

Oral and Maxillofacial Surgery/Fourth Year

2024-2025

د.ياسر رياض الخناق

Infective Endocarditis

Infective endocarditis (IE) is defined as a microbial infection of the endothelial surface of the heart or heart valves that most often occurs in proximity to congenital or acquired cardiac defects. Its intracardiac effects include severe valvular insufficiency, which may lead to intractable congestive heart failure and myocardial abscesses, therefore, emphasis has long been directed toward its prevention.

Although bacteria most often cause these diseases, fungi and other microorganisms may also cause infection; thus, the term infective endocarditis (IE) is used to reflect this multimicrobial origin.

Classification

IE is classified based on:

- ❖ The causative microorganism (e.g., streptococcal endocarditis, staphylococcal endocarditis, candidal endocarditis)
- ❖ The type of valve that is infected (e.g., native valve endocarditis [NVE], prosthetic valve endocarditis [PVE]).
- ❖ The source of infection; whether community acquired or hospital acquired, or whether the patient is an intravenous (IV) drug user or not.

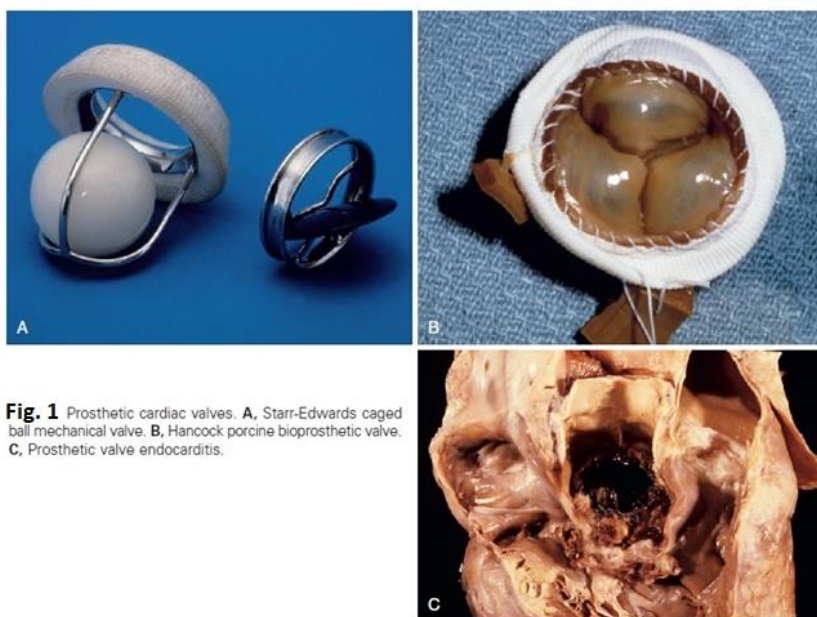


Fig. 1 Prosthetic cardiac valves. **A**, Starr-Edwards caged ball mechanical valve. **B**, Hancock porcine bioprosthetic valve. **C**, Prosthetic valve endocarditis.

Etiology

- ✓ Streptococci are the most common cause of IE 30%-65%, of which streptococci viridans (alpha-hemolytic streptococci), which are normal constituents of the oral flora and gastrointestinal tract, remain the most common cause of community acquired NVE.
- ✓ Staphylococci are the cause of at least 30%-40% of cases of IE; mostly coagulase-positive Staphylococcus aureus which is the most common pathogen in IE associated with IV drug abuse, it is also the most common pathogen in nonvalvular cardiovascular device infections.
- ✓ In some recent studies, S. aureus has emerged as the most common cause of IE and rates of viridans streptococci have decreased.
- ✓ Other microbial agents that less commonly cause IE such as the HACEK group (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella), Pseudomonas aeruginosa, Corynebacterium, Bacteroides fragilis, and fungi.

Predisposing conditions attributed to IE include:

- Mitral valve prolapse 25%-30%.
- Aortic valve disease 12%-30%.
- Congenital heart disease 20%-37%.
- Degenerative valve disease 1-5%
- Prosthetic valve 25%-30%.
- Intravenous drug abuse 5%-20%.
- No identifiable condition 25%-47%.

