CARDIOGENIC SHOCK AND THE HAEMODYNAMIC EFFECTS OF ARRHYTHMIAS

A. P. RAE AND I. HUTTON

CARDIOGENIC SHOCK

Cardiogenic shock or pump failure may be defined as severe circulatory failure resulting from a primary defect in the pumping function of the heart, most commonly from acute myocardial infarction, and less commonly from end-stage congestive cardiomyopathy. It is essential that surgically correctable lesions leading to pump failure are identified and these would include papillary muscle dysfunction with mitral incompetence, rupture of the interventricular septum and pericardial effusion with cardiac tamponade.

Pump failure results primarily from impairment of left ventricular function when greater than 40% of the left ventricle becomes infarcted and is associated usually with triple vessel coronary disease (Harnayaran et al., 1970). A less common but important cause is right ventricular dysfunction secondary to right ventricular infarction (Cohn et al., 1974; Lorell et al., 1979). It is important to recognize this pathology, as treatment is more likely to be successful than when the left ventricle is involved.

The haemodynamic consequences of arrhythmias and conduction problems will be discussed later but, clearly, ventricular performance will be further impaired in the presence of both tachy- and bradyarrhythmias, particularly when there is asynchrony between atrial and ventricular contraction.

Clinical features

Pump failure is a complication of acute myocardial infarction in 15% of patients and is still the major cause of death in hospital. The patient is orthopnoeic, cyanosed, vasoconstricted, cold, peripheral pulses are weak and arterial pressure is reduced with a small pulse pressure. Perfusion of vital organs is impaired, as reflected by drowsiness and oliguria. Heart sounds are faint and additional sounds with tachycardia (S₃ and S₄ gallop rhythms) are common. Confirmatory evidence includes metabolic acidosis, hypoxia and hypercapnia. Disturbances of cardiac rhythm and conduction are common and a chest x-ray confirms the presence of pulmonary oedema.

Haemodynamics

The use of the balloon directed pulmonary artery catheter allows detailed haemodynamic assessment and thus the appropriate therapy for patients with pump failure (Swan et al., 1970). Cardiac output is invariably low, in spite of an increased left ventricular filling pressure, but LV end-diastolic volume may be only modestly increased and the high LV end-diastolic pressure is secondary to a reduction in left ventricular compliance (Diamond and Forrest, 1971). The low cardiac output results in a reduced stroke volume, left ventricular ejection fraction and systemic hypotension. The neuro–humoral responses secondary to increased sympathetic activity include increases in concentrations of circulatory catecholamines, cortisol, renin, angiotensin II and aldosterone, which all lead to increased systemic vascular resistance and fluid retention.

The typical haemodynamic picture of pump failure in acute myocardial infarction consists of a systolic arterial pressure of less than 80 mm Hg with a mean arterial pressure of 60 mm Hg, pulmonary capillary wedge pressure (PCWP) of > 18 mm Hg, a tachycardia of 100 beat min⁻¹, a cardiac index of < 1.8 litre min⁻¹ m⁻² and a systemic vascular resistance of > 2000 dyn s cm⁻⁶.

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Relief of pain with i.v. diamorphine is mandatory and this may also reduce excess vagal stimulation manifested by sinus bradycardia and other bradyarrhythmias. Oxygen is administered by face mask, cardiac arrhythmias are treated promptly and acid–base balance corrected.

The principles in the management of pump failure are:
2. Maintain an optimal preload or left ventricular filling pressure.
3. Reduce systemic vascular resistance and thus impedance to left ventricular ejection by the use of vasodilators.
4. Enhance intrinsic contractility by the use of inotropic agents.

Preload is an important determinant of ventricular performance and, in the context of acute myocardial infarction with the left ventricular compliance problem, the ideal filling pressure is between 18 and 20 mm Hg. If volume expansion is indicated, this is best provided by fructose or plasma volume expanders. Conversely, in the presence of radiological evidence of pulmonary oedema or a PCWP of > 20 mm Hg, i.v. diuretics such as frusemide should be given. The consequent diuresis reduces intravascular volume overload and, in addition, diuretics have venodilating properties, thus further reducing ventricular filling pressure (Hesse, Nielsen and Lund-Jacobsen, 1975).

Inotropic Agents
The use of inotropic agents to stimulate the depressed myocardium in cardiac failure is based on the premise that, although the myocardium is depressed, there is residual contractile reserve which is still capable of being stimulated, thus improving overall myocardial function. There is evidence that inotropes may be of benefit in the short term, but none that the long term administration of such agents will improve symptoms or influence mortality.

The ideal inotropic agent should increase stroke volume and cardiac output without undue increase in heart rate. These benefits should be maintained chronically with no loss of efficacy. The compound should be devoid of unacceptable side-effects, particularly the provocation of arrhythmias, and should also be suitable for oral administration.

Cardiac glycosides
There is no good evidence that digitalis, or other cardiac glycosides, are useful inotropic agents in the treatment of pump failure unless the underlying cardiac rhythm is atrial fibrillation (Swan et al., 1969).

Catecholamines
Catecholamines increase contractility by stimulating the β₁ receptor on the myocardial cell surface. Most of the catecholamines currently available can only be given i.v., and stimulation of these β₁ receptors, in addition to an inotropic effect, produces a positive chronotropic action with an increase in heart rate. This combination increases myocardial oxygen consumption and the increased heart rate, by reducing diastolic filling time, may compromise coronary arterial perfusion. β₂ receptor stimulation produces vasodilatation of vascular smooth muscle, with reduction in systemic arterial pressure and vascular resistance. β₂ receptor stimulation results in vasoconstriction of vascular beds, with an increase in systemic pressure and vascular resistance.

The clinical usefulness of the standard amines—adrenaline, noradrenaline and isoprenaline—is limited by their relative chronotropic and peripheral receptor effects.

Noradrenaline has marked α agonist properties which may be beneficial in hypotension, but this is offset by increases in afterload and myocardial oxygen consumption, thereby compromising left ventricular function and reducing peripheral organ perfusion (Vatner, Higgins and Braunwald, 1974).

