ACUTE MYOCARDITIS

Viral infection is the most common cause and the main culprits are the Coxsackie viruses and influenza viruses A and B. Myocarditis may occur several weeks after the initial viral infection. Myocarditis may be heralded by a 'flu'-like illness.

ECG changes are common but non-specific. Biochemical markers of myocardial injury (e.g. troponin I and T, creatine kinase) are elevated in proportion to the extent of damage.

Echocardiography may reveal left ventricular dysfunction which is sometimes regional (due to focal myocarditis),

In most patients the disease is self-limiting and the immediate prognosis is excellent. However, death may occur, due to a ventricular arrhythmia or rapidly progressive heart failure. Myocarditis has been reported as a cause of sudden and unexpected death in young athletes. There is strong evidence that some forms of myocarditis may lead to chronic low-grade myocarditis or dilated cardiomyopathy.

In most cases only supportive therapy is available. Treatment for cardiac failure or arrhythmias may be required and patients should be advised to avoid intense physical exertion because there is some evidence that this can induce potentially fatal ventricular arrhythmias. Clinical trials have failed to demonstrate any benefit from treatment with corticosteroids.

CARDIOMYOPATHY

DILATED CARDIOMYOPATHY

This condition is characterised by dilatation and impaired contraction of the left (and sometimes the right) ventricle; left wall thickness is normal or reduced.

The differential diagnosis includes coronary artery disease . Alcohol is an important aetiological factor. At least 25% of cases are inherited as an autosomal dominant trait. A late autoimmune reaction to viral myocarditis is thought to be an aetiological factor.

Arrhythmia, thromboembolism and sudden death are common and may occur at any stage; sporadic chest pain is a surprisingly frequent symptom. The ECG usually shows non-specific changes but echocardiography is useful in establishing the diagnosis.

Treatment is aimed at controlling the resulting heart failure. Some patients may be considered for implantation of a cardiac defibrillator and/or cardiac resynchronisation therapy.

HYPERTROPHIC CARDIOMYOPATHY

This is common form of cardiomyopathy, , and is characterised by inappropriate left ventricular hypertrophy with malalignment of the myocardial fibres. The hypertrophy may be generalised or confined largely to the interventricular septum (asymmetric septal hypertrophy)or other regions (e.g. apical hypertrophic cardiomyopathy, a variant which is common in the Far East).

Heart failure may develop because the stiff non-compliant ventricles impede diastolic filling. Septal hypertrophy may also cause dynamic left ventricular outflow tract obstruction (hypertrophic obstructive cardiomyopathy, or HOCM) and mitral regurgitation due to abnormal systolic anterior motion of the anterior mitral valve leaflet. Effort-related symptoms (angina and breathlessness), arrhythmia and sudden death are the dominant clinical problems.

The condition is a genetic disorder with autosomal dominant.

Symptoms and signs are similar to those of aortic stenosis, except that in hypertrophic cardiomyopathy the character of the arterial pulse is jerky

clinical deterioration is often slow. Sudden death typically occurs during or just after vigorous physical activity; indeed, hypertrophic cardiomyopathy is the most common cause of sudden death in young athletes. Ventricular arrhythmias are thought to be responsible for many of these deaths.

RISK FACTORS FOR SUDDEN DEATH IN HYPERTROPHIC CARDIOMYOPATHY

- A history of previous cardiac arrest or sustained ventricular tachycardia
- Recurrent syncope
- An adverse genotype and/or family history
- Exercise-induced hypotension
- Multiple episodes of non-sustained ventricular tachycardia on ambulatory ECG monitoring
- Marked increase in left ventricular wall thickness

Beta-blockers and the rate-limiting calcium antagonists (e.g. verapamil) can help to relieve angina, there is no pharmacological treatment that is definitely known to improve prognosis. Arrhythmias are common and often respond to treatment with amiodarone. Outflow tract obstruction can be improved by partial surgical resection (myectomy) or by iatrogenic infarction of the basal septum (septal ablation) using a catheter-delivered alcohol solution. An implantable cardiac defibrillator (ICD) should be considered in patients with clinical risk factors for sudden death. Digoxin and vasodilators may increase outflow tract obstruction and should be avoided.

RESTRICTIVE CARDIOMYOPATHY

In this rare condition, ventricular filling is impaired because the ventricles are stiff. This leads to high atrial pressures with atrial hypertrophy, dilatation and later atrial fibrillation. Amyloidosis is the most common cause of restrictive cardiomyopathy.