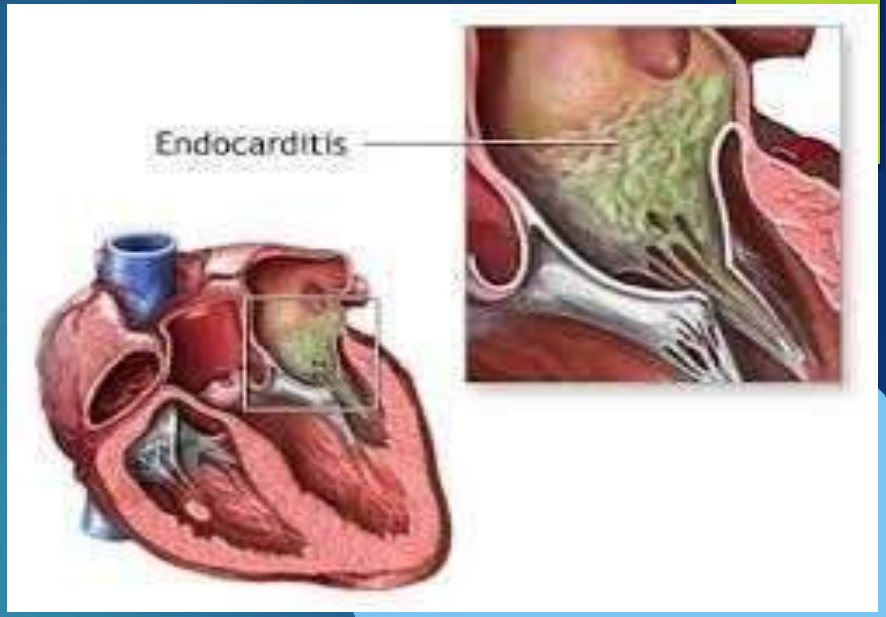
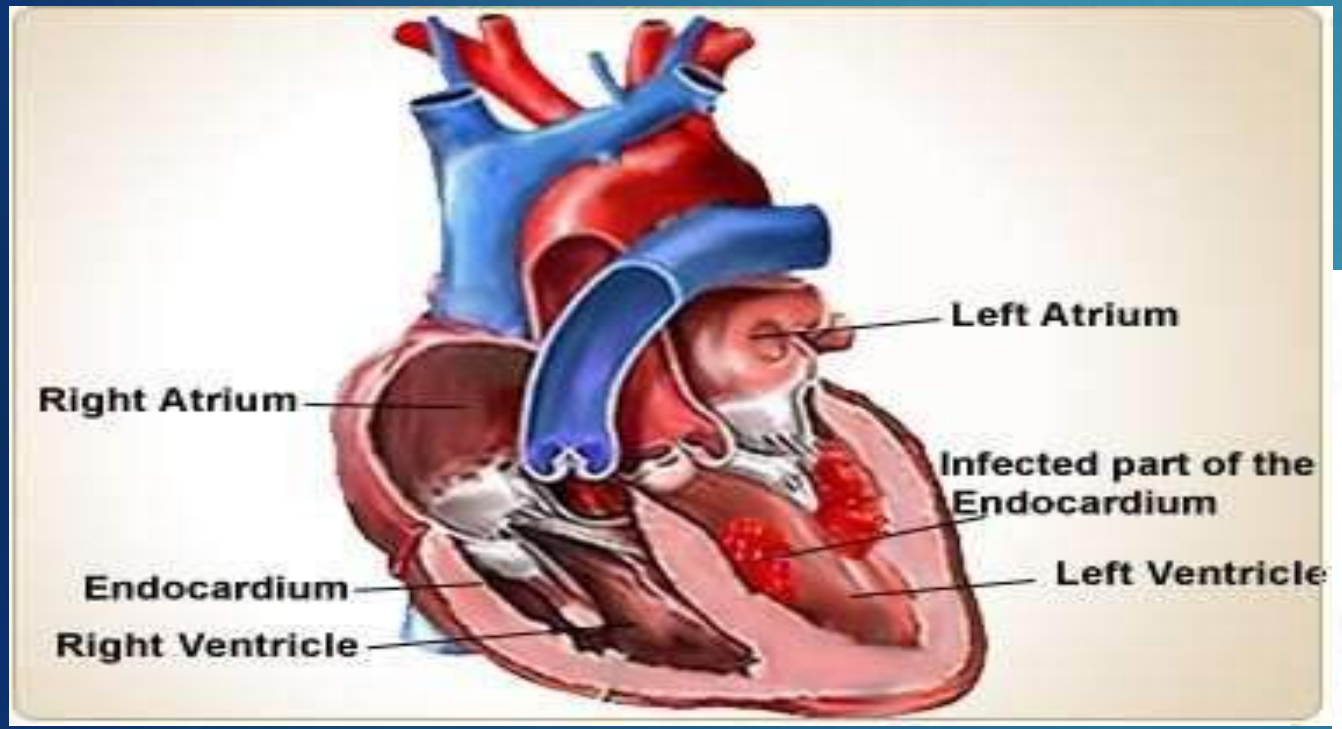
Endocarditis

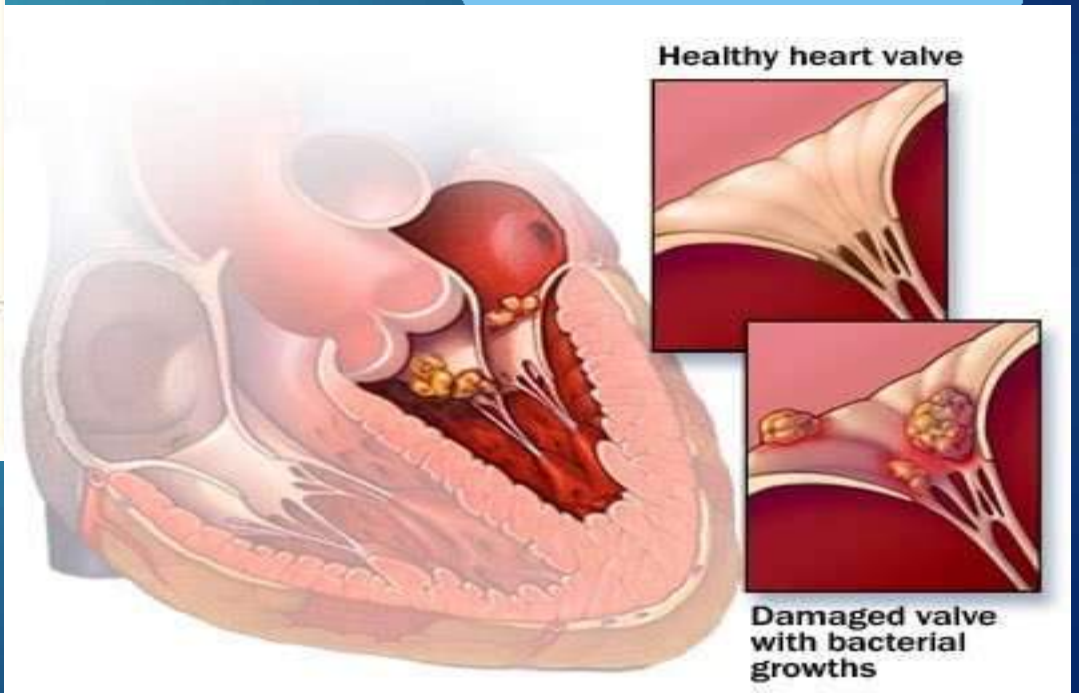
INFECTIVE ENDOCARDITIS

- Infection of the endocardial surface of the heart, which may include one or more heart valves, the mural endocardium, or a septal defect.
- Varieties of IE that were uncommon in the early antibiotic era have become prominent.
- Cases of NIE, IVDA IE, and PVE have markedly increased.
- Valvular infections have entered the era of IE caused by intravascular devices and procedures.

ENDOCARDITIS







Definitions

	Surgery/necropsy	Echocardiography
Vegetation	Infected mass attached to an endocardial structure or on implanted intracardiac material.	Oscillating or non-oscillating intracardiac mass on valve or other endocardial structures, or on implanted intracardiac material.
Abscess	Perivalvular cavity with necrosis and purulent material not communicating with the cardiovascular lumen.	Thickened, non-homogeneous perivalvular area with echodense or echolucent appearance.
Pseudoaneurysm	Perivalvular cavity communicating with the cardiovascular lumen.	Pulsatile perivalvular echo-free space, with colour-Doppler flow detected.
Perforation	Interruption of endocardial tissue continuity.	Interruption of endocardial tissue continuity traversed by colour-Doppler flow.
Fistula	Communication between two neighbouring cavities through a perforation.	Colour-Doppler communication between two neighbouring cavities through a perforation.
Valve aneurysm	Saccular outpouching of valvular tissue.	Saccular bulging of valvular tissue.
Dehiscence of a prosthetic valve	Dehiscence of the prosthesis.	Paravalvular regurgitation identified by TTE/TOE, with or without rocking motion of the prosthesis.

S. Aureus mitral valve vegetation, anterior leaflet

Acute

- Toxic presentation
- Progressive valve destruction & metastatic infection developing in days to weeks
- Most commonly caused by S. aureus

Subacute

- Mild toxicity
- Presentation over weeks to months Rarely
- leads to metastatic infection
- Most commonly S. viridans or enterococcus

EPIDEMIOLOGY AND ETIOLOGY

- Infective endocarditis is an uncommon, but not rare, infection affecting about 10,000 to 20,000 persons annually in the United States.
- accounts for approximately 1 in every 1,000 hospital admissions.
- The mean male-to-female ratio is 1.7:1.

Predisposing factors to bacterial infective endocarditis

1. Dental manipulation and disease
2. Instrumentation (urinary tract, GI tract, IV infusions)
3. Cardiac surgery
4. Injection drug use

PROSTHETIC VALVES

- 7-25% of cases of infective endocarditis
- The rates of infection are the same at 5 years for both mechanical and bioprostheses, but higher for mechanical in first 3 months
- Cumulative risk: 3.1% at 12 months and 5.7% at 60 months post surgery
- Onset:
  - within 2 months of surgery ***early*** and usually hospital acquired
  - 12 months post surgery ***late*** onset and usually community acquired

Aetiological Agents

4. Gram-negative rods

- HACEK group
  - *Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae*.
  - Fastidious oropharyngeal GNBs
  -
- *E. coli, Klebsiella* etc
  - Uncommon
- *Pseudomonas aeruginosa*
  - IVDA
- *Neisseria gonorrhoae*
  - Rare since introduction of penicillin

Aetiological Agents

5. **Others**
   - Fungi
     - *Candida* species, *Aspergillus* species
   - **Chlamydia**
   - **Bartonella**
   - **Legionella**

Clinical Manifestations

Symptoms

- Fever, sweats, chills
- Anorexia, malaise, weight loss

Signs

- Anemia (normochromic, normocytic)
- Splenomegaly
- Microscopic hematuria, proteinuria
- New or changing heart murmur, CHF
- Embolic or immunologic dermatologic signs
- Hypergammaglobulinemia, elevated ESR, CRP, RF

