Nitrates and other agents
Sublingual glyceryl trinitrate (300-500 μg) is a valuable first-aid measure in threatened infarction, and intravenous nitrates (nitroglycerin 0.6-1.2 mg/hour or isosorbide dinitrate 1-2 mg/hour) are useful for the treatment of left ventricular failure and the relief of recurrent or persistent ischaemic pain. Large-scale trials have shown that there is no evidence of a survival advantage from the routine use of oral nitrate therapy, oral calcium antagonists or intravenous magnesium in patients with acute MI.

COMPLICATIONS OF INFARCTION:
Arrhythmias:
Ventricular fibrillation
Ventricular tachycardia
Accelerated idioventricular rhythm
Ventricular ectopics
Atrial fibrillation
Atrial tachycardia
Sinus bradycardia (particularly after inferior MI)

Heart block
Pain relief, rest and the correction of hypokalaemia can all play a major role in the prevention of arrhythmias.

Ventricular fibrillation
This occurs in about 5-10% of patients who reach hospital, and is thought to be the major cause of death in those who die before receiving medical attention.

Prompt defibrillation will usually restore sinus rhythm.
Moreover, the prognosis of patients with early ventricular fibrillation (within the first 48 hours) who are successfully and promptly resuscitated in this way is identical to the prognosis of patients with acute MI that is not complicated by ventricular fibrillation.

Atrial fibrillation
This is common, frequently transient and may not require treatment.
However, if the arrhythmia causes a rapid ventricular rate with severe hypotension or circulatory collapse, cardioversion by means of an immediate synchronised DC shock should be considered.
In other situations, digoxin or β-blockers are usually the treatment of choice.
Atrial fibrillation (due to acute atrial stretch) is often a feature of impending or overt left ventricular failure, and therapy may be ineffective if heart failure is not recognised and treated appropriately. Anticoagulation may be required if AF persists.

Sinus bradycardia
This does not usually require treatment, but if there is hypotension or haemodynamic deterioration, atropine (0.6 mg i.v.) may be given.
Atrioventricular block complicating inferior infarction is usually temporary and often resolves following thrombolytic therapy; it may also respond to atropine (0.6 mg i.v. repeated as necessary).

However, if there is clinical deterioration due to second-degree or complete atrioventricular block, a temporary pacemaker should be considered.

Atrioventricular block complicating anterior infarction is more serious because asystole may suddenly supervene; a prophylactic temporary pacemaker should be inserted.

Ischaemia

Post-infarct angina occurs in up to 50% of patients. Most patients have a residual stenosis in the infarct-related vessel despite successful thrombolysis, and this may cause angina if there is still viable myocardium downstream; nevertheless, there is no evidence that routine angioplasty improves outcome after thrombolysis.

Patients who develop angina at rest or on minimal exertion following MI should be managed in the same way as patients with unstable angina who are thought to be at high risk.

Acute circulatory failure

Acute circulatory failure usually reflects extensive myocardial damage and indicates a bad prognosis.

All the other complications of MI are more likely to occur when acute heart failure is present.

Pericarditis: This may occur at any stage of the illness but is particularly common on the second and third days.

The patient may recognize that a different pain has developed even though it is at the same site, and that this pain is positional and tends to be worse or is sometimes only present on inspiration.

A pericardial rub may be audible.

Non-steroidal and steroidal anti-inflammatory drugs should be avoided in the early recovery period as they may increase the risk of aneurysm formation and myocardial rupture.

Opiate-based analgesia should be used.

The post-myocardial infarction syndrome (Dressler’s syndrome)

is characterized by persistent fever, pericarditis and pleurisy, and is probably due to autoimmunity.

The symptoms tend to occur a few weeks or even months after the infarct and often subside after a few days; prolonged or severe symptoms may require treatment with high-dose aspirin, an NSAID or even corticosteroids.

