GUIDELINES ON THE DIAGNOSIS AND MANAGEMENT OF PERICARDIAL DISEASES

ESC Task Force
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Full text guidelines: www.escardio.org
Pocket Guidelines: Available per request from the ESC
Guidelines on the Diagnosis and Management of Pericardial Diseases

MAIN TOPICS

- Acute pericarditis
- Pericardial effusion and cardiac tamponade
- Constrictive pericarditis
- Viral pericarditis
- Bacterial pericarditis
- Pericarditis in renal failure
- Autoreactive pericarditis and pericardial involvement in systemic autoimmune diseases
- The post-cardiac injury syndrome
- Postinfarction pericarditis
- Traumatic pericardial effusion
- Haemopericardium in aortic dissection
- Neoplastic pericarditis
- Pericardial diseases in pregnancy
- Drug- and toxin-related pericardial disease
• **Level of evidence A**: Multiple randomised clinical trials or meta-analyses.

• **Level of evidence B**: A single randomised trial or non-randomised studies.

• **Level of evidence C**: Consensus opinion of the experts.
Class I: evidence and/or general agreement that a given procedure or treatment is useful and effective.

Class II: conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: Weight of evidence/opinion is in favour of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.
ACUTE PERICARDITIS
ACUTE PERICARDITIS

ECHOCARDIOGRAPHY

TAMPONADE or PE >20 mm in diastole

Suspected purulent, TBC or neoplastic effusion

PERICARDIOCENTESIS

PERICARDIAL DRAINAGE (best with cardiac catheterisation)

SUBXIPHOID PERICARDIOTOMY AND DRAINAGE

FOLLOW-UP ECHOCARDIOGRAPHY

NO TAMPONADE PE 10-20 mm in diastole

INTRAPERICARDIAL THERAPY

PERICARDIOSCOPY AND PERICARDIAL/EPICARDIAL BIOPSY

Symptomatic management
Hospitalisation and exercise restriction
Pain management
- Ibuprofen, 300-800 mg tid or qid
- Colchicine, 0.5 mg bid

NO TAMPONADE PE <10 mm in diastole
### ACUTE PERICARDITIS

#### Diagnostic pathway and sequence of performance

<table>
<thead>
<tr>
<th>OBLIGATORY (class I, level of evidence B for all procedures):</th>
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</thead>
<tbody>
<tr>
<td><strong>Auscultation</strong></td>
</tr>
<tr>
<td><strong>ECG</strong></td>
</tr>
<tr>
<td><strong>Echocardiography</strong></td>
</tr>
<tr>
<td><strong>Blood analyses</strong></td>
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<tr>
<td><strong>Chest x-ray</strong></td>
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</tbody>
</table>

* Typical lead involvement: I, II, aVL, aVF, and V3-V6.  
* Cardiac troponin is detectable in 32.2-49%. An increase beyond 1.5 ng/ml is rare (7.6-22%).
**ACUTE PERICARDITIS**

Diagnostic pathway and sequence of performance  
*(level of evidence B for all procedures)*

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
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<tbody>
<tr>
<td><strong>MANDATORY IN TAMPONADE (class I)</strong>, OPTIONAL IN LARGE/RECURRENT EFFUSIONS OR IF PREVIOUS TESTS INCONCLUSIVE (class IIa) IN SMALL: EFFUSIONS (class IIb):**</td>
<td>Pericardiocentesis and drainage Pericardial fluid cytology, and cultures, PCRs and histochemistry for determination of infection or neoplasia</td>
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<tr>
<td><strong>OPTIONAL OR IF PREVIOUS TESTS INCONCLUSIVE (class IIa):</strong></td>
<td></td>
</tr>
<tr>
<td>CT</td>
<td>Effusions, peri-, and epicardium</td>
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<tr>
<td>MRI</td>
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</tr>
<tr>
<td>Pericardioscopy, pericardial biopsy</td>
<td>Establishing the specific aetiology</td>
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</tbody>
</table>
ACUTE PERICARDITIS

Symptomatic management

• Exercise restriction

• Hospitalisation to determine the aetiology and observe for tamponade as well as the effect of treatment.

