Prosthetic valves

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- Diseased heart valves can be replaced with mechanical or biological prostheses. The three most commonly used types of mechanical prosthesis are the ball and cage, tilting single disc and tilting bi-leaflet valves. All generate prosthetic sounds or clicks on auscultation. Pig or allograft valves mounted on a supporting stent are the most commonly used biological valves. They generate normal heart sounds. All prosthetic valves used in the aortic position produce a systolic flow murmur.
- All mechanical valves require long-term anticoagulation because they can cause systemic thromboembolism or may develop valve thrombosis or obstruction; the prosthetic clicks may become inaudible if the valve malfunctions.

- Biological valves have the advantage of not requiring anticoagulants to maintain proper function; however, many patients undergoing valve replacement surgery, especially mitral valve replacement, will have AF that requires anticoagulation anyway. Biological valves are less durable than mechanical valves and may degenerate 7 or more years after implantation, particularly when used in the mitral position. They are more durable in the aortic position and in older patients, so are particularly appropriate for patients over 65 undergoing aortic valve replacement.

Prosthetic valve dysfunction

- Symptoms or signs of unexplained heart failure in a patient with a prosthetic heart valve may be due to valve dysfunction, and urgent assessment is required. Metallic valves can suffer strut fracture and fail, causing catastrophic regurgitation. Alternatively, they may thrombose and cause systemic thromboembolism or valve obstruction, especially in the presence of inadequate anticoagulation. Biological valve dysfunction is usually associated with the development of a regurgitant murmur and may begin to develop 8–10 years after implantation.

16.92 Anticoagulation targets and prosthetic heart valves

Mechanical valves	Target INR
Ball and cage (e.g. Starr–Edwards) **Tilting disc** (e.g. Bjork–Shiley)	3.0–4.0
Bi-leaflet (e.g. St Jude)	2.5–3.0
Biological valves with atrial fibrillation	2.0–3.0

(INR = international normalised ratio)

Infective endocarditis

- Infective endocarditis is caused by microbial infection of a heart valve, the lining of a cardiac chamber or blood vessel, or by a congenital anomaly. Both native and prosthetic valves can be affected. The most common causes of infective endocarditis are streptococci and staphylococci but other organisms may also be involved.
- Epidemiology :
- Underlying condition was rheumatic heart disease in 24% of patients, congenital heart disease in 19%, and other cardiac abnormalities such as calcified aortic valve or floppy mitral valve in 25%. The remaining 32% were not thought to have a pre-existing cardiac abnormality.
- Bacterial endocarditis is a serious illness; the case fatality is approximately 20% even with treatment, and is even higher in those with prosthetic valve endocarditis and those infected with antibiotic-resistant organisms.

- Pathophysiology:
- Infective endocarditis typically occurs at sites of pre-existing endocardial damage, but infection with particularly virulent or aggressive organisms such as Staphylococcus aureus can cause endocarditis in a previously normal heart.
- Staphylococcal endocarditis of the tricuspid valve is a common complication of intravenous drug use.
- Many acquired and congenital cardiac lesions are vulnerable, particularly areas of endocardial damage caused by a high-pressure jet of blood, such as ventricular septal defect, mitral regurgitation and aortic regurgitation, many of which are haemodynamically insignificant. In contrast, the risk of endocarditis at the site of haemodynamically important low pressure lesions, such as a large atrial septal defect, is minimal.

- Infection tends to occur at sites of endothelial damage because they attract deposits of platelets and fibrin that are vulnerable to colonisation by blood-borne organisms. The avascular valve tissue and presence of fibrin and platelet aggregates help to protect proliferating organisms from host defense mechanisms.
- Valve regurgitation may develop or increase if the affected valve is damaged by tissue distortion, cusp perforation or disruption of chordae. Extracardiac manifestations, such as vasculitis and skin lesions, may occur as the result of either emboli or immune complex deposition. Mycotic aneurysms may develop in arteries at the site of infected emboli.
- Splenic and renal infarctions may occur.

