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ARREST AFTER CARDIAC SURGERY Introduction

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The incidence of cardiac arrest after cardiac surgery is around 0.7–8.0%, with 17–79% survival rates. VF accounts for up to 50% of arrests; tamponade and major bleeding account for most others. Multiple variables may dictate di ff erences in the management of cardiac arrest after cardiac surgery when compared with other situations. Therefore, EACTS published guidelines for resuscitation of cardiac arrest after cardiac surgery , which are summarised below .

Acyanotic congenital heart disease

Acyanotic congenital heart disease

- Patent ductus arteriosus The ductus arteriosus, a normal fetal communication, facilitates the shunting of oxygenated blood from the pulmonary artery to the aorta, away from the lungs. Normally , functional closure of the ductus occurs within a few hours of birth; it is abnormal - if it persists beyond the neonatal period. The ductus closes in response to an increase in peripheral oxygen saturation and a drop in the resistance of the pulmonary circulation as the lungs expand; this causes the ductal tissue to contract through a prostaglandin inhibition mechanism. A cyclo-oxygenase inhibitor (e.g. indomethacin) may be used therapeutically to close the ductus in the first few weeks of life. In premature - babies the ductus is more likely to remain patent for longer or permanently . In the isolated case of PDA, there is a left-to-right shunt of blood, resulting in a high pulmonary blood flow . Small shunts usually cause few symptoms and signs apart from the continuous machinery murmur in the left second intercostal space. Larger ducts cause cardiac failure and can uncommonly lead to shunt reversal with cyanosis and clubbing. The diag - nosis is best confirmed by echocardiography with colour flow - Doppler imaging. After 6 months of age, PDA closure is rare. Most should - be closed by preschool age, regardless of symptoms, if the risks of infective endocarditis, left ventricular failure or , rarely , Eisenmenger syndrome are to be avoided. In the adult, surgical treatment is indicated if there is a persistent left-to-right shunt, - even in the presence of reversible pulmonary hypertension. In - the premature infant, if medical treatment to close the duc - tus is unsuccessful, the PDA may be treated by percutaneous interventional cardiology techniques using an umbrella or coil duct occlusion device. If the PDA is very large or the patient very small, surgical closur e via a left thoracotomy is preferred. This can be accomplished by either ligation or division of the PDA. The operative mortality rate is low and outcome gener - ally very good. Coarctation of the aorta - This accounts for 6–7% of congenital heart disease and is defined as a haemodynamically significant narrowing of the aorta, usually in the descending aorta just distal to the left subclavian artery , around the area of the ductus arteriosus (Figure 59.24). The coarctation typically puts a pressure load on the left ventricle, which can ultimately fail. The upper body is well perfused but the lower body , including the kidneys, is poorly perfused, leading to fluid overload, excess renin secre tion and acidosis. Coarctation usually a ff ects boys and, if it occurs in girls, is suggestive of Turner syndrome. In the neonatal period, coarctation (‘infantile’ or preductal coarctation) presents with symptoms of heart failure. The child may appear well in the first few days of life because the coarcta tion is bypassed by the ductus arteriosus and o xygenated blood reaches the systemic circulation. As the ductus closes, the child

becomes progressively more unwell. In adult-type coarctation (juxtaductal or slightly postductal) obstruction is gradual with complications dev eloping in adolescence or early adulthood. Hypertension is a common presenting problem in older chil dren – often upper body hypertension only with development of enormous collateral vessels that may cause rib-notching and flow murmurs over the scapula. Other symptoms include prominent pulsation in the neck, tired legs or intermittent claudication on exercise. Clinical examination of the pulses may demonstrate a radio-femoral delay and a murmur that is continuous and heard best over the thoracic spine or below the left clavicle. The chest radiograph classically demonstrates rib-notching because of dilated posterior intercostal vessels. The heart is usually of normal size in the older child and shows a classical ‘three sign’ replacing the typical aortic knuckle . The upper part of the three sign is the dilated left subclavian, the middle part is the narrowing at the coarctation site and the lower part is the poststenotic dilatation of the descending aorta. presents with cardiac failure, often requiring vigorous medical treatment, including the administration of prostaglandin to reopen the ductus and general resuscitation before corrective surgery . Definitive treatment is usually surgical repair via a left thoracotomy . Coarctation presenting in the child or later typically requires surgical repair, as most patients die before the age of 40 years because of the associated complications. Percutaneous stenting is currently the standard treatment for adults with isolated coarctation. Without correction, the majority of deaths are caused by heart failure, infective endocarditis, rupture of the aorta or haemorrhagic stroke. The preoperative hypertension may not resolve despite surgical repair. Atrial septal defects An ASD is a defect in the septum between the left and right atria leading to a left-to-right shunt, the significance of which is determined by the size of the defect and the relative compli - ance of the ventricles. The development of the atrial septum is complex and abnormalities of development lead to three commonly recognised ASDs (Figure 59.25). The most common type is an ostium secundum ASD. The anomaly is caused by failure of the septum prim um to develop, leading to incomplete coverage of the ostium secundum. These defects are usually asymptomatic in childhood, with symptoms developing insidiously , typically presenting in middle age with congestive cardiac failure secondary to pulmonary hyperten - sion or with atrial arrhythmias. In ostium primum ASD, the anomaly is a form of partial atrioventricular canal defect or endocardial cushion defect. The abnormalities are confined to the atrial septum and are - caused by the endocardial cushions failing to develop and so close the ostium primum part of the interatrial se ptum. The defect is associated with abnormalities of the mitral valve, lead - ing to mitral regurgitation. There is a relatively high incidence of this abnormality in trisomy 21 (Down syndrome). Typically , - -

Coarctation Subclavian artery Ligamentum arteriosum Ascending aorta Pulmonary Intercostal artery

arteries Internal thoracic artery Descending aorta Figure 59.24 Coarctation of the aorta. Coarctation causes severe obstruction of blood /f_l ow in the descending thoracic aorta. The descending aorta and its branches are perfused by collateral channels from the axillary and internal thoracic arteries through the intercostal arteries (arrows). SVC Sinus venosus or superior Tricuspid caval defect valve Fossa ovalis defect Atrioventricular defect IVC Coronary sinus Figure 59.25 Atrial septum viewed from

the right. The fossa ovalis is a useful reference point; the most common defect is in this area and is called a fossa ovalis (or ostium secundum) defect. A defect near the atrioventricular junction may be part of the spectrum of atrioventric

ular septal defects; defects near the entry of the superior vena cava (SVC) are commonly associated with anomalies of venous drainage into the atria. IVC, inferior vena cava.

hood, with dyspnoea, recurrent chest infections and, if pulmo nary hypertension develops, cyanosis. A sinus venosus ASD is a rare defect and is the result of failure of partition of the pulmonary and systemic venous cir culations. These defects are most commonly located high in the atrial septum at the junction of the superior vena cava and the right a trium. They are frequently associated with anoma lous pulmonary venous drainage, with right superior pulmo nary veins draining into the superior vena cava or right atrium directly . Summary box 59.12 Atrial septal defects (ASDs) /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Closure is performed during the first decade of life, even in the absence of symptoms, to avoid late-onset right ventric ular failure, endocarditis and paradoxical emboli. In adults, closure is still appropriate for symptomatic improvement and avoidance of complications. The traditional method of clo sure in volves open-heart surgery with CPB and closure of the defect, either directly with sutures, as with most secundum defects, or, if the defect is large, using a pericardial or synthetic patch. Closure of small to moderate ASDs using percutaneous catheter-delivered devices in the cardiology catheter labora tory is increasingly common. Primum atrioventricular defect repairs may require additional mitral valve repair. The opera tive mortality rate for isolated atrioventricular defect repairs is <1%, with an excellent prognosis. Surgical correction of com plete atrioventricular canal defects, with closure of the ASD and ventricular septal components and mitral v alve repair, is possible, but with a higher surgical mortality rate. Ventricular septal defects A VSD is a defect in the interventricular septum that allows left-to-right shunting of blood. VSDs account for 20–30% of congenital heart

disease and a ff ect approximately 2 in 1000 live births. They may occur in isolation or as part of a more complex set of cardiac abnormalities (e.g. tetralogy of Fallot, Henri Louis Roger , 1809–1891, physician, Hôpital Sainte-Eugene, Paris, France. - Types of ventricular septal defects (VSD) - /uni25CF - - /uni25CF /uni25CF /uni25CF complete atrioventricular canal defect). Four major anatomical types of VSD are described, based on the anatomical subsec - tions of the interventricular septum (Figure 59.26). The VSD permits a ventricular left-to-right shunt, with subsequent right ventricular volume overload and increased pulmonary blood flow . This may lead to progressive pul - monar y oedema and congestive cardiac failure. Persistently elevated pulmonary blood flow and pulmonary vascular resis - tance also lead to irreversible pulmonary h ypertension. They may eventually result in reversal of flow across the defect and Eisenmenger syndrome. The clinical presentation reflects the magnitude of the left-to-right shunt, which, in turn, depends on the size of the VSD and the pulmonary and systemic vascu - - lar resistances. Small defects may close or cause little systemic disturbance (maladie de Roger); infants are asymptomatic with normal development. In the first 5 years, up to 50% of VSDs - close spontaneously . Clinically , a loud pansystolic murmur can - - -

Common defects Ostium secundum: fossa ovalis defect (approximately 70% of ASDs) Ostium primum: atrioventricular septal defect (approximately 20% of ASDs) Sinus venosus defect: often associated with anomalous pulmonary venous drainage (approximately 10% of ASDs) Patent foramen ovale: common in isolation, usually no left-to- right shunt (not strictly an ASD) Rarer defects Inferior vena cava defects: a low sinus venosus defect and may allow shunting of blood into the left atrium Coronary sinus septal defect: also known as unroofed coronary sinus, with the left superior vena cava draining to the left atrium as part of a more complex lesion Perimembranous (conoventricular) defect The most common defect (70–80%), usually located within the membranous septum and may extend to the tricuspid valve annulus or the base of the aortic valve Muscular (trabecular) defect Occurs in 10% of cases and is located within the membranous septum and can be multiple Atrioventricular (inlet) defect Also called an atrioventricular canal-type defect; occurs in 5% of cases and is located in the atrioventricular canal beneath the tricuspid valve Subarterial (outlet) defect Occurs in 5–10% of cases and lies within the conal septum immediately beneath the pulmonary valve annulus Subarterial Perimembranous Atrioventricular Muscular Figure 59.26 Ventricular septum viewed from the right, showing the characteristic sites of ventricular septal defects.

flow between the ventricles. Large defects typically present with congestive cardiac failure in the first 2 months of life. Because of the size of the VSD, ventricular pressures are equalised and often only a soft systolic murmur is detected. If left untreated, pulmonary hypertensive changes start from about 1 year of age. Eisenmenger syndrome, secondary to shunt reversal in such cases, may become evident in the second decade of life. Echocardiography confirms the diagnosis and can estimate the degree of shunting across the defect. Cardiac catheteri sation can quantify right and left cardiac pressures and the degree of pulmonary hypertension, as well as demonstra step-up in oxygen saturation between left and right ventricles. Generally , surgical closur e is indicated for large defects; when there is failure to respond to medical therapy; for left-to-right shunts of >2:1; when there are signs of increasing pulmonary vascular resistance; and in the presence of complications of VSD. These include: (i) aortic regurgitation, which occurs in about 5% of defects; (ii) infundibular stenosis, which tends to be progressive and leads to shunt reversal; and (iii) infective endocarditis, often presenting with pneumonia or pleurisy as the infected ‘emboli’ in a

VSD with a typical left-to-right shunt flows into the pulmonary circulation.

Aetiology

Aetiology

There is often no obvious aetiology; most abnormalities appear to be multifactorial with both genetic and environmental influ ences. There are well-recognised associations. Jacqueline Anne Noonan , 1928–2020, pediatric cardiologist, the University of Kentucky College of Medicine, Lexington, KY , USA, described this condition in 1963. Mary Clayton Holt , 1924–1993, cardiologist, The London Hospital for Women and Children, London, UK. Samuel Oram , 1913–1991, cardiologist, King’s College Hospital, London, UK. Holt and Oram described this syndrome in a joint paper in 1960. John Langdon Haydon Down , 1828–1896, physician, The London Hospital, London, UK. John Hilton Edwards , 1928–2007, Professor of Genetics, University of Oxford, Oxford, UK. Klaus Patau , 1908–1975, German-born American geneticist, University of Wisconsin–Madison, Madison, WI, USA. Henry Hubert Turner , 1892–1970, Professor of Medicine, The University of Oklahoma, Oklahoma City , OK, USA. Harry Klinefelter , 1912–1990, American rheumatologist and endocrinologist, first described the syndrome in 1942. Angelo M DiGeorge , 1921–2009, Professor of Pediatrics, Temple University , Philadelphia, PA, USA. John CP Williams , b. 1922, New Zealand born cardiologist, described the condition in 1961. Recognised associations with congenital heart disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Maternal (environmental) factors Infection: rubella Disease: systemic lupus erythematosus, diabetes mellitus, maternal phenylketonuria Drugs/medications: alcohol abuse, warfarin, phenytoin, lithium, thalidomide Genetic factors Single gene defects: Marfan, Noonan and Holt–Oram syndromes; numerous single-gene disorders Chromosomal defects: trisomy 21 (Down syndrome), trisomy 18 (Edwards syndrome), trisomy 13 (Patau syndrome), Turner syndrome, Klinefelter syndrome Deletions: DiGeorge and Williams syndromes

Aortic dissection

Aortic dissection

This occurs when a defect or flap occurs in the intima of the aorta, resulting in blood tracking into the aortic tissues, splitting the medial layer and creating a false lumen. It most commonly occurs in the ascending aorta or, less often, just distal to the left subclavian artery . It is also more common in men, typically those aged 50–70 years, and in Afro-Caribbean patients. Aetiology It usually occurs as a spontaneous or sporadic event, often in a patient with a history of hypertension. Other important associations include Marfan syndrome and pregnancy . Michael Ellis DeBakey , 1908–2008, American cardiac surgeon, Baylor College of Medicine, Houston, TX, USA. Predisposing factors for aortic dissection /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Clinical features The presentation is often with tearing interscapular pain not unlike the pain of myocardial ischaemia, and it may be di ffi cult to distinguish between the two. The extent of arterial dissection may produce widespread symptoms and signs. The dissection can extend distally down the aorta and spi - ral to involve: /uni25CF the renal arteries (renal pain and renal failure); /uni25CF the mesenteric arteries (abdominal pain and bowel isch - aemia); /uni25CF the spinal arteries (paraplegia); - /uni25CF the iliac arteries (leg pain, pallor, loss of or reduced pulses and acute limb ischaemia). - The dissection may track proximally to involve: /uni25CF the head and neck vessels (symptoms and signs of a stroke or transient ischaemic attack); /uni25CF the coronary vessels (MI); /uni25CF the aortic root (aortic regurgitation). The dissection may also result in aortic wall rupture into the pericardium (cardiac tamponade) or mediastinum (left haemothorax). Classification - There are two classifications, both of which are limited in their application but widely used. The DeBakey classification is based on the pattern of dissection, whereas the Stanford clas - sification is based on whether the ascending aorta is involved (Figure 59.28). Investigations The diagnosis is suspected based on the clinical presentation and careful history taking. Diagnosis is confirmed by CT , which is the standard method for diagnosis. Other imaging modalities such as TOE or MRI can be utilised in cases where the CT is equivocal (Figure 59.29). Management Initial management of all types of aortic dissection includes blood pressure control (which is usually high at presentation)

Age Hypertension Marfan syndrome Pregnancy Other connective tissue disorders, e.g. Ehlers–Danlos syndrome, giant cell arteritis, systemic lupus erythematosus Coarctation of the aorta Turner or Noonan syndromes Aortic cannulation site following previous cardiac surgery (iatrogenic)

and strict pain management, followed by prompt referral for specialist management. The advent of specialist regional centres and regional referral pathways has been shown to improve outcomes in these patients.

