Ischaemic heart disease presenting as arrhythmias

A V Ghuran and A J Camm
Department of Cardiological Sciences, St George's Hospital Medical School, London, UK

Despite considerable progress in management over the recent years, coronary artery disease (CAD) remains the leading cause of death in the industrialised world. It is estimated that CAD is responsible for causing 152,000 deaths per year in the UK and one in eight deaths world-wide. Many of these deaths are attributed to the development of ventricular tachyarrhythmias during periods of myocardial ischaemia or infarction. Myocardial ischaemia is characterised by ionic and biochemical alterations, creating an unstable electrical substrate capable of initiating and sustaining arrhythmias, and infarction creates areas of electrical inactivity and blocks conduction, which also promotes arrhythmogenesis. The purpose of this chapter is to review some of the metabolic changes associated with cardiac ischaemia, their relevance to electrophysiological instability, and the clinical manifestation and management of some of the more common arrhythmias that follow cardiac ischaemia. Particular attention is given to the peri-infarction period (arbitrarily accepted as within 48 h of the index myocardial infarction) as arrhythmias are most likely to be seen around this time, and are considered to be non-indicative of long-term prognosis. In contrast, arrhythmias developing in the post-infarction period (after 48 h) have been demonstrated to be associated with an adverse outcome. Regardless of the anti-arrhythmic therapy used in treating peri- and post-infarction arrhythmias, it is presumed that patients who had a myocardial infarction or who have left ventricular dysfunction will also receive other appropriate therapies, such as aspirin, β-blockers, cholesterol lowering agents and angiotensin converting enzymes inhibitors.

Factors contributing to arrhythmias during acute myocardial ischaemia

Biochemical and electrophysiological factors

Acute myocardial ischaemia is accompanied by significant intracellular and extracellular ionic and metabolic alterations of the myocardial syncytiun. Extracellular changes include: elevated potassium, lysophosphoglycerides
and adenosine concentrations, increased lactate and carbon dioxide production, acidosis, and catecholamine release. Concomitantly, intracellular changes include: acidosis, elevated cyclic adenosine monophosphate (cAMP), and elevated concentrations of calcium, magnesium, and sodium ions. These biochemical and metabolic changes alter inward and outward transmembrane ionic current fluxes, causing profound alterations of the resting membrane and action potential characteristics of the myocyte. Changes such as depolarisation of the resting membrane potential, diminished upstroke velocity, slowed conduction, decreased excitability, shortening of the action potential...
duration, altered refractoriness, dispersion of repolarisation, and abnormal automaticity, can all occur.

The resultant biochemical and electrical changes do not all occur at once but evolve temporally, providing the electrophysiological trigger and anatomic substrate necessary to induce arrhythmias through virtually all known arrhythmogenic mechanisms (Fig. 1). A history of a previous myocardial infarction with scar formation further contributes to this arrhythmogenic milieu. The presence of myocardial fibrosis causes slowing of cardiac conduction, resulting in re-entry circuits and subsequent ventricular desynchronisation.

**Autonomic nervous system**

The pathophysiological role of the autonomic nervous system (ANS) in arrhythmogenesis has been firmly established both experimentally and clinically. Within minutes of myocardial ischaemia there is a striking surge of sympathetic nerve activity caused by a combination of pain, anxiety and reflex activation, which has been demonstrated to be inversely related to left ventricular ejection fraction. A general increase in circulating catecholamines can also aggravate myocardial ischaemia, because of positive chronotropic and inotropic actions, therefore establishing a vicious circle.

A relative excess in sympathetic over vagal activity is generally pro-arrhythmic because of alterations of the electrophysiological properties of the specialised conducting tissue and the cardiac myocyte (Table 1). Consequently, the risk of developing supraventricular and ventricular tachyarrhythmias is increased.