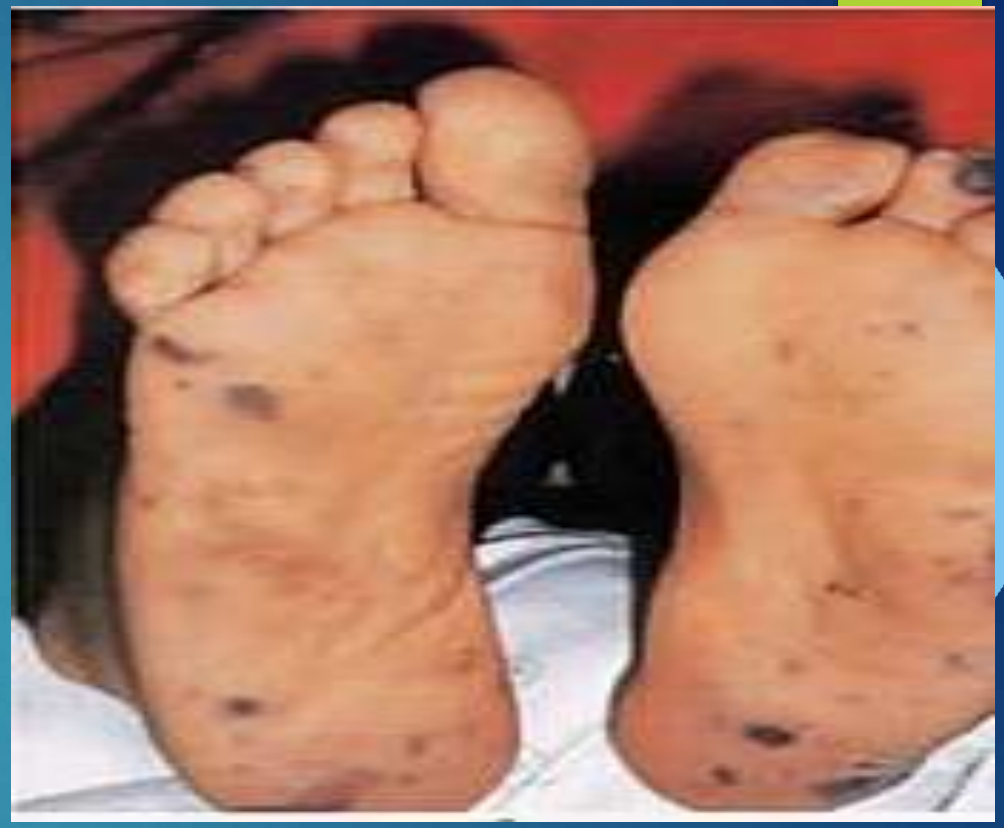
Pathogenesis

- **Bacteraemia**
  - **Transient bacteraemia occurs when a heavily colonised mucosal surface is traumatised**
    - **Dental extraction**
    - **Periodontal surgery**
    - **Tooth brushing**
    - **Tonsillectomy**
    - **Operations involving the respiratory, GI or GU tract mucosa**
    - **Oesophageal dilatation**
    - **Biliary tract surgery**

Siteof Infection

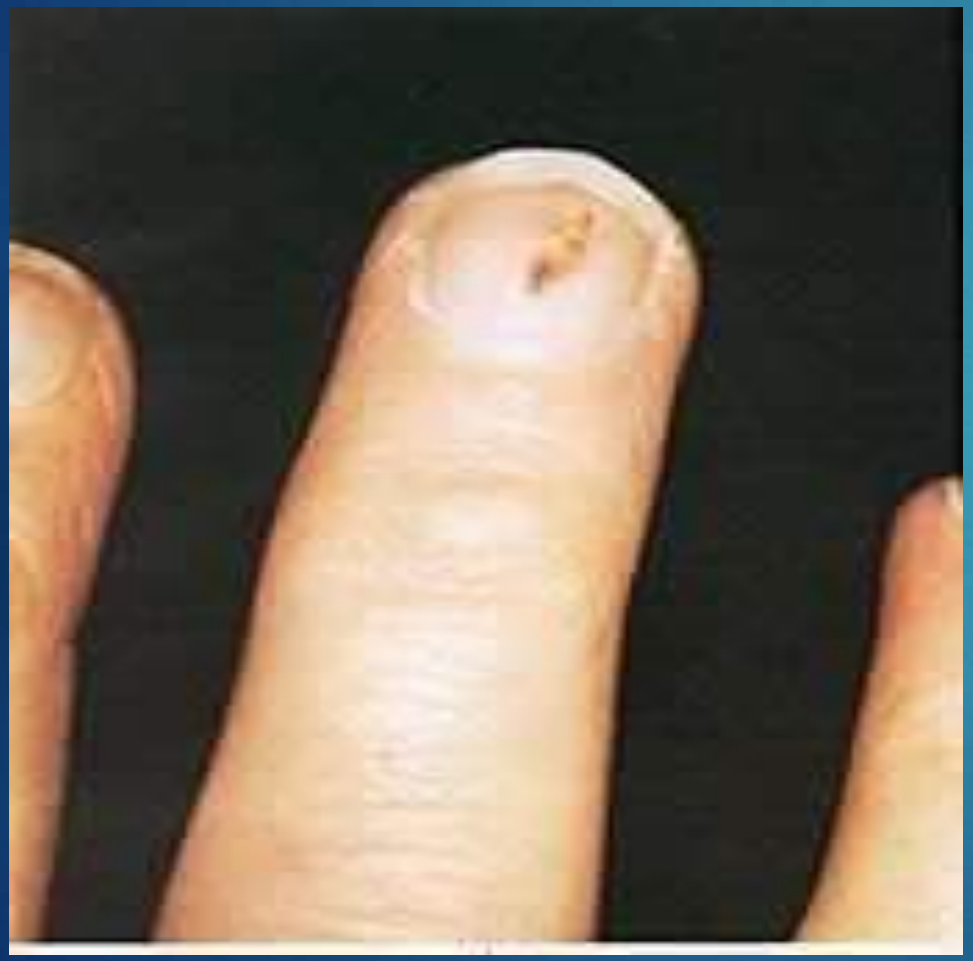
- **Aortic valve more common than mitral**
- **Aortic:**
  - **Vegetation usually on ventricular aspect, all 3 cusps usually affected**
  - **Perforation or dysfunction of valve**
  - **Root abscess**
- **Mitral:**
  - **Dysfunction by rupture of chordae tendineae**

Janeway Lesions



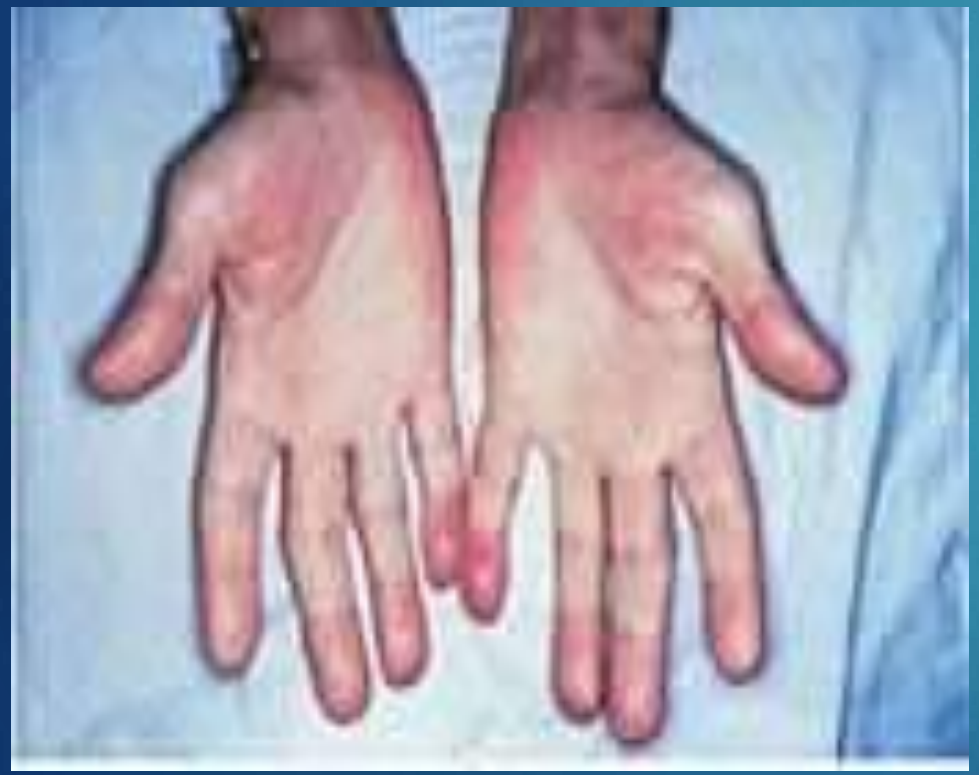
Janeway lesions—Hemorrhagic, painless plaques on the palms of the hands or soles of the feet. These lesions are believed to be embolic in origin.

Splinter Hemorrhage



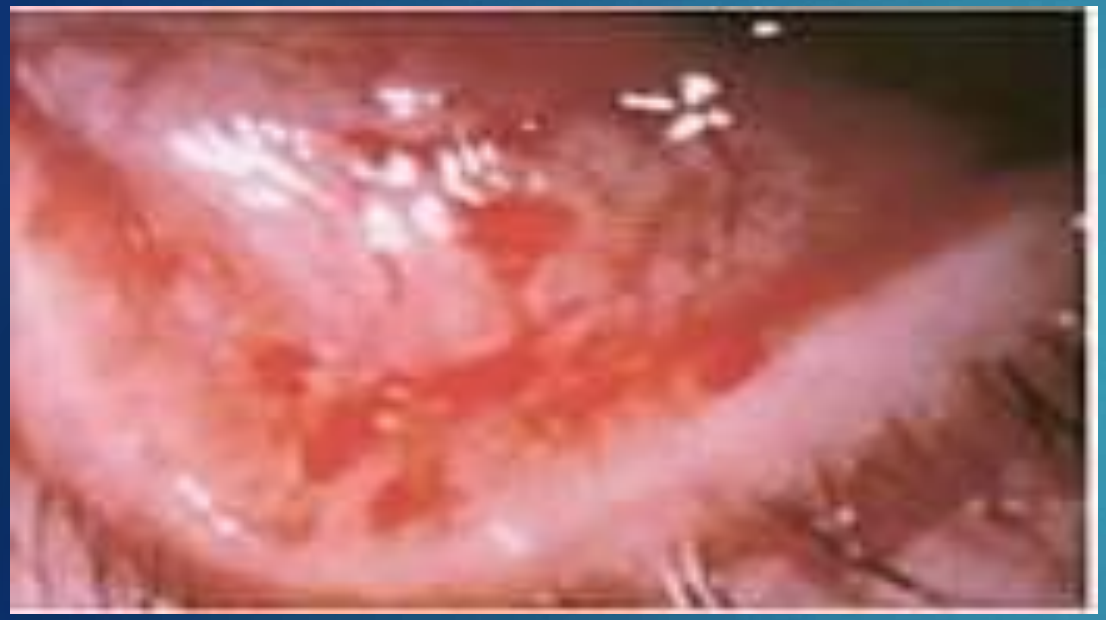
***Splinter hemorrhages*— Thin, linear hemorrhages found under the nail beds of the fingers or toes.**

Osler’s Nodes



Osler nodes **— Purplish or erythematous subcutaneous papules or nodules on the pads of the fingers and toes. These lesions are 2 to 15 mm in size and are painful and tender.**

