PREOPERATIVE ASSESSMENT OF THE PATIENT WITH CARDIOVASCULAR DISEASE

P. Foëx

The morbidity and mortality of anaesthesia and surgery are increased by cardiac diseases. It is essential, therefore, to recognize the presence of cardiovascular diseases and to assess their severity in order to prevent serious complication during anaesthesia and the period after operation. Relatively silent cardiac or vascular diseases are often first diagnosed during the preoperative visit (Kyei-Mensah and Thornton, 1974; Frost, 1976). Where the diagnosis has already been made it is important to evaluate the severity of heart disease, to determine whether or not medical treatment could further improve cardiac function (Howat, 1971; Friedlander, 1973) and to consider the pathophysiology of the disease. The optimal anaesthetic management and the decision on invasive monitoring rest on the correct diagnosis of the disease and of its repercussions on the circulation. Detailed medical history and careful clinical examination, supplemented by electrocardiogram and chest x-ray, form the basis of evaluation before operation and may have to be complemented by echocardiography, cardiac catheterization and angiography.

ISCHAEMIC HEART DISEASE

Coronary heart disease causes more than 160,000 deaths each year in the United Kingdom. The frequency of coronary artery disease is very high and the disease may not be clinically apparent unless several vessels are obstructed. Cardiac function may be impaired in almost asymptomatic patients. Presence of coronary heart disease causes a two- to three-fold increase of perioperative mortality and a 10-fold increase in coronary occlusion after surgery. The risk of postoperative infarction is particularly high when surgery takes place within the first 2 months of infarction, reinfarction is common and its mortality is about 60%. Reinfarction is more frequent after thoracic or abdominal surgery, prolonged operation and after episodes of intraoperative hypo- and hypertension (Steen, Tinker and Tarhan, 1978). These factors had already been identified in 1964 by Chamberlain and Edmonds-Seal in their study of electrocardiographic abnormalities after operation. The long-term prognosis of infarction is worse in patients who have developed arterial hypertension, atrial or ventricular arrhythmias or transient renal failure (Luria et al., 1976); their operative risk also is likely to be increased.

Coronary heart disease must be suspected even in the absence of symptoms when predisposing factors are noted. The risk of developing major coronary events is increased two-fold by smoking, four-fold by the combination of smoking and hypertension, and eight-fold when smoking, hypertension and hyperlipidaemia are all present (Stamler and Epstein, 1972). Diabetes, occlusive vascular events and family history of ischaemic heart disease are other important predisposing factors.

The diagnosis of coronary heart disease rests on a history of angina pectoris or electrocardiographic evidence of myocardial ischaemia or infarction. Angina, described as a retrosternal, painful sensation of pressure, constriction or tightness, radiating towards left arm, lower jaw or throat, is easily recognized. The frequency and duration of attacks should be noted. An increased frequency or duration of angina or a reduced exercise tolerance indicate the development of unstable angina and the operative risks may be similar to those of patients with recent infarction.

Between anginal attacks, clinical examination may reveal a fourth heart sound, an enlarged left ventricle and a weak apex contraction reflecting reduced wall motion. Signs of left ventricular failure may be observed.

P. Foëx, M.A., D.Phil., D.M.(Geneva), Nuffield Department of Anaesthetics, Radcliffe Infirmary, Woodstock Road, Oxford.

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The electrocardiogram can be completely normal between attacks of angina unless the patient has suffered previous myocardial infarction. However, in 80% of patients it is abnormal. Nonspecific abnormalities such as signs of left ventricular hypertrophy, bundle branch blocks and atrio-ventricular conduction disorders must be taken seriously; they may be caused by coronary artery disease. Specific abnormalities include pathological Q waves, abnormal ST segments and J point (fig. 1) (Chaitman et al., 1979) and ischaemic alterations of T waves, and confirm the diagnosis. The unexpected regression of R waves in the precordial leads may indicate the presence of non-transmural infarction and is not always recognized. Acute ischaemic alterations of the electrocardiogram suggesting myocardial infarction can be observed in the absence of symptoms: silent infarction is far from exceptional in patients with vascular disease and increases the operative risks to unacceptable levels. Comparison of sequential e.g. tracings is important to detect recent myocardial ischaemia and also to identify persistent ST segment elevations associated with ventricular aneurysms. Further evaluation of cardiac function, for example by echocardiography, is necessary when ventricular aneurysm is suspected. Arrhythmias must be recognized. In the context of ischaemic heart disease, ventricular premature beats are associated with sudden death (Kotler et al., 1973), left ventricular dysfunction (Sharma, Ballantyne and Goldstein, 1974) and reduced ejection fraction (Schultze et al., 1975), their treatment should be considered before surgery.

