# Infective endocarditis (pediatrics)

**Infective Endocarditis** (IE) is a disease caused by an infectious agent that affects the endocardium, heart valves and adjacent structures. Inflammation can be caused by bacteria, fungi, chlamydia, rickettsia or viruses.

## Risk Factors

Risk factors in children:

- congenital heart defects;
- rheumatic heart defects (rare);
- iatrogenic – long-term central venous catheters;
- intravenous drug use;
- bicuspid aortic valve;
- mitral valve prolapse with regurgitation;
- degrees cardiac operations using conduits and vascular prostheses, with artificial valves.**Cite error: Invalid <ref> tag; invalid names, e.g. too many**

## Classification

- IE native flaps,
- IE of drug addicts (predisposes to tricuspid valve involvement with risk of pulmonary embolism),
- IE of valve prostheses (early/late onset - limit 2 months after surgery).

The division of IE into acute and subacute form is already obsolete and is not used. Division according to the inducing agent is recommended. Microorganisms with low virulence (e.g. α-hemolytic streptococci) usually cause a "subacute" form, on the contrary, *Staphylococcus aureus* and other pyogenic bacteria cause "acute" forms.

## Risks of infective endocarditis

**high risk;**

- valve prostheses (for life),
- degrees heart surgery (up to 6 months after surgery),
- aortic defects,
- Tetralogy of Fallot,
- mitral insufficiency,
- PDA,
- VSD,
- CoA,
- Marfan syndrome,
- History of IE.

**medium risk;**

- mitral stenosis,
- tricuspid defects,
- mitral prolapse,
- hypertrophic cardiomyopathy.

## Clinical picture

We always suspect IE in high-risk patients (see above) with febrile condition. It is always necessary to keep in mind the mitigated forms of IE at p.o. treatment with antibiotics, which was administered out of embarrassment, fevers may then subside. Endocarditis is most often manifested by ``temperatures *and* ``*nonspecific problems* such as myalgia, arthralgia, headaches, fatigue. If the disease lasts longer, we find splenomegaly, the skin has a color with a touch of white coffee (caffé au lait). Other late symptoms are embolization manifestations on the periphery: splinter-like subungual hemorrhages, petechiae on the skin or subconjunctivally, red spots on the palms (*Janeway's spots*), painful induration on the tips of the fingers (*Osler's nodes*). Embolization may reveal fundus examination (hemorrhagic retinal lesions = Roth spots) or hematuria. In up to 30% of patients, the first clinical symptom of IE may be an acute embolic cause. The basin of the internal carotid artery is most often affected. Clinical symptoms are hemiplegia, aphasia, mental disorders, rarely blindness with retinal artery involvement. In general, IE of the left heart causes embolization to the periphery with subsequent ischemia, infarction in sterile emboli, abscesses in infectious emboli, or mycotic aneurysm. Embolization from the right heart to the lungs is often asymptomatic due to the good filtering properties of the lungs, or symptoms of pulmonary embolism with subsequent cough, auscultatory and X-ray findings on the lungs are manifested.

Sometimes there may even be a picture of *Löhlein's nephritis* with hematuria, proteinuria and a decrease in glomerular filtration. It is a manifestation of microembolization to the kidneys or a consequence of focal or diffuse glomerulonephritis, which causes deposits of immune complexes in the glomeruli. Up to 20% of children have neurological symptoms: meningitis, brain abscesses, toxic encephalopathy.

Significant findings are a newly formed or changed *heart murmur* due to valve involvement, rarely the inflammatory process can affect the conduction system of the heart and cause AV block. Heart failure is the most common cause of deathi.

⚠

## Diagnostics

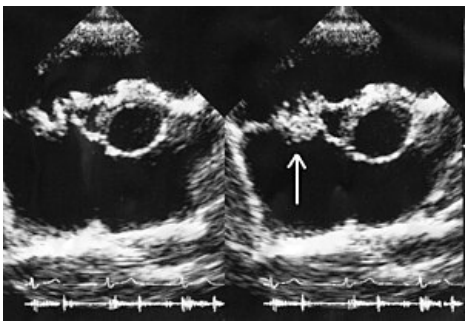
### Laboratory examination

From common laboratory findings, high sedimentation, leukocytosis, microscopic hematuria, proteinuria, positive rheumatoid factor, elevated CIK values are indicative of the diagnosis of IE. We often find anemization and hypergammaglobulinemia. Correct collection of **blood culture'** is crucial for the diagnosis and treatment of IE. We take 3 blood cultures within 24 hours, and in case of negative findings on the second day of incubation, another 2 blood cultures are taken. In case of clinical suspicion of IE, blood cultures are also taken from subfebrile or afebrile patients. Blood culture negativity may be due to previous antibiotic therapy, IE caused by rickettsiae, chlamydiae, viruses, or slow-growing organisms. Molecular biological methods such as PCR bring further possible precision in the diagnosis of IE. Negative blood cultures can also support the diagnosis of sterile thrombotic endocarditis occurring most often in antiphospholipid syndrome.