I II III (DeBakey) Type B (Stanford) Type A Figure 59.28 Stanford classi /f_i cation of aortic dissections according to whether the ascending aorta is involved (type A) or not (type B). This is simpler than the DeBakey classi /f_i cation (types I, II and III). Figure 59.29 Computed tomography

scan showing acute dissection of the descending thoracic aorta. F , false lumen; T, true lumen.

Aortic valve disease

Aortic valve disease

Approximately two-thirds of all valve surgery performed in the UK is for aortic valve disease, which remains common despite rich countries. - Aortic stenosis The commonest cause of aortic stenosis in adults is an acquired, degenerative, calcific process that results in immobile aortic valve cusps. Progressive fibrosis and calcification of a congeni - tally abnormal valve can mimic this degenerative process. The usual congenital abnormality is commissural fusion, leading to a bicuspid aortic valve, which occurs in approximately 1% of the population (Figure 59.17). - Pathophysiology A pressure gradient develops between the left ventricle and the aorta, with the ventricle adapting to this systolic pressure overload by an increase in wall thickness or hypertrophy . This adaptive response is an attempt to normalise left ventricular wall stress in the face of increased left ventricular systolic pressure and may maintain a normal cardiac output, prevent left ventricular dilatation and avoid significant symptoms for a number of years. Eventually , myocardial function is a ff ected and, together with insu ffi cient left ventricular hypertrophy to normalise wall stress (load mismatch), ventricular contractility is reduced. As aortic stenosis worsens, cardiac output cannot increase with exertion and eventually becomes insu ffi cient at rest. The reduction in ventricular contractility leads to an irreversible decline in left ventricular function, with dilata tion and a rise in left ventricular end-diastolic pressure, to the point of overt left heart failure. The severity of aortic stenosis is shown in Table 59.5 . Clinical features Patients are often asymptomatic until decompensation occurs, typically presenting with dyspnoea and angina due to the increased oxygen needs of the hypertrophied left ventricle, reduced coronary filling and inadequate exertional cardiac output. Patients often describe feeling light-headed or ‘near’ syncope on e ff ort. Arrhythmias can also occur. Auscultation demonstrates an ejection systolic murmur that is typically harsh and best heard over the aortic area with radiation to the carotids. The murmur may become quieter with reduced cardiac output in critical stenosis. The apex beat may be displaced in late disease along with signs of cardiac congestion (Figure 59.18).

(a) (b) Anterior Anterior Anterior Posterior Posterior Bicuspid Rheumatic Posterior Senile calci /f_i ed Figure 59.17 (a) Formaldehyde-treated aortic valve (normal tricuspid con /f_i guration). (b) Aortic stenosis, different pathologies. TABLE 59.5 Classi /f_i cation of the severity of aortic stenosis. Mild Moderate Severe Valve area

> 1.5 1.0–1.5 <1.0 2 (cm) Mean gradient <20 20–40 40 (mmHg) 3.0–4.0 4.0 Velocity (m/s) 2.6–2.9 (<2.5 found in aortic sclerosis) Velocity ratio 0.50 0.25–0.50 <0.25

↑ Investigations /uni25CF ECG : there is left ventricular hypertrophy with tall R waves in the lateral leads and ST depression with inverted T waves – (‘strain pattern’). /uni25CF Chest radiography may

be normal. Cardiomegaly and pulmonary congestion may be seen with left ventricu lar failure. Poststenotic dilatation of the aorta is occasion ally seen (Figure 59.19). /uni25CF Echocardiography confirms the diagnosis and colour flow Doppler imaging allows assessment of the aortic valve gradient, valve area and evaluation of left ventricular dimensions. /uni25CF Coronary angiography : to investigate the coronary arteries in patients >40 years of age. Indications for surgery Medical management focuses on the avoidance of systemic hypotension and arterial vasodilatation, which may reduce myocardial perfusion pressure and provoke ischaemia. The natural history of symptomatic patients with aortic stenosis is dismal, with 10-year mortality around 80–90%. The risk of sudden death is related to the severity of stenosis. Surgery is indica ted in asymptomatic patients with severe stenosis and impaired left ventricular function or when the patient is undergoing concomitant procedures such as CABG. An abnormal blood pressure response to exercise (lo w blood pressure) is also a sign that there is limited reserve in asymp tomatic patients. Aortic regurgitation The causes of aortic regurgitation can be classified according to the speed of development of the regurgitant jet (acute or chronic) or according to the anatomical location of pathology (valve leaflet or aortic wall). The causes of acute aortic regur gitation include infective endocarditis, aortic dissection and trauma. The common causes of chronic aortic regurgitation include degeneration leading to aortic root and/or annular dilatation, congenital bicuspid valve and previous rheumatic fever or endocarditis. Causes are shown in Table 59.6 Pathophysiology In acute aortic regurgitation, backflow of blood increases - ventricular load. It causes a sharp rise in left ventricular - end-diastolic pressure, premature closure of the mitral valve and inadequate forward left ventricular filling. The result is sudden haemodynamic deterioration and acute respiratory compromise. In chronic aortic regurgitation, the left ventricle dilates as a result of volume load, and eccentric hypertrophy is a com - pensatory mechanism to maintain cardiac output. Systolic and diastolic function is abnormal, and sudden deterioration can occur. Clinical features Longstanding aortic regurgitation is usually asymptomatic until left ventricular failure develops, when exertional dyspnoea (predominantly) or angina may develop. A wide pulse pressure due to a reduction in diastolic pressure and a collapsing pulse (water hammer pulse) are commonly seen. - - .

180/0– 10–15 + mmHg mmHg + + + Hypertrophied + + with raised systolic pressure Figure 59.18 Features and pathophysiology of aortic stenosis. Haemodynamic changes in aortic stenosis. Aorta with poststenotic dilatation. Figure 59.19 Chest radiograph in aortic stenosis. TABLE 59.6 Causes of aortic regurgitation. Acute aortic regurgitation Chronic aortic regurgitation Lea /f_l et abnormalities Bicuspid aortic valve Infective endocarditis Calci /f_i c degeneration Prosthetic valve dysfunction Fen /f_l uramine usage (appetite Traumatic lea /f_l et rupture suppressant) Aortic wall abnormalities Aortic wall dissection Calci /f_i c degeneration Aortic trauma Marfan syndrome, Ehlers– Danlos Aortic root dilatation Rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis

/uni2191 /uni2191 Other manifestations of the wide pulse pressure include visible capillary pulsation of the nail bed (Quincke’s sign), pulsatile head bobbing (de Musset’s sign), visible arterial pulsation in the neck (Corrigan’s sign), a ‘pistol shot’ sound on auscultating over the femoral artery (Traube’s sign) and uvular pulsation (Müller’s sign). The apex is displaced laterally and is often visible and hyperdynamic or ‘thrusting’ in nature because of the left ventricular hypertrophy . Auscultation reveals a high- pitched early diastolic murmur best heard at the left sternal edge (Figure 59.20). Investigations /uni25CF ECG : there is left ventricular hypertrophy and sometimes a

‘strain pattern’. /uni25CF Chest radiography : cardiomegaly can be seen if the left ventricle is dilating; sometimes, the aortic shadow may also indicate dilatation. /uni25CF Echocardiography : this allows assessment of the un derlying cause and severity of aortic regurgitation and en ables the diameter of the aortic root as well as left ventric ular dimensions to be determined. Colour flow Doppler imaging quantifies the size of the regurgitant jet. /uni25CF Coronary angiography : to investigate the coronary arteries in patients >40 years of age. Indications for surgery Medical therapy with vasodilator drugs for relief of dyspnoea or angina improves forward stroke volume and reduces regur gitant volume. It is important to note that symptomatic relief does not alter the need for valve surgery . The indications for surgery include sev ere regurgitation in symptomatic patients. Asymptomatic patients with severe aortic regurgitation and left ventricular dysfunction should also be o ff ered surgery . V alve replacement should also be Heinrich Irenaeus Quincke , 1842–1922, Professor of Medicine, Kiel, Germany . Louis Charles Alfred de Musset , 1810–1857, French poet and playwright in whom the sign, traditionally , was first noticed. Sir Dominic John Corrigan , 1802–1880, physician, Jervis Street Hospital, Dublin, Ireland. Ludwig Traube , 1818–1876, physician, The Charité, Berlin, Germany . Friedrich von Müller , 1858–1941, physician, Munich, Germany . if they are undergoing cardiac surgery for any other reason, or when there is evidence of progressiv e left ventricular dilatation (left ventricular end-systolic diameter >50 /uni00A0 mm). Aortic valve replacement is recommended if there is a decrease in systolic function. Summary box 59.10 Causes of aortic valve disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Aortic valve surgery Unlike mitral valve surgery , there are few occasions when the aortic valve can be repaired and usually the valve requires replacement. However, in neonates and children, aortic valve - repair or valvotomy is well established. Percutaneous aortic - balloon valvotomy also has a role in children, but appears to - only result in temporary benefit in adult aortic valve disease. Aortic valve replacement Aortic valve replacement is performed through a median sternotomy or mini-sternotomy on CPB. The aorta is cross- clamped and opened proximally to reveal the diseased valve. Cardioplegic solution is infused into the coronary arteries to arrest the heart in diastole. The valve is excised, leaving the annulus in situ but removing as much calcific debris as possible. - The annulus is sized and the mechanical or biological valve is then placed into position at the level of the native annulus and the aortotomy is closed. The operative mortality rate for elective aortic valve sur - gery is 2–3%. It is higher in emergency surgery , surgery for endocarditis and in older patients.

LAP + 120/0– + mmHg + + + + Figure 59.20 Haemodynamic consequences of aortic regurgitation. The left ventricle dilates and hypertrophies and there is a diastolic murmur. LAP , left atrial pressure. Stenosis Congenital Rheumatic heart disease Acquired calci /f_i cation and /f_i brosis of valve Regurgitation Rheumatic heart disease Infective endocarditis Congenital In /f_l ammatory: Systemic lupus erythematosus Rheumatic ankylosing spondylitis Dilatation of aortic root: Marfan syndrome Dissection Systemic disease: Syphilis Ulcerative colitis

MI (2%) and heart block requiring a permanent pacemaker (<1%). The major determinant of late survival after aortic valve surgery is preoperative left ventricular function. The 5-year survival rate is approximately 75–85%, with the major ity of late deaths related to myocardial factors. Transcatheter aortic valve implantation Although aortic valve replacement is still the gold standard treatment, a significant number of patients a ff ected by severe aortic stenosis do not undergo surgery because they are high risk (owing to age, frailty or heart failure) or because they are a ff

ected by concomitant comorbidities that noticeably increase the operative risk. In such patients TA VI is an attrac tive alternative to standard aortic valve replacement. Other indications include heavily calcified (‘porcelain’) ascending aorta and the presence of severe congenital thoracic wall distortion. T he advances in TA VI techniques and the currently available evidence suggests that TA VI can be an option in intermediate-risk patients. There are di ff erent approaches for valve implantation; the most commonly used are transapical (retrograde) and trans luminal (antegrade). /uni25CF Transapical approach . In transapical TA VI, the car diac apex is prepared through a small left anterolateral mini-thoracotomy using a purse-string or a crossing suture reinforced by pledgets. The device is advanced in the left ventricle between the purse-string sutures. T his approach reduces the risk of calcium dislodgement due to the pas sage of a sti ff transluminal device into a diseased aortic arch. /uni25CF Transluminal approach . This can be carried out via direct access to the aorta, or femoral or subclavian arteries. This is a useful technique for patients with previous cardiac surgery; however, the presence of poor access because of peripheral vascular disease, small vessel diameters, tortu ous vessels, aortic disease or previous aortic surgery con traindicates this approach. Whichever approach is used, a balloon catheter is advanced into the left ventricle over a guidewire and positioned at the aortic valve orifice. The existing aortic valve is dilated in order to make r oom for the prosthetic valve. Rapid right ventricular pacing is used to interrupt cardiac output through the exist ing aortic valve and to reduce movement during implantation. The new valve, mounted on a metal stent, is manipulated into position and is either self-expanding or deployed using balloon inflation. Deployment leads to obliteration of the e xisting aor tic valve. Complications associated with TA VI include mortality (5–18% at 30 days), mild-to-moderate aortic regurgitation (30–50%), stroke (3–9%), perioperative open conversion (9–12%), vascular complica tions (10–15%), atrioventricular block (4–8%) and access artery problems such as bleeding or thrombosis. A recent MI (<3 months), severe pulmonary Robert E Gross , 1905–1988, Surgeon-in-Chief, Cardiovascular Surgery , Children’s Hospital, Boston, MA, USA. Etienne Arthur Louis Fallot , 1850–1911, Professor of Medicine, Marseilles, France. contraindications for transapical TA VI. Interestingly , recent multicentre trials have demonstrated that the role of TA VI may be o ff ered to intermediate-risk patients, with satisfactory - mid-term outcomes.