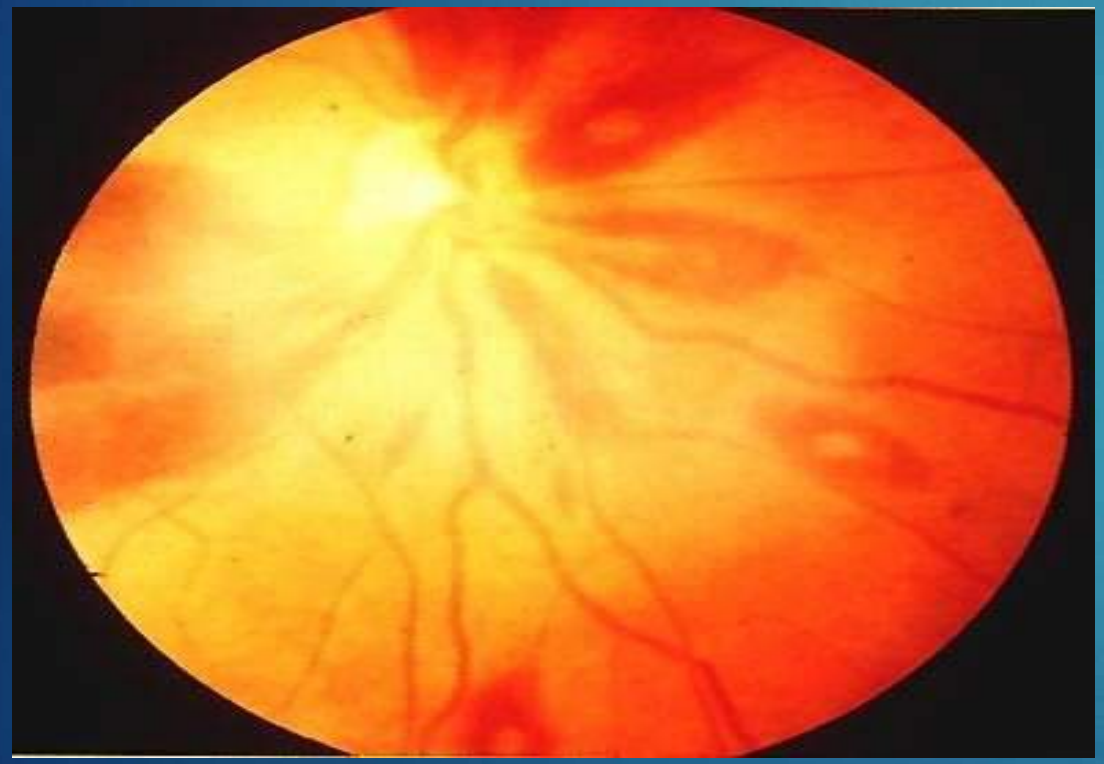
Petechiae



***Petechiae*—Small (usually 1 to 2 mm in diameter), erythematous, painless, hemorrhagic lesions.**

These lesions appear anywhere on the skin but more frequently on the anterior trunk, buccal mucosa and palate, and conjunctivae..

Roth’s Spots



***Roth spots*—Retinal infarct with central pallor and surrounding hemorrhage.**

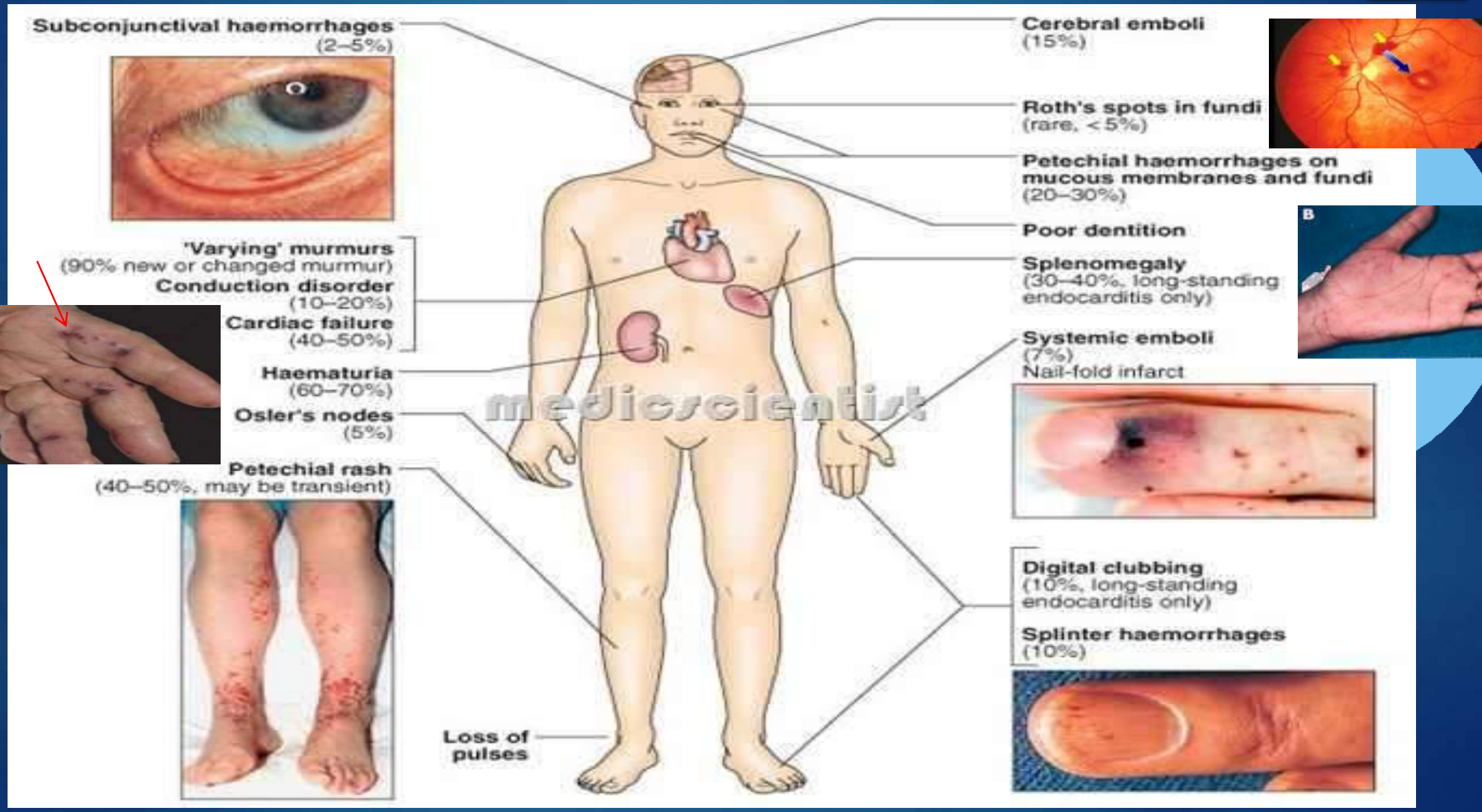
Pretest Probability

High risk patients (pretest probability > 4%):

- **prosthetic valves**
- **congenital heart diseases**
- **previous endocarditis**
- **new murmur**
- **(new onset) heart failure**
- **Other stigmata of endocarditis (see next slide for physical exam).**

Low Risk: EVERYONE ELSE!

Physical Exam findings



Investigations

1. **Blood culture**
2. **Echo**
   - **TTE**
   - **TOE**
1. **FBC/ESR/CRP**
2. **Rheumatoid Factor**

Diagnosis: Duke Criteria

- In 1994 a group at Duke University standardised criteria for assessing patients with suspected endocarditis
- Include

-Predisposing Factors

-Blood culture isolates or persistence of bacteremia

-Echocardiogram findings with other clinical, laboratory findings

Modified Duke's criteria

Definite IE

Pathological criteria

- Microorganisms demonstrated by culture or on histological examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen; or
- Pathological lesions; vegetation or intracardiac abscess confirmed by histological examination showing active endocarditis

Clinical criteria

- 2 major criteria; or
- 1 major criterion and 3 minor criteria; or
- 5 minor criteria

Possible IE

- 1 major criterion and 1 minor criterion; or
- 3 minor criteria

Rejected IE

- Firm alternate diagnosis; or
- Resolution of symptoms suggesting IE with antibiotic therapy for ≤4 days; or
- No pathological evidence of IE at surgery or autopsy, with antibiotic therapy for ≤4 days; or
- Does not meet criteria for possible IE, as above

Definition Of Different Criteria

Major criteria

1. **Blood cultures positive for IE**
   a. Typical microorganisms consistent with IE from 2 separate blood cultures:
      - *Viridans streptococci, Streptococcus gallolyticus (Streptococcus bovis), HACEK group, Staphylococcus aureus*; or
      - Community-acquired enterococci, in the absence of a primary focus; or
   b. Microorganisms consistent with IE from persistently positive blood cultures:
      - ≥2 positive blood cultures of blood samples drawn >12 h apart; or
      - All of 3 or a majority of ≥4 separate cultures of blood (with first and last samples drawn ≥1 h apart); or
   c. Single positive blood culture for Coxiella burnetii or phase I IgG antibody titre >1:800

2. **Imaging positive for IE**
   a. Echocardiogram positive for IE:
      - Vegetation;
      - Abscess, pseudoaneurysm, intracardiac fistula;
      - Valvular perforation or aneurysm;
      - New partial dehiscence of prosthetic valve.
   b. Abnormal activity around the site of prosthetic valve implantation detected by ^{18}F-FDG PET/CT (only if the prosthesis was implanted for >3 months) or radiolabelled leukocytes SPECT/CT.
   c. Definite paravalvular lesions by cardiac CT.

Minor criteria

1. Predisposition such as predisposing heart condition, or injection drug use.
2. Fever defined as temperature >38°C.
3. Vascular phenomena (including those detected by imaging only): major arterial emboli, septic pulmonary infarcts, infectious (mycotic) aneurysm, intracranial haemorrhage, conjunctival haemorrhages, and Janeway's lesions.
4. Immunological phenomena: glomerulonephritis, Osler's nodes, Roth's spots, and rheumatoid factor.
5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE.