In the early peri-infarction period, cardiac autonomic reflexes can be triggered depending on the site of the myocardial infarction. For instance, acute inferoposterior myocardial ischaemia or infarction often results in bradycardia and hypotension, whereas anterior myocardial ischaemia more frequently evokes tachycardia and hypertension. There is a greater density of vagal afferent receptors in the inferoposterior wall of the left ventricle, which may be responsible for causing an enhanced

---

**Table 1** Electrophysiological effects of the sympathetic nervous system

- Shifts pacemaker from sinus node to junctional region
- Increases Purkinje fibre automaticity
- Alters P wave morphology and shortens QT interval
- Shortens PR interval
- Increase after-depolarisations (facilitating triggered activity)
- Enhances re-entry during acute myocardial ischaemia
- Decreases ventricular fibrillation threshold
Ventricular arrhythmias

The mechanisms of ventricular arrhythmias in acute myocardial ischaemia and infarction have been mainly studied using animal models, and have been shown to occur in several distinct phases. The acute phase, which occurs roughly 2–30 min following coronary artery occlusion when changes are still reversible, demonstrates a bimodal distribution and is divided into phases 1a and 1b. Phase 1a arrhythmias occur between 2–10 min. Although several mechanisms have been proposed to explain these arrhythmias, the pathophysiology is most likely to be related to alterations in cellular electrophysiology and re-entrant mechanisms. Phase 1b arrhythmias occur 10–30 min after acute coronary occlusion and may be related to local accumulation of catecholamines and increased automaticity. The second or delayed phase of ventricular arrhythmias occurs up to 72 h after coronary artery occlusion, with a peak incidence between 12–24 h. These arrhythmias may be caused by abnormal automaticity within surviving Purkinje fibres, triggered activity arising from Purkinje fibres, or re-entry mechanisms involving either the Purkinje fibres or the ischaemic myocardium. Chronic phase arrhythmias developing after 72 h are usually due to re-entry mechanisms.

Ventricular premature complexes (VPCs)

VPCs commonly develop during periods of ischaemia. In the early peri-infarction period, the incidence of VPCs has been reported to vary between 10–93%. They are usually asymptomatic and their presence in the peri-infarction period, regardless of frequency and complexity (bigeminy, multiformity, etc) bears no relation to mortality or the development of sustained ventricular tachyarrhythmias. In contrast, their presence in the post-infarction period (usually >10 per h) is a strong predictor of all cause and arrhythmic mortality.
Treatment
Numerous trials have compared prophylactic anti-arrhythmic drugs with placebo for the treatment of VPCs in the peri-infarction and post-infarction periods following a myocardial infarction. Although there may be a reduction in VPCs' frequency, none of the agents administered (with the exception of β-blockers) have conclusively reduced cardiovascular or all-cause mortality and some anti-arrhythmic drugs may facilitate arrhythmic death\textsuperscript{10–14}. VPCs are treated conservatively by alleviation of any on-going cardiac ischaemia, and correction of electrolyte and metabolic disturbances. β-Blockers should be administered as early as possible to avoid the pro-arrhythmic effects of sympathetic stimulation.

Ventricular arrhythmias
Ventricular tachycardia (VT) is defined as three or more consecutive cardiac depolarisation arising below the atroventricular node, with an RR interval of less than 500 ms (>120 beats/min). VT is estimated to occur in 3–39\% of patients in the peri-infarction period\textsuperscript{7,8}. The presentation of ventricular tachycardia during acute myocardial infarction depends on the rate of tachycardia, and on left ventricular function. Significant haemodynamic compromise can occur if the tachycardia is fast and sustained, and when there is left ventricular dysfunction. VT increases myocardial oxygen demand, which may result in exacerbation of ischaemia and possible infarct extension. Occasionally, VT is the presenting feature of an otherwise silent myocardial infarction (the presence of a scar provides a stable substrate capable of maintaining a re-entrant tachycardia mechanism).

VT is conventionally classified according to its temporal and morphological characteristics. VT is described as non-sustained (NSVT), if the duration is less than 30 s, and sustained if it lasts more than 30 s or requires termination within 30 s because of haemodynamic compromise. VT is described as being ‘monomorphic’ if the QRS complexes have one morphology; multiple monomorphic if there are two or more runs of different QRS morphologies, but each run has a uniform QRS complex; and polymorphic if the QRS morphology is variable during one episode (Fig. 3).