• Pain management
ACUTE PERICARDITIS

Pain management

- NSAIDs are the mainstay (level of evidence B, class I).
- Ibuprofen is preferred (rare side-effects, favourable impact on the coronary flow, and the large dose range: 300-800 mg every 6-8 h)
- Aspirin 300-600 mg every 4-6 h
- Indomethacin should be avoided in elderly patients (flow reduction in the coronary arteries).
- Gastrointestinal protection must be provided.
**ACUTE / RECURRENT PERICARDITIS**

Treatment and prevention of recurrences

- Colchicine (0.5 mg bid) added to an NSAID or as monotherapy is well tolerated with fewer side effects than NSAIDs (level of evidence B, class IIa).

- Percutaneous balloon pericardiotomy in recurrences resistant to medical treatment (level of evidence B, class IIb).
Treatment and prevention of recurrences

- **Corticosteroids**
  - only in patients with poor general condition or in frequent crises (level of evidence C, class IIa).
  - The recommended regimen is: prednisone 1-1.5 mg/kg, for at least one month.
  - If patients do not respond adequately, azathioprine (75-100 mg/day) or cyclophosphamide can be added.

- **Pericardiectomy** - only in frequent and highly symptomatic recurrences resistant to medical treatment (level of evidence B, class IIa).
CARDIAC TAMPONADE
CARDIAC TAMPONADE

Clinical presentation

- Elevated systemic venous pressure
  - Jugular venous distension is less notable in hypovolemic patients or in “surgical tamponade”.
  - An inspiratory increase or lack of fall of the pressure in the neck veins (Kussmaul’s sign) indicates constriction
- Hypotension
  - Occasional pts are hypertensive especially if they have pre-existing hypertension
- Pulsus paradoxus
  - Absent in tamponade complicating atrial septal defect and significant aortic regurgitation.
- Tachycardia
  - HR may be < 100 b/min in hypothyroidism and in uremic patients
- Dyspnoea or tachypnoea with clear lungs
CARDIAC TAMponade

Precipitating factors

- **Drugs**
  - Cyclosporine
  - Anticoagulants
  - Thrombolytics, etc.

- **Injury**
  - Recent cardiac surgery
  - Indwelling instrumentation
  - Blunt chest trauma

- **Malignancies**

- **Connective tissue disease**

- **Renal failure**

- **Septicaemia**
CARDIAC TAMPOONADE

Electrocardiogram

- Can be normal or
- Non-specifically changed (ST-T wave)
- Electrical alternans (QRS, rarely T)
- Bradycardia (end-stage)
- Electromechanical dissociation (agonal phase)
CARDIAC TAMPONADE

Chest X-ray

- Enlarged cardiac silhouette with clear lungs
CARDIAC TAMPOONADE

M-mode/2D echocardiogram

- Diastolic collapse of the anterior RV free wall
  - Can be absent in RV hypertrophy or RV infarction.
CARDIAC TAMPOONADE

M-mode/2D echocardiogram

- RA collapse
- LA and very rarely LV collapse
- Increased LV diastolic wall thickness "pseudohypertrophy"
- VCI dilatation (no collapse in inspirium)
- "Swinging heart"

CARDIAC TAMponade

Doppler echocardiography

- Tricuspid flow increases and mitral flow decreases during inspiration (reverse in expiration)
- Systolic and diastolic flows are reduced in systemic veins in expirium and reverse flow with atrial contraction is increased.

M-mode colour Doppler

- Large respiratory fluctuations in mitral/tricuspid flows

CARDIAC TAMпонАDе

Cardiac catheterisation

- Confirmation and quantification of the haemodynamic compromise.
- Documenting haemodynamic improvement after pericardiocentesis.
- Detection of the coexisting haemodynamic abnormalities (LV failure, constriction, pulmonary hypertension).
- Detection of associated coronary artery disease or cardiomyopathy.

Murgo 1982
CARDIAC TAMPOONADE

Cardiac catheterisation - haemodynamics

• Confirmation and quantification of the haemodynamic compromise:
  – RA pressure is elevated (preserved systolic x descent and absent or diminished diastolic y descent).
  – Intrapericardial pressure is also elevated and virtually identical to RA pressure (both pressures fall in inspiration).
  – RV mid-diastolic pressure elevated and equal to the RA and pericardial pressures (no dip-and-plateau configuration).
  – PA diastolic pressure is slightly elevated.
  – LV systolic and aortic pressures may be normal or reduced.
CARDIAC TAMponade

RV/LV angiography
- Atrial collapse
- Small hyperactive ventricular chambers.

Coronary angiography
- Coronary compression in diastole.