Echocardiography

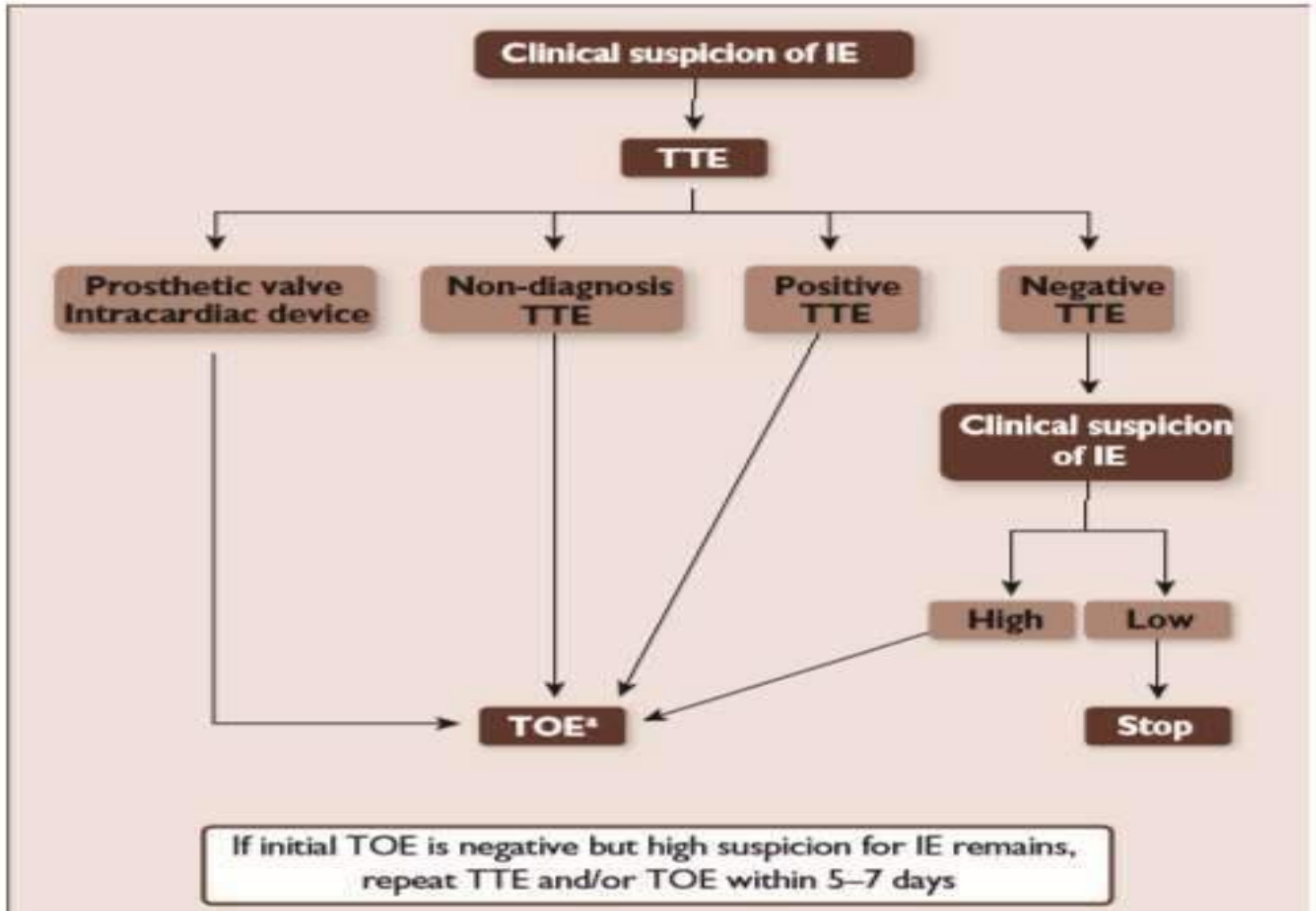
- **Trans Thoracic Echocardiography (TTE)**
  - **r apid, non-invasive– excellent specificity(98%) but poor sensitivity**
  - **obesity, chronic obstructive pulmonary disease and chest wall deformities**
- **Transesophageal Echo (TOE)**
  - **more invasive, sensitivity up to 95%, useful for prosthetic valves and to evaluate myocardial invasion**
  - **Negative predictive valve of 92%**
  - **TOE more cost effective in those with *S. aureus***

catheter-associated bacteraemia and bacteraemia/fever and recent IVDA

ECHO CARDIOGRAPH IN DIAGNOSIS

Recommendations	Class[a]	Level[b]
A. Diagnosis		
• TTE is recommended as the first-line imaging modality in suspected IE.	I	B
• TOE is recommended in all patients with clinical suspicion of IE and a negative or non-diagnostic TTE.	I	B
• TOE is recommended in patients with clinical suspicion of IE, when a prosthetic heart valve or an intracardiac device is present.	I	B
• Repeat TTE and /or TOE within 5–7 days is recommended in case of initially negative examination when clinical suspicion of IE remains high.	I	C
• Echocardiography should be considered in *Staphylococcus aureus* bacteraemia.	IIa	B
• TOE should be considered in patients with suspected IE, even in cases with positive TTE, except in isolated right-sided native valve IE with good quality TTE examination and unequivocal echocardiographic findings.	IIa	C

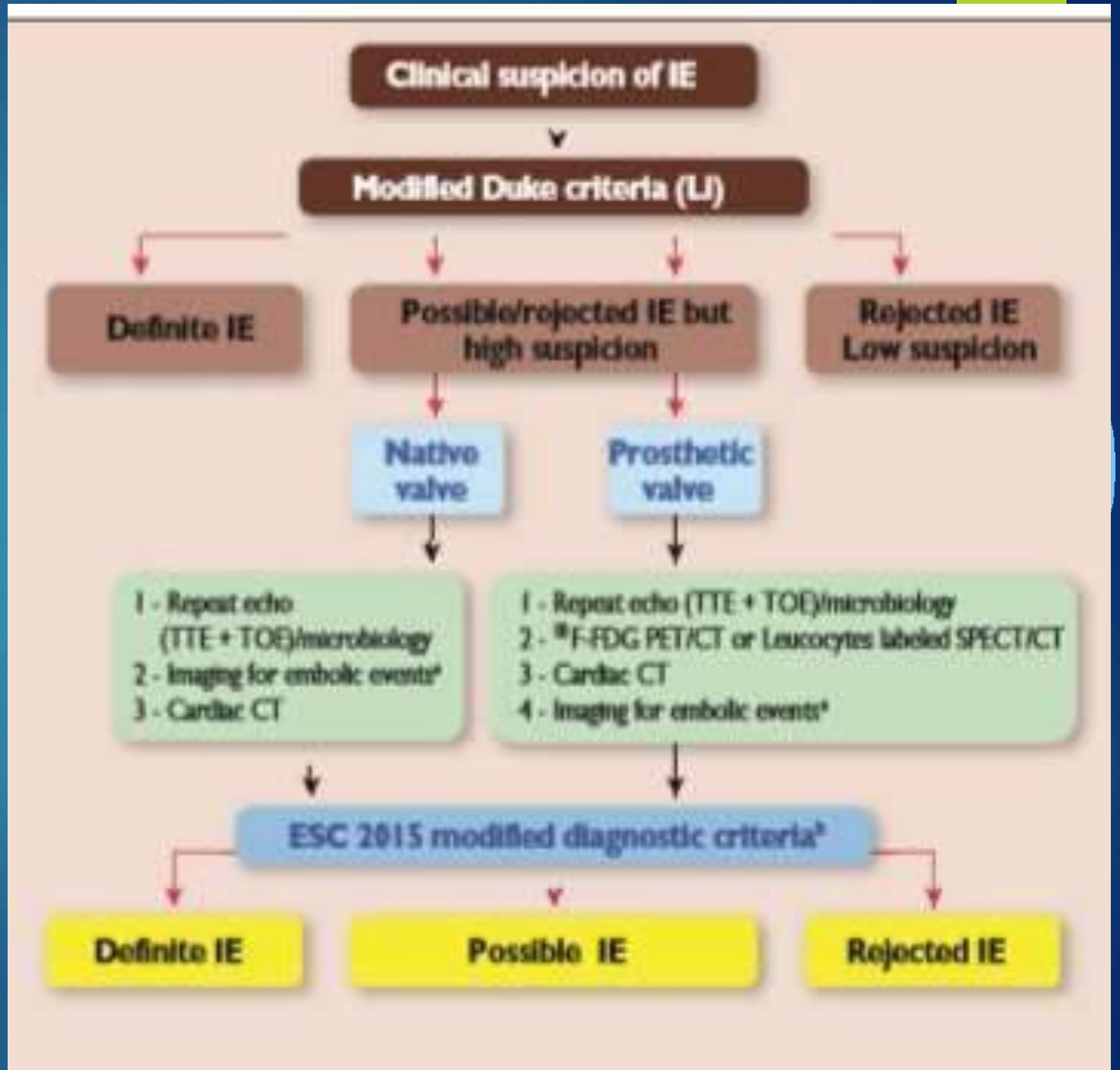
Indication For Echocardiography In I.E.



IE = infective endocarditis; TOE = transoesophageal echocardiography; TTE = transthoracic echocardiography.

[a]TOE is not mandatory in isolated right-sided native valve IE with good quality TTE examination and unequivocal echocardiographic findings.

ESC Algorithm For Diagnosis Of I.E.



Goals of Therapy

1. **Eradicate infection**
2. **Definitively treat sequel of destructive intra-cardiac and extra-cardiac lesions**

The etiologic agents

- Streptococci: 60%-80% .
- Viridans streptococci: 30%-40% .
- Other streptococci: 15%-25%.
- Enterococci: 5%-18% .
- Coagulase-positive organisms: 10%-27%
- Coagulase-negative organisms: 1 %-3%
- Gram-negative aerobic bacilli: 1% -13%
- Fungi: 2%-4% Staphylococci: 20%-35%

ANTIBIOTIC TREATMENT

Antibiotic treatment - oral streptococci and streptococci bovis- penicillin susceptible

Antibiotic	Dosage and route	Duration (weeks)	Class[b]	Level[c]	Ref.[d]	Comments
Strains penicillin-susceptible (MIC ≤ 0.125 mg/L) oral and digestive streptococci						
Standard treatment: 4-week duration						
Penicillin G	12–18 million U/day i.v. either in 4–6 doses or continuously	4	I	B	6,8, 135–139	Preferred in patients > 65 years or with impaired renal or VIII (vestibulocochlear) cranial nerve functions. 6-week therapy recommended for patients with PVE
or Amoxicillin[e]	100–200 mg/kg/day i.v. in 4–6 doses	4	I	B		
or Ceftriaxone[f]	2 g/day i.v. or i.m. in 1 dose	4	I	B		
	Paediatric doses:[g] Penicillin G 200,000 U/kg/day i.v. in 4–6 divided doses Amoxicillin 300 mg/kg/day i.v. in 4–6 equally divided doses Ceftriaxone 100 mg/kg/day i.v. or i.m. in 1 dose					
Standard treatment: 2-week duration						
Penicillin G	12–18 million U/day i.v. either in 4–6 doses or continuously	2	I	B	6,8, 127, 135–138	Only recommended in patients with non-complicated NVE with normal renal function.
or Amoxicillin[e]	100–200 mg/kg/day i.v. in 4–6 doses	2	I	B		
or Ceftriaxone[f]	2 g/day i.v. or i.m. in 1 dose	2	I	B		
combined with Gentamicin[h]	3 mg/kg/day i.v. or i.m. in 1 dose	2	I	B		
or Netilmicin	4–5 mg/kg/day i.v. in 1 dose	2	I	B		Netilmicin is not available in all European countries.
	Paediatric doses:[g] Penicillin G, amoxicillin, and ceftriaxone as above Gentamicin 3 mg/kg/day i.v. or i.m. in 1 dose or 3 equally divided doses					
In beta-lactam allergic patients[i]						
Vancomycin[j]	30 mg/kg/day i.v. in 2 doses	4	I	C		6-week therapy recommended for patients with PVE
	Paediatric doses:[g] Vancomycin 40 mg/kg/day i.v. in 2 or 3 equally divided doses					
Str						

Penicillin resistance :

Strains relatively resistant to penicillin (MIC 0.250–2 mg/l)[k]						
Standard treatment						
Penicillin G *or* Amoxicillin[e] *or* Ceftriaxone[f] combined with Gentamicin[h]	24 million U/day i.v. either in 4–6 doses or continuously 200 mg/kg/day i.v. in 4–6 doses 2 g/day i.v. or i.m. in 1 dose 3 mg/kg/day i.v. or i.m. in 1 dose	4 4 4 2	I I I I	B B B B	6,8, 135, 136	6-week therapy recommended for patients with PVE
In beta-lactam allergic patients[i]						
Vancomycin[j] with Gentamicin[k]	30 mg/kg/day i.v. in 2 doses 3 mg/kg/day i.v. or i.m. in 1 dose **Paediatric doses:**[g] As above	4 2	I I	C C		6-week therapy recommended for patients with PVE

