Paramedic Resource Manual

CARDIOVASCULAR SYSTEM

SECTION THREE

2014 Update by
Ontario Base Hospital Group Education Subcommittee

OBJECTIVES: CARDIOVASCULAR SYSTEM

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. discuss the features of cardiac muscle and relate these features to the function of cardiac muscle.

2. discuss the electrophysiology of cardiac muscle which differentiates it from other muscle tissue.

3. describe the conductive pathways in the heart and discuss the process of impulse conduct ion through these pathways.

4. define cardiac output and discuss the factors that affect its control.

5. describe the structure and function of arteries and veins.

6. discuss the role of the autonomic nervous system in relation to the cardiovascular system.

7. discuss the factors affecting the control of blood pressure.

8. discuss the effects of the exercise on the cardiovascular system.

9. relate to above objectives to common clinical situations.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.

GLOSSARY

BLOOD FLOW	The quantity of blood (in millilitres or litres) that passes a given point in a period of time (minute).
BLOOD PRESSURE	Force exerted by the blood against any unit area of the vessel wall.
CARDIAC CYCLE	A complete heartbeat, consisting of the contraction and relaxation of both atria, and the contraction and relaxation of both ventricles.
CARDIAC OUTPUT	The amount of blood pumped by the ventricles in one minute. Normally approximately 5 litres (70 mL/kg). Cardiac output = stroke volume x heart rate.
CONDUCTION SYSTEM OF THE HEART	Consists of the SA node, the internodal pathways, the AV node, the bundle of His, bundle branches and the Purkinjie fibers.
DIASTOLIC PRESSURE	The blood pressure during diastole. Diastolic pressure reflects the state of constriction of the blood vessels.
ECG	A graphic record of the electrical activity of the heart.
FRANK STARLING'S LAW OF THE HEART	Within limits, the longer the heart muscle fibers are stretched at the beginning of contraction, the stronger is their contraction.
PERIPHERAL RESISTANCE	Resistance to blood flow imposed by the force of friction between blood and the walls of the vessels – depends on the viscosity (thickness) of the blood and the diameter of the vessel. Also called afterload. Mathematically, resistance depends on: $\frac{\text{viscosity x length of vessel}}{\text{radious}^4}$
PULSE	The alternate expansion and recoil of an artery.
PULSE PRESSURE	Systolic pressure minus diastolic pressure.
STROKE VOLUME	The amount of blood ejected from the ventricle with a single contraction of the ventricles
SYSTOLIC PRESSURE	The peak in blood pressure during systole. Systolic pressure is a reflection of blood volume.

INTRODUCTION

The cardiovascular system, consisting of a pump (the heart), and a distribution and collection system (arteries and veins), performs a number of important functions in the body including:

1. Transportation of oxygen and other nutrients to the cells.

2. The removal of carbon dioxide and other waste products from the tissues.

3. Distribution of hormones and other substances required for physiological control.

4. Control of heat transfer.

Because the system must perform effectively under a wide variety of circumstances, it must have mechanisms to adapt to the changes that occur. Tissue needs very with activity, and the cardiovascular system adjusts to these needs in a variety of ways.

A basic knowledge of the anatomy and physiology of the cardiovascular system is not just of theoretical interest. It is essential in dealing with clinical situation, such as:

- interpretation of vial signs
- recognition of shock states
- management of the patient with cardiovascular disease
- interpretation of electrocardiograms
- understanding of the role and effects of medications.

The first step in treating a patient is recognition of the abnormalities that are present. To do this effectively, one must first understand normal function and adaptive mechanisms.

THE HEART AND ITS CONDUCTION SYSTEM

The heart is a four-chambered, muscular organ, approximately the size of a closed fist, which lies in the thorax at the level of the fifth to eighth thoracic vertebrae. There are three layers to the heart:

- endocardium (lining)
- myocardium (muscle)
- epicardium (outer layer).

The heart consists to two atria and two ventricles. Functionally the ventricles are the main contractile force of the pump; the atria act primarily as entrances to the ventricles, although they also pump weakly to move the blood into the ventricles. As 70% of the blood passes into the ventricles before atrial contraction, this role is a minor one.

The Atrio-ventricular valves (AV valves), the mitral valve and tricuspid valve, have guide-wires called chordae tendinae attached to the edges of the valve leaflets and anchored to the papillary muscles which attach to the ventricular walls. These attachments prevent the valves from being forced back into the atria (regurgitation) during ventricular contraction.

Clinical vignette

Occasionally in acute myocardial infarction (AMI) a papillary muscle may infarct and rupture suddenly, leading to inability of an AV valve to close properly during ventricular contraction. Consequently, blood leaks back into the atria (regurgitation), and when this occurs with the mitral valve, regurgitation combined with impaired left ventricular contraction in the setting of AMI may lead to blood backing up into the lungs which may lead to acute pulmonary edema (APE).

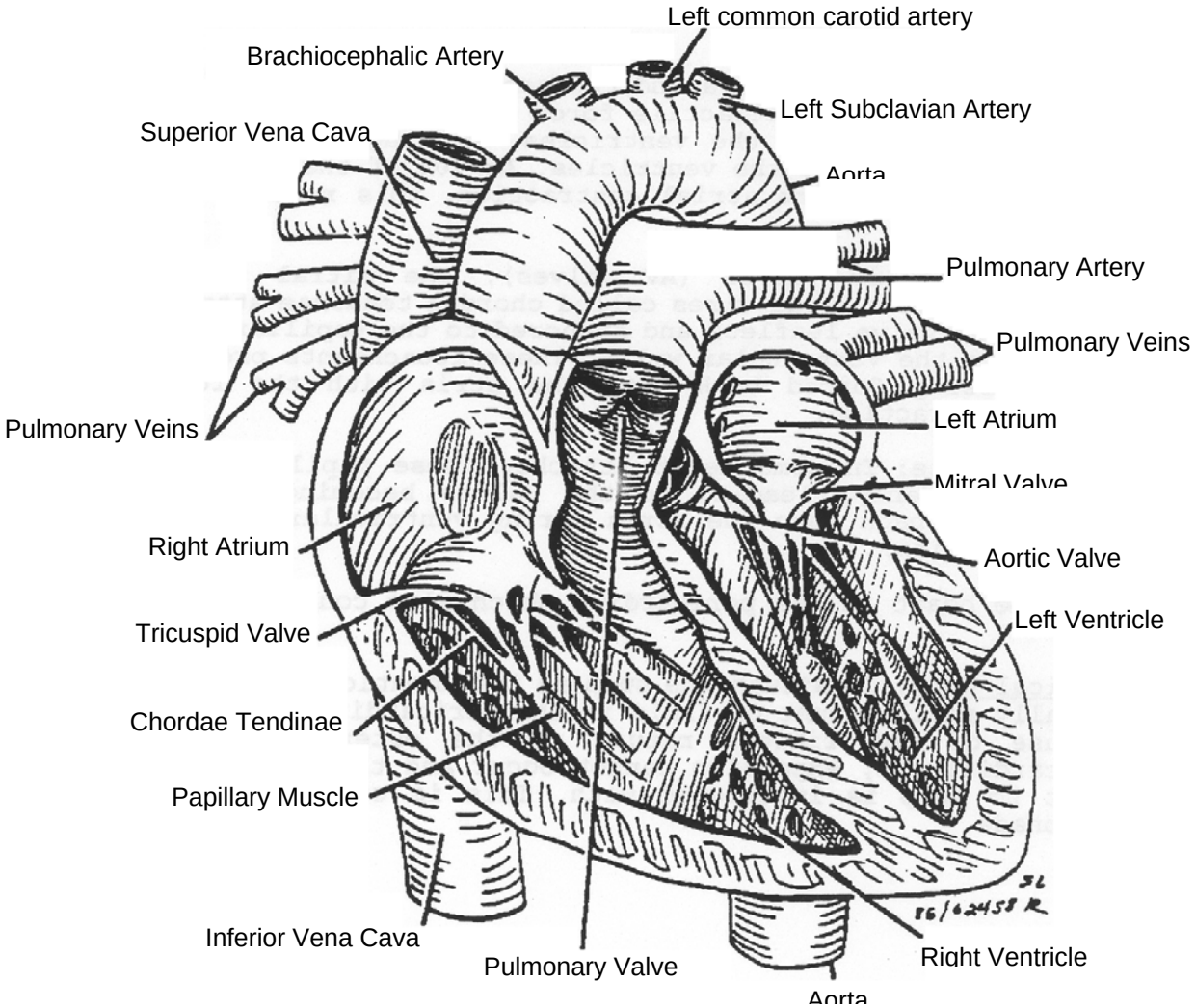
Enclosing the heart is the pericardium which also consists of three layers.

Clinical vignette

In situations such as inflammation or trauma, the normally thin layer of serous fluid in the pericardial space may increase. Because of the inelastic fibrous nature of the outer layer of the pericardial sac, stretching of the pericardium to accommodate fluid build up is limited and may begin to interfere with the heart's ability to pump. This is called pericardial tamponade.

Chronically, as much as 1-2 liters may accumulate in the pericardial sac. Acutely however, a sudden increase in pericardial fluid by as little as 150 ml may be fatal.

