary artery perfusion (the coronary arteries come off the aorta just above the aortic valve)

Collapse or sudden death may result if heart muscle ischeamia causes dysrythmias

The pathophysiology of aortic incompetence

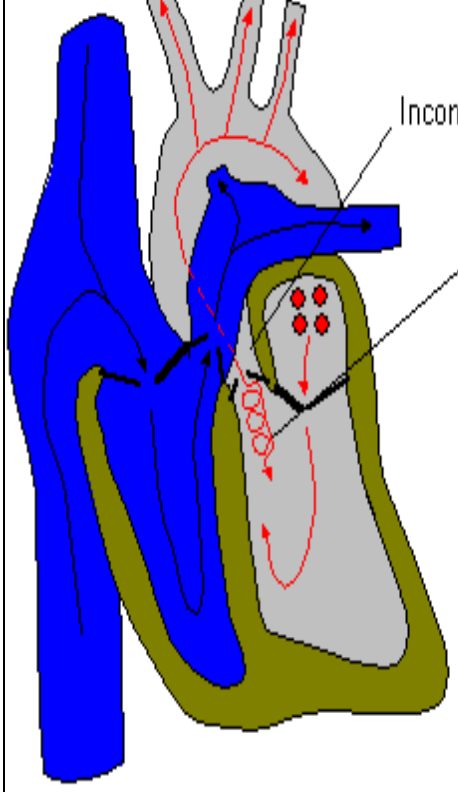
Wide pulse pressure - the difference between the systolic and diastolic blood pressures

Aortic incompetence may be rheumatic in aetiology. Some bicuspid aortic valves are incompetent

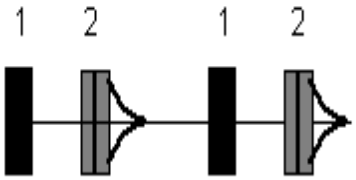
Incompetent aortic valve

Turbulent blood flow

The incompetence is caused by damage to the valves themselves (as in rheumatic damage or in certain autoimmune diseases) or by dilation of the aortic valve ring (as classically occurs in syphilis)

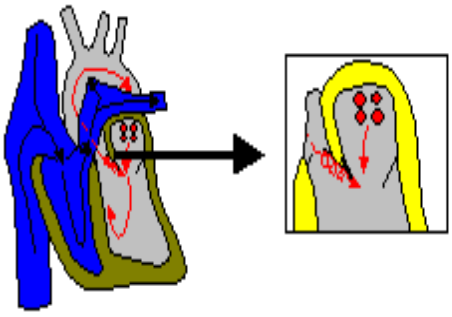


Displaced apex beat (because of volume overload of left ventricle)

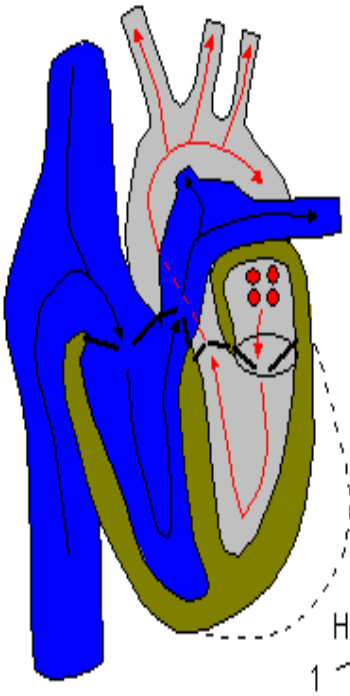


A high-pitched decrescendo murmur best heard at the lower end of the stenum with the patient leaning forward and holding their breath in expiration

On occasion blood regurgitating from the aortic valve can impinge on the mitral valve to cause a mitral-type murmur (the Austin-Flint murmur)



Mitral incompetence



Causes of mitral incompetence include:
Dilatation of the valve ring if the left ventricle dilates
Damage to the heart valve
Damage to the restraining tissues of the mitral valve

Turbulent blood flow

Valvular incompetence

Dilated left ventricle associated with volume overload

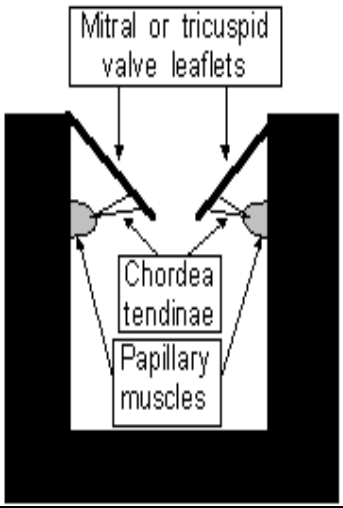
The extra regurgitant blood that has to return to the left ventricle can cause a third heart sound and/or a short mid-diastolic murmur

Heart sounds

1 2

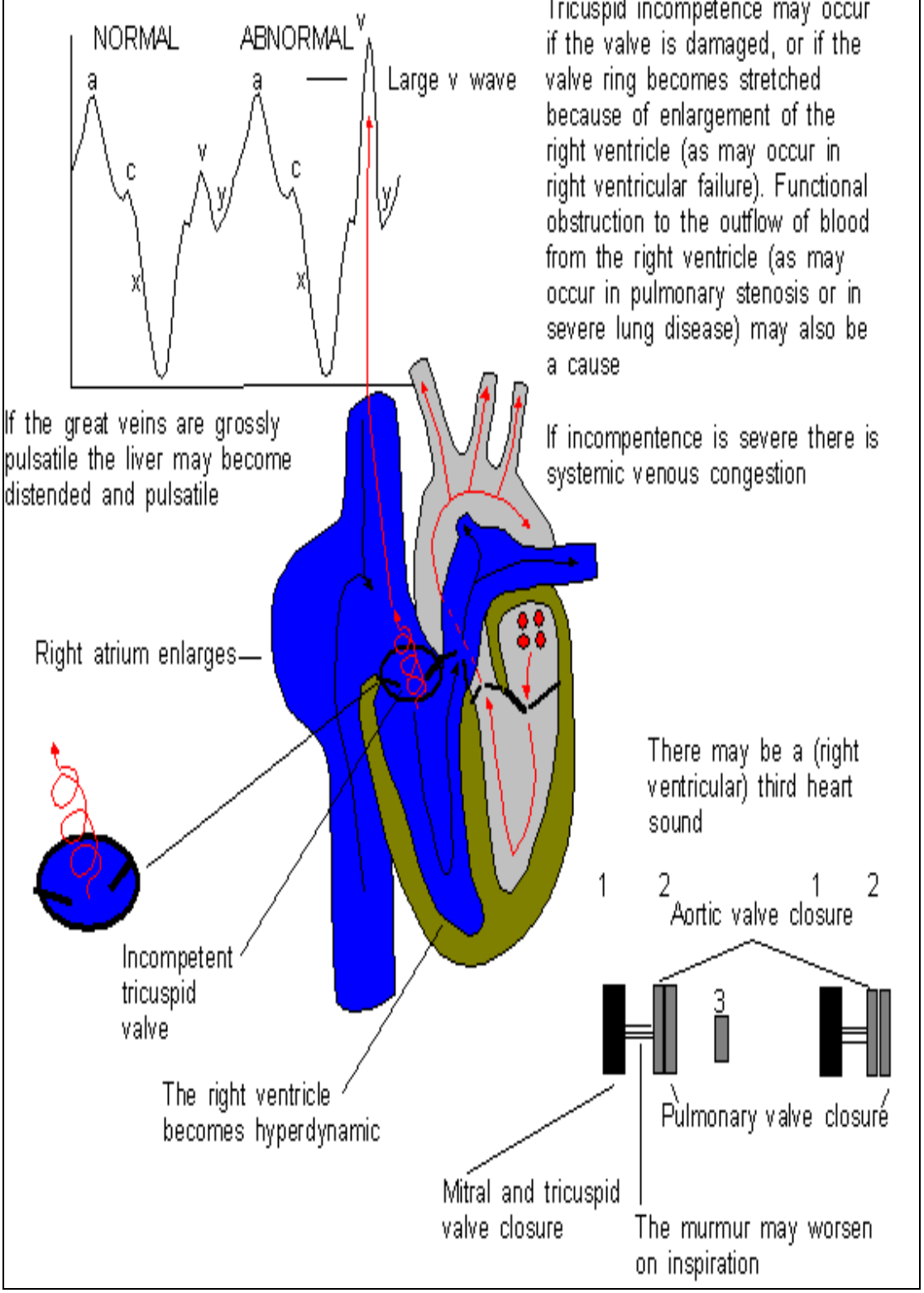
There is a pan-systolic apical murmur which radiates to the axilla because the direction of abnormal blood flow is mostly towards the back