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CARDIAC TAMPONADE

Computer tomography

- No visualisation of subepicardial fat along both ventricles, which show tube-like configuration and anteriorly drawn atrias

Chiles et al. Radiographics 2001
PERICARDIOCENTESIS
Classes of recommendations for pericardiocentesis

Class I

- Cardiac tamponade
- Effusions >20 mm in echocardiography (diastole)
- Suspected purulent or tuberculous pericardial effusion

Class IIa

- Effusions 10-20 mm in echocardiography in diastole for diagnostic purposes other than purulent pericarditis or tuberculosis (pericardial fluid and tissue analyses, pericardioscopy, and epicardial/pericardial biopsy)
- Suspected neoplastic pericardial effusion
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PERICARDIAL EFFUSION / TAMPOODADE

Classes of recommendations for pericardiocentesis

Class IIb

• Effusions <10 mm in echocardiography in diastole for diagnostic purposes other than purulent, neoplastic or tuberculous pericarditis

Contraindications (Class III)

• Aortic dissection
• Relative contraindications include uncorrected coagulopathy, anticoagulant therapy, thrombocytopenia <50000/mm3, small, posterior, and loculated effusions.
• If the diagnosis can be made otherwise or the effusions are small and resolving under anti-inflammatory treatment.
Pericardiocentesis guided by fluoroscopy

- Current and reliable echocardiography before the procedure
- Cardiac catheterisation laboratory.
- Local anaesthesia.
- Subxiphoid approach (long needle directed towards the left shoulder at a 30° angle to the skin).
Pericardiocentesis guided by echocardiography

- Bedside, intensive care unit, cardiac cath. lab., or operating theatre.
- Echocardiography should identify the shortest route where the pericardium can be entered intercostally (usually in the sixth or seventh rib space in the anterior axillary line).
- Intercostal arteries should be avoided.
PERICARDIAL EFFUSION / TAMPONADE

How to perform pericardiocentesis

- Strict aseptic conditions, ECG, and blood pressure monitoring have to be provided.

- Direct ECG monitoring from the puncturing needle is not an adequate safeguard.

- Right-heart catheterisation can be performed simultaneously, allowing exclusion of constriction.
PERICARDIAL EFFUSION / TAMPONADE

How to perform pericardiocentesis

- The needle approaches pericardium slowly.
- Steady manual aspiration is essential.
- Stop the needle as soon as the effusion is aspirated.
- Exchange for soft J-tip guidewire and after dilatation for a multi-holed pigtail catheter.
• Drain the fluid in <1 l steps to avoid the acute right-ventricular dilatation.

• Perform prolonged pericardial drainage (several days) until <25 ml/day.
PERICARDIAL EFFUSION ANALYSES

Should be ordered according to the clinical presentation

CLASS I

- **Suspected malignant effusion**: CYTOLOGY.

- **Suspected tuberculous effusion**: ACID-FAST BACILLI STAINING, mycobacterium CULTURE (preferably with radiometric growth detection e.g., BACTEC-460), adenosine deaminase, IFN-gamma, pericardial lysozyme, PCR analyses

- **Suspected bacterial infection**: at least three cultures of pericardial fluid for aerobes and anaerobes as well as three blood cultures. Positive cultures should be followed by sensitivity tests for antibiotics.
PERICARDIAL EFFUSION ANALYSES

Should be ordered according to the clinical presentation

CLASS IIa

- **Viral vs. autoreactive pericarditis:** PCR analyses for cardiotropic viruses.

- **Suspected neoplastic pericarditis:** Tumour markers (CEA, AFP, CA 125, CA 72-4, CA 15-3, CA 19-9, CD-30, CD-25...).

- **Benign reactive mesothelial cells vs. adenocarcinoma:** Combination of epithelial membrane antigen, CEA, and vimentin immunocytochemical staining.
PERICARDIAL EFFUSION ANALYSES
Should be ordered according to the clinical presentation
CLASS IIb

- **Exudate vs. transudate:**
  - Pericardial fluid specific gravity (>1015)
  - Protein level (>3.0 g/dl; fluid/serum ratio >0.5)
  - LDH (>200mg/dL; serum/fluid >0.6), and
  - Glucose (exudates vs. transudates = 77.9±41.9 vs. 96.1±50.7 mg/dl)
CONSTRUCTIVE PERICARDITIS
Clinical presentation

- Severe chronic systemic venous congestion
- Jugular venous distension
- Hypotension
- Low pulse pressure
- Abdominal distension
- Oedema
- Muscle wasting
Guidelines on the Diagnosis and Management of Pericardial Diseases

CONstrictive Pericarditis

Thickening of the pericardium

- Not always equal to constrictive physiology
- May also be absent in proven constriction (18% of 143 surgically proven cases, Talreja et al. Circulation 2003).

