The Burden of Coronary Artery Disease

Atherosclerosis
Angina Pectoris
Acute Coronary Syndrome
Myocardial Infarction
Revascularisation
Arrhythmias

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Cardiovascular disease has a major impact on the health of the nation and on the health service. Figures quoted in the BHF 2015 Statistics document state that UK wide cardiovascular disease kills 154, 225 men and women of all ages. Of this figure, a quarter can be attributed to Stroke. In 2009, CVD cost the UK economy £19 billion (Townsend et al. 2012). For the first time however, the single biggest cause of death is Cancer, with mortality figures of 167, 268. With improvements in interventions, the death rates associated with CAD continues to decline, with a fall in acute MI mortality rates of over 50% between 2002 and 2010 (Smolina et al 2012). This translates to approximately 1 in 5 male deaths and 1 in 10 female deaths (Townsend et al 2012).

The reduction in incidence and mortality rates for MI is likely to be associated with smoking related legislation and cessation as well as pharmacological and interventional treatments (Knight and Timmis 2011). Reducing the associated morbidity and mortality of CAD however remains a key objective of all health services within the UK today, with strategies focusing on the continued reduction of deaths of those under the age of 75 years and closing the socio-economic gap which sees those living in the most deprived 5th of the population at a 50% greater risk of cardiovascular death (British Heart Foundation 2009). Cardiovascular disease (CVD) is not only a national issue but is a global health problem, with CVD being estimated as becoming the leading cause of death and disability in the world by 2020, surpassing that of current infectious diseases (Omoigui 2007).

The Coronary Arteries

The word ‘coronary’ is derived from the Latin word meaning ‘crown’ as the coronary arteries encircle the heart. The two main arteries of the heart are the right and left coronary arteries which originate from the base of the aorta. The right coronary artery (RCA) travels down the heart feeding the whole of the right ventricle and the lower aspect of the left ventricle through smaller branches.

The arterial wall has three layers ('tunica' from the Latin for coat): The outer collagen and elastic layer (tunica externa or adventitia), the middle smooth muscle layer (tunica media), and the inner endothelial layer (tunica interna or intima).

The left coronary artery (LCA) enters as the left main stem (LMS) and splits into two. The left anterior descending (LAD) supplies the anterior and lateral part of the left ventricle as well as the septum, while the circumflex (LCx) supplies the posterior aspect of the left ventricle (Figure 1). This is the most common situation but the actual amount and area supplied by each vessel may vary from individual to individual (Tortora and Grabowski 1996).

Atherosclerosis

The primary cause of CAD (Woods et al. 2000) is atherosclerosis. Atherosclerosis comes from the Greek 'athero' meaning gruel or wax. This relates to the necrotic core at the base of the plaque. 'Sclerosis' signifies induration or firming, and refers to the plaques cap which penetrates the luminal edge (Ladich et al. 2012).

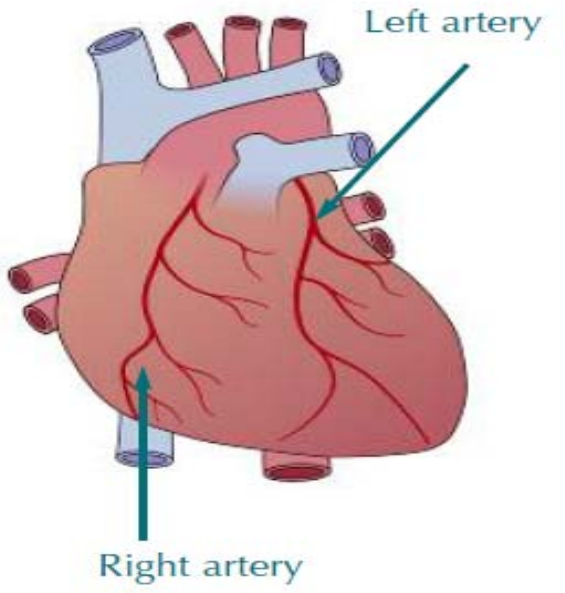

Figure 1. Left and right artery of the heart.

Atheroma develops when LDL cholesterol (LDL-c), which congregates within the tunica intima of the vessel wall, becomes oxidised. Macrophages engulf the oxidized LDL-c becoming foam cells. These cells form the framework of fatty streaks within the tunica intima (Figure 2).

There are various etiological principles which are associated with endothelial injury, including: hypertension, diabetes, hypercholesterolemia, obesity and free radical activity derived from activities such as smoking. Beyond established CAD risk factors, atherosclerosis is now thought to be associated with an inflammatory mediated disease resulting from complex interactions between leucocytes, platelets and the cells of the vessel wall (Omoigui 2007).

Disease Progression

Atherosclerosis develops over the course of several decades, often beginning in the early teenage years (Piers et al. 2008). It is often a diffuse disease which may remain asymptomatic for many years (Tuzcu and Schoenhagen 2003). The progression from asymptomatic disease to occlusive disease is associated with atherothrombosis. This occurs when the atherosclerotic plaque is disrupted triggering the clotting cascade and thrombus formation. Plaque rupture is implicated in acute cardiac events, acute coronary syndrome (ACS) and sudden cardiac death.

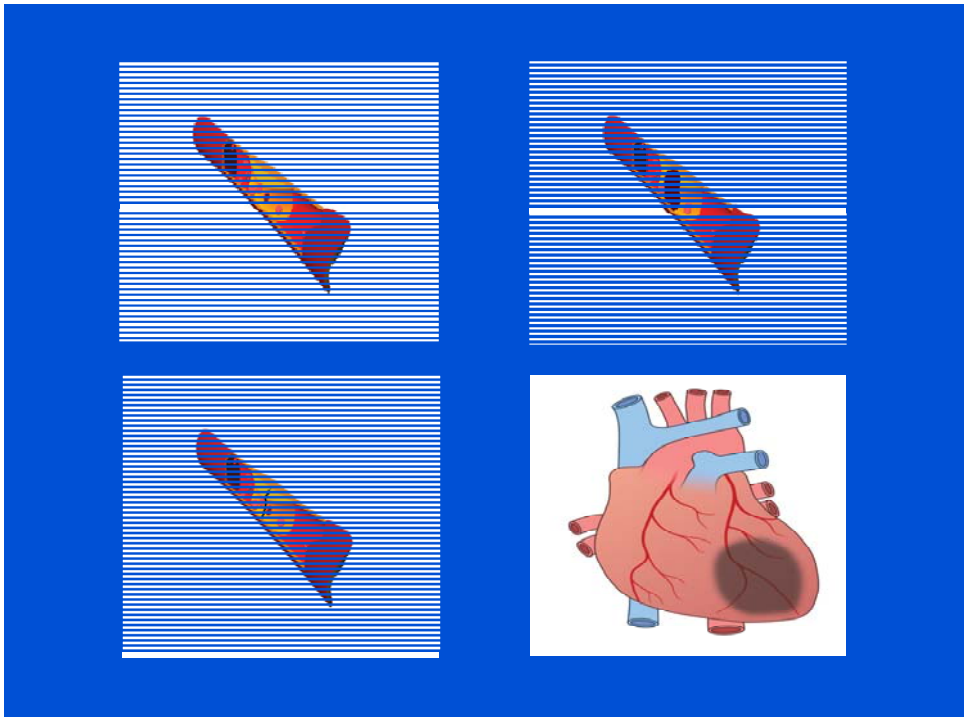

Figure 2. Plaque rupture.

Plaque rupture is believed to be the most common cause of atherothrombosis, accounting for around 70% of all events (Fuster et al. 2005). These plaques exhibit thin caps, large necrotic and cholesterol rich cores, as well as evidence of inflammation and remodelling.

Diffuse plaques and the identification of focal lesions can be explained through the exploration of the remodelling response. The initial cardio-vascular response is to accommodate the plaque load by expanding the vessels size. This takes place with minimal reduction in lumen size. This process is known as positive remodelling (Figure 3). In many cases lesions are non-occlusive but are vulnerable to rupture. In fact most lesions which evolve into acute MI would be considered non-obstructive with a mean diameter of around 48% (Moreno 2010). Over recent years the ability to assess the vulnerability of coronary plaques has continued to advance. Techniques such as computed tomography angiography, intravascular ultrasound and coronary magnetic resonance angiography may allow vessel walls to be assessed for various pathologies such as: plaque remodelling, calcification, core composition and cap thickness.