FIGURE 1: THE HEART – VALVES, CHAMBERS, MAJOR VESSELS



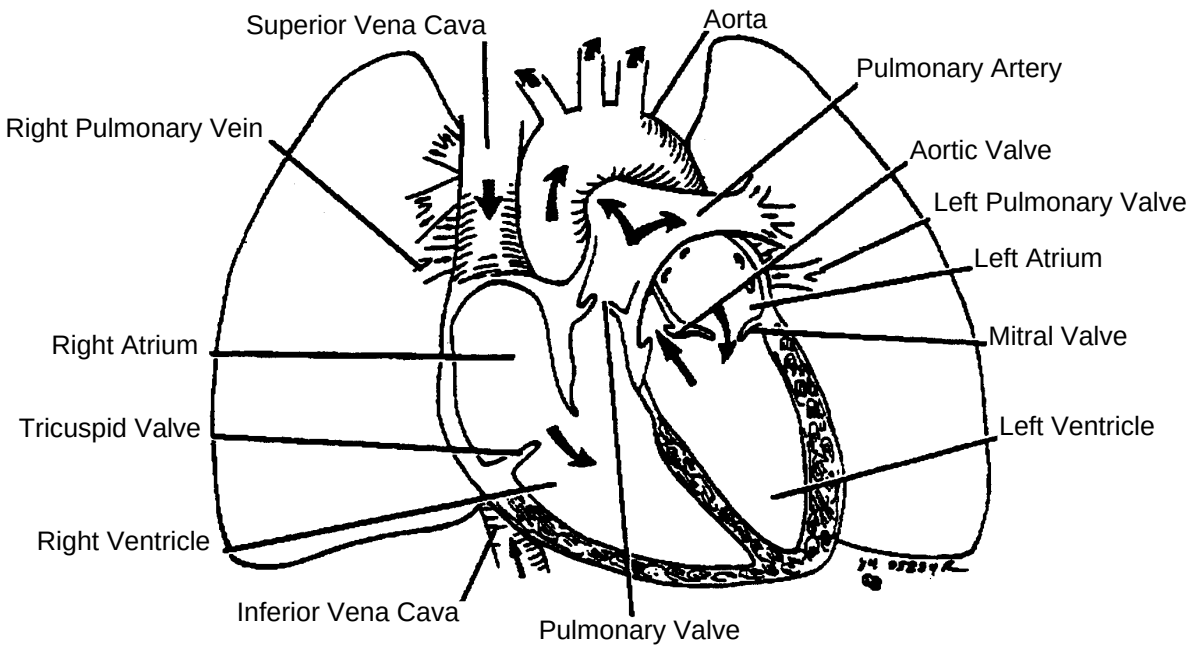
The right atrium receives blood from the superior and inferior vena cavae, which forms most of its upper and lower walls. It also receives the blood from the coronary veins. From the right atrium, blood passes into the right ventricle through the tricuspid valve, and from there it is pumped a short distance through the pulmonary valve into the pulmonary artery to the lungs. Blood then passes through the lungs for oxygenation.

From the lungs, the blood passes into the heart via the four pulmonary veins which enter the left atrium laterally. The blood then passes through the mitral valve into the left ventricle and out the aortic valve to the systemic circulation. The origin of the coronary arteries, which supply the blood to the heart, lies just distal to the aortic valve.

Clinical vignette

Because the right ventricle pumps blood only a short distance to the lungs, it has a much thinner wall than the left ventricle. Consequently, the right ventricle's force of contractility is very dependant on changes in preload (Starling's Law – i.e. within physiological limits, the great the heart muscle is stretched, the greater its force of contractility.

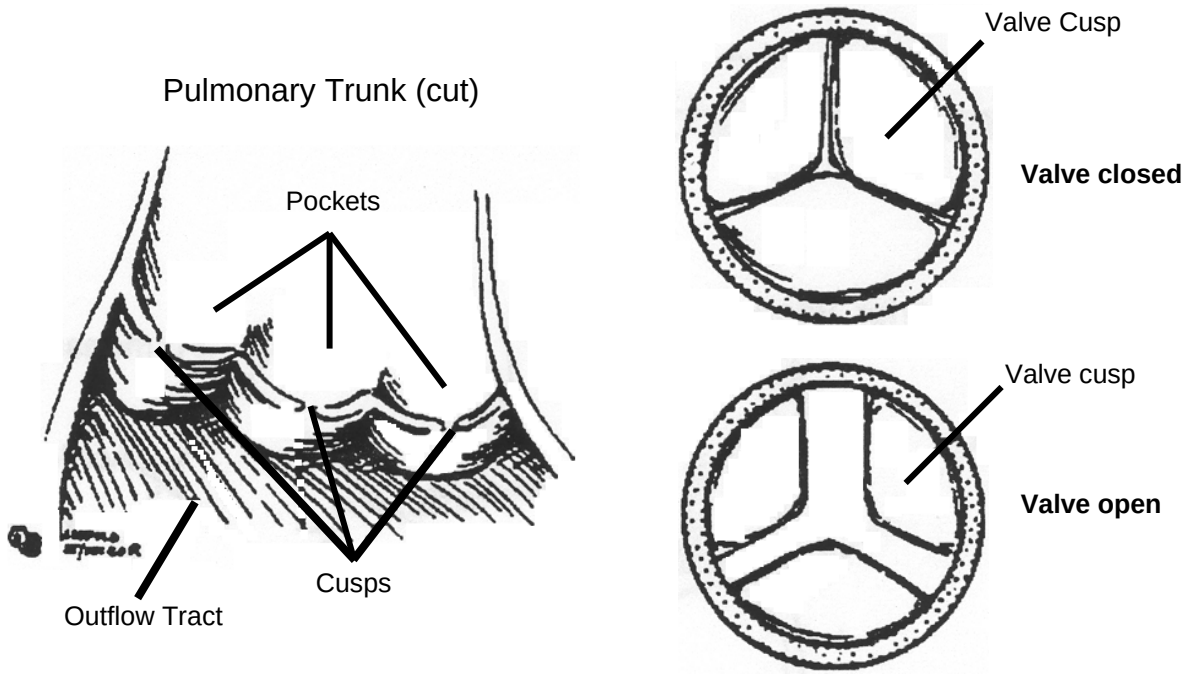
FIGURE 2: FLOW OF BLOOD THROUGH THE HEART



The four valves of the heart function passively, dependent on pressure gradients for their opening and closure. The mitral valve has two leaflets, the others three. The aorta and pulmonary arteries are dilated just distal to the valves (forming out-pouchings called sinuses), to prevent the leaflets from sticking to the walls of the vessels.

The normal heart sounds results from the vibrations of the closure of the valves. The first heart sound is from the closure of the atrio-ventricular valves and the second heart sound is from the closure of the aortic and pulmonary valves.

FIGURE 3: PULMONARY VALVE



NEUROMUSCULAR ELECTROPHYSIOLOGY

The heart consists of three types of muscle – atrial, ventricular and specialized conductive tissue. The muscles of the atria and ventricles are like skeletal muscle in most ways, but distinguishable by the presence of intercalated disks between the cells. These disks allow impulse to travel from cell to cell faster than in other parts of the body. The specialized conductive tissue contracts minimally and transmits impulses at a faster rate.

Electrical potential exist across all cell membranes. Some cells, such as those in nerve and muscle, can transmit impulses along their membranes, i.e. they are excitable.

All cell membranes are surrounded by electrolyte solutions, inside and outside, containing 155 mEq/L of anions (negatively charged ions) and cations (positively charged ions). Excesses of anions over cations immediately inside the cell membrane and of cations over anions immediately outside the cell membrane, generate the membrane potential. This can be electrolyte solutions in a car battery. The excesses of anions and cations occur as a result of active transport or diffusion.

An example of active transport is the sodium pump, which actively transports sodium from the inside of the cell to the outside of the cell. Because sodium is a cation, the transport leads to a positive charge on the outside of the cell and a negative charge on the inside of the cell.

Diffusion depends on the permeability of the membrane (ability of a substance to pass through the membrane) and the concentrations of various ions on either side of the membrane.

At rest, by means of the sodium and potassium pumps and diffusion, the resting membrane potential is approximately - 85 mV (millivolts), with a higher concentration of potassium inside the cell and a higher concentration of sodium outside the cell.

Factors that can create such a change include:

- electrical stimulation
- chemicals
- mechanical damage to the cell
- heat, cold.

At the time of the action potential, sodium permeability changes suddenly. Sodium enters the cell leading to the inside of the cell becoming positive. Sodium is then pumped back out of the cell, returning the cell membrane to its resting state.

Sodium, potassium, and the other electrolytes diffuse through pores in the cell membrane called channels. The change in the permeability to sodium, as occurs with an action potential, is related to the opening of gates that are sensitive to changes in electrical current. These gates or channels are normally closed. Calcium is felt to play a role in closing the gates which control access to the sodium channels. Channels also exist for calcium and magnesium, a familiar concept because of the recent development and use of calcium channel blockers in cardiac patients, e.g. Diltiazem.

In excitable tissue, an action potential at any one point on the cell membrane leads to the adjacent portion of the membrane becoming excited as well. Thus, the action potential along the cell is transmitted which leads to the creation of an impulse. Once an impulse has been generated, it will travel along the entire membrane. If, however, the fiber is in an abnormal state at the time the impulse reaches it, the action potential may not generate sufficient voltage to stimulate the adjacent area – e.g. as seen in heart block dysrhythmias.

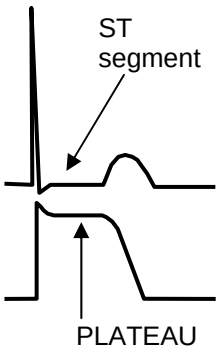
The repolarization process involves the sodium and potassium pumps and is thus an energy requiring process.

Some excitable tissue does not repolarize immediately, but remain on a plateau for a few milliseconds before repolarization occurs. This is the situation with cardiac muscle resulting in contraction of the heart muscle for the duration of the plateau.

All excitable tissues can discharge repeatedly if the threshold for stimulation is reduced to a low enough level. All muscles

Clinical vignette

The action potential represents depolarization of a single cell. When you compare the action potential to the electrocardiogram which looks at depolarization of the entire myocardium, you can see that the Plateau phase (continuous influx of calcium) occurs during the ST segment of the ECG. In other words, the heart is in a state of contraction during this brief period to eject all of the blood from the ventricles.



do this when the calcium concentration is low enough. Repetitive discharges normally occur within smooth muscle and the heart.

