BLOOD PRESSURE & THE CARDIAC CYCLE

Aim

Explain blood pressure and the cardiac cycle and their relationship to health and fitness.

BLOOD PRESSURE

Blood pressure describes the pressure exerted on the walls of blood vessels by the blood within. It is measured in millimetres of mercury (mmHg). For example, a blood pressure measurement of 100mmHg means blood vessel pressure equals the pressure created by a column of mercury 100mm high.

Factors Affecting Blood Pressure

There are three main factors which affect blood pressure:

1. Cardiac output

Cardiac output refers to the amount of blood in litres pumped out of the heart per minute. It is calculated by taking the stroke volume (the amount of blood in millilitres pumped out into the circulation by the left ventricle each time it contracts) and multiplying it by the number of times the heart beats each minute. This will give a cardiac output in ml/minute. Dividing the ml by 1000 will give you the litres/minute.

Cardiac Output (L/min) = Stroke Volume (ml/beat) x Heart Rate (beats/min)

Blood pressure changes are directly linked to changes in cardiac output. A larger cardiac output means that a larger volume of blood is being forced out of the heart and into the aorta. This is like if you have a small room, with a few people in it there is little contact and little pressure on the walls of the room. However if you stuff the room full of people they will be squashed together and against the walls, putting pressure on them.

2. Peripheral resistance

This is the most important factor affecting blood pressure. Peripheral resistance refers to the resistance blood encounters as it flows through blood vessels. Blood vessels are like pipes – the thinner the blood vessel the higher the pressure must be to push the blood through it. Importantly, blood vessels can constrict, narrowing vessels and increasing blood pressure. Some causes of peripheral resistance are:

- plaque build up on the inside of vessels
- old age which reduces the elasticity of blood vessels, making them less able to constrict and dilate in response to differing volumes of blood
- blood clots which narrow the blood vessels
- drugs or medications that affect vasoconstriction/dilation

3. Blood volume

Most adults have about 5 litres of blood in their circulatory system. But in cases of severe blood loss or dehydration, this can be drastically reduced. A major drop in blood volume causes blood pressure to drop. The reverse is also true. If blood volume increases, for example if the body retains too much water, then blood pressure rises. The blood volume is regulated by the renal system. In a dehydrated person, any water that arrives at the kidneys in the blood will be retained and returned to the blood. In a well hydrated person, any excess water will be eliminated by the kidneys.

How blood pressure is taken

An inflatable pressure bag is wrapped around the upper arm. The bag is connected to a pressuremeasuring instrument called a sphygmomanometer. The operator manually pumps up the bag with air until the circulation of the arm's main artery (the brachial artery) is interrupted. The pressure in the bag is then slowly released until it equals the systolic pressure in the artery, indicated by blood once again moving through the vessel. This makes a 'thumping' sound. The systolic pressure is indicated on the sphygmomanometer and recorded. Next, the blood pressure in the arm's main artery drops to equal the lowest pressure, which is the diastolic pressure. This is the pressure at which the thumping sound is no longer heard. This figure is also recorded. The operator may take a couple of readings to get the true picture. This is because many people tend to tense up during the procedure, and nervous tension may temporarily boost the blood pressure.

THE CARDIAC CYCLE

People who have done prior studies of the cardiovascular system may find some of this is revision.

The beating of the heart is simply the contracting and relaxing of the heart walls. The heart is able to function independently of any outside control because the beating of the heart is controlled by impulses sent out by two centres of specialised cardiac cells. Contraction of the atrium is governed by the Sino-atrial node while the ventricles are controlled by the atrio-ventricular node. Both these nodes are situated in the muscular heart wall.