When either the medical history is suggestive of coronary heart disease or predisposing factors are recorded but the electrocardiogram is normal, it is often suggested that confirmation of the diagnosis may be obtained using an exercise e.g. The test is considered positive if ST segment depression greater than 0.1 mV develops. The test is considered both specific and sensitive, but carries a risk and must be discontinued if angina develops. However, when compared with coronary angiography, the exercise test may give false positive as well as false negative results (Borer et al., 1975). Because of these limitations, it is often possible to evaluate the severity of ischaemic heart disease only in terms of frequency of attacks of angina, of limitation of physical activity and a history or evidence of myocardial infarction. It must be stressed that the persistence of angina after myocardial infarction increases postoperative morbidity and mortality respectively two- and four-fold by comparison with those of patients with well compensated infarction (Ponka, 1977).

Abnormalities of wall motion (hypokinesia, akinesia and dyskinesia) are frequently observed in patients with coronary heart disease and can be detected by ventriculography (fig. 2) and by non-invasive techniques (Swan, 1979). In up to 60% of patients minor abnormalities of wall motion can be detected at rest, while in up to 86% of patients more pronounced ventricular wall dysfunction occurs during exercise (Lindsay et al., 1980) and the ejection fraction is markedly reduced. The non-invasive techniques rely on radionuclides (radionuclide cineangiography), ultrasonics (bi-dimensional echocardiography) and magnetic inductance (cardiokymography), and are of proven value (Borer et al., 1977; Righetti et al., 1980; Silverberg et al., 1980). With these techniques systematic studies of wall function will become easier and will contribute greatly to the assessment of patients with coronary disease. This may become even more relevant if experimental studies showing that anaesthetic agents can cause dysfunction of myocardium supplied by a narrowed coronary artery (Lowenstein et al., 1979; Cutfield et al., 1980; Francis et al., 1980) are confirmed in man.
Assessment before operation must include a review of the patient's medication. The problems associated with digitalis and adrenergic beta-receptor antagonists will be discussed separately. Calcium antagonists have been introduced relatively recently in the treatment of coronary heart disease, particularly where coronary artery spasm appears to play an important role. They are inhibitors of inward calcium currents of smooth muscle and cardiac muscle cells. They cause peripheral and, most importantly, coronary vasodilatation, but they limit the amount of calcium made available for interaction with the contractile proteins and, therefore, reduce myocardial contractility and myocardial oxygen demand. There are few data on the interactions between calcium antagonists and anaesthesia (Patschke et al., 1977), but the reduction of vascular resistance accompanied by myocardial depression may cause hypotension.

Anaemia is another risk factor. It causes a hyperdynamic state of the circulation accompanied by a large increase in myocardial oxygen consumption. Oxygen supply to the myocardium is adequate only as long as coronary blood flow is increased. In the case of narrowed coronary vessels, anaemia may cause wall dysfunction (Hagl et al., 1977) and its presence increases the risk of complications.

In ischaemic heart disease, autoregulation of the coronary circulation is impaired by the narrowing of the coronary vessels. The major contributing factors to imbalance of oxygen supply and oxygen demand and thus to myocardial ischaemia, are:

1. hypotension (reduction of flow through narrowed vessels can be greater than the reduction of oxygen demand);
2. hypertension (increased demand cannot be met by similar increases of flow);
3. tachycardia (demand is increased and supply is reduced because of the shorter duration of diastole);
4. left ventricular overload (increased oxygen demand as a result of higher ventricular diastolic pressure, while oxygen supply is decreased because of the reduction of the coronary perfusion pressure, the difference between aortic diastolic and ventricular diastolic pressure).

HYPERTENSIVE HEART DISEASE

Systemic arterial pressure in the adult represents a continuum without sharp dividing lines between normo- and hypertension. However, it is generally agreed that values of arterial pressure greater than 150 mm Hg systolic and 90 mm Hg diastolic on three successive occasions establish the diagnosis of arterial hypertension. The frequency of arterial hypertension varies between 13% and 41% of the population (National Health Survey, 1964; Tibblin, 1972) and the frequency of cardiovascular complications is proportional to the level of hypertension (Anderson, 1978). Severe arterial hypertension (greater than 180/100 mm Hg) is observed in as many as 11% of surgical patients (Kyei-Mensah and Somanathan, 1976).
Straightforward determination of cuff arterial pressure is easy, yet approximately 50% of hypertensive patients are not diagnosed, 50% of those diagnosed are untreated and 50% of those treated are inadequately controlled. Thus, arterial hypertension remains one of the commonest causes of cardiovascular deaths. Approximately 60% of hypertensive patients die from acute myocardial infarction, 30-40% of a cerebrovascular accident and about 10% from other causes (Hollander, 1973; Bulpitt et al., 1979).

The symptoms and signs of arterial hypertension relate to involvement of the main target organs: heart, brain and kidneys, and depend upon the primary or secondary nature of the disease.

The cardiac complications of arterial hypertension are coronary artery disease and left ventricular hypertrophy. Clinical examination may reveal an enlarged heart and signs of left ventricular failure. The chest x-ray may confirm left ventricular hypertrophy and may reveal aortic calcifications often associated with coronary artery lesions. The electrocardiogram will confirm the presence of left ventricular hypertrophy with or without strain. Disorders of intraventricular conduction, signs of myocardial ischaemia and of myocardial infarction are frequently observed.

The cerebral complications of arterial hypertension include transient ischaemic attacks and major cerebrovascular accidents; they will be discussed separately.