C A R D I A C M A S S E S

C A R D I A C M A S S E S

Cardiac masses can be either thrombus (blood clots) or tumours. Thrombus can be found in patients with poor left ventricular function or longstanding AF , as well as in patients with proximal pulmonary embolus, in either the ventricles or the left atrium. Cardiac tumours can be either benign or malignant, which in turn can be secondary (from lung, oesophagus, breast, etc.) or primary . J Aidan Carney , b. 1934, County Roscommon, Ireland, pathologist at the Mayo Clinic, described a syndrome of myxomas, spotty pigmentation and endocrine overactivity in 1985. This is the most common benign cardiac mass in adults. Myxo - mas are neoplasms of endocardial origin, often appearing as pedunculated masses most commonly seen in the left (75%) or the right (20%) atria. They are rarely found in the ventricles (Figure 59.31). Myxomas are associated with a congenital disorder (Carne y complex) in 5% of cases. They usually pres - ent with symptoms related to blood flow obstruction through heart valves or systemic embolisation. Treatment is by surgical excision and recurrence rates are usually <5%.

CARDIOPULMONARY BYPASS

CARDIOPULMONARY BYPASS

CPB was first used successfully in 1953 by Gibbon and has since - revolutionised cardiac surgery . It can be used in any procedure in which the heart and lungs need to be stopped temporarily and their function replaced artificially . Before Gibbon’s work, heart surgery was mostly confined to epicardial procedures or e - crude trauma repair. However, valve surgery under direct vision was not possible, nor were the precise reconstructions needed to treat extensive coronary artery disease (CAD). Much of the success of modern CPB is attributable to the development of new biomaterials and sophisticated oxygenating devices, as well as a greater understanding of the pathophysiological consequences of CPB.

CONGENITAL HEART DISEASE Introduction

CONGENITAL HEART DISEASE Introduction

Congenital heart diseases are abnormalities of cardiac struc - ture that are present from birth. Such developmental abnor - malities of the heart typically arise in the third to eighth week - of gestation. The first operation for congenital heart disease was patent ductus arteriosus (PDA) ligation b y Gross in 1938. With the development of neonatal CPB, improved myocardial protection and microsurgical techniques, an increasing number of corrective and palliative operations are possible.

CORONARY ARTERY BYPASS SURGERY

CORONARY ARTERY BYPASS SURGERY

-

Cardiac arrest with ‘non-shockable’ rhythm

Cardiac arrest with ‘non-shockable’ rhythm

Cardiac surgical patients with a non-VF/ventricular tachycar dia (VT) arrest commonly have tamponade, tension pneumo thorax or severe hypovolaemia. Prompt treatment is associated with an excellent outcome. Resternotomy should be performed promptly if connecting the pacemaker and atropine fail to resolve the arrest, especially if a prolonged period of CPR is needed, which will be better performed by internal massage.

Cardiopulmonary bypass circuit

Cardiopulmonary bypass circuit

Once the circuit is connected (Figure 59.1) the CPB machine (‘pump’) gradually takes over circulation and ventilation. Once - tion or full flow is established (the required cardiac output depends on many factors, including the patient’s body surface area and temperature), the ventilator is stopped and the heart can be - culation and stopped. Blood is isolated from the rest of the cir drained from the heart to the venous reservoir using a siphon e ff ect (gravity) as it is usually placed 50–70 /uni00A0 cm below the level of the heart and oxygenated using an oxygenator that allows gas exchange across its membrane. Oxygenated blood is then pumped back to the patient by the bypass machine via the aortic cannula. The patient’s core temperature can be lowered if needed by passing the returning blood through a heat exchanger, - reducing the metabolic demands of the tissues. The degree of cooling is managed according to the severity and complexity of the surgical procedure as well as the surgeon’s preference. Suction pumps can be used to keep the operative field clear. V ents, which are small cannulae that are inserted during sur - gery and connected to the CPB circuit, are used to keep the heart empty by draining any blood that accumulates inside the heart during surgery .

Figure 59.1 The cardiopulmonary bypass circuit shown here in use during ‘on-pump’ cardiac surgery.

Classification

Classification

Congenital heart disease can be broadly classified according to the presence or absence of cyanosis, although the distinction is not always clear-cut. Central cyanosis – blueness of the trunk and mucous membranes – results from levels of deoxygenated haemoglobin of >3–5 /uni00A0 g/dL in the arterial circulation. Cyanotic congenital heart diseases make up 25% of cases (8 or 9/1000 live births) and are usually more complex, - although they do include simple defects. Cyanotic congenital cardiac lesions can involve: monary blood flow . Many lesions consist of septal defects in conjunction with a right-sided obstructive lesion, pro ducing obligatory right-to-left shunts. The most common cause of this is the tetralogy of Fallot. /uni25CF Parallel systemic and pulmonary blood flow . If there is no mixing this is incompatible with life; neonates have a pat ent foramen ovale or VSD that allows some mixing of the two circulations at this level. The most common example of this is TGV . /uni25CF Defects in the connections of the heart in which there is mixing of the systemic and pulmonary flows. An exam ple of such a complex lesion is total anomalous pulmonary venous drainage (TAPVD). Acyanotic congenital heart diseases represent 75% of cases and are usually less complex. They result in an increase in the work imposed on the heart because of either: /uni25CF A left-to-right shunt with increased pulmonary blood flow , causing an increase in volume work of the heart. Examples include PDA, ASD and VSD. /uni25CF Obstruction of blood flow across a left-sided heart valve, such as aortic stenosis, or in the aorta itself, as occurs with coarctation of the aorta, leading to an increase in pressure and work of the heart. Typically , acyanotic congenital heart disease presents as heart failure in infancy because of pulmonary congestion caused by increased pulmonary blood flow or increased pul monary venous blood pressure resulting from an obstructive lesion. The common acyanotic cardiac defects can also present as a murmur in infancy or later. Tetralogy of Fallot - This is the most common cyanotic congenital heart disease in children surviving to 1 year and accounts for about 4–6% of all congenital heart diseases. The four intracardiac lesions - originally described (Figure 59.22) were: /uni25CF VSD; /uni25CF overriding aorta; /uni25CF pulmonary (infundibular or subpulmonary) stenosis; /uni25CF right ventricular hypertrophy . - There may be no initial clinical signs, but, as pulmonary stenosis progresses, cyanosis typically develops within the first year of life. Squatting is an adaptation by the child to hypoxic spells , increasing systemic vascular resistance and the venous return to the heart. Consequently blood is diverted into the pulmonary circulation, increasing oxygenation. Lethargy and tiredness are also common. Plain radiography classically demonstrates a ‘boot-shaped’ heart with poorly developed lung vasculature. The diagnosis is confirmed with echocardi - ography . Surgical correction is the mainstay of treatment and is usually carried out at 4–6 months of age, when possible. Repair is achieved using a patch to close the VSD and resection of the obstructing infundibular se ptum. Surgical results are good, with a late survival rate of 95% at 5–10 years following correction of tetralogy , an operative mortality rate for a repair of between 5% and 10% and an incidence of reoperation fol - - lowing tetralogy repair of 5–10%. Transposition of the great vessels This is the second most common cyanotic congenital heart

disease and most common cause of cyanosis from a congenital cardiac defect discovered in the newborn period. TGV results from abnormal development, with the aorta arising from the right ventricle and the pulmonary artery from the left ventricle (Figure 59.23). The resulting transposition causes pulmonary and systemic circulations to run in parallel rather than in series; oxygenated pulmonary venous blood returns to the lungs and desaturated systemic venous blood is pumped around the body . The situation is incompatible with life and mixing of the

1 Narrowing of the pulmonary valve 3 Displacement of aorta over ventricular septal defect 2 Thickening of wall of right ventricle 4 Ventricular septal defect opening between the left and right ventricles Figure 59.22 Fallot’s tetralogy. Four abnormalities that result in insuf /f_i ciently oxygenated blood being pumped to the body. Aorta Pulmonary Right artery atrium Left ventricle Right ventricle

Figure 59.23 Transposition of the great vessels.

foramen ovale or VSD. Patients often present with severe central cyanosis occur ring within 48 hours of birth. However, if there is a large ASD or VSD there may be minimal cyanosis initially . Typically , progr ess is poor and, as pulmonary vascular resistance declines in the neonatal period, high pulmonary flow develops, with cardiac enlargement and left ventricular failure. The chest radiograph shows pulmonary plethora, with the heart having an ‘egg on its side’ appearance, with a small pedicle (aorta in front of pulmonary artery). Cardiac echocar diography is su ffi cient to confirm the diagnosis and delineate the ana tomy . Many infants will die without treatment within 1 month of birth. Initial stabilisation can be achieved by performing percutaneous balloon septostomy to increase the systemic arterial oxygen saturation. Alternatively , intravenous pros taglandins can be administered to keep the PDA open and increase systemic–pulmonary shunting. Arterial switch repair is currently the standard operation and is typically carried out within the first few weeks of life. Long-term outcomes of the operation are excellent and many patients achieve good exer cise tolerance; however, some patients will require reoperation for neopulmonary stenosis. Total anomalous pulmonary venous drainage TAPVD accounts for l–2% of congenital heart disease. In TAPVD, the pulmonary venous drainage has disconnected from the left atrium and drains into the systemic venous circu lation at some other point (inferior vena cava, superior vena cava, coronary sinus or right atrium). TAPVD presents after the first week of life with cyanosis that is mild to moderate depending on pulmonary flo w . Infants with high pulmonary flow develop cardiac failure, recurrent chest infections, failure to thrive and feeding di ffi culties. If high pulmonary flow is associated with a large ASD, cyanosis is often minimal and the lesion is tolerated well. If there is additional venous obstruc tion, cyanosis presents at birth with dyspnoea and pulmonary oedema. Echocardiography and cardiac (pulmonary) angiog raphy are necessary to confirm the diagnosis and delineate the anomalous drainage. The surgical principle is to re-estab lish the pulmonary venous drainage into the left atrium. The exact operative tech nique depends on the anatom y and type of TAPVD. The long ter m results for survivors of the operation are generally good. Late death following repair is uncommon but, when it occurs, it is often caused by intimal fibroplasia of the pulmonary veins awa y from the anastomosis. Eisenmenger syndrome Eisenmenger syndrome is becoming less common due to development of corrective techniques for congenital heart disease with fewer patients developing a fixed increase in their pulmonary vascular resistance. It follows reversal of a left-to right shunt, that occurs with, for example, a ASD or VSD, such Victor Eisenmenger , 1864–1932, Austrian physician who described this condition in 1897, but the term ‘Eisenmenger syndrome’ was introduced in 1958 by an Australian cardiologist, Paul Hamilton Wood (1907–1962). cyanosis. These congenital anomalies (ASD, VSD) cause an - increase in flow and higher right-sided pressures, which lead to compensatory right ventricular hypertroph y and a subsequent rise in pulmonary artery pressure. Increasing pulmonary hypertension leads to equalisation of pressures either side of the shunt but, at some point, the right-sided pressures will exceed those on the left side, resulting in shunt reversal and desaturated blood entering the left side of the circulation. Cyanosis and dyspnoea are the most common clinical features. - Closure of the shunt is contraindicated if pulmonary hyper - tension is irreversible because the right-to-left shunt now serves to decompress the pulmonary circulation.

Clinical manifestations

Clinical manifestations

The principal symptoms of IHD are chest pain or angina, breathlessness, fatigue, peripheral oedema, palpitations and syncope. The severity of symptoms and the extent to which the symptoms interfere with everyday activities and quality of life are important aspects of the clinical history . An assessment of risk factors should be included. Clinical examination follows and, although often normal, any evidence of myocardial isch aemia such as new murmurs or heart sounds associated with heart failure or stigmata of associated disease, such as diabetes or peripheral vascular disease, should be noted. Summary box 59.4 Risk factors for IHD /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Smoking Hypertension Diabetes mellitus Family history of IHD Advancing age Obesity Male gender Reduced physical activity Hyperlipidaemia

Complications of CPB

Complications of CPB

CPB is a complex technique requiring careful interaction and communication between surgeon, anaesthetist and perfusionist to ensure patient safety . Di ffi culties can occur during cannu lation (aortic dissection or atrial injury), at the start of CPB (oxygenator failure) and at the end of CPB (coagulopathy). Other complications can occur following blood exposur e to the non-physiological surface of the CPB circuit. This leads to the William Dressler , 1890–1969, cardiologist and Director of Cardiology at Maimonides Medical Center, Brooklyn, NY , USA. rise to a post-CPB systemic inflammatory response syndrome (SIRS) that can lead to multiorgan failure. Recent improved under standing of the impact of CPB on coagulation and the inflammatory response (SIRS) has resulted in the development of smaller ‘mini’ CPB circuits, which have demonstrated some advantages in ter ms of reduced post-CPB inflammatory responses and blood transfusion requirements. Alternative methods include surgery ‘o ff -pump’ on a beating heart without the use of CPB; this has some advantages but its use remains restricted to coronary artery bypass grafting (CABG).