The membrane, in the resting state of a pacemaker cell is constantly permeable to sodium. Sodium slowly enters the cell, lessening the negativity within the cell. A point is reached where the current inside the cell is sufficiently positive (reaches threshold) to generate an action potential (depolarize).

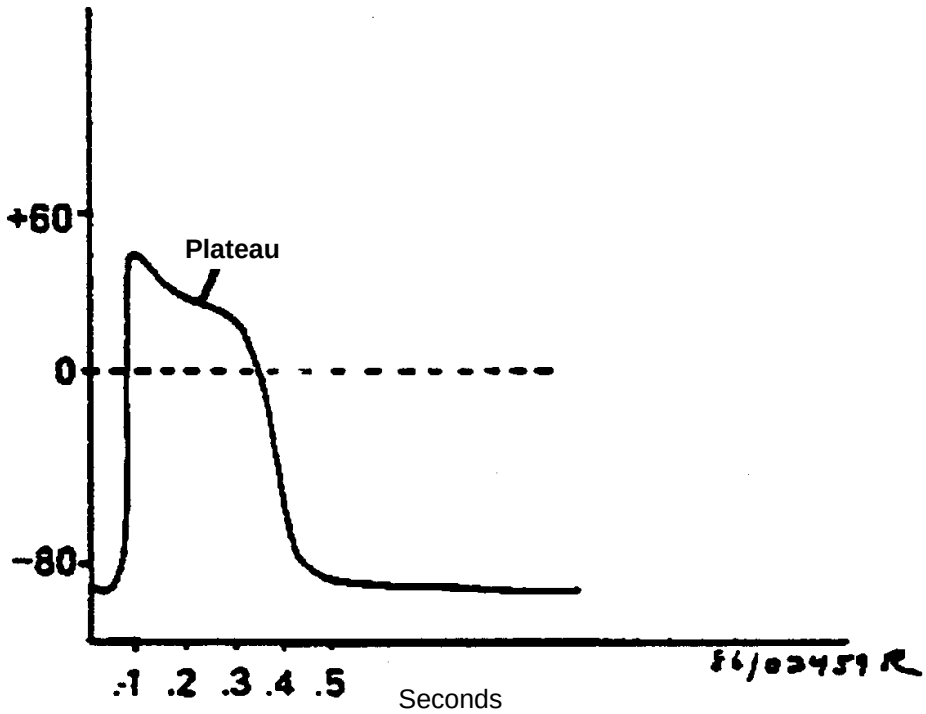
Pacemaker cells, such as the sino-atrial (SA) Node, have a tendency to generate action potentials rhythmically thus resulting in rhythmic contraction of the heart.

Cardiac muscle has a refractory period which is a period immediately after the action potential when the muscle cannot respond to another impulse. The refractory period for ventricular muscle is longer than that for atrial muscle.

Clinical vignette

Because of the difference in refractory time, the atria can beat faster than the ventricles. The Atrioventricular (AV) Node also has a longer than usual refractory period and acts as a gatekeeper between the atria and ventricles. With a very rapid atrial rhythm, the ventricles may only respond to an impulse being transmitted from the atria every few beats. e.g. atrial tachycardia with 2:1 or 3:1 block or atrial fibrillation.

FIGURE 4: ACTION POTENTIAL FOR CARDIAC MUSCLE

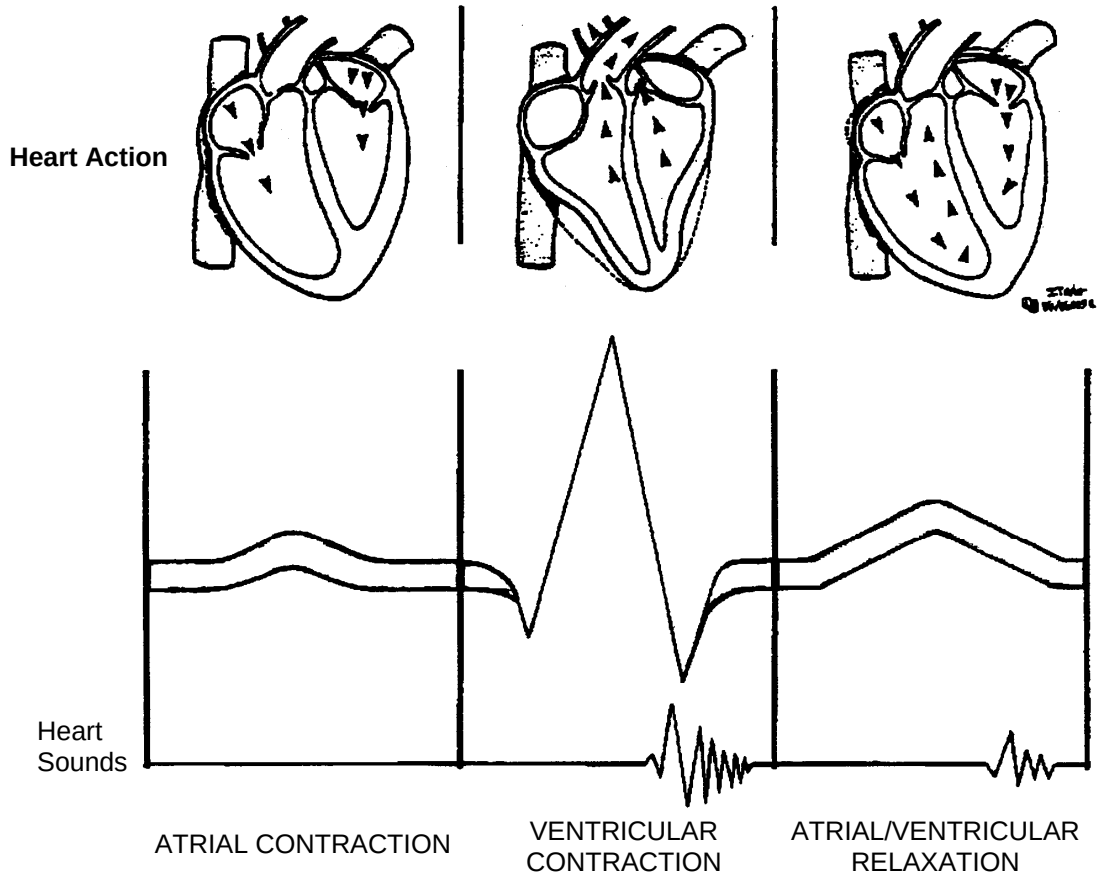


Refractory periods can be functional (absolute) meaning that the muscle cannot respond to any stimulus, or relative, meaning that the muscle may respond to a strong impulse (e.g. premature ventricular complex).

The period of time from one heart contraction to the next (or one P wave to the next) is call the cardiac cycle. The contraction phase is called systole, and the relaxation phase is called

diastole (Figure 5). Systole is actually comprised of atrial and ventricular contraction. Since ventricular contraction is responsible for the pulse, systole usually refers to the ventricular contraction.

FIGURE 5: CARDIAC CYCLE



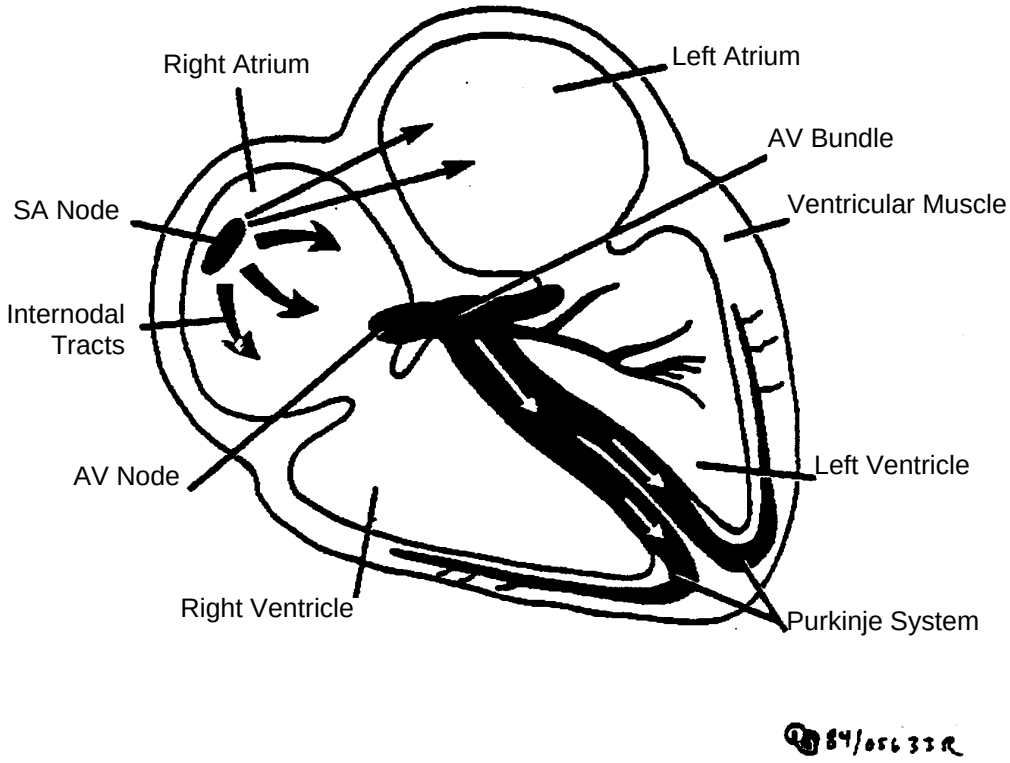
The cardiac impulse originates in the sino-atrial node (SA node), the first in the network of specialized conduction tissue, which is located in the posterior portion of the right atrium just beneath, and medial to, the entrance of the superior vena cava. Most cardiac fibers are capable of automaticity, an ability to generate action potentials spontaneously, but the SA node has a higher intrinsic rate than the other tissue and, for this reason, functions as a normal pacemaker of the heart.