16.94 Microbiology of infective endocarditis

Pathogen	Of native valve (n = 280)	In injection drug users (n = 87)	Of prosthetic valve	
			Early (n = 15)	Late (n = 72)
Staphylococci	124 (44%)	60 (69%)	10 (67%)	33 (46%)
Staph. aureus	106 (38%)	60 (69%)	3 (20%)	15 (21%)
Coagulase-negative	18 (6%)	0	7 (47%)	18 (25%)
Streptococci	86 (31%)	7 (8%)	0	25 (35%)
Oral	59 (21%)	3 (3%)	0	19 (26%)
Others (non-enterococcal)	27 (10%)	4 (5%)	0	6 (8%)
***Enterococcus* spp.**	21 (8%)	2 (2%)	1 (7%)	5 (7%)
HACEK	12 (4%)	0	0	1 (1%)
Polymicrobial	6 (2%)	8 (9%)	0	1 (1%)
Other bacteria	12 (4%)	4 (5%)	0	2 (3%)
Fungi	3 (1%)	2 (2%)	0	0
Negative blood culture	16 (6%)	4 (5%)	4 (27%)	5 (7%)

(HACEK = ***H****aemophilus aphrophilus* – now known as *Aggregatibacter aphrophilus*–***A****ggregatibacter actinomycetemcomitans*; ***C****ardiobacterium hominis*; ***E****ikenella corrodens*; and ***K****ingella kingae*)

Adapted from Moreillon P, Que YA. Infective endocarditis. Lancet 2004; 363:139–149.

Clinical features

- Endocarditis can take either an acute or a more insidious ‘subacute’ form. There is considerable overlap, however, because the clinical pattern is influenced not only by the organism but also by the site of infection, prior antibiotic therapy and the presence of a valve or shunt prosthesis. The subacute form may abruptly develop acute life-threatening complications, such as valve disruption or emboli.