Mechanical complications

Part of the necrotic muscle in a fresh infarct may tear or rupture, with devastating consequences:

Papillary muscle damage may cause acute pulmonary oedema and shock due to the sudden onset of severe mitral regurgitation, which presents with a pansystolic murmur and third heart sound.
In the presence of severe valvular regurgitation, the murmur may be quiet or absent. The diagnosis can be confirmed by Doppler echocardiography, and emergency mitral valve replacement may be necessary.

**Rupture of the interventricular septum**

This usually presents with sudden haemodynamic deterioration accompanied by a new loud pansystolic murmur radiating to the right sternal border, but may be difficult to distinguish from acute mitral regurgitation.

However, patients with an acquired ventricular septal defect tend to develop right heart failure rather than pulmonary oedema

Doppler echocardiography and right heart catheterisation will confirm the diagnosis. Without prompt surgery, the condition is usually fatal.

**Rupture of the ventricle** may lead to cardiac tamponade and is usually fatal.

**Embolism:**

Thrombus often forms on the endocardial surface of freshly infarcted myocardium; this may lead to systemic embolism and occasionally causes a stroke or ischaemic limb.

Venous thrombosis and pulmonary embolism may occur but have become less common with the use of prophylactic anticoagulants and early mobilisation.

**Impaired ventricular function:**

Acute transmural MI is often followed by thinning and stretching of the infarcted segment (infarct expansion); this leads to an increase in wall stress with progressive dilatation and hypertrophy of the remaining ventricle (ventricular remodelling).

As the ventricle dilates, it becomes less efficient and heart failure may supervene. Infarct expansion occurs over a few days and weeks but ventricular remodelling may take years; heart failure may therefore develop many years after acute MI. ACE inhibitor therapy reduces late ventricular remodelling and can prevent the onset of heart failure

**A left ventricular aneurysm** develops in approximately 10% of patients. Can lead to:

Heart failure.

ventricular arrhythmias.

mural thrombus.

systemic embolism.

Other clinical features include a paradoxical impulse on the chest wall.

persistent ST elevation on the ECG.

An unusual bulge from the cardiac silhouette on the chest X-ray.

Echocardiography is usually diagnostic.

Surgical removal of a left ventricular aneurysm carries a high morbidity and mortality but is sometimes necessary.
LATE MANAGEMENT

Patients who have survived an MI are at risk of further ischemic events. Management should therefore aim to identify those at high risk and introduce effective secondary prevention.

**Lifestyle modification:**

Stop smoking
Regular exercise
Diet (weight control, lipid-lowering)

**Secondary prevention drug therapy:**

Antiplatelet therapy (aspirin and/or clopidogrel)
β-blocker
ACE inhibitor
Statin

Additional therapy for control of diabetes and hypertension

**Rehabilitation**

If the exercise test is negative and the patient has a good effort tolerance, the outlook is good, with a 1-4% chance of an adverse event in the next 12 months.

In contrast, patients with residual ischaemia in the form of chest pain or ECG changes at low exercise levels are at high risk, with a 15-25% chance of suffering a further ischaemic event in the next 12 months.

Coronary angiography, with a view to angioplasty or bypass grafting, should therefore be considered in any patient with spontaneous ischaemia, significant angina on effort, or a strongly positive exercise tolerance test

**Arrhythmias**

The presence of ventricular arrhythmias during the convalescent phase of MI may be a marker of poor ventricular function and may herald sudden death. Although empirical anti-arrhythmic treatment appears to be of no value and even hazardous, selected patients may benefit from sophisticated electrophysiological testing and specific anti-arrhythmic therapy (including implantable cardiac defibrillators).

**Secondary prevention**

Smoking: The 5-year mortality of patients who continue to smoke cigarettes is double that of those who quit smoking at the time of their infarct.

**Hyperlipidaemia**

Lipids should be measured within 24 hours of presentation because there is often a transient fall in blood cholesterol in the 3 months following infarction

Dietary advice should be given but is often ineffective. HMG CoA reductase enzyme inhibitors (‘statins’) can produce marked reductions in total (and LDL) cholesterol and have been shown to reduce the subsequent risk of death, reinfarction, stroke and the need for revascularisation.

