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Myocardial infarction: part 2

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Aims and intended learning outcomes

The aim of this article is to examine the complications that can arise after a myocardial infarction (MI), and their management. The tests and investigations that should be undertaken after an MI are discussed.

After reading this article, you should be able to:

- Describe the electrical conduction system of the heart.
- State the complications that might occur after an MI.
- Understand the implications of patient observations in the detection of complications.
- Identify appropriate treatment and nursing care for complications that can arise.
- Understand the investigations and interventions that might be requested.

Introduction

Part one of this article discussed the causes and development of an MI, and the nursing care and rehabilitation of an uncomplicated event. It is useful to distinguish between complicated and uncomplicated infarcts, as patients who have an uncomplicated MI have an excellent prognosis and are suitable candidates for early mobilisation and discharge, whereas most deaths occur in those who have experienced complications (Kinney and Packa 1996).

This article examines the complications that can and often do arise after an MI, and their treatment. In the light of the government's recommendations in the *National Service Framework for Coronary Heart Disease* (DoH 2000), the specific tests and investigations that

should be undertaken after an MI are also discussed.

Cardiac conduction

Part one discussed the anatomy and physiology of the heart in relation to coronary circulation. Without an electrical conduction system, however, the heart would not beat. The heart's electrical conduction system consists of the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, left and right bundle branches and the Purkinje fibres (Fig. 1).

The electrical activity can be illustrated by examining an electrocardiogram (ECG) (Fig. 2). An impulse is initiated by the SA node. The cells of the SA node are autorhythmic and can generate an impulse spontaneously, without nervous innervation. It is the cells of this node that set the heart rate and for this reason it is called the pacemaker. The impulse spreads throughout both atria and causes them to contract (systole), producing the P-wave on the ECG. After flowing through the atria, the electrical impulse reaches the AV node. Conduction is delayed slightly through this node as a safety mechanism, to allow time for the atria to contract fully. The time taken for the wave to pass from its origin in the SA node, across the atria through the AV node into ventricular muscle, is called the PR interval.

The impulse then enters the bundle of His, a specialised conducting pathway that passes into the intraventricular septum and divides into the right and left bundle branches. The impulse passes down the bundle of His and the right and left bundle branches to reach

Myocardial infarction: part 2	45-53
Multiple-choice questions and submission instructions	54
Practice profile assessment guide	55
Practice profile	27



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In brief

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Summary

Complications can and often do arise following myocardial infarction. Patients can be offered several tests and treatments. Nurses have an important health promotion role at all stages.

Key words

- 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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Fig. 1. Electrical conduction of the heart

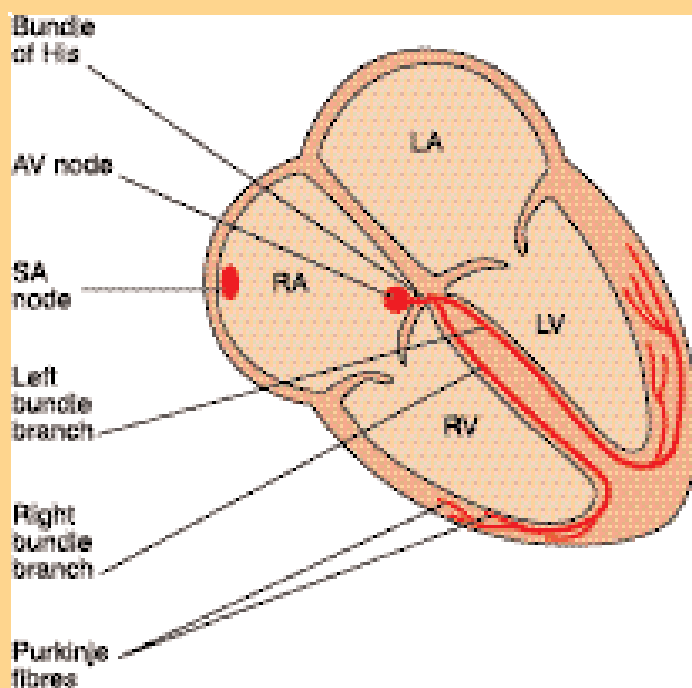
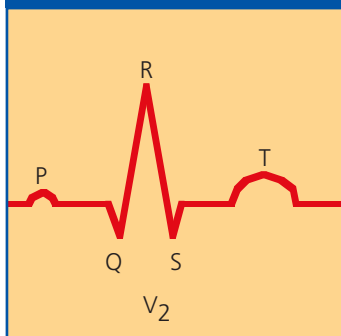


Fig. 2. An electrocardiogram



the apex of the ventricles. The conducting pathway ends by dividing into Purkinje fibres that distribute the wave of depolarisation rapidly throughout both ventricles, which contract simultaneously. The depolarisation of the ventricles is seen as the QRS complex on the ECG, and is usually complete within 0.12 seconds.

