

FRACP- Cardiology answers

2000 Paper

Question 1

(B)

Diastole is the period from closure of the aortic valve to closure of the mitral valve. It consists of 4 phases:

- 1) Isovolumetric relaxation (aortic closure to mitral valve opening)
- 2) Early rapid filling due to transmitral pressure difference. This phase involves energy dependent myocardial relaxation and contributes to 80% of ventricular filling
- 3) Slow filling
- 4) Atrial contraction, which contributes to 15% of filling

Diastolic dysfunction may be considered as impaired LV filling or impaired diastolic function. Impaired LV filling may be due to either pericardial disease (constriction or tamponade) or mitral stenosis. Impaired diastolic function is due to impaired ventricular relaxation (eg. ischaemia), increased compliance (LV hypertrophy due to hypertension, HOCM or AS) or restriction of the LV (infiltrative disease eg amyloid or cardiomyopathy)

Diagnosis usually relies on echocardiography. Technetium-99 labelled red cells can be used during angiography to derive the left diastolic volume measurements. Doppler echo of transmural flow enables non-invasive serial measurements of diastolic function. As it takes longer for the LV pressure to decrease below the left atrial pressure, an altered transmitral flow wave pattern is seen with an increased isovolumetric relaxation time.

Decreased LV compliance results in reduced LV filling and reduced stroke volume. Initially this manifests as low output cardiac failure, however as diastolic dysfunction worsens LV filling pressures are increased, resulting in pulmonary congestion. Symptoms of heart failure may occur in the presence of normal systolic function.

Treatment includes treating the underlying cause (eg. revascularisation for ischaemia) and ACE inhibitors for concomitant systolic impairment. There is a paucity of clinical trials of treatments for isolated diastolic dysfunction; however beta blockers have been shown to improve LV filling by reducing heart rate and increasing the duration of diastole. This has the added advantage of reducing myocardial oxygen demand.

Question 2

(D)

The International Registry of Acute Aortic Dissection (IRAD published paper in JAMA Feb 16, 2000.

They review 464 patients between Jan 96 to Dec 98, 62.3% with Type A dissection and reviewed their presenting history, physical findings and management.

Abrupt onset of symptoms	84.8%
Chest pain	72.7%
Hypotensive on examination	8%
Auscultated murmur of AR	31.6%
AR on imaging	50% of type A = 31%
Pulse deficit	15.1%
Nonspecific ST segment T wave abN	41.4%

Question 3

(B)

Low risk patient going for low risk surgery

Clinical history taking and examination, with particular reference to a history of IHD, current exercise tolerance, presence of cardiac failure and presence of severe aortic stenosis, remain the mainstays of preoperative cardiac risk assessment and are the best predictors of outcome after surgery. Currently there is no evidence that routine tests have a role as screening tests, apart from an ECG, a CXR if clinical signs suggest pulmonary pathology, cardiomegaly or heart failure, and possibly an exercise test if the patient's exercise history is difficult to obtain. Additional tests should be requested for clinical indications alone.

Investigations, including echocardiography for valvular heart disease, should be done for clinical reasons.

Echocardiography should be considered for patients with symptomatic valvular disease who are undergoing moderate or high risk surgery, particularly if there is clinical evidence of moderate to severe AS. This information can be helpful for the anaesthetist in guiding the choice of anaesthetic agent. There is no need to investigate patients undergoing low risk surgery. Indications for valvular surgery are the same for all patients whether they are seen preoperatively or routinely. Aortic valve replacement surgery is recommended prior to routine surgery in patient with severe AS who are candidates for valvular surgery. Only one study has been published on the outcome of patients with severe aortic stenosis who underwent non-cardiac surgery. They did remarkably well with careful anaesthesia and postoperative care. Patients at low cardiac risk or those undergoing a low risk procedure should not need further investigation.

Where possible surgery should be deferred in all high risk patients until the risks have been controlled. This may mean controlling heart failure, waiting longer after MI or considering intensive medical therapy or revascularisation in a patient with unstable IHD.

Patients with moderate cardiac risk need careful assessment if they are undergoing high risk surgery, particularly vascular surgery, and are unable to exercise. The choice of additional investigations (cardiac catheterisation, DSE, or thallium scanning) depends on the clinical indications and the local availability and expertise in performing the tests. It is harder to clinically assess patients with renal disease, diabetes with evidence of end organ disease, or difficulty mobilising, particularly due to arthritis or PVD. Additional investigations are likely to be useful in such patients if they are about to have high risk surgery. There is no evidence that additional testing is helpful for moderate risk patients undergoing moderate or low risk surgery.

Cardiac drugs, particularly beta blockers and aspirin, where possible should be continued throughout the perioperative period.

High risk patients should be monitored in HDU/ICU settings immediately after surgery.

Question 4

(C)

HYPERTROPHIC CARDIOMYOPATHY

This disease is characterized by left ventricular hypertrophy, typically of a nondilated chamber, without obvious cause such as hypertension or aortic stenosis (Fig. 239-CD3). Two features of the disease have attracted the greatest attention: (1) heterogeneous left ventricular (LV) hypertrophy, often with preferential hypertrophy of the interventricular septum resulting in asymmetric septal hypertrophy (ASH); and (2) a dynamic left ventricular outflow tract pressure gradient, related to a narrowing of the subaortic area as a consequence of the midsystolic apposition of the anterior mitral valve leaflet against the hypertrophied septum, i.e., systolic anterior motion (SAM) of the mitral valve (Fig. 239-CD4). Initial studies of this disease emphasized the dynamic "obstructive" features, and it has been termed idiopathic hypertrophic subaortic stenosis (IHSS), hypertrophic obstructive cardiomyopathy (HOCM), and muscular subaortic stenosis. It has become clear, however, that only about one-quarter of patients with hypertrophic cardiomyopathy demonstrate an outflow tract gradient. The ubiquitous pathophysiologic abnormality is not systolic but rather diastolic dysfunction (Chap. 232), characterized by increased stiffness of the hypertrophied muscle that results primarily from an abnormality in calcium handling with attendant intracellular calcium overload. This results in elevated diastolic filling pressures and is present despite a hyperdynamic left ventricle.

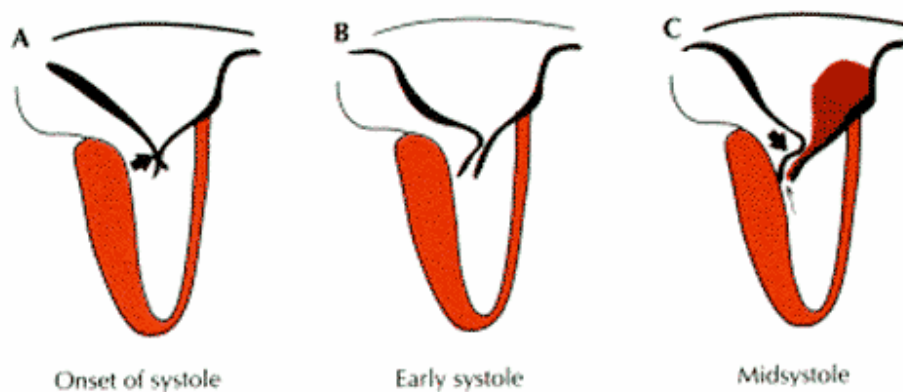


FIGURE 239-cd4: Functional anatomy of mitral leaflet systolic anterior motion and mitral regurgitation in subaortic obstructive hypertrophic cardiomyopathy (HCM).

