The two main coronary arteries (right and left) (Fig 1) begin at the root of the aorta (the aortic cusp). In most people the right coronary artery (RCA) serves the sinoatrial and atrioventricular nodes. The main stem usually comes down the right ventricle from the front, curving to the back via a right lateral approach until it stops where the right and left ventricles meet – the posterior interventricular groove (Tortora and Grabowski, 2002; Seiler, 2003). The branches that leave the RCA are:

- The conus artery – almost at the base where the RCA leaves the aorta;
- The right ventricle (RV) branch – heads to the anterior of the RV and to the side area. It has sub-branches serving the atrium. There are three atrial branches (anterior, posterior and intermediate);
- The RV branch then divides into two sub-branches. The poster lateral ventricular branch moves to the side and back of the RV while the posterior descending artery heads towards the posterior and apex of the RV and has sub-branches supplying blood to the heart septum (Reese et al, 2002).

The left coronary artery (LCA) main stem bifurcates in most people at the front of the left ventricle (LV) into the left anterior descending (LAD) and the left circumflex, which curves around the posterior (back) surface of the LV (Tortora and Grabowski, 2002; Seiler, 2003). The LAD is a continuation of the LCA main stem and supplies blood to the anterior heart wall and the septum via the diagonal branches and septal branches.

The circumflex curves around the left atrium to the side and back of the LV, and divides to create the obtuse marginal branches. Some people have a third large branch, the left intermediate artery (or ramus intermedius) giving the effect of a trifurcation of the main stem. This artery supplies blood to the lateral area of the LV wall (Reese et al, 2002).

Hyperlipidaemia

The British population has one of the world’s highest average serum cholesterol levels – two-thirds have a blood cholesterol level of >5.2 mmol/l (Brewer, 2004). Hyperlipidaemia denotes raised levels of one or more of the lipids at the following blood levels (Brewer, 2004; Yokoi et al, 2004):
This classification for chest pain patients fall into Acute coronary syndrome (ACS): Unstable angina (high risk); Unstable angina (high risk); NSTEMI – non-ST-segment elevation myocardial infarction – partial thickness or subendocardial; STEMI – ST-segment elevation myocardial infarction – full-wall thickness.

The level of blood lipids varies with sex, ethnic origin and/or country and age. They are regulated via the liver and are constantly changing. Early onset coronary atheroma is caused mainly by familial hypercholesterolaemia and early detection and management is critical (Yokoi et al, 2004). Coronary atheroma due to raised cholesterol is thought to account for approximately 45 per cent of all cardiac deaths. In the UK, one in four men and one in six women die from coronary heart disease (Brewer, 2004). These factors should be considered when managing patients with elevated lipid levels (Brewer, 2004; Yokoi et al, 2004).

Dietary management – only beneficial in people requiring a 5–10 per cent reduction in lipid levels who are motivated to achieve their target levels;

Lifestyle advice – including weight reduction, increasing exercise, smoking cessation, diet, and reducing excessive alcohol intake. Health promotion and well-being are critical factors to help people to change often long-standing lifestyles that can be difficult to change in a socioeconomic context;

Reductase inhibitors/statins (most commonly used) or a fibrate (for specific cases).

In patients with coronary artery disease, and as a preventive measure for those with related risk factors, clinical trials demonstrated that statins reduced major coronary events and had beneficial effects for coronary lumen diameter (Yokoi et al, 2004). High-density lipoproteins are also considered beneficial in reducing atherosclerosis as they carry excess cellular cholesterol back to the liver for synthesis (Brewer, 2004). The progression of coronary atheroma is illustrated in Fig 2.

Acute coronary syndrome (ACS)
This classification for chest pain patients fall into four major categories (Docherty, 2003):

Unstable angina (low risk);

Unstable angina (high risk);

NSTEMI – non-ST-segment elevation myocardial infarction – partial thickness or subendocardial;

STEMI – ST-segment elevation myocardial infarction – full-wall thickness.

Although three-lead electrocardiogram (ECG) monitoring is required for these patients, it should be supplemented by a 12-lead ECG recording and interpretation (see part four in this series). However, the main principles behind ST-segment interpretation include (Seiler, 2003; Docherty, 2003):

ST depression is seen when the coronary arteries are narrowed and there is a resultant ischaemic chest pain – as in unstable angina. This can also be accompanied by a T-wave inversion.

ST depression is also seen in NSTEMI, and biochemical markers (such as troponin or CKMB) should help in determining whether the depression is related to angina or an infarct.

ST elevation is seen in STEMI and indicates a full occlusion of a coronary artery (Fig 3).

ECG monitoring
Patients who present with acute onset chest pain (with or without previous history) should be placed on a three-lead ECG monitor for 24–48 hours initially then reassessed (Docherty, 2000). The ECG can usually be discontinued when the patient has been pain-free for 24 hours or more. The only exception to ECG monitoring is for patients with known angina, where – in the absence of other critical factors – the condition is stable and does not warrant ECG monitoring (Nicholson, 2004). The ECG can help nurses to detect abnormalities and changes in the cardiac system such as (Nicholson, 2004; Docherty, 2003):

Electrolyte imbalance;

Ischaemia or hypoxia caused by an acute coronary event or anaemia;

Arrhythmias caused by electrolyte imbalance, hypoxia or drugs and therapeutic agents.

The heart rate should generally be 60–100 beats per minute and nurses must ensure heart rate alarms are individualised and set to the patient’s condition, and the normal rhythm identified and documented (Docherty, 2002). Sinus rhythm is normal for most people, although atrial fibrillation is also acceptable, especially in older people. Any ECG rhythm change or concern should be followed rapidly by a blood pressure and pulse check, a 12-lead ECG recording and senior staff alerted accordingly (Docherty, 2002). Both bradycardia and tachycardia can lead to a significant drop in the patient’s blood pressure, cardiac output and level of consciousness (Docherty, 2002).

The ECG three-lead positioning is shown in Fig 4 and nurses should be familiar with the equipment in their own workplace. Electrodes should be changed every 24–48 hours to ensure good contact. Lead I is normally used for monitoring as it provides the most upright deflection (Docherty, 2000). The right electrode should be placed at the right shoulder, the yellow electrode at the left shoulder, and the green or black electrode below the sternum or left lateral – the sternal area is preferred as it will not interfere with defibrillation position if that is required (Docherty, 2000).

The patient should be educated on the rationale and purpose of the machine. For those patients who can ambulate (that is, pain free), telemetry ECG monitoring should be considered, or a portable ECG monitor on a stand.

REFERENCES