Antibiotic treatment - staphylo-cocci

Antibiotic	Dosage and route	Duration (weeks)	Class[i]	Level[i]	Ref.[h]	Comments
Native valves						
Methicillin-susceptible staphylococci						
(Flu)cloxacillin or oxacillin	12 g/day i.v. in 4–6 doses	4–6	I	B	6,8, 128, 135, 136, 158	Gentamicin addition is not recommended because clinical benefit has not been demonstrated and there is increased renal toxicity
	Paediatric doses:[d] 200–300 mg/kg/day i.v. in 4–6 equally divided doses					
Alternative therapy* Cotrimoxazole[a]	Sulfamethoxazole 4800 mg/day and Trimethoprim 960 mg/day (i.v. in 4–6 doses)	1 i.v. + 5 oral intake	IIb	C		*for *Stahylococcus aureus*
with						
Clindamycin	1800mg/day i.v. in 3 doses	1	IIb	C		
	Paediatric doses:[d] Sulfamethoxazole 60 mg/kg/day and Trimethoprim 12 mg/kg/day (i.v. in 2 doses) Clindamycin 40 mg/kg/day (i.v. in 3 doses)					
Penicillin-allergic patients[b] or methicillin-resistant staphylococci						
Vancomycin[b] **	30–60 mg/kg/day i.v. in 2–3 doses	4–6	I	B	6,8, 135, 136	**Cephalosporins** (cefazolin 6 g/day or cefotaxime 6 g/day i.v. in 3 doses) are recommended for penicillin-allergic patients with non-anaphylactic reactions with methicillin-susceptible endocarditis
	Paediatric doses:[d] 40 mg/kg/day i.v. in 2–3 equally divided doses					
Alternative therapy:** Daptomycin[c,f]	10 mg/kg/day i.v. once daily	4–6	IIa	C		**Daptomycin** is superior to vancomycin for MSSA and MRSA bacteraemia with vancomycin MIC > 1 mg/L
	Paediatric doses:[d] 10 mg/kg/day i.v. once daily					
Alternative therapy* Cotrimoxazole[a]	Sulfamethoxazole 4800 mg/day and Trimethoprim 960 mg/day (i.v. in 4–6 doses)	1 i.v. + 5 oral intake	IIb	C		*for *Stahylococcus aureus*
with						
Clindamycin	1800mg/day IV in 3 doses	1	IIb	C		

Empirical treatment

Empirical treatment of acutely severe ill patient

Antibiotic	Dosage and route	Class[b]	Level[c]	Comments
Community-acquired native valves or late prosthetic valves (≥12 months post surgery) endocarditis				
Ampicillin with (Flu)cloxacillin *or* oxacillin with Gentamicin[d]	12 g/day i.v. in 4–6 doses 12 g/day i.v. in 4–6 doses 3 mg/kg/day i.v. or i.m. in 1 dose	IIa	C	Patients with BCNIE should be treated in consultation with an ID specialist.
Vancomycin[d] with Gentamicin[d]	30–60 mg/kg/day i.v. in 2–3 doses 3 mg/kg/day i.v. or i.m. in 1 dose	IIb	C	For penicillin-allergic patients
Early PVE (<12 months post surgery) or nosocomial and non-nosocomial healthcare associated endocarditis				
Vancomycin[d] with Gentamicin[d] with Rifampin	30 mg/kg/day i.v. in 2 doses 3 mg/kg/day i.v. or i.m. in 1 dose 900–1200 mg i.v. or orally in 2 or 3 divided doses	IIb	C	Rifampin is only recommended for PVE and it should be started 3–5 days later than vancomycin and gentamicin has been suggested by some experts. In healthcare associated native valve endocarditis, some experts recommend in settings with a prevalence of MRSA infections >5% the combination of cloxacillin plus vancomycin until they have the final S. aureus identification

SURGICAL MANAGEMENT

Indication and timing of surgery in left sided valve infective endocarditis

Indications for surgery	Timing[a]	Class[b]	Level[c]
1. Heart failure			
Aortic or mitral NVE or PVE with severe acute regurgitation, obstruction or fistula causing refractory pulmonary oedema or cardiogenic shock	Emergency	I	B
Aortic or mitral NVE or PVE with severe regurgitation or obstruction causing symptoms of HF or echocardiographic signs of poor haemodynamic tolerance	Urgent	I	B
2. Uncontrolled infection			
Locally uncontrolled infection (abscess, false aneurysm, fistula, enlarging vegetation)	Urgent	I	B
Infection caused by fungi or multiresistant organisms	Urgent/ elective	I	C
Persisting positive blood cultures despite appropriate antibiotic therapy and adequate control of septic metastatic foci	Urgent	IIa	B
PVE caused by staphylococci or non-HACEK gram-negative bacteria	Urgent/ elective	IIa	C
3. Prevention of embolism			
Aortic or mitral NVE or PVE with persistent vegetations >10 mm after one or more embolic episode despite appropriate antibiotic therapy	Urgent	I	B
Aortic or mitral NVE with vegetations >10 mm, associated with severe valve stenosis or regurgitation, and low operative risk	Urgent	IIa	B
Aortic or mitral NVE or PVE with isolated very large vegetations (>30 mm)	Urgent	IIa	B
Aortic or mitral NVE or PVE with isolated large vegetations (>15 mm) and no other indication for surgery[e]	Urgent	IIb	C

COMPLICATIONS OF ENDOCARDITIS

- **Cardiac :**
  - congestive cardiac failure-valvular damage, more common with aortic valve endocarditis, infection beyond valve→ CCF, higher mortality, need for surgery, A-V, fascicular or bundle branch block, pericarditis, tamponade or fistulae
- **Systemic emboli**
  - Risk depends on valve (mitral>aortic), size of vegetation, (high risk if >10 mm)
  - 20-40% of patients with endocarditis,
    - risk decreases once appropriate antimicrobial therapy started.

PROGNOSTIC FACTORS

Predictors of poor outcome in patient with infective endocardiris

Patient characteristics
- Older age
- Prosthetic valve IE
- Diabetes mellitus
- Comorbidity (e.g., frailty, immunosuppression, renal or pulmonary disease)

Clinical complications of IE
- Heart failure
- Renal failure
- >Moderate area of ischaemic stroke
- Brain haemorrhage
- Septic shock

Microorganism
- *Staphylococcus aureus*
- Fungi
- Non-HACEK Gram-negative bacilli

Echocardiographic findings
- Periannular complications
- Severe left-sided valve regurgitation
- Low left ventricular ejection fraction
- Pulmonary hypertension
- Large vegetations
- Severe prosthetic valve dysfunction
- Premature mitral valve closure and other signs of elevated diastolic pressures


Thank you

Pericardial Diseases

Acute Pericarditis Etiology

Infectious
- **Viral**
- **Bacterial**
- **TB**

Noninfeccious
- **Post MI (acute and Dresslers)**
- **Uremia**
- **Neoplastic disease**
- **Post radiation**
- **Drug-induced**
- **Connective tissue diseases/autoimmune**
- **traumatic**

Infectious

- Viral (idiopathic)
  - Echovirus, coxsackie B
  - Hepatitis B, influenza, IM, Caricella, mumps
  - HIV, TB
  - Bacterial (purulent)
    - Pneuococcus, staphlococci
    - fulminant

Neoplastic

Breast
Lung
Lymphoma
Primary pericardail tumors rare
Hemmorrhagic and large

Radiation

- Dose > 4000rads
- Local inflammation

Autoimmune

- SLE
- RA
- PSS (40% may develop)

Drugs-lupus like

- Hydralazine
- Procaimamide
- Phenytoin
- Methyldopa
- Isoniazid

Drugs- not lupus

- Minoxidil
- Anthracycline antineoplastic agents

Pathogenesis and Pathology

Inflammatory
- **Vasodilation**
- **Increased vascular permeability**
- **Leukocyte exudation**

Pathology
- **Serous-little cells**
- **Serofibrinous – rough appearance / scarring common**
- **Purulent – intense inflammation**
- **Hemmorrhagic – TB or malignancy**

Clinical

- Chest pain
  - Radiate to back
  - Sharp and pleuritic
  - Positional – worse lying back
- Fever
- Dyspnea due to pleuritic pain

Exam

Friction rub

- **Diaphragm leaning forward**
- **1, 2 or 3 components**
  - **Ventricular contraction, relaxaltion, atrial contraction**
- **intermittent**

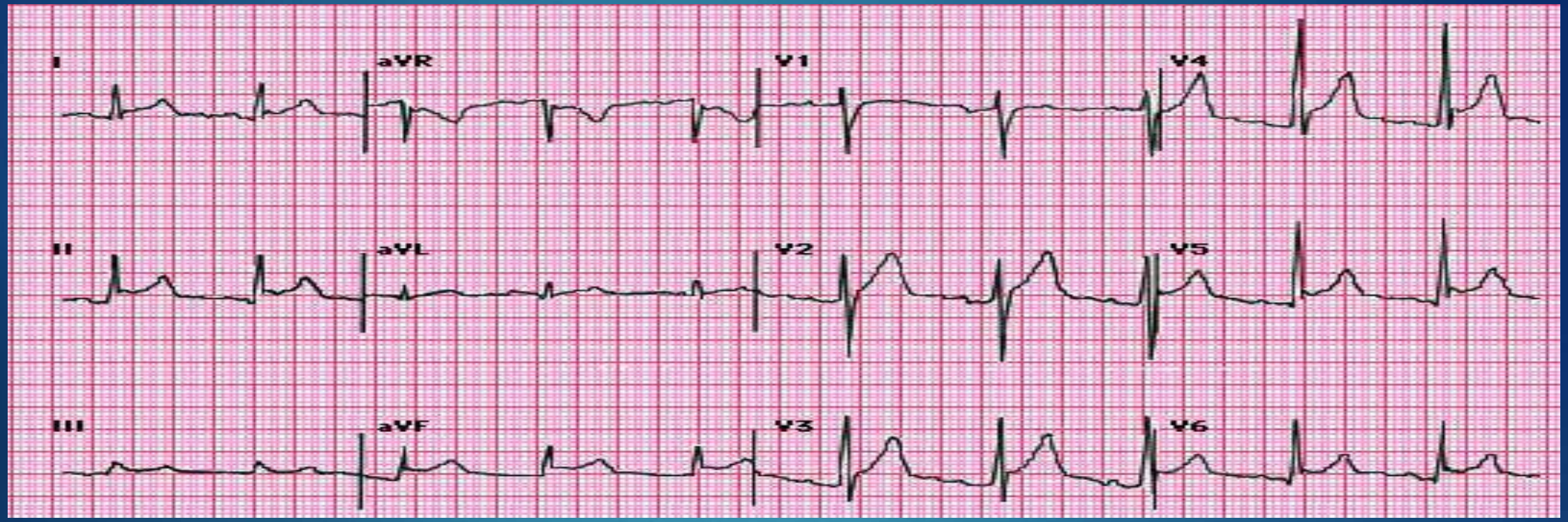
Diagnostic

Clinical history
ECG
- **Abn in 90%**
- **Diffuse ST elevation**
- **PR depression**

Echocardiography
- **Effusion**

PPD
Autoimmune antibodies
Evaluate for malignancy

EKG



Electrocardiogram in acute pericarditis showing diffuse upsloping ST segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR segment deviation (positive in aVR, negative in most other leads). ST segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR segment changes are due to an atrial current of injury which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

