Endocarditis refers to inflammation of the endocardium, the inner layer of the heart (including the heart valves). Endocarditis can be:

- infective (e.g. bacterial, fungal)
- non-infective (e.g. Libman–Sacks endocarditis in systemic lupus erythematosus).

The characteristic lesion in endocarditis is the vegetation, a mass of inflammatory material which can include fibrin, platelets, red and white blood cells and (where present) micro-organisms.

**Infective endocarditis**

In the past, infective endocarditis has been classified as acute or subacute (‘SBE’, subacute bacterial endocarditis) but this terminology is outdated and should no longer be used. Although infective endocarditis is rare (fewer than 10 cases per 100 000 population every year) it is nevertheless a serious and dangerous condition, with a mortality of around 20 per cent.

Infective endocarditis starts with organisms reaching the endocardium either via a bacteraemia or directly via surgery or device placement. The organisms adhere to the endocardium and as they invade the local tissues a vegetation begins to form. Left untreated, they cause local tissue destruction (e.g. valvular regurgitation) and can also lead to abscess and/or fistula formation.

The single most common causative organism is *Staphylococcus aureus*; other commonly encountered organisms are listed in Table 17.1.
The clinical features of infective endocarditis (Table 17.2) can be subtle and sometimes will have been present for several weeks, so a high index of suspicion is needed to avoid missing the diagnosis. Be particularly alert to the possibility of infective endocarditis in those at risk (see above), and/or those with a history of invasive procedures or intravenous drug use.

<table>
<thead>
<tr>
<th>Table 17.1 Common causes of infective endocarditis</th>
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<tbody>
<tr>
<td><strong>Bacterial</strong></td>
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<tr>
<td>Staphylococcus aureus</td>
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<tr>
<td>Streptococcus viridans</td>
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<tr>
<td>Streptococcus intermedius</td>
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<tr>
<td>Pseudomonas aeruginosa</td>
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<tr>
<td>HACEK organisms</td>
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<tr>
<td>Bartonella</td>
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<tr>
<td>Coxiella burnetii</td>
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<tr>
<td><strong>Fungal</strong></td>
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<tr>
<td>Candida</td>
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<tr>
<td>Aspergillus</td>
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<td>Histoplasma</td>
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</table>

**Clinical features of infective endocarditis**

The clinical features of infective endocarditis (Table 17.2) can be subtle and sometimes will have been present for several weeks, so a high index of suspicion is needed to avoid missing the diagnosis. Be particularly alert to the possibility of infective endocarditis in those at risk (see above), and/or those with a history of invasive procedures or intravenous drug use.

<table>
<thead>
<tr>
<th>Table 17.2 Clinical features of infective endocarditis</th>
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<tbody>
<tr>
<td><strong>Symptoms</strong></td>
</tr>
<tr>
<td>Fever</td>
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<tr>
<td>Fatigue</td>
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<tr>
<td>Anorexia</td>
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<tr>
<td>Weight loss</td>
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<tr>
<td>Flu-like symptoms</td>
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<tr>
<td><strong>Signs</strong></td>
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<tr>
<td>Fever</td>
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<tr>
<td>Heart murmur</td>
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<tr>
<td>Splinter haemorrhages</td>
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<tr>
<td>Janeway lesions</td>
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<tr>
<td>Osler’s nodes</td>
</tr>
<tr>
<td>Roth spots</td>
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<tr>
<td>Peripheral emboli</td>
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</tbody>
</table>

The Duke (or modified Duke) criteria can be helpful in cases of diagnostic uncertainty (Durack et al. 1994). Blood cultures are the mainstay of diagnosis, and at least three sets should be taken from different sites at different times. Blood cultures may be negative because of prior antibiotic treatment or the presence of fastidious (difficult to culture) organisms.

Echocardiography plays a valuable role in identifying:

- presence of vegetations
- valvular destruction
- associated abscess, fistula or perforation.
'Major' echo criteria supporting a diagnosis of infective endocarditis are the presence of oscillating structures (vegetations), the presence of an abscess, new valvular regurgitation and dehiscence of a prosthetic valve.

Transthoracic echo (TTE) can, at best, detect vegetations down to a minimum size of 2 mm (and is known to miss the majority of vegetations <5 mm). The superior image quality of transoesophageal echo (TOE) makes it more sensitive and specific than TTE, particularly in cases of prosthetic valve endocarditis and in the detection of abscesses. TTE has an overall sensitivity in detecting vegetations of ≈50 per cent, whereas the sensitivity of TOE is ≥90 per cent. TOE should be considered in cases where the TTE is negative or inconclusive (particularly if the clinical suspicion of infective endocarditis is high), or where there is a suspicion of prosthetic valve endocarditis, right heart endocarditis or a cardiac abscess. It is important to note that a negative echocardiogram (even TOE) does not rule out a diagnosis of infective endocarditis and it is prudent to include this, when appropriate, in your echo report.