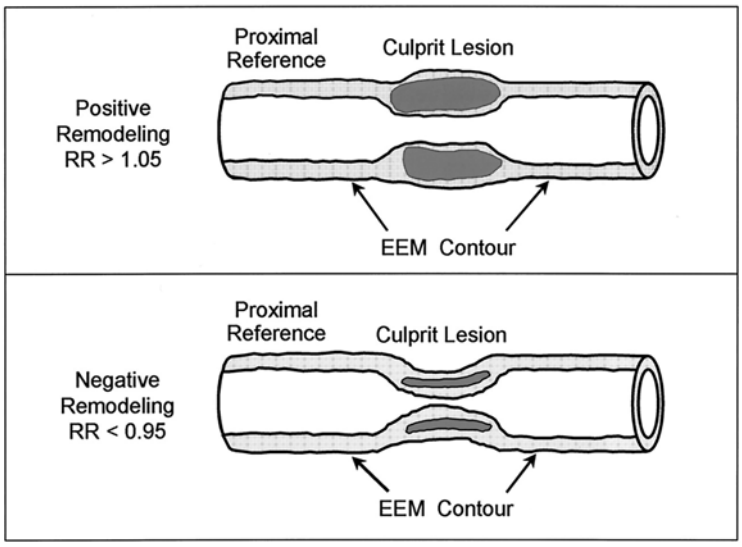

Figure 3. Positive and negative arterial remodelling (Schoenhagen et al. 2001)

Predicting Sudden Cardiac Events

The knowledge of what triggers a sudden cardiac event is the first step in being able to predict who is most at risk.

A key cause of atherothrombosis and sudden cardiac event is **plaque erosion**. Erosion tends to occur more frequently in younger women and cigarette smokers with the pathophysiology of atherothrombosis being linked to a systemic predisposition to thrombosis rather than a local mechanism. Identifying areas within coronary vasculature which are prone to erosion has so far proven to be difficult (Newby 2010).

Biomarkers such as interleukins, tissue factor, fibrinogen and highly sensitive C-reactive protein (hsCRP) have been used to predict unstable angina and those at risk of MI (Berneis et al. 2010), while the ability to measure carotid intima thickness has also been considered a valuable predictive marker (Corrado et al. 2009).

Elevated inflammatory markers have been linked to the presence of multiple lesions as has coronary artery calcification scoring (Piers et al. 2008; Corrado et al. 2009). Calcification can be detected using electron-beam computed tomography and this score can be used to assist with the prediction of cardiovascular events and mortality independent of additional risk stratification. This may lead to improved detection of those predisposed to plaque rupture and continue to assist practitioners in the identification of those at greatest risk of CVD and events.

(See chapter on predicting cardiovascular risk for more details on risk factors.)

Ischaemia

Myocardial ischaemia occurs when there is an imbalance in myocardial perfusion to myocardial demand. Although tissue hypoxia primarily refers to the lack of oxygen, substrates (glucose and free fatty acids) also play a key role in the pathogenesis of myocardial injury (Kones 2010a). Myocardial demand is affected by individual heart rate, blood pressure, and myocardial wall tension. In normal coronary arteries, 75% of the oxygen is absorbed from the circulating blood Adjusting to decreased myocardial oxygenation cannot therefore be easily achieved by increasing oxygen uptake (Kones 2010a).

The physiological response via the auto-regulatory system is to increase coronary blood flow via vessel dilation. In healthy coronary arteries the vessel walls may expand 4-6 times the diameter of their normal resting capacity (Kones 2010b). This process may also be enhanced through pharmacological interventions. The normal physiological response is however limited in those with atherosclerosis. Adequate perfusion of the myocardium is also influenced by the coronary microvasculature. Assessing these small vessels cannot however be achieved by standard angiography.

Angina Pectoris

Angina is a symptom described as a transient discomfort in the chest which is generally associated with CAD due to reduced myocardial perfusion without necrosis

(NHS Centre for Reviews and Dissemination 1997). It is further defined by the general course of symptom relief, manifestation, radiation and duration of the discomfort felt.

Angina is usually characterised by a retrosternal (behind the breast bone) sensation which may be described by the patient as tight, squeezing, pressing or heavy.

Although angina is often referred to as 'chest pain' many patients may describe it as other types of discomfort rather than pain (De Bono et al. 1993).

Angina may radiate to both sides of the chest and to the arms (particularly the left arm), shoulder, back, neck, mid abdomen and jaw or teeth (McGillion et al 2008). Occasionally the discomfort originates in these areas and does not affect the chest at all. The symptoms of angina may be accompanied by weakness, fatigue, nausea, diaphoresis and dyspnoea; in some patients it is shortness of breath that is the main symptom. Differential diagnosis may include gastrointestinal, musculoskeletal, pulmonary and panic disorders (Kones 2010a).

Stable angina- Angina is considered stable if symptoms are experienced over several weeks, are induced by specific and predictable circumstances and there is an absence of changes in symptom characteristics and deterioration (McGillion et al 2008).

Stable angina is primarily precipitated by physical activity such as exercise, cold and/or windy weather, digesting a heavy meal or when under emotional stress, with symptoms easing within 1-5 minutes of ceasing activity or more rapidly if nitrates have been used (Kones 2010a).

Many people have adapted to their symptoms by curtailing their activity to avoid the frequency of their symptoms. This however may lead to reduced exercise capacity and restricted social interaction, having a fundamental impact on the individual's quality of life (Xie et al, 2008).

This type of angina is primarily associated with significant obstructive lesions, around 50% vessel occlusion of the left main coronary artery or a stenosis of 70% or greater in other major epicardial vessels.

Unstable angina – see page 13 Acute Coronary Syndromes section.

Silent ischaemia- Even though angina is considered a common symptom of myocardial ischaemia, its prevalence may be underestimated, with two thirds of

those with stable angina experiencing no symptoms during ischaemia and 15-30% of MIs being silent (Deedwania and Carbajal, 1991). It is known that those who experience angina symptoms are also likely to have asymptomatic episodes (Mulcahy 2005).

Why some episodes of ischaemia are symptomatic while others occur without detection remains unclear. As the identification of these individuals is hampered by the lack of traditional clinical symptoms, unrecognised events and delayed treatment may lead to poorer prognosis (Kones 2010a).

Diagnosis

Although angina episodes are considered reversible, they should not be thought of as benign as the prognosis of those with angina in relation to future cardiac events or cardiac related death is similar to those who have experienced acute MI or revascularisation, with general health status being noted as being poorer (Buckley and Murphy 2009).

It is important to be aware that around 12% of those discharged from an emergency department following an episode of chest discomfort will go on to develop an acute MI (Buckley and Murphy 2009). The use of highly sensitive biomarkers such as Troponin (see Myocardial Infarction section this chapter) and development of rapid access chest pain services may however result in enhanced care, reducing inappropriate hospital admissions and discharges.

For those with new on-set chest pain, the primary objective is to diagnose if ischaemia is present and if this is due to an obstructive or non-obstructive lesion. Investigation aims to risk stratify individuals in order to deliver effective management strategies.

First line investigations will include obtaining a full medical history, routine blood sampling, resting electrocardiography and exercise electrocardiography (exercise tolerance testing or ETT). Graded exercise testing is carried out using the Bruce Protocol and Duke Treadmill score.. This information along with any other signs or symptoms experienced during exertion, such as arrhythmia, will aid diagnosis and offer guidance towards risk stratification, the necessity for further investigation and treatment options. Symptom severity may not however directly correlate to the extent of the disease (Kones 2010a).

In some cases, exercise testing may be considered inappropriate if, for example: over 1mm ST segment depression is detected on resting ECG, an MI has occurred within the previous two days, unstable angina is suspected or there is poor rhythm control. Two days prior to exercise testing, heart rate control medication may be stopped with Digoxin being stopped for around a week prior to investigation.

It is important to note that exercise testing is imperfect, with false negative and positive results being found in around one third of the tests carried out (Kones 2010a). It is however inexpensive, simple and often easily accessed.

Some people who require ETT may be unable to carry it out due to reduced physical capacity or physiological factors which may increase their risk if they exercise to a high level of exertion. In these circumstances pharmacological stress testing may be required. Here medication such as adenosine, dipyridamole or dobutamine may be used to induce increased cardiac blood flow, with perfusion imaging then being used to detect areas of reduced blood flow. Additional investigations to aid diagnosis may also include echocardiography, magnetic resonance imagining or magnetic resonance coronary angiography, computed tomographic coronary angiography and intravascular ultra sound. Current guidelines suggest that most individuals with suspected CAD undergo non-invasive testing, but around 15% of patients still do not have this done (Kones 2010a). In some areas additional functional and quality of life screening tools such as the World Health Organisation's Rose Angina Score (Rose 1962), the Seattle Angina questionnaire (Spertus et al. 1995) or the York Angina Beliefs questionnaire (Furze et al. 2003) may also be utilised in the assessment and diagnostic process.

Angina management

There are three key aims in the management of those with stable angina:

- Reducing or stabilising the number of episodes and severity of the episodes to improve symptom control and quality of life
- Risk stratifying in order to prevent or limit future cardiovascular events
- Stabilising and reducing atherosclerotic plaque formation

(Kones 2010b)

Pharmacological Intervention: Beta-blockers are generally used as a first line therapy for the relief of stable anginal symptoms. Those who are unable to tolerate beta-blockers may be treated with a rate limiting calcium channel blocker or a long-acting nitrate or Nicorandil (Scottish Intercollegiate Guidelines Network [SIGN] 2007b). Sublingual Glyceryl Trinitrate is prescribed for the immediate relief of symptoms and before performing activities which are known to bring on angina (SIGN 2007b).