Isoprenaline, a β receptor agonist, is a potent inotrope, but this effect is compromised by its powerful chronotropic effect, resulting in tachycardia and provocation of arrhythmias (Beregovich et al., 1972). In addition, β₂ mediated systemic vasodilatation, although improving myocardial performance secondary to afterload reduction, may also be disadvantageous by diverting increased cardiac output to the large vascular splanchnic and skeletal muscle beds and away from vital organs (Gunnar and Loeb, 1972). Reduction in diastolic arterial pressure may also result in a reduction in coronary perfusion pressure.

Adrenaline has both β and α effects, the latter predominant at higher doses, resulting in periph-
eral vasoconstriction and reduced organ perfusion, especially to the kidney (Gunnar and Loeb, 1972).

It was on the basis of these limitations in the clinical application of the sympathomimetic amines, that dopamine and subsequently dobutamine were developed.

**Dopamine**

Dopamine, an endogenous precursor of noradrenaline, has both a direct sympathomimetic action and, by releasing noradrenaline from its stores in the sympathetic nerve terminal, an indirect action. The pharmacology of dopamine has been reviewed by Goldberg (1972). The cardiac effects are mediated by β1 stimulation, but it has less chronotropic activity relative to inotropic activity than has isoprenaline (Tsai, Langer and Trendelenburg, 1967). At low doses it has a specific vasodilating effect on the renal vasculature, which has also been demonstrated in the mesenteric and to some extent the coronary vascular beds. The vasodilating effect results from stimulation of a structurally specific “dopaminergic” receptor (McDonald and Goldberg, 1963). Dopamine has been shown to have weak β2 activity in the dog, but in man this effect is difficult to detect. At higher doses, dopamine behaves similarly to noradrenaline, having a predominant vasoconstrictor effect on peripheral vascular beds mediated by α receptor stimulation (McDonald and Goldberg, 1963). The overall effect of dopamine is particularly dose dependent, allowing relative separation of the different receptor effects. Studies in normal subjects (McDonald et al., 1964) demonstrated that, at doses of dopamine 0.5–2.0 μg kg⁻¹ min⁻¹, renal vasodilatation produces an increase in renal blood flow with little effect on other haemodynamic variables. As the infusion rate is increased to 5 μg kg⁻¹ min⁻¹, the effect on the β1 receptor becomes apparent, with an increase in cardiac contractility and output, but with little heart rate response until infusion rates in the range 5.0–10.0 μg kg⁻¹ min⁻¹ are used. The effects of α stimulation are noted at these higher rates, especially at 10 μg kg⁻¹ min⁻¹ and above, with the development of an increase in systemic arterial pressure and systemic vascular resistance which tends to counteract the renal vasodilatation with a concomitant reduction in renal blood flow. Although coronary sinus blood flow is increased at the low- to mid-dose range as a result of modest coronary vasodilatation and increased cardiac output, at the higher dose range, coronary sinus flow decreases as a result of coronary vasoconstriction secondary to stimulation (Goldberg, 1972).

Haemodynamics and renal function can be improved in the pump failure patient. Cardiac output and stroke volume are increased but, at doses greater than 10 μg kg⁻¹ min⁻¹, increases in heart rate and, more importantly, systemic vascular resistance occur (Holzer et al., 1973; Ichard et al., 1983). The effects of inotropic agents on myocardial metabolism and oxygen usage have also to be considered, and dopamine increases myocardial oxygen consumption (Mueller, Evans and Clynes, 1978).

In the context of acute myocardial infarction there is always a balance between oxygen supply and demand in the tissues surrounding the infarct area, and increase in oxygen usage causes progression of ischaemia to necrosis and thus extends the size of the infarct. The major determinants of myocardial oxygen consumption are: heart rate; myocardial wall tension (determined by LV systolic volume and LV end-diastolic volume); intrinsic myocardial contractility.

**Dobutamine**

Dobutamine, a derived analogue of dopamine, was synthesized by Tuttle and Mills (1975). Their aim was to develop a compound which selectively increased cardiac contractility without adverse vasoconstriction and arrhythmogenicity. In isolated organ and animal experiments, dobutamine has been shown to have marked β1 effects with weak β2 and minimal α activity (Vatner, McRitchie and Braunwald, 1974). It has no effect on dopaminergic receptors (Goldberg, Hsieh and Resnekov, 1977) and is a directly acting sympathomimetic with no indirect mode of action. In dogs, with infusion rates of dobutamine 1–32 μg kg⁻¹ min⁻¹, progressive increases in cardiac output and dP/dtmax were noted. The increase in heart rate and decrease in vascular resistance resulted from β1 stimulation. Systolic arterial pressure increased, but mean and diastolic pressures remained unchanged. There were no significant increases in renal or mesenteric blood flow, but there was a dose-related increase in femoral blood flow as a result of the reduction in vascular resistance and increase in cardiac output (Robie and Goldberg, 1975). The effects of dobutamine are similar to those produced by isoprenaline, although the β2 peripheral effect is less, as is the chronotropic effect. Jewitt and colleagues (1974)
compared the effects of isoprenaline and dobutamine infused at rates of 2.5–10 μg kg\(^{-1}\) min\(^{-1}\) in 10 patients and demonstrated a dose related increase in cardiac output from 4.3 litre min\(^{-1}\) to 6.1 litre min\(^{-1}\), with an increase in stroke volume and \(dP/dt_{\text{max}}\). Unlike isoprenaline, which produces significant tachycardia, dobutamine produced little effect on heart rate. Gillespie and colleagues (1977) infused dobutamine 1–40 μg kg\(^{-1}\) min\(^{-1}\) to 16 patients with acute myocardial infarction for 24 h and reported an increase in cardiac output from 4.9 litre min\(^{-1}\) to 6.0 litre min\(^{-1}\), without significant change in heart rate or systemic arterial pressure. PCWP decreased from 22 to 17 mm Hg. There was no evidence of increase in ventricular ectopy in these patients.