Coronary artery anatomy

Coronary artery anatomy

The coronary arteries are branches of the ascending aorta, arising from ostia in the aortic sinuses above the aortic valve, the right from the anterior sinus and the left from the left posterior sinus (Figure 59.2). Summary box 59.3 Coronary artery bypass surgery /uni25CF /uni25CF Left coronary artery The left main coronary artery , which arises from the aortic root, can be the site of significant stenosis (‘left main stem disease’) and carries the worst prognosis in terms of survival without surgery . The artery is inaccessible at its origin and therefore grafts are anastomosed to its branches, the left anterior descending (LAD) artery or anterior interventricular artery and obtuse/marginal (OM) branches of the circumflex artery . The LAD artery is the most frequently diseased coro nary artery and most often bypassed during CABG surgery . Right coronary artery The right coronary artery (RCA) passes from its origin ante riorly between the right atrial appendage and the pulmonary trunk and courses in the atrioventricular groove around the margin of the right ventricle. It usually forms an anastomosis with the circumflex artery at the junction of the right and left atria and the interventricular septum (the crux) on the back of the heart. It continues as the posterior descending artery or interventricular artery . Common sites of stenosis of the RCA are in its proximal portion or at the bifurcation or crux. In the presence of disease a t the bifurcation, a graft can be anastomosed distally to the posterior descending artery . Anatomical dominance is determined by the artery that supplies the posterior descending artery . In approximately 90% of cases the posterior descending artery arises from the RCA, a pattern referred to as right dominance. The posterior descending artery can also arise from the circumflex artery , a pattern referred to as left dominance, which occurs in approx - imately 10% of cases. Co-dominance describes the situation in which there are two posterior descending arteries, one each arising from the right coronary and circumflex arteries; the incidence is around 5%.

Randomised controlled trials have con /f_i rmed improvement in survival following CABG for certain groups of patients Randomised controlled trials have con /f_i rmed

symptomatic bene /f_i ts (relief of angina) following CABG Left atrium Circum /f_l ex branch of left coronary artery Pulmonary cum /f_l ex veins Posterior ventricular branches Inferior vena cava Right coronary artery Posterior Atrioventricular descending node artery artery (b) (a) Anterior surface of the heart; (b) base and diaphrag

Development of the heart and fetal

Development of the heart and fetal

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Diagnosis

Diagnosis

Antenatal diagnosis is occasionally possible, with severe defects detected in utero at 16–18 weeks. If an infant has suspected congenital heart disease, a diagnostic evaluation begins with an accurate history from the parents and specific questions about maternal health and drug use. A detailed family history is important because some defects are familial. Clinical exam - - ination may reveal a murmur, evidence of heart failure, failure to thrive and cyanosis. In addition, congenital heart disease can present with hypertension, an arrhythmia, evidence of polycy - thaemia or a thromboembolic event. Investiga tion is much the same as for the adult patient and, with fetal echocardiography available, cardiac catheterisation is now avoided whene ver possible. -

Emergency resternotomy for ventricular fibrillation

Emergency resternotomy for ventricular fibrillation or pulseless ventricular tachycardia

A precordial thump may be successful if performed within 10 seconds of the onset of VF or pulseless VT; however, this should not delay cardioversion by defibrillation. In VF or pulseless VT , emergency resternotomy should be performed after three failed attempts at defibrillation.

Emergency resternotomy

Emergency resternotomy

After the identification of cardiac arrest, basic life support according to the Advanced Life Support guidelines should be initiated while preparing for emergency resternotomy . Emergency resternotomy may be required in 0.8–2.7% of all patients undergoing cardiac surgery . Emergency resternotomy is a multipractitioner procedure, which should be rapidly performed with a full aseptic technique. Preparation for emergency resternotomy /uni25CF A gown and gloves should be donned in a sterile fashion, but opening should not be delayed in the arrest situation. /uni25CF The drape is applied, ensuring that the whole bed is cov ered (if an all-in-one sterile drape is used then there is no need to prepare the skin with antiseptic). /uni25CF The scalpel is used to cut the sternotomy incision, includ ing all sutures, deeply down to the sternal wires. edges will separate a little, which may relieve tamponade. /uni25CF Suction is used to clear excessive blood or clot. /uni25CF The retractor is placed between the sternal edges and the sternum opened. /uni25CF If cardiac output is restored expert assistance should then be summoned. If there is no cardiac output, the position of any grafts should be carefully identified and internal cardiac massage and internal defibrillation performed, if required. Internal cardiac massage This is a potentially dangerous procedure. Risks include avulsion of a bypass graft, with the LIMA being at particular risk, and right ventricular rupture, especially if it is thin or distended. Therefore, it is important to carefully remove any clot and identify structures at risk such as grafts before placing - hands around the heart. There are several methods of internal - massage; however, the two-hand technique is the safest. Two-hand technique The heart should be inspected to locate the internal mammary and other grafts if present, followed by removal of any blood clots. The right hand is passed over the apex of the heart and then advanced round the apex to the back of the heart, palm up and hand flat. The left hand is then placed flat onto the anterior surface of the heart and the two hands squeezed together at a rate of 100 per minute. Flat palms and straight fingers are important to avoid an unequal distribution of pres - sure onto the heart, thereby minimising the chance of trauma. If there is a mitral valve replacement or repair, care should be taken not to lift the apex by the right hand, as this can cause a posterior ventricular rupture.

FURTHER READING

FURTHER READING

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HISTORICAL PERSPECTIVE

HISTORICAL PERSPECTIVE

Prior to 1925, when Sir Henry Souttar reported the first mitral commissurotomy in the British Medical Journal , heart surgery was thought to be impossible. Souttar wrote that the heart should be as amenable to surgery as any other organ, and the main problem was maintenance of blood flow , particularly to the brain, during surgery . The first real advances occurred in the late 1940s and early 1950s, driven by surgeons who gained confidence and experi ence under the pressures and opportunities provided by war. This was follo wed by the development of cardiopulmonary Sir Henry Sessions Souttar , 1875–1964, surgeon, The London Hospital, London, UK. John Heysham Gibbon , 1903–1973, worked at Je ff erson University , Philadelphia, PA, USA. bypass (CPB) in the mid-1950s, which permitted longer, mor e complex surgery . Recently , the outlook of patients with con - genital, valvular and degenerative heart disease has improved drastically because of advances in the range, complexity and technical expertise in cardiac surgery .

The role of surgery in valvular heart disease • The role of surgery in congenital heart disease • The management of aortic pathology • The management of pericardial disease • The principles of cardiopulmonary resuscitation after • cardiac surgery

Incidence

Incidence

Cardiac defects are the most common congenital abnormalities in the UK; the incidence of significant cardiac abnormalities is 8 per 1000 live births. Many spontaneous abortions or stillbirths have cardiac malformations or chromosomal abnor malities associated with structural heart defects. In neonates and children with congenital heart disease, 15% will have more than one cardiac abnormality and 15% will have another extracardiac abnormality .

Indications for surgery

Indications for surgery

The decision to o ff er CABG is based on the balance between expected benefit and potential risks to the patient. Two issues need to be addressed when determining surgical suitability: the appropriateness of revascularisation and the relative merits of CABG versus the alternative PCI. Current best evidence shows that revascularisation can be readily justified on symptomatic grounds in patients with persistent limiting symptoms (angina or angina equivalent) despite optimal medical therapy and/or on prognostic grounds in certain anatomical patterns of disease. The myocardial revascularisation guidelines of the Euro - pean Society of Cardiology and the European Association for Cardio-Thoracic Surgery (EACTS) can be useful for identify - ing patients with certain angiographic features who can benefit from surgery , such as patients with comple x coronary anatomy or left main stem disease. Summary box 59.6 Indications for surgery /uni25CF /uni25CF /uni25CF . In /uni25CF Acute coronary syndromes - Substantial benefit is gained with an early invasive revascu - larisation strategy with PCI or surgery or both. After defining the anatomy with angiography , a decision about the type and extent of intervention can be made. Angiography in combina - - tion with ECG changes often identifies the culprit lesion and - PCI may be used to treat it. In patients who become stab le after an episode of ACS, the indications for CABG are similar to those for pa tients with stable chronic disease (see Summary box 59.6).

> “ 50% stenosis of the left main stem (‘critical left main stem disease’) 50% stenosis of the proximal left anterior interventricular artery Three main coronary arteries diseased (‘triple-vessel disease’) Two-vessel disease including the proximal LAD Moderate/signi /f_i cant Severe Occluded 50–69% 70% reduction Complete occlusion 75% 90% 100%

and CABG. The benefits of PCI in patients with non-ST seg ment elevation occur with early intervention whereas the ben efits of CABG are greatest when patients undergo surgery after several days of medical stabilisation. However, emergency CABG ma y be indicated for unstable patients with left main stem, multivessel disease and failed PCI. Surgery for the complications of myocardial infarction MI leads to myocyte necrosis that usually heals by formation of scar tissue but may lead to rupture of the ventricular wall. Free rupture of the ventricle is usually fatal. V entricular septal rupture typically presents 3–7 days after infarction with pulmonary oedema, a pansystolic mur mur and haemodynamic instability . Advances in reperfusion therapy such as early access to angiography/PCI services have r educed the incidence to <1%. Diagnosis is usually confirmed with echocardiography , and repair can be performed with a pericardial or artificial Dacron patch in addition to CABG for diseased vessels supplying viable myocardium. Such sur gery is usually associated with significant mortality owing to the associated impairment of the ventricular function. Mitral valve papillary muscle necrosis causes acute mitral regurgita tion.

Diagnosis is made by echocardiograph y , and right heart catheterisation may be required in the presence of poor right ventricular function and high pulmonary pressure . Mitral valve intervention in addition to CABG is usually necessary , but the mortality rate is higher than in valve intervention for non-ischaemic disease. V entricular aneurysm may occur fol lowing partial-thickness necrosis of the ventricular wall if the free wall is replaced with non-contractile fibrous tissue. Left ventricular function is a ff ected because the fibrous wall bal loons out during systole and reduces stroke volume. Repair is undertaken using CPB, and CABG and mitral valv e replace ment may also be necessary . Acute failure of percutaneous coronary angioplasty Since the advent of intracoronary stents, the need for emer gency CABG following complications of PCI is low at <1%. The mortality rate of CABG in this group is significantly higher than in the elective setting.

Initiating cardiopulmonary bypass Arterial cannula

Initiating cardiopulmonary bypass Arterial cannulation

Conventionally , a perfusion cannula is inserted into the ascend ing aorta. Two purse-string sutures are usually placed in the selected area for cannulation after manual or epiaortic scan inspection to ensure that it is clear from severe calcifica atherosclerotic lesions that can prevent safe cannulation or lead to increased risk of postoperative complications such as stroke. The aortic cannula is checked for size and inserted into the aorta between the purse-string sutures and secured by tight ening them. Air is excluded and the cannula connected to the bypass circuit. Alternatively , when it is either inadvisable (aortic dissection), impractical (aortic root surgery) or impossible (severe adhesions or porcelain [calcified] aorta) to cannulate the aorta, alternative cannulation sites can be used, such as the femoral or the axillary artery . The axillary approach has recently been gaining more popularity as it provides more physiological blood flow in the aorta (antegrade) than femoral cannulation, in which blood flow is opposite to normal physi ological conditions (retrograde), and can be utilised to provide selective cerebral perfusion in complex aortic operations. Axillary cannulation has the theoretical advantage of r educing thromboembolic events compared with femoral cannulation. This is related to the di ff erences in the direction of blood flow as flow in femoral cannulation is from the descending aorta to the heart, which means increasing the chances of mobilising calcified plaques from the aorta to the head and neck vessels.

Introduction

INTRODUCTION

Cardiac surgery has developed at a rapid pace since the first procedures in the 1920s. Driven by trauma innovations during the post-war period, the specialty has seen a massive expansion in the range and complexity of conditions treated. Initially thought to be inoperable, surgery for both acquired and congenital heart disease is now commonplace. There are a variety of techniques to address both ischaemic heart disease (IHD) and valvular disease. These ar e often performed in con junction with cardiology colleagues, and minimally invasive approaches are now complementary to surgical techniques. Surgical correction of congenital defects has given rise to a specialty in its own right, and many patients who would pr viously have succumbed to heart disease in infancy now have normal life expectancy . In addition, there are a range of allied technologies that are improving the survival of both adult and paediatric patients undergoing car diac surgery . Transplantation, mechanical assistance devices and extracor poreal circuits are continuing to have improved outcomes and ensure that cardiac surgery is becoming accessible to more patients than ever. Introduction

- Before the 1950s, surgical attempts to treat CAD through grafting of non-coronary flow to the myocardium was via peri - cardial or omental adhesions, with limited success. From the - 1960s onwards, the importance of aortocoronary saphenous vein grafts and the value of the internal mammary (internal thoracic) artery were incr easingly recognised. Outcomes of CABG surgery were carefully scrutinised and, by the 1970s, multiple large, prospectively randomised, multicentre trials were conducted. All trials showed that a subset of patients had - improved survival after surgery , compared with other treat - ments. With the advent of percutaneous coronary intervention (PCI) in the 1980s, the patient population undergoing CABG has changed, becoming progressively sicker but often with the most to gain. Over the last decade, there have been major adv ances in PCI, including the use of several generations of drug-eluting stents, as well as biodegradable stents, in an attempt to reduce restenosis. Although the role of CABG in - the treatment of IHD has been questioned, several multicentre randomised trials carried out comparing CABG with PCI with he drug-eluting stents have clearly shown that CABG remains the gold standard operation in certain groups of patients, such as those with left main stem disease, three-vessel coronary disease, diabetes or those at high risk. - Summary box 59.2 Potential complications of CPB /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF

Coagulopathy Myocardial depression Infection Neurological dysfunction Air embolism Postcardiotomy syndrome (similar to Dressler’s) Gastrointestinal complications (bowel and Pulmonary injury liver ischaemia/pancreatitis) Systemic organ dysfunction Microembolisation (eyes, Vascular injury brain)

Superior vena cava Left coronary artery Branch to Left atrium sinoatrial Left atrial node branch Right atrial branch Cir Right Left atrium marginal branch Right coronary artery Diagonal branches Right Left marginal Left anterior ventricle branch Apex descending artery Figure 59.2 The heart, showing the distribution of the left and right coronary arteries. matic surface of the heart.