If symptoms are not controlled with a beta-blocker, a calcium channel blocker should be added (rate limiting calcium channel blockers are only be used with caution when combined with a beta-blocker) (SIGN 2007b). Other medications may include Ivabradine and Ranolazine.
Patients with stable angina due to atherosclerosis will also receive aspirin and statin therapy and may be considered for an angiotensin-converting enzyme inhibitor.

Invasive Management: Coronary artery bypass grafting (CABG) and percutaneous coronary interventions (PCI) are both appropriate options for those with anginal symptoms who fall into the moderate or high risk categories as determined by the GRACE scoring system. See NICE clinical guideline 94 (2010c) for more details. Cardiologists may vary their advice due to individual diagnosis and comorbidities and developments in percutaneous angiography and stent production has seen a rise in the complexity and number of lesions tackled at any one time.

For further information on revascularisation procedures see page 23. N.B.
Patients should ONLY participate in the Heart Manual programme if revascularisation has taken place and/or symptoms have stabilised.

Acute Coronary Syndrome (ACS)

The underlying cause of CAD is atherosclerosis and how this condition manifests itself varies (National Institute for Health and Clinical Excellence [NICE] 2010a, NICE 2010c). For some individuals a slow progression of atherosclerotic lesions results in decreased vessel patency and ischaemic symptoms, while others may experience a sudden event with limited or no prior symptoms detected. These sudden events are known collectively as acute coronary syndrome (ACS).

Classification

ACS encompasses a spectrum of unstable CAD, from unstable angina to MI. All have a common aetiology in the form of sudden thrombus formation on a damaged and inflamed plaque (NICE 2010c; SIGN 2013).The principles behind the presentation, investigations and management are similar, with some very important distinctions depending on the diagnosis; unstable angina or clinical myocardial infarction (Figure 4) (Fox et al. 2004).

If the volume of thrombus is extensive, causing total occlusion of the vessel lumen, myocardium distal to the obstruction will experience a rapid decline in perfusion and myocyte necrosis will ensue. If however the thrombus is insufficient to fully occlude the artery or occurs temporarily, then damage is likely to be less severe or intermittent. In these circumstances, myocyte necrosis is still likely to occur but to a lesser extent than ST segment elevation myocardial infarction (STEMI) (NICE 2010c). It should however be pointed out that 30% of coronary arteries appear to reperfuse spontaneously (without intervention) within 12 hours of plaque rupture. This has been associated with endogenous fibrinolysis (Newby 2010). Late presentation particularly in those who have experienced what may be considered ‘silent events’ may therefore hold as poor a prognosis as those clearly recognised as presentation with STEMI (Valenci et al. 2011).

In patients who experience sudden clinical symptoms which appear cardiac in nature such as angina, dyspnoea, diaphoresis, nausea or emesis, the underlying pathology may be similar but may not result in myocardial necrosis. In this situation, the syndrome is classified as unstable angina (NICE 2010c).

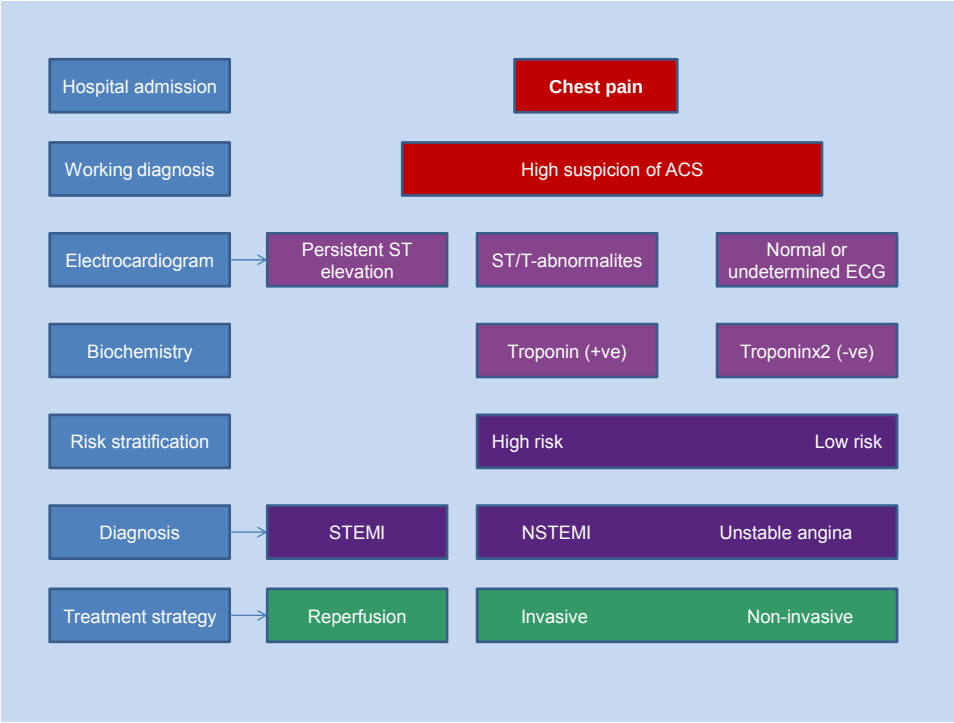

Figure 4. The spectrum of acute coronary syndrome (Fox et al. 2010).

Myocardial Infarction (MI)

In 2007 the European Society of Cardiology (ESC) published their 'Universal definition of myocardial infarction' in the European Heart Journal (Thygesen et al. 2007). Within this paper the ESC describe how an MI may be considered a minor or even undetected event for some people living with lifelong chronic cardiovascular disease while for others it presents as a catastrophic event.

Historically, MI has been determined by electrocardiographic changes, signs and symptoms as well as biochemical markers. As medicine has advanced, the clinician's ability to detect even minimal myocyte necrosis has developed due to the implementation of highly sensitive and specific biochemical markers and advanced imaging techniques. MI can now be clearly defined by area of damage, size, the circumstances resulting in cell necrosis e.g. spontaneous or procedure induced and the timing of the event i.e. acute, evolving, healing or healed.

Myocyte damage does not occur immediately following plaque rupture but evolves over a period of time from around 20 minutes to 2-4 hours or longer depending on the presence of collateral circulation, intermittent occlusion and cellular sensitivity to decreased perfusion. During this time the process may be considered 'evolving'

when presenting within the first 6 hours, 'acute' 6 hours to 7 days, 'healing' between 7 and 28 days and 'healed' at around 29 days. It takes around 5-6 weeks for myocardial damage to heal (Thygesen et al. 2007).

The area of damage can be detected most accurately by using imaging techniques. The use of these methods may also allow the amount of damage to be assessed with smaller infarcts being considered less than 10% of the left ventricle, moderate 10-30% and large >30% (Thygesen et al. 2007).

The clinical classification allows the clear differentiation of the different types of MI:

- Type 1 refers to spontaneous plaque rupture, erosion, fissuring or dissection
- Type 2 occurs secondary to increased oxygen demand or decreased oxygen supply, such as experienced in those with coronary artery spasm or hypo/hypertension
- Type 3 is related to sudden death, which occurs before clinical assessment can be made or following post mortem
- Type 4 is associated with PCI and/or stent thrombosis
- Type 5 refers to those events which occur with CABG

Diagnosis

The diagnosis and classification of ACS is dependent upon the clinical presentation of the patient. The assessment will include past medical history and presenting clinical signs and symptoms, electrocardiographic (ECG) changes and cardiac specific biochemical markers.

Obtaining a brief past medical history will help to determine if the pain experienced is likely to be cardiac in origin. Information such as a previous history of cardiac chest pain, the presence of cardiovascular risk factors (diabetes, dyslipidaemia, hypertension, family history, smoking), a history of ischaemic heart disease including current treatment and previous interventions can help to guide the clinical decision making process. Evaluation of the patient's blood glucose is also an important part

of early admission assessment, as elevated blood glucose has been identified as a strong independent risk factor (SIGN 2013).

One of the key issues in managing patients is the need to reduce the delay from symptom onset to first seeking medical assistance. Finnegan et al (2000) describe the reasons for this as being associated with people's perception of what they expected the symptoms of a heart attack to be. This included describing pain associated with a cardiac event as severe, sharp or crushing rather than the ambiguous sensations that many people experience. There was also a lack of appreciation of risk factors, with individuals underestimating the likelihood of experiencing a cardiac episode. Both of these misconceptions had led to delayed help seeking behaviour which is linked to increased myocardial damage and mortality (Newby 2010). The average delay of those with an ACS has been found to be between 2 and 6 hours, with those with non ST segment ACS (see below) delaying the longest (Goldberg et al. 2002).