**VEGETATIONS AND JET LESIONS**

Vegetations occur most commonly on heart valves. However, they can also occur anywhere where a high velocity jet of blood flow (‘jet lesion’) occurs between a high-pressure and low-pressure chamber, impinging on the endocardium and potentially resulting in endothelial injury and establishing a focus for infection. Examples include the high velocity jets found in ventricular septal defect (VSD) or persistent ductus arteriosus (PDA).

**Echo assessment of infective endocarditis**

A full echo study should be performed and any structural abnormalities that predispose to infective endocarditis noted. According to the National Institute for Health and Clinical Excellence (2008), patients at risk of developing infective endocarditis include those with:

- acquired valvular disease including stenosis or regurgitation
- valve replacement
- structural congenital heart disease, including surgically corrected or palliated conditions (except isolated atrial septal defect and fully repaired VSD or PDA, and endothelialized closure devices)
hypertrophic cardiomyopathy
previous infective endocarditis.

Look carefully for any features of infective endocarditis:

- vegetations
- valvular destruction
- abscess
- fistula.

**Vegetations**

The characteristic echo appearance of a vegetation is of an echogenic mass, irregular in shape, attached to the ‘upstream’ side of a valve leaflet (i.e. the atrial side in the case of the mitral and tricuspid valves, the ventricular side for the aortic and pulmonary valves). Vegetations can be attached to any part of the valve, but most commonly at the coaptation line. Vegetations move with the leaflet but in a more chaotic (‘oscillating’) manner. It is common for a vegetation to prolapse through the valve as it opens.

Vegetations vary in size, often being just a few mm in diameter but sometimes reaching 2–3 cm. Vegetations resulting from fungal infections (e.g. *Candida, Aspergillus*) are usually much bigger than bacterial vegetations, and can be so big that they are mistaken for a cardiac tumour. Fungal endocarditis is rare and is more likely to occur in patients who are immunosuppressed.

In order of decreasing frequency, the valves affected by infective endocarditis are the mitral (Fig. 17.1), aortic, tricuspid and pulmonary. More than one valve can be affected. Right heart endocarditis is much commoner in patients who are intravenous drug users, and can also occur in association with right-sided devices such as pacemaker leads.

If vegetations are present, describe their:

- location (which valve(s), and which parts of the valve, are affected)
- mobility (e.g. immobile, oscillating)
- size.

Because infective endocarditis usually occurs on a valve that is already abnormal, you must also describe any underlying valvular disease as well as looking for any evidence that the infection is causing valvular destruction (see below).
Infective endocarditis commonly occurs on a heart valve that is already abnormal, and a pre-existing abnormality (e.g. myxomatous mitral valve disease, nodular aortic cusp thickening) can be mistaken for vegetations, or can make the recognition of existing vegetations more difficult. Patients who are left with sterile vegetations following a previously treated episode of endocarditis can pose a difficult diagnostic challenge.

Cardiac tumours and thrombi can also be mistaken for vegetations (and vice versa). Benign structures that can be mistaken for vegetations include Lambl’s excrescences and the Eustachian valve (see Chapter 21). Remember too that not all vegetations are infective in nature (p. 241).

**Valvular destruction**
Infective endocarditis can cause valvular destruction, leading to regurgitation either through distorting normal valve closure or through perforation of a valve leaflet (Fig. 17.2). Valvular regurgitation should be assessed and described as for native valvular regurgitation, as outlined in Chapters 13–15. Valvular stenosis as a result of infective endocarditis is much rarer, and usually results from obstruction of the valve orifice by a large vegetation.
Prosthetic valve endocarditis can be challenging to diagnose, particularly using TTE. Imaging of the valve itself is often suboptimal because of shadowing and reverberation of the echo signal by the prosthesis. Furthermore, prosthetic valve infections typically occur at the sewing ring rather than on the leaflets, making it harder to identify any vegetations. Look carefully for any signs of dehiscence (‘splitting open’) of the sewing ring around the valve, allowing regurgitant blood flow around the valve prosthesis (paravalvular regurgitation).

A TOE study is therefore usually appropriate in order to obtain better resolution of the prosthesis and any associated abnormalities. Prosthetic valve endocarditis is difficult to treat with antibiotics alone and early re-do surgery is usually required, particularly in the first 12 months after the valve was first implanted.