Drawing of a transesophageal echocardiogram (frontal long-axis plane) demonstrating the anterior and superior motion of the anterior mitral leaflet to produce mitral leaflet-septal contact and failure of leaflet coaptation in midsystole. A. At the onset of systole, the coaptation point (arrow) is in the body of the anterior and posterior leaflets rather than at the tip of the leaflets, as in normal subjects. During early systole (B) and midsystole (C) there is anterior and superior movement of the residual length of the anterior mitral leaflet (thick arrow in C), with septal contact and failure of leaflet coaptation (thin arrow in C) with consequent mitral regurgitation directed posteriorly into the left atrium (dotted area).

[From ED Wigle, AD Kitching, H Rakowski: Hypertrophic cardiomyopathy, in WH Abelmann (volume ed):

Cardiomyopathies, Myocarditis, and Pericardial Disease, in E Braunwald (series ed): Atlas of Heart Diseases, vol 2. Philadelphia: Current Medicine, 1994, p. 2.9. Adapted from LE Grigg, ED Wigle, WG Williams, et al: Transesophageal Doppler echocardiography in obstructive hypertrophic cardiomyopathy: clarification of pathophysiology and importance in intraoperative decision making. J Am Coll Cardiol 20:42-52, 1992.]

The pattern of hypertrophy is distinctive in hypertrophic cardiomyopathy and differs from that seen in secondary hypertrophy (as in hypertension). Most patients have striking regional variations in the extent of hypertrophy in different

portions of the left ventricle, and the majority demonstrate a ventricular septum whose thickness is disproportionately increased when compared with the free wall. Other patients may demonstrate disproportionate involvement of the apex or left ventricular free wall; 10 percent or more of patients have concentric involvement of the ventricle. All, however, show a bizarre and disorganized arrangement of cardiac muscle cells in the septum, with disorganization of the myofibrillar architecture, whether or not a systolic intraventricular pressure gradient is present, along with a variable degree of myocardial fibrosis and thickening of the small intramural coronary arteries.

About half of all cases of hypertrophic cardiomyopathy have a positive family history compatible with autosomal-dominant transmission. About 40 percent of these are associated with mutations of the beta cardiac myosin heavy chain gene on chromosome 14, with certain mutations associated with more malignant prognoses. About 15 percent have a mutation of the cardiac troponin T gene, 10 percent a mutation of myosin binding protein L, and about 5 percent a mutation of the tropomyosin gene. The remainder of familial cases presumably are due to mutations of other genes. Echocardiographic studies have confirmed that about one-third of the first-degree relatives (i.e., parents, siblings, and children) of patients with familial hypertrophic cardiomyopathy have evidence of the disease, although in many of these patients the extent of hypertrophy is mild, no outflow tract pressure gradient is present, and symptoms are not prominent. Since the hypertrophic characteristics may not be apparent in childhood and often appear first in adolescence, a single normal echocardiogram in a child does not entirely exclude the presence of the disease.

In contrast to the obstruction produced by a fixed narrowed orifice, such as valvular aortic stenosis, the pressure gradient in hypertrophic cardiomyopathy, when present, is dynamic and may change between examinations and even from beat to beat. Obstruction appears to result from further narrowing of an already small left ventricular outflow tract by SAM of the mitral valve against the hypertrophied septum. While SAM may be found in a variety of other conditions besides hypertrophic cardiomyopathy, it is always found when obstruction is present in hypertrophic cardiomyopathy. Three basic mechanisms are involved in the production of the dynamic pressure gradient: (1) increased left ventricular contractility, (2) decreased ventricular volume (preload), and (3) decreased aortic impedance and pressure (afterload). Interventions that increase myocardial contractility, such as exercise, isoproterenol, and digitalis glycosides, and those that reduce ventricular volume, such as the Valsalva maneuver, sudden standing, nitroglycerin, amyl nitrite, or tachycardia, all may cause an increase in the gradient and the murmur. Conversely, elevation of arterial pressure by phenylephrine, squatting, sustained handgrip, augmentation of venous return by passive leg raising, and expansion of the blood volume all increase ventricular volume and ameliorate the gradient and murmur.

Clinical Features

Many patients with hypertrophic cardiomyopathy are asymptomatic and may be relatives of patients with known disease. Unfortunately, the first clinical manifestation of the disease may be sudden death, frequently occurring in children and young adults, often during or after physical exertion. In symptomatic patients, the most common complaint is dyspnea, largely due to increased stiffness of the left ventricular walls, which impairs ventricular filling and leads to elevated left ventricular diastolic and left atrial pressures. Other symptoms include angina pectoris, fatigue, syncope, and near-syncope ("graying-out spells"). Symptoms are not related to the presence or severity of an outflow gradient. Most patients with gradients demonstrate a double or triple apical precordial impulse, a rapidly rising carotid arterial pulse, and a fourth heart sound. The hallmark of obstructive hypertrophic cardiomyopathy is a systolic murmur, which is typically harsh, diamond-shaped, and usually begins well after the first heart sound, since ejection is unimpeded early in systole (Fig. 239-CD5). The murmur is best heard at the lower left sternal border as well as at the apex, where it is often more holosystolic and blowing in quality, no doubt due to the mitral regurgitation that usually accompanies obstructive hypertrophic cardiomyopathy.

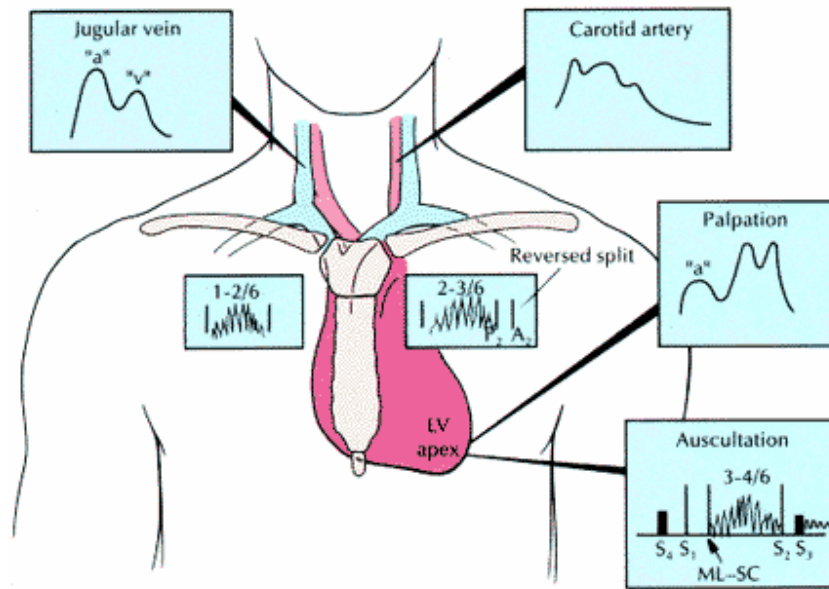


FIGURE 239-cd5: Physical examination in subaortic obstructive HCM.