Treatment
The treatment of ventricular tachyarrhythmias should target both the cause of the arrhythmia (upstream approach) as well as the arrhythmic expression of the pathology (downstream approach). In other words, in patients with significant coronary artery disease, revascularisation and haemodynamic optimisation should be considered in the first instance to prevent ventricular arrhythmias and their complications.
Accelerated idioventricular rhythm (slow ventricular tachycardia)

This rhythm is caused by an abnormally firing ventricular focus, which usurps sinus node pacemaker dominance and further depress sinoatrial node automaticity (Fig. 2). By definition, the heart rate is less than 120 beats/min. It occurs very commonly during myocardial infarction and has been shown to be particularly associated with reperfusion of the myocardium following thrombolytic therapy.

Treatment

This rhythm is usually benign and has no adverse effect on mortality. Most episodes are transient and require no treatment. If the rhythm causes haemodynamic compromise, for example due to loss of atrioventricular synchrony, increasing the atrial rate with atropine or atrial pacing is indicated.

NSVT

The incidence of NSVT (monomorphic) in the peri-infarction period has generally been reported to occur in 1–7% of patients\(^\text{15,16}\). Limited data are available regarding the prognostic significance of NSVT in the peri-infarction period. Earlier studies have suggested that NSVT had no adverse effect on either in-hospital or 1-year survival\(^\text{15,17}\). However, a recent study by Cheema et al\(^\text{18}\) has found that the time of onset of NSVT from admission (\(\geq 13\) h and \(\leq 24\) h) as well as specific NSVT characteristics (association with previous MI or faster heart rates) were significant predictors of long-term survival.
Substantial data have demonstrated that NSVT in the post-infarction period increases the risk of sudden cardiac death by at least 2-fold\textsuperscript{19,20}. This risk is further increased to more than 5-fold in patients with left ventricular dysfunction (ejection fraction <0.40)\textsuperscript{19,20}.

**Treatment**

Apart from the early administration of β-blockers (Table 2), the administration of anti-arrhythmic drugs for the treatment of asymptomatic NSVT in the peri-infarction period should generally be avoided. If episodes of NSVT are frequent, rapid, prolonged or associated with significant symptoms, the administration of lidocaine or amiodarone can be considered.

Although the occurrence of NSVT in the post-infarction period is a prognostic marker, its suppression or treatment with anti-arrhythmic drugs has been disappointing\textsuperscript{11–14}. CAMIAT was a randomised double-blind placebo control trial designed to investigate the effect of amiodarone on the risk of resuscitated ventricular fibrillation or arrhythmic death among survivors of myocardial infarction with frequent VPCs (≥10 VPCs/h) or at least a short run of NSVT (defined as ≥3 beats at a rate of 100–120 per min, or 3–10 beats at a rate of >120 per min)\textsuperscript{13}. Although amiodarone reduced arrhythmic mortality, there was no corresponding reduction in all-cause mortality. Subsequent analyses of CAMIAT as well as the European Myocardial Infarct Trial (EMIAT) have demonstrated that the combination of amiodarone plus a β-blocker may have a synergistic interaction with a consequent reduction in all-cause and arrhythmic mortality\textsuperscript{21}. Further adequately powered, prospective studies are needed to confirm this interaction.

In the late post-infarction period, the advent of implantable cardioverter defibrillators (ICD) has changed clinical practice. Patients with a history of coronary artery disease, NSVT, left ventricular dysfunction (ejection fraction <0.40) and inducible VT on electrophysiological testing, ICD implantation has been shown to reduce significantly arrhythmic and all-cause mortality\textsuperscript{22,23}. The treatment of NSVT outside this carefully selected patient group remains less defined.