The comparative effects of dopamine and dobutamine have been investigated in patients with heart failure. Stoner, Bolen and Harrison (1977) compared patients to whom dobutamine was infused at a rate of 10 μg kg\(^{-1}\) min\(^{-1}\) with a comparable group of patients infused with dopamine. Although both drugs increased cardiac output by similar increments, the peripheral effects were qualitatively and quantitatively different. Dobutamine increased both cardiac output and stroke volume and reduced systemic vascular resistance, the latter effect being beneficial to cardiac performance. A further benefit was the decrease in PCWP and, therefore, preload. The heart rate response for each increment in cardiac output was higher with dopamine, as was the mean arterial pressure, implying that myocardial oxygen consumption was greater with dopamine. This would tend to favour the use of dobutamine in patients with pump failure secondary to coronary artery disease. Similar conclusions were reached by Loeb, Bredobis and Gunnar (1977), who infused dopamine and dobutamine at doses producing similar increments in cardiac output and noted the differences in the peripheral effects—PCWP and vascular resistance reduced with dobutamine, but with dopamine 5 μg kg\(^{-1}\) min\(^{-1}\) PCWP increased in eight of 13 patients and in several subjects arterial hypoxaemia, dyspnoea and early pulmonary oedema occurred. In a single crossover trial in 13 patients with heart failure from cardiomyopathy, Leier and colleagues (1978) assessed the dose related haemodynamic effects of both dopamine and dobutamine. Dobutamine produced increases in cardiac output and decreases in PCWP and SVR at infusion rates up to 10 μg kg\(^{-1}\) min\(^{-1}\), whereas with dopamine at rates greater than 4 μg kg\(^{-1}\) min\(^{-1}\) there was an increase in ventricular ectopy and heart rate.

In summary, therefore, both dopamine and dobutamine are powerful inotropic agents, and are more selective in effect than previously available sympathomimetic amines. Each agent has its own pharmacological profile and its selection depends on the haemodynamic characteristics of the individual patient. In the setting of pump failure, especially secondary to coronary artery disease, for a single agent, on the available evidence dobutamine would be the agent of choice (Gunnar and Loeb, 1983).

Vasodilators

Mechanism of action

Vasodilator therapy in pump failure is based on the principle of afterload reduction. Studies on isolated heart muscle have demonstrated that increasing the load against which muscle shortens (the afterload) decreases both the velocity and magnitude of the shortening. Conversely, if the load is reduced, the muscle shortens further and with greater velocity. Translated to the intact heart this explains how a decrease in resistance to left ventricular ejection increases stroke volume and cardiac output.

Systolic arterial pressure is the most easily measured index of left ventricular afterload, but is a very crude approximation. A better assessment is aortic impedance, which reflects both flow and pressure in the aorta but cannot be measured easily in patients, and the nearest approximation is systemic vascular resistance. Reduction in systemic vascular resistance and aortic impedance is the mechanism whereby ventricular performance is improved by vasodilators. A further benefit to be obtained from vasodilator therapy is by increasing left ventricular diastolic compliance and thus reducing the increased filling pressure.

The generalized increase in systemic vascular resistance found in pump failure maintains systemic arterial pressure. Excessive arteriolar constriction and precapillary constriction may reduce tissue flow and oxygen delivery, despite adequate perfusion pressure. Not only is cardiac output further reduced by this increase in afterload, but myocardial oxygen consumption is considerably increased. Despite the obvious benefits of vasodilators in chronic cardiac failure, their use in the management of pump failure remains controversial. The major problems relate to reduction in diastolic arterial pressure leading
to further impairment of coronary perfusion and thus extending ischaemic myocardial damage.

Intravenous vasodilators with a rapid onset of action and a short half-life are indicated, and these include nitroprusside, phentolamine and nitroglycerin. Nitroprusside acts directly on vascular smooth muscle to cause both arterial and venous dilatation (Palmer and Lessiter, 1975). The initial dose is 0.5 \( \mu g \) kg\(^{-1} \) min\(^{-1} \), which is increased gradually if haemodynamic improvement is maintained (Franciosa et al., 1972). Although cardiac output and stroke volume increase, neither short term nor long term prognosis has been improved by vasodilator therapy (Cohn, Mathew and Franciosa, 1974; Chatterjee et al., 1976).

The use of nitroglycerin has been largely restricted to the patient with mild left ventricular dysfunction or uncomplicated myocardial infarction, and no definite conclusions have been reached with respect to prognosis in this group of patients.

The combination of the inotropic action of dobutamine and the vasodilating properties of nitroprusside can be of salutary benefit in improving haemodynamics in the patient with pump failure but, again, there is no evidence that prognosis can be improved.

**Mechanical Circulatory Support**

**Intra-aortic balloon counterpulsation**

The major indications for this procedure are in patients in whom pump failure results from a mechanical problem such as acute mitral regurgitation secondary to papillary muscle dysfunction or rupture of the interventricular septum. It is also particularly appropriate in the patient recovering from cardiopulmonary bypass with reversible depression of the myocardium and in whom recovery of ventricular function is anticipated.

The technique consists of the retrograde insertion of a balloon catheter to the ascending aorta distal to the left subclavian artery. The ECG is used to synchronize the inflation of a 30- or 40-ml balloon during diastole and the deflation of the balloon before left ventricular ejection. The advantages of this technique over pharmacological therapy are augmentation of coronary perfusion by increasing diastolic perfusion pressure, and reduction in afterload improving cardiac output and reducing myocardial oxygen usage.

Complications include lower limb ischaemia, femoral and renal arterial emboli, haemolysis, infection and haemorrhage at the insertion site in the femoral artery.

The results of intra-aortic balloon counterpulsation in pump failure have demonstrated that temporary haemodynamic improvement can be achieved with increases in cardiac output, decrease in PCWP, improved tissue perfusion and increased urine output (Scheidt, Wilner and Mueller, 1973; Weber and Janicki, 1974; Willerson et al., 1975).

Intra-aortic balloon counterpulsation has had little impact on decreasing mortality in patients with pump failure, but has been useful in stabilizing the haemodynamic status before cardiac surgery. Mechanical support should only be utilized if there is a possibility of successful cardiac surgery for the correction of mechanical defects.

**Left ventricular assist devices**

In the patient recovering from cardiopulmonary bypass, in whom recovery of ventricular function is anticipated, the use of left ventricular devices has been advocated. These consist of a pump with outlets to the thoracic aorta and left ventricular apex; thus the patient's left ventricle performs little work and almost the whole left ventricular output is handled by the assist device (Pierce et al., 1981).

