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Myocardial infarction: part 1

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Aims and intended learning outcomes

The aim of this article is to provide a background to myocardial infarction (MI) and to develop your knowledge of the health requirements for a patient following MI. After reading this article, you should be able to:

- State the main predisposing factors in the development of an MI.
- Describe the signs and symptoms that a patient suffering an MI might present.
- Identify the priorities in nursing management during the first 12 hours of admission.
- State the contraindications and complications of thrombolysis.
- Discuss the heath education requirements for a patient following an MI.
- Discuss the implications of the government's *National Service Framework for Coronary Heart Disease* (DoH 2000) regarding aspects of rehabilitation.

Introduction

Coronary heart disease (CHD) is one of the biggest killers in England (DoH 2000). More than 1.4 million people suffer from angina and 300,000 have heart attacks each year. The *National Service Framework for Coronary Heart Disease* (DoH 2000) introduces 12 standards with milestones and targets that are intended to be audited, in an attempt to ensure equal access to services and predictable and equal standards of care, regardless of postcode or socio-economic factors. The standards cover all areas of CHD, from prevention through to treatment, revascularisation procedures and rehabilitation.

This article looks at the causes, signs and symptoms, treatment, care, and recovery of a patient with an 'uncomplicated' MI, in the context of the national service framework. The second article will discuss the patient with a 'complicated' MI within the same context.

Anatomy and physiology of the heart

The heart is a small muscular organ, roughly the size of an adult's fist. It is located in the thoracic cavity immediately above the diaphragm and between the lungs. The heart beats an average of 70 times per minute throughout the person's life and moves more than 8,183 litres of blood per day. The heart has two main functions:

- To circulate oxygenated blood to the tissues via the high-pressure general (systemic) circulation.
- To pump deoxygenated blood to the lungs through the low-pressure pulmonary circulation, where gaseous exchange occurs.

The heart consists of four chambers: the left and right atria and the left and right ventricles. The atria are smaller than the ventricles and have thinner walls, because the atria serve as low-pressure storage chambers rather than pumps that eject the blood into the circulation. The ventricles propel the blood all the way through the pulmonary or systemic circulation and are strong enough to overcome the pressures in these systems.

Deoxygenated blood enters the right atria via the inferior and superior vena cava, on its journey

In brief

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Summary
Informed nursing care is crucial in effective treatment and rehabilitation of the patient following MI. The *National Service Framework for Coronary Heart Disease* should be integrated into nursing practice.

Keywords
- Heart disorders: nursing
- Heart disorders: rehabilitation

These key words are based on subject headings from the British Nursing Index. This article has been subject to double-blind review.

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back to the lungs to collect more oxygen. It moves from the atria into the right ventricle passing through the tricuspid valve, so called because it has three cusps or flaps. The valves in the heart prevent back flow of blood. From here the blood flows through the semi-lunar pulmonary valve into the pulmonary arteries, which take the blood to the lungs. The pulmonary arteries are the only arteries in the body to carry deoxygenated blood. In the lungs, carbon dioxide from the blood moves into the alveoli and oxygen moves from the alveoli into the blood. This is known as gaseous exchange. The freshly oxygenated blood is then returned to the left atria via the four pulmonary veins, the only veins in the body to carry oxygenated blood. It then flows through the mitral (bicuspid) valve into the left ventricle. From here it is propelled through the aortic valve and into the high-pressure systemic circulation via the aorta.

The action of the heart valves is significant in the correct functioning of the heart. If the valves become stenosed or incompetent, this leads to increased cardiac work and loss of efficiency and can result in heart failure if untreated. The mitral and aortic valves are most often affected. Both can be surgically replaced if necessary.

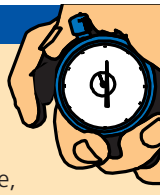
The wall of the heart is also of significance in overall functioning. It is made up of three distinct layers: the inner endocardium, middle myocardium and outer pericardium.

The endocardium is endothelial tissue that lines the heart and extends to cover the valves. It also forms the lining of vessels joining and leaving the heart. The smoothness of this tissue ensures a steady flow of blood and prevents turbulence, which would damage the vessel walls.