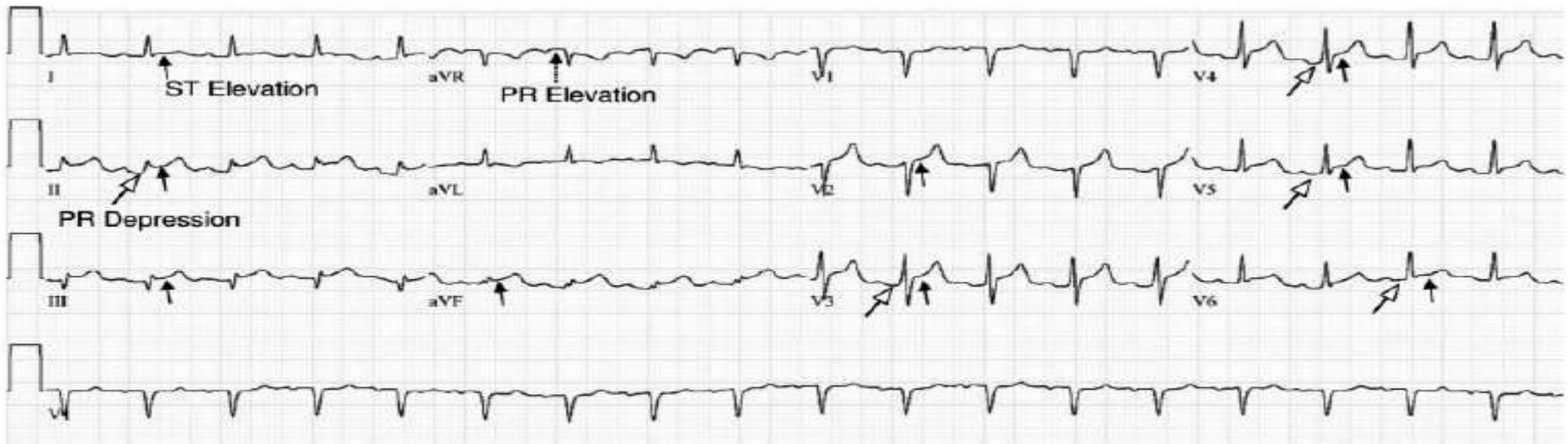


Figure 2. ECG demonstrating typical features seen on presentation of acute pericarditis. There is diffuse ST elevation and PR depression except in aVR, where there is ST depression and PR elevation.

(***Circulation.*** **2006;113:1622-1632.)**

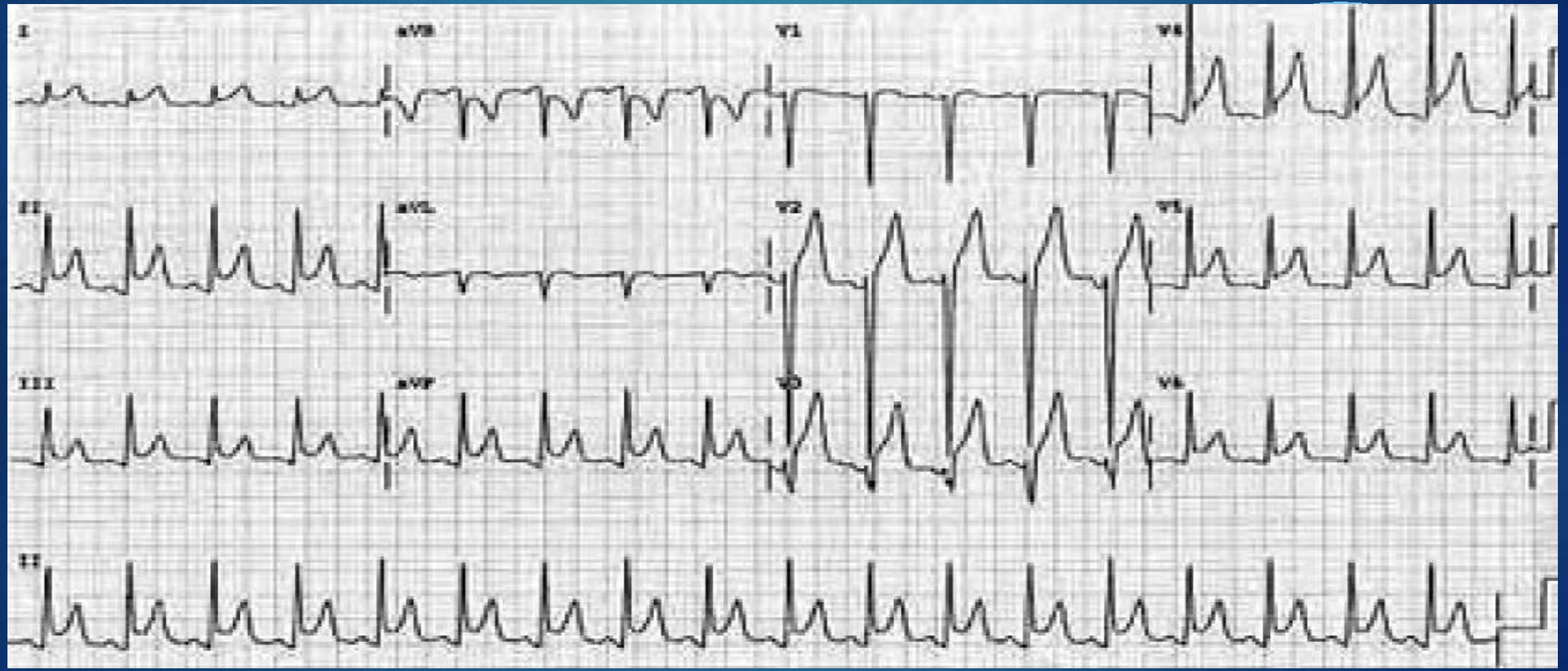

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

TABLE 2. Differentiation of Pericarditis From Myocardial Ischemia/Infarction and Pulmonary Embolism

	Myocardial Ischemia or Infarction	Pericarditis	Pulmonary Embolism
Chest pain			
Character	Pressure-like, heavy, squeezing	Sharp, stabbing, occasionally dull	Sharp, stabbing
Change with respiration	No	Worsened with inspiration	In phase with respiration (absent when the patient is apneic)
Change with position	No	Worse when supine; improved when sitting up or leaning forward	No
Duration	Minutes (ischemia); hours (infarction)	Hours to days	Hours to days
Response to nitroglycerin	Improved	No change	No change
Physical examination			
Friction rub	Absent (unless pericarditis is present)	Present in 85% of patients	Rare; a pleural friction rub is present in 3% of patients
ECG			
ST-segment elevation	Localized convex	Widespread concave	Limited to lead III, aVF, and V_1
PR-segment depression	Rare	Frequent	None
Q waves	May be present	Absent	May be present in lead III or aVF or both
T waves	Inverted when ST segments are still elevated	Inverted after ST segments have normalized	Inverted in lead II, aVF, or V_1 to V_4 while ST segments are elevated

Adapted with permission from Lange and Hillis.[10] Copyright 2004, Massachusetts Medical Society.

(***Circulation.*** **2006;113:1622-1632.)**

Treatment

ASA or NSAIDs
- **Avoid NSAID in MI**

Colchicine

Steroids - avoid
- **May increase reoccurance**

TB – Rx TB

Purulent – drainage of fluid + antibiotics

Neoplastic- drainage

Uremic - dialysis

Pericardial Effusion

From any acute pericarditis
Hypothyriodism- increased capillary permeability
CHF- increased hydrostatic pressure
Cirrhosis- decreased plasma oncotic pressure
Chylous effusion- lymphatic obstruction
Aortic Dissection