Investigations

Investigations

Non-invasive methods of diagnosis Resting electrocardiography As a baseline test, a 12-lead resting electrocardiogram (ECG) often provides the first indication of ischaemic cardiac disease and is essential in the acute clinical setting. However, it may be normal even in the presence of severe multivessel coronary disease. Evidence of previous myocardial infarction (MI) is indicated by Q waves and/or non-specific ST- and T-wave changes and angina by ST depression. Troponin and cardiac isoenzymes These are useful in assessing patients with an acute coronary syndrome (ACS), which is the umbrella term for STEMI (ST elevated myocardial infarction), non-STEMI and unstable angina, especially when the diagnosis is in doubt. Standard enzyme measurement such as troponin, creatine kinase myocardial band and lactate dehydrogenase can also aid both diagnosis and prognosis. Exercise tolerance testing (ETT) is a valuable technique for assessing myocardial ischaemia, both for diagnostic purposes - and as a prognostic tool. However, an abnormal exercise test must be interpreted in the light of the probability of CAD and the physiological response to exercise as measured by the percentage of the maximum predicted heart rate achieved. A positive test with evidence of ischaemia on the ECG (ST depression of ≤ 2 /uni00A0 mm) does not always indicate IHD, and a negative test does not always exclude its presence. ETT should be avoided in patients with cardiac disorders such as aortic - stenosis. Echocardiography Performed through either a transthoracic or transoesophageal approach, echocardiography is valuable for the evaluation of ventricular function and regional wall motion, as well as valvular lesions. Transoesophageal echocardiography provides essential real-time information intraoperatively . Stress echocardiography can detect regional wall motion abnormalities brought on by exercise or the use of dobuta - mine or dipyridamole. It is reliable in identifying viable myo - cardium. Impaired but recoverable myocardium possesses a - functional reserve that allows it to be temporarily recruited into action, whereas scar tissue does not. The development of real- time three-dimensional echocardiograph y (RT3DE) with the ability to carry out valve reconstruction from di ff erent aspects has recently revolutionised preoperative surgical planning in patients with complex valvular lesions. Radionuclide studies and cardiac magnetic resonance imaging The main type of radionuclide study used is myocardial perfusion scanning using specific radioisotopes (such as thal - lium-201) to assess the significance of coronary disease and viability of the myocardium. Cardiac magnetic resonance imaging (MRI) can be per - formed to ev aluate the ischaemic burden of coronary disease (using pharmacological agents to stress the heart) and to provide details of tissue viability when using gadolinium as a contrast agent. Close gap MRI is also very useful in assessing cardiac tumours, pericarditis and other structural heart diseases. Positron emission tomography Positron emission tomography (PET) provides information on myocardial perfusion, metabolism and cell membrane func - tion. Positron-emitting isotopes are used to label physiological substances, which can measure the regional distribution of these substances. PET is valuable in the diagnosis of CAD, particularly when the more widely available imaging modalities are inconclusive. It can identify injured but viable myocardium that is potentially salvageable by revascularisation. Computed tomography With the development of ECG-gated computed tomography (CT) scanners,

multislice high-resolution CT imaging may become an alternative to coronary angiography . It allows assessment of coronary disease, particularly proximal CAD, and gives some information about the degree of coronary artery calcification (calcium score) that is very helpful when stratifying patients to determine which ones will benefit from more invasive coronary angiography . It is also useful in patients in whom angiography is challenging (e.g. di ffi cult anatomy). Invasive methods of diagnosis Coronary angiography Selective coronary angiography remains the gold standard diagnostic technique for accurate diagnosis of the presence and extent of CAD (Figure 59.3). In spite of the availability of newer imaging techniques such as cardiac MRI, selective coronary angiography provides high image quality , demonstrating the extent, severity and location of coronary artery stenoses and the quality and size of the distal coronary arteries. Di ff erent categories of coronary disease are shown in Table 59.1 addition, angiography can assess ventricular function and provide the cardiac surgeon with information to determine operability , operative risk and probability of success. Coronary angiography only outlines the coronary anat omy; it does not demonstrate ischaemia and it carries an overall complication rate of less than 1%. However, flow measurement across a stenotic area, using techniques such as fractional flow reserve, has been e ff ectiv e in predicting those patients who are likely to benefit from revascularisation. Moreover, intravascu lar ultrasound can provide more detailed information regard ing the degree of stenosis, especially in left main stem disease. A reduction in the luminal diameter of ≥ 70% usually means an inability to increase coronary flow above resting values. Coronary angiography /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 59.3 Coronary angiogram demonstrating severe stenosis in the left main stem prior to bifurcation of the left anterior descending and circum /f_l ex arteries. The arrow indicates the area of severe stenosis. TABLE 59.1 Luminal stenosis of coronary arteries and angiographic /f_i ndings. Minimal Mild Angiographic degree of 0% 20–49% stenosis Luminal cross-sectional 0% 40–60% stenosis Gold standard for imaging coronary anatomy Demonstrates extent, severity and location of stenosis Demonstrates quality and size of distal arterial tree Aids diagnosis of ischaemia Evaluates suitability for surgery Aids in prognostic assessment

Ischaemic heart disease

Ischaemic heart disease

IHD is a major cause of morbidity and mortality in resource- rich countries. The underlying pathology is usually atheroscle - rosis of the coronary arteries. Pathophysiology Atherosclerosis is the process underlying the formation of focal - obstructions or plaques in large- and medium-sized arteries. It is a chronic inflammatory process resulting from interactions between plasma lipoproteins, leukocytes (monocyte/macro - phages, T lymphocytes), vascular endothelial cells and smooth - muscle cells. Di ff erent progressive stages of atherosclerosis exist; namely /uni25CF The fatty streak . The first evidence of atherosclerosis can be found in children aged 10–14 years. This appears streak consists of smooth muscle cells, which are filled with cholesterol, and foam cells (lipid-laden macrophages). /uni25CF Fibrous plaque . A fibrous plaque consists of large num bers of smooth muscle cells, foam cells and leukocytes. As the fibrous plaque grows, it projects into the vessel lumen, causing narrowing that, in turn, can lead to ischaemia or infarction. /uni25CF Complicated lesion . This occurs when the fibrous plaque ruptures, provoking activation of the coagulation cascade and the formation of thrombus. The end result is often a calcified ulcerated plaque with areas of haemor rhage and thrombus.

Learning objectives

Learning objectives

To provide an overall view of: The principles of cardiopulmonary bypass • Incisions, conduits and valve options in cardiac surgery • The role of investigation and preoperative assessment in • planning surgery The management of coronary heart disease •

Mitral valve disease

Mitral valve disease

- Mitral regurgitation Any pathological process a ff ecting the mitral valve apparatus may lead to mitral regurgitation. As such, there are many causes of regurgitation and they can be broadly classified into four headings. They are shown in Table 59.4 . Pathophysiology There is an important distinction between acute and chronic mitral regurgitation. The former is usually the result of

Prosthetic valve Streptococcus spp. Aggregatibacter spp., Cardio spp.) Coagulase-negative Staphylococcus spp. Staphylococcus aureus Enterococci Candida Non-tuberculous mycobacteria

ischaemic papillary muscle rupture or following infective endocarditis, whereas the latter is the result of longstanding myxomatous degeneration or fibroelastic changes in the leaflets. In acute mitral regurgitation, the left ventricle ejects blood back into a small, poorly compliant left atrium, imposing a sud den volume load on the left atrium during ventricular systole. This leads to an abrupt rise in left atrial pr essure followed a rise in pulmonary venous pressure and pulmonary oedema. Chronic mitral regurgitation progresses slowly , allowing compensatory left ventricular dilatation and hypertrophy , and atrial dilata tion without significant increase in pressure, pro tecting the pulmonary circulation. As the disease advances left atrial pressure begins to rise, leading to a rise in pulmonary venous pressure and progressive pulmonary congestion, with eventual congestive cardiac failur e. Clinical features In acute mitral regurgitation, the patient is usually unwell, presenting with clinical and radiological evidence of acute ↑ John Brereton Barlow , 1924–2008, South African cardiologist. Bernard Jean Antonin Marfan , 1858–1942, physician, L’Hôpital des Enfants-Malades, Paris, France, described this syndrome in 1896. Edward Ehlers , 1863–1937, Professor of Clinical Dermatology , Copenhagen, Denmark. Henri Alexandre Danlos , 1844–1912, dermatologist, Hôpital St Louis, Paris, France. Christian Johann Doppler , 1803–1853, Professor of Experimental Physics, Vienna, Austria, enunciated the ‘Doppler principle’ in 1842. pulmonary oedema and a loud apical pansystolic murmur. Patients with mild chronic mitral regurgitation are usually asymptomatic. With progressive pulmonary congestion and left ventricular failure, the patient develops fatigue, exertional - dyspnoea and orthopnoea. The development of AF with left atrial dilatation is common. The enlarged left ventricle leads to by a heaving apical impulse and a pansystolic murmur. Investigations /uni25CF ECG : may show left atrial hypertrophy (bifid P waves, - known as ‘P mitrale’), left ventricular hypertrophy and AF . /uni25CF Chest radiography : there may be cardiomegaly with prominent pulmonary vasculature. /uni25CF Echocardiography : this is often combined with colour flow Doppler imaging, which shows the severity of the re - gurgitant jet of mitral regurgitation. /uni25CF Coronary angiography : in patients >40 years of age to investigate the coronary arteries. /uni25CF Cardiac MRI : increasingly popular as it can give detailed information on structure and function. Indications for surgery Indications for surgery in patients with primary mitral regurgitation include severe symptoms or associated changes in left ventricular function or dimension (e.g. left ventricular end-systolic

diameter). Evidence suggests that changes in this setting are usually associated with significant mortality if not corrected (Figure 59.13). It is also recommended to treat severe mitral disease if a patient is undergoing cardiac surgery for a di ff erent reason. Surgical treatment of primary mitral regurgitation usu - ally involves valve repair. When repair is not feasible, valve replacement with attempts to preserve the subvalvular appa - ratus should be considered. The treatment of ischaemic mitral regurgita tion remains controversial and current evidence suggests that patients with

Degenerative causes Ventricular causes Barlow’s disease (myxomatous Transient ischaemia and degeneration) dynamic regurgitation Calci /f_i cation of the lea /f_l ets or annulus Myocardial infarction resulting in papillary muscle rupture Marfan/Ehlers–Danlos syndromes Cardiomyopathy and annular and other connective tissue disorders dilatation 120/80 mmHg + + + 120/ + mmHg Figure 59.13 Features and pathophysiology of mitral regurgitation. There is a loud parasystolic murmur and the left atrium enlarges. The left ventricle enlarges as a consequence of volume overload. Autoimmune and infective Other causes causes Infective endocarditis Trauma (rarely) Rheumatic fever (post- Congenital defects such as streptococcal throat infection) isolated mitral cleft Associated with certain medications (those containing ergotamine) Radiotherapy

replacement, while patients with moderate regurgitation should usually undergo repair along with CABG if indicated. Mitral stenosis The most common cause of mitral stenosis worldwide remains rheumatic fever, despite the fact that the incidence of overt rheumatic fever in resource-rich countries has decreased. During the healing phase of acute rheumatic fever, the valve leaflets become adherent to each other at their free border so that the commissures become obliterated, narrowing the valve orifice. Symptoms of mitral stenosis usually develop more than 10 years after the acute attack. Pathophysiology Mitral stenosis slows diastolic ventricular filling and left atrial pressure rises to maintain cardiac output. This leads to atrial hypertrophy and dilatation. Pulmonary congestion results from the rise in left atrial pressure with time. Although the lungs are protected against pulmonary oedema by constriction of the pulmonary vessels, this adaptive response, along with the passive ‘back pressure’ generated by the rise in left atrial pressure, leads to pulmonary hypertension (>25 /uni00A0 mmHg). This leads to an increased demand on the right ventricle with eventual right heart failure and tricuspid regurgitation. The development of AF is common and can lead to a significant reduction in cardiac output. AF predisposes to thrombi form ing in the left atrium, which may embolise to the systemic circulation. Summary box 59.9 Causes of mitral valve disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Clinical features Some patients may remain asymptomatic for years and then present with symptoms when the heart is stressed by events such as pregnancy , fever, chest infection or with the onset of AF . The common symptoms are fatigue and dyspnoea on exer tion, which result from the combination of reduced forward flow and increased back pressure. The resulting pulmonary congestion adds to breathlessness and may produce a cough or haemoptysis. In sev ere mitral stenosis, there may also be a right ventric ular heave due to right ventricular hypertrophy in response to pulmonary hypertension. Auscultation may reveal an opening - snap soon after the second heart sound, as the diseased valve is opened forcibly by the high pressure in the left atrium. The reverse happens when the valve closes and there is a loud ‘tap - ping’ first heart sound. In addition, a rumbling mid-diastolic murmur can be heard. The duration of the murmur is related to the severity of the mitral stenosis, increasing in length as the stenosis becomes more severe. Investigations /uni25CF ECG may show left atrial enlargement or AF , right axis deviation or

other signs of right ventricular hypertrophy (tall QRS complexes in the right ventricular leads V1–3). /uni25CF Chest radiography : there is a small aortic outline and a prominent pulmonary artery . The left atrium is enlarged (sometimes to an enormous degree) along with upper lobe diversion as a result of the raised pulmonary venous pressure. The right ventricle also appears enlarged (Figure 59.14). /uni25CF Echocardiography , in combination with colour flow Doppler imaging, allows assessment of the flow across the valve and, therefore, the degree of stenosis. Transoesoph - ageal echocardiography (TOE) may be better at assessing valve morphology and excluding the presence of an atrial thrombus. /uni25CF Coronary angiography : to investigate the coronary arteries. - /uni25CF Cardiac MRI . /uni25CF Right heart catheterisation . Indications for surgery Medical management includes anticoagulation in patients - with AF or left atrial enlargement. Tachyarrhythmias should

Stenosis Rheumatic heart disease (common) Calci /f_i cation of valve or chordae tendinae Congenital (rare) Regurgitation Rheumatic heart disease Valve prolapse Left ventricular dilatation or hypertrophy Ischaemia Bacterial endocarditis Figure 59.14 Chest radiograph of longstanding mitral stenosis, showing a massive left atrium.