**Right Ventricular Infarction**

The haemodynamic and therapeutic features of right ventricular infarction were first recognized by Cohn and colleagues in 1974. In 40–70% of patients with inferior myocardial infarction, there is evidence of right ventricular involvement detected by radionuclide techniques and echocardiography (Wackers et al., 1978). The clinical syndrome of right ventricular infarction which consists of right ventricular dysfunction, accompanied by low cardiac output and hypotension, occurs in only 3–8% of all infarctions (Lorell et al., 1979). Compared with patients with pump failure from left ventricular infarction, these patients have a better prognosis, with a mortality of 40–50%.

Heart block and bradyarrhythmias are common and restoration of atrial transport function using A–V sequential pacing can increase cardiac output and arterial pressure (Love et al., 1984). The addition of dobutamine further increases cardiac output and stroke volume and thus improves prognosis. Clinical recognition and haemodynamic confirmation of right ventricular infarction is thus of the utmost importance in this group of patients.
In summary, pump failure complicating myocardial infarction still carries a mortality of 80–100%, despite advances in haemodynamic monitoring, inotropes, vasodilator therapy and mechanical circulatory assistance. This high mortality is related to extensive loss of contracting left ventricular myocardium, but there is a subset of patients who may benefit from emergency cardiac surgery. The recognition of right ventricular dysfunction is important, as the prognosis in this group of patients is better than with left ventricular dysfunction, although the clinical presentation may be similar.

Any reduction in mortality requires therapy which will limit the ultimate extent of infarcted tissue, thus preventing the development of pump failure. The most promising approach would appear to be the use of thrombolytic therapy to reperfuse the myocardium during the very early stages of developing infarction and thereby prevent the onset of pump failure.

HAEMODYNAMIC CONSEQUENCES OF ARRHYTHMIAS

The impact of arrhythmias on cardiac performance is not only dependent on the effect of the arrhythmia per se, but is also related to the underlying cardiac status. Arrhythmias which may be tolerated in patients with “normal” hearts may have severe deleterious consequences in patients with reduced cardiac function and impaired cardiac reserve. The haemodynamic sequelae are the end-result of the effects of the arrhythmia on various interdependent factors. For discussion purposes, these factors will be dealt with separately.

Heart rate

Heart rate is a major determinant of cardiac function and is probably the main compensatory mechanism in the normal individual for adjustment of cardiac output to meet physiological demands (Braunwald, Sonnenblick and Ross, 1980).

With inappropriate bradycardia, cardiac output can be maintained within certain limits by a compensatory increase in stroke volume. This mechanism may be significantly compromised in patients with impaired myocardial function. This reflex response is dependent to some extent on the acuteness of development of the bradycardia. For example, in chronic complete heart block this mechanism has time to develop and the patient may be asymptomatic at low heart rates, whereas at relatively similar rates in acute heart block, cardiac decompensation may occur.

In tachycardia, the shorter R–R interval is a result of a shortening in the duration of the diastolic filling time, with relative preservation of systolic time. This reduction in diastolic filling time produces a decrease in end-diastolic volume with consequent reduction in cardiac output as determined by the Starling curve. In addition, since coronary flow occurs during diastole, blood supply to the myocardium may be compromised.

As in the case of the response to bradycardia, the limits within which other compensatory mechanisms can be invoked are narrowed, especially if ventricular compliance is abnormal or further limitation to ventricular filling, such as mitral stenosis, co-exists.

A–V synchrony

Optimum cardiac function is dependent on the synchronization of atrial and ventricular contraction (Braunwald and Fram, 1961; Kosowsky et al., 1968). Impairment of this sequencing may be an important component of the resultant haemodynamic response to arrhythmias. Atrial contribution augments ventricular filling, producing an increase in end-diastolic volume and therefore an increase in cardiac output. Although, in normal individuals, this may be of minor importance, in patients with impaired diastolic compliance, such as results from ventricular hypertrophy, loss of atrial transport becomes a significant factor (Rahimtoola et al., 1975).

In tachycardias, for example atrial fibrillation, this deleterious consequence is compounded further by the shortening of ventricular filling time.

Disturbance of the synchronized contraction of the ventricles may also assume importance in patients with underlying heart disease. Some recent studies have suggested that asynchronous ventricular contraction (development of bundle branch block) can influence overall cardiac performance (Rolfe et al., 1984).

Contractility

Intrinsic myocardial contractile function requires an adequate nutrient supply to provide energy stores for the working myocardium. In the setting of disturbed haemodynamic function, disturbance of this factor can further compromise
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Cardiac performance. Nutritional supply to the myocardium is provided by coronary arterial blood flow which, as previously discussed, may be reduced during tachycardia to an extent such that imbalance in myocardial oxygen demand–supply can impair myocardial function (Thadani, Chiong and Parker, 1980).

With co-existing obstructive coronary artery disease, this reduction in blood flow may become critical, with the development of myocardial ischaemia/infarction. In addition to the direct consequences on cardiac performance, such imbalance in myocardial oxygen supply may produce ischaemia-related arrhythmias, with worsening of the overall haemodynamic status.

Indirect effects

As a result of the impairment in cardiac performance secondary to the arrhythmia, disturbances in acid–base balance and electrolyte upset may further compromise cardiac function and worsen the arrhythmia.

Indications for Treatment

In the intensive care situation, there is no definitive plan of treatment that can be applied to a particular arrhythmia. This section provides general guidelines to the management of arrhythmias, but it should be stressed that each case must be individualized to take into consideration the specific problems operative at that time.

The decision to treat an arrhythmia depends on the specific type of arrhythmia; the haemodynamic compromise produced by the arrhythmia; and the potential for the arrhythmia to degenerate into a more malignant type.

The main aims of any therapeutic approach should be not only the conversion of the arrhythmia to sinus rhythm, but also the maintenance of sinus rhythm. In certain cases, this cannot be achieved and amelioration of the haemodynamic consequences may have to be accepted (e.g. control of ventricular response by digoxin in atrial fibrillation).

Before the institution of therapy is considered, attention must be paid to the treatment and correction of provocative factors and underlying disease states, if possible. This includes withdrawal, or dose reduction, of concomitant therapy with drugs which are potentially arrhythmogenic, such as the sympathomimetic amines, dopamine and dobutamine.