Abscess
A valvular infection can spread to involve surrounding tissues, particularly in prosthetic valve endocarditis. On echo, an abscess appears as an
echolucent or echodense area in the valve annulus or surrounding tissues. TOE has a much greater sensitivity for detecting perivalvular abscesses than TTE.

Abscesses occur more commonly around the aortic valve than the mitral valve, although an aortic root abscess can sometimes spread to affect the anterior mitral valve leaflet. A mitral valve abscess typically affects the posterior annulus, appearing on echo as a thickened and echodense region.

If an abscess is present, describe its:

- location (which valve is affected, and where the abscess lies in relation to the valve)
- size.

**Fistula**

An aortic root abscess (or pseudoaneurysm) may rupture into a neighbouring chamber (usually the right ventricle, but sometimes one of the atria) to form an abnormal communication or fistula. Fistulas can be multiple.

A fistula can be demonstrated using colour Doppler and continuous wave (CW) Doppler to show abnormal flow arising in the aortic root and flowing through the fistula. Describe where the fistula arises and which chambers it connects. Describe too its haemodynamic effects, such as consequent chamber dilatation and/or dysfunction.

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**SAMPLE REPORT**

The aortic valve cusps are thickened with mildly reduced mobility and there is mild aortic stenosis (peak gradient 24 mmHg). There is a mobile ‘oscillating’ echogenic nodule, measuring 7 mm in diameter, attached to the ventricular surface of the right coronary cusp. There is moderate aortic regurgitation into a non-dilated left ventricle, with good systolic function. No evidence of abscess or fistula formation is seen. The appearances are in keeping with a vegetation on the aortic valve.

**Management of infective endocarditis**

The treatment of infective endocarditis usually requires a prolonged (4–6 week) course of antibiotics, chosen where possible according to the antibiotic sensitivities of the causative organism. Cardiac surgery is necessary in about half of patients with infective endocarditis. Ideally, in stable patients, surgery
should be performed only once the infection has been successfully treated with antibiotics to minimize the risk of recurrent infection. However, it is not always possible to delay surgery if patients are unstable or at high risk of complications (e.g. embolization). Early surgery should be considered for:

- haemodynamic instability/heart failure as a result of acute aortic or mitral regurgitation
- persistent infection (fever and bacteraemia despite treatment with appropriate antibiotics for 7–10 days)
- development of a perivalvular abscess or fistula
- infective endocarditis on a prosthetic valve
- fungal infections
- difficult to treat organisms (e.g. *Brucella*)
- high embolic risk:
  - recurrent emboli despite appropriate antibiotics
  - large vegetation (>10 mm) and a single embolic event
  - very large mobile vegetation (>15 mm).

**Follow-up echo**

Patients being treated for infective endocarditis require regular follow-up echo studies to monitor progress and to watch for complications. With effective treatment the vegetations may gradually shrink and become less mobile. The sudden disappearance of a vegetation between studies should raise a suspicion that the vegetation has broken free and embolized elsewhere.

Even when an episode of infective endocarditis has been fully treated, (sterile) vegetations may remain visible on echo. This can make the diagnosis of recurrent infection challenging in patients who have suffered an episode of endocarditis previously – clinical evidence of active infection, particularly on blood cultures, is the key to diagnosis in such cases.

**Prevention of infective endocarditis**

Patients at risk of infective endocarditis (see above) should receive guidance on good oral hygiene and how to recognize the symptoms of infective endocarditis, and also information on the risks of invasive procedures, but antibiotic prophylaxis is no longer routinely recommended.

**Non-infective endocarditis**

Not all vegetations occur as a result of infection, a fact that emphasises the importance of taking the patient’s clinical history (and in particular the results of blood cultures) into account when making a diagnosis of infective (versus non-infective) endocarditis.
Non-infective endocarditis has also been termed non-bacterial thrombotic endocarditis (NBTE) or, historically, marantic endocarditis. The vegetations that occur are sterile, and are composed mainly of fibrin and platelets. Non-infective endocarditis can result from:

- trauma to the valve leaflets (e.g. from an intracardiac catheter)
- circulating immune complexes
- vasculitis
- hypercoagulability
- mucin-producing adenocarcinomas.

Non-infective endocarditis occurring in systemic lupus erythematosus is called Libman–Sacks endocarditis (also known as ‘verrucous’ endocarditis), and in this condition, the vegetations mainly consist of immune complexes and mononuclear cells. The mitral and aortic valves are most commonly affected, although just about any part of the endocardium can be involved. The vegetations are usually small, irregular and immobile (compared with the vegetations in infective endocarditis). Libman–Sacks endocarditis is usually asymptomatic, but can present with valvular regurgitation or, less commonly, stenosis. There is also a risk of embolization, although this is uncommon.

**FURTHER READING**