be treated using pharmacological agents such as digoxin to avoid decompensation and cardiac failure. Diuretics may also provide some benefit. The first-line invasive intervention is balloon valvuloplasty (PMBV); surgery is indicated for severely symptomatic patients who are unsuitable for PMBV or in whom PMBV failed. The prognosis is determined by the severity of the stenosis, the size of the atrium, the presence of AF and rising pulmonary artery pressure (Figure 59.15 Surgical options include mitral valve repair or mitral valve replacement. Formerly common surgical procedures such as closed or open commissurotomy are now rarely performed. Mitral valve operations Approaches to the mitral valve vary; commonly a median sternotomy or, occasionally , right thoracotomy is performed (‘mini’-mitral surgery). The valve can be approached directly through the left atrium in the interatrial groove, through the right atrium and then the interatrial septum or through the left atrial appendage. Mitral valve repair Restoration of normal valve function and preservation of the mitral apparatus is preferable to replacement in specific groups of patients, as it can be associated with improved long-term ventricular remodelling and function. This approach reduces the bleeding complications associated with anticoagulants. The functional classification system developed by Carpentier serves as a guideline in valve reconstruction. It classifies mitral insu ffi ciency into one of three groups according to the ampli tude of the leaflet motion and provides a useful framework for the mechanisms of failure of the mitral valve. As a rule, several valvular lesions or abnormalities are involved in a functional abnormality , with specific techniques developed to correct each lesion. At surgery , the anatomy of the valvular apparatus and sub valvular structures is carefully inspected. The extent of annular dilatation, leaflet prolapse and c hordal dysfunction is assessed. Repair should respect rather than resect tissues, restoring a Alain Carpentier , b. 1933, cardiothoracic surgeon, Hôpital European Georges Pompidou, Paris, France. good coaptation surface between the two leaflets. The mitral valve repair can employ various techniques, including insertion of a prosthetic ring annuloplasty (Figure 59.16); triangular or quadrangular resection of the leaflet; use of a sliding plasty; chordal shortening; chordal transposition; and neochordea implantation. Many techniques exist, indicating that no one technique). addresses all possible findings in mitral regurgitation. Valve repair o ff ers better preserv ation of ventricular function and avoids prolonged anticoagulation, and valve-related complica - tions such as PVE or structural dysfunction. Recent advances in surgical techniques and

the development of di ff erent types of rings has led to increased use of mitral valve repair with excellent results, making it the standar d operation. The oper - ative mortality is 1–3%. One of the major issues related to mitral repair is the incidence of regurgitation recurrence, which varies between series but can be up to 30% at 5 year s. This is related to which leaflet is repaired and the amount of foreign material used in the repair (patch). Mitral valve replacement When valve repair is not feasible, mitral valve replacement is necessary . This usually involves a median sternotomy and access to the left atrium on CPB. The diseased valve is excised and a suitably sized mechanical or bioprosthetic valve is implanted. The atriotomy is closed following de-airing of the left heart. Intraoperative TOE can be used to assess adequate valve function. - The mortality rate for elective mitral valve replacement may be up to 5%, depending largely on the state of the myocar - dium and the general condition of the patient. Common seri - ous in-hospital complications include stroke (<3%) and renal or patients following failure (3%). The longer term prognosis f mitral valve replacement is generally good in comparison with - the natural history of mitral valve disease . Indeed, more recent evidence suggests that patients with ischaemic severe mitral regurgitation can benefit more from valve replacement than from repair.

120/0–5 mmHg Enlarged 20+ left atrium mmHg Thrombus Figure 59.15 Pathophysiology of mitral stenosis. The aorta and left ventricle are relatively small because of chronically reduced cardiac output. The atrium is enlarged and may /f_i brillate, become stagnant and contain a thrombus. The ventricle /f_i lls with a turbulent jet that may be detected as a diastolic murmur or a thrill at the apex. Figure 59.16 Operative view of the mitral valve repair using a Carpentier–Edwards annuloplasty ring (courtesy of A Murday, FRCS).

® The MitraClip is a device used to reduce mitral valve regur gitation. The method involves suturing of the leaflets of the mitral valve together so that regurgitation into the left atrium is prevented. The valve continues to open through the sides of the suture and theref ore blood continues to flow into the left ventricle. Access is usually from the groin where a catheter is inserted in the femoral vein to the right atrium. The left atrium is accessed by making a septal puncture. Although this method is less invasive and associated with rapid recovery and reduced in-hospital stay , it is however technically demanding and long term durability of the results of the device is unknown. ® Data suggest that the MitraClip may be suitable for a small subset of high-risk patients (e.g. chronic heart failure), but the vast majority are better served by surgery that leaves them with substantially less mitral regurgitation.

Myocardial protection

Myocardial protection

Once CPB has been established, the ascending aorta is usually cross-clamped to obtain a bloodless operative field. The heart ceases ejecting and becomes anoxic owing to inhibition of coro - nary blood flow . Permanent myocardial damage can develop within 15–20 minutes, therefore most cardiac operations require some form of myocardial protection. Tec hniques of myocardial protection and the operative management of the myocardium have had a significant impact on the complexity of cardiac surgery . Methods of myocardial protection include intracoronary infusion of a cardioplegic solution (antegrade), infusion via the coronary sinus (retrograde), intermittent cross-clamp fibrillation and total circulatory arrest. Cardioplegia solutions vary in temperature, pH, osmolality and the presence of red cells. Potassium is the most commonly used arresting agent, stopping the heart in diastole by (4–10°C) isotonic crystalloid or blood solutions aid myocardial protection by reducing metabolic requirements through local hypothermia. Warm cardioplegic solutions, on the other hand, may facilitate better myocardial recovery postoperatively by aiding activation of intramyocardial enzymes. Cardioplegia solutions will need to be given repeatedly every 15–20 minutes during surgery . Other cardioplegia solutions that can be given as a single dose are usually reserved for more complex and longer operations. Intermittent cross-clamp fibrillation is a technique in which intermittent ventricular fibrillation (VF) is induced by a small electrical charge. The heart does not eject and is relatively still but not bloodless . The aorta is cross-clamped to render the heart ischaemic. The heart can tolerate short periods (10–20 minutes) of ischaemia, providing it is reperfused when the cross-clamp is released and allowed to beat following cardio version for short periods. Total circulatory arrest is necessary when visibility and clar ity of the operative field is crucial, as in paediatric surgery or in surgery of the ascending arch of the aorta. CPB is established and the core body temperature reduced to 15–18°C (pro found h ypothermia). The metabolic rate of all body organs is reduced by 50% with every 7°C drop in temperature. Using this technique, circulatory arrest (in w hich the CPB machine is switched o ff) can be tolerated for up to 20–30 minutes. Additional cerebral protection can be provided with ice packs placed around the head, pharmacological agents such as thio pental or steroids and cerebral perfusion techniques that allow for longer arrest times. Discontinuing cardiopulmonary bypass At the end of the procedure, air must be meticulously excluded from the cardiac chambers (de-airing). Once perfusion is restored to the coronary arteries (by removing the cross-clamp) the heart may beat spontaneously . If VF is present, cardio version may be required. Epicardial pacing wires are usually placed to treat postoperative bradycardia or heart block. T patient is rewarmed, acidosis and hypokalaemia are corrected and ventilation is restarted. The heart gradually takes over the circulation while the arterial flow from the CPB machine is reduced (‘weaning from bypass’). When the blood pressure is acceptable and the surgeon is confident that the heart func tion is adequate, CPB is discontinued and anticoagulation is reversed by administering protamine and the cannulae are removed.

Outcomes

Outcomes

If type A dissection is untreated, the mortality rate is 50% within 48 hours and 75% within 1–2 weeks, whereas patients with type B dissections have a better prognosis. Surgical mortality is variable but is around 20–25% for proximal aortic dissection. The overall survival rate for patients leaving hospital, regardless of the type of dissection, is around 80% at 5 /uni00A0 years and 40% at 10 years.

PERICARDIAL DISEASES

PERICARDIAL DISEASES

There is a fibrous envelope covering the heart and separating it from the mediastinal structures. This includes a parietal layer and allows the heart to move with each beat. It can be left wide open after cardiac surgery without any ill e ff ects; however, there are a number of conditions a ff ecting the pericardium that may present to the surgeon.

Pericardial effusion

Pericardial effusion

There is continuous production and resorption of pericardial fluid; if this balance is disturbed, a pericardial e ff usion may develop. If the pressure exceeds the pressure in the atria, compression will result in reduced venous return and compro - mised circulation. This state of a ff airs is called tamponade. A gradual build-up of fluid (e.g. malignant infiltration) may be well tolerated for a long period before tamponade occurs, and the pericar dial cavity may contain up to 2 litres of fluid. Acute tamponade (from penetrating trauma, coronary angiography or postoperatively) may occur in minutes with small volumes of blood. The clinical features are low blood pressure with a raised jugular venous pressure and paradoxical pulse. Kuss - - maul’s sign is a characteristic pattern that is seen when the jugular venous pressure rises with inspiration as a result of the impaired venous return to the heart. - Emergency treatment of pericardial tamponade is aspira - tion of the pericar dial space. A wide-bore needle is inserted under local anaesthesia to the left of the xiphisternum, between the angle of the xiphisternum and the ribcage (Figure 59.30). The needle is advanced towards the tip of the scapula into the pericardial space. An ECG electrode attached to the needle will indicate when the heart has been touched. This will relieve the situation temporarily until the cause of the tamponade is established. Penetrating wounds of the heart usually require exploration through a median sternotomy . Emergency room thoracotomy is rarely required. Chronic tamponade is usually a result of malignant infiltration of the pericardium (usually secondary carcinoma from breast or bronchus) or, very occa sionally , uraemia or connective tissue disease. Treatment some times requires a pericardial window between the pericardial space and the pleural or peritoneal space.

Figure 59.30 (a) Pericardial aspiration through the subxiphoid region. (b) Site of needle insertion for pericardial aspiration.

Pericarditis

Pericarditis

Infection and inflammation may also a ff ect the pericardium. Acute pericarditis usually occurs following a viral illness. Treat ment is with non-steroidal anti-inflammatory drugs and bed rest (in case there is an underlying myocarditis). Acute purulent pericarditis is uncommon but requires urgent drainage and intravenous antibiotics, with attention to the underlying cause. Chr onic pericarditis is an uncommon condition in which the pericardium becomes thickened and non-compliant. The heart cannot move freely and the stroke volume is reduced by the constrictive process. The central venous pressure is raised and the liver becomes congested. P eripheral oedema and asci tes are also a feature. Treatment is surgical and is aimed at relieving the constriction.

Postoperative complications

Postoperative complications

Bleeding Significant bleeding occurs in approximately 2–3% of patients. Rarely , acute cardiac tamponade or profound hypotension may occur in the early postoperative period and requires emergency resternotomy . Arrhythmias The most common postoperative arrhythmia is sinus tachy cardia, closely followed by atrial fibrillation (AF). AF occurs in around 30–60% of patients undergoing CABG and often spontaneously reverts to sinus rhythm. Treatment includes correction of potassium (>4.5 /uni00A0 mmol/L), the use of β -b lockers, amiodarone or digoxin and, if necessary , cardioversion. pacing wires inserted intraoperatively may be required in the postoperative period. Poor cardiac output state Myocardial function typically declines in the first few hours - following cardiac surgery , presumably in response to isch - aemia/reperfusion-type injury . Inotropic agents are often required at this time to support heart function and maintain the circulation. Occasionally , the patient develops a persistent low cardiac output state. The clinical manifestations include poor peripheral perfusion, low urine output, a de veloping metabolic acidosis and low blood pressure. There are several mechanisms that may cause this compli - cation in the early postoperative period, including depressed myocardial contractility , reduced preload, increased afterload and a disturbance in heart rate or rhythm. Treatment is aimed at the under lying cause but generally includes oxygenation, optimising preload, reducing afterload, managing any rhythm disturbances and impro ving contrac - tility . If the low cardiac output state persists, the heart may require pharmacological or mechanical support. - Pharmacological support Di ff erent agents can be used to support patients after surgery by altering the systemic vascular resistance, increasing the heart rate and increasing the force of myocardial contractility . Commonly used pharmacological agents include dopamine, dobutamine, adrenaline (epinephrine) and noradrenaline (norepinephrine). Mechanical support If low cardiac output persists despite inotropic support, the heart may require mechanical support while it recovers its function. Mechanical support can be achieved using an intra-aortic balloon pump (IABP), ventricular assist device (V AD) or extracorporeal membrane oxygenation (ECMO). An IABP is a device that is inserted, either percutaneously or under direct vision, into the common femoral artery . It is advanced into the aorta until its tip lies just distal to the aortic arch vessels (Figure 59.7). Balloon filling and emptying is trig - gered by the ECG, deflating during ventricular systole (reduc - ing afterload) and inflating in diastole (displacing blood into the coronary arteries retrogradely). A V AD is a mechanical circulatory supporting device used to replace the function of a failing heart. It can be used as a short-term measure typically f or patients recovering from heart attacks or heart surgery (bridge) or as a long-term support for patients with congestive heart failure (destination). Current V AD devices are all continuous flow and have been shown to be superior to pulsatile flow devices. Blood is exposed in these devices to a non-biological surface that can activate proinflam - matory and coagulation cascades, leading to strokes and bleed - ing. Another important complication associated with V AD is infection. ECMO is another circulatory support device that is similar to CPB; it can be established using venous access only (VV-ECMO) or venous and arterial

access (V A-ECMO). Indications for ECMO include neonates and adults with

(a) Balloon Systole Diastole (b) (b) R R T T P P ECG Aortic pressure Balloon in /f_l ation Diastole Systole Diastole Figure 59.7 Intra-aortic balloon pump counterpulsation. (a) The bal loon de /f_l ates during systole and thereby lowers systemic resistance. It in /f_l ates during diastole and increases coronary perfusion in addition to augmenting the systemic blood pressure. (b) The pressure changes and phases of the electrocardiogram (ECG) are

shown.

support or as a temporary stabilisation method for patients who may need a V AD (bridge therapy). Neurological dysfunction Stroke leading to a focal neurological deficit occurs in approxi mately 2% of patients following CABG. Embolisation, probably originating from the aortic arch or heart chambers, is the most common mechanism for territorial infarcts, with hypoperfu sion leading to wa tershed infarcts. Di ff use neurological injury may also occur, leading to subtle cognitive abnormalities in memory , concentration and attention. Wound infection Significant deep wound infection resulting in sternal dehiscence and mediastinitis occurs in around 0.5–2% of patients. This is associated with significant morbidity , with a prolonged hospital stay and further surgical interventions for debridement and/ or rewiring of the sternum. It has a significant mortality rate of up to 40%. Wound infections are more common in those with diabetes, dialysis patients, smokers, patients with high transfusion requirements and the obese. Mortality In the UK, the mortality rate for patients undergoing CABG is 1–3%. Multiple factors have been demonstrated to a ff ect mortality after CABG, including age, gender, existing morbid ities, left ventricular function and the use of LIMA.