Irrespective of serum cholesterol concentrations, all patients should receive statin therapy after MI. Recent evidence suggests that patients with serum LDL cholesterol concentrations
greater than 3.2 mmol/l (∼120 mg/dl) benefit from more intensive lipid-lowering (e.g. atorvastatin 80 mg daily).

Other risk factors
Maintaining an ideal body weight,
taking regular exercise,
and achieving good control of hypertension and diabetes may all improve the long-term outlook.

Mobilisation and rehabilitation.
There is histological evidence that the necrotic muscle of an acute myocardial infarct takes 4-6 weeks to become replaced with fibrous tissue, and it is conventional to restrict physical activities during this period.
When there are no complications, the patient can sit in a chair on the second day,
walk to the toilet on the third day,
return home in 5 days
and gradually increase activity with the aim of returning to work in 4-6 weeks.
The majority of patients may resume driving after 4-6 weeks.

Drug therapy
Aspirin and clopidogrel: Low-dose aspirin therapy reduces the risk of further infarction and other vascular events by approximately 25% and should be continued indefinitely if there are no unwanted effects.
Clopidogrel should be given in combination with aspirin for the first 4 weeks.
If patients are intolerant of aspirin, clopidogrel is a suitable alternative.

Beta-blockers
Continuous treatment with an oral β-blocker has been shown to reduce long-term mortality by approximately 25% among the survivors of acute MI. Unfortunately, a significant minority of patients do not tolerate β-blockers because of bradycardia, atrioventricular block, hypotension or asthma.
Patients with heart failure,
irreversible chronic obstructive pulmonary disease
or peripheral vascular disease derive similar if not greater secondary preventative benefits from β-blocker therapy if they can tolerate it, and should not be denied this treatment.

ACE inhibitors:
Several clinical trials have shown that long-term treatment with an ACE inhibitor (e.g. enalapril 10 mg 12-hourly or ramipril 2.5-5 mg 12-hourly
can counteract ventricular remodelling,
prevent the onset of heart failure,
improve survival and reduce hospitalisation.
The benefit of treatment is greatest in those with overt heart failure (clinical or radiological) but extends to patients with asymptomatic LV dysfunction and those with preserved LV function.

In patients intolerant of ACE inhibitor therapy, angiotensin receptor blockers (e.g. valsartan 40-160 mg daily or candesartan 4-16 mg daily) are suitable alternatives and are better tolerated. Patients with acute MI complicated by heart failure and LV dysfunction appear to benefit from additional aldosterone receptor antagonism (e.g. eplerenone 25-50 mg daily).

Device therapy: Implantable cardiac defibrillators are of benefit in preventing sudden cardiac death in patients who have severe left ventricular impairment (ejection fraction ≤ 30%) after MI.

PROGNOSIS: In almost one-quarter of all cases of MI, death occurs within a few minutes without medical care.

Half the deaths from MI occur within 24 hours of the onset of symptoms and about 40% of all affected patients die within the first month.

The prognosis of those who survive to reach hospital is much better, with a 28-day survival of more than 80%.

Early death is usually due to an arrhythmia but later on the outcome is determined by the extent of myocardial damage.

Unfavourable features include poor left ventricular function, atrioventricular block and persistent ventricular arrhythmias.

The prognosis is worse for anterior than for inferior infarcts. Bundle branch block and high enzyme levels both indicate extensive myocardial damage. Old age, depression and social isolation are also associated with a higher mortality. Of those who survive an acute attack, more than 80% live for a further year, about 75% for 5 years, 50% for 10 years and 25% for 20 years.

MYOCARDIAL INFARCTION IN OLD AGE:

Atypical presentation: often with anorexia, fatigue or weakness rather than chest pain.

Case fatality: rises steeply. Hospital mortality exceeds 25% in those over 75 years old, which is five times greater than that seen in those aged less than 55 years.

Survival benefit of treatments: not influenced by age. The absolute benefit of evidence-based treatments may therefore be greatest in older people.

Hazards of treatments: rise with age (e.g. increased risk of intracerebral bleeding after thrombolysis) and is due partly to increased comorbidity.