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An Overview on Endocarditis

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1. Introduction

The endocardium is the innermost layer of tissue that lines the chambers of the heart. Its cells are embryologically and biologically similar to the endothelial cells that line blood vessels.

The endocardium underlies the much more voluminous myocardium, the muscular tissue responsible for the contraction of the heart. The outer layer of the heart is termed epicardium. Whole heart is surrounded by a small amount of fluid enclosed by a fibrous pouch called the pericardium.

Recently, it has become evident that the endocardium, which is primarily made up of endothelial cells, can have control over myocardial function. This modulating role is separate from the homeometric and heterometric regulatory mechanisms that control some myocardial contractility. Furthermore, the endothelium of the myocardial (heart muscle) and capillaries, which is also closely appositioned to the cardiomyocytes (heart muscle cells) are involved in this modulatory role. Thus, the cardiac endothelium (both the endocardial endothelium and the endothelium of the myocardial capillaries) controls the development of the heart in the embryo as well as in the adult, for example during cardiac hypertrophy. Additionally, the contractility and electrophysiological environment of the cardiomyocytes are regulated by the cardiac endothelium. This function recently known, it is extremely important to us when there are some injuries cardiac affecting to the myocardial infarction.

The endocardial endothelium may also act as a kind of blood-heart barrier (analogous to the blood-brain barrier), thus controlling the ionic composition of the extra-cellular fluid in which the cardiomyocytes bathe.

All inflammation of the endocardium is called, therefore, endocarditis.

Depending on how extensive of the inflammation, endocarditis can be: localized (most often) or generalized.

As in every inflammation, the cause of endocarditis can be infectious (virus, bacteria, parasites and so...) - bacteria is the most common - or non-infectious.

During depolarization, the impulse is carried from endocardium to epicardium, and during repolarization, the impulse moves from epicardium to endocardium. In infective endocarditis,

the endocardium (especially the endocardium lining the heart valves) is affected by originating agent, (The majority, an infectious agent).