Talreja et al. Circulation 2003
CONSTRUCTIVE PERICARDITIS

Transient forms

Haley et al. J Am Coll Cardiol 2004
Electrocardiogram

Can be normal or:

- Low QRS voltage
- Generalized T-wave inversion/flattening
- LA abnormalities
- Atrial fibrillation
- AV block
- Intraventricular conduction defects
- Pseudoinfarction pattern (rarely)
CONSTRUCTIVE PERICARDITIS

Chest X-ray

- Pericardial calcifications
- Pleural effusions

Constrictive Pericarditis

- Pericardial thickening and calcifications
- Indirect signs of constriction

M-mode/2D echocardiogram

Oxantenko et al, J Clin Gastroenterol 2002
Indirect echo signs of constriction

- RA & LA enlargement with normal ventricles, and systolic function.
- Early pathological outward and inward movement of the interventricular septum ("dip-plateau phenomenon")
- Flattering waves at the LV posterior wall
- LV diameter is not increasing after the early rapid filling phase.
- VCI and the hepatic veins are dilated with restricted respiratory fluctuations.
• Restricted filling of both ventricles with respiratory variation (>25% over the AV-valves).
  – In mixed constriction-restriction and increased atrial pressures respiratory changes are <25%.
  – In atrial fibrillation flow velocity pattern is inconclusive, but hepatic diastolic vein flow reversal in expirium is observed.
• Provocation test with head-up tilting or sitting position with decrease of preload may unmask the constrictive pericarditis.
“Dip and plateau” or “square route” sign in the RV and/or LV pressure curve.

- Equalisation of LV/RV end-diastolic pressures.
- In occult constriction rapid infusion of 1-2 l of normal saline may reveal the diagnosis.
Guidelines on the Diagnosis and Management of Pericardial Diseases

CONSTRICTIVE PERICARDITIS

RV/LV angiography

- The reduction of RV & LV size and increase of RA & LA size
- Dip-plateau - rapid early filling with stop of further enlargement in diastole

Coronary angiography

- Indicated in all patients over 35 years and in pts with a history of mediastinal irradiation, regardless of the age.
CONSTRUCTIVE PERICARDITIS

CT / MRI

- Thickened and/or calcified pericardium
- Tube-like configuration of one or both ventricles
- Enlargement of one or both atria
- Narrowing of one or both atrio-ventricular grooves
- Congestion of the caval veins

Guidelines on the Diagnosis and Management of Pericardial Diseases

Courtesy of R. Maksimović and T. Dill
### Guidelines on the Diagnosis and Management of Pericardial Diseases

**CONSTRICTIVE PERICARDITIS VS. RESTRICTIVE CARDIOMYOPATHY**

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<thead>
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<th>RESTRICTIVE CARDIOMYOPATHY</th>
<th>CONSTRUCTIVE PERICARDITIS</th>
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<tbody>
<tr>
<td><strong>Physical findings</strong></td>
<td>Kussmaul’s sign ±, apical impulse +++</td>
<td>Kussmaul’s sign +, apical impulse -</td>
</tr>
<tr>
<td></td>
<td>S₃ (advanced), S₄ (early disease), regurgitant murmurs ++</td>
<td>pericardial knock+, regurgitant murmurs -</td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>Low voltage, pseudoinfarction, left-axis deviation, AF, conduction disturbances</td>
<td>Low voltage (&lt;50%)</td>
</tr>
<tr>
<td><strong>Chest radiography</strong></td>
<td>No calcifications</td>
<td>Calcifications may be present</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(low diagnostic accuracy)</td>
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</tbody>
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## Guidelines on the Diagnosis and Management of Pericardial Diseases