On palpation, a spike-and-dome arterial pulse can often be felt in the carotid artery. On palpation of the left ventricular (LV) apex, there may be a triple apex beat caused by a palpable left atrial gallop and a double systolic impulse. On auscultation, at or just medial to the LV apex, there is a late onset, diamond-shaped systolic murmur of grade 3 to 4/6 in intensity; caused by both the subaortic obstruction and the concomitant mitral regurgitation. There is often a short diastolic inflow murmur after the third heart sound. Rarely, a mitral leaflet-septal contact (ML-SC) sound may be heard preceding the systolic murmur at the apex. Reversed splitting of the second heart sound may occur. In nonobstructive HCM, there is often a third or fourth heart sound at the apex. The jugular venous pulse frequently reveals a prominent a-wave that rises on inspiration, reflecting RV diastolic dysfunction. HCM, hypertrophic cardiomyopathy.

[From ED Wigle, AD Kitching, H Rakowski: Hypertrophic cardiomyopathy, in WH Abelmann (volume ed): *Cardiomyopathies, Myocarditis, and Pericardial Disease*, in E Braunwald (series ed): *Atlas of Heart Diseases*, vol 2. Philadelphia: Current Medicine, 1994, p. 2.17.]

Laboratory Evaluation

The electrocardiogram commonly shows left ventricular hypertrophy and widespread, deep, broad Q waves that suggest an old myocardial infarction. Many patients demonstrate arrhythmias, both atrial (supraventricular tachycardia or atrial fibrillation) and ventricular (ventricular tachycardia), during ambulatory (Holter) monitoring. Chest roentgenography may be normal, although a mild to moderate increase in the cardiac silhouette is common. The mainstay of the diagnosis of hypertrophic cardiomyopathy is the echocardiogram (Fig. 239-CD6), which demonstrates left ventricular hypertrophy, often with the septum 1.3 or more times the thickness of the high posterior left ventricular free wall. The septum may demonstrate an unusual ground glass appearance, probably related to its abnormal cellular architecture and myocardial fibrosis. SAM of the mitral valve is found in patients with pressure gradients. The left ventricular cavity typically is small in hypertrophic cardiomyopathy, with vigorous posterior wall motion but reduced septal excursion. A rare form of hypertrophic cardiomyopathy, characterized by apical hypertrophy, is often associated with giant negative T waves on the electrocardiogram and a "spade-shaped" left ventricular cavity on angiography; it usually has a benign clinical course. Radionuclide scintigraphy with thallium 201 frequently reveals evidence of myocardial perfusion defects even in asymptomatic patients.

Although cardiac catheterization is not required to diagnose hypertrophic cardiomyopathy, the two typical hemodynamic features are an elevated left ventricular diastolic pressure due to diminished left ventricular compliance and, when obstruction is present, a systolic pressure gradient between the body of the left ventricle and the subaortic region. When a gradient is not present, it often can be induced by provocative maneuvers such as infusion of isoproterenol, inhalation of amyl nitrite, or the Valsalva maneuver.

Treatment

Competitive sports and probably strenuous activity should be proscribed. Beta-adrenergic blockers often are used and may ameliorate angina pectoris and syncope in one-third to one-half of patients to some degree. Resting intraventricular pressure gradients usually are unchanged, although these drugs may limit the increase in the gradient that occurs during exercise. It is not known whether beta-adrenergic blockers offer any protection against sudden death. It is not established whether any antiarrhythmic agent, for that matter, is effective in this setting. However, amiodarone appears to be effective in reducing the frequency of supraventricular as well as life-threatening ventricular arrhythmias. Verapamil and diltiazem

may reduce the stiffness of the ventricle, reduce the elevated diastolic pressures, increase exercise tolerance, and, in some instances, reduce the severity of outflow tract gradients, although adverse side effects occur in about one-quarter of patients. Disopyramide has been used in some patients to reduce left ventricular contractility and the outflow gradient. Dual-chamber permanent pacing recently has gained favor because it improves symptoms and reduces the outflow gradient in some patients with severe symptoms, presumably by altering the pattern of ventricular contraction. (See Concise Review: Pacing in Hypertrophic Cardiomyopathy) Infarction of the interventricular septum induced by ethanol injections into the septal artery has also been reported to reduce obstruction. The insertion of an implantable automatic defibrillator should be considered in patients surviving cardiac arrest and those with high-risk ventricular tachyarrhythmias. A surgical myotomy/myectomy of the hypertrophied septum may result in lasting symptomatic improvement in about three-quarters of operated patients, but the mortality of 3 to 5 percent limits the operation to severely symptomatic patients with large pressure gradients who are unresponsive to medical management. The effect of any of these therapies on the natural history is not clear. Digitalis, diuretics, nitrates, and beta-adrenergic agonists are best avoided if possible, particularly in patients with known left ventricular outflow tract pressure gradients.

Question 5

(C)

Question 6

Question 7

(C)

ACUTE PERICARDITIS

Acute pericarditis, by far the most common pathologic process involving the pericardium, may be classified both clinically and etiologically (Table 240-1). Pain, a pericardial friction rub, electrocardiographic changes, and pericardial effusion with cardiac tamponade and paradoxical pulse are cardinal manifestations of many forms of acute pericarditis and will be considered prior to a discussion of the most common forms of the disorder.

Chest pain is an important but not invariable symptom in various forms of acute pericarditis (Chap. 13); it is usually present in the acute infectious types and in many of the forms presumed to be related to hypersensitivity or autoimmunity. Pain is often absent in a slowly developing tuberculous, postirradiation, neoplastic, or uremic pericarditis. The pain of pericarditis is often severe. It is characteristically retrosternal and left precordial, referred to the back and the trapezius ridge. Often the pain is pleuritic consequent to accompanying pleural inflammation, i.e., sharp and aggravated by inspiration, coughing, and changes in body position, but sometimes it is a steady, constricting pain that radiates into either arm or both arms and resembles that of myocardial ischemia; therefore, confusion with myocardial infarction is common. Characteristically, however, the pericardial pain may be relieved by sitting up and leaning forward and is intensified by lying supine. There is often a pleural component, with aggravation of the pain with coughing and deep inspiration. The differentiation of acute myocardial infarction from acute pericarditis becomes perplexing when, with acute pericarditis, the serum transaminase and creatine kinase levels rise, presumably because of concomitant involvement of the epicardium. However, these enzyme elevations, if they occur, are quite modest, given the extensive electrocardiographic ST-segment elevation in pericarditis.

The pericardial friction rub is the most important physical sign; it may have up to three components per cardiac cycle and is high-pitched, scratching, and grating, as described in Chap. 227; it can sometimes be elicited only when firm pressure with the diaphragm of the stethoscope is applied to the chest wall at the left lower sternal border. It is heard most frequently during expiration with the patient in the sitting position. The rub is often inconstant and the loud to-and-fro leathery sound may disappear within a few hours, possibly to reappear the following day.