**Sustained VT**

Peri-infarction sustained VT has an incidence of 0.3–1.9%\textsuperscript{15,24}. It is associated with a higher in-hospital mortality, but is not considered to be a prognostic factor among hospital survivors\textsuperscript{15}. The occurrence of sustained monomorphic VT is an uncommon arrhythmia in the peri-infarction period. When present, it usually signifies previous myocardial scarring or may be a sign of extensive myocardial damage\textsuperscript{24}.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Loading dose</th>
<th>Maintenance</th>
<th>Comments</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Incremental doses of 0.5 mg every 3–5 min up to a maximum of 2 mg.</td>
<td>Orally: 100 mg twice daily</td>
<td>Atropine should be used with caution in the setting of acute myocardial infarction because of the protective effects of parasympathetic stimulation against ventricular arrhythmias and infarct extension</td>
<td>Bradyarrhythmias</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>IV: 2.5 mg over 2–4 min, may repeat every 5 min up to 15 mg</td>
<td>IV: 0.25–0.5 mg/min</td>
<td>Hypotension, bronchospasm, negative inotrope and chronotrope. Acts synergistically with digoxin for rate control in atrial fibrillation. Avoid if heart rate &lt;60 per min or if PR &gt;0.24 s</td>
<td>Treatment of supraventricular, atrial and ventricular tachyarrhythmias</td>
</tr>
<tr>
<td>Propranolol</td>
<td>IV: 0.5–1 mg every 5 min to a maximum 0.15–0.2 mg/kg</td>
<td>Orally: 40–240 mg/day in 3–4 divided doses</td>
<td>As for metoprolol</td>
<td>As for metoprolol</td>
</tr>
<tr>
<td>Atenolol</td>
<td>IV: 2.5–10 mg at a rate of 1 mg/min</td>
<td>Orally: 100 mg/day</td>
<td>As for metoprolol</td>
<td>As for metoprolol</td>
</tr>
<tr>
<td>Esmolol</td>
<td>IV: 0.5 mg/kg/min</td>
<td>IV: 0.05–0.2 mg/kg/min</td>
<td>Very short half-life, as for metoprolol</td>
<td>As for metoprolol</td>
</tr>
<tr>
<td>Digoxin</td>
<td>IV/orally: 0.25–0.5 mg every 6–8 h up to 1 mg/24 h</td>
<td>IV/orally: 0.125–0.5 mg/day</td>
<td>Peak effects may take up to 2 h</td>
<td>Ventricular rate control for atrial fibrillation, especially if left ventricular dysfunction</td>
</tr>
<tr>
<td>Verapamil</td>
<td>IV: 2.5–10 mg over 2 min. Can repeat dose after 30 min</td>
<td>IV: 0.125 mg/min</td>
<td>Acts synergistically with digoxin, increase digoxin levels, hypotension, bradycardia, negative inotrope</td>
<td>Treatment of supraventricular and atrial tachyarrhythmias</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>IV: 0.25–0.35 mg/kg/min over 2 min</td>
<td>IV: 5–15 mg/h</td>
<td>As for verapamil</td>
<td>As for diltiazem</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>IV: 300 mg, made up to 20 ml with 5% dextrose (can be given peripherally).</td>
<td>Orally: 90–240 mg/day in divided doses</td>
<td>Can increase digoxin and warfarin levels. Contra-indications: sinus or AV node disease (unless fitted with a pacemaker) iodine sensitivity, pregnancy, breast feeding, thyroid dysfunction (relative)</td>
<td>Treatment of supraventricular, atrial and ventricular tachyarrhythmias especially if associated left ventricular dysfunction</td>
</tr>
<tr>
<td>Procainamide</td>
<td>12–17 mg/kg over 30–60 min (20–30 mg/min)</td>
<td>2–4 mg/min</td>
<td>Caution in patients with renal insufficiency. Reduce dose or discontinue if QT interval is prolonged by 60 ms above baseline or more than 500 ms</td>
<td>Treatment of supraventricular, atrial and ventricular tachyarrhythmias</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1–1.5 mg/kg</td>
<td>1–4 mg/min. Reduce after 24 h to 1–2 mg/min</td>
<td>Additional boluses of 0.5–0.75 mg/kg every 5–10 min as needed, to a maximum of 3 mg/kg</td>
<td>Treatment of ventricular tachyarrhythmias</td>
</tr>
</tbody>
</table>
In the setting of acute myocardial infarction, polymorphic VT is not usually related to QT interval prolongation, sinus bradycardia, pauses or electrolyte abnormalities. When present, it usually implies recurrent myocardial ischaemia. It has been reported to occur in 0.3–2% of patients in the peri-infarction period\textsuperscript{15}. The prognosis is similar to patients with sustained VT.