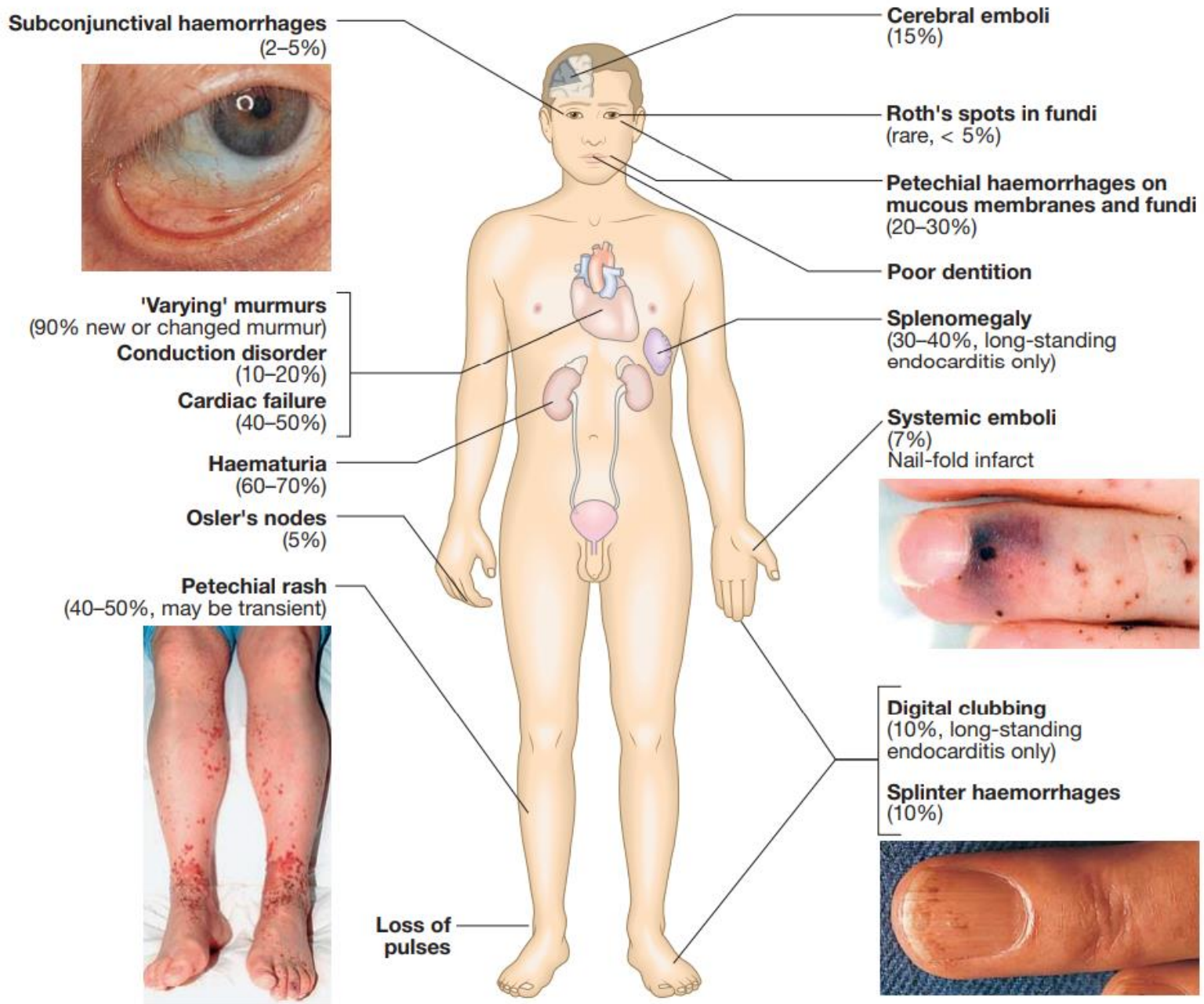

Fig. 16.89 **Clinical features that may be present in endocarditis.** *Insets (Petechial rash, nail-fold infarct) From Newby D, Grubb N. Cardiology: an illustrated colour text. Edinburgh: Churchill Livingstone, Elsevier Ltd; 2005.*

16.95 Diagnosis of infective endocarditis*

Major criteria

Positive blood culture

- Typical organism from two cultures
- Persistent positive blood cultures taken >12 hrs apart
- Three or more positive cultures taken over >1 hr

Endocardial involvement

- Positive echocardiographic findings of vegetations
- New valvular regurgitation

Minor criteria

- Predisposing valvular or cardiac abnormality
- Intravenous drug misuse
- Pyrexia ≥38°C
- Embolic phenomenon
- Vasculitic phenomenon
- Blood cultures suggestive: organism grown but not achieving major criteria
- Suggestive echocardiographic findings

*Modified Duke criteria. Patients with two major, or one major and three minor, or five minor have definite endocarditis. Patients with one major and one minor, or three minor have possible endocarditis.

- Subacute endocarditis
- This should be suspected when a patient with congenital or valvular heart disease develops a persistent fever, complains of unusual tiredness, night sweats or weight loss, or develops new signs of valve dysfunction or heart failure. Less often, it presents as an embolic stroke or peripheral arterial embolism. Other features include purpura and petechial haemorrhages in the skin and mucous membranes, and splinter haemorrhages under the fingernails or toenails. Osler's nodes are painful, tender swellings at the fingertips that are probably the product of vasculitis; they are rare. Digital clubbing is a late sign.
- The spleen is frequently palpable; in Coxiella infections, the spleen and the liver may be considerably enlarged. Non-visible haematuria is common. The finding of any of these features in a patient with persistent fever or malaise is an indication for re-examination to detect hitherto unrecognised heart disease.

- Acute endocarditis

This presents as a severe febrile illness with prominent and changing heart murmurs and petechiae. Clinical stigmata of chronic endocarditis are usually absent. Embolic events are common, and cardiac or renal failure may develop rapidly. Abscesses may be detected on echocardiography. Partially treated acute endocarditis behaves like subacute endocarditis.

- Post-operative endocarditis

This may present as an unexplained fever in a patient who has had heart valve surgery. The infection usually involves the valve ring and may resemble subacute or acute endocarditis, depending on the virulence of the organism. Morbidity and mortality are high and revision surgery is often required. The range of organisms is similar to that seen in native valve disease, but when endocarditis occurs during the first few weeks after surgery it is usually due to infection with a coagulase-negative staphylococcus that was introduced during the perioperative period.

- Investigations

Blood culture :

Three to six sets of blood cultures should be taken prior to commencing therapy and should not wait for episodes of pyrexia. The first two specimens will detect bacteraemia in 90% of culture-positive cases. A meticulous aseptic technique is essential. Taking discrete sets of blood cultures from peripheral sites at intervals of ≥ 6 hours reduces the risk of misdiagnosis due to contamination with skin commensals. Isolation of a typical organism in more than one culture provides strong evidence in favour of the diagnosis. An in-dwelling line should not be used to take cultures. Both aerobic and anaerobic cultures are required.

- Echocardiography

It is key for detecting and following the progress of vegetations, for assessing valve damage and for detecting abscess formation. Vegetations as small as 2–4 mm can be detected by transthoracic echocardiography, and even smaller ones (1–1.5 mm) can be visualised by trans-oesophageal echocardiography (TOE), which is particularly valuable for identifying abscess formation and investigating patients with prosthetic heart valves. Vegetations may be difficult to distinguish in the presence of an abnormal valve; the sensitivity of transthoracic echo is approximately 65% but that of TOE is more than 90%. **Failure to detect vegetations does not exclude the diagnosis.**

- Elevation of the ESR, a normocytic normochromic anaemia, and leucocytosis are common but not invariable. Measurement of serum CRP is more reliable than the ESR in monitoring progress. Proteinuria may occur and non-visible haematuria is usually present.
- The ECG may show the development of **AV block (due to aortic root abscess formation)** and occasionally infarction due to emboli. The chest X-ray may show evidence of cardiac failure and cardiomegaly.