The electrocardiogram in acute pericarditis without massive effusion usually displays changes secondary to acute subepicardial inflammation (see Fig. 228-18, p. 1245). There is widespread elevation of the ST segments, involving two or three standard limb leads and V2 to V6, with reciprocal depressions only in aVR and sometimes V1. Usually there are no significant changes in QRS complexes, except for some reduction in voltage in patients with large pericardial effusions. After several days, the ST segments return to normal, and only then do the T waves become inverted. In contrast, in acute myocardial infarction, reciprocal depression of ST segments is usually more prominent; QRS changes occur, particularly the development of Q waves, as well as notching and loss of the amplitude of R waves; and T-wave inversions usually occur within hours before the ST segments have become isoelectric. Sequential electrocardiograms are useful in distinguishing acute pericarditis from acute myocardial infarction. In the latter, elevated ST segments return to normal within hours. Early repolarization is a normal variant and also may cause widespread ST-segment elevation, most prominent in left precordial leads. However, in this condition the T waves are usually tall and the ST/T ratio is under 0.25, but it exceeds this number in acute pericarditis. Depression of the PQ segment (below the TP segment) also is common and reflects atrial involvement. With large pericardial effusions, the QRS voltage is reduced; atrial premature beats and atrial fibrillation are sometimes noted.

Question 8

(B)

Table 246-2 in Harrison's states that 92-94% of hypertensives have essential hypertension, and that the prevalence of renovascular hypertension is 1-2% of hypertension. Approximately 15% of patients with essential hypertension have plasma renin levels above the normal range. It has been suggested that plasma renin plays an important role in the pathogenesis of hypertension in these patients. However, most studies have found that saralasin (a substance that, like losarten, acts as a competitive antagonist of angiotensin 2 significantly reduces BP in less than half of these patients. This finding has led some investigators to believe that the elevated renin levels and BP may both be secondary to an increase in adrenergic system activity. It has been proposed that, in patients with angiotensin dependent high renin hypertension whose pressures are lowered by angiotensin 2 antagonists, the mechanism responsible for the increase in renin, and therefore for the hypertension is the nonmodulating effect.

Question 9

(D)

Terfenadine undergoes extensive metabolism in the liver by cytochrome P450 3A4. Erythromycin is a competitive 3A4 enzyme inhibitor. Patients with impaired hepatic function or those receiving treatment with potent inhibitors of hepatic oxidation (eg. erythromycin or ketoconazole; or having conditions leading to QT prolongation may experience QT prolongation and/or VT at the recommended dose. Loratidine, despite also being cleared by CYP 3A4 does not have this arrhythmogenic effect. Sotalol, despite prolonging the QT interval is 80-90% excreted unchanged in the urine, the rest in the faeces. No hepatic metabolism. Cimetidine is a non-competitive inhibitor of CYP3A4 but no arrhythmogenic effects

Question 10

(C)

Cigarette smoking is not a risk factor for the development of hypertension; however, hypertensives who smoke are at a greater risk of developing malignant hypertension and of dying from hypertension. The beneficial effects of diet on blood pressure can be maximized by avoiding high intake of NaCl and ensuring adequate intake of fruits, vegetables, and fat-free and low-fat dairy products. Such diets are rich in potassium, calcium, magnesium, and protein and low in total fat, saturated fat, and cholesterol. Although not discussed here, additional nutritional strategies for optimizing the effect of diet on blood pressure are prevention and treatment of obesity, and for people who drink alcohol, avoiding consumption of >2 drinks per day.

Question 11

(C)

Question 12

(A)

Insulin resistance, hyperinsulinaemia and glucose intolerance is reported to be atherogenic. Diabetes, IGT, and high normal levels of glycosylated Hb in the Framingham study were powerful contributors to atherosclerotic cardiovascular events in study participants. In this study, the risk of CHD in participants younger than 65 years was typically double in men with diabetes and triple in women with diabetes compared with non diabetic counterparts. These relative risks were slightly lower but still increased in participants with diabetes who were older than 65 years. Patients with diabetes have a greater burden of atherogenic risk factors than patients without diabetes, including elevated BP, raised triglycerides, increased total/HDL cholesterol ration, hyperuricaemia, elevated fibrinogen and LVH. The risk of CHD in patients with diabetes varies widely with the intensity of these risk factors.

Persons with diabetes and hypertension are noted in the Framingham study to be especially at risk of unrecognised MI, necessitating periodic surveillance with routine ECG.

Hypertension, obesity, insulin resistance, hyperinsulinaemia, hypertriglyceridaemia and low HDL cholesterol levels tend to coexist as an "insulin resistance syndrome" and accelerate atherogenesis

FRACP 1999 (paper 1)

Question 1

(A)?

General population prevalence ranges between 3 and 20 individuals per 1000, increasing to between 80 and 160 individuals per 1000 among those aged 75 years or over. These data consistently shows the pronounced influence of age, such as the doubling by decade effect on incidence recorded in the Framingham study.

The incidence and the prevalence of heart failure in NZ has not been firmly established, but are almost certainly similar to other Western countries. There is a close relationship between heart failure and age. With progressive aging of the New Zealand population it must be presumed that the prevalence of heart failure will increase.

In terms of aetiology, long term followup shows that hypertension is the most common identifiable risk factor. In Framingham, there has been no change in the frequency of hypertension as the attributable cause of heart failure during 4 decades of observation.

Despite reductions in the age related incidence of myocardial infarction and improved control of blood pressure, the prevalence of heart failure does not seem to be falling and may be rising.

Question 2

(B)

The contractile process of cardiac muscle begins just after the start of depolarisation and lasts about 1.5x as long as the action potential. The role of Ca²⁺ in excitation-contraction coupling is similar to its role in skeletal muscle. However, it is the influx of extracellular calcium that is triggered by the activation of the dihydropyridine channels in the T system, rather than depolarisation per se that triggers release of stored calcium from the sarcoplasmic reticulum.

Question 3

(E)

Steady state is reached after 4 – 5 half lives

Question 4

(E)

Predictors of mortality in idiopathic dilated cardiomyopathy

Although clinical, hemodynamic, and ventriculographic features are helpful in determining risk in large populations, the assessment of prognosis for an individual patient remains problematic. Notwithstanding the associated risks of ventricular arrhythmias and embolic complications, the prognosis is most closely related to the severity of left ventricular dysfunction. Although the relation is not linear, a greater degree of ventricular enlargement typically portends a poorer prognosis. The left ventricular ejection fraction is also a powerful, independent predictor of prognosis. Whereas the severity of left ventricular dysfunction can be correlated with outcome, (13,14,30,31,34,35,36,37,38,39) the relation between the ejection fraction and survival is weaker in more homogeneous populations, particularly when the ejection fraction falls below 25 percent