Impairment of renal function is more difficult to assess clinically. A history of nocturia may indicate chronic renal involvement, but there may be no sign of advanced renal disease until uraemia develops. Blood urea and creatinine concentrations are significantly increased only when 70% of total renal function has been lost. Serum electrolytes, urea and creatinine should be measured together with creatinine clearance. Careful urinalysis is also necessary, with close attention to proteinuria, abnormal urinary sediment and inability to concentrate urine.

The retina is another target organ. Diminution of the calibre of small vessels, segmental constriction of the arterioles and arteriovenous nipping are all observed in hypertensive patients. Flame-shaped haemorrhages and glistening white exudates indicate a more advanced stage, while papilloedema may herald the final stage of the disease.

About 90% of hypertensive patients suffer from primary or essential hypertension and only fewer than 10% of secondary hypertension. However, enquiry should be directed to determine the nature of arterial hypertension, since the latter will influence the anaesthetic management of the patient. Renal causes fall into three categories: renal parenchymal diseases, renovascular disease, and other severe renal diseases. A full evaluation of renal function must be obtained. It must be remembered that renal function will be further impaired during the period after operation and that fluid and electrolyte balance will be particularly difficult to maintain. An important hazard in these patients is the development of hyperkalaemia.

Endocrine causes of hypertension form another important group. The excessive release of catecholamines by the adrenal medulla, in cases of phaeochromocytoma, may be paroxysmal or stable. Patients suffering from phaeochromocytoma, may present with typical paroxysms of hypertension, accompanied by tachycardia, palpitations, malaise, apprehension and sweating. In other patients the increase of arterial pressure is stable and associated with heat intolerance and signs of hypermetabolism. Confirmation is obtained from studies of plasma catecholamines and determinations of excretion of vanillylmandelic acid (VMA), catecholamines and metanephrine. If the presence of a phaeochromocytoma is not recognized and the patient is not prepared adequately, anaesthetic and surgical manoeuvres are likely to cause life-threatening complications such as myocardial infarction, intracerebral haemorrhage and cardiac failure.

Arterial hypertension may result from over production of gluco- and mineralocorticoids by the adrenal cortex. The clinical features of Cushing's disease are easily recognized and it is important to be aware of the possibility of diabetes, hypokalaemia and osteoporosis, because these are common complications. Low potassium stores combined with muscle wasting caused by increased protein catabolism result in muscle weakness. This may severely interfere with adequate pulmonary ventilation in the period after operation. In primary hyperaldosteronism (Conn's syndrome), the excessive production of aldosterone promotes sodium retention and causes losses of potassium and hydrogen ions. The hypokalaemic alkalosis facilitates the development of arrhythmias and the low potassium stores are responsible for muscle weakness. In both Cushings syn-
PREOPERATIVE ASSESSMENT

drome and Conn's syndrome, repletion of the potassium stores is essential before surgery.

Amongst other causes of arterial hypertension, aortic coarctation must also be considered. Blood flow to the territories distal to the obstruction, particularly to the kidneys, is maintained because of arterial hypertension and because of collateral channels connecting vessels above and below the obstruction. The diagnosis is often made on routine physical examination revealing arterial hypertension, forceful carotid pulsation, delayed or absent femoral pulses, collateral vessels around the scapula and signs of left ventricular enlargement. Patients with aortic coarctation are at risk of developing congestive heart failure, bacterial endocarditis, hypertensive encephalopathy and cerebrovascular accidents. Hypertensive responses to anaesthetic or surgical manoeuvres could precipitate life-threatening complications.

Treatment of arterial hypertension has improved the life expectancy and reduced the frequency of cerebrovascular and renal complications. Withdrawal of antihypertensive therapy may cause severe exacerbations of the hypertensive disease (Katz, Croneau and Barash, 1976; Bruce, Croley and Lee, 1979), while maintenance of treatment up to and including the day of anaesthesia and surgery makes the cardiovascular state of the hypertensive patients more stable (Prys-Roberts, Meloche and Foex, 1971). This attitude is now accepted and it is very important to understand the pharmacology of antihypertensive agents. Thiazides are widely used in the treatment of hypertension and may induce hypokalaemia, hyperuricaemia and hyperglycaemia. Noradrenaline-depleting drugs (guanidine analogues and rauwolfia alkaloids) decrease the effects on the circulation of indirect-acting pressor amines such as ephedrine, while they exaggerate the effects of direct-acting pressor amines (noradrenaline, methoxamine, metaraminol, phenylephrine). Vasodilators tend to cause tachycardia. Adrenergic beta-receptors are widely used in the treatment of hypertension and will be discussed later. Clonidine causes central sympathetic inhibition; sudden withdrawal may cause severe rebound hypertension (Hansson and Huynor, 1973) and thus it is essential to continue the normal dosage throughout the period of operation, if necessary by i.v. or i.m. administration (Bruce, Croley and Lee, 1979; Kaukinen, Kaukinen and Bérola, 1979).