**CONSTRICTIVE PERICARDITIS VS. RESTRICTIVE CARDIOMYOPATHY**

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<tr>
<td><strong>2D-Echocardiography</strong></td>
<td>Small LV cavity with large atria</td>
<td>Normal wall thickness</td>
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<tr>
<td></td>
<td>Increased wall thickness</td>
<td>Pericardial thickening, prominent early diastolic filling with abrupt displacement of IVS</td>
</tr>
<tr>
<td></td>
<td>sometimes present (especially thickened interatrial septum in amyloidosis)</td>
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<tr>
<td></td>
<td>Thickened valves and granular sparkling (amyloidosis)</td>
<td></td>
</tr>
<tr>
<td><strong>Tissue Doppler Echocardiography</strong></td>
<td>Peak early velocity of longitudinal expansion (peak Ea) of ≥8.0 cm/s (89% sensitivity and 100% specificity)</td>
<td>Negative</td>
</tr>
</tbody>
</table>
CONSTRUCTIVE PERICARDITIS VS. RESTRICTIVE CARDIOMYOPATHY

<table>
<thead>
<tr>
<th>DOPPLER STUDIES</th>
<th>RESTRICTIVE CARDIOMYOPATHY</th>
<th>CONSTRUCTIVE PERICARDITIS</th>
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<tr>
<td>Mitral inflow</td>
<td>No respiration variation of mitral inflow E wave velocity, IVRT E/A ratio &gt;2, short DT, diastolic regurgitation</td>
<td>INSPIRATION: decreased inflow E wave velocity, prolonged IVRT EXPIRATION: opposite changes, short DT, diastolic regurgitation</td>
</tr>
<tr>
<td>Pulmonary vein</td>
<td>Blunted S/D ratio (0.5), prominent and prolonged AR No respiration variation, D wave</td>
<td>S/D ratio = 1, INSPIRATION: decreased PV S and D waves EXPIRATION: opposite changes</td>
</tr>
<tr>
<td>Tricuspid inflow</td>
<td>Mild respiration variation of tricuspid inflow E wave velocity, E/A ratio &gt;2, TR peak velocity, no significant respiration change</td>
<td>INSPIRATION: increased tricuspid inflow E wave velocity, increased TR peak velocity, EXPIRATION: opposite</td>
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### DOPPLER STUDIES

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<tr>
<td><strong>Hepatic veins</strong></td>
<td>Blunted S/D ratio, increased inspiratory reversals</td>
<td>INSPIRATION: minimally increased HV, S and D EXPIRATION: opposite changes</td>
</tr>
<tr>
<td><strong>Inferior vena cava</strong></td>
<td>Plethoric</td>
<td>Plethoric</td>
</tr>
<tr>
<td><strong>Colour M-mode</strong></td>
<td>Slow flow propagation</td>
<td>Rapid flow propagation ($\geq 100$ cm/s)</td>
</tr>
<tr>
<td><strong>Mitral annular motion</strong></td>
<td>Low-velocity early filling ($&lt; 8$ cm/s)</td>
<td>High-velocity early filling ($\geq 8$ cm/s)</td>
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### Guidelines on the Diagnosis and Management of Pericardial Diseases

#### CONSTRICTIVE PERICARDITIS VS. RESTRICTIVE CARDIOMYOPATHY

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<tr>
<td>Cardiac catheterisation</td>
<td>Dip and plateau, LVEDP often &gt;5 mmHg, greater than RVEDP, but may be identical, RV systolic pressure &gt;50 mmHg, RVEDP &lt; 1/3 RVSP</td>
<td>Dip and plateau, LVEDP usually equal, INSPIRATION: Increase in RV systolic pressure, DECREASE in LV systolic pressure, with EXPIRATION, opposite changes</td>
</tr>
<tr>
<td>EMB</td>
<td>May reveal specific cause of restrictive cardiomyopathy</td>
<td>May be normal or show nonspecific hypertrophy or fibrosis</td>
</tr>
<tr>
<td>CT/MRI</td>
<td>Pericardium usually normal</td>
<td>Pericardium thickened or calcified.</td>
</tr>
</tbody>
</table>
ECHOCARDIOGRAPHY

If symptomatic for >2 years

CHRONIC PERICARDIAL EFFUSION

CONSTRICITIVE PERICARDITIS

EFFUSIVE-CONSTRICITIVE PERICARDITIS

RECURRENT PERICARDITIS

Symptomatic management
Hospitalisation and exercise restriction
Pain management
- Ibuprofen, 300-800 mg tid or qid
- Colchicine, 0.5 mg bid
- Prednisone 1-1.5 mg qd

CLINICAL SUSPICION FOR:

Congestive heart failure therapy

CARDIAC CATHETERIZATION

PERCUTANEOUS BALLOON PERICARDIOTOMY

PERICARDIECTOMY
CONSTRUCTIVE PERICARDITIS

Pericardiectomy

- The only treatment for permanent constriction.
  - Antero-lateral thoracotomy
  - Median sternotomy (faster access to the aorta and right atrium for extracorporeal circulation).