Pathophysiology

IE is thought to be the result of a series of complex interactions of several factors involving endothelium, bacteria, and the host immune response. The sequences of events include:

1. Injury or damage to an endothelial surface, most often of a cardiac valve leaflet.
2. Fibrin and platelets then adhere to the roughened endothelial surface and form small clusters or masses called nonbacterial thrombotic endocarditis (NBTE), these masses are sterile and do not contain microorganisms.
3. With the occurrence of a transient bacteremia, bacteria can be seeded into and adhere to the mass.
4. Additional platelets and fibrin are then deposited onto the surface of the mass, which serves to protect the bacteria that undergo rapid multiplication within the protection of the vegetative mass.

5. The clinical outcome results from:

- Local destructive effects of intracardiac (valvular) lesions.
- Embolization of vegetative fragments to distant sites, resulting in infarction or infection.
- Hematogenous seeding of remote sites during continuous bacteremia.

- Antibody response to the infecting organism with subsequent tissue injury caused by deposition of preformed immune complexes or antibody/complement interaction with antigens deposited in tissues.

Signs and symptoms

The clinical presentation may be varied; the interval between the presumed initiating bacteremia and the onset of symptoms of IE is estimated to be less than 2 weeks in more than 80% of patients.

- ✓ Fever (most common).
- ✓ Heart murmur.
- ✓ Petechiae of the palpebral conjunctiva, the buccal and palatal mucosa, and extremities.
- ✓ Osler's nodes (small, tender, subcutaneous nodules that develop in the pulp of the digits). **Named after Sir William Osler (1849-1919).** They are caused by immune-complex deposition.
- ✓ Janeway lesions (small, erythematous or hemorrhagic, macular nontender lesions on the palms and soles). **Named after Edward Janeway (1841-1911).** They are caused by septic emboli which deposit bacteria, forming micro-abscesses of the dermis with marked necrosis and inflammatory infiltrate not involving the epidermis.
- ✓ Splinter hemorrhages in the nail beds.
- ✓ Roth spots (oval retinal hemorrhages with pale centers). Caused by immune complex mediated vasculitis. **Named after Moritz Roth, a Swiss Pathologist (1839-1914).**
- ✓ Splenomegaly
- ✓ Clubbing of the digits.
- ✓ Positive blood cultures in most cases. Although up to 30% of cases of IE are initially found to be "culture negative," especially in patients who have taken antibiotics prior to the diagnosis of IE.



Fig. 3 Osler node in infective endocarditis. (From Fowler VG



Fig. 2 Petechiae in infective endocarditis.



Fig. 4 A Roth spot in the retina in infective endocarditis

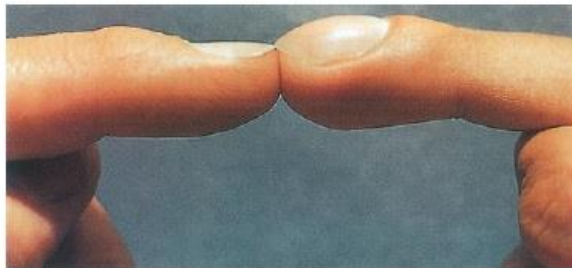


Fig. 5 Nail clubbing may appear within a few weeks of development of infective endocarditis.

Diagnosis

Duke criteria were developed to facilitate diagnosis of IE. These criteria are categorized as major and minor.

❖ Major criteria:

- Positive blood cultures.
- Evidence of endocardial involvement (e.g., positive echocardiography, presence of new valvular regurgitation, myocardial abscess).

❖ Minor criteria:

- Predisposing heart condition or IV drug use.
- Fever.
- Vascular phenomena, including embolic events.
- Immunologic phenomena.
- Microbiologic evidence other than positive blood culture.

Definitive diagnosis of IE requires the presence of two major criteria, one major and more than three minor criteria, or five minor criteria.