The middle myocardial layer is made of highly specialised cardiac muscular tissue. The structure of this tissue allows for the generation of an action potential and the wave of contraction to pass easily across the myocardium. The myocardium is dependent on oxygen for its energy requirement (aerobic respiration), and performance is, therefore, seriously impaired by oxygen deficiency. The myocardium is at its thickest over the ventricles where sufficient force has to be generated to push the blood into a high-pressure circulation. The force of the myocardial contraction is proportional to the degree of stretching of the ventricular muscle fibres. This is known as Starling's law of the heart and can be illustrated by thinking of an elastic band. The more stretch applied to the band, the greater the recoil when it is let go, so, as the ventricle fills, the fibres are stretched and the next contraction is more powerful.

The heart is surrounded by a double-layered sac called the pericardium. The outer sac enclosing the heart is the fibrous pericardium. This is lined with an inner serous layer – the parietal pericardium. The visceral pericardium (epicardium) is a second layer found as part of the cardiac wall. A potential space between the two layers contains serous fluid, that lubricates and prevents friction as the heart contracts. The heart is prevented from overdistension and damage by the outer fibrous layer.

TIME OUT 1



Before reading on, draw a diagram of the heart and label the following structures: left and right atria, left and right ventricles, pulmonary valve, pulmonary arteries, pulmonary veins, aortic valve, aorta, mitral and tricuspid valves, inferior and superior vena cava, endocardium, myocardium, pericardium. Check your answer using an anatomy and physiology text of your choice.

Blood supply to the heart As with any organ of the body, the heart itself requires a good blood supply if it is to function. This is separate from the blood flowing through the heart. Knowledge of this coronary circulation is vital to the understanding of MI. The myocardium receives its blood supply from the right and left coronary arteries – the first branches from the aorta, running in grooves on the surface of the heart. The left coronary artery divides to form the circumflex and anterior intraventricular arteries, and the right coronary artery divides to form the marginal and posterior intraventricular arteries. This arrangement ensures that the myocardial cells receive adequate oxygen and nutrients for efficient functioning.

According to Brooker (1998), the heart receives about 250ml of blood per minute, with myocardial cells extracting more oxygen from the blood than any other body structure. Most of the blood reaches the myocardium during diastole, the period when the heart is relaxed before the next contraction. This is because the blood vessels are compressed during systole (when the heart contracts). When the heart needs more oxygen, the coronary arteries dilate, increasing blood flow. Deoxygenated blood leaves the heart via the cardiac veins, some of which form the coronary sinus that returns blood directly to the right atrium.

Predisposing factors for CHD

Although CHD was targeted in the government's White Paper *Saving Lives: Our Healthier Nation* (DoH 1999), the *National Service Framework for Coronary Heart Disease* (DoH 2000) goes one step further by setting auditable milestones and goals in key areas. Standards two and three relate to reducing heart disease in the population. The standards adopt two approaches: first, lowering the average level of risk factors in the population, and second, identifying high-risk people and offering appropriate advice and treatment.

According to the framework, CHD is 'common, frequently preventable and largely fatal' (DoH 2000), responsible for more than 110,000 deaths in England in 1998, including 41,000 under the age of 75. Age is important as the target for reduction in mortality from CHD, stroke and related disorders of 40 per cent by the year 2010, established in *Saving Lives: Our Healthier Nation* (DoH 1999), referred to the under-75 age group. The key areas identified for intervention in the national service framework are reducing smoking, promoting healthy eating, promoting physical activity and reducing obesity, although several other factors are known to contribute to the development of CHD. Box 1 lists possible predisposing factors for CHD.

Smoking has long been known to be a factor in the development of CHD. According to Smeltzer and Bare (2000), nicotine decreases blood flow to the extremities and increases heart rate and blood pressure via stimulation of the sympathetic nervous system, causing vasoconstriction. In addition, it raises the chances of clot formation by increasing the aggregation of platelets. Porth (1998) also suggests that the toxic components of cigarette smoke damage the endothelial lining of the blood vessels, that might initiate the development of atherosclerosis. Because carbon monoxide from the cigarette binds readily with haemoglobin, it deprives oxygen of some of its transport mechanism and thus the tissues of oxygen. So, while the heart has to work harder, oxygen supply is actually reduced.