Postoperative management

Postoperative management

Antibiotic prophylaxis Currently the National Institute for Health and Care Excellence recommends that prophylactic antibiotics are not required for patients with prosthetic valves undergoing dental procedures. Other leading European bodies have recently supported the above recommendation. Antithrombotic therapy All patients with mechanical valves require anticoagulation, usually started on the first or second postoperative day . Use of anticoagulants with biological valves is based largely on the manufacturer’s guidance. Direct oral anticoagulants such as apixaban and rivarox - aban are not currently licensed for use with mechanical pros - thetic valves. Warfarin is currently the drug of choice and the target international normalised ratio (INR) should be adapted to patient risk factors and thrombogenicity of the prosthesis with evidence supporting a lower INR target for aortic valves; however, the range of INR can vary between 2.5 and 4.

Postoperative recovery

Postoperative recovery

The majority of patients are extubated a few hours postoper atively and remain in the ICU for 24 hours. In some centres, ‘fast tracking’ appropriate patients allows earlier transfer to a recovery area or high-dependency unit. Discharge is routinely 4–8 days after surgery .

Preparation for surgery

Preparation for surgery

Clinical assessment Before CABG, the severity and stability of the patient’s IHD, the presence of significant valvular disease and the status of left ventricular function should be properly evaluated. Any comorbid risk factors for IHD should be documented and, in particular, the state of coexisting diseases assessed. Attention is paid to the presence of carotid artery disease, peripheral vascu lar disease, respiratory status, preoperative diabetic control and presence of associated diabetic complications, significant renal dysfunction or coagulopathy . All medications taken by Edgar Van Nuys Allen , 1900–1961, Professor of Medicine, Mayo Clinic, Rochester, MN, USA. - surgery (e.g. antiplatelet agents, including aspirin; anticoag - - ulants; and oral hypoglycaemics). Others, including diuretics and angiotensin-converting enzyme inhibitor s, are stopped at the discretion of the surgeon. Cardiac and antihypertensive medications should be taken preoperatively . Risk assessment Myocardial revascularisation by CABG is appropriate when the expected benefits (i.e. survival or health outcomes) exceed the expected negative consequences of the procedure. There - fore, objective methods for risk assessment are essential to determine the patient’s suitability for surgery and to provide patients with adequate information for informed consent. V arious scoring systems have been developed for risk strati - - fication in cardiac surgery , including the EuroSCORE II and the Society of Thoracic Surgeons (STS) score. EuroSCORE II is the system most commonly used in the UK and takes into account di ff erent factors such as age and gender, coexisting conditions such as diabetes and peripheral vascular disease and the proposed operation. -

Primary malignant cardiac tumours

Primary malignant cardiac tumours

These are extremely rare and less common than secondary malignancies. They include angiosarcoma, rhabdomyosar - - coma and leiomyosarcoma. Patients usually have advanced - disease when they are discovered, and they are associated with poor outcomes even with multimodality treatment (surgery and chemoradia tion). - -

Figure 59.31 Transthoracic echocardiography view of a left atrial (LA) myoma (arrow). RA, right atrium.

Prosthetic valve dysfunction and complications

Prosthetic valve dysfunction and complications

Structural valve failure Structural failure rates for the currently used bioprosthetic valves, although rare in those over 70 years of age, can reach 60% after 15 years. Structural failure of a mechanical - valve is generally uncommon. Recently , a new generation of bioprosthetic valves has been introduced with a novel leaflet preparation method. These valves are associated in theory with more durability and can be used for the younger cohort of patients requiring prosthetic valve replacement. The increased utilisation of transcatheter aortic valve inser - tion (TA VI) means that many patients with degenerative pros - thetic valve disease can have a new valve inserted inside the old valve without the need for reoperation. This has encouraged many young patients to select a bioprosthetic valve as the pre - fer red choice for replacement. Paravalvular leak Early-onset paravalvular leaks usually result from technical di ffi culties at insertion. Late-onset leaks can occur and may be precipitated by an episode of endocarditis or by leaflet degen - eration. The leak can cause haemolytic anaemia or haemo - dynamic compromise and the valve may need replacement. Recent improvements in catheter techniques have resulted in the ability to close small areas of paravalvular leak with special occlusion devices, thus reducing the need for r eoperation. Thrombosis and thromboembolism Thrombus formation is the most common complication of a mechanical valve (Figure 59.12). The risk of thromboembo - lism is greater with a mitral valve (mechanical or biological) than with one in the aortic position. The incidence of thromboembolism in current mechanical valves is 0.5–3% per patient-year. Management depends on the extent of the thrombosis and valve dysfunction and can include either thrombolysis or surgery . Prosthetic valve endocarditis The incidence of prosthetic valve endocarditis (PVE) is 2–4%. The risk is lifelong and is at its greatest in the first 3 months after surgery . The incidence of PVE is higher with mechanical and bioprosthetic valves and lowest with homograft and auto graft valves. The diagnosis is suspected following symptoms of septicaemia, development of a new murmur or a septic embolus. It is confirmed with echocardiography , which may show vegetations and even abscess formation. A high index of suspicion is required and early multiple blood cultures are needed to confirm the diagnosis, identify the infective organism and choose appropriate antibiotic therapy . The most common organisms in prosthetic and native valve endocarditis are shown in Table 59.3 . antibiotic therapy . Serial echocardiography to assess extent of infection and involvement of surrounding myocardial tis - sue, as well as functional assessment of the infected valve, may help in optimising decisions on timing of surgical intervention. Multidisciplinary team discussion is essential. The principle of surgical treatment is radical debridement of all infective tissue followed by reconstruction of any defects in the ann ulus and replacement. The prognosis of PVE remains poor, with an overall mortality rate of over 20%.

Figure 59.12 Thrombus (marked T and indicated with arrows) on the moving components of a ball-and-cage valve. TABLE 59.3 Common organisms in infective endocarditis. Classi /f_i cation of organism Native valve Gram-negative bacteria Streptococcus viridans/milleri HACEK (Haemophilus spp., bacterium , Eikenella , Kingella Gram-positive bacteria Staphylococcus aureus/epidermidis Streptococcus faecalis Other Candida Histoplasma Aspergillus

Rhabdomyoma

Rhabdomyoma

Cardiac tumours in children are incredibly rare (<0.2% of the population), although this is the most common benign cardiac tumour. It usually presents with symptoms related to valve dysfunction or arrhythmias. There are usually multicentric pedunculated masses in either or both ventricles. Rhabdomy - oma is associated with tuberous sclerosis in >50% of the cases. Treatment is usually by surgical excision.

Selection of conduit

Selection of conduit

- Venous grafts The long saphenous vein is the most commonly used venous conduit as it is straightforward to harvest, provides good length and is easy to handle. Historical studies showed a limited long - term patency rate for long saphenous vein grafts (50–60% at 10 - years). However, recent studies suggest that early postoperative use of lipid-low ering agents and antiplatelet agents such as low-dose aspirin can improve vein graft long-term patency . In - assessing the patient preoperatively , the legs should be checked for varicose veins. Alternative vein conduits include the short - saphenous vein or upper limb veins such as the cephalic vein; however, these grafts are associated with poorer long-term patency rates. Arterial grafts The left internal mammary artery (LIMA), or internal thoracic - artery , has become the conduit of choice for LAD grafting. Evidence from the mid-1980s to the present day suggests a 10-year patency rate of >95%, with a lower reoperation rate. As this arterial conduit avoids the late complication of vein graft atherosclerosis, interest has focused on the use of bilateral internal mammary artery grafts although there is currently no supporting evidence for this. The use of the radial artery as an alternative arterial bypass graft has undergone a recent revival. This has been driv en by the belief that total arterial revascularisation (avoiding venous conduits) might improve long-term results of coronary surgery . Di ff erent studies have demonstrated excellent patency rates at 1 and 5 years with this strategy . When assessing a patient in - whom a radial artery harvest is planned, an Allen’s test should be performed. The alternative would be vascular assessment of the radial and ulnar arteries with ultrasound. Allen’s test /uni25CF /uni25CF /uni25CF

Figure 59.4 Open long saphenous vein harvesting is performed through an incision starting anteriorly to the medial malleolus of the ankle, extending to the groin if necessary. Figure 59.5 A pedicled left internal mammary artery is dissected off the chest wall and divided distally after systemic heparinisation. It is left attached to the subclavian artery proximally. The patient repeatedly clenches and unclenches the /f_i st while the surgeon compresses both radial and ulnar arteries digitally at the wrist; this empties blood from the hand The hand is then relaxed and compression of the ulnar artery is released; the speed of returning colour to the hand is assessed If colour returns in 5–7 seconds, patency and collateral /f_l ow from the ulnar artery are con /f_i rmed and it is safe to harvest the radial artery

Surgical anatomy

Surgical anatomy

Heart valves serve to maintain pressure gradients between cardiac chambers, thus ensuring a unidirectional flow of blood through the heart. The aortic valve is tricuspid, with semilunar leaflets attached to the aortic wall at the annulus, the aortic sinuses being above the base of each leaflet, two of which form the origin or ostium of the coronary arteries. The intrinsic shape of the aortic semilunar valve allows blood to leave the ventricle during systole and prevents regurgitation during diastole. If disease leads to disruption of the leaflets or the annulus, valve function will be a ff ected. The mitral valve is bicuspid; the anterior cusp is larger in area and lies between the orifices of the mitral and aortic valves. The leaflets, like those of the aortic valve, are attached to an annulus. T he leaflets join at two commissures and are supported by a subvalvular apparatus, consisting of chordae tendinae and papillary muscles. The papillary muscles con tract in ventricular systole, pulling the cusps towards the atrio ventricular orifice and holding blood within the ventricle. The proper functioning of the mitral valve depends on the integrity of the annulus, leaflets, chordae and papillary muscles. If sur gical re pair is required, these structures should be preserved whenever possible (Figure 59.9). Dwight Harken , 1910–1993, American surgeon. In June 1948, in Boston, MA, USA, Harken successfully introduced a cardiovalvulotome through the left atrial appendage and into the heart of a 27-year-old with severe mitral stenosis. In 1950, he developed and implanted the first stainless steel cage prosthesis in the aortic position. Albert Starr , b. 1926, formerly Professor of Surgery , The University of Oregon, OR, USA. Inventor of the world’s first durable artificial mitral valve; winner of Lasker award in 2007 – an award given by the Lasker Foundation in the USA to a person (or persons) who has made major contributions to medical science or who has performed public service on behalf of medicine. The decision to either repair or replace a valve depends on the underlying pathology , severity of disease and quality and/ or involvement of the supporting structures. Generally , repair is favoured when possible in mitral valve disease, particularly in degenerative mitral regurgitation, where it has been shown to have good long-term outcomes. Repair is the operation of choice in tricuspid valve disease, but aortic valve surgery generally involves replacing the diseased valve (Table 59.2). Important factors in selecting the procedure and prosthe - - sis include patient choice, age, existing comorbidities and the need for anticoagulation. Because of uncertainties about its longevity , most surgeons use a bioprosthetic (biological) valve in patients o ver 60 years. The need for anticoagulation with warfarin may have an impact on choice of valve, particularly in women of childbearing age, the elderly , the presence of con - genital or acquired bleeding diathesis and when there is the need for further major surgery .

Pulmonary valve Aortic valve Tricuspid Mitral valve valve Figure 59.9 Four valves of the heart.

Surgical approach to the heart

Surgical approach to the heart

Median sternotomy is the main approach during cardiac surgery . An incision is made from the suprasternal notch to the xiphisternum. The sternum is divided in the midline and retracted, exposing the thymus superiorly and pericardium inferiorly . The atrophic thymus remains relatively vascular. The thymus and pleurae are dissected from the pericardium, which is opened. Before cannulation for CPB, the patient is fully heparinised. Other incisions can be used, including - limited upper or lower sternotomy and left or right anterolateral thoracotomy (in minimally invasive operations or descending aortic surgery). Alternative uses of CPB /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Rewarming in hypothermia Resuscitation in severe respiratory failure As an adjunct in pulmonary embolectomy Single- and double-lung transplantation In cardiopulmonary trauma Certain non-cardiac surgical procedures (e.g. resection of highly vascular tumours or those invading large blood vessels; e.g. the inferior vena cava in renal tumours)

Surgical options

Surgical options

Type A (or type I and II) dissections Those involving the ascending aorta usually require surgical intervention. The chest is opened through a median sternot omy and CPB is commenced, often with core cooling down to 18°C based on the technique used. The aorta is cross-clamped as high up the ascending aorta as possible and opened. Cardio plegic solution is infused into the coronary ostia to arrest the heart in diastole. If the intimal tear is present and localised, the ascending aorta is excised with the tear and replaced with a synthetic g raft. The distal anastomosis is performed with circulatory arr est. Recently there have been attempts to carry out endovascular stenting of type A dissections with variable Adolf Kussmaul , 1822–1902, Professor of Medicine at, successively , Heidelberg, Erlangen, Freiburg and Strasbourg, Germany . the setting of acute type I dissection is debatable and is based on the clinical picture and surgical experience. Type B (or type III) dissections Initially , these are best managed medically with antihyperten - sive drugs and monitoring on an acute care unit. Intervention is indicated in complicated cases if the pain increases (signalling impending rupture) or fails to resolve; or when the dissection is associated with evidence of malperfusion (organ, limb or cutaneously placed neurological symptoms). The use of per endovascular stents is currently the standard intervention of choice in patients with complicated type B dissection, and surgery is reserved for the rare case that is not suitable for stenting.