Complications

- Heart failure as a result of severe valvular dysfunction.
- Embolization of vegetation fragments leads to stroke, MI, pulmonary embolism. Emboli also may involve other systemic organs, including the liver, spleen, kidney, and abdominal mesenteric vessels.
- Renal dysfunction is also common and may be due to immune complex glomerulonephritis or infarction.
- The overall mortality rate despite treatment is 10%-30%

Medical Management

Generally, it consists of antibiotics and surgery. The most widely used antibiotics include penicillin, ceftriaxone, gentamicin and vancomycin while most staphylococcus aureus organisms that produce betalactamase respond to nafcillin and oxacillin and for strains resistant to oxacillin, vancomycin is combined with rifampin and gentamicin.

Surgical intervention may be necessary to facilitate a cure for IE or to repair damage caused by the infection.

Dental management

- The dentist should identify from history taking, those patients with cardiac conditions that increase risk for IE and should remain alert and refer the patient with signs or symptoms of IE to physician. This would apply whether or not the patient has received prophylactic antibiotics for dental procedures.
- The establishment and maintenance of optimal oral hygiene are critically important in these patients.
- The basic assumption is that IE is most often due to bacteremia that results from an invasive dental procedure, and that through the administration of antibiotics prior to those procedures, IE could be prevented. But studies have shown that bacteremia can also result from many normal daily activities such as tooth brushing, flossing, using toothpicks, using oral water irrigation devices, and chewing emphasizing the need to maintain good oral hygiene and eradicating dental/oral disease for decreasing the frequency of bacteremia produced by normal daily activities.
- Preoperative dental evaluation and necessary dental treatment should be provided whenever possible before initiation of cardiac valve surgery or replacement or repair of congenital heart disease in an effort to decrease the incidence of IE.
- Cardiac Conditions Associated with the Highest Risk of Adverse Outcome from Endocarditis for which Prophylaxis with Dental procedures is recommended:
 1. Prosthetic cardiac valve
 - Presence of cardiac prosthetic valve
 - Transcatheter implantation of prosthetic valves
 - Cardiac valve repair with devices, including annuloplasty, rings, or clips
 - Left ventricular assist devices or implantable heart
 2. Previous infective endocarditis

3. Congenital heart disease (CHD)
- ✓ *Unrepaired cyanotic* CHD, including those with palliative shunts and conduits.
 - ✓ *Completely repaired CHD with prosthetic material or device* by surgery or catheter intervention during the first 6 months after the procedure. Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.
 - ✓ *Repaired CHD with residual defects* at the site or adjacent to the site of a prosthetic patch or prosthetic device, which inhibits endothelialization.
 - ✓ Surgical or transcatheter pulmonary artery valve or conduit placement.
4. Cardiac transplantation recipients who develop cardiac valvulopathy.
- American Heart Association (AHA) recommended antibiotic prophylaxis for many patients with heart murmurs caused by valvular disease (e.g., mitral valve prolapse (MVP), rheumatic heart disease) in an effort to prevent infective endocarditis.
 - IE antibiotic prophylaxis is recommended only for patients listed above who undergo any dental procedure that involves the manipulation of gingival tissues or the periapical region of a tooth and for those procedures that perforate the oral mucosa.
 - The following procedures and events **do not** need prophylaxis: routine anesthetic injections through noninfected tissue, restorative dentistry, taking dental radiographs, placement of removable prosthodontic or orthodontic appliances, adjustment of orthodontic appliances, placement of orthodontic brackets, shedding of deciduous teeth, suture removal, fluoride treatment and bleeding from trauma to the lips or oral mucosa.

➤ Antibiotic prophylaxis regimens

Situation	Agent	Regimen: Single dose 30-60 Minutes before Procedure	
		Adult	Child
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2g IM or IV	50 mg/kg IM or IV
	Cefazolin or Ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergic to Penicillins or Ampicillin Oral	Cephalexin	2 g	50 mg/kg
	Azithromycin or Clarithromycin	500 mg	15 mg/kg
	Doxycycline	100 mg	>45 kg, 100 mg <45 kg, 4.4 mg/kg
Allergic to Penicillins or Ampicillin and cannot take oral medications	Cefazolin or Ceftriaxone	1 g IM or IV	50 mg/kg IM or IV