Obesity is also a known risk factor for CHD. Huether and McCance (1996) state that upper-body obesity is a higher indicator of risk than lower-body obesity. Obesity also predisposes an individual to hypertension and hyperlipidaemia, other important risk factors for CHD. Hypertension can precipitate or exacerbate atherosclerosis by bringing trauma to the arterial walls, causing them to narrow. This again increases the workload of the heart and its oxygen demand. Hyperlipidaemia might develop as a result of a high fat dietary intake or from systemic disease such as pancreatitis, diabetes mellitus, or hypothyroidism.

Physical activity is identified in the framework as a method of delaying or preventing CHD by reducing blood pressure, decreasing the urge to smoke or eat, improving carbohydrate metabolism and psychological outlook.

Diabetes mellitus is also known to predispose CHD, and is particularly prevalent among the Asian community (Porth 1998). It is often associated with increased lipid levels, obesity and hypertension and can be more of a risk for women than men. Alcohol is also implicated as it often increases body weight and systolic blood pressure. It also has a direct cardiotoxic effect when taken in excess (Huether and McCance 1996).

There are several other factors considered to contribute to CHD. Porth (1998) suggests that in terms of age, men over 45 and women over 55 or who have premature menopause without oestrogen replacement, are considered at higher risk. Huether and McCance (1996) say that personality type also determines risk, citing persons with suppressed anger or hostility as more likely to develop CHD.

Perhaps one of the predisposing factors that little can as yet be done to alter, is a genetic predisposition that accounts for a significant number of 'early' deaths (under age 55 in men and 70 in women) from CHD (Huether and McCance 1996). Huether and McCance suggest that those who come from families with a strong history of heart disease or stroke due to atherosclerosis are at greater risk. They also say that cholesterol levels are the most predictive risk factors for CHD, and that familial hypertension is significant.

Box 1. Possible predisposing factors for CHD

- Smoking
- Hypertension
- Obesity
- Alcohol
- Gender
- Diabetes mellitus
- Genetic predisposition
- Hyperlipidaemia
- Physical inactivity
- Personality

Pathophysiology of MI

Having looked at the predisposing factors for CHD, we will now look at how an MI actually develops. Acute myocardial infarction (AMI) can be considered the end point of the CHD–myocardial ischaemia continuum (Huether and McCance 1996). CHD is a condition of decreased perfusion of the myocardium due to occlusion of one or more coronary arteries by atherosclerosis, thrombosis or spasm (Hansen 1998). This means that a blockage in a coronary artery reduces the blood and, therefore, oxygen supply, to a portion of heart muscle, reducing its ability to pump effectively.

A condition called atherosclerosis underlies nearly all CHD. It is defined by Hansen (1998) as

a: '... pathological process of arterial wall damage and gradual occlusion of the lumen of the artery, associated with infiltration of lipids and the formation of a characteristic lesion (plaque)'.

The artery wall consists of three layers: an inner *tunica intima*, a middle *tunica media* and an outer *tunica adventitia*. The atheromatous plaque begins to build up under the tunica intima, causing a gradual narrowing of the lumen of the artery leading to partial or complete occlusion. According to Porth (1998), although fixed atherosclerotic plaques in the coronary arteries might progress to complete occlusion, they do not usually precipitate AMI, probably because the heart develops a collateral circulation to compensate for the blocked artery. However, as the plaque develops, it becomes vulnerable to rupture.

Rupture of the plaque exposes a 'foreign' surface to the blood, that can lead to the triggering of inappropriate thrombosis (clotting) by mediators of the inflammatory response. It is this resultant blood clot that can result in the total occlusion of the artery.

Although infarction is usually a result of atherosclerotic plaque disruption, it might also occur because of severe coronary artery spasm as in the condition known as Prinzmetal's angina (Julian and Cowan 1992). Coronary artery spasm is a less well-understood phenomenon usually associated with mild to moderate plaque deposition, but it might also occur in non-atherosclerotic coronary arteries. The spasm will also slow blood flow through the vessel, again promoting thrombosis. Many factors have been shown to provoke spasm, including exposure to cold, adrenaline, noradrenaline and histamine, and coronary investigations such as diagnostic angiography.