The mitral valve prolapse syndrome "floppy mitral valve syndrome" may be casued by chordae that are too long or if the mitral valve ring is calcified

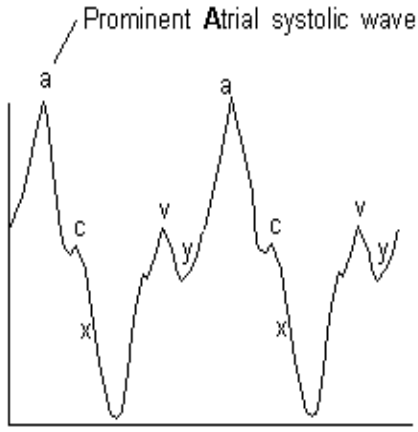


Papillary muscle damage or ischaemia causes dysfunction possibly with incompetence of the valve (mild dysfunction in inferior myocardial infarction gives rise to mild mitral incompetence but papillary muscle rupture usually produces fatal incompentence (unless surgical intervention occurs)

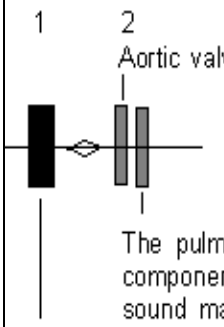
The pathophysiology of tricuspid incompetence



Pathophysiology of pulmonary stenosis



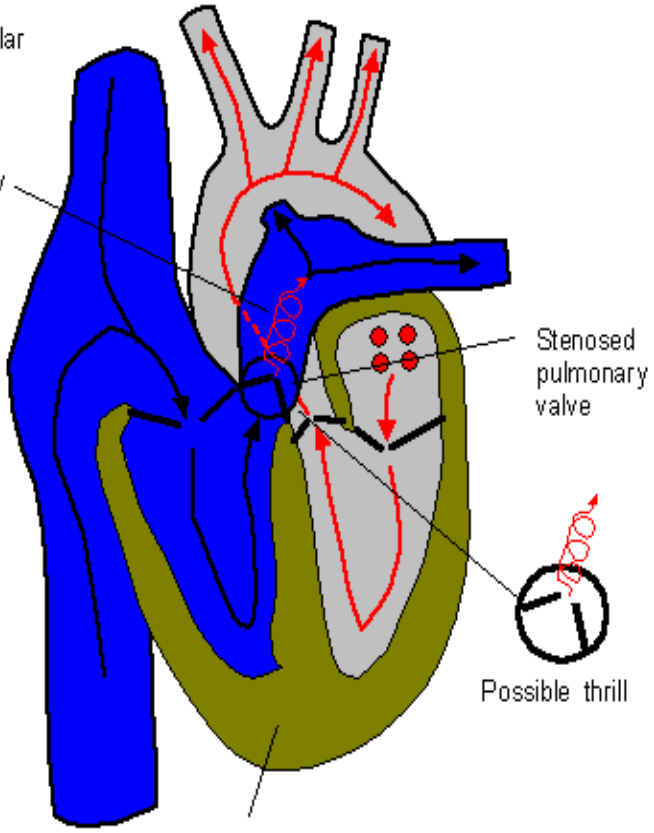
Blood may also be held up in the right atrium causing prominent jugular venous pressure (a waves) to be seen



Aortic valve closure

The pulmonary valve component of the second sound may be delayed

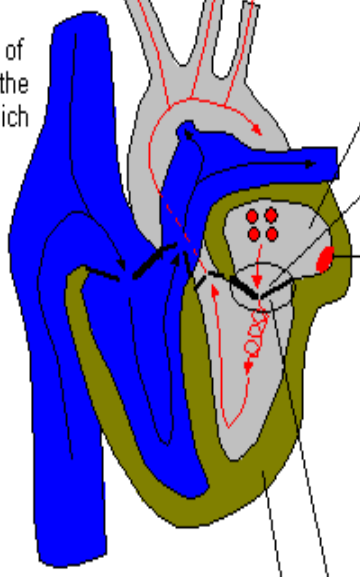
Mitral and tricuspid valve closure



With severe stenosis blood will be held up in the right ventricle the muscle of which may therefore become hypertropied and palpable.

The pathophysiology of mitral stenosis

With severe mitral stenosis blood cannot return from the lungs and the right ventricle may also fail because of increased pressure in the pulmonary arteries (which carry systemic venous blood to the lungs for oxygenation)



The left atrium dilates as there is a *volume* overload of blood waiting to enter the left ventricle

Mitral valve edges become adherent, progressively stiffen and may calcify

Blood clots may form in the dilated atrium. If such clots break off they will block peripheral arteries (systmeic emboli).

Loud first heart sound caused by the thickened, sometimes calcified, valve banging shut

No displacement of apex beat in solitary mitral stenosis. A diastolic thrill may be palpable

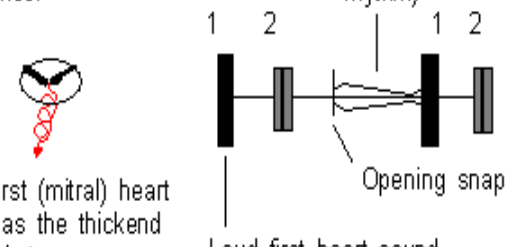
Mitral stenosis is usually rheumatic in aetiology. The left heart may fail to pump blood efficiently and signs of left heart failure may develop.

Stenosed mitral valve

The left ventricle does not become enlarged or dilated because there is no volume or pressure overload - the left ventricle is protected by the stenosed mitral valve

If there is combined mitral stenosis and incompetence the presence of an opening snap suggest dominance of the stenosis, whereas a third heart sound suggests dominance of the incompetence.

Rumbling, low-pitched mid diastolic murmur with pre-systolic accentuation (only if the patient is in sinus rhythm)