The ideal situation is that of treated, well controlled hypertension. What should be the attitude for untreated hypertensive patients? In those with moderate hypertension (diastolic arterial pressure between 90 and 100 mm Hg), and in the absence of clinical signs of cardiac, renal or cerebrovascular involvement, there is no evidence that treatment of arterial hypertension reduces the frequency of complications after surgery (Goldman and Caldera, 1979). In those with severe hypertension, elective surgery should be postponed and hypertension brought under control. In emergencies haemodynamic responses to anaesthesia can be minimized by the careful use of anaesthetic agents and of cardiovascular drugs, (including, where necessary, vasodilators and adrenergic beta-receptor antagonists) with monitoring of e.g., arterial pressure and, in some cases, the pulmonary wedge pressure (Prys-Roberts and Meloche, 1980).

VASCULAR DISEASE

Patients with peripheral vascular disease suffer from a generalized disease and the life-threatening complications include myocardial infarction and cerebrovascular accidents.

Cerebrovascular disease

Looking for signs of previous cerebrovascular accidents is imperative, but not sufficient to assess patients with cerebrovascular disease. The patients should be questioned carefully regarding any history of transient disturbances of neurological function. Transient ischaemic attacks have a brief duration, tend to resolve spontaneously without residue and usually recur. When the initiating lesion is present in the distribution of the carotid arteries, a history of brief contralateral hemiparesis or monoparesis may be elicited, at times accompanied by monocular blindness on the side of the lesion. When the stenotic lesion is in the vertebrobasilar distribution, the most common manifestations are vertigo, often accompanied by dimming or loss of vision and "drop attacks". Particular attention should be paid to reduced carotid artery pulsation and to carotid artery bruits. These signs could indicate the presence of carotid artery stenosis. Cerebral infarction could follow the development of hypotension during operation (Tufo, Ostfeld and Shekelle, 1970; Carney et al., 1977). Like postoperative myocardial infarction, the
prognosis of postoperative stroke is very poor with about 50% mortality (Carney et al., 1977). The risk of stroke after operation is increased by previous cerebrovascular accidents (Corman, 1979).

Cerebrovascular disease as a result of surgically accessible lesions of the carotid arteries may be discovered in patients with severe coronary artery disease requiring coronary artery surgery. Prophylactic carotid endarterectomy has been advocated as a preliminary to coronary artery surgery. Mortality of carotid artery surgery is very high in these patients (Braithwaite, 1975). When coronary artery and carotid artery surgery are performed during the same operation, the frequency of cardiac and cerebrovascular complications is low (Morris et al., 1978) and this must be borne in mind when cerebrovascular surgery is contemplated in patients with severe coronary heart disease.

Peripheral vascular disease

Oclusive vascular disease is most commonly caused by atherosclerosis or embolic occlusion. Thromboangiitis obliterans, collagen disease and other types of arteritis are much less frequent. In the case of aorto-iliac atheroma, the stenosis or occlusion is situated in the lower part of the abdominal aorta below the renal arteries. In case of femoro-popliteal atheroma, the lesions develop near the junction of the middle and distal thirds of the femoral artery and then extend proximally up the vessel as far as its femoris profunda branch. The clinical features of peripheral vascular disease of the lower extremities include intermittent claudication, diminution or absence of pulses, systolic bruits, ischaemic skin changes and muscle wasting. Claudication pain is classically felt in the calf (gastrocnemius and soleus), in the sole of the foot and, less frequently, in thighs and buttock. Claudication pain occurs after a definite amount of exercise. In the early stages of the disease hyperaemic responses are delayed but still adequate, and the patient is able to continue to walk. In more severe cases the patient must stop. Rest pain is a very ominous sign with regard to survival of the affected limb. All pulses should be felt and their intensity recorded. Oscillometry gives a crude indication of the amplitude of arterial pulsation, while examination with ultrasound gives a non-invasive quantitative estimation of blood flow. Auscultation for bruits should be performed over the entire length of the aorta. A bruit in the region of the umbilicus may indicate distal aortic or common iliac disease; in the mid-epigastrium and laterally it may reveal renal artery stenosis, and in the subxyphoid area it may relate to coeliac artery stenosis.

In the presence of severe peripheral vascular disease, the patient’s activity can be so limited that even very severe coronary heart disease may remain asymptomatic. This should not be overlooked in the preoperative assessment.

Valvular heart disease

Only the basic pathophysiology of the most common valvular diseases will be described.

Aortic stenosis

Aortic stenosis causes a chronic pressure overload of the left ventricle characterized by concentric hypertrophy of the ventricular wall. The hypertrophic ventricle has reduced compliance. Myocardial oxygen demand is increased because of increased left ventricular mass, increased left ventricular pressure and prolonged ejection time, while coronary blood flow and oxygen supply are decreased by intramyocardial pressure (decreasing systolic coronary flow and impeding subendocardial blood flow) and by associated coronary artery disease. While tolerance to anaesthesia need not be reduced by mild or moderate asymptomatic aortic stenosis, it is greatly reduced by severe symptomatic aortic stenosis (Goldman et al., 1977). Depression of the myocardium by volatile anaesthetic agents and sudden reductions of peripheral vascular resistance may reduce coronary perfusion and cardiac output dramatically. Because of the relatively fixed stroke volume, bradycardia causes marked reductions of cardiac output; tachycardia compromises coronary flow by reducing the duration of diastole. Because of the reduced diastolic compliance, the atrial contribution to ventricular filling is an important determinant of cardiac output and arrhythmias such as junctional rhythms are poorly tolerated. Similarly, even modest changes in circulating volume have an adverse effect on cardiac performance.