Occlusion of one or more of the coronary arteries or their branches results in decreased perfusion (oxygen supply) of the areas of the myocardium served by those vessels. When oxygen supply is insufficient to meet the metabolic demands of the affected area, myocardial ischaemia occurs. Prolonged ischaemia results in MI and death of affected myocardial cells.

The extent of the damage depends on the degree and duration of the occlusion. Because the coronary arteries penetrate the myocardium at a 90° angle, the smallest vessels are found deep in the muscle next to the endocardium. Tissues served by these vessels are therefore the first to be deprived, leading to what is known as a subendocardial infarct. With more significant occlusion, myocardial damage can extend through the entire thickness of the myocardium in the affected area known as a transmural infarction.

Ischaemic damage to cells triggers an inflammatory response and necrosis of myocardial cells releases intracellular enzymes into the blood that can be detected by blood testing, and used as confirmation of infarction. MI results in lethal ischaemic injury to a central zone of myocardium. Around that zone is a region of 'stunned' myocardial cells that are damaged but still viable. These cells are dysfunctional for a time, but recover if perfusion is restored and drug therapy is instituted that stimulates contraction. Myocardial damage can become complete and irreversible within three to four hours or less unless the infarct zone is adequately perfused by collateral circulation, or the occluded artery is opened by medical or surgical intervention.

According to Huether and McCance (1996), cardiac cells can only withstand ischaemic conditions for around 20 minutes before cellular death takes place. After only 30 to 60 seconds of hypoxia, changes are visible on an electrocardiograph (ECG). After eight to ten minutes of decreased blood flow, the affected myocardium becomes cyanotic and cooler. Myocardial oxygen reserves are used up quickly (within eight seconds) after coronary flow has ceased, and the heart then has to resort to producing energy without oxygen (anaerobic respiration), which is an inefficient process. This leads to the accumulation of hydrogen ions and lactic acid that further compromises the myocardium. Oxygen deprivation is also accompanied by electrolyte disturbances, specifically the loss of potassium, magnesium and calcium from the cells. Deprived of these vital nutrients and oxygen, the cells lose their contractility and reduce the heart's ability to keep pumping.

According to Huether and McCance, the myocardium in health takes up quantities of adrenaline and noradrenaline (catecholamines) from the blood; however, in the presence of arterial occlusion, the myocardial cells release catecholamines leading to imbalances between sympathetic and parasympathetic nervous system function, irregular heart beat and heart failure. Catecholamines also mediate the release of glycogen, glucose and stored fat from body cells and within one hour of onset of AMI, plasma concentrations of free fatty acids and glycerol begin to rise. Noradrenaline is responsible for elevation of blood sugar levels by stimulating the liver and skeletal muscle cells and suppressing pancreatic beta cell activity, thus reducing insulin secretion and elevating blood glucose levels even further. This can be detected around 72 hours after an acute MI (Huether and McCance 1996).

Porth (1998) suggests that myocardial infarctions are three times more likely to occur during the early morning hours (6am-12noon). This implies that physiological factors in the early morning, including surges in coronary artery tone and blood pressure, might promote plaque disruption and subsequent platelet aggregation and, thus clot formation within the artery.

TIME OUT 2

From what you have just learnt, how would you explain to a patient in simple terms what an MI is and how it is caused?

Signs and symptoms of MI

The first sign that a patient suffering an MI usually presents with is acute chest pain. It is typically described as feeling like a tight band around the chest and radiating down into the left arm. In the author's experience, 'crushing' and 'suffocating' are often used to describe the pain. Some patients also experience pain in the neck and jaw area. Onset is usually abrupt, the pain commencing within 60 seconds of the onset of ischaemia. The patient will usually be extremely frightened. The precise manifestation of symptoms often depends on the extent and location of the infarction, that in turn depends on the location of the blockage and the specific artery that has been occluded. Although pain is usually severe, up to 25 per cent of patients might suffer a 'silent infarction' in which no pain is experienced (Hansen 1998). These are often older patients or those who have diabetes, with diminished capacity to feel pain.