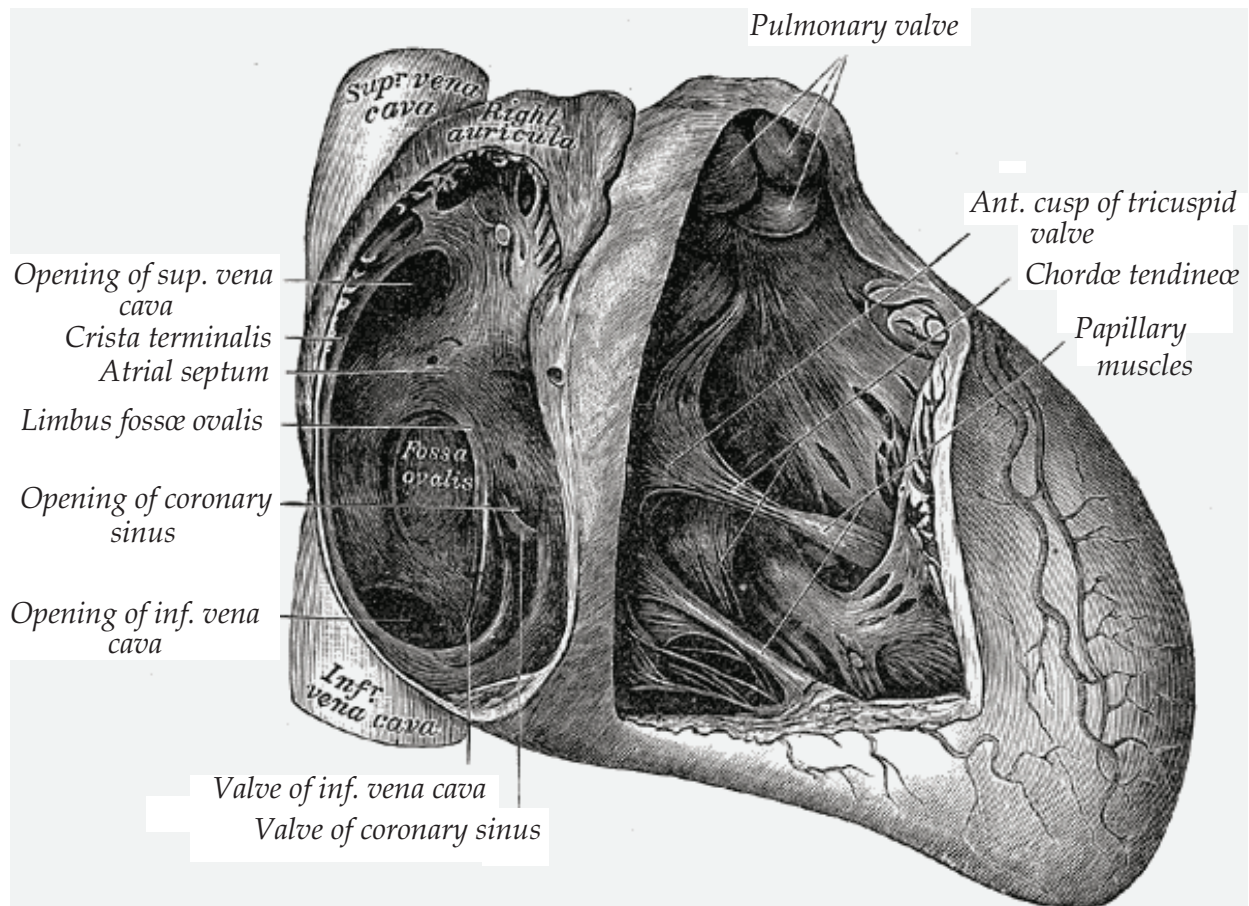


Fig. 1. Interior of right side of heart. (modified from Gray's)

Infective endocarditis is the most common form of endocarditis and its valves, the most affected structures, over all the mitral valve: The agents are usually bacterial, but other organisms can also be responsible.

The valves of the heart do not receive any dedicated blood supply. As a result, defensive immune mechanisms (such as white blood cells) cannot directly reach the valves via the bloodstream. If an organism (such as bacteria) attaches to valve surface and forms vegetation, the host immune response is blunted. The lack of blood supply to the valves also has implications on treatment, since drugs also have difficulty reaching the infected valve.

Normally, blood flows smoothly through these valves. If they have been damaged - from rheumatic fever, for example - the risk of bacterial attachment is increased.

Historically, infective endocarditis has been clinically divided into *acute* and *subacute* presentations (because untreated patients tended to live longer with the subacute as opposed to the acute form). This classifies both the rate of progression and severity of disease.

Subacute bacterial endocarditis (SBE) is often due to streptococci of low virulence and mild to moderate disease, which progresses slowly over weeks and months and has low propensity to haematogenous seed extracardiac sites.

Acute bacterial endocarditis (ABE) is a fulminant infection over days to weeks, and is more likely due to *Staphylococcus aureus* which has much greater virulence, or disease-producing capacity and frequently causes metastatic infection. (Currently, the terms short incubation -meaning less than about six weeks-, and long incubation -greater than about six weeks- are preferred).

Infective endocarditis may also be classified as culture-positive or culture-negative. Culture-negative endocarditis can be due to micro-organisms that require a longer period of time to be identified in the laboratory, such organisms are said to be fastidious because they have demanding growth requirements, or due to absence of an organism as in marantic endocarditis. Some pathogens responsible for culture-negative endocarditis include *Aspergillus* species, *Brucella* species, *Coxiella burnetii*, *Chlamydia* species, and HACEK bacteria. Another possible reason for culture negativity, even with the more typical pathogens, is prior antibiotic treatment.

Endocarditis can also be classified by the side of the heart affected:

Patients who inject narcotics or other drugs intravenously may introduce infection, which will travel to the right side of the heart classically affecting the tricuspid valve, and most often caused by *S. aureus*.

In other patients without a history of intravenous exposure, endocarditis is more frequently left-sided. Another form of endocarditis is nosocomial endocarditis which is when the patient is diagnosed with endocarditis and has had hospital care one month prior to the incident and is usually secondary to IV catheters, Total parenteral nutrition lines, pacemakers, and so.

Besides, Endocarditis can have a classification according its affected valve types: The distinction between native-valve endocarditis and prosthetic-valve endocarditis is clinically important. Prosthetic valve endocarditis can be early (< 60 days of valvular surgery) or late (> 60 days of valvular surgery).

Early prosthetic-valve endocarditis is usually due to intraoperative contamination or a postoperative bacterial contamination, which is usually nosocomial in nature. Late prosthetic valve endocarditis is usually due to community acquired microorganisms.