The ST segment is the transient period when no electrical current can be passed through the myocardium. It is measured from the end of the S wave to the beginning of the T wave. It is particularly important in the diagnosis of an MI and ischaemia. The T wave on the ECG is representative of the ventricles relaxing or returning to their resting electrical state, ready for the next impulse.

The pattern generated is sinus rhythm. A patient's heart rate and rhythm can be monitored easily by the application of electrodes from a cardiac monitor to the patient's chest. Several positions can be used for the electrodes, each one allowing a different view of the heart. The position in Figure 3 is lead II, which allows a good view of the QRS complex and P wave, and is the one most commonly used by nurses (Sheppard and Wright 2000). Figure 4 is modified chest lead I (MCLI). This also allows a good view of the QRS complex and P wave, but has the advantage that the leads will not interfere with the positioning of the defibrillation paddles if they are required.

TIME OUT 1

Before reading on, write down a simple description of the P wave, QRS complex, PR interval and T wave.

**The 12-lead ECG**

The cardiac monitor enables us to look at the heart from one angle, but the 12-lead ECG allows a bigger picture, from which the diagnosis and location of an MI can be made. Depression of the ST segment is a sign of ischaemia, and elevation of the ST segment (Fig. 5) indicates an emerging MI (Sheppard and Wright 2000). T wave changes are seen in ischaemia, either flattening or inversion, but are also seen within days of an MI.

Because the 12-lead ECG gives a complete picture of the electrical activity of the heart, it is possible to locate the area of infarction or ischaemia. Table 1 illustrates which leads correspond to specific areas of the heart.

TIME OUT 2

Nurses and support workers are increasingly undertaking ECG recording, which inevitably speeds up the process. It is important, therefore,

that nurses learn to interpret the ECG so that treatment, if required, is not delayed. From what you have read and with the aid of an appropriate textbook, look at the 12-lead ECG (Fig. 6):

- What specific changes can you see?
- What does it indicate?
- What is your diagnosis?

**Cardiac arrhythmias following MI**

Up to 90 per cent of MI patients will experience cardiac arrhythmias (Huether and McCance 1996). Arrhythmias present for several reasons:

- Ischaemia.
- Hypoxia.
- Electrolyte abnormalities.
- Drug toxicity.
- Lactic acidosis.
- Alteration of conduction pathways.
- Conduction abnormalities.
- Haemodynamic abnormalities.

Arrhythmias range from occasional missed or rapid beats to serious conduction disturbances that can seriously affect the pumping ability of the heart, leading to heart failure, cardiac arrest and death.



Kinney and Packa (1996) believe that the disturbances most in need of intervention include ventricular fibrillation (VF), ventricular tachycardia (VT), second- or third-degree heart block and new-onset atrial fibrillation. Tachycardia that persists for more than 24 hours in the absence of fever might be an indicator of heart failure. VF (Fig. 7) and VT (Fig. 8) without a pulse constitute cardiac arrest and should be treated using the appropriate current UK Resuscitation Council Basic and Advanced Life Support protocols (www.resus.org.uk).

Ventricular tachycardia with a pulse is three or more ventricular ectopic beats in rapid succession (Bennett 1987). The rate is usually between 120 and 250bpm and the QRS complex will be broad (more than 0.12 seconds duration). Episodes can be self-terminating or sustained. Sustained VT usually occurs at a heart rate of 150-250bpm and is remarkably well tolerated by some patients. Symptoms of VT vary from mild palpitations to dizziness, fainting and cardiac arrest. The heart cannot continue to beat rapidly for prolonged periods without compromising its performance, and without treatment can progress to VF and cardiac arrest. VT is common after MI, particularly in the first 24 hours (Nolan *et al* 1998), and often results from reperfusion of the myocardium during or following thrombolytic therapy. It is not likely to recur and is not associated with poor prognosis. VT that occurs after the first 24 hours is usually related to scar tissue. It is usually indicative of a large infarct and is more likely to recur. VT should be treated in accordance with current UK Resuscitation Council Peri-arrest Arrhythmia protocol (www.resus.org.uk). Depending on the patient's condition, this can include intravenous medication or the use of elective cardioversion.

First-degree heart block manifests as prolongation of the PR interval and is common following inferior MI. Forty per cent of patients with inferior MI and first-degree block will develop short, self-terminating episodes of second-degree (Wenckebach) or complete heart block (Nolan *et al* 1998). In anterior MI, first-degree block is often a sign of extensive myocardial necrosis. It requires no specific treatment, but close monitoring is required for the development of other blocks.

Second-degree heart block is intermittent failure to conduct atrial impulses to the ventricles, leading to dropped beats (Bennett 1987). Second-degree block is subdivided into Mobitz Type I (Wenckebach) and Mobitz Type II blocks.

Mobitz Type I (Fig. 9) is characterised by a progressive increase in conduction time over several beats until an impulse is completely

blocked, and the corresponding QRS complex does not appear. This phenomenon repeats itself with gradual lengthening of the PR interval over three-to-six beats, until a P wave occurs without a QRS complex following it. The block is usually localised to the AV node, periodic and of shorter duration than Mobitz type II. It is commonly seen following an inferior MI and does not often cause haemodynamic compromise.