**Treatment**

Rapid treatment of sustained VT is mandatory because of the deleterious effect on cardiac output, the exacerbation of myocardial ischaemia, and the risk of degeneration into ventricular fibrillation. If there is haemodynamic compromise, synchronised direct current cardioversion (DCC) should be implemented. If the patient is haemodynamically stable, pharmacological termination can be attempted. The ACC/AHA has recommended either: amiodarone, procainamide or lidocaine to treat peri-infarction sustained monomorphic VT (Table 2)\textsuperscript{15}. Although lidocaine, which has an acceptable safety profile, has been traditionally used for treating stable monomorphic VT, studies have suggested that it is relatively ineffective for termination of VT\textsuperscript{26} and less effective against VT than IV procainamide\textsuperscript{27} or IV sotalol\textsuperscript{28}. Studies investigating the use of amiodarone to treat haemodynamically stable VT are minimal; however, it is effective in treating unstable VT and VF\textsuperscript{29} and consequently it is considered an acceptable agent to treat stable VT. The new *Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*\textsuperscript{30} has de-emphasised the use of lidocaine as a first-line agent for the treatment of stable monomorphic VT, recommending either intravenous sotalol or intravenous procainamide as first-line agents in patients with normal left ventricular function. In patients with left ventricular dysfunction, either amiodarone or lidocaine is recommended as a first-line agent as they cause the least additional impairment of LV function\textsuperscript{30}. Hypokalaemia and hypomagnesaemia should be corrected by ensuring plasma concentrations $\geq 4$ mmol/l and 2 mmol/l, respectively.

If not already administered or contra-indicated, patients with recurrent VT should be established on intravenous amiodarone. In patients who continue to display arrhythmias despite the use of amiodarone at recommended doses, supplemental infusions can be considered\textsuperscript{29}. The use of drug combinations (amiodarone plus either β-blocker, lidocaine or procainamide) may be helpful during the early phases of dosing, until amiodarone has reached higher myocardial concentrations\textsuperscript{29}.

Burst overdrive pacing can also be used to terminate refractory VT. Overdrive pacing is performed by inserting a temporary pacing wire in the right ventricle and pacing 10–20 beats/min faster than the VT rate, for approximately 20–50 beats. Atrial or ventricular pacing at rates marginally higher than the intrinsic sinus rate (physiological overdrive) can help prevent bradycardia-related ventricular arrhythmias.
Peri-infarction polymorphic VT is uncommon and its treatment is the same for sustained VT. However, these arrhythmias are less responsive to class 1 agents and may be suppressed with intravenous amiodarone. As these arrhythmias are usually associated with recurrent ischaemia, treatment should include strategies to reduce ischaemia, such as adequate doses of β-blockers and emergency PTCA/CABG surgery.

Ventricular fibrillation

Ventricular fibrillation is characterised by rapid, disorganised, multiple re-entrant wavelets in the ventricle, resulting in no uniform ventricular contraction and no cardiac output. Untreated, the arrhythmia is lethal and it is the main mechanism of sudden cardiac death. It has been reported to occur in 3% of acute MI with approximately 60% of episodes occurring within 4 h and 80% within 12 h.

Treatment

The only definitive treatment for VF is defibrillation. Initially, these rhythms are readily treatable; however, the chances of successful defibrillation diminish rapidly with time, declining by 5–10% per min. Therefore, the priority is to minimise any delay between the onset of cardiac arrest and the administration of defibrillation shocks. The treatment of VF is now standardised to local and national resuscitation protocols. Results of the recently published ARREST study, showed that the early use of intravenous amiodarone after 3 failed DC shocks, can increase the number of survivors.

In treating peri-infarction ventricular tachyarrhythmias (VT and VF), it is common clinical practice to continue anti-arrhythmic drug infusions for 24–48 h and to discontinue the infusion provided there is no arrhythmic recurrence. In patients with refractory ventricular arrhythmias or electrical storms, high dose β-blockade coupled with atrial or dual chamber pacemaker therapy, light anaesthesia and artificial ventilation may be life-saving. Urgent referral for coronary revascularisation and/or implantable cardioverter defibrillation should be considered for patients who develop ventricular arrhythmias >48 h after the index MI or in whom the arrhythmias persist beyond this point. In patients who are adequately revascularised and who refuse device therapy, pace mapping to identify the arrhythmogenic zone combined with radiofrequency ablation has been shown to be an acceptable strategy in that small proportion of patients with a single tachycardia focus on the endocardial surface.