Aortic stenosis associates a loud, rough, systolic murmur (second right interspace) with thrill, absent second aortic sound and slow-rising pulse. The apex thrust is forceful. Electrocardiographic signs of left ventricular hypertrophy and strain are present.
**Aortic regurgitation**

Aortic insufficiency imposes a chronic volume load to the left ventricle as the regurgitant flow from the aorta is added to normal ventricular filling from the left atrium. This increased load causes eccentric ventricular hypertrophy (wall thickness and size increase) and the ventricle becomes more compliant. The low aortic diastolic pressure may reduce coronary perfusion. Bradycardia accentuates regurgitation by increasing the duration of diastole, and is poorly tolerated. Resistance to left ventricular ejection being a major determinant of forward left ventricular flow, increases in peripheral vascular resistance decrease cardiac output and may precipitate cardiac failure.

Aortic insufficiency may be difficult to detect. The high-pitched diastolic murmur may be heard only when the patient leans forward. Wide pulse pressure and collapsing pulse are observed. The electrocardiogram reveals signs of left ventricular hypertrophy and strain.

**Mitral stenosis**

Resistance to blood flow through the narrowed mitral orifice causes chronic increase of left atrial pressure. The increased atrial pressure is reflected into the pulmonary circulation where pulmonary venous congestion and perivascular oedema develop, followed by hypertrophy and intimal sclerosis of the pulmonary vessels. Gradually, the signs of pulmonary venous congestion (haemoptysis, pulmonary oedema) are replaced by signs of systemic venous congestion (hepato-megaly, peripheral oedema). In severe mitral stenosis, stroke volume is relatively fixed and bradycardia can result in severe reductions of cardiac output. Tachycardia reduces the diastolic filling time and causes large increases in left atrial pressure, facilitating the development of pulmonary oedema. This risk is particularly important in case of fast atrial fibrillation. Administration of digitalis before operation has been advocated to minimize the risk of fast atrial fibrillation (Friedlander, 1973).

Physical signs of mitral stenosis include a loud, high-pitched first heart sound, exaggerated second pulmonic sound, short opening click and diastolic rumble with presystolic reinforcement. The electrocardiogram will confirm atrial hypertrophy (P mitrale) as long as sinus rhythm is present. Right axial deviation is usual.

**Mitral regurgitation**

During systole a large regurgitant flow occurs in the direction of the left atrium. The heart enlarges to accommodate the volume load and both atrial and ventricular compliance increase. With a very distended left atrium, large regurgitant flows are accommodated with little change of atrial pressure and the pulmonary vascular complications are minimized. However, in case of acute mitral regurgitation as a result of papillary muscle rupture or dysfunction or as a result of chordal rupture, pulmonary hypertension and right ventricular overload may develop very rapidly. Arterial hypertension increases the regurgitant flow and precipitates cardiac failure.

Physical signs of mitral regurgitation include faint apical first sound, blowing pansystolic murmurs extending into the axilla and strong apex beat. E.C.G. and chest x-ray confirm left atrial and left ventricular hypertrophy.

**Patients with valve prosthesis**

Replacement of diseased heart valves causes subjective improvement in 90% of the survivors. Radiological signs of cardiomegaly and of pulmonary hypertension, when previously observed, decrease gradually. In some patients slight stenosis of the replaced valve will develop; dysfunction because of thrombi or displacement may occur; thromboembolic accidents and mild haemolysis may develop. The frequency of embolic accidents is reduced by well controlled anticoagulation. During the first year after tricuspid or mitral valve replacement, anticoagulants should not be stopped unless haemostasis is likely to be very difficult or bleeding very dangerous. Changing from oral to parenteral anticoagulation with heparin has been recommended (Friedlander, 1973). The most serious risk in patients with valve prosthesis is that of endocarditis.

**Prevention of endocarditis**

The risk of endocarditis relates to host factors, characteristics of micro-organisms and degree of bacteraemia (Sipes, Thompson and Hooke, 1977). At highest risk are patients with left-sided valvular lesions, prosthetic valves, small ventricular septal defects, patent ductus arteriosus, tetralogy of Fallot and aortic coarctation. The presence of arteriovenous fistulae (haemodialysis shunts) and previous endocarditis increase the risk of infection. The frequency of bacteraemia is very high.
during prostatectomy, periodontal surgery, dental extraction, tonsillectomy, burns surgery and surgery of infected areas. Bronchoscopy with a rigid bronchoscope, nasotracheal intubation or endotracheal suction may cause bacteraemia in up to 15% of patients (Everett and Hirshmann, 1977). In patients at risk of developing endocarditis, all possible sites of infection must be investigated and eradicated before surgery. Antibiotic therapy should be based on all available data concerning the micro-organisms responsible for the focal infection. In the absence of infection, prophylactic antibiotic therapy is instituted. The recommendations of the Committee on Prevention of Rheumatic Fever and Bacterial Endocarditis of the American Heart Association (1977) are summarized in table I.