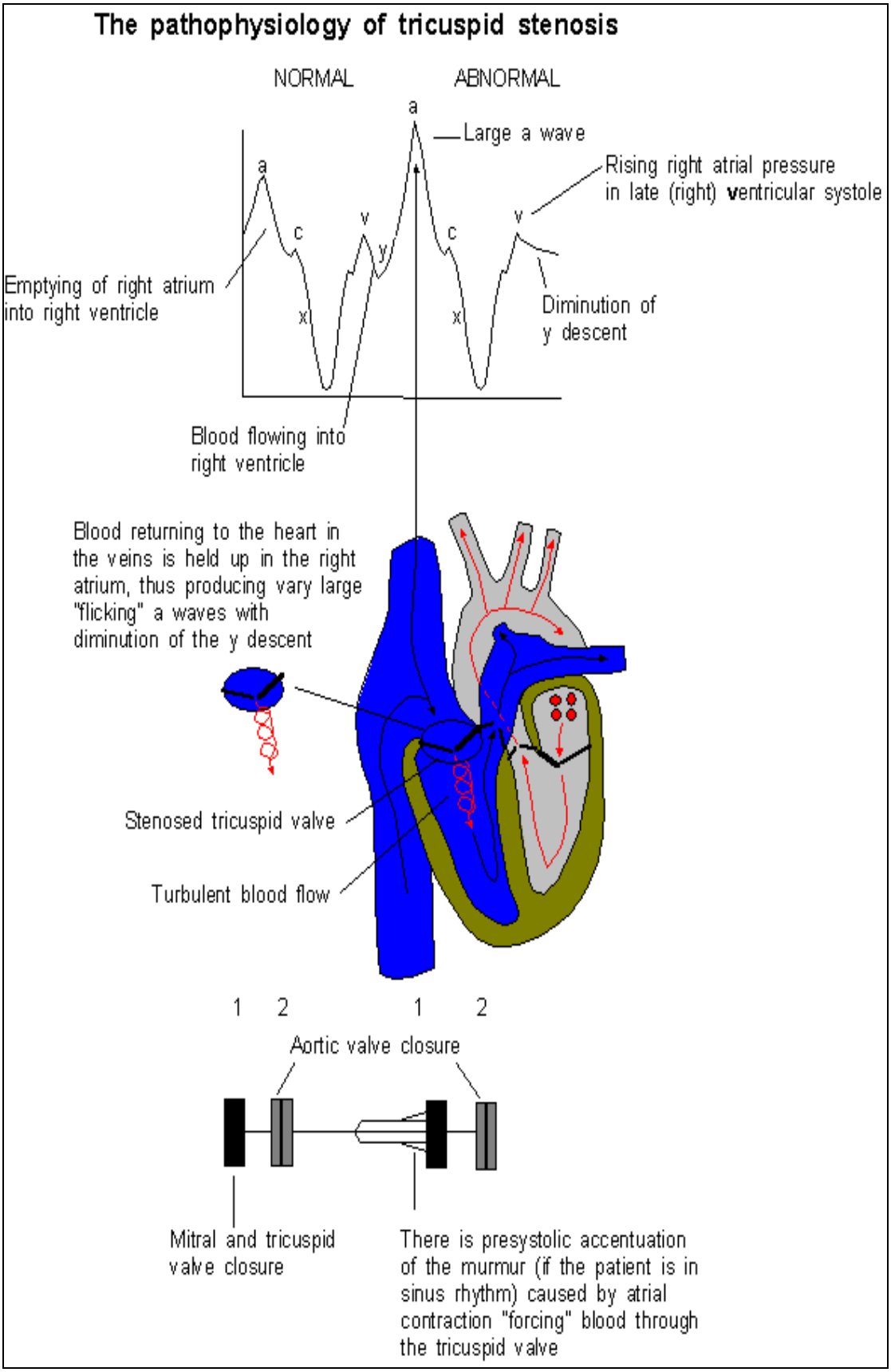


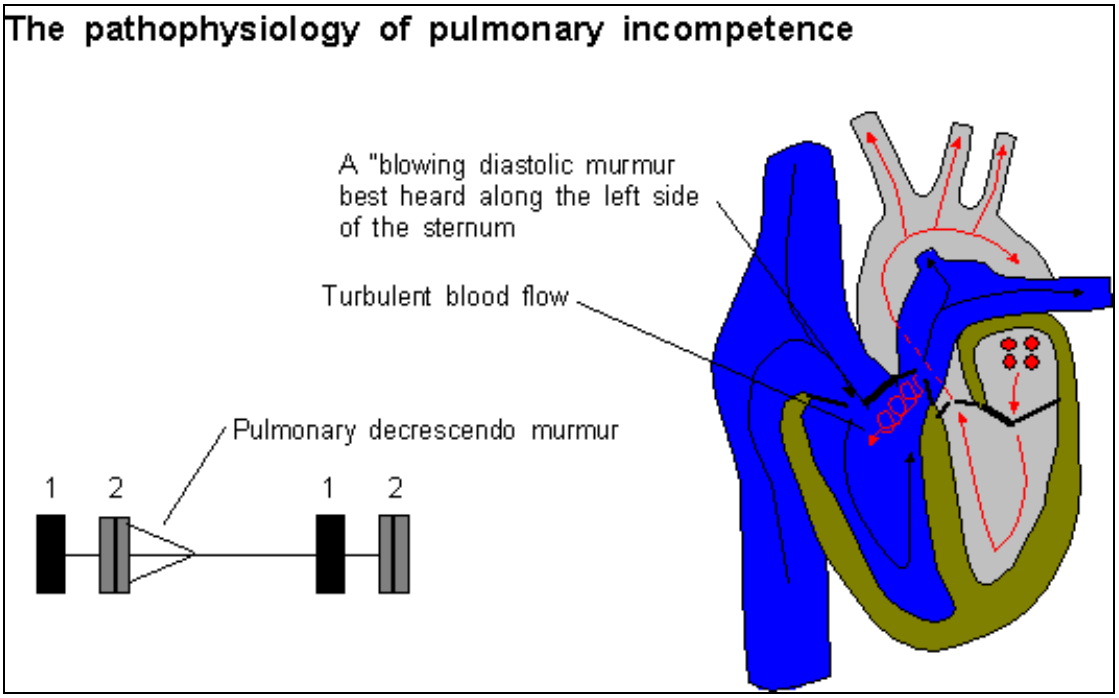
Loud first (mitral) heart sound as the thickend valve shuts

Opening snap

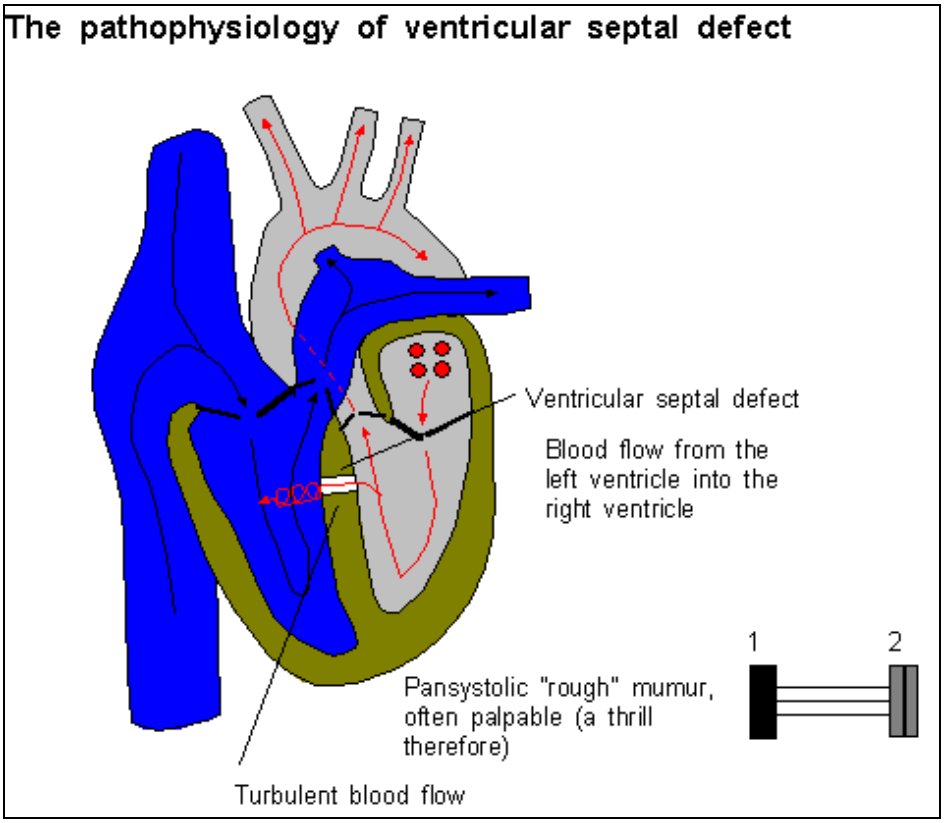
Loud first heart sound

There will be no murmur if blood flow across the valve is minimal


The pathophysiology of tricuspid stenosis
NORMAL
ABNORMAL
a
Large a wave
Rising right atrial pressure in late (right) ventricular systole
a
c
v
x
y
c
v
x
Emptying of right atrium into right ventricle
Diminution of y descent
Blood flowing into right ventricle
Blood returning to the heart in the veins is held up in the right atrium, thus producing vary large "flicking" a waves with diminution of the y descent
Stenosed tricuspid valve
Turbulent blood flow
1
2
1
2
Aortic valve closure
Mitral and tricuspid valve closure
There is presystolic accentuation of the murmur (if the patient is in sinus rhythm) caused by atrial contraction "forcing" blood through the tricuspid valve



Ventricular septal defects (VSD)

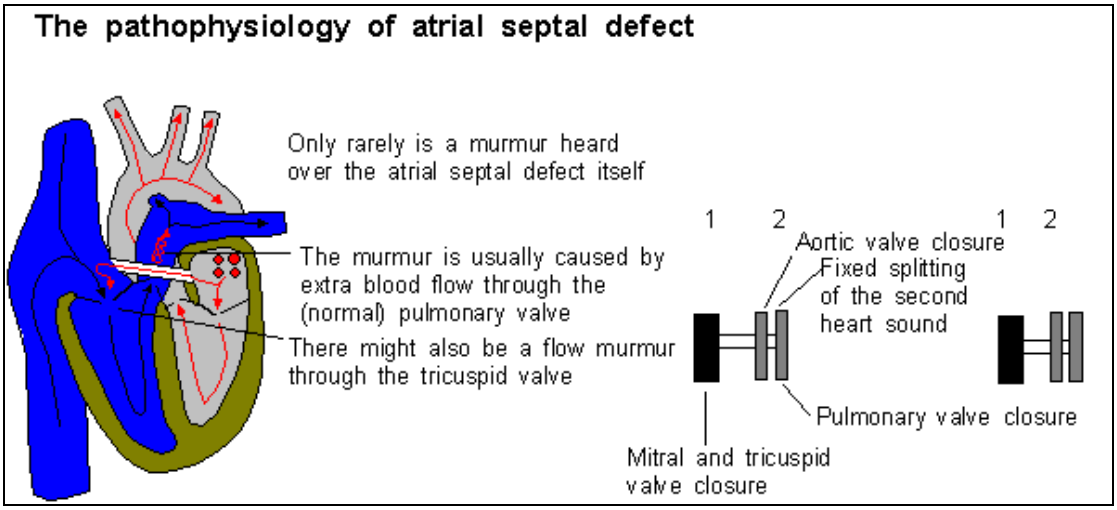


Blood is forced through the defect from left to right because left ventricular pressure is higher than right ventricular pressure. The smaller the defect, the louder and higher the pitch of the murmur. Both ventricles may become volume overloaded and may

dilate (the left ventricle because of the extra blood returning from the lungs for the second time). If there is a reversed shunt (right to left flow through a ventricular septal defect, known as the Eisenmenger complex) as may occur if there is severe obstruction to blood flow to the lungs there may be cyanosis because the deoxygenated "cyanosed" right ventricular blood enters the left ventricle and thus into the general circulation (at some stage in the development of a reversed shunt the murmur will disappear because the pressures in the two ventricles will be equal and there will be no blood flow across the defect).