Clinical features associated with a more favorable prognosis include an NYHA functional class below IV, (38,39,40,47) a relatively young age, (13,17,20,30) and female sex (21). Syncope, (12) persistent S3 gallop or right-sided heart failure on physical examination, (34,43) and either first- or second-degree atrioventricular block or a left bundle-branch block on the electrocardiogram predict a poor prognosis (35,36). Hyponatremia (serum sodium concentration, <137 mmol per liter) is a marker of more advanced disease and a poorer prognosis (47,48). Although elevated plasma concentrations of norepinephrine, (47,49) atrial natriuretic factor, (47) and renin (47) have prognostic value, they are seldom used clinically. Ventricular arrhythmias and sudden death are both common features of dilated cardiomyopathy, yet the prognostic importance of ventricular arrhythmias remains unclear. Ambulatory 24-hour electrocardiographic monitoring has confirmed the virtually universal occurrence of premature ventricular beats (35,53,54,55,56) and has detected asymptomatic nonsustained ventricular tachycardia in 40 percent of patients (range, 20 to 60 percent) (35,55,56,57,58,59). As in ischemic heart disease, there is an inverse correlation between the severity of ventricular arrhythmia and the left ventricular ejection fraction (56). A review of 15 published series has shown that 12 percent of patients with IDC died suddenly and that sudden death accounted for 28 percent of all deaths (range, 8 to 51 percent) (14,19,30,35,36,38,47,53,54,55,56,57,58,59,60). Despite the use of beta-blockers, vasodilators, and amiodarone, this percentage has not decreased appreciably in recent years. **Syncope is a clinical feature strongly predictive of sudden death** (61,62). Although virtually all studies have confirmed an association between asymptomatic nonsustained ventricular tachycardia and both the severity of hemodynamic abnormalities and overall cardiac mortality, (30,35,47,53,54,55,56,57,60) the patients at greatest risk of sudden death or in need of antiarrhythmic therapy cannot yet be prospectively identified

FRACP 1999 (Paper 2)

Question 1

(A)

Question 2

(E)

See answer to question 3 FRACP 2000

Question 3

(B)

Age related cardiovascular physiological changes include

- ◆ Decreased arterial compliance with increased systolic blood pressure
- => Hypotensive response to an increase in heart rate, volume depletion or loss of atrial contraction
- ◆ Decreased adrenergic response
- => Decreased cardiac output and HR response to stress
- ◆ Decreased baroreceptor sensitivity and decreased SA node automaticity
- => Impaired blood pressure response to standing and volume depletion

Question 4

(A)

ECG shows inferoposterior ST elevation MI

Question 5

(D)

Calcium is involved in the initiation of contraction of smooth muscle, as it is in skeletal muscle. However visceral smooth muscle has a very poorly developed sarcoplasmic reticulum, and the increase in intracellular calcium concentration that initiates contraction is due primarily to calcium influx from the ECF via voltage gated calcium channels. In addition, the myosin in smooth muscle must be phosphorylated for activation of the myosin ATPase. Phosphorylation and dephosphorylation of myosin also occur in skeletal muscle, but phosphorylation is not necessary for activation of ATPase. In smooth muscle, calcium binds to calmodulin, and the resulting complex activates calmodulin dependent myosin light chain kinase. This enzyme catalyses the phosphorylation of the myosin light chain on serine at position 19. The phosphorylation allows myosin ATPase to be activated, and actin slides on myosin producing contraction. This is in contrast to skeletal and cardiac muscle where contraction is triggered by the binding of calcium to troponin C.

Question 6

(A)

See explanation to FRACP 2000 Question3

Question 7

(A)

See explanation to FRACP 2000 Question3

Question 8

(C)

FRACP 1998

Question 1

(B)

Figure 1: Pathways of Lipid Transport.

Cholesterol is absorbed from the intestine and transported to the liver by chylomicron remnants, which are taken up by the low-density lipoprotein (LDL)-receptor-related protein (LRP). Hepatic cholesterol enters the circulation as very-low-density lipoprotein (VLDL) and is metabolized to remnant lipoproteins after lipoprotein lipase removes triglyceride. The remnant lipoproteins are removed by LDL receptors (LDL-R) or further metabolized to LDL and then removed by these receptors. Cholesterol is transported from peripheral cells to the liver by high-density lipoprotein (HDL). Cholesterol is recycled to LDL and VLDL by cholesterol-ester transport protein (CETP) or is taken up in the liver by hepatic lipase. Cholesterol is excreted in bile. The points in the process that are affected by the five primary lipoprotein disorders -- familial hypertriglyceridemia (FHTG), familial combined hyperlipidemia (FCHL), remnant removal disease (RRD, also known as familial dysbetalipoproteinemia), familial hypercholesterolemia (FH), and hypoalphalipoproteinemia -- are shown.

The effects of drug therapy can also be understood from these pathways. Statins decrease the synthesis of cholesterol and the secretion of VLDL and increase the activity of LDL receptors. Bile-acid-binding resins increase the secretion of bile acids. Nicotinic acid decreases the secretion of VLDL and the formation of LDL and increases the formation of HDL. Fibrates decrease the secretion of VLDL and increase the activity of lipoprotein lipase, thereby increasing the removal of triglycerides. Adapted from Knopp. (12)

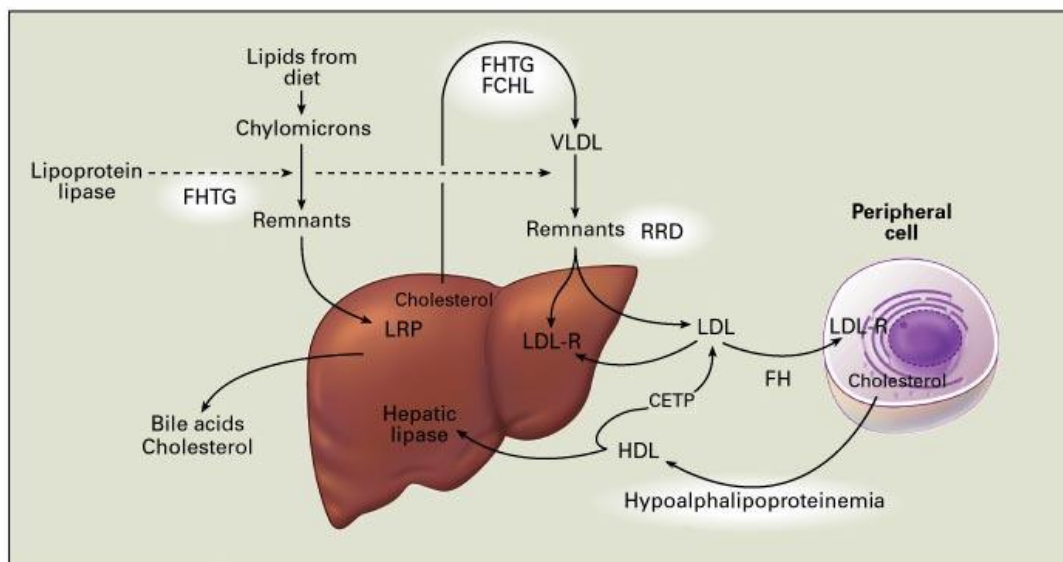


Figure 3: Mechanisms of Action of Nicotinic Acid.

Nicotinic acid inhibits the mobilization of free fatty acids (FFA) from peripheral adipose tissue to the liver. As a consequence of this decrease or an additional hepatic effect, the synthesis and secretion of very-low-density lipoprotein (VLDL) are reduced, and the conversion of VLDL to low-density lipoprotein (LDL) is decreased. (96) Nicotinic acid can also increase serum high-density lipoprotein (HDL) cholesterol concentrations by up to 30 percent; the mechanism responsible for this effect is unknown. Reproduced from Knopp et al. (100) with the permission of the publishers.