**TABLE I. Prophylaxis of bacterial endocarditis in adults**

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Dental procedures and upper airway surgery</th>
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<tbody>
<tr>
<td>A</td>
<td><strong>Combined oral-parenteral penicillin.</strong> Aqueous crystalline penicillin G 1 mega unit i.m. mixed with procain penicillin G 600 000 units i.m., given 30–60 min before procedure. Penicillin V 500 mg 6-hourly for next 48 h.</td>
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<tr>
<td></td>
<td><strong>Oral penicillin.</strong> Penicillin V 2 g, 30–60 min before procedure. Follow with 500 mg 6-hourly for 48 h.</td>
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<td></td>
<td><strong>Patients allergic to penicillin.</strong> Erythromycin 1 g, 1.5–2 h before procedure followed by 500 mg 6-hourly for 48 h, or Vancomycin (see below).</td>
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<tr>
<th>Regimen B (recommended for patients with prosthetic valves)</th>
<th>Gastrointestinal and genito-urinary surgery and instrumentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) <strong>Penicillin plus streptomycin.</strong> Aqueous crystalline penicillin G 1 mega unit i.m. mixed with procain penicillin G 600 000 units i.m. plus streptomycin 1 g. i.m. given 30–60 min before procedure. Penicillin V 500 mg orally, 6-hourly for next 48 h.</td>
<td></td>
</tr>
<tr>
<td>(2) <strong>Patients allergic to penicillin.</strong> Vancomycin 1 g i.v. over 30–60 min started 30–60 min before procedure and followed by erythromycin 500 mg orally 6-hourly for 48 h.</td>
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**Arrhythmias and Conduction Disorders**

Arrhythmias and heart block are often associated with organic heart disease.

**Arrhythmias**

Atrial arrhythmias are common and often considered relatively benign. However, atrial premature beats may precede the development of atrial tachycardia or atrial fibrillation; a wandering pacemaker may indicate the presence of organic heart disease or digitalis intoxication. Atrial fibrillation and flutter are usually associated with organic heart disease or with hyperthyroidism. Fast ventricular rates should always be brought back to normal values. The perioperative management of patients with atrial fibrillation is made difficult by the poor tolerance to fluid load. During major surgery monitoring of pulmonary wedge pressure is recommended (Sørensen and Engell, 1978).

Ventricular premature beats are often associated with organic heart disease or digitalis toxicity (particularly when they assume the pattern of bigeminy). Ventricular ectopic beats occurring during repolarization (R on T phenomenon) may start ventricular tachycardia or fibrillation and require immediate treatment. It must be stressed that atrial and ventricular arrhythmias contribute significantly to the cardiac risks of anaesthesia (Goldman et al., 1977).

**Long QT interval**

The upper limit for QT interval when corrected for heart rate is usually given as 0.44 s. Its prolongation may be associated with ventricular arrhythmias, syncope and sudden death. Prolongation of QT may be congenital (deafness, long QT, syncope, sudden death) or acquired. Prolongation of QT may be associated with electrolyte imbalance (hypokalaemia, hypomagnesaemia, hypocalcaemia) and with administration of drugs (quinidine, procainamide, phenothiazines, tricyclic antidepressants) or with hypothermia, cerebrovascular disease, neck surgery. The overall mortality of untreated symptomatic patients is high. If abnormally long QT interval is observed, contributing factors should be treated and beta-adrenergic receptor antagonists could be used (Schwartz and Wolf, 1978; Moss and Schwartz, 1979).

**Heart Block**

Heart block may be functional (vagal stimulation, administration of drugs including digitalis, quinidine, beta-adrenergic receptor antagonists), but more commonly is organic. Classified in three degrees, blocks range from slowing of atrio-
ventricular conduction (1st degree) to complete atrioventricular dissociation (3rd degree). The 2nd degree heart block is subdivided into Möbitz I, (or Wenckebach) type, characterized by progressive lengthening of the PR interval, and Möbitz II type characterized by regularly spaced missing ventricular contractions whilst the PR interval is constant. In this block, the lesion is situated below the A–V node and evolution into complete heart block is expected (fig. 3).

**Bifascicular blocks** are intraventricular conduction defects that may evolve into complete heart block. The bundle of His can be divided into three fascicles: right bundle branch, left anterior fascicle of the left bundle branch and left posterior fascicle of the left bundle branch. Complete left bundle branch block is, by definition, bifascicular. Right bundle branch block may be monofascicular or bifascicular if either left anterior or left posterior fascicles of the left bundle branch are also involved. When right bundle branch block is associated with left anterior fascicle block, the electrical axis shows an axial deviation of $-75^\circ$ or greater; involvement of the left posterior fascicle causes an axial deviation of greater than $110^\circ$ (fig. 4).