The SA node fibers are continuous with the atrial fibers, so that any action potential generated in the SA node spreads to the remainder of atrial tissue. In addition to this generalized spread of the action potential, there are also internodal pathways between the SA node and the atrio-ventricular node (AV node) with faster conduct ion than normal atrial tissue.

The impulse is delayed at the AV node, resulting in slower conduction through these fibers. This delay in conduction between the atria and the ventricles allows the atria to contract before the ventricles.

From the AV node, the impulse travels down the Bundle of HIS which bifurcates into the right and left bundle branches. The impulse then reaches the into the Purkinje network in the apex of the heart. The Purkinje fibers form a network of small fibers which spread through the ventricular chambers (Figure 6).

FIGURE 6: CONDUCTION SYSTEM



Clinical vignette

The Electrocardiogram (ECG) is a graphic representation of the heart's electrical activity. It records the electrical voltage generated by the heart and transcribes it. The P wave represents depolarization of the atria, the QRS represents depolarization of the ventricles and the T wave represents repolarization of the ventricles.

Although it is expected that if the atria and ventricles depolarize (as seen on the ECG) that they will contract, one cannot assume that contraction has taken place unless a corresponding pulse is palpated.

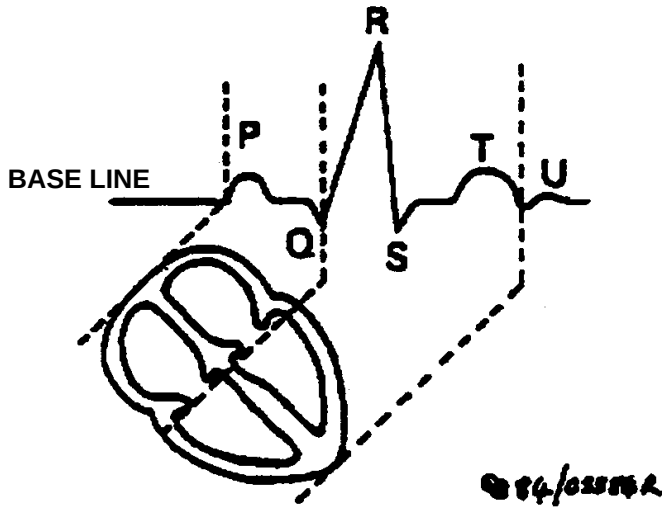
The physiological delay at the AV node is represented by the PR interval. The QRS complex of the ECG is the depolarization of the ventricles, and occurs immediately before ventricular contraction. The T wave is the pattern of repolarization of the ventricles, and occurs just prior to the end of contraction.

The cardiac cycles is thus represented on the ECG as the period between two P waves.

CONTROL OF CARDIAC FUNCTION

The cardiac function is controlled by both intrinsic and extrinsic factors.

FIGURE 7: ELECTRICAL/ANATOMICAL RELATIONSHIP



The major intrinsic factor is venous return. In cardiac muscle, the greater the stretch, the greater the force of contractility. What this means is that within a physiological range, the heart will pump whatever blood it receives (the venous return). This is the Frank Starling Law of the Heart, which can also be stated as “the greater the diastolic filling (the filling of the ventricles during diastole), the greater the cardiac output”.

The extrinsic factors governing cardiac function relate primarily to the autonomic nervous system, which controls not only the rate, but also the degree of contractility of the heart.

The atria are extensively innervated by both sympathetic and parasympathetic (vagal) fibers. The ventricles contain predominately sympathetic fibers, with few parasympathetic fibers.

In general:

- **Sympathetic** stimulation **increases** both the rate and the strength of contraction.
- **Parasympathetic** stimulation **decreases** both the rate and the strength of contraction.

The autonomic nervous system involvement in cardiac control originates in the vasomotor centre of the brain. The sympathetic and parasympathetic nervous systems work in a check-and-balance manner to increase or decrease heart rate and force of contractility based on the body's metabolic demands at the moment. The autonomic nervous system has been compared to the two footed driver – i.e.

- you can go faster by applying more pressure to the gas pedal (sympathetic), or
- by simply taking you foot off the brake pedal (parasympathetic inhibition)
- you can go slower by taking your foot off the gas pedal (sympathetic inhibition), or
- by applying more pressure to the brake pedal.

Other external factors which govern cardiac function include but are not exclusive to electrolytes and temperature. Electrolytes in the circulation affect heart rate and cardiac contractility because of the role they play in the generation of action potential and the contractions of muscle. High levels of potassium lead to a dilated, flaccid heart with a slower heart rate and delayed transmission through the AV node. High levels of calcium lead to spastic contraction of the heart. Elevated sodium depresses cardiac function. Temperature increase leads to an increase in heart rate and contractility.

TABLE 1
FACTORS AFFECTING CARDIAC FUNCTION

FACTOR	EFFECT
INTRINSIC: Venous Return	Greater the venous return Greater the cardiac output
EXTRINSIC: Sympathetic Stimulation	Increases heart rate Increases contractility Increases conduction velocity
Parasympathetic	Decreases heart rate Decreases contractility Decreases conduction
High Potassium	Decreases heart rate Decreases contractility
High Calcium	Increases contractility
High Sodium	Decreases contractility
High Temperature	Increases contractility Increases heart rate

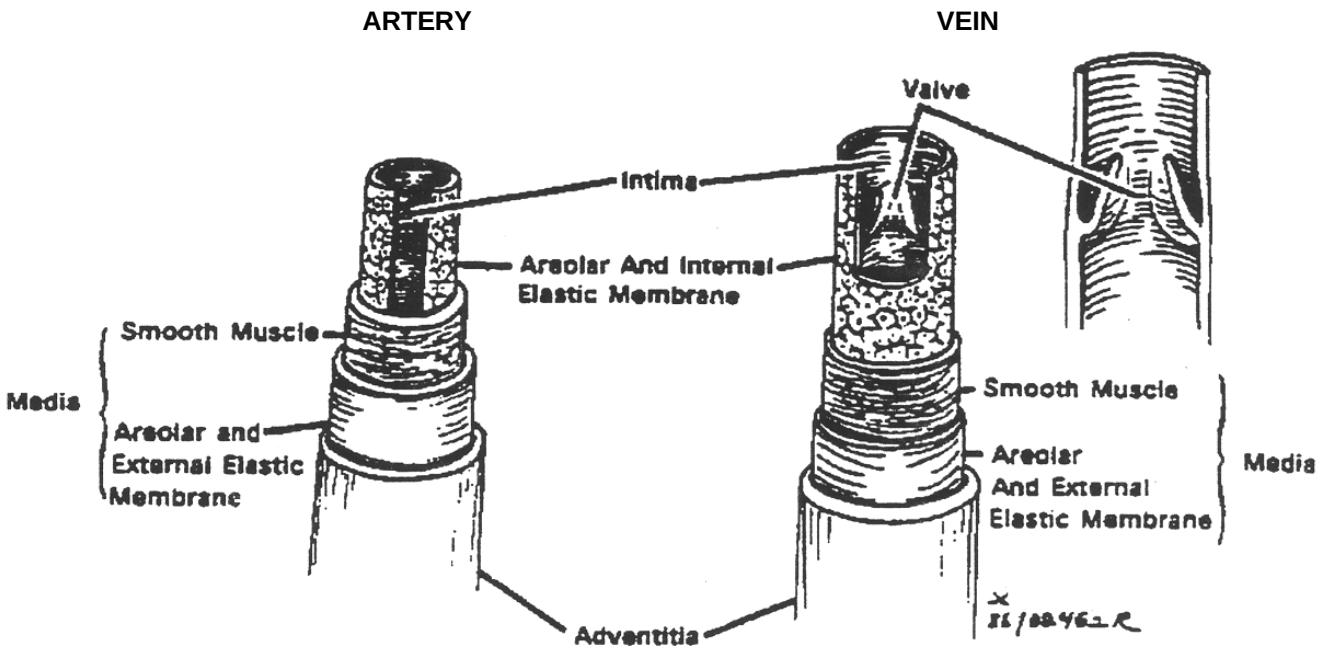
ARTERIES AND VEINS

The arteries function primarily as a distribution system for the supply of nutrients to tissues. With repeated branching, arteries become progressively smaller, from the aorta (with a diameter of 2.5 cm) to the arterioles (with a diameter of 0.1 mm). In the majority of organs the branching occurs in a fashion to provide anastomotic channels which may function as alternate routes of supply in the event of obstruction of one of the vessels. End arteries are those where a single vessel provides the flow to a structure with few or no alternate routes.

Clinical vignette

An example of an "End Artery", and its significance, is the central retinal artery of the eye. Obstruction of the central retinal artery by either embolus or thrombus, deprives the retina of nutrients and leads to visual loss in the eye involved.

FIGURE 8: ARTERIES AND VEINS



Arteries are elastic muscular tubules whose walls consist of three layers:

- Intima (the smooth inner lining of endothelial cells)
- Media (the thick middle layer of muscle and elastic fibers)
- Adventilita (the outer layer of fibrous, collagenous tissue).

The arterioles are the last branches of the arterial system, and act as control valves for releasing blood to the capillaries.