Clinical presentation may be the same as any ischaemic episode. Symptoms may include retrosternal pain or discomfort which may be described by the patient as tightness, squeezing or heaviness. Symptoms may radiate to the arms, shoulders, back, neck, abdomen and jaw. Pain which persists for longer than 15 minutes or is unresponsive to nitrate administration is more likely to be associated with ACS (NICE 2010a). Additional symptoms frequently described include: nausea with or without emesis, diaphoresis, dyspnoea and a heightened sense of anxiety.

The immediate management of patients with ACS is strongly determined by ECG changes, in particular the presence or absence of ST segment elevation. Those presenting with > 1 mm ST elevation in at least two adjacent limb leads, > 2mm ST elevation in at least two contiguous pre-cordial leads or a newly diagnosed onset of left bundle branch block are likely to be diagnosed as ACS with clinical MI (STEMI) (SIGN 2013). ECG leads can then be utilised to assist in the identification of the offending artery by assessing the leads which correspond to the area of ischaemia and the normal vessel/s which perfuse the myocardium (Table 2).
ECG changes which are more indicative of non ST segment elevation myocardial infarction (NSTEMI) or unstable angina are ST segment depression, T wave changes such as inversion and the presence of pathological Q waves (Q waves which are >25% of the R wave and/or >0.04 seconds, >2mm deep,) (Hampton 2008).

Table 1. ECG leads, corresponding arteries and myocardium supplied (Shoulders-Odom 2008)

Leads	Myocardium	Artery
II, III, AVF	Inferior wall	Right coronary artery
I, AVL, V_5, V_6	Lateral wall	Left circumflex
V_1, V_2	Septal wall	Left anterior descending (proximal)
V_2, V_3, V_4	Anterior wall	Left anterior descending
V_2R, V_3R, V_4R	Right ventricle	Right coronary (proximal)
V_7, V_8, V_9	Posterior wall	Posterior descending

One of the main diagnostic tools used to guide ACS classification, is the use of serum concentration of cardiac specific biochemical markers, such as troponin I or T. Troponin is a regulatory protein involved in muscle contraction in skeletal and cardiac muscle cells. When cardiac muscle is damaged, troponin leaks out of cells into the circulation. Troponin T (cTnT) and I (cTnI) are cardiac specific and are extremely sensitive to myocardial injury and damage. The detection of troponin I or T is therefore a strong indication that myocardial damage has occurred. It should be noted however, that additional causes can result in a raised troponin, so further investigations and clinical presentation are still essential to diagnosis. The reasons for this are not always clear but troponin release in heart failure could be explained by increased wall strain (Januzzi Jr et al 2012) and myocarditis due to inflammation. Troponin levels can be detected within 4 to 6 hours of initial damage and stay elevated for around a week (4-7 days troponin I, 10-14 days troponin T).

The use of troponin as a method of detecting myocardial damage is far superior to previous biochemical markers such as creatine and kinase, as they are non-cardiac specific and may be detected due to other myocyte damage e.g. after a muscular sprain or strain. They also help detect the extent of damage so high levels indicate greater damage and lower amounts less damage.

Even a modest increase in troponin however is associated with a substantial increase in risk of mortality (Table 3).

Table 2. Current definitions and prognosis of acute coronary syndrome according to troponin T concentration (SIGN 2013)

	12 hr serumTroponinTconcentration(ng/ml)		
	<0.01	≥0.01and<1.0	≥1.0
BCS definition	ACS with unstable angina	ACS with myocyte necrosis	ACS with clinical myocardial infarction
ESC/ACC definition	unstable angina	myocardial infarction	myocardial infarction
WHO definition	unstable angina	unstable angina	myocardial infarction
30-daymortality (Casey, Faxon 2004)	4.5%	10.4%	12.9%
6-monthmortality (Casey, Faxon 2004)	8.6%	18.7%	19.2%

The more sensitive Troponin I has been used in many health boards, but following recent advances even this is now being superceded by a high-sensitivity cardiac Troponin I (cTnI) assay in many areas (Shah et al 2013). There are obvious benefits in being able to rule out MI earlier, with better patient assessment having implications for reducing unnecessary admissions. NICE (2010a) guidelines currently advise that Troponin should be checked on admission and 10-12 hours after the onset of symptoms to determine peak Troponin rise. Reflecting the increased sensitivity of the new assay, it has been recommended that Troponin can be determined on admission and at 3-6 hours after admission irrespective of symptom onset (Thygesen 2012), however, if symptoms are suggestive of ACS further testing will be required.

A recent study by Shah et al (2015) revealed that standard Troponin assays with a single (higher) diagnostic threshold (i.e. not sex specific) under diagnosed MI in women, resulting in sex inequalities in ongoing referral, invasive treatments, and ultimately clinical outcomes. It would also result in them missing out on cardiac rehabilitation. The improved precision of the high sensitive assay means that the 99th percentile reference point is defined at a lower level than previously. Having a more sensitive assay, and setting a lower diagnostic threshold for women, will result in the lower Troponin concentrations due to reduced left ventricular mass, being detected (Shah et al 2015). Diagnostic thresholds for men are now >34 ng/l and >16 ng/l for women.

Acute Coronary Syndrome Management

The way in which patients are managed is currently guided by various factors: the time of symptom onset, delay in seeking medical assessment, clinical presentation, local service provision and the prediction of clinical outcomes.

Adverse clinical outcome is associated with numerous physiological factors. These may include:

- the individual's age
- the presence and severity of ECG changes
- the detection and level of biochemical markers
- cardiogenic shock
- elevated heart rate
- arrhythmias in particular ventricular changes
- atrial fibrillation or cardiac arrest
- diabetes and diabetic control
- renal impairment
- evidence of previous cardiovascular related event or disease

(NICE 2010c)

Used independently, these clinical features may not offer an accurate estimation of the individual's absolute risk. Guidelines therefore advocate the use of validated risk scores such as PREDICT, GRACE, PURSUIT or TIMI risk scoring systems (NICE 2010a; SIGN 2007c). These scoring systems combine clinical factors in order to accurately distinguish between high and low risk patients while estimating the actual risk of short or long term adverse events. The scores may predict outcomes such as survival, re-infarction, need for revascularisation and other serious complications (NICE 2013). When used in practice, these scores may assist clinical decision making by balancing the benefits of treatment with the possibility of adverse events such as bleeding or stroke.

Non-ST Elevation Acute Coronary Syndromes - In the absence of ST segment elevation but troponin positive as detected within hospital dictated clinical levels, a diagnosis of NSTEMI is likely to be given, while those who display cardiac symptoms

but are troponin negative are given the diagnosis 'unstable angina'. In these circumstances, patients are unlikely to receive emergency reperfusion unless clinical signs or symptoms persist or recur. Patients with non STEMI syndromes are therefore treated pharmacologically to manage symptoms, increase cardiac perfusion, support myocardial recovery and prevent the occurrence of further events. This will include the administration of analgesia (morphine), anti-emetics (metoclopramide), anti-anginal therapy (nitrates), anti-platelet (aspirin, clopidogrel) and anti-thrombin therapies, depending on treatment options as well as current and future risk of bleeding. Additional oxygen may also be required.

Further interventions should be guided by clinical risk scoring and should include additional pharmacological therapy (beta blockade, ACE inhibitor and statin therapy), PCI or CABG (SIGN 2013). Pre-discharge or follow up assessment may include echocardiogram to assess left ventricular function and stress testing.

In the past, the perception of NSTEMI has been considered benign when compared to STEMI. This is however inaccurate as the 2 month prognosis of those post-NSTEMI is worse than those experiencing STEMI (Knight and Timmis 2011).

Prior to discharge, all patients should be offered advice and information about their diagnosis and clinical follow up. Cardiac rehabilitation, including lifestyle change and secondary prevention strategies, should be discussed including ongoing medical therapy. Patient specific support should then be arranged and referrals made as appropriate.

ST Elevation Acute Coronary Syndromes – The management of those presenting with STEMI is highly dependent on the first 12 hours from symptom detection, with myocardial necrosis beginning 15-30 minutes after vessel occlusion (Van de Werf et al. 2008).

It is often during this early phase where life threatening arrhythmias require rapid intervention and valuable myocardium can be preserved through the appropriate utilisation of reperfusion therapies. Reperfusion may be achieved either pharmacologically via the administration of thrombolyic agents or mechanically via primary percutaneous coronary intervention (PPCI).

Thrombolytic therapy

The use of streptokinase (SK) and its role in the management of those with acute MI became fully recognised following the publication of the GISSI trial in 1986 (Gruppo Italiano per lo Studio della Streptochinasi Nell'Infarto Miacardico (GISSI) 1986). This study not only recognised streptokinase as an effective fibrinolytic agent but set out a clear protocol for its use in managing those with acute MI. Streptokinase is a derivate of streptococci bacteria. Because it is a bacterial product, the body has the ability to develop immunity towards it. The repeated use of streptokinase is therefore discouraged as its effectiveness is likely to be limited and the possibility of an allergic reaction increased (Now rarely used).