Atrial septal defect

In atrial septal defects oxygenated blood returns from the lungs into the left atrium, passes through the defect into the (lower pressure) right atrium and thence (again) to the lungs. Cyanosis thus does not occur in uncomplicated atrial septal defect.



In theory atrial septal defect murmurs should be heard during ventricular diastole when the atria will be in systole (as if the murmur were exclusively caused by passage of blood through the defect between the right and left atrium). *In practice* a murmur generated at the defect itself is rarely audible because of the low pressures involved and the largeness of most defects. The murmur of atrial septal defect occurs during ventricular systole and is caused by the augmented blood volume passing through the pulmonary valve (thus simulating a pulmonary stenosis murmur). The augmented blood flow through the right heart may also cause a tricuspid flow murmur during right ventricular filling.

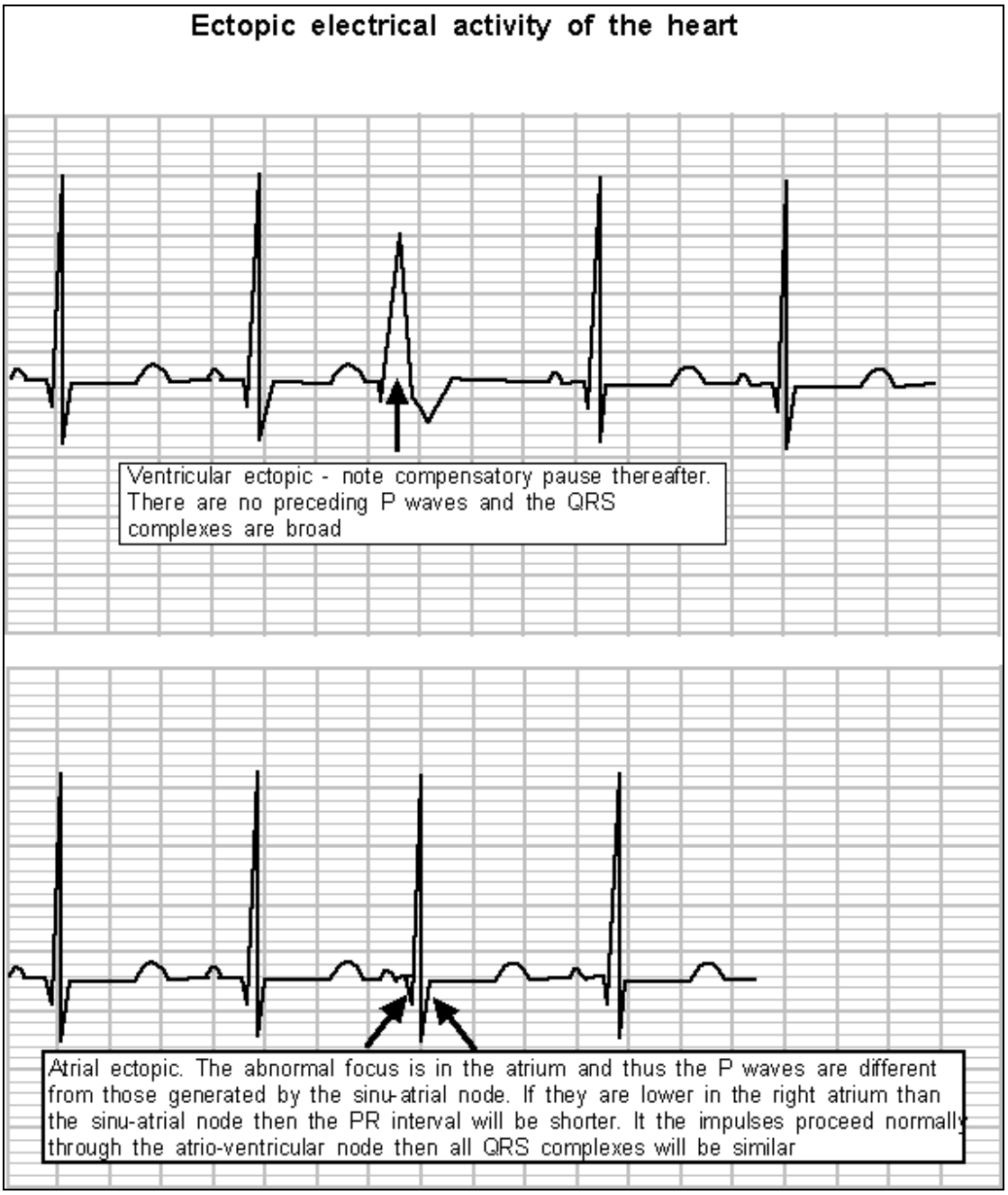
Normally the second heart sound is split in inspiration with the aortic valve closing before the pulmonary valve because of the higher aortic pressures. During inspiration more blood can be accommodated in the lungs, this extra blood passes into the right ventricle during ventricular relaxation which then caused prolonged right ventricular contraction (it takes longer to eject the increased amount of blood). The prolonged contraction time delays pulmonary valve closure. Thus a normal second heart sound may be split and the split is normally accentuated during inspiration.

With an atrial septal defect the two atria can be regarded as one chamber and blood flow into both ventricles is similar. Both ventricular contraction times are therefore similar and both valve closures are delayed equally. Thus there is a split second sound

which does not vary with respiration (fixed splitting). The right ventricle may also enlarge.

Heart rate

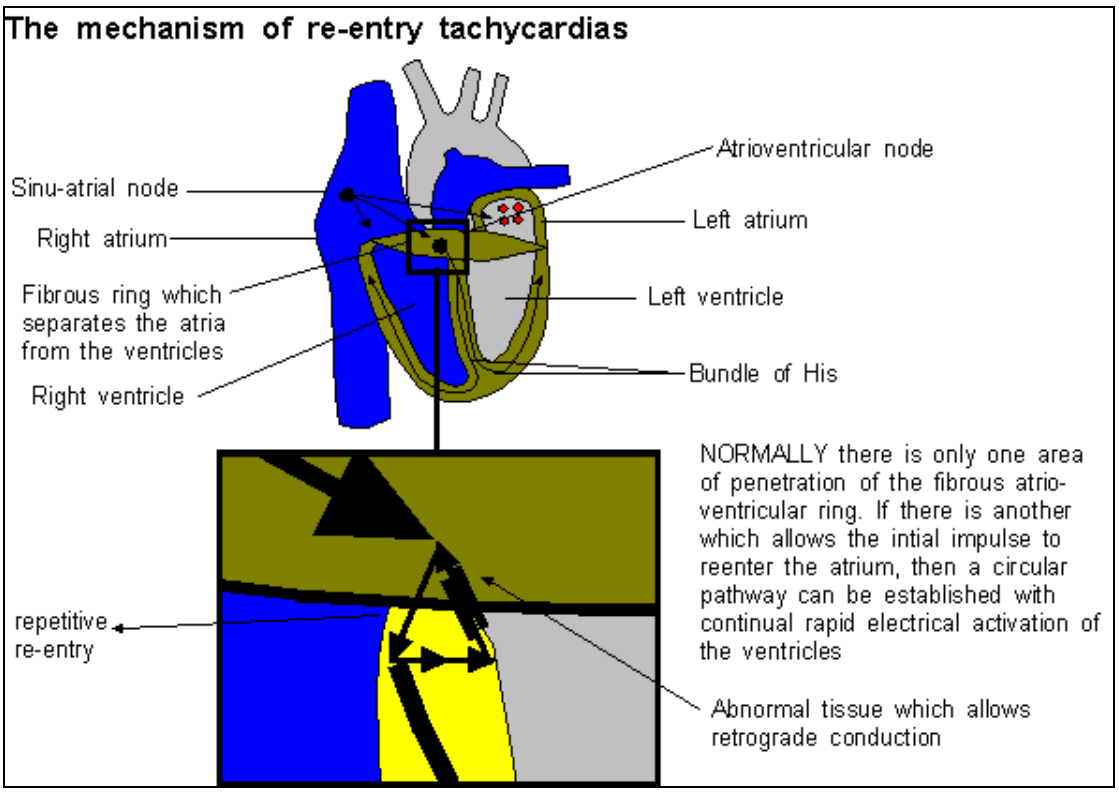
Heart muscle is intrinsically rhythmical and the supervisory contraction impulses travel via specially adapted muscle, not nerves. The contraction impulses, and thus heart rate, are usually controlled by the sino-atrial node in the right atrium. Ectopic beats (also known as extrasystoles) are contractions initiated in the atria or ventricles, atrio-ventricular node, or bundle of
His.



