Aortic valve disease

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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.

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