- ✓ Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.
- ✓ The adverse effect profile demonstrated with the prophylactic use of clindamycin led to its elimination from the current AHA recommendations.
- ✓ In patients who are already taking penicillin or amoxicillin for eradication of an infection or for long-term secondary prevention of rheumatic fever are likely to have streptococcus viridans that are relatively resistant to penicillin or amoxicillin. Therefore:
 - Azithromycin, clarithromycin or doxycycline should be selected for prophylaxis if treatment is immediately necessary. Cephalosporins should be avoided because of cross resistance.
 - An alternative approach is to wait for at least 10 days after completion of antibiotic therapy before administering prophylactic antibiotics. (Evidence exists from prospective studies indicating that amoxicillin-resistant VGS can persist for 24 days after a single dose of 2 g amoxicillin; thus waiting at least 24 days may have additional benefit.)
- In case of prolonged dental procedures (longer than 6 hours) it is advisable to administer an additional prophylactic dose (same dose).
- Prior to cardiac valve surgery or replacement or repair of congenital heart disease, it is recommended that preoperative dental evaluation be performed and necessary dental treatment provided whenever possible in an effort to decrease the incidence of late PVE caused by viridans group streptococci.

Rheumatic fever and rheumatic heart disease

Rheumatic fever is an autoimmune inflammatory process that develops after pharyngeal infection with group A beta-hemolytic streptococci (*Streptococcus pyogenes*). It predominantly affects children between 5-15 years. Rheumatic fever may occasionally be followed by chronic rheumatic carditis with permanent cardiac valvular damage that appears to be immunologically mediated tissue damage, which may lead to fibrosis and distortion of the cardiac valves (chronic rheumatic heart disease).

Clinical manifestations

The clinical manifestations of acute rheumatic fever are so variable that the diagnosis is made only if at least two of the major criteria are fulfilled

Diagnostic criteria	
Major	Minor
Carditis	Pyrexia
Polyarthritides	Arthralgia
Chorea	Previous rheumatic fever
Erythema marginatum	Raised ESR and C-reactive protein
Subcutaneous nodules	Characteristic ECG changes

- A sore throat may be followed after about 3 weeks by an acute febrile illness with multiple joints pain (migratory arthralgia) which heals without permanent damage in about 3 weeks.
- Cerebral involvement causing spasmodic involuntary movements (Sydenham chorea, St. Vitus dance).
- A characteristic rash (erythema marginatum).
- Lung involvement.
- Subcutaneous nodules (usually around the elbows).
- The most serious cardiac complication is subendocardial inflammation, particularly along the lines of closure of the mitral and aortic valve cusps, resulting in the formation of fibrinous vegetations and later scarring, fibrotic stiffening and distortion of the heart valves, often causing mitral valve and/or aortic valve stenosis. This is essentially a mechanical, hemodynamic disorder, in which the defective valves may become infected at any time, leading to infective endocarditis. Cardiac failure can develop, often after many years.

Medical management

- ✓ Prompt antimicrobial treatment of streptococcal sore throat (within 24 hours of onset) prevents the development of rheumatic fever in most cases.

Commented [WU1]: Chorea is a movement disorder that causes involuntary, irregular, unpredictable muscle movements. The disorder can make you look like you're dancing (the word **chorea** comes from the Greek word for "dance") or look restless or fidgety. **Chorea** is a movement problem that occurs in many different diseases and conditions.

- ✓ After an attack of rheumatic carditis, there is a risk of recurrence and continuous antibiotic prophylaxis becomes necessary to lessen the risk of permanent cardiac damage. The drug of choice is usually oral phenoxymethyl penicillin until the age of 20. For those allergic to penicillin, sulfadimidine should be given.

Dental management

- Acute rheumatic fever patients are exceedingly unlikely to be seen during an attack but emergency dental treatment may be necessary.
- Patients with a history of rheumatic fever but without cardiac involvement are treated as a normal person.
- Most patients with chronic rheumatic heart diseases are anticoagulated and they should be managed after determining their prothrombin time and INR and the treatment can be done under local anesthesia with vasoconstrictor in consultation with the physician. Conscious sedation with nitrous oxide may be given if cardiac function is good and with the approval of the physician.
- Indications for prophylactic antibiotics are only for the high-risk patients mentioned in the dental management of IE.
- American Heart Association (AHA) recommended antibiotic prophylaxis for many patients with heart murmurs caused by valvular disease (e.g., mitral valve prolapse (MVP), rheumatic heart disease) in an effort to prevent infective endocarditis.