False positivity of blood cultures is caused by contamination during non-sterile sampling. It is most often coagulase negative staphylococci, corynebacteria, transiently colonizing enterobacteria, pseudomonads, etc. The etiological agent is evidenced by repeated findings, isolation of the same strain from other biological samples and a corresponding clinical picture.

### Echocardiography

Echocardiography is a very valuable method for confirming IE. This examination reliably reveals vegetation on the endocardium and valves and is also important in monitoring the development of possible valvular regurgitation or other heart findings. For unclear findings, we also use transesophageal echocardiography. Echocardiographic diagnosis of IE on an artificial valve is very difficult, in which vegetations are often hidden in the shadow of a strong signal caused by echodense material.



Tricuspid valve vegetation (ECHO)

⚠

### Special examination

When embolization into the pulmonary or systemic flow is suspected, special imaging examinations CT scan, MRI are indicated to prove or rule out septic emboli or abscesses.

We always consult a dentist or ENT doctor to rule out focal infection. Remediation of the infectious site is carried out during the treatment of IE.

### Diagnostic criteria

Currently, the criteria proposed by Durack from Duke University in the USA (the so-called Duke criteria) are recommended for the diagnosis of IE, which are based on a combination of clinical, laboratory and echocardiographic findings. According to these criteria, patients with suspected IE are divided into 3 categories: *proven IE, possible IE* and *ruled out IE*.

***Definition of IE Criteria (Duke Criteria):***

**main criteria**

- *positive blood culture:* from 2 different blood samples a typical microorganism for IE was detected (*Streptococcus viridans* including nutritional variants or *Streptococcus bovis*, or microorganisms of the *HACEK* group; *Staphylococcus aureus* ' or *Enterococcus spp.*, if no other primary source of infection was detected) / repeatedly positive blood cultures, if there was: the same finding in 2 blood cultures taken within 12 hours or more or the same finding in 3 or 3 out of 4 blood cultures, if the interval between the first and last sampling was greater than 1 hour
- *signs of endocardial involvement:* echocardiographic findings corresponding to IE (fluttering intracardiac structures on the valve or on surrounding structures at the site of accelerated blood flow, or on foreign material for which there is no other anatomical explanation; abscess; newly formed paravalvular dehiscence at the site of artificial valves) / newly formed valve regurgitation

**secondary criteria**

- *predisposition:* structural heart disease, abusus i.v. drugs,
- "temperatures" ≥ 38.0° C,
- *vascular symptoms:* embolization, septic pulmonary infarction, intracranial hemorrhage, conjunctival hemorrhage, and skin petechiae,
- *immunological symptoms:* glomerulonephritis, Osler nodules, Roth spots, rheumatoid factor,

- *microbiological finding:* a positive blood culture that does not meet the main criteria above, or serological evidence of active infection consistent with IE,
- "Echocardiographic finding" corresponding to IE, but not meeting the above main criteria.

***Diagnosis of IE (Duke Criteria):***

**proven IE**

- pathological criteria (at least 1 criterion): proven microorganism by culture or histologically in vegetation or embolization of vegetation or intracardiac abscess, or histological evidence of active IE in vegetation or in intracardiac abscess,
- clinical criteria: 2 main criteria, or 1 main and 3 minor criteria, or 5 minor criteria.

**maybe IE**
1 main + 1 minor criteria / 3 minor criteria

**IE excluded**
proven other diagnosis explaining the symptoms of the disease / disappearance of IE symptoms during 4 days of antibiotic treatment / absence of IE findings during surgery or at autopsy after antibiotic treatment during 4 days

## Therapy

For ``empirical treatment *or when the blood culture is negative, we choose a combination of oxacillin 200 mg/kg/day every 4 hours + gentamicin 3 mg/kg/day every 12 hours IV, for patients allergic to penicillins then vancomycin 40 mg/kg /day for 6 hours + gentamicin 3 mg/kg/day for 12 hours i.v.*

With a positive blood culture, we take into account the type of microbe detected, its sensitivity and MIC. As a rule, when penicillin-sensitive streptococci are detected, we administer Penicillin G 200,000–400,000 I.U./kg/d i.v. at 4 p.m., possibly + gentamicin. When enterococci and other resistant streptococci are detected, we administer ampicillin 200–300 mg/kg/day every 6 hours i.v. + gentamicin. Gram-negative IE (HACEK) is treated with a combination of III cephalosporins. generation, e.g. ceftriaxone 100 mg/kg/day every 12 hours i.v. or ampicillin 200–300 mg/kg/day IV every 6 hours + gentamicin 3 mg/kg/day IV every 12 hours, Fungal IE is treated with amphotericin B: the initial test dose is 0.1 mg/kg, if it is well tolerated, we increase the dose to 0.5 mg/kg for 1 day and continue for at least 6–8 weeks with a maintenance dose of 1 mg/kg/d i.v. It is usually necessary to supplement the surgical procedure (replacement of infected cstupidly, excision of infected tissue). Aminoglycosides are administered for 14 days (longer administration is associated with a high risk of nephrotoxicity), other antibiotics for 4–6 weeks.