The available therapeutic modalities for the treatment of arrhythmias include vagal manoeuvres, drugs, pacing techniques and d.c. cardioversion. D.c. cardioversion is the most effective procedure for the acute termination of sustained tachycardia and is the preferred choice in the setting of hypotension and heart failure.

It should be remembered that all anti-arrhythmic drugs have potentially adverse effects and these must be considered in the selection of therapy. These effects include:

(1) Direct negative inotropic effect (depressant) on myocardial function.

(2) Potential for proarrhythmia with consequent development of different or more malignant arrhythmias (Velebit et al., 1982).

(3) Possibility of interaction with concomitant drug therapy.

In addition, the effects of a changing haemodynamic situation on the metabolism of the anti-arrhythmic drug should be appreciated. Table I summarizes the indications and dose regimens for commonly used anti-arrhythmic drugs.

Sinus tachycardia

Sinus tachycardia usually reflects increased sympathetic drive and occurs commonly in many diverse situations. In most cases it does not compromise cardiac function and settles spontaneously with treatment of the underlying problem. In the intensive care situation, provocative factors are frequently anxiety and pain.

In patients with depressed ventricular function, sinus tachycardia is a compensatory reflex response to maintain cardiac output, and treatment is directed towards improving cardiac performance with diuretics, vasodilators, etc. In the setting of acute myocardial infarction, persistent tachycardia, by increasing myocardial oxygen consumption, may be a factor in extension of the infarct and treatment with a β-adrenoceptor blocker may be beneficial (Sobel et al., 1972; Yusuf et al., 1980). This approach, however, should be used with caution, since this type of therapy may worsen underlying myocardial dysfunction, and preferably haemodynamic monitoring should be used to evaluate the haemodynamic response. Metoprolol, initially 5 mg up to 15 mg i.v. can be used, depending on the response.

Sinus node re-entrant tachycardia and the non-paroxysmal form of sinus tachycardia will not be discussed, since they are uncommon in this context.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>I.v. dose regimens</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Amiodarone</td>
<td>Atrial tachycardia</td>
<td>300 mg infused over 30 min followed by an infusion of up to 1000 mg over 24 h</td>
<td>Preferably administered through a central venous catheter because of risk of phlebitis</td>
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<td>Re-entrant junctional tachycardia</td>
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<td></td>
<td>Atrial flutter</td>
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<td></td>
<td>Atrial fibrillation</td>
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<td></td>
<td>Ventricular tachycardia</td>
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<tr>
<td>Digoxin</td>
<td>Atrial flutter*</td>
<td>0.5–1.0 mg by slow i.v. injection or infusion</td>
<td>Maintenance dose reduction in renal failure</td>
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<tr>
<td></td>
<td>Atrial fibrillation*</td>
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<tr>
<td>Disopyramide</td>
<td>Atrial tachycardia</td>
<td>2 mg kg¹ over 5 min to a maximum of 150 mg. Maintenance infusion up to 800 mg/24 h</td>
<td>May precipitate heart failure</td>
</tr>
<tr>
<td></td>
<td>Re-entrant junctional tachycardia</td>
<td></td>
<td>May cause urinary retention</td>
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<tr>
<td></td>
<td>Atrial fibrillation</td>
<td></td>
<td>Dose reduction in renal failure</td>
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<tr>
<td>Flecaainide</td>
<td>Atrial tachycardia</td>
<td>For rapid effect, i.v. bolus of 2 mg kg¹ over 10 min. 30 min indicated in less acute states or if cardiac failure present. Maximum bolus dose is 150 mg I.v. infusion of 1.5 mg kg¹ in 1st hour followed by 0.25 mg h¹</td>
<td>May precipitate heart failure</td>
</tr>
<tr>
<td></td>
<td>Re-entrant junctional tachycardia</td>
<td></td>
<td>Should not be administered if A-V block unless pacemaker back-up available</td>
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<tr>
<td></td>
<td>Atrial fibrillation</td>
<td></td>
<td>Dose reduction in renal failure</td>
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<td></td>
<td>Ventricular tachycardia</td>
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<tr>
<td>Lignocaine</td>
<td>Ventricular tachycardia</td>
<td>100-mg bolus i.v. followed by an infusion of 4 mg min⁻¹ for 30 min. 50-mg bolus repeated after 15 min if required. Infusion rate reduced to 2–3 mg min⁻¹ maintenance</td>
<td>Should not be administered if A-V block (see above)</td>
</tr>
<tr>
<td>Metroprolol</td>
<td>Sinus tachycardia</td>
<td>5-mg bolus—repeated up to a maximum of 15 mg</td>
<td>May precipitate hypotension and heart failure</td>
</tr>
<tr>
<td></td>
<td>Re-entrant junctional tachycardia</td>
<td></td>
<td>Concomitant verapamil therapy a relative contraindication</td>
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<td>Atrial fibrillation*</td>
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<tr>
<td>Ouabain</td>
<td>Atrial flutter*</td>
<td>0.25–0.5-mg bolus or infusion. 5-mg bolus repeated if necessary</td>
<td>See digoxin</td>
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<td></td>
<td>Atrial fibrillation*</td>
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<tr>
<td>Verapamil</td>
<td>Multifocal atrial tachycardia</td>
<td>5-mg bolus repeated if necessary after 5–10 min up to maximum of 15–20 mg</td>
<td>May precipitate hypotension and heart failure</td>
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<td></td>
<td>Re-entrant junctional tachycardia</td>
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<td>Concomitant β-adrenoreceptor blocker therapy a relative contraindication</td>
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<td>Atrial flutter*</td>
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**Ectopic atrial rhythm**

Ectopic atrial rhythm describes a rhythm which is similar to sinus in respect of rate, but the P wave on the surface ECG has a different morphology, depending on the intra-atrial site of the focus. It usually produces no haemodynamic embarrassment and does not require treatment.