- Primary installation of cardiopulmonary bypass is not recommended (diffuse bleeding following systemic heparinisation).

- Areas of strong calcification or dense scaring may be left as islands to avoid major bleeding.
PERICARDECTOMY FOR CONSTRICTIVE PERICARDITIS

Major complications

• Acute perioperative cardiac insufficiency (should be treated by fluid substitution and catecholamines, high doses of digitalis, and intraaortic balloon pump in most severe cases).
• Ventricular wall rupture.
• Mortality (in properly selected cases 6-12%, but >40% in unselected patients).

Long term results

• If indication for surgery was established early, long-term survival after pericardectomy corresponds to that of the general population.
PERICARDIECTOMY FOR VARIOUS PATHOANATOMICAL FORMS OF CONSTRICTION

- Exclusion of patients with extensive myocardial fibrosis and/or atrophy significantly reduces the mortality rate.
VIRAL PERICARDITIS
VIRAL PERICARDITIS

Diagnosis

- Not possible without the evaluation of pericardial effusion and/or pericardial/epicardial tissue, preferably by PCR or in-situ hybridisation (level of evidence B, class IIa).

- A four-fold rise in serum antibody levels (two samples within 3-4 weeks) is suggestive but not diagnostic for viral pericarditis (level of evidence B, class IIb).
VIRAL PERICARDITIS

Management

- In most cases the disease is self-limiting and no specific treatment is necessary.
- Symptomatic treatment for chest pain, and eventual rhythm disorders and congestive heart failure is indicated.
In patients with chronic or recurrent symptomatic pericardial effusion and confirmed viral infection, the following specific treatment is under investigation:

- **CMV pericarditis**: hyperimmunoglobulin – one daily 4ml/kg on day 0, 4, and 8; 2 ml/kg on day 12 and 16;
- **Coxsackie B pericarditis**: Interferon alpha or beta 2.5 million IU/m² surface area s.c. 3 x per week;
- **Adenovirus and parvovirus B19 pericarditis**: immunoglobulin treatment: 10 g intravenously at day 1 and 3 for 6-8 hours.
Guidelines on the Diagnosis and Management of Pericardial Diseases

PERICARDITIS IN AIDS

Management

• Symptomatic treatment
• Pericardiocentesis in large effusions/tamponade
• Standard (prolonged) anti-tuberculous regimens for TBC pericarditis in AIDS
• Use of rifampicin is precluded (if pts are treated with protease inhibitors or non-nucleoside reverse transcriptase inhibitors).
• Corticoid therapy as an adjunct to tuberculostatic treatment is allowed (level of evidence A, class I).
BACTERIAL PERICARDITIS
Guidelines on the Diagnosis and Management of Pericardial Diseases

BACTERIAL PERICARDITIS

Diagnosis

- Pericardiocentesis must be promptly performed.
- Pericardial fluid should undergo Gram, acid-fast, and fungal staining, followed by cultures for aerobes, anaerobes, and M. tuberculosis (preferably with radiometric growth detection).
- Drug sensitivity testing is essential for treatment selection.
TUBERCULOUS PERICARDITIS

Diagnosis

- PCR analyses
- Adenosine deaminase >40 IU/L
- Interferon-gamma >200 pg/L
- Pericardial lysozyme >6.5 microg/dL

Cost-effective only if the pre-test probability is high (populations with high incidence of tuberculosis).
**Urgent pericardial drainage**

**Intravenous antibiotic therapy** (e.g. vancomycin 1 g bid, ceftriaxone 1-2 g bid, and ciprofloxacin 400 mg/day (MIC and MBC need to be considered))