Mobitz Type II (Fig. 10) is characterised by an occasional dropped QRS complex without the preceding lengthening of the PR interval. The dropped beats can occur irregularly, or every second, third or fourth beat, which is called 2:1, 3:1 and 4:1 block, respectively. The block in conduction occurs beneath the AV node (in the bundle branches or bundle of His) and haemodynamic upset is common. It often accompanies inferior or anteroseptal MI, and progression to complete heart block is common. Treatment includes the use of atropine and a temporary pacemaker, however, in many cases a permanent pacemaker is required.

In third-degree block (Fig. 11), there is no conduction of P waves to the ventricles; the atria and ventricles are working independent of each other. In patients with acute inferior MI, third-degree block requires pacing if the patient is symptomatic or haemodynamically compromised. In acute anterior MI, the development of third-degree block usually indicates an extensive infarct and a poor prognosis (Houghton and Gray 1997). Temporary pacing is recommended, therefore, regardless of the patient's condition.

Atrial fibrillation (AF) (Fig. 12) is the most common arrhythmia occurring above the ventricles following MI (Nolan *et al* 1998). It is frequently associated with acute anterior MI and usually implies a poor prognosis. Nolan *et al* also state that post-infarct AF occurs because of atrial infarction or stretch, thus generating multiple re-entry circuits within the atrium. The chaotic atrial activity occasionally manages to pass through the AV node and cause the ventricles to depolarise, resulting in irregular and narrow QRS complexes. The treatment depends on the rate and the associated features. The patient might experience chest pain, heart failure, low blood pressure and impaired consciousness and direct current (DC) cardioversion might be considered. Fifty per cent of post-infarct AF episodes last less than 30 minutes (Nolan *et al* 1998), and no treatment is necessary. If AF does persist but the patient is relatively asymptomatic, drug treatment such as intravenous amiodarone might suffice and can convert the patient back to sinus rhythm.

Sudden death is an obvious complication of

Fig. 3. Lead II electrode position

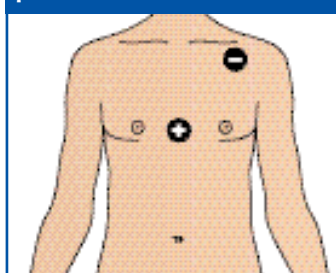


Fig. 4. Modified chest lead I electrode

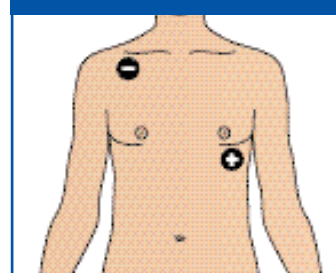


Fig. 5. Elevation of the ST segment

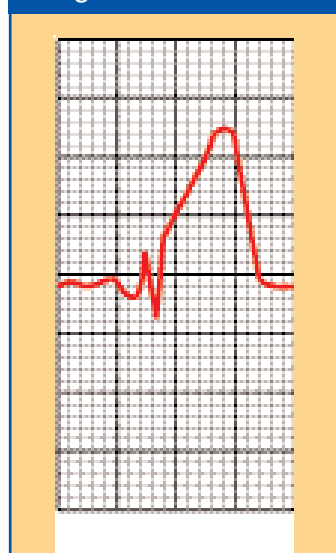




Table 1. ST segment elevation in MI

Lead containing ST segment elevation	Location of MI
V ₁ -V ₄	Anterior
I, aVL, V ₄ -V ₆	Lateral
I, aVL, V ₁ -V ₆	Anterior lateral
V ₁ -V ₃	Anterior septal
II, III, aVF	Inferior
I, aVL, V ₅ , V ₆ , II, III, aVF	Inferolateral
(Houghton and Gray 1997)	

MI, occurring within one hour of symptom onset and usually attributed to fatal arrhythmias (Porth 1998). Porth suggests that 30-50 per cent of people with acute MI die from VF within the first few hours after symptoms develop. The recent move by the government to promote widespread placing of defibrillators in the public domain is, therefore, a potentially life-saving measure. Defibrillation has also become a common skill for most nurses, further emphasising and recognising the benefit of early defibrillation in saving lives.