**Atrial premature beats**

Atrial premature beats usually are haemodynamically insignificant and treatment is not indicated. However, if the premature beat acts as a trigger factor for sustained tachycardias such as supraventricular tachycardia or atrial flutter/fibrillation, suppressive therapy is required either
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Atrial tachycardia is usually the result of abnormal automaticity of an ectopic focus and in adults is frequently observed in toxic states. Medical therapy tends to be ineffective, although amiodarone and type I agents including flecainide have been successful in a proportion of patients (Creamer, Nathan and Camm, 1985). In the presence of digoxin toxicity, atrial tachycardia is frequently associated with A–V block (PAT with 2:1 A–V block) (fig. 1).

A particular form of automatic atrial tachycardia is multifocal atrial tachycardia, which occurs not uncommonly in patients with underlying chronic lung disease, especially during acute exacerbations of their illness (Shine, Kastor and Yurchak, 1968; Wang et al., 1977). It has been suggested that this type of tachycardia may be related in part to the use of theophylline-like drugs (Levine, Michael and Guarnieri, 1985a).

It is characterized electrocardiographically by varying P–P, P–R and R–R intervals, with the P wave of varying morphology (fig. 2). If the tachycardia is fast, it may be extremely difficult to differentiate this rhythm from atrial fibrillation, which is important, since multifocal tachycardia is frequently resistant to anti-arrhythmic therapy including digoxin. If not recognized, there is a tendency to prescribe increasing doses of digoxin with the possibility of development of digoxin toxicity. With treatment of the underlying illness and correction of blood-gas tensions, acid–base balance and electrolyte upset, the tachycardia...
tends to terminate spontaneously. Recently, it has been shown that verapamil administered i.v. may be effective in terminating this tachycardia (Levine, Michael and Guarnieri, 1985b). Pacing techniques and cardioversion are not usually of benefit, since the rhythm is suppressed only transiently.

**Non-paroxysmal junctional tachycardia.** In general clinical practice the most common form of junctional tachycardia is that related to digoxin toxicity. The rate of tachycardia is normally less than 130 beat min\(^{-1}\) and is dissociated from the atrial activity. Although the tachycardia is regular, exit block can occur, especially of the Wenckebach type, producing an irregular rhythm. On closer inspection, however, it can be seen that there is a regularity to the varying cycles (allorhythmia, group-beating) which reveals the true nature of the arrhythmia. Its recognition is important, not only because of the possibility of haemodynamic compromise, but also because it may be a portent for the development of more malignant digoxin-toxic arrhythmias. With withdrawal of digoxin and correction of hypokalaemia, if present, the tachycardia normally subsides spontaneously.

**Re-entrant supraventricular (junctional) tachycardia.** The most common underlying mechanism for paroxysmal supraventricular tachycardia is re-entry (Josephson, 1978; Wu et al., 1978). The requirements for re-entry to occur are the presence of two electrophysiologically distinct pathways: slowed conduction in one pathway and antegrade block in the other. Under these conditions, if a premature beat is conducted down the unblocked pathway sufficiently slowly to allow recovery of the blocked pathway, the impulse can return up this pathway to complete a re-entrant circuit and initiate a tachycardia.

The implications of this mechanism are that premature beats are usually the initiating factor, and that a critical balance in the two limbs of the circuit are necessary for the tachycardia, for both initiation and sustenance.

The most common re-entrant tachycardias are A-V nodal re-entrant tachycardia (AVNRT) and A-V re-entrant tachycardia (AVRT). In the former, the re-entrant circuit is within the A-V node itself, and in the latter the re-entrant circuit involves the A-V node in one limb and an accessory atrioventricular band of myocardium bypassing the A-V node as the other limb. In the majority of cases of A-V re-entrant tachycardia, the impulse uses the accessory pathway in the retrograde direction with the A-V conducting system anterogradely and therefore both these tachycardias have a normal narrow QRS morphology (fig. 3). The presence of an accessory pathway cannot be diagnosed during the tachycardia but, in sinus rhythm, because of impulse conduction bypassing the normal delaying properties of the A-V node, ventricular pre-excitation may be present. This is recognized by
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FIG. 3. Re-entrant (junctional) tachycardia with narrow QRS complex.

the short P–R interval with widened QRS complex resulting from a delta wave seen in the Wolff–Parkinson–White syndrome. Not uncommonly, however, the accessory pathway cannot conduct anterogradely (therefore gives no evidence of pre-excitation in the ECG) and the participation of an accessory pathway is revealed only by electrophysiological study (Pritchett et al., 1978).

These re-entrant tachycardias usually have a rate of 170–220 beat min⁻¹ and, in normal individuals, palpitation may be the only symptom. In patients with compromised ventricular function, these tachycardias may precipitate hypotension and cardiac failure.

Since in both these tachycardias the A–V node is part of the circuit, vagal manoeuvres may be very effective in terminating them. These manoeuvres include carotid sinus massage, Valsalva and diving reflex. The effect of these manoeuvres can be enhanced by elevating the lower limbs.

Similarly, anti-arrhythmic drugs which slow impulse conduction through the A–V node can perturb the circuit sufficiently to terminate the tachycardia. Intravenous verapamil is probably the most effective agent for the acute termination of these tachycardias, although it should be used with caution in patients with evidence of cardiac decompensation. This adverse feature is worsened in the presence of β-adrenoceptor blockers and in this situation probably verapamil should not be used (Kieval et al., 1982). Both digoxin and β-adrenoceptor blockers also act by their effect on the A–V node and the latter, especially, are effective when administered i.v.

The alternative pharmacological approach is to use drug therapy which perturbs the retrograde limb of the re-entrant circuit. Despite the intranodal site of this limb in AVNRT, it responds similarly to the accessory pathway. In this case, the Type I anti-arrhythmic agents, procainamide, disopyramide and flecainide, can be used i.v. Prophylactic therapy may not be required in the first instance unless the arrhythmia is life threatening or produces severe haemodynamic effects. If it is required, the agents used for termination can also be used for prophylaxis. If
the patient is already receiving prophylactic therapy, it is rational to use for acute termination a therapy the main action of which is on the opposite limb of the circuit.

In circumstances in which the patient is suffering recurrent episodes and anti-arrhythmic therapy is either ineffective or more particularly producing adverse effects, pacing techniques can be used. A standard transvenous pacing line can be positioned in the right atrium and the tachycardia frequently terminated by overdrive pacing. This involves pacing the atrium at a rate faster than the tachycardia until it captures and then switching off. A potential complication of this approach is the induction of atrial fibrillation, although frequently this will terminate spontaneously.