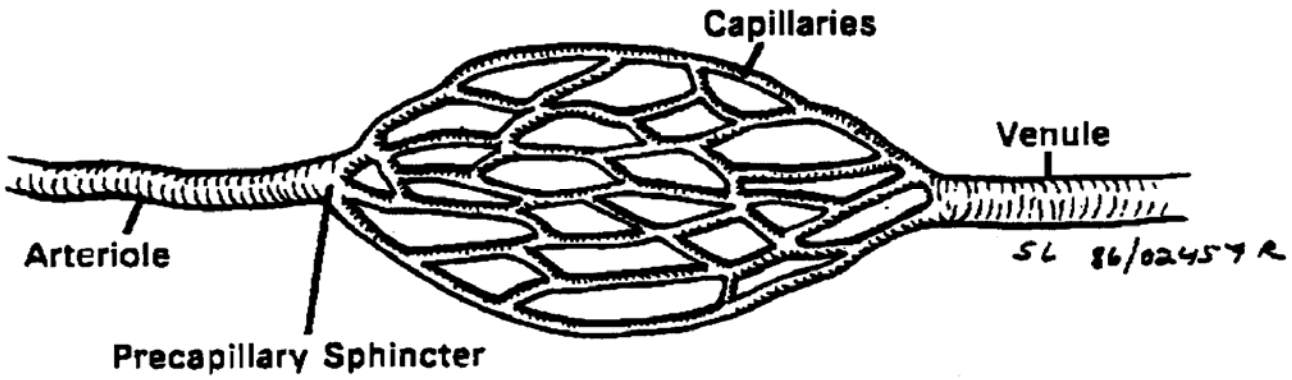
The **veins** have a major role as a storage system **(capacitance system)**, as well as serving as a collecting system to return blood to the heart. Like arteries, their walls are three layers thick. However, being a lower pressure system there is far less muscular and elastic tissue within their walls, and the entire medial layer is far less pronounced.

In contrast to arteries, veins have valves which consist of cup-shaped projections of epithelial tissue, resembling the semi-lunar valves of the heart. These are especially prominent in the long extremity veins. The valves serve to promote unidirectional blood flow. When the muscles contract, the veins are compressed and the blood is pushed in a forward direction.

The smooth muscle in both arteries and veins is under the control of the autonomic nervous system.

The exchange of nutrients takes place at a capillary level. Capillaries are supplied by

FIGURE 9: CAPILLARIES



arterioles and drained by venules. They are small vessels with walls consisting of only endothelial cells on a basement membrane. The diameter of capillaries is measured in micrometres (a micrometer is 1/1,000 of a millimeter). Their structure allows the ready exchange of nutrients and waste materials through the vessel walls. There are no smooth muscle cells in the capillaries, and they are not under the control of the autonomic nervous system.

PRACTICAL ANATOMY

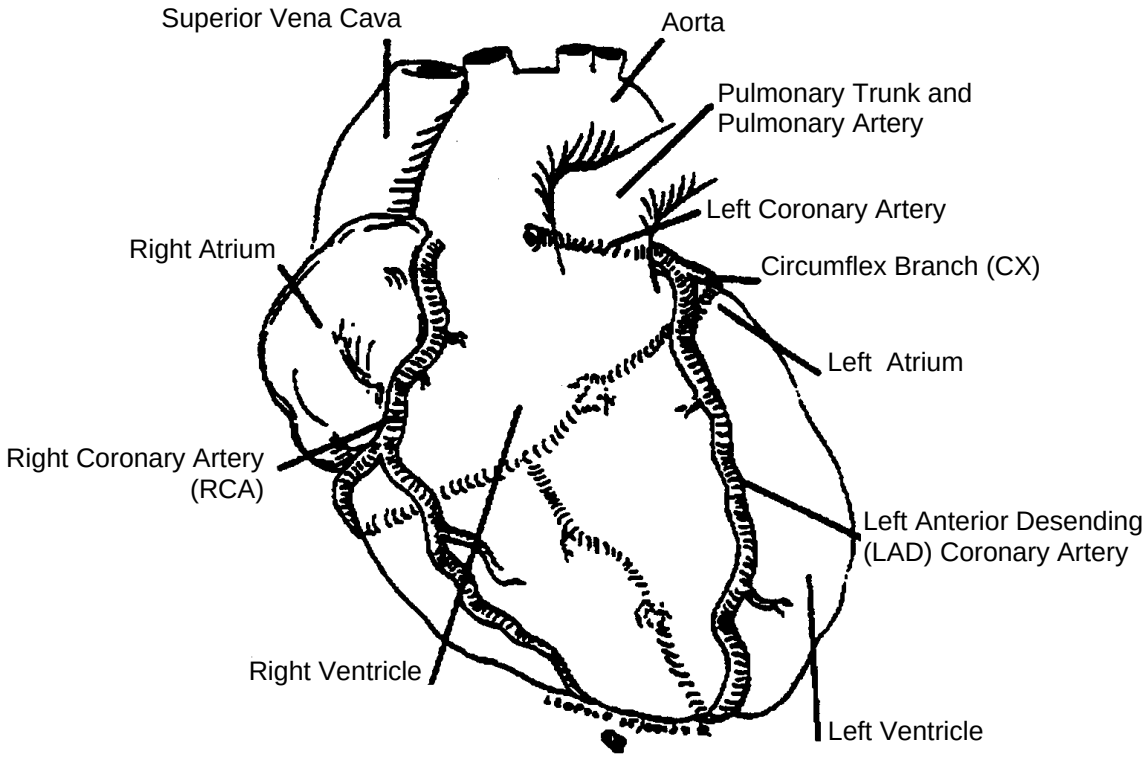
No attempt will be made to review the entire anatomy of the cardiovascular system. Rather, certain practical points will be mentioned.

In the course of practising Paramedicine, it is worthwhile to have access to an anatomy and physiology book, as it is surprising how often something will arise which can be explained by an understanding of the anatomy and physiology. It is far easier to remember anatomy when it has acquired relevance through a case, and it is worthwhile taking advantage of this sort of opportunity.

CORONARY ARTERIES

It is of value to know the circulation of the coronary arteries in order to understand some of the problems that can arise with occlusion of one of the arteries, e.g. myocardial infarction. The two main arteries are the right coronary artery (RCA) and the left coronary artery. The left divides, near its origin, into the left anterior descending (LAD) and the left circumflex (CX).

FIGURE 10: CORONARY ARTERIES



As mentioned previously, these arise from the sinuses just distal to the aortic valve and travel along the surface of the heart. Cardiac muscle is supplied entirely by coronary vessels. There is no nutrient or gas exchange across the endocardial surface within the heart's chambers. There are normally few or no anastomoses between the coronary vessels, making many of them End Arteries in the terms of occlusion (Figure 10).

The coronary arteries also supply the SA and AV nodes and the conduction system of the heart, so occlusion of these vessels can lead to ischemia of the nodes or conduction system, and subsequent arrhythmias.

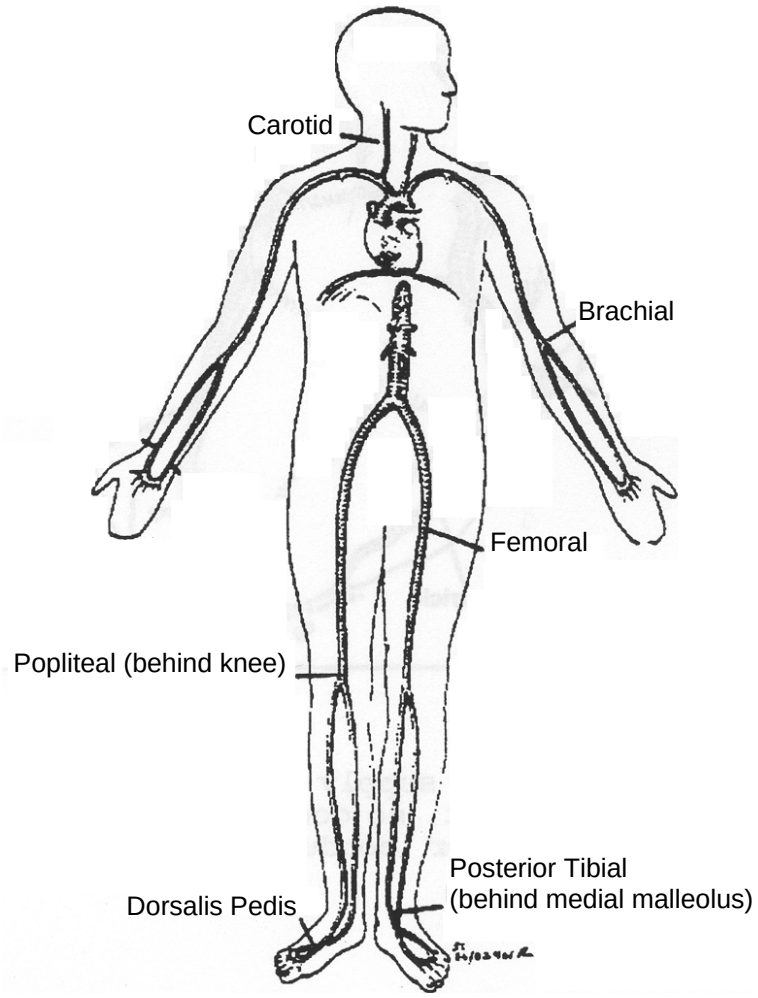
PUSLE POINTS

Points where arteries are readily palpable are important in detecting a pulse in a situation where the blood pressure is low enough to prevent effective use of a sphygometer and where a fracture, crush injury or embolus may have obstructed flow to a vessel or make use of a cuff impossible. Common pulse points (Figure 11) are:

- Carotid
- Brachial
- Radial and ulnar
- Posterior tibial
- Dorsalis pedis
- Femoral.

The popliteal pulse (posterior leg behind the knee) is also palpable, but can be difficult to feel owing to the anatomy of the area.