The cardiac cycle is the time and events occurring from the start of one heart beat to the start of the next heartbeat. The heartbeat itself is then made up of different events. Systole is the period of time when the ventricles are contracting, forcing blood into the pulmonary artery and aorta. When taking someone's blood pressure, the systolic pressure is first number and gives the maximum arterial pressure caused by left ventricular contraction. Diastole is the period of time between ventricular contractions when the heart muscle is relaxed and the ventricles are filling. The diastolic pressure is the second number recorded when measuring blood pressure and gives the minimum arterial pressure during this relaxation period. The two pressures are given in mmHg (millimetres mercury) and a normal healthy blood pressure ratio is 120/80.

It is essential that once blood has been forced into the large arteries, pressure is maintained. This is achieved in the following ways:

- the closure of the valves to prevent the back flow of blood into the heart
- the elasticity of the walls of the arteries. During systole, the walls expand to accommodate the
 extra volume of blood. During diastole, the walls recoil in order to maintain the pressure on the
 blood inside the artery.

Heart Muscle Cell Contraction

The muscle cells making up the heart walls are known as cardiomyocytes (literally this term means heart (cardio) muscle (myo) cells (cytes)). The contraction of these cells is controlled by electrical impulses, like all muscle cells. However the heart has its own, independent pace makers, the sino-atrial and atrio-ventricular nodes. These nodes fire off electrical impulses but they do so without direction from the brain and nervous system. This means your heart does not need to be told by the brain to beat. The brain and nervous system do however have some control over the heart beat, when stimuli are received and interpreted by the brain to require a change in heart rate or some other factor.

To understand how the cardiomyocytes contract we first need to understand the cell biology of these cells. Like all cells, they are enclosed by a semi-permeable plasma membrane. The plasma membrane is polarised, that means, there is a difference in the net charge on one side of the membrane compared to the other side (the intracellular and extra cellular environments have different charges). This is due to differing concentrations of different ions (charged molecules) between the extracellular fluid and the cytoplasm. Cardiomyocytes also have a nucleus, and not surprisingly, a relatively high number of the energy producing mitochondria as well as Golgi apparatus, and other organelles. However, they also have a muscle specific organelle known as the sarcoplasmic reticulum. The sarcoplasmic reticulum, along with the plasma membrane polarisation are vital for muscle cell contraction.

As an overview, muscle contraction occurs following stimuli from a nerve cell. The effect of the stimuli is to depolarise the plasma membrane, which in turn results in a series of events inside the cell that result in the cell contracting. For cardiomyocytes, the action potential, or stimulus, they receive results in membrane depolarisation by causing an influx of calcium ions into the cardiomyocyte. The calcium ions (which carry a charge of 2+) move into the cell through voltage gated calcium channels. There are a number of steps in the contraction of a cardiomyocyte:

1. The resting myocyte maintains its negative membrane potential of approximately -85mV (millivolts) due to passive movement of potassium ions (K+) out of the cell. This occurs because there is a much higher concentration of K+ inside the cell, compared to outside the cell. The membrane is at this point much less permeable to sodium ions (Na+) which are found in much higher concentration outside the cell than inside it.

2. A stimulus is received by the cardiomyocyte, from a nerve. This changes the extracellular environment and triggers an uptake by the cell of sodium ions (Na+). This is because the extracellular concentration of Na+ is much higher than the intracellular concentration and the arrival of the stimulus at the cardiomyocyte opens voltage gated Na+ channels, allowing Na+ to flood into the cell. This process adds positive charge to the inside of the cell, making it closer to the charge outside the cell. This is membrane depolarisation.

3. As the stimulus from the nerve finishes, the Na+ channels on the cardiomyocyte close. Calcium ions (Ca2+) now enter the cell through voltage dependent calcium channels. Although Na+ has entered the cell, there is still a difference in charge between the cytosol and the outside environment, so calcium moves into the cell. This influx of calcium also occurs due to the relatively low concentration of free calcium ions inside the cell compared to outside. In the polarised cell, the free calcium concentration in the cytosol is about 1×10^4 (0.0001) millimoles per litre (mmol/L), compared to the average 2mmol/L in the extracellular fluid. The combination of Na+ and Ca2+ influx takes the membrane potential past zero.