Nausea and/or vomiting accompanied by general epigastric discomfort are also signs associated with MI. Nausea and vomiting are thought to be related to the severity of the pain and by reflex stimulation of the vomiting centre in the medulla, by pain fibres. The presence of epigastric pain is often interpreted by the patient as indigestion, an interpretation that might lead to a delay in the patient seeking medical attention.

Pain and the stimulation of the sympathetic arm of the autonomic nervous system (which is responsible for the 'fight or flight' state) are responsible for several other signs that might be present in a patient presenting with an MI. These signs include tachycardia, anxiety, restlessness and a feeling of impending doom. The patient's skin is often pale and clammy as a result of peripheral vasoconstriction as the body diverts the blood supply towards the vital organs, the brain and heart. The impaired pumping ability of the damaged myocardium might also lead to hypotension.

In the infarcted area itself, the death of myocardial cells causes inflammation and the release of molecular markers and enzymes into the extracellular fluid.

MI causes a severe inflammatory response that ends in wound repair. Damaged cells undergo degradation, and scar tissue is synthesised. Within 24 hours, white blood cells infiltrate the necrotic area causing the white cell count to rise (leukocytosis) and a fever might develop within 24 hours and continue for three to seven days (Porth 1998). Initially, the newly deposited collagen matrix is mushy and vulnerable. However, after around six weeks the necrotic area is completely replaced by scar tissue, that according to Huether and McCance (1996) is strong, but cannot contract and relax like healthy myocardial tissue.

A diagnosis of MI should be based on a history of prolonged chest pain and other signs of distress, together with ECG changes characteristic of MI, and the presence of raised levels of specific enzymes in the blood, that confirm myocardial injury.

TIME OUT 3

Mr Clarke is on his way from the A&E department to be admitted to your coronary care unit. He is 46 years old and accompanied by his wife. The provisional diagnosis is that he has had an AMI. From what you have learnt describe what signs and symptoms you might reasonably expect Mr Clarke to have and what has caused them. Explain what equipment you will need to have ready for Mr Clarke's arrival and why you might need it.

Investigations and observations

Accurate and regular clinical observations in patients presenting with symptoms of MI are fundamental in patient care. Blood pressure should be monitored as an indication of how well the heart is coping and is also an important warning of the onset of complications following an infarct. Initially, this might be required every 15 minutes, particularly in the presence of certain intravenous therapy aimed at reducing the clot (antithrombolytics). Temperature should also be recorded as this can be raised following an

infarct due to the body's inflammatory response. It might also be an indicator of complications such as pericarditis. The patient's pulse rate is an indication again of the demands being placed on the heart. The harder the heart has to pump, the greater will be the need for oxygen. As already stated, the patient's blood sugar should also be recorded. This is often raised following a traumatic event such as MI (known as stress related hyperglycaemia), however, as diabetes is a known predisposing factor in the development of MI, it could be that the patient has undiagnosed diabetes, which will require investigation and treatment.

Oxygen saturation levels will also need to be monitored via a pulse oximeter. This looks at the actual saturation of haemoglobin with oxygen within the red blood cells. Supplemental oxygen must be given according to the recorded levels to prevent further stress on the damaged heart by increasing the oxygen content of the blood perfusing the coronary arteries.

Cardiac monitoring is required as a method of observing the patient's heart rate and rhythm. Although not a painful procedure, this can be distressing for the patient and the relatives. A reassuring explanation of the process must be given to the patient. The importance of good patient communication cannot be stressed enough. Anxiety will only be heightened if the patient is not informed about his or her condition and the reason for the ongoing interventions. This increases the strain on an already compromised heart muscle.

A further important investigation in the diagnosis of MI is the 12-lead electrocardiograph (ECG). This test is often carried out by nurses and is important not only in the diagnosis, but in finding the location of the infarcted area.

Several blood tests are necessary following a suspected MI. Electrolyte levels must be obtained as levels of potassium and sodium in particular have an effect on the excitability of cardiac cells. During the first five to six hours following an MI there is a shift in the distribution of sodium and potassium ions and an associated increased risk of cardiac arrhythmias. This might be life threatening if not treated. Blood glucose levels, lipid levels and a full blood count (FBC) will also be required.