FIGURE 11: PULSE POINTS



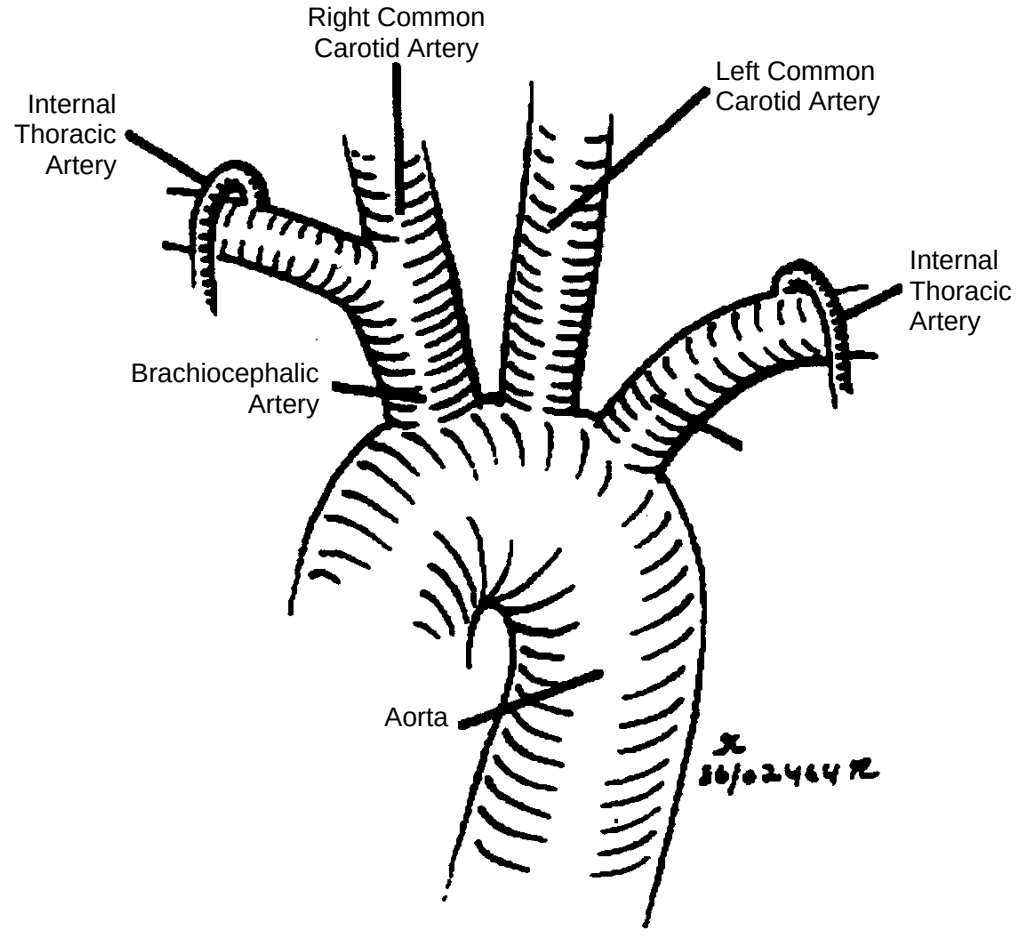
INTERCOSTAL VESSELS

Should it be necessary to insert a needle into the chest of a patient with a tension pneumothorax, then it is useful to know that the nerve, artery and vein of the intercostal space travel on the under surface of the ribs. For this reason, A needle (or a chest tube in the hospital setting) should be inserted at the top s of the rib, to avoid damaging the artery and causing intrathoracic bleeding. A needle thoracostomy is performed in the 2nd intercostal space at the mid-clavicular line.

AORTIC BRANCHES

The major branches of the aorta are useful to know to assist in the detection of a thoracic aortic dissection in the upper portion of the aorta. In this condition, the blood pressure in one arm may be lower compared to the other as blood may dissect between the intimal and medial layers along the aorta and into one of the subclavian arteries– e.g. It will be lower on the left side if blood dissects into the left subclavian artery. A blood pressure difference of $\geq$ 15 mmHg systolic in one arm compared to the other is considered sygnificant. This is commonly a major clue to the diagnosis of thoracic aortic dissection and is one reason why understanding of the anatomy is important.

FIGURE 12: MAJOR BRANCHES ASCENDING AORTA



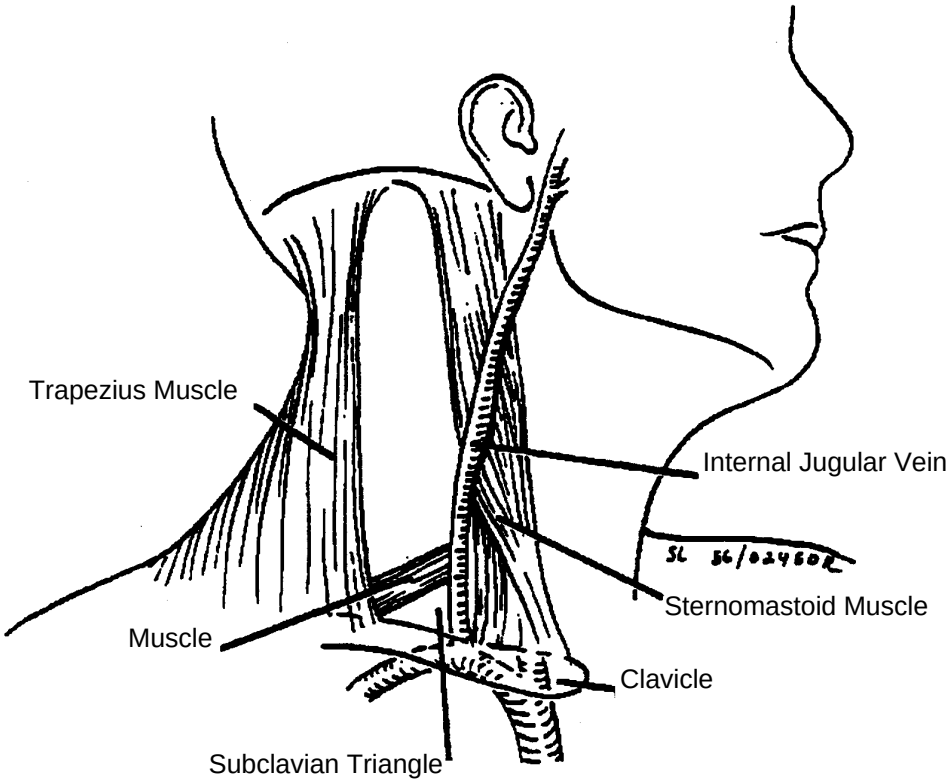
JUGULAR VEIN

Recognition of jugular venous distention is important in a number of clinical situations, such as heart failure and pericardial tamponade. Therefore, it is important to know the location of the jugular vein in the neck (Figure 13).

Clinical caveat

In 3rd degree AV block and some instances of ventricular tachycardia, you may see irregular jugular venous distension (Cannon A waves) as the atria and ventricles contract at different rates (asynchronously). This means that at times the atria will contract against closed AV valves and the blood will back up into the jugular veins. Look at the neck veins in these dysrhythmias.

FIGURE 13: EXTERNAL JUGULAR VEIN



It can be worthwhile to review the relationship of some of the major arteries to bony structures to understand how a person can fracture a clavicle and lose a radial pulse, or injure the popliteal artery with a dislocation of the knee.

By relating the anatomy to practical situations, it is not only easier to recall information, but also allows you to anticipate what structures might have been injured in a particular situation.

PHYSIOLOGY OF THE CIRCULATION

REGULATION OF BLOOD FLOW

Blood flow is regulated by three sets of factors:

- Local
- Nervous
- Humoral.

LOCAL CONTROL

The prime local determinant of blood flow in most tissues is the partial pressure of oxygen. When the oxygen concentration is low, due to increased metabolism and increased oxygen consumption, the pre-capillary sphincters open, leading to increased flow in the tissues. The opposite occurs in the pulmonary circulation where if a group of alveoli are obstructed, the surrounding vessels will constrict and blood will be shunted to other areas of the lungs for gas exchange.

In a situation of high local concentration of oxygen, the pre-capillary sphincters close. This may relate to the fact that the smooth muscle contraction is an energy requiring process. Thus, when the oxygen is low, the necessary energy to cause contraction and closure of the sphincter is lacking, and the muscle relaxes.

Two special situations exist where other factors are prime determinants of sphincter smooth muscle activity:

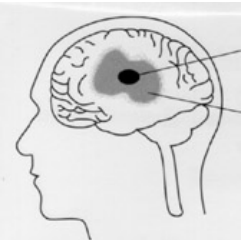
1. In the kidney, the flow increases in response to an elevated level of sodium in the serum, or to an elevated level of end products of protein metabolism.

2. In the brain, flow increases in the presence of an elevated level of carbon dioxide ($PaCO_2$) or hydrogen ions.

Clinical vignette

Although $PaCO_2$ is a potent cerebral vasodilator and may contribute to the increase in intracranial pressure (ICP) in the setting of a head injury, hyperventilation is not indicated in the prehospital setting unless hypercarbia is suspected, and even then, it should be done briefly. Ideally, $ETCO_2$ monitoring should be available to ensure CO_2 is kept to the low end of normal – i.e. approximately 33 mmHg.

Hyperventilation of the head injured patient has gone out of vogue in the prehospital setting because of the risk of the “watershed effect”



the vessels within the injured area are damaged

constricting the vessels surrounding the damaged area from hyperventilation results in blood flow into the damaged area resulting in worsened edema and further secondary brain damage

NERVOUS FACTORS

Two major features dominate the nervous system control of blood flow in tissue. First, the control has a rapid onset (within 1 second), and second, nervous system control overrides local factors, affecting larger areas of the body.

The sympathetic nervous system exerts the most important influence over the peripheral vascular system. Vasomotor fibers of the sympathetic system leave the spinal cord through all thoracic as well as the upper two lumbar vertebrae. These fibers pass into the sympathetic chain, and are then distributed to the vessels through the peripheral sympathetic nerve fibers and the spinal nerves. Sympathetic stimulation alters peripheral vascular resistance and therefore alters flow. As the system also controls the venous system, it is capable of changing the volume in the capacitance system.