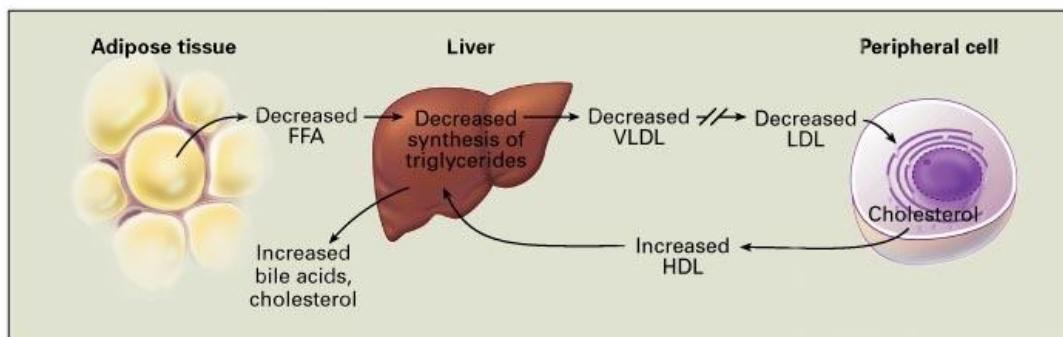


TABLE 1. PRIMARY LIPOPROTEIN DISORDERS AMENABLE TO TREATMENT WITH DIET AND DRUG THERAPY.*

DISORDER	MECHANISMS	COMPLICATIONS	TREATMENT†
Familial hypertriglyceridemia‡	Decreased serum triglyceride removal resulting from decreased LPL activity Increased hepatic secretion of triglyceride-rich VLDL	Pancreatitis at triglyceride concentrations >2000 mg per deciliter (22.6 mmol/liter); low risk of CAD	Diet and weight loss Fibrate Nicotinic acid n-3 fatty acids Oxandrolone
Familial combined hyperlipidemia‡	Increased hepatic secretion of apolipoprotein B-containing VLDL and conversion to LDL Accumulation of VLDL, LDL, or both, depending on efficiency of their removal	CAD, PVD, stroke	Diet and weight loss Statin Nicotinic acid Fibrate§
Remnant removal disease (familial dysbetalipoproteinemia)	Increased secretion of VLDL Impaired removal of remnant lipoproteins resulting from homozygosity (ϵ_2/ϵ_2) or heterozygosity (ϵ_2/ϵ_3 or ϵ_2/ϵ_4) for apolipoprotein E ϵ_2	PVD, CAD, stroke	Diet, weight loss Fibrate§ Nicotinic acid Statin
Familial or polygenic hypercholesterolemia	Diminished LDL-receptor activity Defective apolipoprotein B that is poorly recognized by LDL receptor	CAD, occasionally PVD, stroke	Diet Statin Bile-acid-binding resin Nicotinic acid
Familial hypoalphalipoproteinemia (low HDL syndrome)¶	Diminished apolipoprotein AI formation, increased removal, increased CETP or hepatic lipase activity	CAD, PVD, (may be associated with hypertriglyceridemia)	Exercise and weight loss Nicotinic acid Fibrate§ Statin

*LPL denotes lipoprotein lipase, VLDL very-low-density lipoprotein, CAD coronary artery disease, PVD peripheral vascular disease, HDL high-density lipoprotein, and CETP cholesterol-ester transfer protein.

†The treatments may be given alone or in combination; the primary treatment is listed first, followed by other treatments in decreasing order of importance.

‡Diabetes mellitus can greatly exacerbate the condition. The hyperlipidemia of diabetes is closest mechanistically to familial combined hyperlipidemia.

§Combined treatment with a fibrate and a statin can increase the risk of myopathy.

¶This disorder is characterized by low concentrations of HDL cholesterol.

Patients with severe hypertriglyceridemia are best treated with diet and a fibrate, alone or in combination with nicotinic acid, n-3 fatty acids, possibly a statin, or as a last resort, an anabolic steroid, to prevent pancreatitis. The presence of hypertriglyceridemia with low serum LDL cholesterol concentrations may not be associated with atherosclerosis. If a patient has vascular disease of any type or a family history of vascular disease, treatment is the same as for familial combined hyperlipidemia (Table 1).

Among patients with familial combined hyperlipidemia, the most appropriate treatment depends on the findings at presentation. Patients with the hypertriglyceridemic form should be treated first with diet and then nicotinic acid, and those with the hypercholesterolemic form should receive dietary therapy and a statin. The most effective therapy in patients with elevations of both serum LDL cholesterol and triglycerides is the combination of nicotinic acid (up to 2000 mg daily) and a statin with dietary therapy. If plain or timed-release nicotinic acid must be discontinued because of adverse effects, a fibrate can be given alone or in combination with a statin. This combination, however, increases the risk of myopathy. Treatment with both nicotinic acid and fibrate causes a shift in the form of LDL from small, dense particles to large, buoyant particles, an effect that is potentially beneficial in patients with combined hyperlipidemia. (43)

Fibrates are the most appropriate treatment for patients with remnant removal disease since these drugs curb the overproduction of VLDL. Such patients are often very sensitive to diet and exercise as well. A statin given in combination with a fibrate increases the removal of remnant lipoproteins. Nicotinic acid can also be given in combination with or as an alternative to a fibrate. Of all the hyperlipidemic disorders, remnant removal disease is the most responsive to drug therapy, as it is to dietary therapy, but the use of drugs in combination is required for best results.

Patients with polygenic or heterozygous familial hypercholesterolemia should be given a statin and placed on the Step II diet. A bile-acid-binding resin can be added to lower the serum LDL cholesterol concentration further. If the serum HDL cholesterol concentration is low, nicotinic acid is the preferred second drug. All three drugs are often required in patients with heterozygous familial hypercholesterolemia.

Patients with hypoalphalipoproteinemia, who have low serum HDL cholesterol concentrations, have a variable response to weight loss, exercise, diet, and lipid-lowering drugs. In patients with hypertriglyceridemia, serum HDL cholesterol concentrations almost always increase as serum triglyceride concentrations fall. Nicotinic acid usually increases serum HDL cholesterol concentrations by 30 percent, fibrates by 10 to 15 percent, statins by 5 to 10 percent, and bile-acid-binding resins by 1 to 2 percent, supporting the rationale for combined drug therapy. Patients who have low serum HDL cholesterol concentrations in isolation probably should not be treated unless they have other risk factors for atherosclerosis, existing heart disease, or a family history of heart disease.

Cardiovascular disease accounts for nearly 50 percent of all deaths in the United States. Clinical trials and pathophysiological evidence support the use of aggressive therapy in patients with arteriosclerotic vascular disease and in those with several risk factors for the disease. Combination therapy with lipid-lowering drugs is advisable, especially in patients with combined hyperlipidemia.

Question 2

(B)

A variety of metabolic and pharmacologic agents alter the ECG and, in particular, cause changes in repolarization (ST-T-U) and sometimes QRS prolongation. Certain life-threatening electrolyte disturbances may be diagnosed initially and monitored from the ECG. Hyperkalemia produces a sequence of changes usually beginning with narrowing and peaking (tenting) of the T waves. Further elevation of extracellular K⁺ leads to AV conduction disturbances, diminution in P-wave amplitude, and widening of the QRS interval. Severe hyperkalemia eventually causes cardiac arrest with a slow sinusoidal type of mechanism ("sine-wave" pattern) followed by asystole. Hypokalemia (Fig. 228-19) prolongs ventricular repolarization, often with prominent U waves. Prolongation of the QT interval (Fig. 228-19) is also seen with drugs that increase the duration of the ventricular action potential--type 1A antiarrhythmic agents and related drugs (e.g., quinidine, disopyramide, procainamide, tricyclic antidepressants, phenothiazines) and type III agents (amiodarone, sotalol). Marked QT prolongation, sometimes with deep, wide T-wave inversions, may occur with intracranial bleeds, particularly subarachnoid hemorrhage ("CVA T-wave" pattern) (Fig. 228-19). Systemic hypothermia (Fig. 228-19) also prolongs repolarization, usually with a distinctive convex elevation of the J point (Osborn wave). Hypocalcemia typically prolongs the QT interval (ST portion), while hypercalcemia shortens it (Fig. 228-20). Digitalis glycosides also shorten the QT interval, often with a characteristic "scooping" of the ST-T-wave complex (digitalis effect).