Congenital heart diseases

Congenital heart diseases (CHD) are the most common type of cardiac diseases present in children. They can broadly be classified as Cyanotic and Acyanotic (non-cyanotic).

❖ Cyanotic CHDs

The cyanosis results from shunting of deoxygenated blood from the right ventricle into the left side of the heart and the systemic circulation (right to left shunt) leading to chronic hypoxemia, they include:

- ✓ Eisenmenger syndrome (**Named after Victor Eisenmenger who described this condition in 1897**).
- ✓ Fallot's tetralogy (**Named after the French physician Etienne-Louis-Arthur Fallot 1850-1911**).
- ✓ Pulmonary atresia.
- ✓ Pulmonary valve stenosis.
- ✓ Total anomalous venous drainage.
- ✓ Transposition of great vessels.
- ✓ Tricuspid atresia.

Patients may crouch to improve venous return, but eventually polycythemia with hemorrhagic and thrombotic tendencies develop, finger and toe clubbing develops but after 3 months of age. If untreated, 40% of patients with cyanotic CHD die within 5 years.

❖ **Acyanotic CHDs**

They are further divided into those with no shunt like; Aortic stenosis, bicuspid aortic valve, coarctation of the aorta, dextrocardia and mitral valve prolapse. The other division of the Acyanotic CHD is those diseases with left to right shunt and these include; Atrial septal defects (ASD), Ventricular septal defects (VSD) and patent ductus arteriosus (PDA).

Some CHD start as Acyanotic diseases and become cyanotic with time. Most of these cardiac defects are well tolerated in utero, and it is only after birth that their anatomic and hemodynamic abnormalities become evident.

CHD is most commonly diagnosed through echocardiography, and confirmed by cardiac magnetic resonance imaging (MRI). Early correction of the congenital defect, often by transvenous catheter techniques, is the treatment of choice. More complex defects may require an operation. Medical treatment may be needed for the management of pulmonary edema, heart failure, polycythemia, infection or emotional disturbances.

Modern surgical and medical care helps children survive into adult life and patients are then often called adult or 'grown-up' CHD. Nevertheless, complications observed in adults who were previously thought to have had successful repair of CHD include arrhythmias, valve disorders and cardiac failure, and residual defects can still predispose to complications such as infective endocarditis.

Dental management

- The most important aspect for dentists to consider is how well the patient's heart condition is compensated. Consultation with the physician is recommended.
- Patients with heart disease should take their medications as usual on the day of the dental procedure, and should bring all their medications to the dental office for review at the time of the first appointment.
- Patients with stable heart disease receiving atraumatic treatment under local anesthesia can receive treatment.
- Late morning or early afternoon appointments are advisable.

- Stress-reduction and good analgesia should be provided.
- Limited use of vasoconstrictor with aspirating syringes.
- Retraction cords containing adrenalin should be avoided.
- Conscious sedation preferably with nitrous oxide can be given with the approval of the physician. General anesthesia should only be provided by expert anesthetists in hospital.
- Bleeding tendencies due to platelet dysfunction or coagulation defects should be evaluated and managed accordingly.
- There may be susceptibility to infective endocarditis, so prophylactic antibiotics should be used in the following cases:
 - ✓ Unrepaired cyanotic CHD, including those with palliative shunts and conduits
 - ✓ Completely repaired CHD with prosthetic material or device by surgery or catheter intervention during the first 6 months after the procedure. Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.
 - ✓ Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device, which inhibits endothelialization.
- Prior to cardiac valve surgery or replacement or repair of congenital heart disease, it is recommended that preoperative dental evaluation be performed and necessary dental treatment provided whenever possible in an effort to decrease the incidence of late PVE caused by viridans group streptococci.

Oral manifestations

- Delayed eruption of both dentitions
- Enamel hypoplasia; the teeth often have a bluish-white 'skimmed milk' appearance and there is gross vasodilatation in the pulps
- Greater caries and periodontal disease activity, probably because of poor oral hygiene and lack of dental attention
- After cardiac surgery, transient small white non-ulcerated mucosal lesions of unknown etiology may appear.