In a healthy individual, a bacteraemia (where bacteria get into the blood stream through a minor cut or wound) would normally be cleared quickly with no adverse consequences. If a heart valve is damaged and covered with a piece of a blood clot, the valve provides a place for the bacteria to attach themselves and an infection can be established.

In the past, bacteremia caused by dental procedures (in most cases due to viridans streptococci, which reside in oral cavity), such as a cleaning or extraction of a tooth was thought to be more clinically significant than it actually was. However, it is important that a dentist or a dental hygienist be told of any heart problems before commencing treatment. Antibiotics are administered to patients with certain heart conditions as a precaution, although this practice has changed in the US, with new American Heart Association guidelines released in 2007, and in the UK as of March 2008 due to new NICE guidelines. Everyday tooth brushing and flossing will similarly cause bacteremia. Although there is little evidence to support antibiotic prophylaxis for dental treatment,

the current American Heart Association guidelines are highly accepted by clinicians and patients.

Another group of causes results from a high number of bacteria getting into the bloodstream. Colorectal cancer (mostly *Streptococcus bovis*), serious urinary tract infections (mostly enterococci), and drug injection (*S. aureus*) can all introduce large numbers of bacteria. With a large number of bacteria, even a normal heart valve may be infected.

A more virulent organism (such as *S. aureus*, but see below for others) is usually responsible for infecting a normal valve.

Intravenous drug users tend to get their right-sided heart valves infected because the veins that are injected enter the right side of the heart, so they will have injured valves on that side that the bacteria can bind to. In rheumatic heart disease infection occurs on the aortic and the mitral valves, on the left side of the heart.

Other factors that increase the risk of developing infective endocarditis are low levels of white blood cells, immunodeficiency or immunosuppression, malignancy, diabetes, and alcohol abuse.

As we have already said, altered blood flow around the valves is a risk factor for obtaining endocarditis. The valves may be damaged congenitally, from surgery, from auto-immune mechanisms, or simply as a consequence of old age. The damaged part of a heart valve becomes covered with a blood clot, a condition known as non-bacterial thrombotic endocarditis (NBTE). Altered blood flow, and thus infective endocarditis, is more likely in high pressure areas.

Consequently, ventricular septal defects create more susceptibility than atrial septal defects. Damaged vascular endothelium will also promote platelet and fibrin deposition, upon which bacteria can take hold. Valvular lesions are a major cause of such damage, as are jet lesions resulting from ventricular septal defects or patent ductus arteriosus.

All patients should fulfill the Duke criteria in order to establish the diagnosis of endocarditis. As the Duke criteria rely heavily on the results of echocardiography, research has addressed when to order an echocardiogram by using signs and symptoms to predict occult endocarditis among patients with intravenous drug abuse and among non drug-abusing patients. Unfortunately, this research is over 20 years old and it is possible that changes in the epidemiology of endocarditis and bacteria such as staphylococci can make the following estimates incorrect.

2. Duke criteria

Established in 1994 by the Duke Endocarditis Society and revised in 2000, the Duke criteria are a collection of major and minor criteria used to establish a diagnosis of endocarditis. A diagnosis can be reached in any of three ways: two major criteria, one major and three minor criteria, or five minor criteria.

Major criteria include:

Positive blood culture with typical IE microorganism, defined as one of the following:

Typical microorganism consistent with IE from 2 separate blood cultures, as noted below:
 Viridans-group streptococci, or
 S. bovis including nutritional variant strains, or
 HACEK group, or
 S. aureus, or
 Community-acquired enterococci, in the absence of a primary focus
 Microorganisms consistent with IE from persistently positive blood cultures defined as:
 Two positive cultures of blood samples drawn >12 hours apart, or
 All of 3 or a majority of 4 separate cultures of blood (with first and last sample drawn 1 hour apart)
 Coxiella burnetii detected by at least one positive blood culture or antiphase I IgG antibody titer >1:800
 Evidence of endocardial involvement with positive echocardiogram defined as
 Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation, or
 Abscess, or
 New partial dehiscence of prosthetic valve or new valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)

Minor criteria include:

Predisposing factor: known cardiac lesion, recreational drug injection
 Fever >38°C
 Evidence of embolism: arterial emboli, pulmonary infarcts, Janeway lesions, conjunctival hemorrhage
 Immunological problems: glomerulonephritis, Osler's nodes
 Positive blood culture (that doesn't meet a major criterion) or serologic evidence of infection with organism consistent with IE but not satisfying major criterion