There is usually only one route, the atrioventricular node, by which contraction impulses can pass from the atria to the ventricles. Thereafter contraction impulses travel down the bundle of His which bifurcates with the *left* branch which divides

early, ultimately reaching the left ventricle whereas the right branch divides later to activate the right ventricle.

The normal (parasympathetic) vagal tone keeps the heart rate at about 70 per minute. A slow heart rate (bradycardia) may be caused by increased vagal tone or drug action. Sino-atrial pacemaker node dysfunction may cause an irregular absence of P waves on electrocardiography (sinus arrest). If the sinoatrial node discharges too slowly, or not at all, then other pacemakers lower down the conduction system may emerge. The electrocardiographic appearance of ectopic beats that occur when an ectopic focus discharges ae shown below.



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Fast heart rates of atrial or atrio-ventricular origin

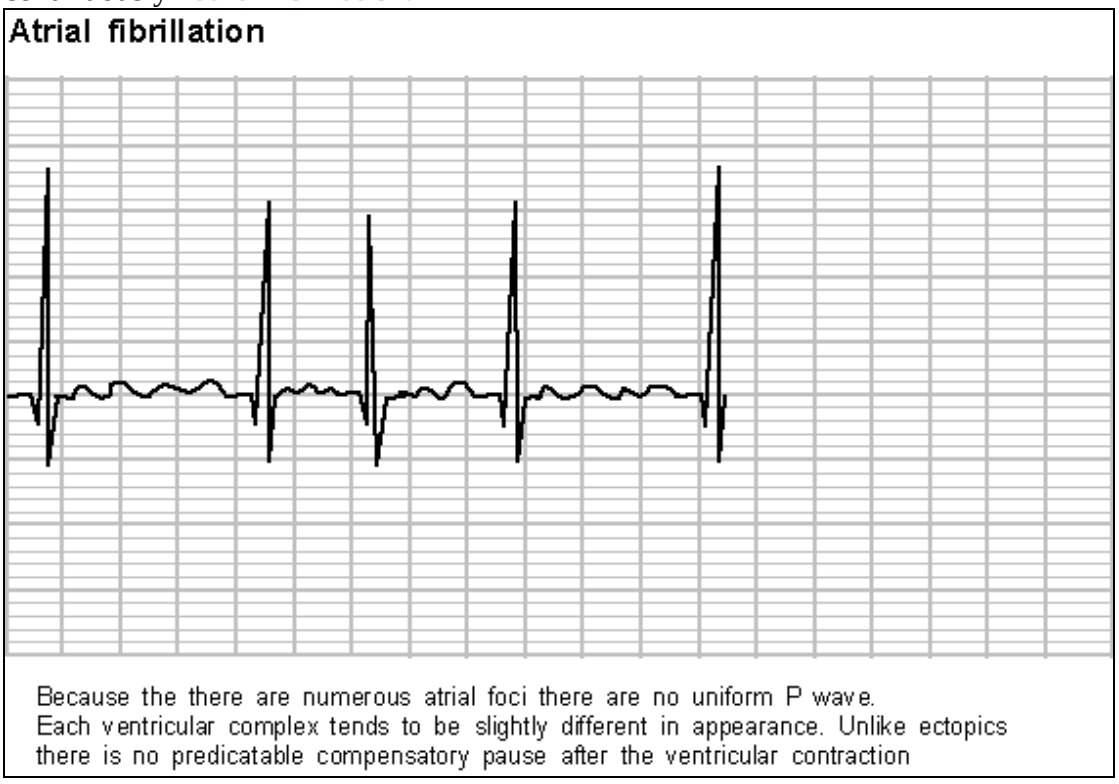
Tachycardias are heart rates above 100 per minute. There are four main mechanisms:

- Rapid sino-atrial or atrio-ventricular node discharge causing rapid repolarisation and reduction of resting membrane potential
- Premature repolarisations reducing the time between successive P waves
- Abnormal re-entry of electrical impulses
- Abnormal impulse conduction along abnormal pathways between the atria and ventricles causing premature excitation of the ventricles with a slurring of the upstroke of the R wave (a delta wave) on the electrocardiogram

Tachycardias arising from above the ventricles (supraventricular tachycardias) usually derive from the sino-atrial or atrio-ventricular nodes. The ventricular conducting systems are usually normal (with normal QRS complexes) unless there is bundle branch block.

Abnormal atrial rhythms

If multiple depolarisations arise from multiple atrial foci then the atria quiver continuously - atrial fibrillation.



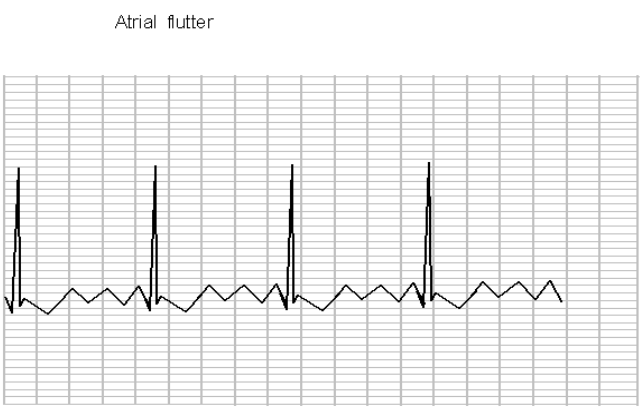
The irregularly irregular depolarisation impulses pass to the atrio-ventricular node which cannot transmit all impulses (after an impulse passes there has to be a refractory period, a delay of at least 0.35 of a second). Additionally the ventricles cannot respond to all such transmitted depolarisation waves and ventricular contractions (and therefore the pulse) become irregularly irregular. Some ventricular contractions, although palpable at the heart apex, do not give rise to sufficient pressure to give a palpable pulse at the wrist. Thus the apex rate may exceed the pulse rate at the wrist "the pulse deficit." The volume is also irregular because of variable ventricular filling at the time of ventricular contraction.

The onset of atrial fibrillation may cause marked decline in heart function especially in some left sided heart valve conditions (aortic stenosis and atrial fibrillation is a particularly ominous combination). Fibrillating atria may develop clots, especially in mitral stenosis, in association with relative stasis of the blood in the left atrium.

Therapy of recent onset atrial fibrillation attempts to restore sinus rhythm by using drugs or electric shock (direct current cardioversion). Therapy of established atrial fibrillation is by control of the ventricular rate, particularly during exertion, usually by using digoxin to maintain a resting rate of about 90 per minute. Anticoagulants may be used to prevent clot formation in the atria.

Atrial flutter is a fast discharge of an atrial focus causing fast saw-tooth P waves on electrocardiography which may revert to sinus rhythm spontaneously or may progress

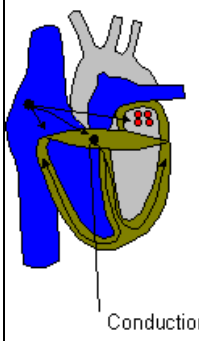
to atrial fibrillation. If the pacemaker focus is low down in the atria the P waves may be inverted.

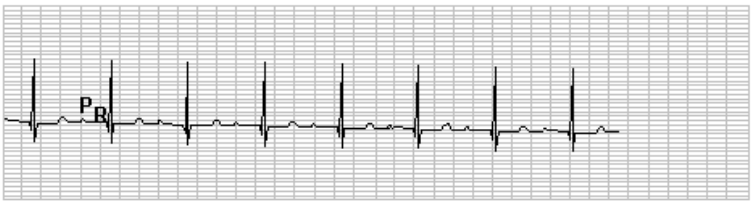


The atrio-ventricular node cannot transmit more than about 200 impulses per minute and thus only a proportion can get through, usually only one in two (less commonly one in three) impulses. Carotid sinus pressure, by increasing vagal tone may reduce the heart rate by half, or by two thirds or by one third.