Fig. 6. 12-lead ECG

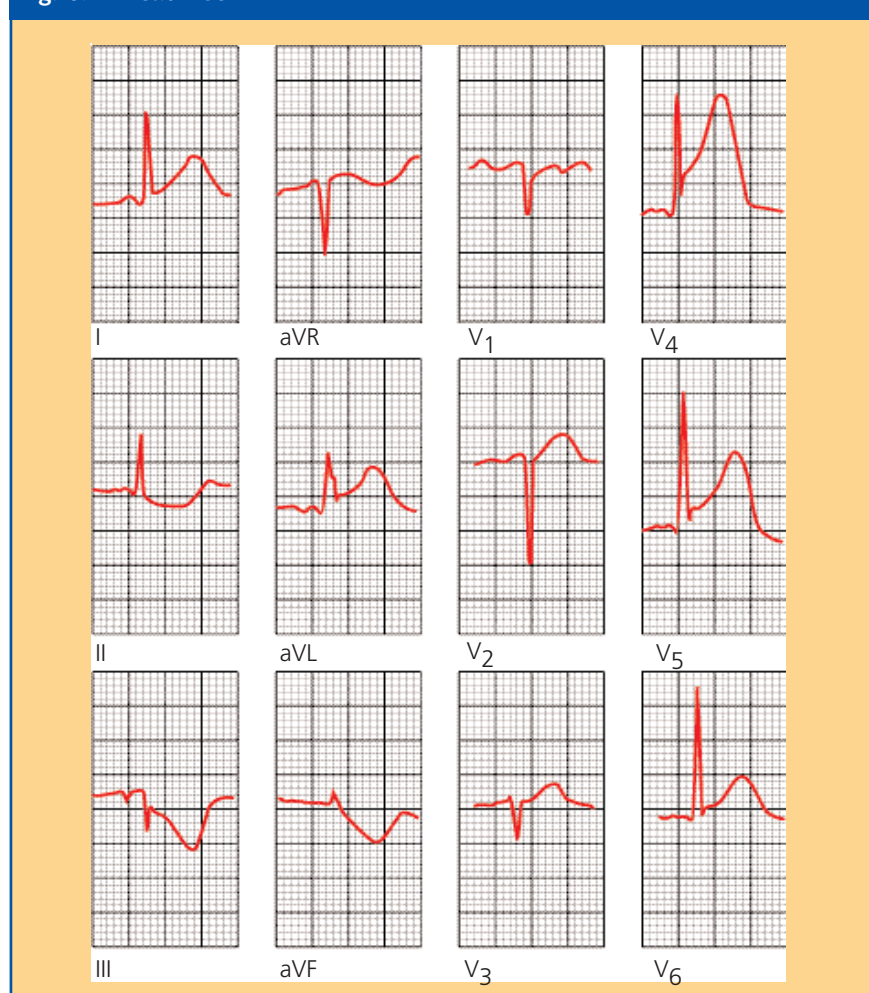
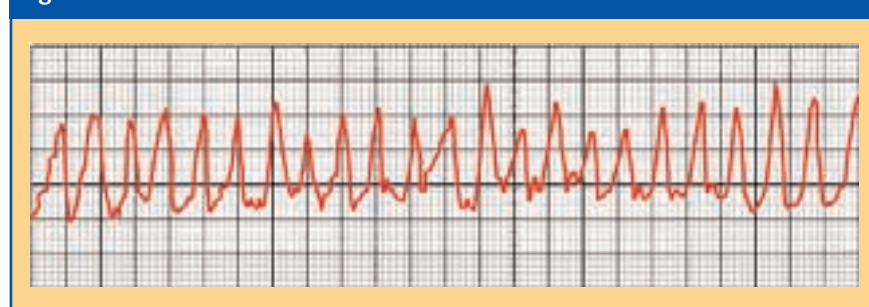


Fig. 7. Ventricular fibrillation



TIME OUT 3

Take some time to familiarise yourself with the Basic and Advanced Life Support and Peri-arrest protocols. If you have access to the internet, these can be found at <http://www.resus.org.uk>. Your local trust resuscitation officer will also be able to supply you with appropriate information and further explanation.



Heart failure

Heart failure is a frequent complication of MI. It is: '...a state in which cardiac output is insufficient to meet the metabolic needs of the body' (Kinney and Packa 1996). Before this definition can be sufficiently understood, it is necessary to explore further the concept of cardiac output. The bloodstream is the transport mechanism from which the cells gain their supplies of nutrients and oxygen that they need to function. The heart will increase or decrease its output in response to the current demand from the tissue cells. This ability to alter cardiac output is central to the maintenance of tissue oxygenation.

Cardiac output is the volume of blood ejected by either ventricle per minute. It is determined by multiplying the heart rate by the stroke volume, which is the amount of blood ejected from the ventricle in one contraction. If a healthy heart beats at a rate of 70 per minute, and the amount ejected by the ventricle with each contraction is 80ml, the cardiac output per minute will be $70 \times 80 = 5,600\text{ml/min}$.

Factors that affect cardiac output include preload, afterload and contractility. Preload is the amount of blood delivered to the ventricles during diastole (venous return). Preload is affected by the amount of blood in the system and the ability of the heart to pump the blood around the body and back to the heart. Afterload is the resistance against which the



ventricle must work. To open the aortic valve and eject its contents into the circulation, the left ventricle must generate enough pressure to overcome the pressure in the aorta. Therefore, increases in the pressure in the aorta will seriously affect the ability of the left ventricle to expel its contents. Contractility is the ability of the myocardium to pump effectively.