Question 3

(B)

Severe **Marfan's syndrome** is characterised by a triad of features:

- 1) long thin extremities frequently associated with other skeletal changes
- 2) reduced vision as the result of dislocations of the lens (ectopia lentis)
- 3) aortic aneurysms that typically begin at the base of the aorta.

The severe form is usually caused by a mutation in a single allele of the fibrillin gene located on chromosome 15. Marfan's has an incidence of 1 in 10000, of which ¼ are sporadic mutations. The disorder is transmitted as an autosomal dominant trait. Other features include:

- Severe chest deformities (pectus excavatum, carinatum or asymmetry)
- Mitral valve prolapse
- Myopia due to elongation of the globe
- Cataracts result from the lens dislocation
- Skin changes include striae over the shoulders and buttocks

Marfan's must be distinguished from other related syndromes

- 1) Homocystinaemia, which can cause ectopia lentis and the same skeletal changes as Marfan's syndrome
- 2) Congenital contractural arachnodactyly, that causes similar skeletal changes but none of the other features of Marfan's
- 3) Familial ectopia lentis, not associated with any of the other features of Marfan's
- 4) Familial aortic aneurysms, that result from a more common autosomal dominant disorder not associated with other features of Marfan's, type 4 EDS, or other known disorders of connective tissue

The **homocystinaemias** are 7 biochemically and clinically distinct disorders, each characterised by an increased concentration of the amino acid homocysteine in the urine. The most commonest form results from reduced activity of cystathionine beta synthase, an enzyme in the transsulfuration pathway that converts methionine to cysteine. Other enzymes may be involved and depending on which, in some instances clinical improvement can be seen following the administration of specific vitamin supplements (pyridoxine, folate, cobalamin)

When homocysteine and methionine accumulate in the body, cysteine synthesis is impaired. Homocysteine interferes with the normal crosslinking of collagen, an effect that plays an important role in the ocular, skeletal and vascular complications.

More than 80 percent of homozygotes for complete cystathionine synthase deficiency develop dislocated optic lenses. This abnormality usually appears by 3 to 4 years of age and often results in glaucoma and impaired visual acuity. Mental retardation occurs in about half of such patients, often accompanied by ill-defined behavioral disturbances. Osteoporosis is a common radiologic finding (seen in two-thirds of patients by age 15) but rarely causes clinical disease. Life-

threatening vascular complications, probably initiated by damage to vascular endothelium, are the major cause of morbidity and mortality. Occlusion of coronary, renal, and cerebral arteries with attendant tissue infarction can occur during the first decade of life. Nearly a fourth of patients die of vascular disease before age 30. These vascular complications seem to be exacerbated by angiographic procedures. Importantly, pyridoxine-responsive patients have milder clinical manifestations and may escape newborn screening and present with ectopia lentis or premature vascular occlusion. Heterozygous carriers for synthase deficiency (about 1 in 70 in the population) and others with high concentrations of plasma homocystine are at increased risk for premature coronary, peripheral, and cerebral vascular disease.

Question 4

(D)

The diagnosis of hypertriglyceridemia is made by determining levels of plasma lipids after an overnight fast. Because of the less certain association of triglycerides with CHD (compared to LDL cholesterol), plasma concentrations greater than the 90th or 95th percentile for age and sex have been used to define hypertriglyceridemia. Isolated elevations of plasma triglycerides can be due to increased levels of VLDL (type IV) or combinations of VLDL and chylomicrons (type V). Rarely, only chylomicron levels are elevated (type I). Plasma is usually clear when triglyceride levels are <4.5 mmol/L (<400 mg/dL) and becomes cloudy when levels are higher and VLDL (and/or chylomicron) particles become large enough to diffuse light. When chylomicrons are present, a creamy layer floats to the top of plasma after storage in the cold for several hours. Tendon xanthomas and xanthelasma do not occur with isolated hypertriglyceridemia, but eruptive xanthomas (Fig. 341-CD5), small orange-red papules, can appear on the trunk and extremities when triglyceride levels are >11 mmol/L (>1000 mg/dL) (i.e., when chylomicronemia is present). At these high levels of triglycerides, the retinal vessels can appear to be orange-yellow in color (lipemia retinalis). Pancreatitis is the major risk associated with plasma triglyceride concentrations >11 mmol/L (>1000 mg/dL).

Question 5

(A) or (B)

The natural history of AS in the adult consists of a prolonged latent period in which morbidity and mortality are very low. The rate of progression of the stenotic lesion has been estimated in a variety of hemodynamic studies performed largely in patients with moderate AS. Cardiac catheterization and Doppler echocardiographic studies indicate that some patients exhibit a decrease in valve area of 0.1 to 0.3 cm² per year; the average rate of change is \sim 0.12 cm² per year. The systolic pressure gradient across the valve may increase by as much as 10 to 15 mm Hg per year. However, more than half of the reported patients showed little or no progression over a 3- to 9-year period. Although it appears that progression of AS can be more rapid in patients with degenerative calcific disease than in those with congenital or rheumatic disease, it is not possible to predict the rate of progression in an individual patient.

Eventually, symptoms of angina, syncope, or heart failure develop after a long latent period, and the outlook changes dramatically. After onset of symptoms, average survival is <2 to 3 years. Thus, the development of symptoms identifies a critical point in the natural history of AS.

Many asymptomatic patients with severe AS develop symptoms within a few years and require surgery. The incidence of angina, dyspnea, or syncope in asymptomatic patients with Doppler outflow velocities 4 m/s has been reported to be as high as 38% after 2 years and 79% after 3 years. Therefore, patients with severe AS require careful monitoring for development of symptoms and progressive disease.

Sudden death is known to occur in patients with severe AS but has rarely been documented to occur without prior symptoms. Recent prospective echocardiographic studies provide important data on the rarity of sudden death in asymptomatic patients. Although sudden death does occasionally occur in the absence of preceding symptoms in patients with AS, it must be an uncommon event—probably $<1\%$ per year.

Indications for Aortic Valve Replacement

In the vast majority of adults, AVR is the only effective treatment for severe AS. However, younger patients may be candidates for valvotomy (see section VI.A.).