**Irrigation with urokinase or streptokinase, using large catheters, may liquefy the purulent exudate**

**Open surgical drainage is preferable.**
TUBERCULOUS PERICARDITIS

Management

- Respiratory isolation in active laryngeal or lung TBC.
- The initial treatment:
  - Isoniazid 300 mg/day
  - Rifampicin 600 mg/day
  - Pyrazinamide 15-30 mg/kg/day
  - Ethambutol 15-25 mg/kg/day.
- Prednisone (1-2 mg/kg/day) may be given simultaneously with antituberculous therapy for 5-7 days and progressively reduced to discontinuation in 6-8 weeks.
- After two months most patients can be switched to two-drug regimen (isoniazid and rifampicin) for the total of 6 months.
Guidelines on the Diagnosis and Management of Pericardial Diseases

TUBERCULOUS PERICARDITIS

Pericardiectomy

- Recurrent effusions
- Constriction (continued elevation of central venous pressure after 4-6 weeks of antituberculous and corticosteroid therapy).

Courtesy of P. Petrović
PERICARDITIS IN RENAL FAILURE
PERICARDITIS IN RENAL FAILURE

Diagnosis

- Chest pain, pericardial friction rub and pericardial effusion in a patient with advanced renal failure.
- Patients on maintenance chronic haemodialysis or peritoneal dialysis can also be affected.
- Heart rate may remain slow (60–80 beats/min) during tamponade, despite fever and hypotension (uremic autonomic impairment).
- No ST/T elevations in ECG due to the lack of the myocardial inflammation.
PERICARDITIS IN RENAL FAILURE

Management

- Frequent (heparin-free) haemodialyses.
- Peritoneal dialysis (no heparinisation), may be therapeutic in pericarditis resistant to haemodialysis, or if heparin-free haemodialysis cannot be performed.
- NSAIDs and systemic corticosteroids have limited success when intensive dialysis is ineffective.
PERICARDITIS IN RENAL FAILURE

Management

- Cardiac tamponade and large chronic effusions resistant to dialysis must be treated with pericardiocentesis (level of evidence B, class IIa).

- Large, non-resolving symptomatic effusions may be treated with intrapericardial instillation of corticosteroids (triamcinolone hexacetonide 50 mg every 6 h for 2-3 days).

- Pericardiectomy is indicated only in refractory, severely symptomatic patients.
PERICARDITIS AND AUTOIMMUNITY
AUTOREACTIVE PERICARDITIS

Diagnosis I

- Pericardial effusion (PE) with >5000/mm³ lymphocytes and mononuclear cells (autoreactive lymphocytic), or antibodies against heart muscle tissue (autoreactive antibody-mediated).

- Inflammation in epicardial/endomyocardial biopsies by >14 cells/mm².

- Exclusion of active viral infection both in PE and endomyocardial/epimyocardial biopsies.
  - No virus isolation.
  - No IgM-titer against cardiotropic viruses in PE.
  - Negative PCRs for major cardiotropic viruses.

B. Maisch et al. Eur Heart J 2002
AUTOREACTIVE PERICARDITIS

Diagnosis II

- TBC, B. burgdorferi, C. pneumoniae, and other bacterial infection excluded by PCR and/or cultures
- Neoplastic infiltration absent in pericardial effusion and biopsy samples
- Exclusion of systemic, metabolic disorders, and renal failure
Guidelines on the Diagnosis and Management of Pericardial Diseases

AUTOREACTIVE PERICARDITIS AND PERICARDIAL INVOLVEMENT IN SYSTEMIC AUTOIMMUNE DISEASES

Management

- Intrapericardial treatment with triamcinolone plus colchicine per os 0.5 mg bid for six months is highly efficient with rare side effects (level of evidence B, class IIa).

- In systemic autoimmune diseases intensified treatment of the underlying disease and symptomatic management are indicated (evidence level B, class I).

- For tapering of prednisone, ibuprofen or colchicine should be introduced early.
POSTPERICARDIOTOMY SYNDROME

Diagnosis

- Chest pain
- Pericardial friction rub
- ECG changes
- PE within days to months after cardiac, pericardial injury or both.

Courtesy of P. Cocco and G. Thiene
POSTPERICARDIOTOMY SYNDROME

Management

- Symptomatic treatment as in acute pericarditis
- In refractory forms long term (3-6 months) oral corticoids or preferably intrapericardial instillation of triamcinolone (300 mg/m²)
- Redo surgery or pericardectomy is rarely needed.
- Primary prevention with short-term perioperative steroid treatment or colchicine is under investigation.
- Warfarin administration in patients with early postoperative PE imposes greatest risk.
POSTINFARCTION PERICARDITIS
(Pericarditis epistenocardica and Dressler’s syndrome)

Diagnosis

- Detection of PE after acute myocardial infarction
- ECG changes are often overshadowed by myocardial infarction changes.
- Postinfarction PE >10 mm is most frequently associated with haemopericardium, and two thirds of these pts may develop tamponade/free wall cardiac rupture.
POSTINFARCTION PERICARDITIS
(Pericarditis epistenocardica and Dressler’s syndrome)
Management I

- **Hospitalisation** to observe for tamponade, differential diagnosis, and adjustments of treatment is needed.