High dose antibiotics are administered by the intravenous route to maximize diffusion of antibiotic molecules into vegetation(s) from the blood filling the chambers of the heart. This is necessary because neither the heart valves nor the vegetations adherent to them are supplied by blood vessels. Antibiotics are continued for a long time, typically two to six weeks. Specific drug regimens differ depending on the classification of the endocarditis as acute or subacute (acute necessitating treating for S. aureus with oxacillin or vancomycin in addition to gram-negative coverage). Fungal endocarditis requires specific anti-fungal treatment, such as amphotericin B. In acute endocarditis, due to the fulminant inflammation empirical antibiotic therapy is started immediately after the blood has been drawn for culture. This usually includes oxacillin and gentamicin IV infusions until the culture sensitivity report with the minimum inhibitory concentration comes, when the therapy can be modified to tailor to the microorganism.

There should be noted that the routine use of gentamicin to treat Staphylococcal endocarditis has been questioned, given the lack of evidence to support its use and the high rate of complications.

In subacute endocarditis, antibiotic treatment is based on the micro organism involved, requiring the culture sensitivity report.

So immediate therapy is mainly focused on symptomatic treatment.

The most common organism responsible for infective endocarditis is viridans-group streptococci, which are highly sensitive to penicillin. High dose IV crystalline penicillin every 4hrs for 2 weeks is recommended and still remains the drug of choice.

Again it is important to note that antibiotic therapy hinges upon the culture sensitivity report.

The short course treatment in patients where the blood culture reveals the causative organism, culture sensitivity reports should be followed to treat the patient,

In addition to usage of two bactericidal antibiotics for a minimum of two weeks as a combination therapy.

Surgical debridement of infected material and replacement of the valve with a mechanical or bioprosthetic artificial heart valve is necessary in patients who fail to clear micro-organisms from their blood in response to antibiotic therapy, or in patients who develop cardiac failure resulting from destruction of a valve by infection. other indications to consider surgery include:

- unstable prosthetic valve or obstruction
- recurrent septic emboli, mycotic aneurysm
- large vegetations
- abscess formation
- early closure of mitral valve
- gram negative species

Infective endocarditis is associated with 25% mortality.

3. Non-infective endocarditis

Nonbacterial thrombotic endocarditis (NBTE) or marantic endocarditis is most commonly found on previously undamaged valves.

As opposed to infective endocarditis, the vegetations in NBTE are small, sterile, and tend to aggregate along the edges of the valve or the cusps.

Also unlike infective endocarditis, NBTE does not cause an inflammation response from the body (confusing, as the suffix "-itis" refers to inflammation).

NBTE usually occurs during a hypercoagulable state such as system wide bacterial infection, or pregnancy, though it is also sometimes seen in patients with venous catheters. NBTE may also occur in patients with cancers, particularly mucinous adenocarcinoma (where Trousseau syndrome can be encountered). Typically NBTE does not cause many problems on its own, but parts of the vegetations may break off and embolize to the heart or brain, or they may serve as a focus where bacteria can lodge, thus causing infective endocarditis.