Effusion Pathophysiology

Pericardium is stiff- PV curve not flat

Above critical volume – rapid increase in pressure

Factors that determine compression

- **Volume**
- **Rate of accumulation**
- **Pericardial compliance**

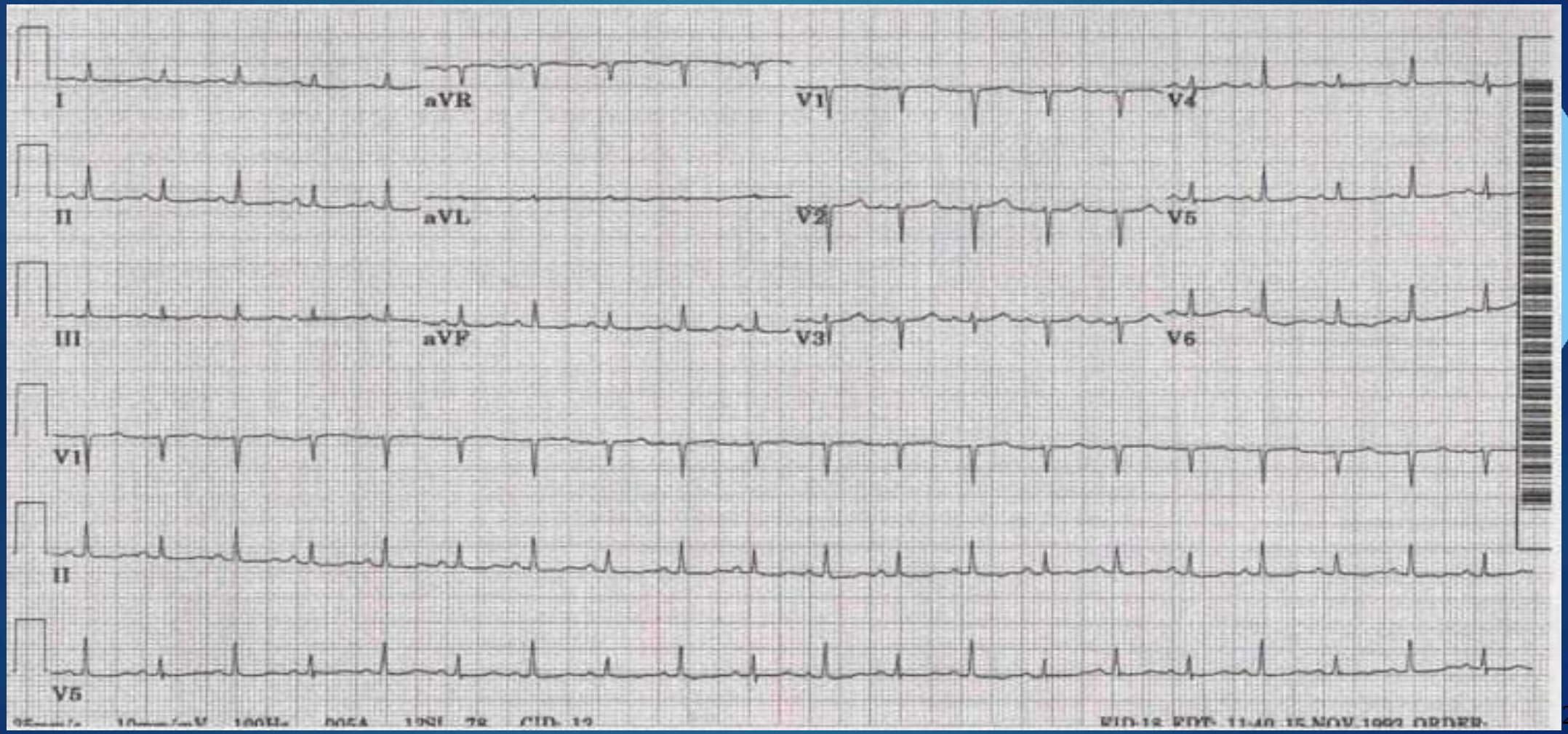
Clinical

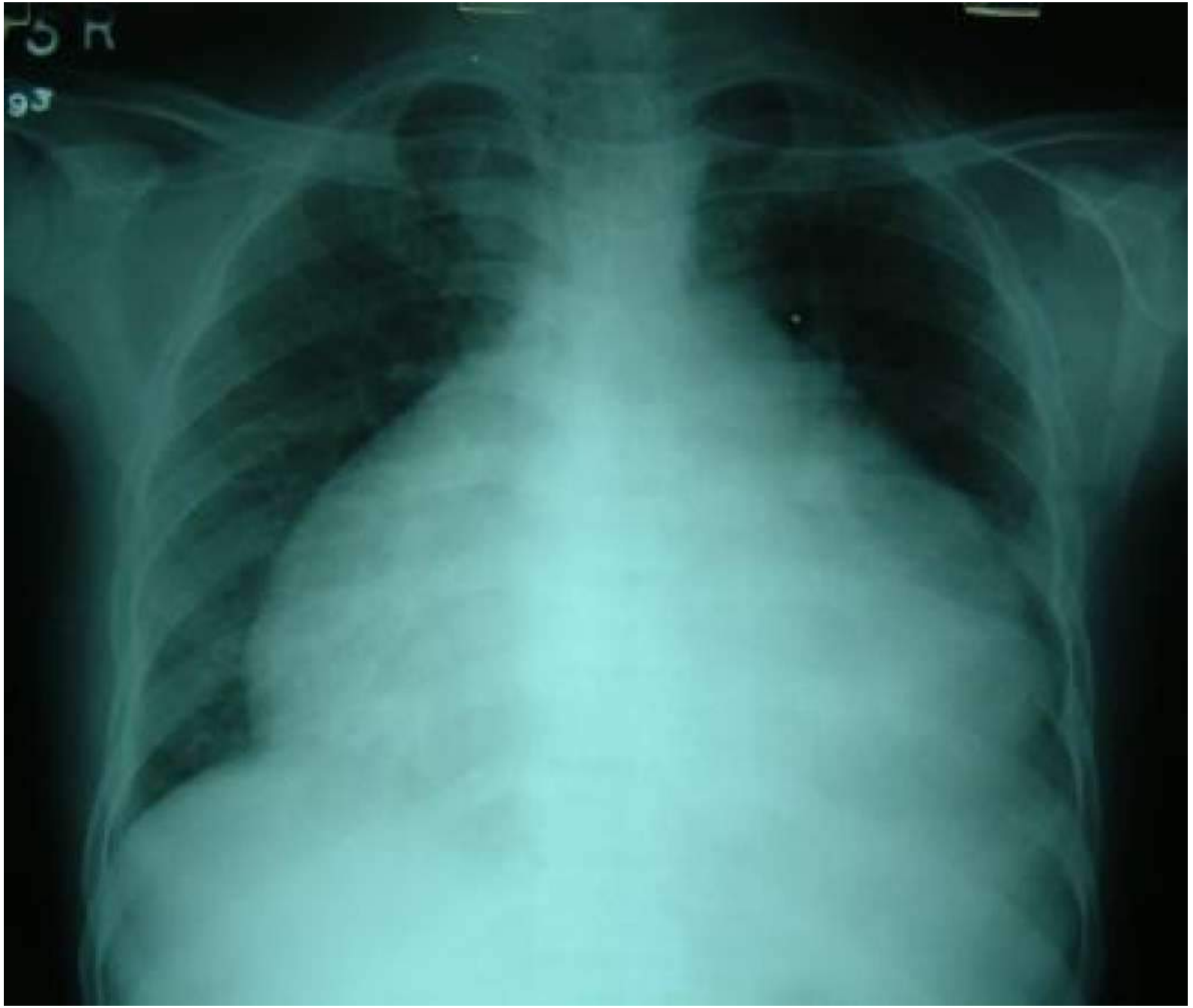
Asymptomatic
Symptoms
    CP, dyspnea, dysphagia, hoarseness, hiccups
Tamponade
Exam
    Muffled heart sounds
    Absence of rub
    Ewarts sign-dullness L lung at scapula
        atelectasis

Diagnostic studies

CXR - > 250 ml fluid globular cardiomegaly
ECG low voltage and electrical alternans
Echocardiogram most helpful
    Identify hemodynamic compromise

ECG low voltage and electrical alternans





Treatment

If known cause- treat that
If unknown- may need pericardiocentesis or pericardial window
Cardiac tamponade is emergency- pericardiocentesis drainage or window

Tamponade

Any cause of effusion may lead to
Diastolic pressures elevate and = pericardial pressure
Impaired LV/RV filling
Increased systemic venous pressure
Decreased stroke volume and C.O.
Shock

Tamponade

Have right side failure with edema and fatigue only if occurs slowly
Key physical findings:
- JVD
- Hypotension
- Small quiet heart

Sinus tachycardia
Pulsus paradoxus- decease in BP > 10 during normal inspiration

Pulsus Paradoxus

Exaggeration of normal

Normally septum moves toward LV with inspiration, with decrease in LV filling

With compression and fixed volume, there is even greater limitation in LV filling and reduced stroke volume

PP also seen in COPD/asthma

Tamponade

Echocardiography

- **Compression of RV and RA in diastole**
- **Can have localized effuison with localized compression of one chamber (RA,LV)**
  - **Effusion post cardiac surgery**
- **Differentiate other causes of low cardiac output**

Cardiac catheterization- definitive

- **Measure pressures- chamber and pericardial equal, and all elevated.**

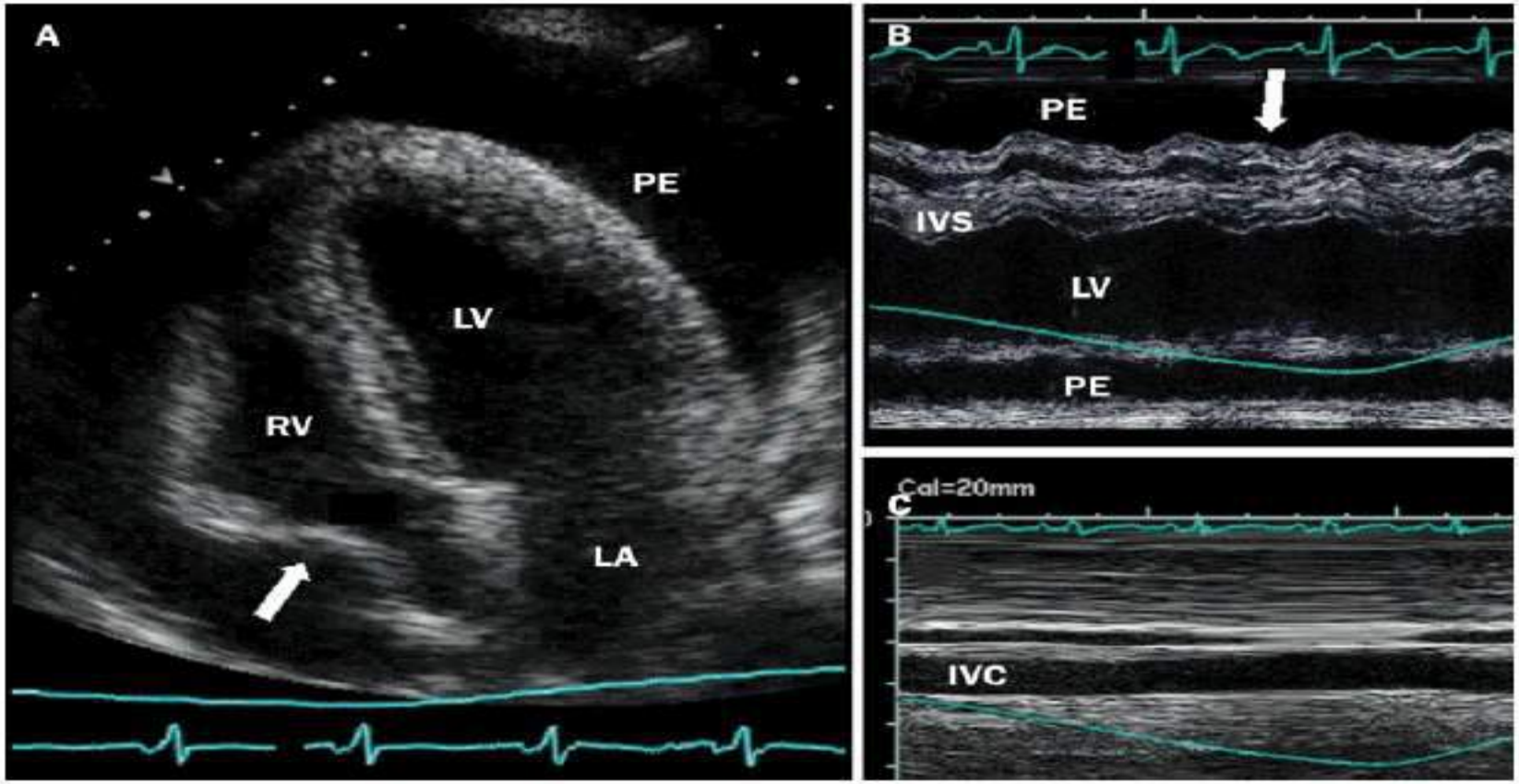


Figure 2: **Echocardiographic images of large pericardial effusion with features of tamponade**

Lancet 2004; 363: 717–27

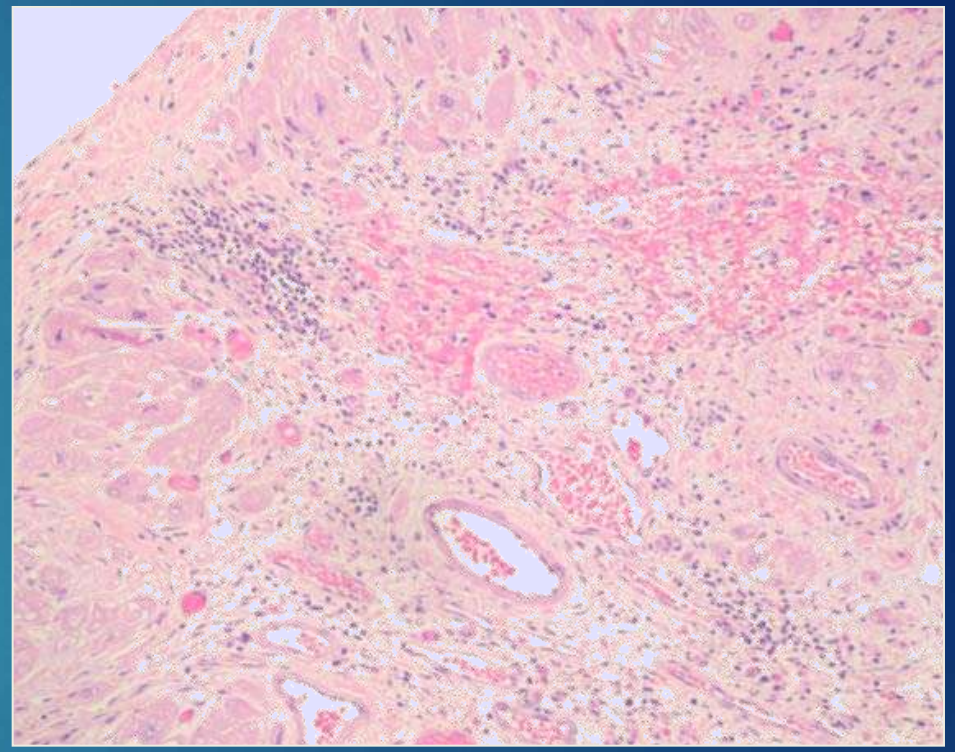
Pericardial Fluid

Stained and cultured
Cytologic exam
Cell count
Protein level
- **pp/sp> 0.5 - exudate**