Current thrombolytic agents include tissue plasminogen activator (tPA). This protein is involved in the breakdown of clots and is found in endothelial cells. Tissue plasminogen activator is an enzyme which can be manufactured (recombinant tissue plasminogen activator - rtPA) and its use can therefore be repeated if necessary. Types of rtPA include alteplase, reteplase and tenectaplase.

As per the use of primary PCI, the effective use of thrombolytic therapies is highly dependent on their early administration with pre-hospital administration advocated unless the provision of PPCI services is available within 90 minutes (Van de Werf et al. 2008) PPCI has been shown to reduce short and long term mortality and major cardiovascular events when compared to thrombolytic therapies (Keeley et al. 2003). However, when there is a prolonged delay in accessing cardiac catheterisation services or when these services are unavailable, the use of pre-hospital thrombolysis is advocated as it reduces all-cause mortality when compared to in hospital administration (Morrison et al. 2000).

The use of thrombolysis must be assessed in line with the possible risks associated with bleeding. Contraindications for its use include: recent surgery, trauma, stroke, aortic dissection etc. It is suggested that around 40-50% of people will be considered unsuitable for this type of treatment, with the largest majority being associated with delayed presentation (>12 hours) (SIGN 2013). The primary objective must therefore be to educate the public in relation to the importance of seeking medical care as soon as possible when a cardiac event is suspected.

Thrombolytic therapy is deemed successful in those who have relief of symptoms with a reduction in ST elevation of >50% within 60-90 minutes post administration (Van de Werf et al. 2008).

Primary Percutaneous Coronary Intervention

PPCI is when an angioplasty is performed during an acute MI. It may or may not include stenting of the culprit lesion and thrombus aspiration. PPCI has been found to be the preferred treatment option for those with STEMI when a highly skilled interventional team can be made available 24 hours a day and 7 days per week (Van de Werf et al. 2008).

In order to achieve optimum myocardial protection, PPCI should be carried out within 2 hours from first medical contact (NICE 2013; SIGN 2013), with some areas working within a time parameter of 90 minutes (Scottish Health Technologies Group 2009). When compared to thrombolytic therapies, PPCI has been noted to be more effective at restoring vessel patency with less incidents of re-occlusion occurrence. This, in turn, has led to improved left ventricular function and improved clinical outcomes (Keeley et al. 2003). The underlying pathology of thrombus formation in those with ACS has also seen the use of thrombus aspiration. The use of this technique has been shown to reduce microvascular obstruction and limits infarction size (Knight and Timmis 2011)

Percutaneous coronary intervention (PCI) may not only benefit those who present early with STEMI but may be advocated in patients who may continue to have limited perfusion after fibrinolysis administration. This is referred to as rescue PCI. Failure to reperfuse may be detected by less than 50% resolution of ST segment elevation 60 – 90 minutes after the administration of the fibrinolysis or when ischaemic symptoms persist even 12 hours after initial symptom detection (Van de Werf et al. 2008).

Concomitant therapies are similar to those with NSTEMI and may include analgesia (morphine), anti-emetics (metoclopramide), oxygen, anti-anginal therapy (nitrates), anti-platelet and anti-thrombin therapies, with the early administration of a beta blocker also being initiated as appropriate.

Anti-platelet strategies include loading doses of both aspirin and clopidogrel, which are followed by maintenance prescribing. There may be regional differences in

duration of therapy. Other anti-platelet therapies include prasugrel and ticagrelor. The main purpose of this is to enhance thrombus resolution while also preventing future thrombotic events. For those who undergo PPCI, the use of anti-platelet therapy is also directed at the prevention of in-stent thrombus formation during the period of vessel endothelialisation. For those who have a drug eluting stent implanted, endothelialisation takes around 9 -12 months compared to bare metal stents which take 1-3 months (Knight and Timmis 2011)

Additional anti-platelet therapy includes the use of intravenous GPIIb/IIIa receptor blockers such as Abciximab prior to PCI. This not only acts as an anti-platelet therapy but may decrease the inflammatory response especially when administered directly within the vessel (Knight and Timmis 2011)

The use of antithrombotic therapy has previously focused primarily on the use of unfractionated heparin or low molecular weight heparin (Enoxaparin) to enhance the thrombus resolution. This is however changing, as Fondaparinux and Bivalirudin offer alternative management strategies in those undergoing urgent PCI (Van de Werf et al. 2008).

It is essential that the facilitators are aware of the management of the patients within their own area as treatments may vary widely. The time between the onset of symptoms and initiation of treatment is a strong determinant of patient treatment, management and outcome. Only those diagnosed with ACS who are clinically stable, should be given the Heart Manual. Those with ACS with unstable angina are currently excluded.

Revascularisation

Revascularisation is a procedure aimed at restoring, adding or augmenting blood flow to an area of the heart with reduced perfusion. In cardiovascular terms it is associated primarily with two types of procedures, PCI developed in 1977 by Andreas Gruentzig and CABG used in clinical practice since the 1960's (Kolh et al 2010).

Percutaneous Coronary Intervention (PCI)

PCI may include a variety of procedures including angioplasty, stent implantation, intra-coronary radiation (brachytherapy) and thromboectomy. PCI is performed by passing a vascular sheath followed by a guide-wire and a cardiac catheter into the coronary artery tree. The catheter is inserted via one of the peripheral arteries and is fed into the coronary arteries via radiological guidance. Traditionally PCI's were carried out via the femoral artery. This has however become less popular over recent years as the use of the radial artery has been shown to result in fewer complications (Ludman 2009). The catheter is positioned next to the lesion and the balloon tip inflated for approximately 30-90 seconds at high pressure (Figure 5). Angioplasty dilates the lumen of the vessel, displacing the offending plaque and overstretching the vessel. A residual stenosis of around 20% is considered an optimal result (Shoulders-Odom 2008). The balloon may need to be inflated and deflated several times to obtain a good result.

The use of intra-coronary stents has seen a reduction in the need for bypass surgery (Casey and Faxon 2004) (Figure 5). There are currently two main forms of stent used today within the UK; bare metal stents and drug eluting stents. These stents are made of metallic alloys (e.g. stainless steel) and when inserted they are expanded to fit the artery, acting as a form of scaffolding to support the vessel as it heals. Bare metal stents were the first type of stents to be used in clinical practice. Their use to increase patency revolutionised the care of those requiring revascularisation. In some cases however, their use became hampered with the development of in-stent restenosis.

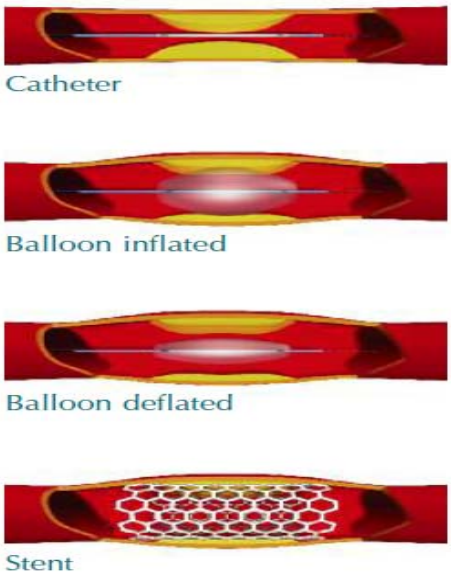

Figure 5. Angioplasty and stent.

Restenosis or recurrence of vessel narrowing is a result of the physiological response to the damage caused to the vessel wall by the mechanical implantation process. Damage occurs in two stages; first the immediate injury causes the development of thrombus and second, an inflammatory immune response takes place resulting in the proliferation of cells in the intima known as neointimal hyperplasia (Shah 2003) All procedures tend to produce some injury with a loss of lumen diameter which is balanced by a general increase in vessel patency (Shoulders-Odom 2008). The response to stent implantation has seen the development of various preventative measures to decrease these normal physiological reactions. This includes the increased use of anti-platelet therapies and the development of drug eluting stents. Restenosis tends to occur within the first 3-12 months, while stent thrombosis occurs more frequently within the acute (first 24 hours) or sub-acute phases (first 30 days) of recovery, although late (3 years) thrombosis has been reported particularly in those receiving drug eluting stents (Shoulders-Odom 2008).