3. The calcium channels close. Potassium ions (K+) move out of the cell and this decrease the voltage inside the cell, bringing it back to the resting membrane potential of about 85mV.

In stage three, inside the cell, the calcium that just entered triggers the release of large stores of calcium ions from the sarcoplasmic reticulum into the cytosol. This process is driven by changed voltage in the cytosol (due to the influx of calcium and sodium ions) which opens calcium channels on the membrane of the sarcoplasmic reticulum and the calcium moves from the area of high concentration (the inside of the sarcoplasmic reticulum) to the area of lower calcium ion concentration (the cytosol).

In the cytosol, there is a network of thin filaments (made up of a core of actin, with tropomyosin twisted around it, and small troponin molecules attached to the tropomyosin) and thick filaments (made up mainly of myosin). The free calcium in the cytosol binds to the toponin molecules on the thin filaments. This causes the tropomyosin to move, exposing binding sites on the thin actin filament for myosin. The myosin head groups of the thick filaments, each of which have ATP bound to them, bind to the thin filament, and this process requires energy, so the ATP is converted to ADP. The break down of ATP to ADP causes a change in the shape of the myosin head, which results in it pulling the thin filament which it has just attached to over the top of the thick filament. This event takes place multiple times, pulling the thin filaments over the thick filaments, shortening the cardiomyocyte and causing contraction of the heart wall.

Contraction of the heart muscle ends when the membrane potential is restored, as this closes the voltage dependent calcium channels in the sarcoplasmic reticulum. This means there is no more calcium entering the cytosol, and what calcium is there is being actively pumped back into the sarcoplasmic reticulum for storage until the next stimulus. Without the calcium binding to troponin, the tropomyosin covers up the myosin binding sites and muscle contraction stops.

The contraction of myocytes is typically broken down into phases, based on the membrane potential:

• Phase 0 (Rapid Depolarisation)

Opening of the voltage gated Na+ channels on the myocyte plasma membrane. Calcium channels open later. Rapid depolarisation of the membrane occurs, changing the membrane potential from about -85mV to about +20mV. The depolarisation is almost entirely due to the Na+ influx; much more Na+ enters the cell than Ca2+.

• Phase 1 (Slight Repolarisation)

Na+ channels close and the membrane potential decreases slightly as K+ ions move out of cells along their concentration gradient. The calcium influx delays repolarisation, allowing contractions to last longer in cardiomyocytes than other contractile cells and also longer than depolarisations in nerve cells.

• Phase 2 (Plateau)

The membrane potential plateaus at about 0mV as Ca2+ slowly enters the cell, while K+ continues to leave.

• Phase 3 (Repolarisation)

The calcium channels on the plasma membrane close. K+ channels are still open, so the membrane potential begins to be restored. Membrane potential goes from approximately 0mV back to the resting potential of about -85mV.

• Phase 4 (Polarised and Resting)

This is the period of time between when the membrane potential has been restored and when depolarisation is triggered by a new electrical impulse. The muscle cells are relaxed and so, this phase relates to diastole, either atrial, when atrial cardiomyocytes are relaxed, or ventricular when the ventricular cardiomyocytes are resting.

Electrical Control of the Heart Muscle Cells

The electrical control of the heart occurs via the sinoatrial node, the atrioventricular node, the bundle of His and.

Sinoatrial Node (SA Node)

The sinoatrial node is found right at the top of the wall of the right atrium, near where the superior vena cava enters. It is composed of modified cardiomyocytes that do not contract but transit electrical impulses much more rapidly than normal cardiomyocytes. The cells of the sinoatrial node initiate the action potential that triggers cardiac muscle contraction. It triggers action potentials without influence from the nervous system; however, the rate and strength of contractions are influenced by feedback from the nervous system and brain. The sinoatrial node is essential the hearts natural pacemaker. How often the cells of the sinoatrial node depolarise and trigger an action potential determines how often your heart will beat.