Sudden cardiac death (SCD) and ICDs

Aborted SCD is, in the majority of cases, caused by life-threatening ventricular tachyarrhythmias. A number of randomised, clinical trials
have now demonstrated the superiority of the ICD over anti-arrhythmic drug therapy (mostly or exclusively amiodarone) in survivors of aborted SCD. On the basis of these studies, treatment with an ICD is considered a class 1 indication (available evidence and general agreement that a device or procedure is beneficial and efficacious) for secondary prevention in: (i) survivors of cardiac arrest not accompanied by an acute myocardial infarction or other reversible cause; (ii) patients with syncope of unknown aetiology and inducible haemodynamically significant VT/VF on electrophysiological testing; and (iii) patients with spontaneous, sustained VT. Patients with associated left ventricular dysfunction (≤35%) derive the most benefit from an ICD.

Aborted SCD is invariably associated with structural heart disease, which in the adult population is most frequently CAD. Although the exact role of cardiac ischaemia in the pathogenesis of SCD is not clearly defined, it is believed to be the trigger that initiates arrhythmogenesis. Therefore, reversal or prevention of ischaemia should avoid the occurrence of the arrhythmia. A recently published study investigating the effects of coronary artery revascularisation in patients with sustained ventricular arrhythmias in the chronic phase of a myocardial infarction demonstrated that arrhythmia recurrence is still high following revascularisation, particularly in patients with reduced left ventricular function. It is, therefore, recommended that survivors of aborted SCD with obstructive CAD and evidence of an arrhythmic substrate (Q waves, depressed left ventricular function, inducible VT/VF) should not only be revascularised, but should also have an ICD implanted. Controversies in management exist in aborted SCD survivors who are demonstrated to have reversible ischaemia and no arrhythmic substrate (no Q waves, preserved left ventricular function and non-inducible VT/VF). Some electrophysiologists believe that these patients are at low risk, and revascularisation and β-blockers may be all that is required, whereas others would recommend implantation of an ICD.

Supraventricular arrhythmias

Sinus bradycardia

Sinus bradycardia (< 60 beats/min) is common, occurring in 25–40% of patients within the first hour of a myocardial infarction. It is more common with inferior wall myocardial infarction and is often due to hypervagotonia. Treatment is only necessary when there are symptoms or evidence of haemodynamic compromise. Most cases respond well to intravenous atropine (0.6–1 mg). Persistent symptomatic bradycardia despite atropine is an indication for temporary cardiac pacing.
Sinus tachycardia

Sinus tachycardia occurs in about 30% of patients with acute myocardial infarction. It can aggravate myocardial ischaemia by increasing myocardial oxygen consumption as well as reducing diastolic coronary artery perfusion time. Sinus tachycardia is also a manifestation of significant ventricular dysfunction, on-going cardiac ischaemia, inadequate analgesia, anxiety, pyrexia and hypovolaemia. Management is aimed at treating the underlying causes. When sinus tachycardia is inappropriately fast, given the physiological state of the patient, slowing of the heart rate with \(\beta\)-blockade is helpful (Table 2), particularly if the patient has evidence of ongoing cardiac ischaemia.

Atrial tachyarrhythmias

The incidence of atrial tachyarrhythmias during the peri-infarction period is estimated at 10–20% \(^{40}\), with atrial fibrillation being the commonest atrial tachyarrhythmia (occurs in 10–15% of cases). Atrial flutter occurs in less than 5% of patients. These arrhythmias usually occur within 72 h of the index infarction with less than 3% arising in the very early phase (<3 h) \(^{41}\).

Atrial fibrillation has been shown to be independently associated with in-hospital and long-term mortality, re-infarction rates, ventricular arrhythmias, advanced atrioventricular conduction disturbances, asystole, cardiogenic shock, and ischaemic strokes. It is also more likely to be associated with extensive coronary artery disease and poor reperfusion of the infarct related artery \(^{42,43}\) and, therefore, the threshold for cardiac catheterisation should be low.

Factors associated with the development of peri-infarction atrial fibrillation include: atrial infarction/ischaemia, sinus node dysfunction, older age, metabolic abnormalities, pericarditis, pericardial effusion, right ventricular infarction, congestive heart failure, higher peak cardiac enzyme concentration, increased heart rate, diabetes mellitus, history of hypertension and inotropic drugs. The development of atrial fibrillation within 24 h is usually associated with inferior wall myocardial infarction from right coronary artery occlusion. In contrast, atrial fibrillation developing more than 24 h afterwards is associated with anterior wall myocardial infarction and left ventricular dysfunction.