Maintaining cardiac output depends on there being sufficient blood in the ventricle before contraction, the heart being able to pump the blood out of the ventricle around the body and back again, and regulation of the amount of resistance that the heart must pump against.

Following an MI, the ability to pump blood around the body can be affected because of injury to the cardiac muscle and the heart can begin to fail. The body, however, responds by setting into motion a series of compensatory mechanisms to protect the cardiac output and keep the tissues oxygenated. These effects are mediated by the sympathetic branch of the autonomic nervous system, which is responsible for the flight-or-fight reaction. Autonomic alterations that affect the heart, arteries and veins result in an increase in systemic vascular resistance and arterial pressure (afterload). Venous tone increases, which in turn increases venous pressure and helps to maintain venous return (preload).

The heart rate will also increase and tachycardia can develop; initially, this will increase cardiac output. If the rate increases too much, however, the amount of time the ventricle has to refill between each contraction will be reduced, resulting in a fall in stroke volume. The increased pumping action also means that the heart needs extra oxygen to increase its performance. This puts further strain on an already damaged heart. The compensatory mechanisms of the autonomic nervous system instigate a vicious circle in an attempt to maintain cardiac output. The heart that is not strong enough to pump sets off a response that makes it work harder.

When cardiac output falls, it can result in poor perfusion of the kidneys leading to reduced urine output. This also results in the kidneys beginning to retain salt and water as an early compensatory mechanism. This triggers the renin-angiotensin mechanism, which results in vasoconstriction and the production of aldosterone from the adrenal gland, causing further sodium retention. Retention of water leads to expansion of the intravascular blood volume, which will eventually lead to oedema. Current treatment for heart failure aims at reducing the fluid overload by the use of diuretics and

Fig. 8. Ventricular tachycardia

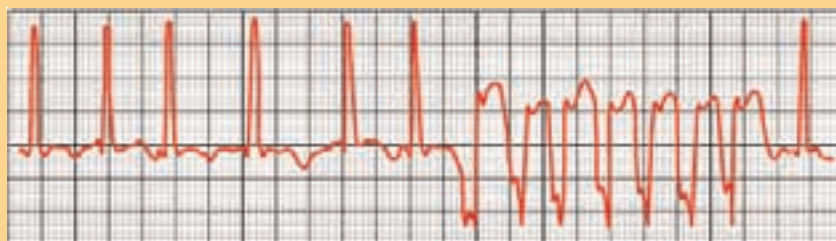


Fig. 9. Mobitz Type I block

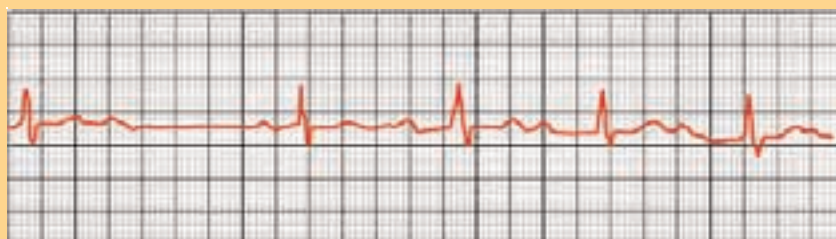


Fig. 10. Mobitz Type II block

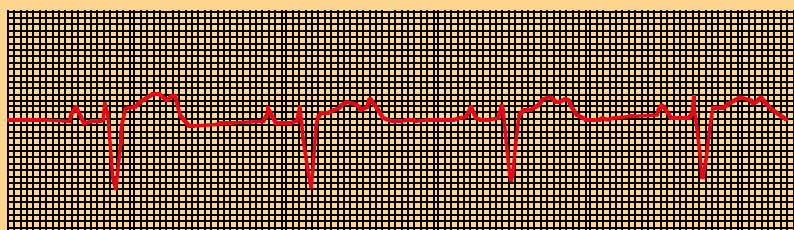


Fig. 11. Third-degree block

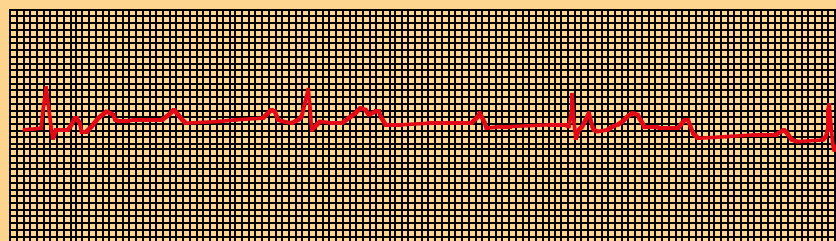
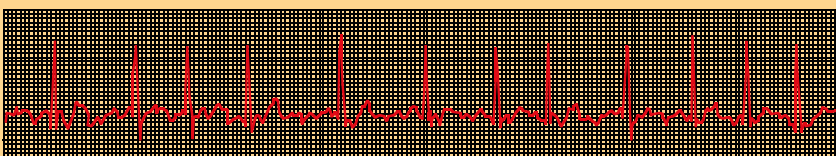


Fig. 12. Atrial fibrillation





ACE inhibitors and by restricting fluid intake.