The use of multi anti-platelet therapies is advocated as each treatment has a different mechanism of action within the clotting cascade. Typical anti-platelet regimes may include a glycoprotein IIb-IIIa receptor blocker (e.g. Abciximab), thienopyridine (e.g. clopidogrel) and salicylate (e.g. aspirin). Although various other therapies may be used particularly as research advocates the use of new preventative treatments (Shoulders-Odom 2008). The recommended duration of therapies also varies

depending on the type of stent implanted, medical history and risk of haemorrhage. The main purpose of these treatments is to enhance thrombus resolution and prevent further thrombotic events within the 1-3 months post bare metal stent implantation and 9-12 months following the deployment of drug eluting stents (Knight and Timmis 2011). Concordance with preventative treatment regimens must therefore be strongly advocated while highlighting the importance of monitoring for signs of bleeding.

The primary aim of drug eluting stents is to slow down the re-endothelialisation process within the arterial lumen and therefore the development of neointimal hyperplasia. This is achieved through the use of two main forms of anti-inflammatory therapies; sirolimus, an immunosuppressant and paclitaxel, a form of chemotherapy (Shoulders-Odom 2008). These drugs are coated on the stent and are delivered locally within the vessel wall. Drug eluting stents have now been shown to significantly reduce the risk of restenosis (Stettler and Wandel et al. 2007).

The use of stents has dramatically changed the way in which people with CAD are managed, with more than 90% of PCI procedures involving the implantation of one or more stents (SIGN 2007b). Newer approaches to PCI continue to be developed in order to push forward the frontiers of cardiovascular care. Current research includes the use of biological materials and **biodegradable stents** along with the adjustment and tailoring of pharmacological therapies (Fox et al. 2010).

PCI post procedure management

Following the procedure, the primary objectives are to monitor for rhythm disturbance, chest pain, control bleeding and monitor the healing of the insertion site. Various methods have been used to aid haemostasis post procedure including manual or mechanical compression and vascular closure devices. These techniques aim to stop haemorrhage while preventing the risk of tissue damage due to reduced perfusion and nerve compression. Complications associated with the sheath removal include haematoma, bleeding, arteriovenous fistula and pseudoaneurysm (Sulzbach-Hoke et al. 2010)Additional care must be taken in those at highest risk, including people with diabetes, hypertension and peripheral vascular disease (Shoulders-Odom 2008).

As the average length of stay post PCI decreases, the need for effective recovery and rehabilitation support increases. The current practice for those undergoing

elective PCI is a day case procedure or overnight stay with the majority (80%) of non-complex patients able to be discharged within this time limit (Heyde et al. 2007). As this becomes wider practice, it is essential that clinicians accurately estimate clinical risk of those with potentially more complex needs. In the future some areas may estimate their patients' risk of complex recovery using four main categories: heart failure, age (>70 years), gender (older women) and those presenting with renal failure. In these patients, the length of stay was noted to be prolonged by about 1 to 6 days (Negassa and Monrad 2011).

As the patient's stay within the clinical setting is frequently short and focuses primarily on rapid clinical recovery, it is important that the patient and their family are made aware of the need for on-going self-monitoring during the recovery phase.
For elective PCI patients (and CABG patients), the Revascularisation Heart Manual is recommended with facilitator support. In particular, emotional support is considered to be important by these patients (Dawkes 2014).

In addition, information provided must include issues such as assessment of the insertion site, activity limitations and possible complications.
Most people will notice a small firm lump around the insertion site with some degree of ecchymosis (bruising). Patients should be reassured that the lump felt is associated with localised tissue injury and haematoma. Expansion of this with any associated increase in localised tenderness, discharge, heat or erythema must be reported as soon as possible. Particular care must be taken in monitoring for signs of more complex vascular complications such as pseudoaneurysm, retroperitoneal haematoma, arteriovenous fistula and atheroembolism (Shoulders-Odom 2008). As the swelling decreases and the haematoma disperses into the superficial tissue, the bruising will expand and change colour frequently ranging from purple/blue to green/yellow as time passes, with the full resolution of haematoma and associated bruising often taking several weeks to settle (Shoulders-Odom 2008).

It is important to reduce activity, particular flexion of the area for the first 48 hours post procedure. After this time, the return to walking and driving can be allowed with the adoption of a paced approached being encouraged. Strenuous activity should however be discouraged for a least a week following the intervention (Shoulders-Odom 2008). Any changes associated with activity should be assessed, in particular swelling and/or pulsation of the insertion site, change in sensation or pallor of the

area or limb. If femoral nerve compression has occurred during the procedure, leg weakness may last over several weeks or even months (Shoulders-Odom 2008).

Additional advice may be required by those who have had implantation of a vascular closure device such as a collagen plug (VasoSeal) or absorbable anchor (Angio-Seal) (Figure 6). Collagen plugs involve the delivery of collagen onto the surface of the femoral artery puncture site. The collagen attracts platelets sealing the area.
Full absorption takes place within 6 weeks. Absorbable anchors use a degradable suture material with a collagen plug to sandwich the vessel between the suture and the plug. These systems take around 90 days to absorb (Shoulders-Odom 2008).

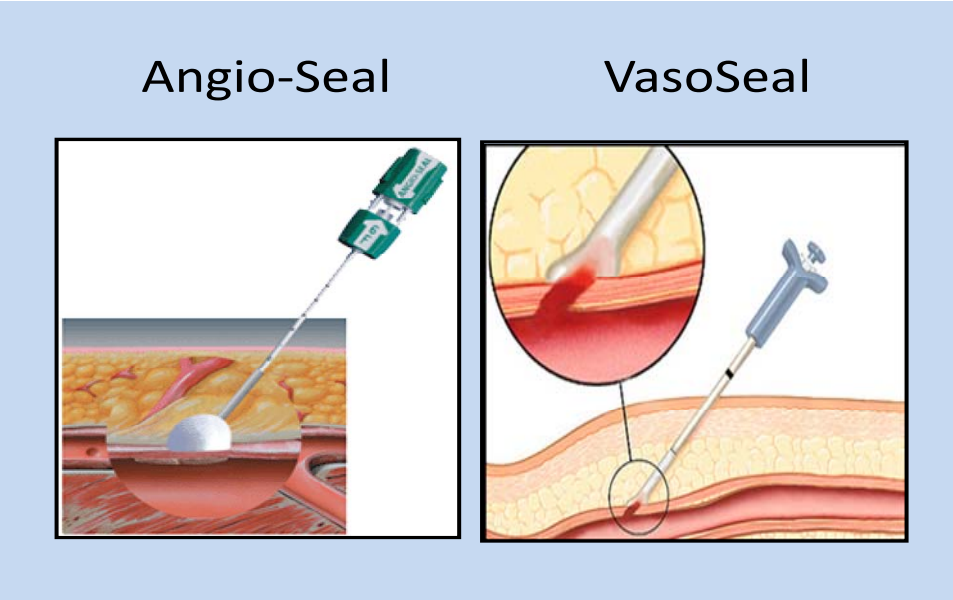

Figure 6. Vascular closure devices (Ludman 2003).

Post-procedure chest pain also occurs frequently (36%) and may be associated with procedural events (Kini et al. 2003). Various mechanisms may lead to symptoms. Abbate et al (2007) divide these into structural or functional causes. Structural causes include restenosis, disease progression and incomplete revascularisation, while functional causes may be associated with coronary microvascular dysfunction, epicardial coronary spasm and vasoconstriction at the stent edge. Pain occurring within the first month was most frequently associated with incomplete revascularisation. Those developing chest pain within 6 months had an increased incidence of restenosis. However, delayed endothelialisation caused by the use of

drug eluting stents may mean that restenosis may take place at a much later date (Abbate et al. 2007).

The main objective when carrying out a clinical assessment must be the differentiation between cardiac and non-cardiac pain, with anyone describing typical angina pain similar to that experienced prior to their intervention being at greatest risk (Abbate et al. 2007). Other pain described includes sharp localised pain which may be associated with what is known as 'stretch-pain'. This post- procedural pain is common, associated with a positive outcome and tends to occur within the acute phase of recovery (Jeremias et al. 1998). Patients can be reassured and can take simple analgesia in the first instance after seeking advice.

Coronary Artery Bypass Graft (CABG)

Coronary artery bypass grafting often referred to as CABG or 'cabbage' has now become a routine surgical procedure throughout the UK, with 22,846 operations being carried out in 2008 (Scarborough et al. 2010). The trends in revascularisation procedures have however changed dramatically over the past decade, from PCI's equalling the number of CABG's carried out in the UK in 1998 (25,083 CABG v 24,889 PCI) to the number of PCI's increasing to more than three times the number of CABG operations performed in 2008 (22, 846 CABG v 80,331 PCI) (Scarborough et al. 2010).

Although PCI has seen a significant reduction in bypass grafts being performed, there remains clear evidence for its usefulness and efficacy in some patients, especially those with left main stem, proximal left anterior descending or three vessel disease and for those who have either complex lesions or when PCI has resulted in incomplete revascularisation (Eagle et al. 2004). Those with diabetes are also recognised as having better long term outcomes when treated with CABG compared to PCI (Kolh et al. 2010; NICE 2011).