Cardiac enzymes have traditionally been the most important diagnostic blood test. The enzymes raised in MI include creatinine kinase (CK) (or its isoenzyme CK-MB), lactic dehydrogenase (LDH) and to a lesser extent, aspartate transaminase (AST) (Houghton and Gray 1997). The levels of these enzymes peak at different times, CK after around 24 hours, AST after 30 hours and LDH after 48 hours and it is, therefore, impossible to wait for these results before commencing treatment. They are also not specifically cardiac in origin and raised levels might well be caused by something as common as an intramuscular injection.

A new and faster method of detecting MI is by looking for increased levels of molecular markers released into the blood as a result of myocardial cell death. These include myoglobin, and cardiac muscle troponins that are part of the muscle actin-myosin unit. The levels of myoglobin, an oxygen carrying protein normally in cardiac and skeletal muscle, usually becomes elevated within one hour after myocardial cell death peaking between four to eight hours (Porth 1998), and is therefore a useful and quick indicator of infarction.

Probably the most obvious and yet most overlooked observations come from communicating with the patient. The patient's colour, temperature on touch and ease of breathing can all be detected by looking, and confusion, mood and psychological state can be ascertained in discussion while carrying out nursing care. Observation of the patient's body language might also help to detect such things as pain and the response to medications.

TIME OUT 4

With reference to Mr Clarke,

- Describe how you would try to relieve the anxieties that Mr Clarke and his wife are expressing about the cardiac monitor.
- Explain what observations you will record on Mr Clarke, why you are recording them and what significance they might have.

Nursing care during the first 12 hours

Porth (1998) suggests that the stages of recovery from MI are closely related to the size of the infarct and the changes that have taken place within the infarcted area. Sudden death is a complication of MI and often occurs within one hour of onset of symptoms. Porth (1998) also states that many people die from fatal arrhythmias such as ventricular fibrillation (which will be discussed in part 2), within the first few hours of onset of symptoms. The care given during the first 12 hours is therefore highly significant.

Nursing observations obviously constitute much of the care for the first 12 hours, the

nature and importance of which have already been discussed.

Pain is probably the most obvious and distressing symptom and one that must be dealt with immediately. The role of the nurse is to ascertain the details regarding the pain, site, severity, duration, and other characteristics, to give medication as prescribed, and monitor the effect. The use of pain charts might be appropriate here. Pain relief during the acute phase takes the form of nitrates (a glyceryl trinitrate 500mcg tablet or sublingual spray) or intravenous opiates (diamorphine 2.5-5mg) depending on the severity. The patient might also feel nauseous and require an intravenous antiemetic such as metoclopramide 10mg.

Oxygen therapy will also be required via mask or nasal cannula and supported by recording oxygen saturation levels. It is well known that oxygen administration dries the oral mucosa and oral hygiene must not, therefore, be neglected. Bed rest must be enforced, as any activity will further increase the heart's oxygen demand.

The patient will obviously require cardiac monitoring during the acute period to assess the heart rhythm. As the patient might be cold and clammy, there could be problems with the electrodes not adhering well to the patient's skin. This can often lead to a straight line on the monitor, that can be misinterpreted as asystole. It is always wise to check a patient before raising the alarm.

Urine output is a good indicator of the ability of the heart to pump effectively and to perfuse the tissues adequately. The patient might be able to urinate without the aid of a penile sheath or catheter, and so hourly urine output monitoring might not be possible. It is important therefore to maintain an accurate fluid balance chart to detect any deterioration in the patient's condition. In terms of elimination, constipation must be prevented, as straining will again place added demand on the heart and cause anxiety for the patient. Often, bed rest dictates that the patient uses a commode at the side of his or her bed during the acute phase, which particularly in a mixed coronary care unit (CCU), could be embarrassing. The patient can be wheeled to the toilet after the first 24 hours if the recovery is uneventful.

Hygiene must not be neglected during this period although patients must not exert themselves in any way. The patient's cannula must also be observed and cared for to remain patent in case of need, and to prevent the site becoming infected. Emotional support should also be given as the patient and his or her relatives will undoubtedly be anxious. Other aspects of care during this period include the generation of a care plan/pathway that should be adapted to the patient's specific needs.