*Sick sinus syndrome* represents a group of disturbances resulting from failure of normal impulse formation in the sino–atrial node. Severe bradycardia, sinus arrest, paroxysmal or chronic atrial fibrillation and tachycardia appear to alternate. Heart block and life-threatening tachycardia may develop.

Anaesthesia may precipitate the development of complete heart block with very slow ventricular rate in patients with conduction disorders. There are several contributing factors to worsening of conduction. Anaesthetic agents may reduce A–V conduction. Anaesthesia often causes arrhythmias and premature beats are known to facilitate the development of blocks. Alterations of potassium concentration (hypocapnic IPPV, blood transfusion) may be induced and will modify cardiac excitability. This is why insertion of a temporary pacemaker is often necessary before elective or emergency surgery (Wynands, 1976; Simon, 1977; Zaidan, 1979). The indication for temporary pacing is almost absolute for 3rd degree and Möbitz type II blocks. In other blocks (1st degree, 1st degree and left bundle branch block, Möbitz type I, bifascicular blocks) the indication for temporary pacing is imperative if patients have presented Stokes–Adams attacks or unexplained fainting or blackouts. Temporary pacing is also indicated in case of severe bradycardia accompanied by cardiac failure, angina or syncope, in case of atrial fibrillation with very slow ventricular rate and in case of sick sinus syndrome. Without pacing, treatment of tachyarrhythmias caused by sick sinus syndrome is difficult and may result in complete heart block. As stressed by Ponka (1977) failure to use a pacemaker when indicated will cause fatalities.

**CONGESTIVE HEART FAILURE**

Left or right ventricular failure, even in their early phases, are major factors of cardiac risks of anaesthesia and surgery (Goldman et al., 1977). Diagnosis and treatment of heart failure are essential before surgery. In an emergency this may necessitate i.v. digitalis and administration of diuretics. The clinical presentation of heart failure depends upon which ventricle is most affected.

In left ventricular failure, pulmonary congestion predominates and its symptoms are dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea. Interstitial oedema stimulates juxtaglomerular J receptors and this causes a pattern of
shallow breathing. The increased effort of breathing results in the sense of shortness of breath. Augmented venous return on lying increases the interstitial oedema and explains the orthopnoea. Development of bronchial oedema explains the intense wheezing of some patients. More difficult to recognize are the early signs of left ventricular failure: coughing on assuming the recumbent position, insomnia and nocturia (in the absence of urinary infection), unexplained permanent tachycardia. Physical examination will usually reveal tachycardia, enlargement of the heart and, if the left ventricle is dilated, the systolic murmur of functional mitral incompetence. An early diastolic heart sound (S₃) is heard. Pulsus alternans (alternation of strong and weak beats while the rhythm is normal) is indicative of severe mechanical failure. Basal rales reveal the accumulation of fluid in alveoli and terminal bronchioles. There is no characteristic electrocardiographic pattern of left ventricular failure; the e.g. abnormalities reflect atrial or ventricular hypertrophy, myocardial ischaemia or infarction, conduction disorders or dysrhythmias. Chest x-ray reveals an enlarged heart shadow, exaggerated apical pulmonary vessels and prominent septal lines (Kerley’s B lines) as a result of basal interstitial oedema and distension of lymphatics.

When right ventricular failure predominates, transudation of fluid occurs in the systemic rather than the pulmonary circulation. Hepatic congestion is a prominent feature, revealed by abdominal discomfort in the right upper quadrant. The jugular veins are distended, the liver is enlarged and hepatojugular reflux is present. Peripheral pitting oedema is observed. The electrocardiogram may reveal signs of right ventricular hypertrophy and strain or signs of pulmonary embolism (S₁, Q₃, right bundle branch block).

In the light of Goldman’s study of the factors of risk, increased jugular venous pressure (indicating
right ventricular failure) or early diastolic third heart sound (left ventricular failure) are important indicators of increased cardiac risk.

The repercussions of left ventricular failure on the pulmonary circulation and on right ventricular function are well recognized. However, only recently has the effect of acute right ventricular failure on left ventricular function been studied (Laver, Strauss and Pohost, 1979). In patients with acute respiratory failure, the combination of hypoxaemia, intermittent positive ventilation and positive end-expiratory pressure can increase resistance or impedance to right ventricular ejection. The failing right ventricle dilates and, because the pericardium has limited compliance, the volume that can be occupied by the left ventricle during diastole is reduced and consequently cardiac output may decrease. This must be borne in mind in the assessment of patients with acute respiratory failure and heart disease.