Although more sophisticated pacing techniques using programmed extrastimulation can also be utilized, this requires specialized expertise and equipment and rarely applies to the intensive care situation.

**Atrial flutter**

During acute illnesses, atrial flutter is not an uncommon arrhythmia. Generally, in atrial flutter, the heart rate is about 150 beat min⁻¹ because of physiological 2:1 A–V block. In toxic states or the presence of drug therapy, the rate may be much more rapid especially if a 1:1 A–V response occurs.

Vagal manoeuvres do not terminate the arrhythmia, but may increase the A–V block with transient slowing of the ventricular response which may facilitate the ECG diagnosis of this arrhythmia (fig. 4).

Attempts at acute termination of atrial flutter with drug therapy are frequently disappointing, although amiodarone i.v. may be effective (Faniel and Schoenfeld, 1983).

As with re-entrant supraventricular tachycardia, overdrive pacing is frequently very effective in terminating atrial flutter (Waldo et al., 1977). Since the atrial rate in atrial flutter is usually about 300 beat min⁻¹, overdrive atrial pacing at rates faster than this is required. This approach is particularly beneficial if the arrhythmia is recurrent.

Atrial flutter is cardioverted easily with low energies of 25–50 J and d.c. cardioversion is the first choice treatment in many patients.

The alternative approach to the treatment of this arrhythmia is to accept the underlying rhythm but improve the haemodynamic state by reducing the ventricular response. Either i.v. digitalis or verapamil may be used. In this situation i.v. ouabain, which has a shorter onset of action than digoxin, may also be used. For longer term therapy, intermittent boluses of these compounds may be administered or oral digoxin or verapamil given.

For oral prophylaxis to maintain sinus rhythm, the type I agents quinidine and disopyramide are effective, but since both these agents may increase A–V nodal conduction, digoxin should be prescribed in addition to prevent 1:1 A–V response if atrial flutter breaks through (Robertson and Miller, 1980). Alternatively, amiodarone by mouth can be used, and with this agent digoxin is not required. In general, however, because of the potential serious side-effects of amiodarone, including pulmonary toxicity, amiodarone is reserved for refractory cases (Zipes, Prystowsky and Heger, 1984).

**Atrial fibrillation**

Atrial fibrillation is one of the most frequent arrhythmias encountered in intensive care. It is frequently transient and caused by electrolyte or acid–base imbalance and responds to the correction of these or treatment of underlying infection. As previously discussed, it has to be differentiated from multifocal atrial tachycardia.

The approach to treatment is similar to atrial flutter, although pacing techniques are ineffective and higher energies for d.c. cardioversion are required. Since the ventricular rate is usually 170–180 beat min⁻¹ and atrial transport is lost,
haemodynamic deterioration may quickly super-
vene, necessitating d.c. cardioversion.

In most situations, a digitalis preparation is used
to control the ventricular response until sponta-
eous cardioversion occurs. In a number of cases
the rhythm can be converted to sinus by i.v.
amiodarone (Faniel and Schoenfeld, 1983). More
recently, i.v. flecainide has also been shown to be
effective in the termination of this rhythm
(Hellestrand et al., 1985). Occasionally, atrial
fibrillation develops because of the acute onset of
heart failure, for example in myocardial infarction,
and the arrhythmia subsides with treatment of the
heart failure with i.v. diuretic therapy.

The presence of continuing atrial fibrillation
should raise the possibility of underlying rheumatic
heart disease or thyrotoxicosis, and these conditions
should be excluded. The development of regularity
of the ventricular response on digoxin suggests the
onset of a junctional rhythm with block (see
nonparoxysmal junctional tachycardia) related to
toxicity and appropriate measures should be taken
(fig. 5). If atrial fibrillation persists, anticoagulation
to prevent systemic embolization should be
considered.

Ventricular premature beats

During the acute phase of myocardial infarction,
ventricular premature beats occur in almost every
patient. Similarly, in acute illnesses or toxic states,
ventricular premature beats (VPB) are frequently
seen, especially if there is hypokalaemia or
acidosis, or both.

Unless the premature beats are very frequent or
occur in bigeminy with underlying bradycardia,
they do not provide haemodynamic embarrass-
ment, and therefore do not require treatment for
this reason.

The prophylactic use of anti-arrhythmic therapy
to suppress VPB in the acute situation is more
controversial. It was previously considered that
frequent VPB, complex forms including R-on-T
and non-sustained ventricular tachycardia were
"warning arrhythmias" and their presence indi-
cated the likelihood of progression to more lethal
types (Lown et al., 1967). In addition, it was
suggested that suppressing these warning ar-
rhythmias using drug therapy prevented the
development of ventricular tachycardia/fibrilla-
tion. Several studies have, however, not confirmed
this precept, except possibly for VPB of the
R-on-T type (El-Sherif et al., 1976; Campbell,
Murray and Julian, 1981). In many intensive care
units prophylactic anti-arrhythmic therapy to
prevent ventricular tachycardia/fibrillation is
provided only for these R-on-T VPB or in the
situation where the patient has already sustained
an episode of ventricular tachycardia/fibrillation.
The presence of VPB otherwise indicates the need

Fig. 5. Development of junctional rhythm in a patient with atrial fibrillation receiving digoxin therapy.
The typically irregularly irregular response progresses to a slow regular response.
for increased surveillance and correction of any precipitating factor.

The most common anti-arrhythmic agent for the suppression of VPB remains lignocaine. Initial administration is by i.v. bolus, followed by an infusion with a supplementary bolus after 20 min. To obtain adequate blood concentrations, an infusion of 3—4 mg min⁻¹ is required and at this rate side effects of confusion, drowsiness and tremor are not uncommon. Because of the hepatic metabolism of lignocaine, the dose should be reduced if there is hypotension or cardiac failure (Stenson, Constantino and Harrison, 1971). If this is ineffective, i.v. disopyramide or procainamide may be considered. Recent studies have also shown that i.v. flecainide is effective in this situation (Hellestrand et al., 1985).