Treatment of atrial tachyarrhythmias

The early treatment of atrial tachyarrhythmias is important as increased ventricular rates and loss of atrial systole result in a significant reduction in cardiac output and an increase in cardiac ischaemia. When there is significant haemodynamic compromise, then DC cardioversion is
indicated. If the patient is haemodynamically stable, early control of the ventricular rate with either a β-blocker, calcium antagonist or digoxin is satisfactory (Table 2). Alternatively, pharmacological cardioversion to sinus rhythm can be attempted with amiodarone or dofetilide. Dofetilide has recently been shown to be effective in cardioverting atrial fibrillation in the post-infarction period in patients with left ventricular dysfunction without affecting all-cause, cardiac and arrhythmic mortality. However, dofetilide is associated with an increased incidence of torsades de pointes and close ECG monitoring is mandatory when it is administered. Although newer class III agents such as ibutilide and azimilide have been successful in the treatment of atrial fibrillation, data concerning their efficacy and safety in the peri-infarction period are currently limited. Elective DC cardioversion should be considered if the patient remains in atrial fibrillation after the acute infarction period has passed. Atrial fibrillation is associated with an increased risk of thrombo-embolism, and provided there are no contra-indications, all patients should be heparinised and considered for oral anticoagulation if atrial fibrillation persists or is paroxysmal.

**Conduction disturbances**

Myocardial ischaemia can produce a broad range of conduction disturbances, involving both the atrioventricular node and infranodal structures. Although early reperfusion with thrombolysis can shorten the duration of AV block and reduce the need for temporary pacing, it has not reduced the incidence of atioventricular block, which has remained relatively constant.

First degree AV block is the most common conduction disturbance occurring in up to 14% of patients with acute myocardial infarction. It is usually associated with inferior myocardial infarction and may be a manifestation of hypervagotonia or functional damage of the AV node. First degree heart block that is below the His bundle is more commonly associated with anterior myocardial infarction and has a worse prognosis. Iatrogenic causes of first degree heart block include drugs such as β-blockers, calcium antagonists and digoxin. Mobitz type 1 heart block (Wenckebach) is present in 4–10% of patients with acute myocardial infarction and accounts for about 90% of patients with second degree heart block. It is usually transient and is more common following inferior infarctions. Mobitz type 2 heart block is less common and is more associated with anterior infarction, indicating damage to the AV junction or His bundle. The QRS complexes are usually wide implying bundle branch involvement and may herald the onset of complete heart block. Complete heart block (CHB) has an incidence of
about 6% and is more common with inferior/posterior infarctions. CHB occurring in association with anterior myocardial infarctions implies extensive myocardial damage and has a worse prognosis. CHB complicating either inferior or anterior wall myocardial infarctions is independently associated with mortality and in-hospital complications.

Conduction disturbances involving the left and right bundle branches occur in 10–24% of patients with acute myocardial infarction. Persistent bundle branch block is an independent marker of mortality, whereas, transient blocks which recover normal conduction during hospitalisation have similar prognosis to patients who never develop this complication.

Left anterior hemiblock occurs in 3–5% of acute myocardial infarctions and mortality is slightly increased. Left posterior hemiblock occurs in 1–2% of cases and, because of its large size, disturbances of this conduction pathway reflect significant myocardial damage, and it is associated with a higher mortality.

Treatment of conduction disturbances

First degree heart block in the peri-infarction period does not require any specific treatment. Similarly, Mobitz type 1 (Wenckebach) heart block does not require specific treatment provided that the ventricular rate is adequate; if there is associated haemodynamic compromise, not responsive to atropine (Table 2), then temporary transvenous ventricular pacing is indicated. Where possible, it is recommended to ensure AV synchronisation by inserting an atrial sensing/pacing electrode. As Mobitz type 2 heart block is at risk of progressing to CHB, the insertion of a temporary pacing wire is recommended. CHB occurring in the context of inferior wall myocardial infarction and hypervagotonia may respond to atropine; otherwise the patient should be temporarily paced. Table 3 summarises the indications for transvenous pacing as recommended by the ACC/AHA.