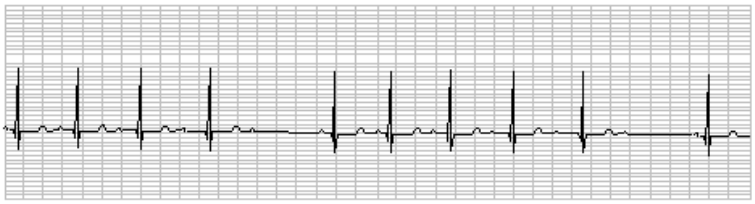
Ventricular dysrhythmias

The various types of heart block



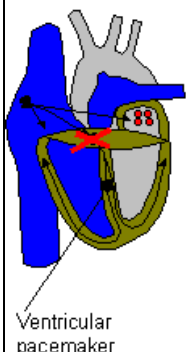


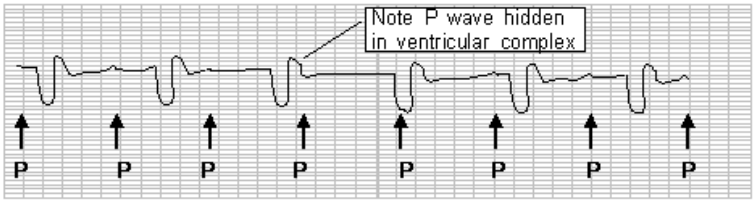
FIRST DEGREE HEART BLOCK. PR interval greater than 0.2 seconds



SECOND DEGREE HEART BLOCK

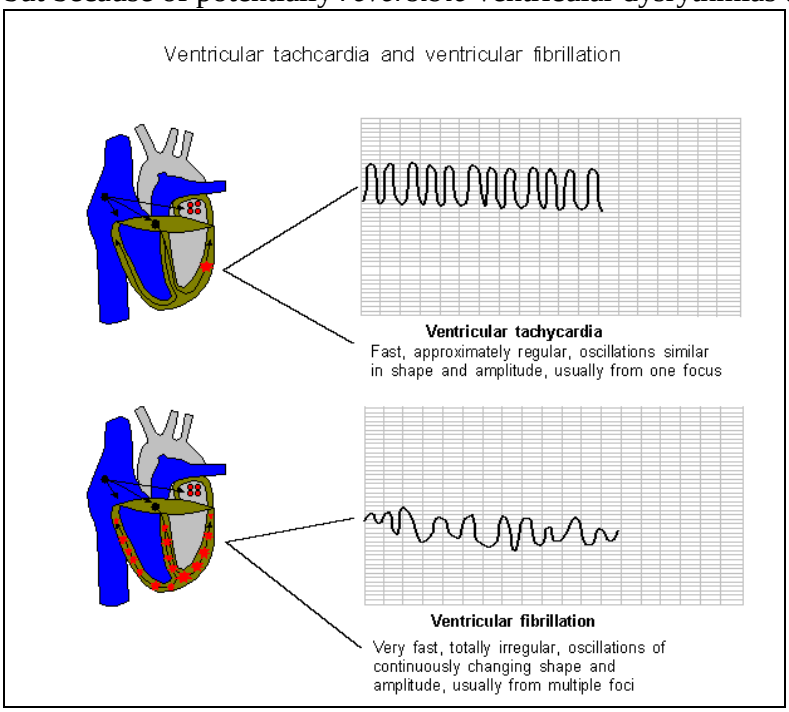
Progressive increase in the PR interval until a P wave fails to elicit a ventricular contraction. The cycle then repeats itself. Usually the problem is in the atrioventricular node. It may progress to third degree heart block. Another type of second degree heart block occurs when an occasional P wave fails to elicit a ventricular contraction





The atrial impulses fail to reach the ventricles (sometimes the atrio-ventricular node becomes the pacemaker but usually a ventricular focus becomes the pacemaker, usually at a regular rate of about 40 beats per minute. In the case illustrated there is a single ventricular focus but multiple foci may develop. In effect the heart beats are initiated by a succession of ventricular ectopics (if this did not happen the heart would stop beating)

Dysrythmias caused by ventricular foci usually have broader QRS complexes than atrially derived impulses and include ventricular tachycardia, a rough saw tooth pattern with a rate of 120-220 per minute. Ventricular fibrillation is rapid irregular uncoordinated electrical activity probably caused by multiple sites of electrical discharge in the ventricles.Ventricular dysrhythmias may cause a precipitous fall in cardiac output with loss of consciousness possibly with a fatal outcome. Each may be "sparked off" by ectopic beats, especially after myocardial infarction. Most people who die after myocardial infarction die not because of the extent of heart muscle death but because of potentially *reversible* ventricular dysrythmias that occur.



Atrioventricular heart block

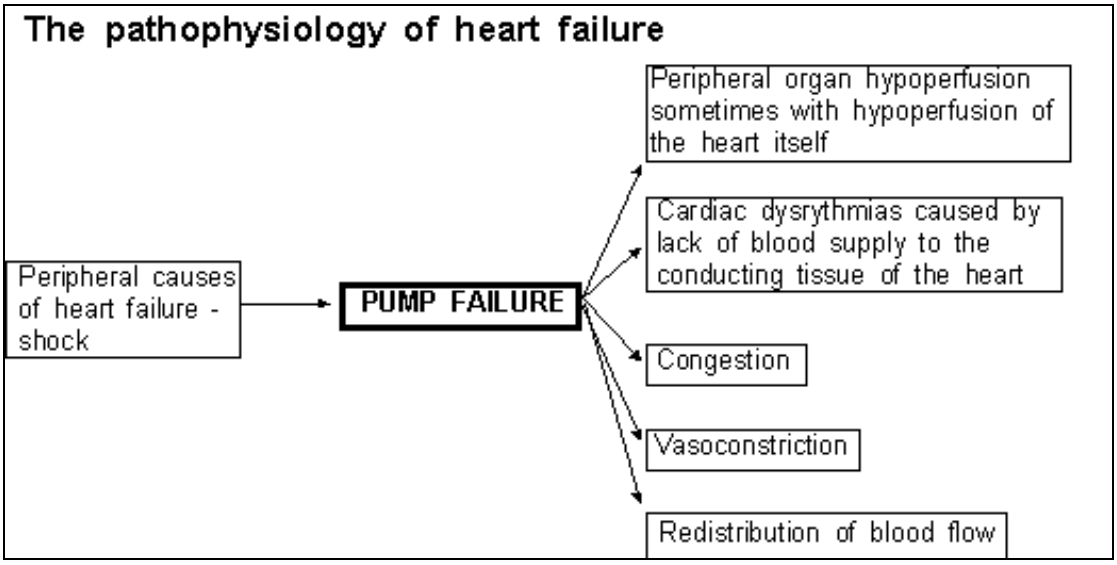
With complete heart block atrial activity is normal but impulses are blocked at atrioventricular node or Budle of His level and do not enter the ventricular muscle. The ventricles then contract at their own rate, about 40 per minute. If the atria (which usually remain in sinus rhythm) continue to contract they may occasionally contract when the (electrically independent) ventricles are contracting thus causing abrupt surges of blood into the jugular veins (known as cannon waves).

Stokes-Adams attacks are abrupt unpredictable episodes when there is no cardiac output usually associated with acute onset complete heart block. Death will occur unless intrinsic ventricular foci emerge or the heart block remits. Patients collapse and go pale because of lack of blood supply then flush as blood surges through the empty arteries which have been dilated by carbon dioxide accumulation during the circulatory stoppage.

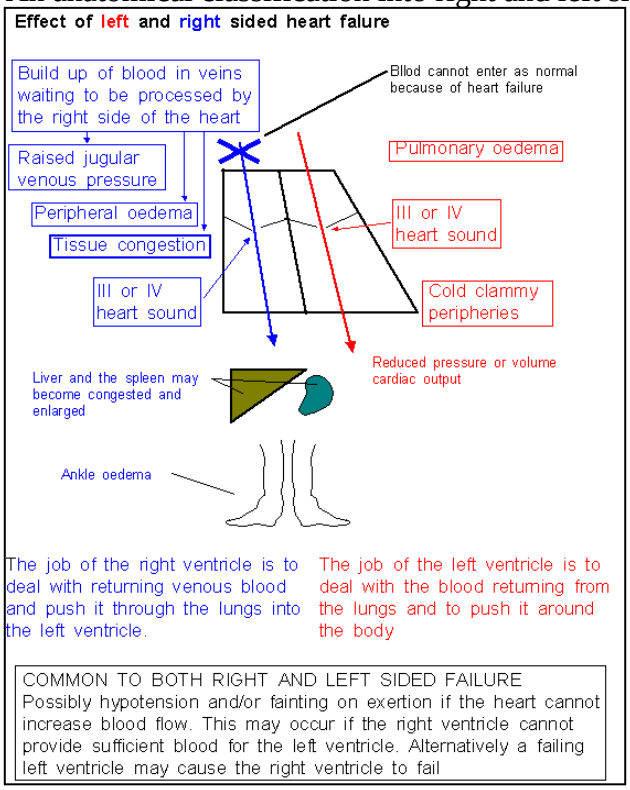
Heart failure

Primary heart failure is circulatory failure caused directly by disorders of the heart itself "pump failure" and excludes disorders of the blood, blood vessels or over-transfusion (although these may cause a secondary failure of the heart). The heart has great physiological reserve and problems are only evident if extensive damage has occurred to the heart itself or if the heart is put under secondary stress. The effects of

heart failure are shown
below.