16.96 Antimicrobial treatment of common causative organisms in infective endocarditis

Antimicrobial susceptibility	Antimicrobial	Dose	Duration	
			Native valve	Prosthetic valve
Streptococci				
Penicillin MIC ≤0.125 mg/L	Benzylpenicillin IV	1.2 g 6 times daily	4 weeks[1]	6 weeks
Penicillin MIC >0.125, ≤0.5 mg/L	Benzylpenicillin IV and gentamicin IV	2.4 g 6 times daily 1 mg/kg twice daily[2]	4 weeks 2 weeks	6 weeks 2 weeks
Penicillin MIC >0.5 mg/L	Vancomycin IV and gentamicin IV	1 g twice daily[3] 1 mg/kg twice daily[2]	4 weeks 4 weeks	6 weeks 6 weeks
Enterococci				
Amoxicillin MIC ≤4 mg/L and gentamicin MIC ≤128 mg/L	Amoxicillin IV and gentamicin IV[2]	2 g 6 times daily 1 mg/kg twice daily[2]	4 weeks 4 weeks	6 weeks 6 weeks
Amoxicillin MIC >4 mg/L and gentamicin MIC ≤128 mg/L	Vancomycin IV and gentamicin IV[2]	1 g twice daily[3] 1 mg/kg twice daily[2]	4 weeks 4 weeks	6 weeks 6 weeks
Staphylococci – native valve				
Meticillin-sensitive	Flucloxacillin IV	2 g 4–6 times daily[4]	4 weeks	–
Meticillin-resistant, vancomycin MIC ≤2 mg/L, rifampicin-sensitive	Vancomycin IV Rifampicin orally	1 g twice daily[3] 300–600 mg twice daily	4 weeks 4 weeks	– –
Staphylococci – prosthetic valve				
Meticillin-sensitive	Flucloxacillin IV and gentamicin IV and rifampicin orally	2 g 4–6 times daily 1 mg/kg twice daily[2] 300–600 mg twice daily	– – –	6 weeks 6 weeks 6 weeks
Meticillin-resistant, vancomycin MIC ≤2 mg/L, rifampicin-sensitive	Vancomycin IV and rifampicin orally	1 g twice daily[3] 300–600 mg twice daily	– –	6 weeks 6 weeks

[1]When conditions in Box 16.97 are met, 2 weeks of benzylpenicillin and gentamicin (1 mg/kg twice daily) may be sufficient. Ceftriaxone 2 g once daily IV/IM can be used instead of benzylpenicillin for those with non-severe penicillin allergy. [2]Pre-dose gentamicin level should be ≤1 mg/L, post-dose 3–5 mg/L. Adjust dose according to levels and renal function. [3]Pre-dose vancomycin level should be 15–20 mg/L. Adjust dose according to levels and renal function. [4]Use 6 times daily if weight >85 kg.
(IV = intravenous; MIC = minimum inhibitory concentration)

Adapted from Gould FK, Denning DW, Elliott TS, et al. Guidelines for the diagnosis and antibiotic treatment of endocarditis in adults: a report of the working party of the British Society for Antimicrobial Chemotherapy. J Antimicrob Chemother 2012; 67:269–289.

16.97 Conditions for the short-course treatment of endocarditis caused by fully sensitive streptococci

- Native valve infection
- Minimum inhibitory concentration (MIC) ≤0.125 mg/L
- No adverse prognostic factors (heart failure, aortic regurgitation, conduction defect)
- No evidence of thromboembolic disease
- No vegetations >5 mm diameter
- Clinical response within 7 days

16.98 Indications for cardiac surgery in infective endocarditis*

- Heart failure due to valve damage
- Failure of antibiotic therapy (persistent/uncontrolled infection)
- Large vegetations on left-sided heart valves with echo appearance suggesting high risk of emboli
- Previous evidence of systemic emboli
- Abscess formation

*Patients with prosthetic valve endocarditis or fungal endocarditis often require cardiac surgery.

- Cardiac surgery with débridement of infected material and valve replacement may be required in a substantial proportion of patients, particularly those with Staph. aureus and fungal infections (Box 16.98). Antimicrobial therapy must be started before surgery.

- **Prevention of Infective endocarditis:**

Until recently, antibiotic prophylaxis was routinely given to people at risk of infective endocarditis undergoing interventional procedures. However, as this has not been proven to be effective and the link between episodes of infective endocarditis and interventional procedures has not been demonstrated, antibiotic prophylaxis is no longer offered routinely.