The risk of mortality following CABG has fallen by around 20% over the last decade in those under the age of 70 (The Royal College of Surgeons of England 2009). Those previously deemed at high risk and excluded from having this intervention, such as people over the age of 75 as well as those with co-morbidities such as diabetes, hypertension and obesity, are now routinely undergoing the procedure.

CABG is a successful treatment for angina in approximately 90% of cases for at least 5-10 years (SIGN 2007b). This may however be increased or reduced depending on other risk factors and the vessels used.

The use of conduits (grafts) to bypass coronary atherosclerotic lesions began in the 1960's. The aim of the procedure was to provide people with CAD with symptomatic relief, increased life expectancy and improved quality of life (Bilal 2011). At this time, the procedure was conducted 'off-pump' until technological advances saw the introduction of perfusion systems allowing the heart to be stopped while continuing to circulate the patient. Since this time, various techniques have been developed in order to reduce patient risk, improve clinical outcomes and promote early recovery. Techniques include off pump surgery where the surgeon works on the beating heart, minimal invasive approaches requiring smaller incisions via thoracotomy and endoscopic techniques. Different approaches require different surgical incisions. A midline sternotomy remains the usual incisional approach although an anterior thoracotomy for bypass of the LAD (left anterior descending) or lateral thoracotomy for marginal vessels may be used when performing off pump procedures (Bilal 2011).

The aim of CABG is to revascularise the myocardium which is poorly perfused by extensively stenosed coronary arteries. To achieve this, the surgeon must obtain a patent and durable conduit. Conduits may be harvested from various sites such as the saphenous vein, radial artery, left and right internal mammary arteries (LIMA or RIMA) also known as the internal thoracic arteries, right gastroepiploic artery, inferior epigastric artery and the splenic artery.

The long saphenous vein lies anterior to the medial malleolus and ascends posteriorly up the tibial border before emptying into the femoral vein. It receives numerous tributaries and contains between 10 and 20 valves. The saphenous vein is frequently used if vascular pathologies of the leg can be excluded such as varicosities, venous insufficiency, or a previous history of deep venous thrombosis. These conduits have an 80-90% patency which decreases over time to around 50% at 10 years (Bilal 2011). Reduced patency within the first year is likely to be associated with technical errors, thrombosis and intimal hyperplasia while graft occlusion occurring later than a year is more likely to be associated primarily with atherosclerosis (Bilal 2011).

The saphenous vein can be obtained via 'open' harvest or via endoscopic retrieval. The use of endoscopic techniques has however been questioned as evidence concerning reduced long term vessel patency has come to light (NICE 2010b). The vein is removed, inspected and then attached to the ascending aorta and below the area of obstruction to 'bypass' the narrowing or occlusion (Figure 7).

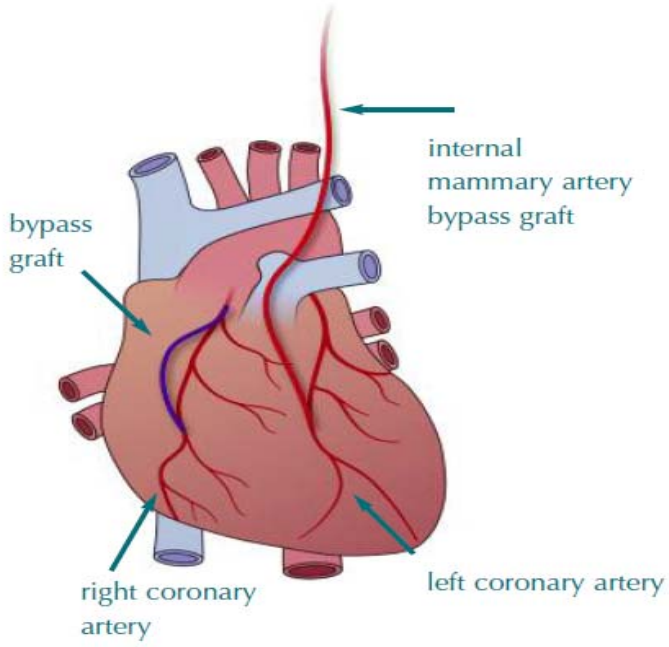

Figure 7. Saphenous vein use in CABG procedure.

Venous donor sites are prone to oedema and localised complications due to reduced venous drainage, as well as lymphatic and soft tissue damage (Jakeman 2010). The accumulation of fluid within the interstitial space can result in reduced tissue perfusion and impaired wound healing. Key strategies to reduce this post-operative complication include the use of compression hosiery, limb elevation, appropriate skin care, nutritional advice, limb exercises and mobilisation (Jakeman 2010).

The internal mammary arteries arise from the subclavian arteries and run down the inside of the chest wall. These arteries are not needed by men and older women and can be directly attached to a coronary artery particularly when the LIMA is used. The preferred artery for bypass with the LIMA is the LAD, as it lies at the front of the heart, close to the sternum and feeds the largest proportion of the left ventricle. The LIMA is a short vessel, making it difficult to reach other arteries. The LIMA has good vessel patency of 98% at year 1 and 90% at 10 years (Bilal 2011).

Right internal mammary artery conduits are also becoming part of routine procedure by some surgeons, although the use of both internal mammary arteries may not be a suitable option for some patients, such as those with diabetes, those with clinical management associated with decreased wound healing or other conditions (Dietl et al. 1995; Taggart 2002). The use of the RIMA is however complex, with questions remaining around which coronary artery has the best long term patency when using this conduit, while radial artery conduits also carry mixed opinion especially as the use of this vessel has been associated with an increased risk of vasospasm (Canver and Yousafzai 2008). The identification of suitable donor vessels and selection of preferred graft sites remains part of a complex decision process with consideration being given to the approach required, the patient's age, vessel availability, surgical vulnerability and any additional co-morbidities which may exist.

One of the main clinical concerns following a procedure requiring sternotomy is the development of a deep chest wound infection affecting the sternum or mediastinal tissues. It has been suggested that obesity, smoking, advanced age, the use of internal mammary conduits, diabetes and repeated chest incision place the patient at greatest risk (Matros et al. 2010). The risk of developing a deep wound infection is between 0.55% and 3 % with mortality rates associated with mediastinitis being between 3% and 35% (Loop et al. 1990). Symptoms of deep chest wound infection include chest wall tenderness, pyrexia, fatigue, shortness of breath, wound exudate and an unstable chest wall.

The most common microbial infections found in Matros et al's study (2010) were associated with staphylococcus epidermidis, methicillin-sensitive staphyloccusaureus and methicillin-resistant staphylococcus aureus. Although staphylococcus epidemidis and aureus are common skin flora, they are associated with over half of mediastinitis infections (Fynn-Thompson and Vander Salm 2004). Similarly, although staphylococcus epidermidis may be considered relatively benign, it has been identified as the most common sternal pathogen, often with minimal signs of systemic infection. It can have a slow onset not being detected until 3 weeks post intervention and is associated with the presence of sternal instability. It is suggested that this initially occurs via a minor cutaneous or subcutaneous infection and spreads into the mediastinal space when the sternum dehiscence disrupts the mechanical barrier (Fynn-Thompson and Vander Salm 2004).

In order to reduce the risks associated with the development of chest wound infections, various modifiable risk reduction strategies may be put in place. These include antibiotic prophylaxis, hyperglycaemic control, preoperative preparation, intra-operative management and specific sternal closure techniques (Fynn-Thompson and Vander Salm 2004).

Surgery takes approximately 4 hours depending on the patient's condition and carries a 2-3% risk of mortality (SIGN 2007b). Cerebrovascular events are a major complication of CABG surgery affecting 1-3% of patients (Bilal 2011). The main cause of this is hypoperfusion and embolic events. Ensuring adequate arterial pressure is therefore of primary concern post intervention in order to maintain cerebral perfusion and therefore protect cerebral function. Neurological complications range from major neurological deficit and coma to deterioration in intellectual function or memory impairment. In those with less severe intellectual impairment, normal activity normally is regained within 3-12 months (Bilal 2011) (see Cognitive Function chapter for more details).

The effect that CABG has on cerebral microvasculature may also be investigated through the development of visual disturbance. This is due to retinal circulation being part of the cerebral circulation. People who have undergone CABG may complain of transient loss of vision, poor reading, loss of colour perception and reduced short and long distance visual acuity (Ascione et al. 2005). In Ascione et al's study (2005) it is suggested that although reduced perfusion may result in perioperative ischaemia, visual disturbance is most likely to be associated with embolisation secondary to aortic manipulation during on pump surgery. Patients are therefore advised to avoid getting a new prescription for glasses within the first 2-3 months post intervention. Sudden visual disturbance should however be dealt with as a matter of urgency. Additional sensory changes noted may include a change in taste and smell.