An anatomical classification into right and left sided heart failure is clinically useful.



Venous blood returning from the body builds up in the right atrium and jugular veins if the **right** ventricle fails to pump sufficient blood through the lungs. This may happen if the lungs have been damaged by certain chronic respiratory diseases (cor pulmonale) or if the right ventricle or its valves are dysfunctional. Abrupt right heart failure produces a raised jugular venous pressure, and often a low blood pressure (because of circulatory impairment). In such situations a raised jugular venous pressure may help to drive the failing right heart.

If the **left** ventricle fails to eject blood returning to it from the lungs into the aorta and the right ventricle continues to pump blood into the lungs then blood builds up in the lungs to initiate pulmonary oedema (this may happen if part of the left heart muscle is dysfunctional or dies), or if there is left heart valve dysfunction or severe hypertension.

Left ventricular failure results in:

- A low blood pressure often with a secondary rise in the heart rate. This fall in blood pressure may be measured using a sphygmomanometer and/or be clinically obvious if the patient faints on standing (because insufficient blood perfuses the brain)
- Pulmonary oedema which forms because the left ventricle cannot deal with blood returning in the pulmonary veins, the pulmonary venous pressure to rise, and when the osmotic pressure is exceeded causing fluid to exude into lung tissue, particularly from the walls of the small airways and alveoli. Gas exchange is thereby impaired
- Cool peripheries caused by diminished peripheral blood flow and reactive arterial contraction in an attempt to maintain the blood pressure
- Patients may be cyanosed as the slow circulation through peripheries enables tissues to extract more oxygen from the haemoglobin in the red blood cells
- Kidney function may decline because of low blood flow or low blood pressure.

Left ventricular failure can cause the right ventricle to fail, either by failure to deal with blood arriving from the right ventricle and/or because an enlarged or dilated left ventricle encroaches into the right ventricular space within the limiting size of the pericardium.

There are two main types of heart failure. *Systolic dysfunction* is mostly caused by deficient blood supply to the ventricles resulting in a reduced left ventricular contractility with a reduction in ventricular ejection. *Diastolic dysfunction* is caused by ventricular hypertrophy or stiffness which results in impaired diastolic relaxation. These patients have normal ventricular ejection of blood but the internal pressures in the ventricles rises disproportionately.

The work for the heart in "systolic type" heart failure can be reduced by agents which either dilate the systemic circulation to decrease afterload (ACE inhibitors), or increase the effectiveness of heart muscle contraction (digoxin), reduce intravascular volume (diuretics) or cause an expanded vascular volume to accommodate the extra fluid that is being retained, thereby taking the strain off the heart (angiotensin converting enzyme inhibitors also dilate the veins to achieve this). With diastolic dysfunction there is less scope for therapeutic intervention, which essentially should be to try to treat the underlying cause. Attempts to increase a poor blood supply to the heart may be one option.

There are several compensatory adjustments that the body can undertake to compensate for a failing heart.

- Fluid and salt retention which increases preload to "drive the heart"
- Sympathetic nervous system activation which increases the rate and force of cardiac contractions
- Activation of the renin-angiotensin system
- Selective diversion of blood to vital organs

If these mechanisms do not solve the problem then their failed attempts may become counterproductive

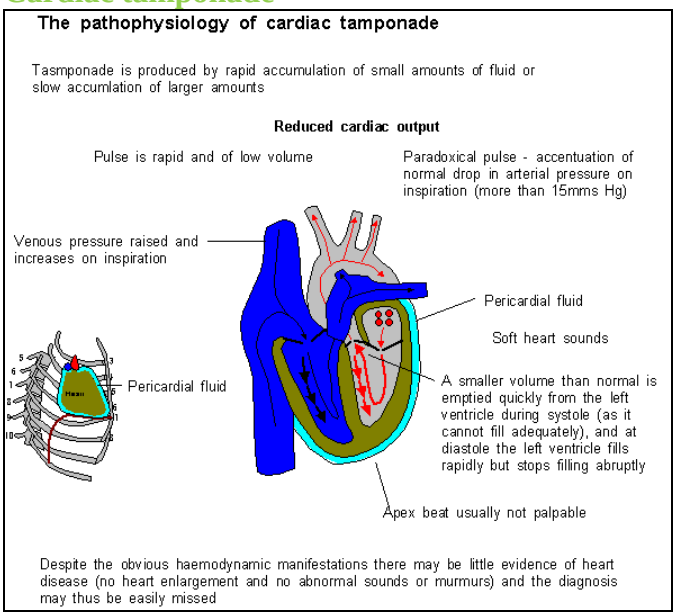
- Excessive fluid and salt retention may cause tissue oedema which 1) impairs tissue perfusion, 2) impairs absorption from the oedematous gut possibly leading to hypoalbuminaemia, 3) causes tissue hypoxia which worsens the function of almost everything or 4) combinations of these
- Vasoconstriction caused by increased sympathetic activity to maintain blood pressure may impair renal function if the heart cannot meet the challenge of an increased peripheral resistance - too high an afterload.

PATHOPHYSIOLOGY OF SOME OTHER HEART CONDITIONS

Congential heart disease

In *cyanotic* congenital (= existing from birth) heart disease there is excess deoxygenated blood in the systemic circulation (because some blood bypasses the lungs), whereas in *non-cyanotic* congenital heart disease blood does go through the lungs (sometimes twice) but at the expense of the systemic circulation.

Cardiac tamponade



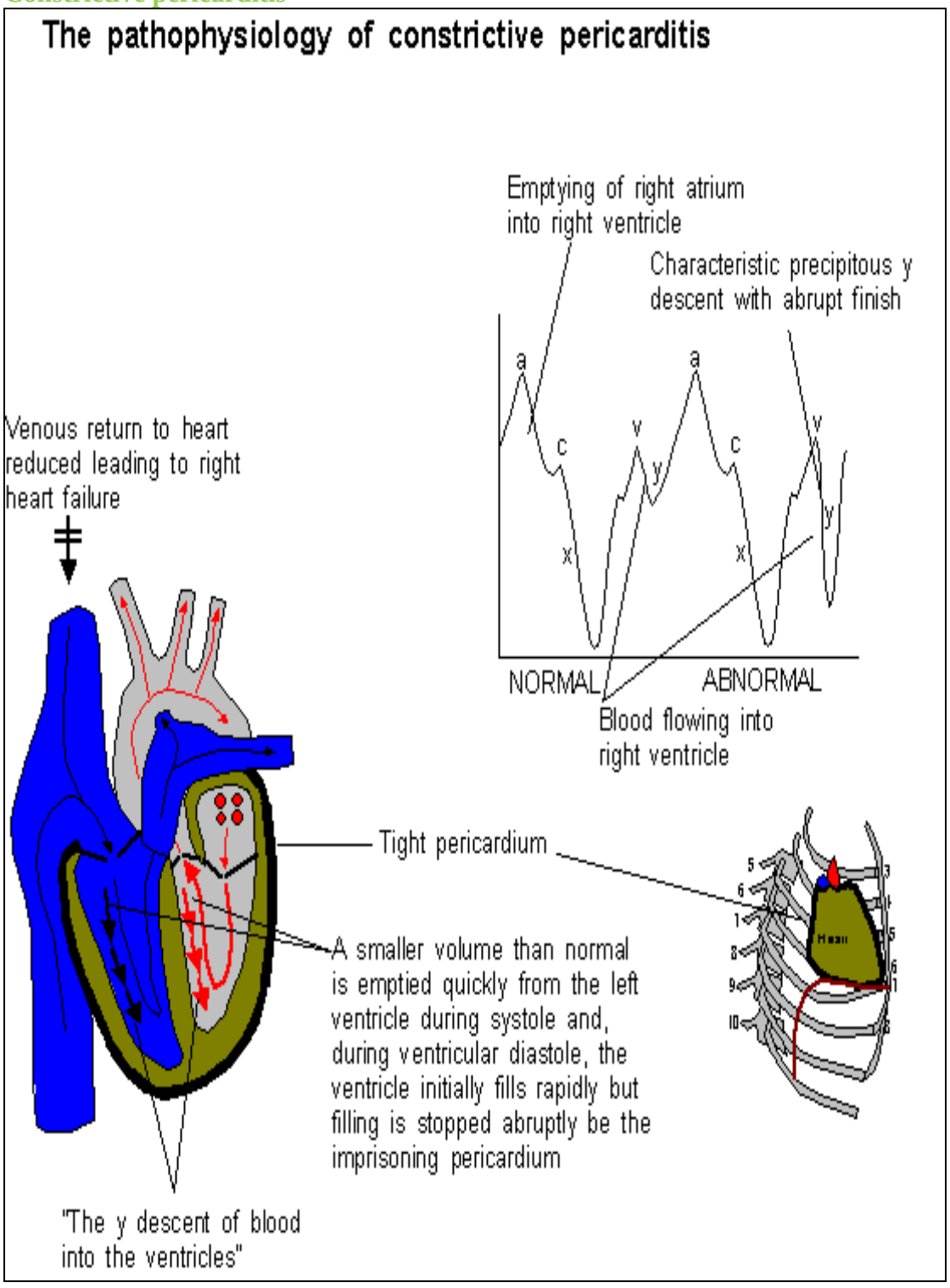
Fluid accumulates between the two layers of pericardium. This fluid preferentially compresses the right atrium and ventricle (which are at a lower internal pressure and thus more easily compressed than their left sided counterparts). Impaired filling of the heart, predominantly the right side during ventricular relaxation, is impaired. The neck veins become distended particularly with inspiration as the expanded lungs compress the heart further. Also less blood returns from the lungs to the left heart and the pulse volume therefore falls in inspiration (pulsus paradoxus). The blood pressure will be low and the heart rate fast because the heart attempts to maintain output by increasing the number of fixed volume contractions. Treatment is usually by drainage of the fluid.