The heart has two pumps, the right and left ventricles. In acute MI, the primary insult is usually to the left ventricle. When the ability of the left ventricle to pump blood is challenged without compromise to the right, temporary imbalance occurs in the output of the two pumps. The right side continues to move blood into the lungs but at the same time, the left side is unable to move blood adequately into the general circulation. The blood, therefore, backs up into the left atrium and increases the pressure in the left side of the heart and the pulmonary vessels. Increased pressure in the pulmonary circulation forces fluid into the pulmonary tissues and alveoli, which impairs gas exchange. This results in dyspnoea – one of the first symptoms of left ventricular failure (LVF). It often occurs during the night (paroxysmal nocturnal dyspnoea), or when lying down, and can be a frightening time for the patient, who might prefer to sleep upright with the support of pillows. It occurs when the patient goes to bed and fluid that has been distributed in the extremities during the day begins to be reabsorbed into the circulating volume (Porth 1998). When this happens, the impaired left ventricle cannot eject the increased volume and, as a result, the pulmonary pressure increases, causing a further shift of fluid into the alveoli.

The cough associated with LVF can be dry and non-productive, but is usually moist. Large quantities of frothy, pink-tinged sputum can be produced, indicating severe pulmonary congestion. Decreased oxygen levels to the brain can result in confusion and restlessness. Anxiety is also heightened for the patient because of the difficulty breathing and knowledge that the heart is not functioning properly. This increases the dyspnoea, which then increases anxiety, creating another vicious circle. Sympathetic nervous stimulation also results in peripheral vasoconstriction in an attempt to conserve oxygen supplies for the vital organs. The patient's skin might appear pale and feel cool and clammy.

Decreased stroke volume will cause an increase in heart rate to compensate, which can lead to palpitations and chest pain and the pulse is weak and thready. Without adequate cardiac output, the patient cannot respond to increased energy demands and will, therefore, become easily tired. Fatigue can also result from the increased energy expended in breathing and the lack of sleep that results from respiratory distress and coughing.

TIME OUT 4

Mr Clarke was admitted to your ward yesterday and diagnosed as having an anterior MI for which thrombolysis was given. His wife has not left him since admission. His chest pain has continued since admission in spite of frequent intravenous diamorphine and he has, therefore, remained in the coronary care unit. His wife calls you over and says that he seems confused and is struggling to breathe. You suspect he might be developing heart failure.



- What nursing observations would you carry out and what would confirm your diagnosis of heart failure?
- From what you have read, list his specific signs and symptoms and suggest the rationale for their occurrence.

Right side failure

When the right side of the heart fails, congestion of the viscera and peripheral tissues predominates, rather than congestion of the pulmonary circulation. This is because the right side of the heart cannot eject blood and cannot, therefore, accommodate all of the blood that is returning from the body via the venous circulation. The signs and symptoms will include oedema of the peripheral extremities, weight gain, liver enlargement, ascites (accumulation of fluid in the abdominal cavity), distended neck veins, anorexia and nausea, nocturia and weakness.

Treatment of heart failure centres on reducing the workload on the heart and increasing the force and efficiency of myocardial contraction. Excessive accumulation of body water can be eliminated by limiting fluid intake, controlling diet and the use of medication, in particular diuretics and ACE inhibitors. Specific treatments for heart failure are prescribed in the framework (DoH 2000).

Cardiogenic shock

Cardiogenic shock occurs when the heart cannot pump sufficient blood to supply the amount of oxygen required by the tissues (Smeltzer and Bare 2000). It can occur because of one significant or multiple smaller infarcts in which over 40 per cent of the myocardium becomes necrotic, a ruptured ventricle, significant valvular dysfunction or at the end stage of heart failure (Smeltzer and Bare 2000). It can also result from cardiac tamponade, pulmonary embolism, cardiomyopathy



or dysrhythmias. Kinney and Packa (1996) suggest a mortality figure of at least 80 per cent during the course of MI, and Hansen (1998) states that the incidence of cardiogenic shock among survivors of MI is likely to be 6-20 per cent, indicating the seriousness of the condition. This places the emphasis on early detection of deteriorating cardiac function and good nursing observations.

The compensatory mechanisms that maintain cardiac output when the heart begins to fail are short-term measures only, and with severe and prolonged heart failure they become counter-productive, further impairing cardiac function. The signs and symptoms of cardiogenic shock are consistent with those of heart failure. The patient might become cyanosed because of stagnation of blood flow and increased extraction of oxygen from the haemoglobin as it passes through the capillary beds. In all cases of cardiogenic shock there is failure to eject blood from the heart, resulting in hypotension. Compensatory mechanisms produce tachycardia, increased systemic vascular resistance and general congestion within the heart. Retention of water and sodium by the kidneys leads to further expansion of the intravascular volume and congestion, both increasing myocardial oxygen demand. Hypotension and poor renal perfusion also result in decreased urine output.