Further complications following CABG include: myocardial dysfunction including arrhythmia which affects around 30% of patients, acute renal failure, cardiac tamponade, aortic dissection and respiratory tract or wound infections (Bilal 2011).

CABG post procedure management

CABG is a highly invasive procedure requiring intubation, sternotomy or thoracotomy in the majority of cases, vessel harvesting as well as invasive monitoring and therapeutic support. In general, most people will be discharged within the first week after their operation. During the early discharge period, core patient self-management information should include:

- breathing techniques
- medication guidance
- fatigue management and sleep hygiene
- activity modification
- resuming physical activity including sex
- wound care
- appetite and nutritional advice
- pain management
- psychological adjustment
- physiological signs and symptoms monitoring
- risk factor prevention

(Frantz et al. 2001; Thoebold and MacMurray 2004; Gao et al. 2009; Lie et al 2010).

For many, the early discharge period can be a much greater challenge than expected. This can lead to increased symptom awareness, resulting in a sense of loss of control, a greater need for social support and increased anxiety (Tolmie et al. 2006; Lie et al. 2010). However, Lie et al (2010) and Theobald and McMurray (2004) report a lack of support and adequate patient information during this crucial stage of recovery. Theobald and McMurray (2004) go on to highlight the need to assess both the needs of the patient and their family caregivers, while reviewing current models of service provision to ensure support is being provided during the early recovery phase within the home setting.

In the qualitative study conducted by Theobald and McMurray (2004) half of the patients experienced complications which they did not expect. These included urinary difficulties, constipation, pneumonia and pleural effusions as well as infected wound sites. Further problems described were fatigue, disturbed sleep, weight loss and a

change in taste and appetite. Change in physical appearance also affected some, with people expressing distress in relation to incision sites and changes in perceived body image. The need for additional support to carry out tasks previously considered simple and routine also required behavioural adjustment, with family members being relied upon to carry out additional household tasks. For some this resulted in marital discord.

It is essential to monitor for signs of arrhythmias, congestive heart failure, bleeding and infection as well as signs of ischaemic chest pain, although the risk of these complications appear to be greatest during the early post-operative period (Lutchmedial and Smilovitch, 2012). From a patient point of view, often the focus is on returning to functional normality. This may include the ability to self-care with personal hygiene, carry out routine tasks such as house work and gardening, driving, returning to physical activity including sex and vocational issues. In this respect, physiological symptoms such as pain, altered taste or loss of appetite, visual disturbance, fatigue and sleep disturbance play an important part in the return to normal function.

Pain is a normal response to trauma. The way in which people detect and manage pain varies from person to person. How people manage their condition following discharge can be ineffective and compliance with therapeutic guidance diverse (Jin et al. 2008). Therapeutic compliance does not only refer to medication but other behaviours which may assist the individual to relieve symptoms and prevent or slow the progress of a condition or disease. (Jin et al. 2008). In relation to poor adherence to medication, the reasons are often multi-factorial but may include a lack of understanding of the disease or therapy, the treatment goals, lack of belief in the value of the medication, fear of adverse effects, cost and forgetfulness (Khanderia et al. 2008). In relation to the recovery of people post cardiac surgery, it is important to utilise a concordant approach, sharing understanding and expectations in order to achieve effective symptom control (Jones et al. 2003). This is not only important for improved pain control but other therapeutic interventions which may enhance recovery.

Hannan et al (2011) analysed the cause of 30 day readmission in 30,953 patients within the state of New York, America following CABG. The study calculated a readmission rate of 16.5%, with rates ranging between 8.3% and 21.1% within hospitals.

The most common cause of readmission was post operative infection (16.7%) followed by the development of heart failure (12.8%). Other complications included cardiac dysrhythmia (6.3%) and chest pain/angina (4.7%). Significant risk factors associated with readmission were identified. These included: increasing age, female gender, elevated BMI and experiencing multiple co-morbidities. Being of African-American race was also identified as an independent risk factor. This may however differ in the UK where ethnic diversities differ. Lessons may none the less be learned and actions taken to identify and support those at greatest risk of readmission.

Arrhythmias

An arrhythmia is classified by the heart rate, rhythm, site (supra-ventricular or ventricular) and the complex size (narrow or broad). Tachy-arrhythmias occur when the heart rate becomes higher than normal i.e. more than100 beats per minute. Brady-arrhythmias occur when the heart rate is much slower than normal, less than 60 beats per minute. The heart may have a normal or abnormal rate with or without an irregular frequency (Kirby 2006).

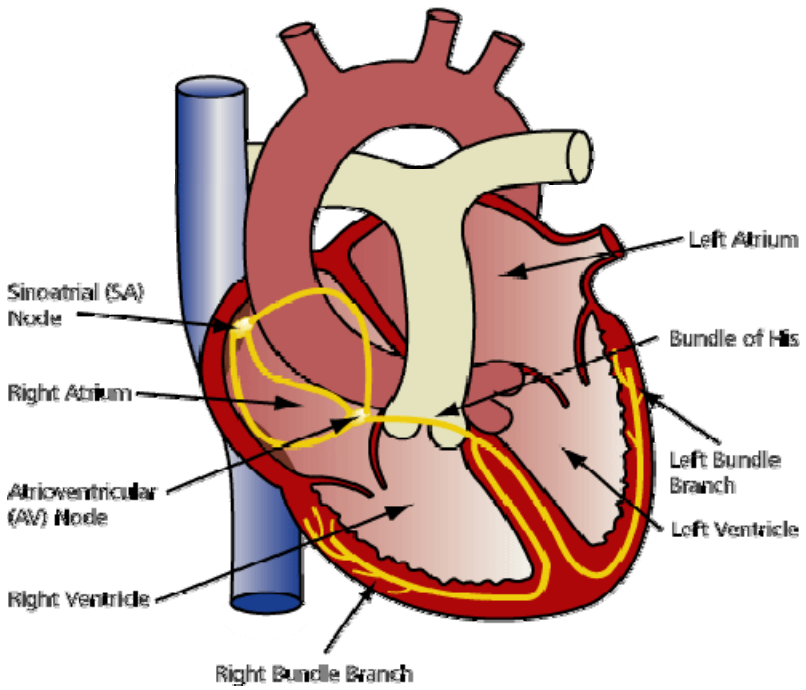

Figure 8. Electrical system of the heart.

An atrial arrhythmia originates in the atrium and is generally less life threatening. These include atrial tachycardia, atrial flutter and atrial fibrillation (AF). Ventricular arrhythmias originate in the ventricles which are the main pumping chambers, often leading to life threatening events or sudden death (Kirby 2006; SIGN 2007a). Sudden death is usually caused by ventricular fibrillation in which all forward pumping activity of the heart ceases within a few seconds of the onset of the arrhythmia. Brady-arrhythmias include various types of heart block. These frequently cause dizziness or syncope (fainting) (Kirby 2006).

Around 7-10% of patients treated with thrombolytic therapy due to an acute MI will develop new AF. The majority of the individuals (70-100%) will return to sinus rhythm by the time of discharge regardless of the treatment strategy (Wong et al. 2002). In acute MI, AF occurs more frequently in those who are elderly, have greater haemodynamic disturbance or have left ventricular impairment. Recurrence of AF is around 20% (Asanin et al. 2006). Sinus Bradycardia occurs in around 40-70% of patients following an MI with some patients requiring permanent pacing due to various types of heart block (Heidbüchel et al. 1994).

There is currently no clear evidence for the prophylactic use of anti-arrhythmics for patients with ACS or acute MI, although medications used for the benefits of reduced mortality including beta-blockers and ACE inhibitors may reduce the incidence in patients following acute MI (Kirby 2006).

AF is a common complication of CAD and occurs in 30-50% of post cardiac surgery patients, often prolonging stay in ICU (Alex et al 2003). The increased risk of haemodynamic instability and thromboembolism/stroke necessitates early recognition and treatment (Alex et al 2003). The prevalence of AF increases with age, affecting around 6% of the over 80 year old population. Factors which increase the predisposition to AF include hypertension, left ventricular hypertrophy or dysfunction and chronic heart failure (CHF) (Kirby 2006). Rate control is the recommended management strategy for those with well tolerated AF, often requiring a combination of medications. These may include beta-blockers, rate-limiting calcium channel blockers or anti-arrhythmics.
Some patients may require cardioversion, ablation therapy or pacing if they remain severely symptomatic or have LV dysfunction with poor rate control or intolerance to drug therapy (SIGN 2007b).

Sustained ventricular arrhythmias, ventricular tachycardia and/or ventricular fibrillation, occur in up to 20% of patients with ACS, with the greatest risk being within the first few hours (SIGN 2007b). These patients are managed with automated external defibrillation and/or anti-arrhythmic drug therapy such as intravenous Amiodarone. Non-invasive assessments of the risk of ventricular arrhythmias are not routinely considered for patients following an MI. Invasive electro-physiological studies are also not routinely recommended (SIGN 2007a).

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