The national service framework is particularly specific regarding the medication that the patient should be prescribed following a definite diagnosis. This includes beta-blockers, which should be continued for at least one year, and ACE inhibitors, which should be reviewed after four to six weeks (unless contraindicated). It also suggests that for patients presenting who are known to have diabetes, insulin-glucose infusion should be considered.

Thrombolysis

If an infarct is highly suspected based on the patient's history, ECG recordings and signs and symptoms, aspirin and thrombolysis might be commenced.

The body will break down the blood clot itself as tissue repair takes place; however, this natural process takes time. Present in the blood is the proenzyme plasminogen. When needed this can be converted into the enzyme plasmin, that degrades fibrin within the clot as well as other clotting factors. This speeds up clot dissolution and hopefully restores blood flow through the affected artery.

The framework states that the standard to be aimed for is for people thought to be suffering from a heart attack to receive aspirin (at least 300mg orally, unless contraindicated), and that thrombolysis should be commenced within 60 minutes of the onset of symptoms, or within 20 minutes of arrival at hospital (door to needle time). The document states that 300,000 people in the UK suffer an MI each year and about 140,000 die (Mockford *et al* 1999). Many of these deaths are apparently avoidable with prompt access to treatment. The provision of aspirin reduces clot formation (an anti-thrombolytic), and reduces the risk of death following MI by about one quarter (DoH 2000). This short 'door to needle time' for the administration of thrombolysis could mean that more thrombolytic therapy is commenced in the A&E department, or in some areas commencement before arrival at hospital might have to be considered.

The common thrombolytic drugs currently used are streptokinase, alteplase (rt-PA), and reteplase. It is the role of the nurse to ensure that the infusion of thrombolytic medication runs as prescribed and that the patient's observations are recorded throughout – blood pressure is particularly important. Care must be

taken when removing the cannula or when giving injections, as normal clotting mechanisms have been interrupted and bleeding may occur.

Streptokinase is an enzyme prepared from beta-haemolytic streptococci and is a potent plasminogen activator. Fever occurs in about one third of all patients receiving streptokinase. Allergic reactions might develop as streptokinase is a protein of bacterial origin and is therefore antigenic (Galbraith *et al* 1999). Also, it cannot be given to the same patient again within six months, as antibody titres are likely to be too high. It is administered intravenously, 1,500,000 units in 100ml of dextrose over an hour.

Alteplase is usually given as a 15mg intravenous bolus followed by 0.75mg/kg over 30 minutes, and 0.5mg/kg over 60 minutes. An infusion of heparin must follow four hours after alteplase commencement; this constitutes a 5,000-unit bolus and 1,000 unit/hour infusion. Allergic reactions to alteplase are less likely as it is of human origin. It is also clot specific, meaning that it only activates the plasminogen within clots, thus minimising episodes of haemorrhage due to generalised activation of plasminogen. The one factor against its use at the moment is its cost compared to streptokinase. One dose of streptokinase would cost in the region of £80 compared to alteplase at £600 (Sheppard and Wright 2000).

Reteplase administration is relatively simple – a ten-unit bolus, followed by a further ten-unit bolus 30 minutes later. Each bolus should be given slowly within two minutes. Reteplase is also followed by heparin, again a 5,000-unit bolus followed by a 1,000 unit per hour infusion beginning after the second bolus.

Tenecteplase is another modified version of the rt-PA which is expected to become available in May 2001. Results from a large clinical field trial have been favourable (ASSENT-2 1999). Tenecteplase has been specifically developed to simplify and speed up administration and can be administered in a five to ten second single bolus dose.

Thrombolysis is particularly beneficial for the following patient groups (Sheppard and Wright 2000):

- Patients with anterior infarction.
- Patients with pronounced ST elevation.
- Patients over 75 years of age.
- Patients with poor left ventricular function.
- Those receiving early administration, within the first hour of symptoms.

Sheppard and Wright (2000) suggest that patients with pain of 12 to 24 hours' duration should only receive thrombolysis providing that they are still in pain, or if their condition appears to worsen.

Not all patients, however, will be suitable for thrombolytic therapy. Because of the mode of action of these 'clot busters', consideration has to be given to patients who have undergone recent surgery or trauma, those with coagulation defects, patients with a history of cerebrovascular disease, especially recent events with any residual disability, and those with recent symptoms of peptic ulceration, where there is the possibility of a new bleed.