Surgical outcome

Surgical outcome

Relief of symptoms If revascularisation is complete, CABG alleviates or improves anginal symptoms in more than 90% of patients at 1 year; this falls to 80% at 5 years and 60% at 10 years. This symptomatic deterioration usually reflects progression of atherosclerotic disease in vein grafts and native coronary arteries. Survival Studies have reported survival rates to be >95% at 1 year, 90% at 5 years, 75% at 10 years and 60% at 15 years. These results may improve in the future because of increased use of arterial conduits and widespread use of dual antiplatelet therapy , β -blockers and lipid-lowering agents. Summary box 59.8 Coronary artery bypass surgery outcome /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF CABG without the use of CPB is gaining popularity and may be combined with a minimally invasive approach or carried out through a conventional sternotomy . It avoids the potential physiological stress associated with CPB and, to some extent, - the aortic manipulation that can lead to neurological injury through atherosclerotic embolisation. Since the introduction of ® cardiac stabilising devices such as the Octopus (Figure 59.8), - o ff -pump coronary artery bypass (OPCAB) grafting has become widespread in the UK and around the world. The advantages of o ff -pump surgery over on-pump have recently been questioned, especially with the development of mini-bypass pumps, which o ff er a closed circuit and minimal non-physiological surface area. This reduces proinflammatory activation but at the same time allows the surgeon to oper - ate on a still, bloodless heart. The disadvantages of OPCAB are mainly related to the quality and number of anastomoses. There is still no evidence to support the superiority of any of the above-mentioned techniques and the final decision is usu - ally based on the surgeon’s skills and the required operation. Minimal access surgery Minimally invasive direct coronary artery bypass (MIDCAB) grafting is performed through a small incision and avoids the invasive aspects of conventional CABG. Through an anterior submammary incision the LIMA can be dissected using a - thoracoscope and grafted to the LAD. More lateral incisions allow access to other coronary vessels, including branches of the circumflex artery . Although not yet evidence based, one approach is to combine MIDCAB (typically LIMA to LAD) with PCI to other less accessible coronary arteries (‘hybrid’ coronary revascularisation).

Mortality Survival 1–3%

> “ 95% at 1 year 90% at 5 years Perioperative infarct 75% at 10 years 2–3% 60% at 15 years Angina Improved in >90% at 1 year 80% at 5 years 60% at 10 years

THE THORACIC AORTA

THE THORACIC AORTA

The most common pathologies a ï¬€ecting the thoracic aorta are aneurysm formation and aortic dissection.

The operation

The operation

Intraoperative monitoring includes continuous central venous pressure and blood pressure recording (via a central line in the internal jugular or subclavian vein and radial artery line, respectively), urine output via a urinary catheter, temperature using a nasopharyngeal probe and continuous ECG monitor - ing. The operation commences with harvesting of the conduits (long saphenous vein from the leg [Figure 59.4] and/or radial artery) while the chest is opened via a median sternotomy and the LIMA is dissected from the chest wall (Figure 59.5). The patient is placed on CPB after heparinisation, the aorta is cross-clamped and the heart arrested with cardioplegia. The /uni00A0 grafts are anastomosed to coronary arteries distal to the stenoses (Figure 59.6). The aortic cross-clamp is removed and the heart is reper - fused with oxygenated blood. A side-biting clamp is applied to the ascending aorta and the proximal anastomoses are com - pleted. Occasionally , the surgeon may opt to carry out the whole operation while the cross-clamp is applied to reduce

Left subclavian artery Ve in grafts Left internal mammary artery Figure 59.6 Completed coronary artery bypass grafts.

warmed and weaned from CPB. The heparin is reversed and the patient is transferred to the intensive care unit (ICU).

Thoracic aortic aneurysms

Thoracic aortic aneurysms

A true aneurysm is a localised dilatation of a blood vessel involving all three layers of the vessel wall, whereas a false aneurysm has compressed supporting tissue as its wall and is usually the result of a defect in the vessel intima (from trauma, dissection or previous surgery). Aneurysms are described as fusiform when the whole circumference is a ff ected or saccular when only part of the circumference is involved. When the whole length of a vessel is a ff ected, the clinical and anatomical situation is referred to as ectasia. Aortic aneurysms can develop anywhere along its length, but thoracic aortic aneurysms, including those that extend into the upper abdomen (thoracoabdominal aneurysms), account for 25%, typically occurring in men in the fifth to seventh decade or younger in those with connective tissue disorders. Although a national UK screening pr ogramme exists for abdominal aortic aneurysm, this is not true for thoracic disease. Aetiology The most common aetiology is atherosclerosis, but connective tissue disorders account for many aneurysms in the aortic root and ascending aorta now that tertiary syphilis is rare. Marfan syndrome is associated with cystic medial degeneration involving the vessel wall and causes widening of the proximal aorta and aortic root, leading to aortic valve insu ffi ciency . Hugh Henry Bentall , 1920–2012, Professor of Cardiac Surgery , The Royal Postgraduate Hospital, Hammersmith, London, UK. Sir Magdi Yacoub , b. 1935, Professor of cardiac surgery , Imperial College, UK. Tirone David , b. 1944, Professor of Surgery , Toronto, Canada. associated with aneurysm formation and dissection include Ehlers–Danlos syndrome, which is associated with a range of complica tions including aortic dissection, joint dislocations, scoliosis and osteogenesis imperfecta. Many aneurysms are asymptomatic and are discovered incidentally on routine chest radiographs. Others present as a space-occupying lesion in the thorax with pain caused by pressure on adjacent structures (v ertebra), hoarseness (left - recurrent laryngeal nerve), dysphagia (oesophagus) and respi - ratory symptoms (left main bronchus). Aortic root aneurysms te a may lead to dilatation of the aortic root annulus and aortic regurgitation. Rupture can lead to cardiac tamponade or haemorrhage into the left pleural space, leading to dyspnoea and, if the tra - c heobronchial airway or oesophagus is involved, haemoptysis or haematemesis. Investigations The diagnosis is confirmed by CT or MRI. Arteriography is not necessary for diagnosis but is often required to demonstrate the relation of the arch vessels to the aneurysm. Indications for surgery Without treatment the aneurysm is likely to expand and ultimately rupture. Important factors to consider when plan - ning treatment are age, comorbidity and coexisting coronary disease. In ascending aneurysms, the presence of progressive aortic valve insu ffi ciency is an important indication for surgery . Other indications in this g roup, including Marfan-related aneurysms, are a diameter of 4.5–5 /uni00A0 cm and the presence of symptoms. In descending aneurysms, indications for surgery include symp - toms, acute enlargement and a diameter of approximately 6 /uni00A0 cm. Surgical options The approach adopted for surgical treatment depends on the location of the aneurysm, but typically involves a median sternotomy , CPB and occasionally cooling the patient to 18°C before cross-clamping the aorta above the aneurysm at the distal ascending aorta just before the innominate artery (Figure 59.27). If the aortic root is involved, the

aorta, together with its annulus and valve, is resected and a composite graft is sutured to the aortic root. The circulation is arrested and, after removal of the aortic cross-clamp, the distal anastomosis is completed. The coronary ostia require reimplantation into the graft (Bentall’s operation). More recently there has been increased interest in valve-sparing root surgery or valve repair and root replacement, which is based on two original tech - niques, namely the remodelling technique described by Magdi Yacoub and the reimplantation technique described by Tirone David. These techniques are associated with reduced throm - boembolic complications, but ar e usually demanding with a small increased risk of requiring reoperation at a later stage. If the ascending aorta is involved, it is resected and replaced with a tube graft. For aortic arch aneurysms, surgery on this section of the aorta is a formidable undertaking because the cerebral and subclavian vessels have to be anastomosed to the graft, either separately or en bloc. Typically , it involves a period of circulatory arrest and some form of cerebral protection. Excision of a descending aortic aneurysm is with graft replace ment under CPB, with exposure via a left thoracotomy or with a heparin-bonded shunt. Increasingly , thoracic aneurysms at the aortic arch or more distal are repaired using a percuta neous approac h via the femoral artery , with insertion of an endovascular stent graft under radiological guidance. Surgical outcome The operative mortality rate is variable depending on the location and type of repair required, but electively is between 5% and 15% and is considerably higher in emergency repairs. Long-term survival depends on underlying pathology but, for ascending aneurysm repairs, the 5-year survival rate is approximately 65%. The major complications of descending aneurysm repairs include paraplegia, renal failure and ventric ular dysfunction.

Figure 59.27 A large thoracic aortic aneurysm.

Types of prosthetic valves

Types of prosthetic valves

Mechanical valves Mechanical valves can be used in any age group to replace any valve (Figure 59.10). They are extremely durable but thrombogenic and patients require systemic anticoagulation, usually with warfarin. The patient should be warned about the risk of haemorrhagic (intracerebral, epistaxis, gastrointestinal bleed) or thrombotic (cerebral infarction) complications. Bioprosthetic (biological) valves - - Bioprosthetic valves include cadaveric homograft (or allograft) valves; autografts , a patient’s own valve; and, most commonly , heterografts (or xenografts) prepared - from animal tissues. All have three semilunar leaflets with central flow , so decreasing pressure gradients and minimising turbulence (Figure 59.11). Heterograft ‘tissue’ valves are the

Figure 59.10 Bilea /f_l et mechanical valve.

most commonly used valves and can be stented with a limited durability of 10–15 years, whereas stentless (or frameless) valves are expected to have less late calcific degeneration but are more technically di ffi cult to insert. Sutureless and rapid deployment valves In recent years, there has been an increase in the number of available valves using rapid deployment and sutureless technol ogy . These valves are quicker to implant as they do not require extensive numbers of sutures (usually three in the case of rapid deployment and none in the case of sutureless). These valves are anchor ed in position with a balloon inflatable stent. This is advantageous in elderly or high-risk patients and in minimally invasive aortic surgery .

Valve repair Mechanical replacement Biological valves Stented Advantages No need for long-term anticoagulation Can be used in younger No need for long-term Good evidence base patients anticoagulation Good history of evidence Mimics ‘natural’ haemodynamics Disadvantages Technically challenging Nidus of infection (endocarditis), can be disastrous Requires anticoagulation Lifespan Excellent long-term Lifespan limited (traditionally 10–15 years, although Variable among durability (patient lifetime) constantly improving) techniques and valve More suited to older patient involved Comments Mostly made of bovine or porcine pericardium Many different types Mostly performed for Gr owing evidence for the use of antiplatelet agents and sizes for a range of mitral valve disease postoperatively scenarios Evidence for other valves is limited Figure 59.11 Porcine heterograft stented valve. Stentless Homograft Does not require anticoagulation Long-term results unknown Technically more Requires specialist challenging to insert expertise Increased complexity of surgery Little evidence, although may be limited Usually taken from deceased donors

VALVULAR HEART DISEASE Introduction

VALVULAR HEART DISEASE Introduction

Early surgical management of valvular heart disease concen - trated on valve repair. The heroic early procedures for valve stenosis were closed and therefore ‘blind’ commissurotomies

® Figure 59.8 Off-pump coronary artery bypass using an Octopus stabiliser to perform the distal anastomosis.

procedures with full visualisation, allowing precise repair and replacement. The first prosthetic valve replacement was performed by Dwight Harken, who replaced an aortic valve, followed by a mitral valve replacement by Starr a year later. Continued improvements in perioperative care, myocardial protection and, in particular, the development of prosthetic heart valves have improved long-term haemodynamic results, provided symptom relief and prolonged survival. The majority of valvular operations involve surgery on the aortic or mitral valve; tricuspid and pulmonary valve surgery is rarely under taken in isolation unless it is part of staged congenital surgery .

Venous cannulation

Venous cannulation

A single purse-string suture is placed around the right atrial appendage and a single ‘two-stage’ venous cannula is placed to establish venous drainage. The venous pipe has end holes that sit in the inferior vena cava and side holes that sit in the right atrium (to drain from the superior vena cava). Alternatively , the superior and inferior venae cavae may be cannulated separately to gain better control over the venous return and facilitate operating on structures in the right ventricle or atrium (‘bicaval’ cannulation). V enous drainage from the femoral vein can o ff er an alternative, particularly during thoracic aortic or minimally invasive procedures.

circulation and circulatory changes at birth

circulation and circulatory changes at birth

- By 12 weeks of fetal life the primitive vascular tube is fully developed. Fetal circulation di ff ers from that of the adult in that the right and left ventricles pump blood in parallel rather than in series. This arrangement allows the heart and head to receive more highly oxygenated blood. This is possible because - of the presence of three structural shunts: the ductus venosus, foramen ovale and ductus arteriosus (Figure 59.21). Soon after birth, pulmonary vascular resistance falls because of the action of breathing and resulting pulmonary vasodilatation. Within 30 minutes of delivery , the ductus arte - riosus constricts in r esponse to increasing blood oxygen levels. The result is a reversal of the pulmonary–systemic pressure - gradient and termination of blood flow from the pulmonary - artery into the aorta. After birth, cutting and tying of the umbilical cord stops venous blood flow from the placenta. This lowers inferior vena ca va pressure and, with falling pulmonary vascular resistance, right atrial pressure falls. The result is closure of the foramen ovale. The abolition of venous return from the placenta also - causes the ductus venosus to close. Closure of the fetal circulatory shunts in the few hours following birth is functional, with complete structural closure typically taking several months. In 20% of adults the structural - closure of the f oramen ovale remains incomplete, but is of no cardiovascular significance. Abnormalities of cardiac structure may arise from the per - sistence of normal fetal channels (PDA, patent foramen ovale), failure of septation (atrial septal defect [ASD], VSD, tetral - ogy of Fallot), stenosis (intracardiac, supravalvular, valvular, infra valvular or extracardiac coarctation of the aorta), atresia or abnormal connections (transposition of the g reat vessels (TGV), total anomalous venous drainage). Fetal echocardiog raphy is now su ffi ciently sensitive to detect intracardiac lesions in the second trimester.

Ductus arteriosus Foramen ovale Ductus venosus Figure 59.21 Fetal circulation.