The electrical impulses (action potentials) from the sinoatrial node trigger co-ordinated contraction of both atria by causing depolarisation of the cardiomyocytes in that area. The impulse travels on to the atrioventricular node.

Atrioventricular Node (AV Node)

The atrioventricular node is found on the bottom wall of the right atrium, along the wall of cardiac tissue that divides the two atria. It is also comprised of modified, non-contractile cardiomyocytes. Its function is to hold up the electrical impulse from the sinoatrial node. This provides a delay that allows the atria to fully contract before the cardiomyocytes of the ventricles are stimulated to contract. This ensures that the ventricles are full of blood before they contract. The electrical impulse then travels on to the bundle of His, also known as the atrioventricular bundle. When the sinoatrial node is damaged, the atrioventricular node can take over as the primary pacemaker for the heart.

Bundle of His (Atrioventricular bundle)

The bundle of His is located immediately inferior to the AV node; in the wall (septum) that divides the ventricles. It branches as it travels down the septum and functions to provide very rapid transmission of the electrical impulse from the AV node to the Purkinje fibres. This function is due to the bundle also being composed of the modified non-contractile cardiomyocytes.

Purkinje Fibres

These are the terminal end of the electrical conduction system of the heart. They run from the base of the septum up and around the outer walls of both ventricles. Like the rest of the conduction system, they are made up of modified cardiomyocytes. (The term fibre here refers to muscle fibres, or cells, not to nerve fibres).

It is the cells of the Purkinje fibres that deliver the electrical impulse that originated in the SA node to the ventricular cardiomyocytes, causing a co-ordinated contraction of both ventricles.



SELF ASSESSMENT

Perform Self Assessment Test 2.1. If you answer incorrectly, review the notes and try the test again

PROBLEMS WITH BLOOD PRESSURE

Normal blood pressure is 120/80 mmHg. The first value (the 120) is a measure of systolic pressure and the second number is the measure of diastolic pressure. When blood pressure drops abnormally low the condition is known as hypotension. When blood pressure is abnormally high the condition is called hypertension. Both conditions can be caused by other problems or can themselves result in further problems.

Systolic hypertension

This is a condition where the diastolic pressure (that produced by atrial contraction) is normal, but the systolic pressure (that produced by ventricular contraction) is abnormally high. Systolic pressure over 140mmHg indicates systolic hypertension. Higher than 160mmHg is severe systolic hypertension. Systolic pressure over 120mmHg is not typically classified as hypertension, but is considered unhealthy.

Diastolic hypertension

This is a condition where the systolic pressure is normal but the diastolic pressure is abnormally high. It is diagnosed when diastolic pressure is above 90mmHg, severe diastolic hypertension occurs when the pressure reaches 100mmHg. Any diastolic pressure over 80mmHg is considered unhealthy.

Hypotension

This is far less common than hypertension and is typically secondary to another condition such as heart contraction abnormalities or direct injury to the heart, injury resulting in loss of large volumes of blood or as the side effect of medications such as beta blockers. Severe overheating can also result in systolic hypotension because it causes the peripheral blood vessels to dilate in order to dissipate heat. This lowers peripheral resistance, in turn reducing blood pressure. Any systolic pressure under 110mmHg is considered systolic hypotension. Any diastolic pressure under 60mmHg is considered diastolic hypotension.

Hypertension can cause damage to the arteries, and can also cause hypertrophy (abnormal overgrowth) of the cardiac muscles that make up the atria and ventricles. People with chronically high blood pressure are more susceptible to strokes and also to renal disease. High blood pressure can also damage organs a situation known as malignant hypertension.

Hypotension can result in damage to the heart and other organs due to oxygen deprivation. It can also cause people to pass out or faint, as occurs with heat stroke, due to lack of blood supply to the brain. In extreme cases it can lead to seizures.

BLOOD PRESSURE AND EXERCISE

From the above we can identify several relationships between exercise and blood pressure

• Exercising increases heart rate.