TIME OUT 5

Mr Clarke has been prescribed reteplase. Understanding both the contraindications and complications of thrombolytic therapy is important when nursing a patient who has had an MI.

- Having looked at the contraindications in a pharmacy textbook, what do we need to know about Mr Clarke's medical history before the drug is commenced and what might be the consequences of ignoring these factors?
- Look at the complications of thrombolysis. As a nurse, how would you monitor him for these complications and what action would you take if they occurred?
- Mr Clarke asks what a 'clot buster' is, and does he have to tell anyone that he has had it. What is your response?

Recovery, discharge and rehabilitation

The length of stay in the CCU will vary between units and in response to the patient's speed of recovery. It is usually after a minimum of 24 hours. If recovery occurs, the patient will be transferred to a medical ward for the remainder of his or her stay and encouraged to regain independence. Gradual mobilisation will take place, and an emphasis will be placed on health promotion and rehabilitation.

The World Health Organization defines cardiac rehabilitation as: '...the sum of activities required to influence favourably the underlying cause of the disease, as well as the best possible physical social and mental conditions, so that people might by their own efforts preserve or resume when lost, as normal a place as possible in the community. Rehabilitation cannot be regarded as an isolated form or stage of therapy but must be integrated within secondary prevention services of which it forms only one facet' (WHO 1993).

The national service framework divides rehabilitation activities into four distinct phases. Phase 1 begins before discharge from hospital and usually takes the form of discussion about the patient's lifestyle in an attempt to identify any risk factors for heart disease, and general information on how to reduce these risks and so prevent a further heart attack. It is often reinforced by a variety of leaflets, videos and cassette tapes directed towards patients and their families, taking into account the socio-economic factors that affect health. It also involves education regarding medications and support groups.

Health promotion is a multidisciplinary activity, that will continue long after discharge with a referral to a health visitor and GP. This is identified as Phase 2 rehabilitation and involves comprehensive assessment of risk, provision of lifestyle advice and a general progress review. It might include the involvement of relevant trained therapists; several studies for example have indicated high levels of depression and anxiety, and problems readjusting following MI, both for the patient and for his or her partner (Bennett and Mayfield 1998, Creed 1999, Glassman and Shapiro 1998).

The internet is a valuable source of current information. The British Heart Foundation website is an excellent place to start (http://www.bhf.org.uk).

One of the rehabilitation activities that is offered to some patients recovering from MI is the cardiac rehabilitation course. This constitutes part of Phase 3 and is recommended to begin four weeks after the cardiac event and should consist of structured exercise sessions to meet the individual's assessed needs. It should also contain access to advice and support from a variety of trained experts in areas such as relaxation, health promotion exercise and psychological matters.

Cardiac rehabilitation courses are available in most hospitals for patients surviving MI. The national service framework suggests that access to such courses should widen with patients being recruited who have been admitted to hospital with other manifestations of CHD such as angina and heart failure. The national service framework also suggests that during the early post-discharge period, resuscitation training might also be offered to family members. While such suggestions are noble in their intention, the implications in terms of resources for hospitals are huge, as current provision is already overstretched.

Phase 4 involves the long-term maintenance of changed behaviours and is mainly carried out by the primary care team. They might choose to involve other parties such as cardiac specialists, psychological services and support groups.

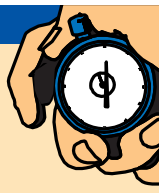
Conclusion

By working through this article you will hopefully now appreciate the broad implications for nursing knowledge that a subject like MI presents. So far, we have examined the predisposing factors for CHD, the development of CHD, the causes and signs and symptoms of an MI, specific treatment and rehabilitation following an MI in the light of the *National Service Framework for Coronary Heart Disease* (DoH 2000). The all-important contribution of competent, informed nursing care has been woven into each section as pivotal in achieving good patient outcomes.

The journey is, however, far from complete. Part 2 will examine the complications that might arise as a result of an MI and the treatments and investigations that patients might be referred for following MI

TIME OUT 6

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you are on page 54.



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