'*Generally* we always choose bactericidal ATB, when combining them we make sure to achieve a synergistic effect. Periodic determination of serum bactericidal activity and monitoring of serum concentration are important, especially with potentially toxic antibiotics (gentamicin, vancomycin). We demonstrate the effectiveness of ATB therapy by eradicating bacteremia in blood culture. In the first 8 weeks after the end of treatment, periodic blood culture control is important, because this is the period when most relapses occur.

In the treatment proposal, we can follow the available guidelines in even more detail, from the current recommendations, for example, according to the American Heart Association ([www.americanheart.org]).

## Prevention

Prevention of IE consists of targeted administration of antibiotics to all at-risk individuals prior to surgical or diagnostic procedures known or suspected to cause transient bacteremia. As a rule, these are procedures in the oral cavity, nasopharynx, digestive or urogenital tract. [1] IE prevention significantly reduced the incidence of this devastating infection after its introduction. The most important thing is to prevent bacteremia in children with structural heart disease. Pay particular attention to the timely treatment of all dental affections, including minor tooth decay, even the first dentition, increased oral hygiene, vigorous therapy of purulent skin affections and respiratory bacterial infections. Prevention of IE, however, does not mean flat-rate treatment of all even non-bacterial infections with antibiotics or permanent administration of antibiotics. Cardiology patients are provided with identification with established principles. In summary, it can be said that the most effective prevention of IE is timely and complete correction of the heart defect.

**diseases requiring routine IE prevention**

- with a congenital heart defect, with the exception of an atrial septal defect,
- with rheumatic or other valvular disease,
- with obstructive form hypertrophic cardiomyopathy,
- with mitral valve prolapse and concomitant regurgitation.[1]

**diseases requiring IE risk prevention**

- the first 6 months after cardiac operations and after interventional catheterization procedures,
- lifelong in patients with an artificial valve including bioprostheses and allografts, after aortopulmonary coupling operations
- in complex cyanotic heart defects (functionally single ventricle, Tetralogy of Fallot, transposition of great

arteries)
- after IE.

**diseases not requiring IE prevention**

- isolated atrial septal defect,
- atrial septal defect and open trachea 6 months after surgery without residual findings,
- mitral valve prolapse without regurgitation,
- past febris rheumatica or Kawasaki disease without valvular involvement,
- functional murmurs,
- implanted pacemaker or defibrillator,
- coronary bypasses.

**performances requiring prevention of IE in patients at risk**

- dental procedures, accompanied by bleeding from the gums or mucous membrane, including professional cleaning of tartar,
- tonsillectomy and adenotomy,
- surgery affecting the mucous membrane of the intestines or the respiratory system,
- bronchoscopy with a rigid bronchoscope,
- dilatation of the esophagus and sclerotization esophageal varices,
- gall bladder surgery,
- cystoscopy and urethral dilation,
- vascularization of the bladder, if an infection is present*,
- urological operations, if there is an infection*,
- prostate surgery,
- incision and drainage of infected tissues*,
- vaginal delivery, if there is an infection*,
- vaginal hysterectomy.

> asterisk – for these procedures, in addition to the recommended prophylaxis, antibiotics are administered according to sensitivity

**performances that do not require IE prevention**

- dental procedures in which there is no bleeding from the gums or mucous membranes, e.g. treatment of tooth decay above the level of the gums,
- loss of the first dentition,
- cardiac catheterization diagnostic,
- endotracheal intubation,
- bronchoscopy with a flexible bronchoscope including biopsy*,
- endoscopic GIT examination including biopsy*,
- transesophageal echocardiography,
- section cesarean,
- if no infection is present: uncomplicated delivery, dilatation of the cervix and curettage, insertion and removal of the intrauterine body*.

> asterisk - except for the risk group of patients

# Links

## Related Articles

- Infective endocarditis
- Heart inflammations (pediatrics): Myocarditis (pediatrics) • Pericarditis (pediatrics)

## References


1. **Cite error: Invalid `<ref>` tag; no text was provided for refs named `KlinPed2012`**


## Source

- HAVRÁNEK, Jiří: *Heart inflammation*. (edited)

This article has been translated from WikiSkripta; ready for the **editor's review**.