An increased heart rate causes increased cardiac output and increased blood pressure. It occurs to meet the increased oxygen and nutrient demand of the working tissues.

• Training can increase stroke volume over time

Increased stroke volume occurs because of the slower resting heart rate. This means there is more time for blood to flow into the ventricles before they contract. This is one of the positive effects of exercise on cardiac health as it means that when the heart is required to increase blood flow to the body it doesn't have to work as hard as each heart beat is more efficient (moves a larger amount of blood).

• Too much exercise without fluid intake can cause dehydration

As you exercise your body temperature rises due to the increased movement and metabolism occurring. Your body responds using several mechanisms, once being sweating. Sweating results in a loss of water. If you aren't replenishing the water, you will become dehydrated. Dehydration results in a smaller volume of blood and this can cause hypotension. This is worsened if you are unable to cool properly, as you body also uses vasodilation to increase the volume of blood in peripheral vessels. This blood transfers heat to the external environment. However, vasodilation also reduces peripheral resistance, magnifying the hypotension caused by dehydration.

Distribution Of Blood Flow

The distribution of blood flow to selected body organs differs during rest and during strenuous exercise is different. The amounts in millilitres per minute are shown in the table below:

	At Rest	Strenuous Exercise
Brain	750	750
Heart	250	750
Skeletal Muscle	1,200	12,500
Skin	500	1,900
Kidney	1,100	600
Abdomen	1,400	600
Other	600	400
Total Blood Flow	5,800	17,500

Distribution of Blood Flow (ml/min)

As the table shows, during exercise, nearly all of the increased cardiac output pumps blood to the skeletal muscles. A considerable increase in blood flow to the heart, illustrates the bodies need for the products needed to regulate normal homeostatic conditions i.e. transferring heat from the inner organs to the skin. The reduction in the blood sent to the kidneys and abdomen demonstrates the body's temporary need for oxygen in other areas of intense use.

DEFINITION Homeostasis

Homeostasis is the term we use to describe the constant state of the internal environment.

REGULATION OF HEART RATE AND BLOOD PRESSURE

The brain is the primary control centre for all organs. However, the heart has its own control centre as well. The sinoatrial node is the heart's natural pacemaker. It is a bundle of nerves located in the superior wall of the right atrium that fires at regular intervals. This firing stimulates the two atria to contract. The nerve signal is passed down to the atrioventricular node. This provides resistance in the electrical circuitry of the heart, holding up the signal. This is what causes the atria to contract first, before the ventricles. Moving on from the AV node, the signal travels down the bundle of His, into the Purkinje fibres which transmit the signal to the cardiac muscle making up the ventricles. The signal stimulates the ventricles to contract.

Sensors

There are two main types of sensors that provide information about the blood.

Baroreceptors

These receptors are found in the major blood vessels (aorta, vena cava, carotid artery) as well as in the atria. They are mechanical receptors that are triggered by stretching of the vessel or chamber walls around them. Stretching occurs with increased blood pressure. Baroreceptor signals are received by the brain, specifically a region in the medulla oblongata known as the cardiovascular (CV) centre. Actions that the body may take in response to their signalling include reduction of peripheral resistance, by vasodilation for example, or reduction of cardiac output.

Chemoreceptor

These are chemical sensors found in both the aorta and the carotid artery. The monitor blood pH, oxygen saturation and carbon dioxide levels in the blood and signal the CV centre in the brain which will then direct responses as appropriate.

PROBLEMS WITH HEART RATE

An average normal resting heart rate in an adult is between 70 and 75 beats per minute (bpm). Women tend to have a slightly higher heart rate than men. Heart rate is most rapid in newborn babies and gradually decreases with age until a person reaches adulthood. It is very easy to find out a persons heart rate. The pulse rate, which is in most cases the same as the heart rate can be determined by compressing a peripheral artery. As you do, you will feel pulses; these correspond to the pumping of blood out of the heart and thus the heart beat. If you count the number of pulses you detect over a set period of time, you can calculate your heart rate in bpm.