**DRUG THERAPY**

**Digitalis**

Administration of digitalis before operation has been advocated to minimize the effects of anaesthetic agents on the myocardium, particularly in patients with previous history of heart failure, left or right ventricular hypertrophy, coronary artery disease, valvular disease, and in patients undergoing cardiac or major pulmonary surgery (Deutsch and Dalen, 1969; Friedlander, 1973). Other authors, however, consider that only heart failure, fast atrial fibrillation or frequent atrial premature beats justify preoperative digitalis (Shelby, 1967). Prevention of arrhythmias by use of digitalis before operation has been advocated in order to minimize the risk of fast supraventricular arrhythmias (Johnson et al., 1976). While Johnson and his colleagues observed a reduction of the frequency of supraventricular arrhythmias (including atrial fibrillation) where digitalis had been administered, other authors have found the frequency of arrhythmias either unchanged (Rose, Glassman and Spencer, 1975) or increased (Tyras et al., 1979). The risk of digitalis-induced arrhythmias is increased by hypokalaemia and by metabolic alkalosis (Brater and Morelli, 1977). Laver and Lowenstein (1980) have summarized their approach as follows:

1. Acute use of digitalis is indicated in case of heart failure;
2. Digitalis is best continued until the evening before operation if the patient with chronic atrial fibrillation has a ventricular rate faster than 80 beat min\(^{-1}\) unless digitalis intoxication is suspected;
3. Use of digitalis before operation may be considered before abdominal or intrathoracic operations in patients with previous myocardial infarction and abnormal ventricular function;
4. Digitalis should not be started only for the purpose of counteracting the cardiac depression caused by anaesthetic drugs.

**Beta-adrenoceptor antagonists**

The adverse consequences of sudden withdrawal of adrenergic beta-receptor antagonists have been well documented. They range from worsening of angina to myocardial infarction and sudden death (Slome, 1973; Alderman et al., 1974). Discontinuation of beta-receptor antagonists 24-48 h before operation makes the circulation more unstable and increases the risk of arrhythmias and of hypertensive crises, which in turn have a detrimental effect on the balance of oxygen demand and oxygen supply (Slogoff, Keats and Ott, 1978). Maintenance of adrenergic beta-receptor blockade throughout the period of operation is beneficial for patients submitting for cardiac or non-cardiac surgery (Prys-Roberts et al., 1973; Boudoulas et al., 1979; Manners and Walters, 1979; Oka et al., 1980). However, the choice of the anaesthetic agent is important. Agents provoking increases of sympathetic activity (cyclopropane, diethylether, fluoro-xene) should be avoided; their negative inotropic action is unmasked by beta-adrenergic receptor blockade. Methoxyflurane and trichloroethylene cause severe myocardial depression in the presence of beta-adrenergic receptor blockade and should also be avoided (Saner et al., 1975; Roberts et al., 1976). An adverse interaction has been observed between enfurane and propranolol (Horan et al., 1977a), but not between enfurane and oxprenolol (Cutfield et al., 1981). Clinical observations suggest that enfurane may be used to supplement nitrous oxide anaesthesia in the presence of beta-adrenergic receptor blockade (Kaplan and Dunbar, 1976). There is no indication of adverse interactions between beta-receptor antagonists and halothane, isoflurane or opiates supplementing nitrous oxide anaesthesia (Prys-Roberts et al., 1973; Horan et al., 1977b; Prys-Roberts, 1979). Beta-adrenergic receptor antagonists pro-
tect the myocardium by reducing the frequency and severity of tachycardia and hypertension. They also protect myocardial function. While they are known to decrease the extent of ischaemic dysfunction after coronary occlusion in experimental animals (Theroux et al., 1976; Vatner et al., 1977) and in man (Norris et al., 1978; Yusuf et al., 1980), they seem to minimize the adverse effect of anaesthesia on the ischaemic myocardium (Cutfield et al., 1981). They play an important role in the safe anaesthetic management of patients with coronary artery disease and arterial hypertension.

The possible hazards of beta-receptor blockade should not be ignored. In patients with cardiac failure, disorders of atrioventricular conduction, asthma or anaemia, beta-receptor blockade may be contraindicated. In the presence of beta-receptor blockade, hypercapnia is likely to cause circulatory depression: the direct negative inotropic effect of carbon dioxide is unmasked. Bradycardia may occur at the time neuromuscular blockade is reversed with neostigmine, particularly if patients are on large doses of beta-adrenergic receptor antagonists (Prys-Roberts, 1979).

OVERALL ASSESSMENT OF RISK

Careful preoperative assessment makes it possible, not only to establish the diagnosis and to evaluate the severity of cardiovascular disease, but also to gain some insight into the risk of cardiac morbidity and mortality. While the classification of physical status according to the American Society of Anaesthesiologists and the classification of heart disease according to the New York Heart Association are useful, they do not allow the anaesthetist to attach a predictive value to symptoms and signs of heart disease. Using discriminant analysis, Goldman and his colleagues (1977) have been able to identify factors that contribute significantly to the cardiac risk of anaesthesia (table II). The relative importance of each factor has been determined and with the resulting multifactorial index, more than 80% of cardiac outcomes have been predicted. All these factors are easily identified during the preoperative visit and it is easy to see the escalation of risk when several abnormalities are present.

The high frequency of life-threatening complications of anaesthesia and surgery in patients with cardiovascular diseases can be overlooked. Very often anaesthesia and the phase immediately after operation appear to be uneventful and cardiac complications occur over the first few days after operation. Anaesthesia, surgery and responses to stress all contribute to these complications, but thorough assessment before operation is essential to minimize their frequency.

REFERENCES