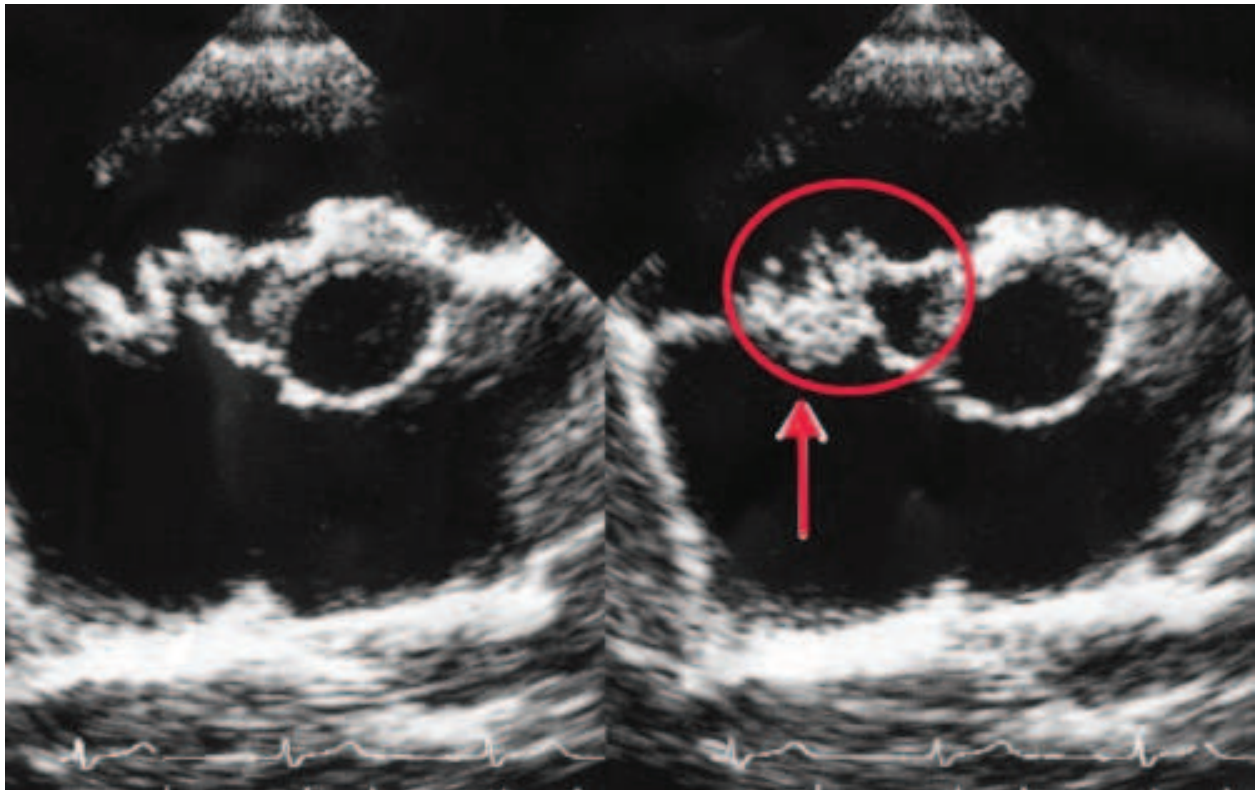


Fig. 2. Vegetation on the tricuspid valve by echocardiography. (Arrow denotes the vegetation)

Another form of sterile endocarditis, is termed LibmanSacks endocarditis; this form occurs more often in patients with lupus erythematosus and is thought to be due to the deposition of immune complexes. Like NBTE, LibmanSacks endocarditis involves small vegetations, while infective endocarditis is composed of large vegetations. These immune complexes precipitate an inflammation reaction, which helps to differentiate it from NBTE. Also unlike NBTE, LibmanSacks endocarditis does not seem to have a preferred location of deposition and may form on the under surfaces of the valves or even on the endocardium.]

Prognosis:

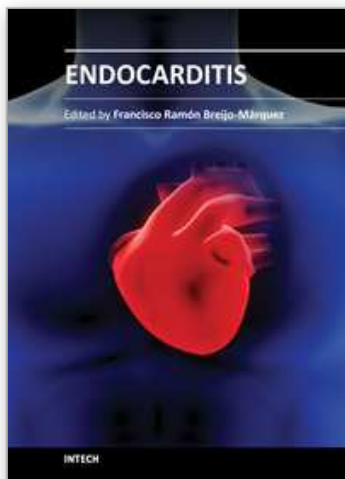
Features suggestive of a worse prognosis are Acute endocarditis (*Staphylococcus aureus*), heart failure, IV drug abuse (often left and right sided disease), prosthetic valve infection, infection of the aortic rather than mitral valve, associated rhythm disturbance.

Subacute bacterial endocarditis (*Streptococcus viridans*) has a better prognosis.

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Endocarditis

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Endocarditis is a disease that occurs as a result of the inflammation of the endocardium. It is an inflammatory process located in the inner lining of the cardiac chambers and native or prosthetic valves. It is characterized by colonization or invasion of the heart valve vegetations composed of platelets forming, fibrin and microcolonies of microorganisms, and occasionally of inflammatory cells. Other structures may also be affected, such as the interventricular septum, chordae tendineae, the mural endocardium or even intra-cardiac implants. The book covers, with scientific rigour, the most prevalent causes and current treatments of endocarditis, as well as the cases when the organs remote from the heart are affected by this disease.

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