Cardiomyopathy

With *hypertrophic* cardiomyopathy there is hypertrophy of the ventricular septum or ventricular muscle. Ventricular contraction is distorted and ventricular filling during ventricular diastole may be impaired. There may be an abrupt carotid pulse because there is an abrupt obstruction to left ventricular emptying. With *restrictive* cardiomyopathy ventricular filling is restricted because of ventricular muscle

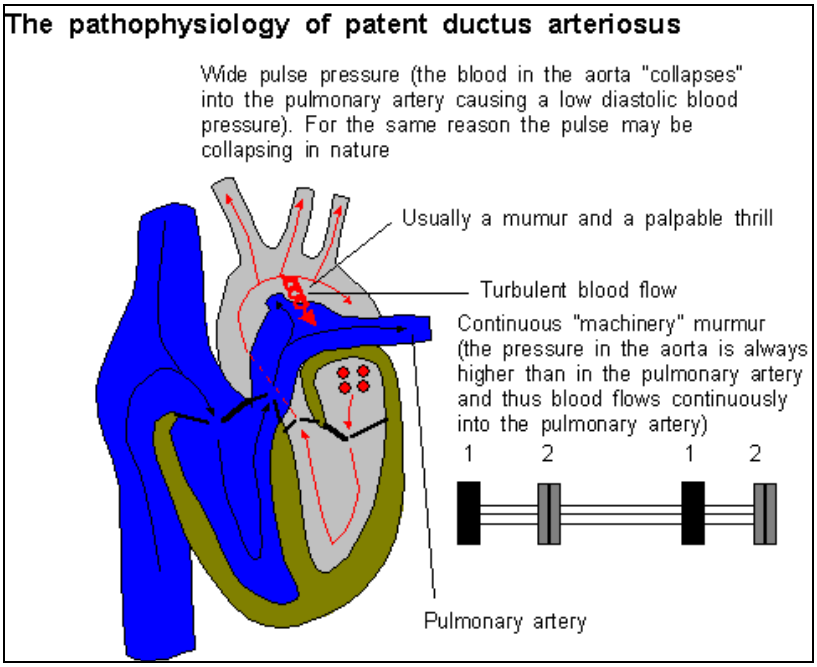
abnormality. This produces symptoms and signs similar to those of constrictive pericarditis.

Constrictive pericarditis



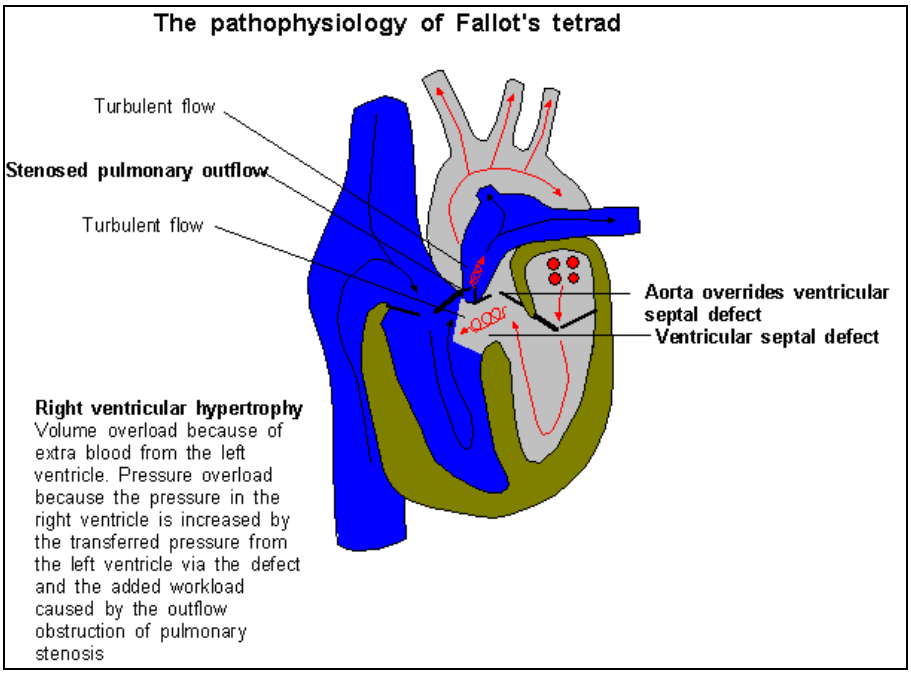
The heart is imprisoned by the tight outer layer of pericardium.

Patent ductus arteriosus



If the ductus arteriosus fails to close at birth then blood will flow *continuously* from the aorta into the pulmonary veins because the aortic pressure after birth is higher than pulmonary vein pressure at all stages of the cardiac cycle. Blood in the aorta is always fully oxygenated and therefore patients do not develop cyanosis. There is a wide pulse pressure because the aortic diastolic pressure is lower than normal due to the continued drainage of blood from the aorta into the pulmonary artery.

Fallot's tetrad



This illustrates one congenital heart problem which is explicable on an embryological basis. The basic feature is that the truncus does not divide appropriately into the aorta and pulmonary arteries with:

- The pulmonary artery being poorly developed
- The aorta straddling the interventricular septum
- Failure of the interventricular septum to develop fully resulting in a ventricular septal defect
- Pressure in the left and right ventricles tends to be equal (a combined effect of the ventricular septal defect and the pulmonary stenosis) with the right ventricle thus becoming hypertrophied

Infective endocarditis

Micro-organisms, almost invariably bacteria, infect heart valves, causing them to be incompetent. Infective endocarditis may have a rapid onset when caused by virulent micro-organisms which may affect normal heart valves. Infective endocarditis caused by less virulent micro-organisms may develop slowly and usually only affects previously damaged valves. Right sided endocarditis is a particular risk for intravenous drug abusers who usually inject into veins.

Ischaemic heart disease

Ischaemia (= a local and temporary deficiency of blood caused by interruption of the blood supply) is almost always associated with coronary artery atheroma. Intrinsic heart muscle disease or overdevelopment of ventricular muscle caused by overwork can outstrip the available blood supply to cause ischaemic symptoms.

Heart muscle has a limited ability to metabolise without oxygen and increased oxygen demands caused by increased work have to be met by increased blood flow brought about by coronary artery vasodilatation (which may be able to compensate for narrowing caused by atheroma).

When heart muscle is starved of oxygen angina (a pressing, constricting discomfort or pain which is brought on by exertion and relieved by rest) may result. Angina may also be felt in the left arm, which has a similar somatic nerve supply as the autonomic supply to the heart. Treatments of an anginal attack includes stopping the causative activity, sitting down and using a nitrate preparation which dilates peripheral arteries and thereby reduces the afterload.

Myocardial infarction

Myocardial infarction (infarction = muscle death) occurs when ischaemic heart muscle dies. Myocardial infarction causes about 200,000 deaths a year in the United Kingdom. Symptoms may be similar to angina but may begin at rest, last for longer (more than 20 minutes) and be more severe. With full thickness myocardial infarction there are usually deep Q waves, ST elevation and T wave inversion.

Treatment of myocardial infarction may include:

- Pain relief
- Aspirin to reduce clotting
- Early attempts to dissolve the thrombus (thrombolysis)
- Interventions include drugs to dilate blood vessels to reduce heart workload, and drugs that reduce heart work by direct action on heart muscle

Pregnancy and the heart

Pregnancy may exacerbate heart failure because in pregnancy there is:

- An increased cardiac output
- An increased blood and extracellular volume
- A slightly increased heart rate
- A 30 percent increase in heart oxygen requirement