The vasomotor centre of the brain acts as the control centre for this system. The upper and lateral portions of the centre, which lie in the reticular substance of the lower brainstem, are tonically active, constantly sending nerve impulses at the basal rate through the vasoconstrictor fibers. This is called sympathetic vasoconstrictor tone and maintains all vessels in a constant state of partial contraction called vasomotor tone. Loss of this tone, such as would occur with a transection of the spinal cord, leads to a significant fall in blood pressure.

Clinical vignette

Patients with a high thoracic or cervical cord injury may be hypotensive, but will not exhibit the typical reflex tachycardia seen in shock. The patient will have a normal pulse and be warm to touch. The normal pulse and warm skin results from the disruption of the sympathetic outflow from the spinal cord and loss of vasomotor tone. Patients are warm and have a low blood pressure because the vessels are dilated. Hemorrhagic shock can be present in these patients as well, however the usual signs of shock will be absent due to the loss of sympathetic tone from the cord injury.

The vasomotor centre controls vasoconstriction and cardiac activity.

Many areas of the brain influence the vasomotor centre, including the cortex of the brain. Feat is an example of the cortex stimulating the vasomotor centre to increase sympathetic outflow. With fear, our heat rate increases, our peripheral vessels constrict and we sweat – all triggered by the sympathetic nervous system. In the same way when we think of exercising, the vasomotor centre receives this message, and our heart and vessels are preparing for this before we actually start the exercise.

HUMORAL FACTORS

Norepinephrine is the substance secreted at the endings of the vasoconstrictor fibers facilitating smooth muscle contraction in vessels. If vasoconstrictor impulses are sent to the adrenal medulla, this leads to the release of **norepinephrine** and **epinephrine** into the blood stream from the medulla, as **circulating catecholamines**.

The sympathetics to skeletal muscle carry dilator as well as constrictor fibers. Stimulation of the motor cortex leads to the release of these vasodilators through the hypothalamus.

Norepinephrine and epinephrine from the adrenal medulla are the major humoral factors involved in flow control. Norepinephrine is a vasoconstrictor and epinephrine dilates vessels to skeletal muscle and cardiac muscle, while causing vasoconstriction in other areas.

The factors controlling blood flow are summarized in Table 2.

TABLE 2
FACTORS AFFECTING BLOOD FLOW

FACTOR	EFFECT
Local:	
Increased oxygen concentration	Vasoconstriction
Decreased oxygen concentration	Vasodilation
Nerves:	
Sympathetic system	Vasoconstriction Vasodilation (skeletal muscle)
Humoral – Adrenal Medulla:	
Norepienphrine	Vasoconstriction
Epinephrine	Vasodilation (except skeletal and cardiac muscle where vasodilates)
Angiotensin	Vasocontriction

BLOOD PRESSURE AND ITS CONTROL

Normally our bodies control our mean arterial blood pressure within the range of 80-120 mm mercury. With age, the mean arterial pressure increases – the systolic component increasing more than the diastolic component. This increase is attributed to atherosclerosis, with loss of the normal elasticity of the vessels.

The control of blood pressure operates on a feedback system, similar to the thermostat in your home which provides a feedback system to the furnace to regulate the heat.

The arterial pressure depends on the cardiac output and the mean arterial resistance, which, from the definitions given in the glossary, is highly dependent on the radius of the vessel – the smaller radius, the higher the resistance. Most of the resistance in the cardiovascular system is at the level of the arterioles.

The means of controlling blood pressure are either:

- Rapid – autonomic nervous system circulating catecholamines
- Intermediate
- Slow – the renal system

There is normally a degree of sympathetic stimulation with vasoconstriction in the peripherial circulation keeping the tone of the vessels above baseline in the same fashion that there is a degree of sympathetic stimulation of the heart.

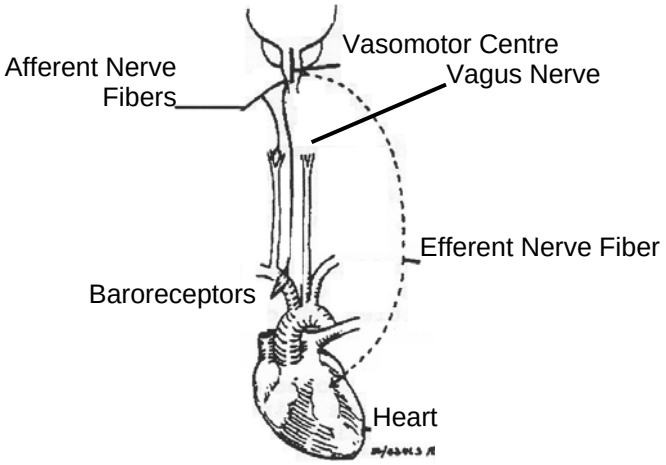
RAPID SYSTEMS

Barorecptors, or pressure receptors, are found in most of the major arteries of the thorax. They are most abundant in the walls of the internal carotids just above the bifurcation of the common carotids and the walls of the aorta.

Stimulation of these receptors by stretch secondary to higher blood pressure sends a message to the vasomotor centre of the medulla (carotid baroreceptors) or up the vagus nerve to the same centre (aortic baroreceptor).

This stimulation leads to inhibition of the vasoconstrictor centre and excitation of the vagal nerve. The end result is a fall in blood pressure due to decrease in heart rate, decrease in contractility and dilation of the peripheral vessels. With a persistent elevation of blood pressure, this system adapts to accept the new level as normal.

FIGURE 14: BARORECEPTORS



Pressure ↑ sensed in AORTIC and CAROTID BARORECEPTORS

↓

Via Afferent Nerves

↓

Medulla

↓

Inhibit Vasoconstrictor Centre
Excite Vagal Centre

↓

Vasodilation
↓ Heart Rate → ↓ Pressure
↓ Contractility

Chemoreceptors (located in the carotid and aortic bodies) are sensitive to a fall in oxygen concentration, a rise in hydrogen ion concentration, or a rise in carbon dioxide. These respond best when blood pressure is low. When stimulated, messages regarding the situation are taken to the vasomotor centre and lead to excitation of the vasomotor centre with the corresponding rise in blood pressure (vasoconstriction, increased heart rate, increased contractility).

The **venous system** is also stimulated in all of these situations. The response of constriction in this system is to decrease the capacitance of the veins. From Starling's Law of the Heart, it is known that the heart will pump (within limits) what it receives, so it follows that increasing the venous return increases the cardiac output, and in turn, the blood pressure. With lack of stimulation, more blood is stored in the venous system, and venous return to the heart is less.

The arterial resistance is called the afterload of the heart, and the venous return is called the preload. These terms can be very useful in understanding abnormalities which occur in the cardiovascular system.

Sympathetic stimulation, as mentioned above, also leads the stimulation of the adrenal medulla to release epinephrine and norepinephrine into the circulation. These circulating cahecholamines excite the heart, constrict most arterioles, and constrict veins.

INTERMEDIATE SYSTEMS

The intermediate systems are those of renin-angiotensin, and vasopressin.

SLOW SYSTEMS

The primary long term system for the control of the blood pressure is the renal system which controls the circulating volume. In general, high blood pressure leads to increased output of water and electrolytes by the kidneys which depletes the extracellular fluid volume, decreases venous return (preload) and cardiac output, and thus lowers the blood pressure. The reverse is true of low blood pressure.

Unlike the other systems, which can adapt to a new level of blood pressure over time, this system remains active.

TABLE 3 FACTORS CONTROLLING BLOOD PRESSURE
FAST ACTING SYSTEMS Barorecptors Chemoreceptors Circulating Catecholamines INTERMEDIATE Renin – Angiotensin Vasopressin SLOW ACTING Renal – body fluid system

CARDIAC OUTPUT

The cardiac output is the quantity of blood pumped from the left ventricle into the aorta per minute. In normal resting conditions in an average adult, cardiac output averages 5 litres/minute (70 mL/kg). In the athlete during exercise, it can increase to over 20 litres/minutes.

Since this quantity of blood depends on the quantity pumped with each beat of the ventricle (stroke volume), and the number of beats per minute (heart rate).

CARDIAC OUTPUT = STROKE VOLUME x HEART RATE

From this it can be seen that the factors controlling the cardiac output will be those controlling the stroke volume and the heart rate. It can also be seen that over a period of time,

CARDIAC OUTPUT = VENOUS RETURN

The heart will normally pump the amount of blood entering the right atrium (within physiological limits). Under normal conditions the venous return (preload) is a prime determinant of cardiac output. When the heart becomes that limiting factor (it can no longer pump the amount of blood it receives), the heart is said to be failing.

Factors that increase the permissive level of the heart to pump what it recieves are increased heart rate and increase contractility. As these occur with sympathetic will increase the cardiac output.

Factor affecting venous return (preload) are:

- Increased preload with:
  - Increased blood volume
  - Increased sympathetic stimulation
  - Contraction of skeletal muscle

- Decreased preload with:
  - Loss of volume
  - Increase pressure in the thoracic cage (normal intrapleural pressure is –4).

Clinical vignette

Every time we take a breath of intrapleural pressure changes. With inspiration, our intrapleural pressure is more negative; with expiration, more positive. In situations, such as asthma, where expiration is compromised by marked narrowing of the airways with expiration, the pressure in the intrapleural space can become considerably more positive and actually impair venous return.

The veins are a low pressure system. Positive pressure can easily collagpse them. In the asthmatic with a high intrapleural pressure, the veins can collapse, so that venous return falls much more than the norm with expiration. This can be detected in a fall in the patient's blood pressure with expiration, a situation called pulsus paradoxus. Why it has been given this name is confusing, as it is not different from normal, but rather and exaggeration of a normal response which all of us have every time we breathe.

With a normal hearth the heart can pump what blood it receives. If some factor lowers the heart's ability to pump, then there will be a backup in the venous system, (heart failure). This leads to elevation of the jugular veins in the neck and can lead to engorgement of the liver, and leakage of fluid into the pulmonary circulation, leading to pulmonary edema.