Variations to Heart Rate

There are a number of variations that can occur to the heart rate.

Bradycardia

An abnormally slow heart rate, typically below 60bpm (in resting adults). There is a vast array of conditions, drugs and medications or abnormalities in the transmission of the electrical signals stimulating heart muscle contractions, which can cause bradycardia. In addition people with above average cardiovascular fitness will have resting heart rates lower than normal adults, and in such people 60bpm may be normal. People with bradycardia will feel weak or faint, may be dizzy and can lose consciousness.

Tachycardia

An abnormally fast heart rate, typically above 100 bpm (in resting adults). When undertaking strenuous activities or exercise you will naturally experience tachycardia. This is a necessary change in heart rate required to ensure proper oxygenation and nutrient supply to organs and muscle tissue. However, it can also be abnormal and dangerous. Rapid beating means the heart muscle itself requires more oxygen and nutrients and it can also cause imbalances in blood gases resulting in alteration in the blood chemistry and pH. Abnormally decreases in blood pressure (hypotension) can result in tachycardia as the body compensates and tries to increase blood flow by pumping blood out of the heart faster.

Fibrillation

Depending on the heart chambers affected fibrillation can be classified as atrial or ventricular. Fibrillation is an abnormal twitching of the heart instead of the usual synchronised, regular contractions. Ventricular fibrillation is the more serious of the two classes of fibrillation, as the twitching ventricles are not able to properly pump blood to the lungs or periphery. A defibrillator is used to shock the heart back into its normal rhythm. If this is not successful the tissues of the body will die from oxygen starvation very rapidly.

• Arrhythmia

A broad term that encompasses any type of abnormal heart rate rhythm caused by problems with the electrical signalling in the heart.

Other Conditions Affecting the Heart

• Myocardial infarction

Myocardial infarction is the correct medical term for a heart attack. They occur when the supply of blood and therefore oxygen and nutrients are cut off from the cells that make up the cardiac muscle tissue. This causes the heart tissue to die due to oxygen starvation. Regions of cardiac muscle tissue that have died are ischemias and they cannot be regenerated.

• Cardiac Tamponade

The heart is enclosed by a protective, lubricating membrane known as the pericardium. It enables the heart to contract and relax constantly without rubbing against other adjacent organs and structure and being damaged or causing damage to them. When the pericardium is damaged mechanically, by infection or inflammatory reactions fluid or blood can accumulate between the pericardium and the heart. The fluid puts pressure on the outside of the heart making it hard for the contracted ventricles to relax. This relaxation is needed to allow blood to flow into the ventricles from the atria. Inadequate ventricular filling leads to poor cardiac output and ineffective supply of blood to the body. This is a life-threatening emergency that requires immediate medical aid.

ELECTROCARDIOGRAMS

These are machines that produce graphs known as electrocardiographs that depict the electrical activity of the heart. To interpret an ECG readout you need to know the following:

• Paper

Special paper is used for electrocardiographs. It is a graph paper with a grid of small squares measuring 1mm x 1mm that are indicative of specific time and voltage values. These small squares form larger squares of 5mm x 5mm.

Horizontal Axis

Along this axis, which runs from left to right, the time is recorded. The horizontal length of each small square on the grid paper is equal to a time period of 40 milliseconds (ms). The horizontal length of each large square is equal to a time period of 0.2 seconds (200ms). These values are based on the standard speed at which the paper is run through the electrocardiogram, 25mm/s.

• Vertical Axis

Along this axis the voltage is recorded. The vertical length of each small square is equal to 0.1 millivolts (mV). The vertical length of each large square is equal to 0.5mV. The amount of voltage measured for different peaks is known as the amplitude.

• Calibration signal

This is the first peak you will see on an electrocardiograph. It is set at an amplitude of 1mV and serves as a reference point.