LDH level
- **p LDH/ s LDH > 0.6 - exudate**

Adenosine Deaminase level - sensitive and specific for TB

Myocarditis



- Inflammatory disease of the myocardium
- Affects children and adults
- Incidence <0.1- 0.6%???
- Acute and Chronic sub-types
- Clinical / histological overlap with dilated cardiomyopathy

Histopathological Definitions

Acute Myocarditis:

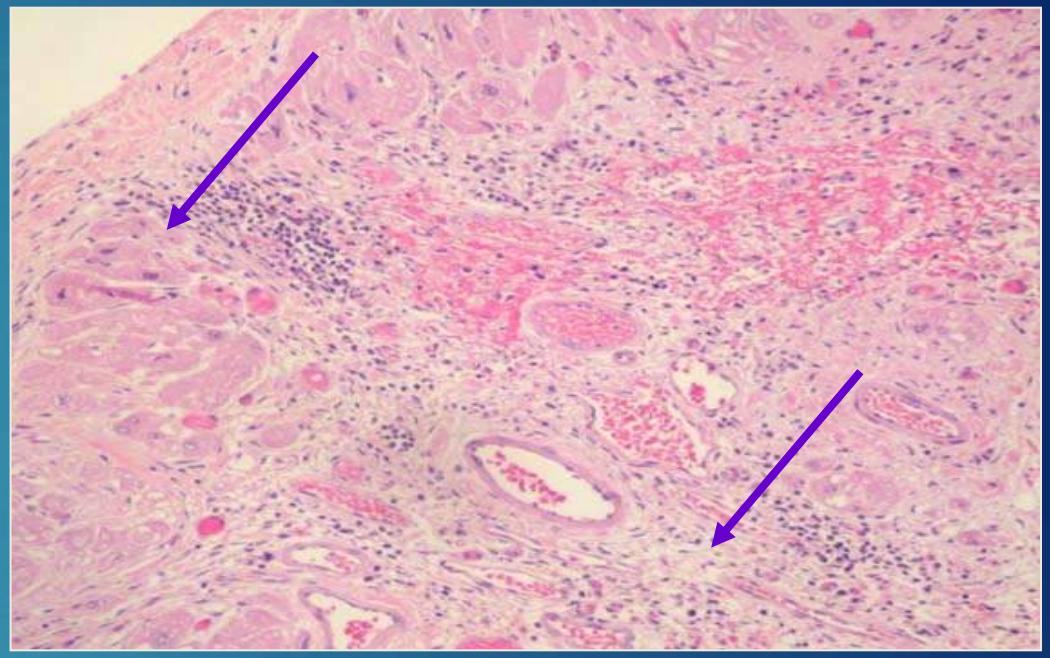
- Inflammation (≥14 lymphocytes / mm^3)
- Myocellular necrosis +/- degeneration

Chronic Myocarditis

- Inflammation without necrosis or degeneration

Dilated Cardiomyopathy

- Myocyte hypertrophy, myofibrillar loss
- Interstitial fibrosis; minimal inflammation

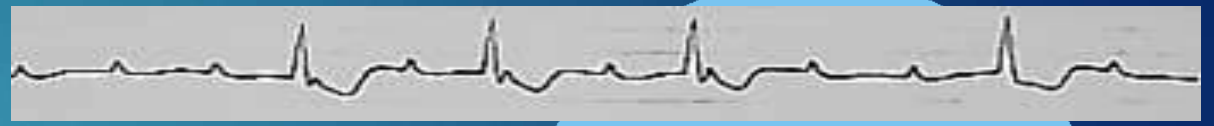


Myocarditis: Presentation in children

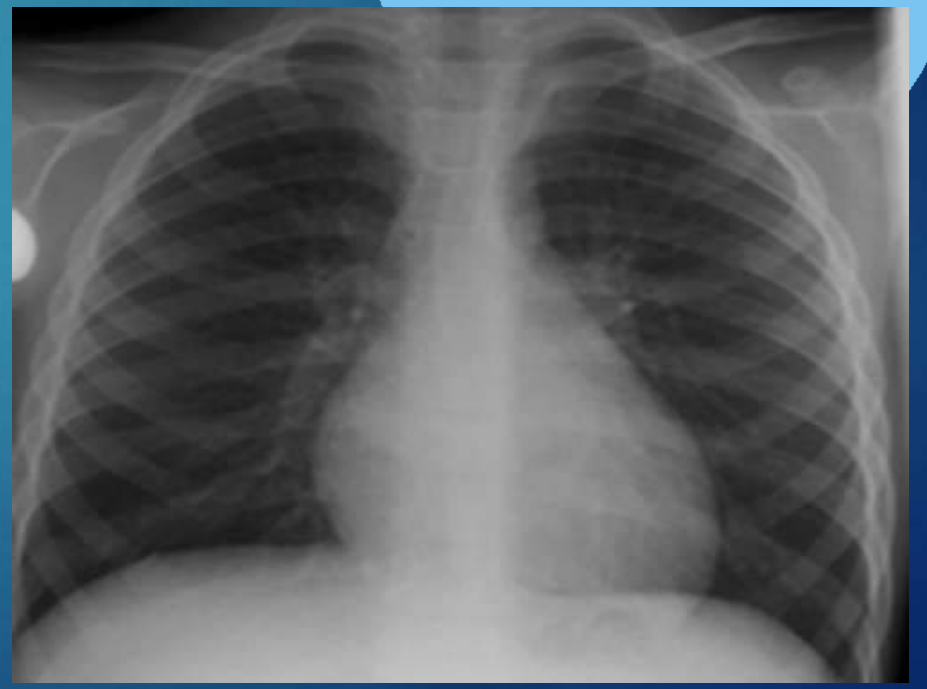
More than half present within the first year of life
Sudden death (acute fulminant myocarditis)
Acute heart failure with cardiogenic shock
Recent onset congestive cardiac failure
Respiratory distress, cough and cold
Arrhythmias

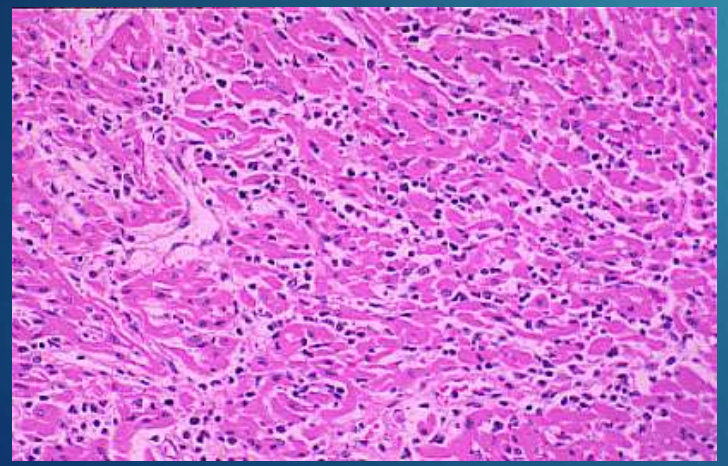
Acute Myocarditis: Clinical Presentation

nausea, vomiting
chest pain
T wave flattening on EKG
arrhythmia



?Signs of CCF





Outcome in fulminant versus acute (non-fulminant) myocarditis

McCarthey RE N Engl J Med 2000; 342:690

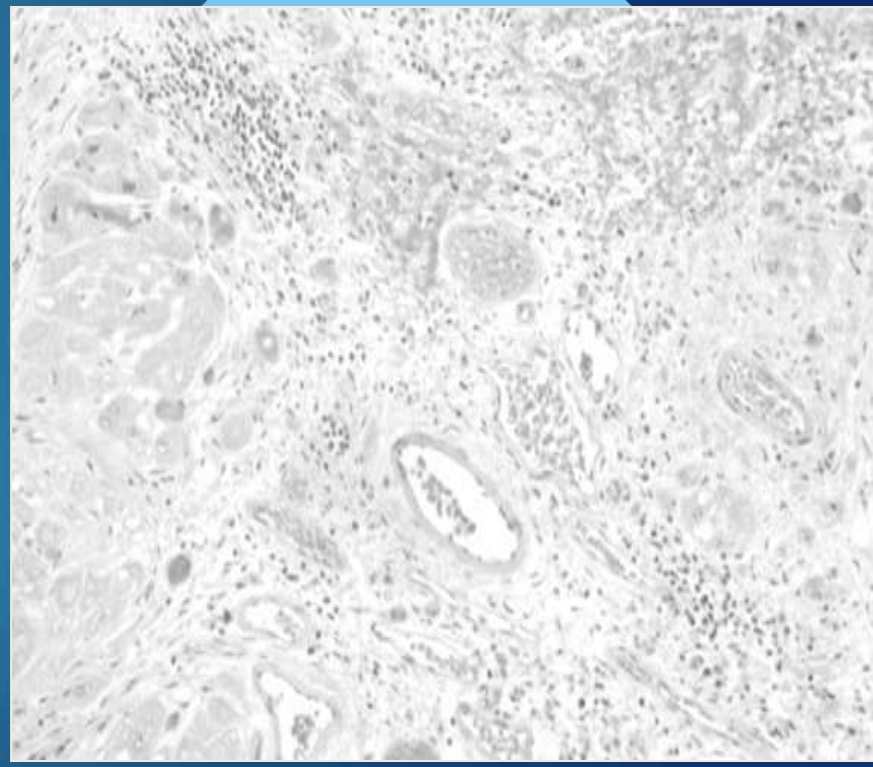
Patients diagnosed with fulminant myocarditis

- ≥5ug/kg/min of dopamine or dobutamine
- left ventricular assist device
- history of viral illness within 2 weeks
- fever
- 1-2 day history of heart failure symptoms

- Viruses –most common cause in children
- Persistent viral infection has been implicated in chronic myocarditis / DCM
- Historically enterovirus thought to be ‘key player’ (all ages)
- PCR amplification of viral genome – valuable tool
  - Tracheal aspirates
  - Myocardial biopsy

Role in children

- Fulminant' Vs 'Acute' presentations – less reliable distinction in children *therefore*
- Diagnostic & prognostic tool
- Histopathology, viral, and metabolic investigations
- Guide to acute therapy
  - Immune, antiviral



Acute Myocarditis: Treatment

- Supportive
  - Afterload reduction (vasodilators)
  - Inotropes
  - ECLS
- Disease specific
  - Immunosuppression

Acute Myocarditis: Treatment

Haemodynamic support: pharmacological

inotropes

milrinone
dopamine
epinephrine
dobutamine

dilators

nitroprusside
captopril

calcium channel blockers

amlodipine

beta blockers

metopropol

NIV in acute myocarditis/DCM

Low cardiac output

↓

Pulmonary oedema

↓

Reduced lung compliance

↓

Increased respiratory work

↓

Increased oxygen consumption

Acute myocarditis

Summary

- Complex disease with major immunological effects
- There is a wide spectrum of disease severity
- Differs in important respects from adult disease
- Immunotherapy remains unproven
- Outcomes are good with supportive therapies