Although atrioventricular block and/or bundle branch block carry an independent risk for mortality and in-hospital complications, the use of pacing during this period does not alter mortality. However, temporary cardiac pacing does serve to protect against hypotension and subsequent ischaemia exacerbation, as well as the development of bradycardia-dependent ventricular tachyarrhythmias. In the setting of inferior myocardial infarction, CHB is self-limiting and usually resolves within the first week but can last up to 14–16 days. Therefore, decisions regarding the placement of a permanent pacemaker in patients with inferior myocardial infarctions should be delayed for at least a week to allow recovery of normal conduction. In contrast, there is a significant risk of
Ischaemic heart disease presenting as arrhythmias

Table 3  Indications for temporary transvenous pacing and permanent pacing in the peri-infarction and post-infarction periods, respectively

TEMPORARY TRANSVENOUS PACING

Established (evidence supporting and/or general agreement)

- Asystole
- Symptomatic bradycardia including sinus bradycardia and type 1 second-degree AV block with haemodynamic compromise, which is medically refractory
- New or age-indeterminate alternating bundle branch block (including RBBB with alternating LAFB/LAPB)
- New or age-indeterminate trifascicular block (RBBB+LAFB+FDHB, RBBB+LPFB+FDHB, or LBBB+FDHB)
- Mobitz type II second-degree AV block

Less established

- New or age-indeterminate bifascicular block (RBBB+LAFB, RBBB+LPFB or RBBB+FDHB)
- New or age indeterminate LBBB
- Atrial or ventricular overdrive/underdrive pacing for recurrent VT
- Recurrent sinus pauses (greater than 3 s) refractory to atropine

PERMANENT PACING

Established (evidence supporting and/or general agreement)

- Persistent second-degree AV block in the His-Purkinje system with bilateral bundle branch block or third degree AV block within or below the His-Purkinje system
- Transient advanced (second or third degree) infranodal AV block and associated bundle branch block
- Symptomatic AV block independent of location

Less established

- Persistent advanced (second or third degree) block at the AV node level

Note: decisions regarding the placement of a permanent pacemaker in patients with inferior myocardial infarction should be delayed for at least 1 week as the majority of conduction disturbances resolve by this time

Note: AV, atrioventricular; RBBB, right bundle branch block; LAFB, left anterior fascicular block; LPFB, left posterior fascicular block; FDHB, first degree heart block. Adapted from Ryan et al39.

asystole in patients with anterior infarction and CHB (even if transient); therefore, permanent pacing is recommended (Table 3).

Conclusions

Cardiac ischaemia causes complex interactions between ionic, metabolic and neurohormonal factors with deleterious effects on cardiac cellular electrophysiology. The end result is the induction and maintenance of supraventricular and ventricular tachyarrhythmias, and conduction disturbances. Despite improvements in the management of myocardial infarction, the arrhythmias generated during myocardial ischaemia contribute significantly to the morbidity and mortality seen in the peri- and post-infarction periods. At present, supraventricular tachyarrhythmias which are often not life-threatening are reasonably controlled with
pharmacological intervention. In contrast, ventricular tachyarrhythmias which are life-threatening are less restrained by pharmacological therapy, and some agents may precipitate arrhythmic death. ICDs remain an effective tool in selected patient groups in the post-infarction period both in terms of primary and secondary prevention. It is imperative to ensure that all patients with CAD are optimally treated for on-going cardiac ischaemia, and are appropriately prescribed some form of antiplatelet therapy, β-blockers, cholesterol lowering therapy and ACE inhibitors. As CAD continues to be prevalent in today's society, it is inevitable that cardiologists and physicians will continue to be challenged with the arrhythmias generated from cardiac ischaemia.

References

12 Teo KK, Yusuf S, Furberg CD. Effects of prophylactic antiarrhythmic drug therapy in acute myocardial infarction. An overview of results from randomized controlled trials. JAMA 1993; 270: 1589–95
Ischaemic heart disease: therapeutic issues


50 Barold SS. American College of Cardiology/American Heart Association guidelines for pacemaker implantation after acute myocardial infarction. What is persistent advanced block at the atioventricular node? Am J Cardiol 1997; 80: 770–4