1. Symptomatic Patients. Patients with angina, dyspnea, or syncope exhibit symptomatic improvement and an increase in survival after AVR. Outcome is similar in patients with normal LV function and those with moderate depression of contractile function. The depressed ejection fraction in many patients in this latter group is caused by excessive afterload (afterload mismatch), and LV function improves after AVR in such patients. If LV dysfunction is not caused by afterload mismatch, then improvement in LV function and resolution of symptoms may not be complete after valve replacement. Survival is still improved in this setting with the possible exception of patients with severe LV dysfunction caused by CAD. Therefore, in the absence of serious comorbid conditions, AVR is indicated in virtually all symptomatic patients with severe AS. However, patients with severe LV dysfunction, particularly those with so-called low gradient AS, represent a difficult management decision (see above). AVR should not be performed in such patients when they do not have anatomically severe stenosis. In patients with severe AS, even those with a low transvalvular pressure gradient, AVR results in hemodynamic improvement and

better functional status.

2. Asymptomatic Patients. Management decisions in asymptomatic patients are more controversial. The combined risk of surgery and late complications of a prosthesis generally exceed the possibility of preventing sudden death and prolonging survival in all asymptomatic patients, as discussed previously. Despite these considerations, some difference of opinion persists regarding indications for AVR in asymptomatic patients. It is reasonable to attempt to identify patients who may be at especially high risk of sudden death without surgery, although data supporting this approach are limited. Patients in this subgroup include those with an abnormal response to exercise (eg, hypotension), LV systolic dysfunction or marked/excessive LV hypertrophy, or evidence of severe AS. However, it should be recognized that such "high-risk" patients are rarely asymptomatic.

3. Patients Undergoing Coronary Artery Bypass Surgery. Patients with severe AS, with or without symptoms, who are undergoing coronary artery bypass surgery should undergo AVR at the time of revascularization. Similarly, patients with severe AS undergoing surgery on other valves (such as mitral valve repair) or the aortic root should also undergo AVR as part of the surgical procedure. Patients with moderate AS (for example, gradient 30 mm Hg) may warrant AVR at the time of coronary artery bypass surgery or surgery on the mitral valve or aortic root. However, controversy persists regarding indications for concomitant AVR at the time of coronary artery bypass surgery in patients with milder forms of AS as discussed in section VIII.D.

Aortic Balloon Valvotomy

Percutaneous balloon aortic valvotomy has an important role in treatment of adolescents and young adults with AS (see section VI.A.) but a very limited role in older adults. Immediate hemodynamic results include a moderate reduction in the transvalvular pressure gradient, but the postvalvotomy valve area is rarely >1.0 cm². However, serious complications occur with a frequency of >10%, and restenosis and clinical deterioration occur within 6 to 12 months in most patients. Therefore, in adults with AS, balloon valvotomy is not a substitute for AVR.

Balloon valvotomy can play a temporary role in the management of some symptomatic patients who are not initially candidates for AVR. For example, patients with severe AS and refractory pulmonary edema or cardiogenic shock may benefit from aortic valvuloplasty as a "bridge" to surgery; an improved hemodynamic state may reduce the risks of AVR. Indications for palliative valvotomy in patients with serious comorbid conditions are less well established, but most patients can expect temporary relief of symptoms despite a very limited life expectancy. Asymptomatic patients with severe AS who require urgent noncardiac surgery may be candidates for valvotomy, but most such patients can be successfully treated with more conservative measures.

Question 6

(A)

Question 7

(B)

See immunology questions and answers

Question 8

(B)

Medical therapy should be initiated as soon as the diagnosis is considered. The patient should be admitted to an intensive care unit for monitoring hemodynamics and urine output. Unless hypotension is present, therapy should be aimed at reducing cardiac contractility and systemic arterial pressure, and thereby shear stress. For acute dissection, unless contraindicated, beta-adrenergic blockers should be administered via the parenteral route, using either intravenous propranolol, metoprolol, or the short-acting esmolol to achieve a heart rate of approximately 60 beats per minute. This should be accompanied by sodium nitroprusside infusion to lower systolic blood pressure to 120 mmHg or less. Recently, labetalol (p. 1388), a drug with both beta- and alpha-adrenergic blocking properties, also has been used as a parenteral agent in the acute therapy of dissection.

Question 9

(D)

Valvular Aortic Stenosis

This malformation occurs three to four times more often in males than in females. The congenital bicuspid aortic valve, which is not necessarily stenotic, is one of the most common congenital malformations of the heart, although it may go undetected in early life. Because bicuspid valves may become stenotic with time or be the site of infective endocarditis, the lesion may be difficult to distinguish in adults from acquired rheumatic or degenerative calcific aortic stenosis.

The dynamics of blood flow associated with a congenitally deformed, rigid aortic valve commonly lead to thickening of the cusps and, in later life, to calcification. Hemodynamically significant obstruction causes concentric hypertrophy of the left ventricular wall and dilatation of the ascending aorta.

Question 10

(A)

This man has had a full thickness myocardial infarction with Q waves. The history of dyspepsia may suggest chronic atheromatous plaque => critical stenosis. Plaque rupture => complete occlusion of LAD.
Idiosyncratic ventricular arrhythmia.

(D) could be a possibility because, 50% circumflex and right coronary lesions are pretty much insignificant to cause symptoms. Could have had 70% LAD stenosis which temporarily occluded => reperfusion => idiosyncratic ventricular rhythm at the time. This seems less likely than A.

Question 11

(E)

Second-degree heart block (intermittent AV block) is present when some atrial impulses fail to conduct to the ventricles. Mobitz type I second-degree AV block (AV Wenckebach block) is characterized by progressive PR interval prolongation prior to block of an atrial impulse (Fig. 230-7A). The pause that follows is less than fully compensatory (i.e., is less than two normal sinus intervals), and the PR interval of the first conducted impulse is shorter than the last conducted atrial impulse prior to the blocked P wave. This type of block is almost always localized to the AV node and associated with a normal QRS duration. It is seen most often as a transient abnormality with inferior wall infarction or with drug intoxication, particularly digitalis, beta blockers, and occasionally calcium channel antagonists. This type of block also can be observed in normal individuals with heightened vagal tone. Although Mobitz type I block can progress to complete heart block, this is uncommon, except in the setting of acute inferior wall myocardial infarction. Even when it does, however, the heart block is usually well tolerated because the escape pacemaker usually arises in the proximal His bundle and provides a stable rhythm. As a result, the presence of Mobitz type I second-degree AV block rarely mandates aggressive therapy. Therapeutic decisions depend on the ventricular response and the symptoms of the patient. If the ventricular rate is adequate and the patient is asymptomatic, observation is sufficient.

In Mobitz type II second-degree AV block, conduction fails suddenly and unexpectedly without a preceding change in PR intervals (Fig. 230-7B). It is generally due to disease of the His-Purkinje system and is most often associated with a prolonged QRS duration. It is important to recognize this type of block because it has a high incidence of progression to complete heart block with an unstable, slow, lower escape pacemaker. Therefore, pacemaker implantation is necessary in this condition. Mobitz type II block may occur in the setting of anteroseptal infarction or in the primary or secondary sclerodegenerative or calcific disorders of the fibrous skeleton of the heart. In so-called high-degree AV block there are periods of two or more consecutively blocked P waves, but intermittent conduction can be demonstrated. Block is usually in the His-Purkinje system, but simultaneous block in the AV node may also be present. Regardless of the site of origin of the escape rhythm, if it is slow and the patient is symptomatic, a cardiac pacemaker is mandatory.

Question 12

(A)

? sat 95% in PACWP

Question 13

(D)