Treatment requires finding a balance between improving cardiac output, reducing the workload and oxygen requirement of the heart and preserving coronary perfusion. Several aggressive interventions can be used for cardiogenic shock caused by acute MI (Porth 1998). Rapid administration of alteplase to dissolve thrombi has been shown to increase aortic pressure and survival significantly. Another suggested intervention is percutaneous transluminal coronary angioplasty (PTCA), dispelling beliefs that infarcting myocardium cannot withstand the rigours of such interventions. The national service framework does, however, suggest that this practice needs further assessment in terms of resource implications and cost-effectiveness (DoH 2000).

Other complications

Prolonged persistent pain is frequently associated with complicated MI, caused by unusually high or secondary increases in cardiac enzyme levels (Kinney and Packa 1996). It is suggested that it is indicative of persistent ischaemia or that the infarction is in the process of extending. There are a variety of ways of treating pain, ranging

from glyceryl trinitrate (GTN) spray to opiates and intravenous nitrates, depending on the patient's response. Pain is usually a sign of increased myocardial demand for oxygen and, therefore, supplemental oxygen will be required.

Pericarditis can further complicate the course of MI. It usually occurs on the second or third day following infarct. The patient will complain of a new type of pain, often described as 'sharp and stabbing', associated with deep inspiration and change of position, particularly leaning forward. A pericardial rub can be present, although it is not always heard in patients with post-MI pericarditis. Dressler's syndrome describes the signs and symptoms associated with pericarditis, that include pain, fever, dyspnoea, ECG changes and white cell elevation. These symptoms can arise between one day and several weeks' post-infarction and are a hypersensitivity reaction to tissue necrosis (Porth 1998). Treatment usually takes the form of non-steroidal anti-inflammatory drugs.

Thromboembolism is also a potential complication of MI, arising either as venous thrombi or occasionally as clots from the wall of the ventricle. Immobility and impaired cardiac function contribute to stasis of the blood in the venous system, therefore, passive exercises should be included in patient care as a means of maintaining adequate circulation and preventing clot formation. The routine use of subcutaneous heparin has also reduced the incidence of thromboembolism in patients with compromised mobility.

The nurse should also be aware of other post-MI complications, such as rupture of the myocardium, the intraventricular septum or a papillary muscle. Myocardial rupture usually occurs on the fourth to seventh day when the injured ventricular tissue is soft and weak. It is often fatal. Necrosis of the septal wall or papillary muscle can lead to the rupture of either of these structures, resulting in worsening ventricular function. Surgical repair might be indicated, preferably when the heart has recovered sufficiently from the infarct.

Cardiac investigations

Investigations that can be undertaken following an MI range from diagnostic tests to determine the extent of the damage, to treatments to improve coronary artery perfusion. Diagnostic tests that are commonly used include exercise ECG, echocardiogram, thallium scanning and cardiac catheterisation.

Exercise ECG The exercise ECG shows the



pattern of the heart's activity during exercise. It also indicates the amount of exercise that can be undertaken and is, therefore, useful in assessing MI patients' suitability for cardiac rehabilitation courses. The test is an ECG, performed while the patient is exercising on a treadmill or stationary bike. The patient is advised to wear suitable clothing and not to have a heavy meal before the test. Patients taking β -blockers can be advised not to take them for one or two days before the test. ECG electrodes are placed on the patient's chest and gentle exercise begins, getting progressively harder. Throughout the test the patient's heart rate, blood pressure and respirations are monitored, and the test might be stopped at any time should the patient develop breathlessness or pain. The test might be negative, which means that there were no unusual or obvious changes on the ECG during exercise, or positive, indicating significant changes. A negative result is reassuring for patients, as it means that they can safely resume physical activity, and the risk of further heart complications is low.

Echocardiogram Bats can fly in the dark by transmitting pulses of sound and listening for echoes reflected from surrounding objects (BHF 1999). A similar principle is used in echocardiography. A probe placed on the chest picks up echoes from various parts of the heart and displays them on a screen. It can take time, but the procedure is painless. The test provides useful information about how the heart muscle has been damaged and is often recommended following MI or heart failure. Doppler echocardiography is another painless test that measures the speed of blood flow in different parts of the heart.

Thallium scanning radionuclide tests, including the thallium scan, are less common. In this investigation, a small, harmless quantity of radioactive substance (isotope) is injected into the blood, often while the patient is exercising on a treadmill or stationary bike. The isotope breaks down rapidly so the radioactive dose is small, similar to two X-rays. Thallium is one of the isotopes used. The test examines the blood flow to the heart muscle, providing more detailed information than the exercise ECG. Thallium scans are accurate and helpful in confirming a diagnosis of coronary heart disease (CHD); however, they are limited to hospitals that have the equipment and expertise to undertake them.