Factors that affect the heart's ability to pump are:

- Increased with:
  - Sympathetic stimulation
  - Hypertrophy (more muscle, as in an athlete)
  - Low systemic arterial pressure
  - Inhibition of the parasympathetics.

- Decreased with:
  - MI
  - Parasympathetic stimulation
  - Inhibition of sympathetic stimulation
  - Myocarditis
  - Cardiac hypoxia
  - Congenital heart disease.

Clinical vignette

In a patient with narrowing of the arteries of the heart from atherosclerosis, there are definite limits on how much oxygen and other nutrients can reach the heart muscle.

While at rest, the amount of oxygen might well be very adequate - but what happens if this patient exercises?

Exercise will require increased cardiac output to supply the needs of the skeletal muscles of the body. To prepare for this, the body stimulates the vasomotor centre and the sympathetic nervous system constricts the peripheral blood vessels, increases the heart rate and increases the contractility of the heart.

The afterload against which the heart must pump is increased, and the preload is increased. The heart must pump more blood against greater resistance to deal with the situation. This increases the work that the heart must do increasing its need for oxygen.

As the heart's arteries are incapable of meeting these needs, the muscle of the heart become ischemic and the patient develops angina – chest pain. If the ability of the heart is greatly impaired, the patient may actually go into pulmonary edema.

Now the patient takes a nitroglycerine tablet before the exercise. The nitroglycerine tablet has the effect of increasing the venous capacitance, lessening the preload of the heart, and reducing the systemic vascular resistance lessening the afterload on the heart. Since the heart has less work to do, with the decrease in preload and afterload, the patient can exercise without experiencing chest pain. Naturally we are not talking about our cardiac patient running a marathon with nitroglycerine, but he is able to do more than he would be able to do without it.

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SELF-ASSESSMENT

Marks

[10] 1. What should you examine in a patient who has fallen on his shoulder and has swelling and tenderness over the clavicle? Give reasons for your examination.

[7] 2. How are arteries and veins different in structure and function?

3. An individual has sustained a major GI bleed, losing a significant amount of his blood volume. Identify the mechanism that will come into play in an attempt to maintain his blood pressure. Describe the physical findings of the examination of the patient that reveal that these are in force.

Complete these tasks using the outline below, filling in the blanks.

[4] a) The body attempts to maintain tissue flow at an acceptable level to deal with the needs. If the patient bled, his _____ would fall, and therefore his _____ would fall. This would lead to a fall in _____ and ultimately a fall in _____.

[7] b) The _____ would detect this fall leading to _____ stimulation. This stimulation leads to an increase in _____ and _____ of the heart. The peripheral cardiovascular system compensates with increased _____ and _____ tone.

[4] c) The vital signs would include _____and the patient would have _____ skin because of _____. The _____ might still be normal if these methods were effective.

d) Over time, the _____would attempt to correct the situation by retaining fluid to replace the lost volume.

[5] e) Since the cardiac output = _____ x _____ the heart rate would increase initially; then a point would be reached where _____ filling was not complete because of the rapid rate. This would lead to a fall in the stroke volume, and eventually a fall in the _____ (because of the rapid heart rate). At this stage, if volume continues to be lost, the _____ will fall in spite of the increase in heart rate.

[2] f) As blood provides oxygen carrying capacity, the fall in blood volume will lead to less oxygen being carried to the tissues, and . metabolism in some tissues where vasoconstriction is intense. The respiratory rate will _____ to keep the acid base balance normal.

[2] g) The patient will have these findings as a result of the compensation mechanisms. He may become confused secondary to the lack of _____ and will demonstrate a _____ in pressure when sat up (as the normal compensation mechanisms will already be in force with the patient supine).

[5] 4. Why could a patient who has sustained a heart attack have a fall in blood pressure during transport? (In a heart attack, a branch of one of the coronary arteries is blocked.)

[8] 5. What are the factors and their effects involved in increasing cardiac output to deal with exercise?

[2] 6. The autonomic nervous system involves which aspects of the cardiovascular control?

[4] 7. Outline the ANS affects (sympathetic and parasympathetic) on each of the above aspects.

[2] 8. Explain the two special features which differentiates cardiac muscle from other muscle.

9. Why is the SA nodes the “pacemaker” of the heart?

10. a) Sympathetic outflow for peripheral vascular control is at the spinal cord level of _____ to _____.

b) Sympathetic control is exerted over which of the following: (more than one answer may be correct)

- Arteries
- Veins
- Capillaries.

___
66 TOTAL

ADVANCED LIFE SUPPORT
PRECOURSE
CARDIOVASCULAR SYSTEM

SELF-ASSESSMENT ANSWERS

1. Key concepts are in bold.

   Since subclavian artery runs underneath the clavicle, if the patient has fractured his clavicle there could be damage to the subclavicle artery. This artery supplies the arm, so it would be important to check the pulses distal to the fracture, namely the branchial pulse on the medial molar side of the elbow, and the radial and ulnar at the wrist.

   One might also check:
   - The blood pressure in both arms to see if they were the same
   - The temperature of the injured arm compared to the other (if the blood supply was less it would be cooler)
   - Function of the muscles in the distal arm
   - Sensation in that area.

2. Key concepts are in bold.

   - Arteries carry blood under more pressure than veins
   - Arterial walls have thicker muscular coats and more elastic tissue than veins
   - Veins have valves to assist in the one way flow, arteries do not
   - Veins have a storage function for blood (one of their major roles), which is different from arteries
   - Arteries have a major role in providing resistance to flow by dilation and constriction. The resistance in veins is much less
   - Arteries carry blood away from the heart, veins carry blood to the heart.

3. a) The body attempts to maintain tissue flow at an acceptable level to deal with the needs. If the patient bled, his blood volume would fall; and therefore his venous return would fall. This would lead to a fall in would fall. This would lead to a fall in cardiac output, and ultimately a fall in blood pressure.

   b) The baroreceptors would detect this fall leading to sympathetic stimulation. This stimulation leads to an increase in rate and contractility of the heart. The peripheral cardiovascular system compensates with increased arterial and venous tone.

   The substance with mediate and regulate blood flow control at this time are collectively called catecholamines.

c) The vital signs would include increased heart rate, and the patient would have cool skin fuse of peripheral vasoconstriction. The blood pressure might still be normal if these methods were effective.

d) Over time, the kidneys would attempt to correct the situation by retaining fluid to replace the lost volume.

e) Since the cardiac output = stroke volume x heart rate the heart rate would increase initially; then a point would be reached where ventricular filling was not complete because of the rapid rate. This would lead to a fall in the stroke volume, and eventually a fall in the cardiac output (because of the rapid heart rate). At this stage, if volume continues to be lost, the blood pressure will fall in spite of the increase in heart rate.

f) As blood provides oxygen carrying capacity, the fall in blood volume will lead to less oxygen being carried to the tissues, and anaerobic metabolism in some tissues where vasoconstriction is intense. The respiratory rate will increase to keep the acid base balance normal.

g) The patient will have these findings as a result of the compensation mechanisms. He may become confused secondary to the lack of adequate circulation to the brain, and will demonstrate a – postural drop in pressure when sat up (as the normal compensation mechanisms will already be in force with the patient supine).

4. The coronary arteries supply the muscle of the heart, and they lack anastomotic channels. For this reason if one of the coronary arteries is blocked, the area supplied by that artery is without oxygen and becomes non-functional.

   Depending on the vessels, this could lead to:

   - Damage to muscle, therefore less effective pumping of the heart, resulting in less stroke volume and a fall in blood pressure.

   - Damage to the conducting system of the heart, therefore development of an arrhythmia with a fall in the heart rate, and/or less co-ordinated pumping of the heart, leading to a fall in blood pressure.

   - Rupture of the heart through the dead muscle, leading to fluid collecting in the pericardium, less effective pumping from the increased pressure on the muscle, and less effective filling due to pressure on the atria, with the end result being a fall in blood pressure.

5. With exercise the cardiac output increases to cope with the increased demands for oxygen and other substrates, and the increased need to remove waste materials.

The thought of exercise leads to an increase in cardiac output. The autonomic nervous system causes increased heart rate and contractility, and increased venous tone. These lead to an increase in cardiac output.

At the onset of exercise, the motor cortex is stimulated leading to sympathetic vasodilation to muscles, increased muscle blood flow, and increased cardiac output (less resistance to flow to muscles). This stimulation also leads to increased heart activity and blood pressure.

The use of the skeletal muscles leads to increased venous return.

The increased muscle metabolism, leads to a fall in the oxygen concentration at the capillary level of the muscles, with this local factor leading to vasodilation there.

6. The autonomic nervous system is involved in the control of both cardiac and vascular function.

7. (½ mark for each effect – given in bold)

Cardiac
- Sympathetic stimulation leads to
  - Increased heart rate
  - Increased contractility
  - Increased conduction speed.

- Parasympathetic stimulation leads to
  - decreased heart rate
  - decreased contractility
  - decreased conduction speed.

Vascular
- Sympathetic stimulation leads to
  - vasoconstriction of arteries
  - vasoconstriction of veins

8. - Plateau on the action potential which causes contraction for the duration of the plateau.

- Repetitive discharges, which lead to a tendency for rhythmic contraction.

9. The SA node has a higher intrinsic rate of discharge than other sites.

10. a) T1 to L2
    b) Arteries and veins

ADVANCED LIFE SUPPORT
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EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading _____ hours
   Self assessment _____ hours
   Total time _____ hours

2. Were the objectives of the module clearly stated?

   [ ] yes [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes [ ] no
   If yes, which items _____
   _____
   _____
   Were they helpful? _____

4. Were the reference notes adequate?

   [ ] yes [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?

   [ ] yes [ ] no
   If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital _____

9. General comments or suggested improvements.