If treatment with these agents fails, i.v. amiodarone should be used, preferably via a central venous catheter, to prevent phlebitis. An initial dose is infused over 30 min, followed by a slower infusion over 24 h (Morady et al., 1983; Mostow et al., 1984).

If maintenance therapy is required for longer than the acute illness, disopyramide, tocainide, mexiletine or flecainide can be prescribed. If oral amiodarone is indicated, a loading dose for 7—14 days is given before reducing to the long-term maintenance dose.

**Ventricular tachycardia**

Although haemodynamic deterioration frequently supervenes with the development of sustained ventricular tachycardia, it should be stressed that this is not always the case. There is a tendency in such situations mistakenly to diagnose supraventricular tachycardia with aberrancy (Wellens et al., 1981). On the surface ECG, a broad complex tachycardia is more likely to be ventricular in origin if the QRS complex is > 0.14 s, the morphology of the QRS is of left bundle branch type, or there is an RS, QR, or qR pattern in V₁ or Q wave in V₆.

If A-V dissociation fusion complexes and captured atrial beats are observed, the diagnosis is almost definitely ventricular tachycardia (fig. 6). Other wide complex tachycardias are very uncommon and, although the surface ECG may provide pointers to the diagnosis, detailed electrophysiological assessment may be required (Miles et al., 1984). These are outwith the scope of this article and will not be discussed further.

The approach to treatment of this tachycardia is entirely dependent on the haemodynamic status. If the tachycardia is being tolerated, anti-arrhythmic drug therapy can be used, using the same guidelines as for the treatment of VPB. If haemodynamic decompensation occurs, d.c. cardioversion should be undertaken, with prophylactic therapy administered thereafter. Occasionally, ventricular tachycardia may be difficult to terminate until the correction of acid-base imbalance or electrolyte upset.

Pacing techniques, including overdrive ventricular pacing and ventricular extrastimulation, can be very effective especially if the tachycardia does not produce immediate haemodynamic collapse, but they require specific expertise and equipment not generally available.

**Idioventricular rhythm**

Not infrequently in acute myocardial infarction, idioventricular rhythm occurs because of slowing of the sinus rate and suppression of junctional pacemakers (fig. 7). Generally, the rate of this rhythm is 50—60 beat min⁻¹, but may occasionally be as fast as 100—110 beat min⁻¹. This has been termed accelerated idioventricular rhythm or "slow VT". It usually is transient, produces no haemodynamic upset and does not require treatment. Intravenous atropine, by accelerating the sinus rate, will terminate this type of arrhythmia.

**Torsade de Pointes**

It is important to distinguish this arrhythmia from ventricular tachycardia. It is characterized by...
Fig. 7. Idioventricular rhythm in a patient with acute myocardial infarction. The upper two tracings demonstrate sinus rhythm and fusion complexes, with a 3-beat run of idioventricular rhythm in the bottom tracing.

Fig. 8. Torsade de Pointes. Spontaneously terminating runs of a fast polymorphic tachycardia with the "twisting" of the QRS axis.
the continual change in axis of the QRS complex through 180° (fig. 8). It is usually rapid, but frequently self-terminating. It occurs with a prolonged Q–T interval and, although there are congenital causes, is more commonly secondary to drug therapy, especially with the type I agents such as quinidine (Jackman et al., 1984).

Treatment of Torsade de Pointes is directed towards shortening the Q–T interval. Anti-arrhythmic drug therapy is withheld and hypokalaemia is corrected. In certain cases, i.v. magnesium sulphate is effective in terminating this arrhythmia (Tzivoni et al., 1984). The tachycardia can also be terminated and suppressed by an i.v. infusion of isoprenaline. The more effective management and treatment of choice is transvenous pacing. If A–V conduction is normal, atrial pacing is preferred because it shortens the Q–T interval more than ventricular pacing. In the case of Torsade secondary to correctable factors, pacing is continued until these factors have been treated.

**Ventricular fibrillation**

The treatment of ventricular fibrillation is d.c. cardioversion, because of the almost immediate development of profound cardiovascular collapse. Occasionally, consciousness can be preserved for a short period by getting the patient to cough. The longer the patient is in ventricular fibrillation before cardioversion, the more difficult it is to terminate and maintain sinus rhythm, because of the rapid development of metabolic disturbance.

If the patient remains in ventricular fibrillation despite cardioversion and correction of acidosis and other provocative factors, i.v. lignocaine should be administered. Once terminated, maintenance lignocaine is usually prescribed.

**Bradyarrhythmias**

Sinus bradycardia usually does not require treatment unless complicated by hypotension, in which case it will respond to i.v. atropine. If repeated doses are required, temporary cardiac pacing should be considered.

Occasionally, ventricular tachyarrhythmias occur as an escape phenomenon if the heart rate is quite slow and, in this case, cardiac pacing by increasing the intrinsic heart rate suppresses these arrhythmias.

In general, because of case of placement and stability of position, transvenous pacing is performed at the right ventricular apex. If, however, there is continuing haemodynamic upset with ventricular pacing, atrial pacing can be used, thus preserving atrial transport (Reiter and Hindman, 1982). This may be of particular importance in patients with right ventricular dysfunction, which can occur in inferior myocardial infarction (Love et al., 1984). Obviously, in patients with A–V block, atrial pacing alone is ineffective and temporary A–V sequential pacing with electrode catheters in both atrium and ventricle is required.

If sinus bradycardia is related to overdosage of β-adrenoceptor blocking therapy, and requires therapy, glucagon or the β-adrenoceptor agonists dopamine, dobutamine or isoprenaline can be used. This may be supplemented by cardiac pacing if required.

**A–V block.** In general, the treatment of A–V block is determined by the haemodynamic consequences. In many cases treatment is not required. Cardiac pacing is the treatment of choice if indicated. Recently, oesophageal pacing techniques have been introduced and for physicians not skilled in transvenous pacing this may be an alternative in the emergency situation until a stable transvenous system can be inserted (Gallagher et al., 1982). In some situations, an isoprenaline infusion can “buy time” until a pacemaker is inserted, although the patient’s rhythm must be carefully monitored in case ventricular arrhythmias occur.

**REFERENCES**


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