Cardiac catheterisation Cardiac catheterisation is used to make important decisions about a patient's treatment, particularly for patients with angina. The purpose of the test is to get

information about the blood pressure within the heart, the function of the pumping chambers and valves and the severity of narrowing in the coronary arteries. The procedure takes place usually as a day case, in a fully equipped angiography suite or X-ray room, and takes between 20 minutes and an hour. A long catheter is inserted into a vein either in the groin or the arm, under local anaesthetic. Using X-ray control, the catheter is advanced through the blood vessels and into the heart. The procedure is visible on a screen and the patient is encouraged to ask questions throughout. Contrast media are injected through the catheter and angiogram X-rays are taken. The test can be particularly stressful and the patient might experience chest pain and a hot, flushing sensation as the contrast media are injected. The catheter is removed when the test is complete, and if the groin was used pressure is applied. The BHF suggests that the first cardiac catheterisation was actually performed by a doctor on himself, thus indicating that under normal circumstances, the test is safe. Serious complications are rare, but it would be wrong to take the risks lightly as many of the patients have serious heart disease. The incidence of cardiac arrest during the procedure is one in 700 (BHF 1999). The test will not, therefore, be recommended unless the benefits are thought to outweigh the risks.

Revascularisation

The framework states that there is evidence that many people with atheromatous plaques and narrowed coronary arteries can have their symptoms relieved or their risk of dying reduced by revascularisation (DoH 2000). The two most commonly used techniques are PTCA and coronary artery bypass grafting (CABG). According to the framework, the UK has high rates of CHD, but lower rates of revascularisation compared to international standards (DoH 2000). Another major difference is the length of time people in the UK have to wait for treatment. Not only are overall rates of revascularisation low, but access to that care is unequal, with gender, geographical and racial variations in revascularisation rates. The documented standard for revascularisation should improve access to care and interventions that the government states are of proven clinical and cost-effectiveness. It aims to increase the overall rate of revascularisation in England and to ensure that the service becomes more equitable in terms of access and quality (DoH 2000).



The framework also states that as coronary revascularisation is a major intervention with risks as well as benefits, each patient will require careful consideration (DoH 2000). It suggests a number of factors that will be used to influence the balance of risk and benefit. Factors that increase the risk are smoking, diabetes, advancing age, recent MI and impaired left ventricular function. The service could be viewed as enhancing inequality of access for some people rather than reducing it.

Coronary angioplasty was first used in 1977 and has now developed rapidly with up to 23,500 angioplasties performed each year in the UK (BHF 1999). The purpose of the angioplasty is to 'squash' the atheroma in the narrowed artery, allowing the blood to flow through with greater ease. A catheter is inserted in the arm or groin as with an angiogram, and an X-ray is used to direct the catheter to the narrowed section. With the catheter in the narrow part, a small balloon is inflated that flattens the atheroma on either side of it. When the balloon is deflated, the channel through the vessel remains enlarged. It is sometimes called a balloon angioplasty. Although the procedure is relatively straightforward, angina-type pain is sometimes experienced while the balloon is inflated, which usually eases when the balloon is deflated.

At least nine out of ten angioplasty operations are successful; however, after the procedure, about one in three arteries narrow again within four to six months (Kinney and Packa 1996). One of the techniques used to prevent this is insertion of a stent, a metal coil or tubular mesh device that is placed in the narrowed area of the vessel to keep it open (Kinney and Packa 1996). Anticoagulation is required to prevent thrombus formation around the stent, which usually means a longer period of hospitalisation than for PTCA. Re-stenosis rates following a single stent insertion fall to between 15 and 30 per cent (Kinney and Packa 1996).

CABG is physiologically and psychologically more traumatic than PTCA. The aim is to bypass the narrowed sections of the coronary arteries surgically by grafting a blood vessel between the aorta and a point in the coronary artery beyond the narrowing. A bypass graft can be created for each of the coronary arteries affected. In some

patients this can mean only one graft, but double, triple or quadruple grafts are more often required. The vessel used for the graft comes from the patient's body. The internal mammary artery is often used, as well as sections of vein from the leg. Eight out of ten patients who undergo CABG experience immediate and lasting relief from angina, while most others say that there has been an improvement (BHF 1999).

TIME OUT 5

Following a remarkable recovery and subsequent discharge from your ward, Mr Clarke has been informed that he is to have an angiogram performed as a day case. He asks you to explain to him and his wife exactly what this entails. Using any method you choose, develop a satisfactory explanation of the procedure for Mr Clarke.



Conclusion

This article has examined the far-reaching effects of a complicated MI. It has included various arrhythmias, heart failure and cardiogenic shock, and has discussed some of the related investigations and treatments that patients can be offered following a complicated MI.

None of the interventions discussed, however, deals with the underlying cause of atheroma. This brings us back to the most fundamental principles of coronary health promotion explored in part one. Although prevention is always better than cure, it is never too late to make lifestyle changes. The role of the nurse in health promotion is just as important following a bypass graft as it is at the first episode of angina, or even when assisting a patient to select food from the hospital menu ■

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 55.



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