• Set up

Electrodes are connected to the electrocardiogram and to the patient. They detect the electrical signals in the heart and send them to the electrocardiogram. The electrocardiogram draws along the moving electrocardiograph paper, moving at a constant horizontal speed of 24mm/s and moving vertically depending on the amplitude of the detected electrical signal.

Interpreting Electrocardiographs

Each cardiac cycle (heart beat) consists of a P wave, PR segment, QRS complex and ST segment, T Wave and TP Segment. Waves are peaks on the graph, segments are periods between events, and the QRS complex is a three part peak complex.

• P Wave

This represents the signal being transmitted from the SA node to the AV node. It can provide information about the atria possible arrhythmias.

• PR Segment

This occurs immediately after the P wave and in normal people it is the same amplitude as that seen during the ST and TP segment. It represents the hold up of the electrical signal at the AV node. The duration of the segment, and its amplitude are used to distinguish a range of cardiac pathologies.

• QRS complex

This is the characteristic large peak on graphs. Following the PR segment the QRS complex represents the transmission of the electrical signal from the AV node, through the bundle of His and Purkinje fibres to the ventricular cardiac muscle. It should be about as long in duration as the P wave, but the amplitude is greater. It is a complicated complex, as the Q and S peaks may be of greater or lesser amplitude than the surrounding PR and ST segments. Additionally, there may not be three clear peaks/troughs in the complex. The specific characteristics of the complex provide information on arrhythmias and ventricular abnormalities and can be indicative of myocardial infarction (heart attack).

• ST Segment

This occurs immediately after the QRS complex and in normal people will have the same amplitude as the PR segment and TP segment. Changes from the normal ST segment are indicative of myocardial infarctions (areas of heart tissue death) and myocardial infarction.

T Wave

This represents the period of time after the stimulation of ventricular contraction. At this point repolarisation of the ventricular cells occurs, resulting in a small amplitude peak. Abnormalities in the T wave may indicate ventricular abnormality, low potassium levels (potassium is required to repolarise the cells) and a variety of other conditions.

TP Interval

This is the time between the end of one heart beat and the start of the next.



SELF ASSESSMENT

Perform Self Assessment Test 2.2. If you answer incorrectly, review the notes and try the test again

SET TASK

- 1. Select three conditions affecting the heart. Research online or in textbooks to determine how the condition may alter electrocardiograph readout. Make notes, draw diagrams.
- 2. When exercising to improve cardiovascular fitness it is important to obtain and then maintain a target heart rate. To calculate your target heart rate you can use the following information:
- Calculate your maximum heart rate (HRmax): HRmax = 220 – Age (in years)
- Select your fitness level:
 Low 50 60% of maximum heart rate
 Average 60 70% of maximum heart rate
 High 75-85% of maximum heart rate
- Your target heart rate is the percentage of your calculated HRmax appropriate for your fitness level.

Having determined your target heart rate, select an exercise activity to undertake for 20 minutes. Ideally use a piece of equipment that has a heart rate monitor such as a treadmill or exercise bike. If you don't have access to any of this equipment, you will need a friend to assist you in taking your heart rate. You will need a thermometer also.

If you have an existing cardiovascular condition or respiratory condition do not undertake the exercise without medical approval. Instead, get the assistance of a healthy friend to undertake the exercise for you.

- Take your pulse rate before you start exercising, this is your resting heart rate. Also take your temperature.
- Start your exercise slowly, with a warm up period of 5 minutes
- At five minutes, take your pulse rate and temperature
- For the next 10 minutes exercise at the maximum level you are comfortably able
- After 5 minutes of take your pulse rate and temperature
- After 10 minutes take your pulse rate and temperature
- After 10 minutes of exercise you should slow down and complete a 5 minute warm down
- At the end of your warm down take your pulse rate and temperature
- At 5, 10 and 15 minutes after you finish the exercise, take your pulse and temperature.