- **Ibuprofen**, which increases coronary flow, is the agent of choice.

- **Aspirin**, up to 650 mg every 4 hours for 2 to 5 days has also been successfully applied (other NSAIDs risk thinning the infarction zone).
Corticosteroid therapy should be used for refractory symptoms only (potential delay in myocardial infarction healing).

In cardiac rupture, urgent surgical treatment is life saving.

If the immediate surgery is not possible pericardiocentesis and intrapericardial fibrin-glue instillation could be an alternative.
PERICARDIAL EFFUSION IN TRAUMA AND AORTIC DISSECTION
TRAUMATIC PERICARDIAL EFFUSION

Management

- Urgent echocardiography, if available TEE
- Rescue pericardiocentesis
- Autotransfusion
- Urgent thoracotomy and surgical repair
Guidelines on the Diagnosis and Management of Pericardial Diseases

HAEMOPERICARDIUM IN AORTIC DISSECTION

Diagnosis

- Echocardiography (both TTE and TEE)
- CT or MRI in complex cases
- Angiography (only in stable patients)
HAEMOPERICARDIUM IN AORTIC DISSECTION

Management

- Pericardiocentesis is contraindicated (risk of intensified bleeding and extension of the dissection).

- Surgery should be performed immediately (evidence level B, class I).
NEOPLASTIC PERICARDITIS
NEOPLASTIC PERICARDITIS

Diagnosis

- Confirmation of the malignant infiltration within the pericardium (cytology, histology, tumour markers).
- In up to 2/3 of pts with documented malignancy PE is caused by non-malignant diseases, e.g. radiation pericarditis, or opportunistic infections.

B. Maisch et al. Eur Heart J 2002
**NEOPLASTIC PERICARDITIS**

**Management I**

- Systemic antineoplastic therapy whenever possible (prevents recurrences in up to 67% of cases)

- Pericardiocentesis to relieve symptoms and establish diagnosis (level of evidence B, class IIa)

- Intrapericardial instillation of a cytostatic/sclerosing agent (level of evidence B, class IIa).
  - Cisplatin (single instillation of 30 mg/m2) is preferred for pericardial metastases of the lung cancer and
  - Intrapericardial instillation of thiotepa (15 mg on days 1, 3, and 5) for breast cancer.
NEOPLASTIC PERICARDITIS

Management II

- Prolonged pericardial drainage is recommended, in all pts with large effusions because of the high recurrence rates (40-70%)(level of evidence B, class I).
- In resistant cases percutaneous balloon pericardiotomy or rarely pericardectomy may be indicated.
- Radiation therapy is very effective in controlling malignant effusion due to radiosensitive tumours (e.g. lymphomas and leukemias), may however, cause myocarditis and pericarditis by itself.
PERICARDIAL DISEASES IN PREGNANCY
PERICARDIAL DISEASES IN PREGNANCY

Diagnosis

- Many pregnant women develop a minimal to moderate clinically silent hydropericardium by the third trimester. Cardiac compression is rare.

- ECG changes of acute pericarditis in pregnancy should be distinguished from the slight ST-segment depressions and T-wave changes seen in normal pregnancy.

- Occult constriction becomes manifest in pregnancy due to the increased blood volume.
• Most pericardial disorders are managed as in nonpregnant pts.

• Caution is necessary while high-dose aspirin may prematurely close the ductus arteriosus.

• Colchicine is contraindicated in pregnancy.

• Pericardiotomy and pericardiectomy can be safely performed if necessary and do not impose a risk for subsequent pregnancies.
• Foetal pericardial fluid can be detected by echocardiography after 20 weeks' gestation (normally $\leq 2$ mm).

• More fluid should raise questions of hydrops foetalis, Rh disease, hypoalbuminemia, and immunopathy or maternally transmitted mycoplasmal or other infections, and neoplasia.
DRUG- AND TOXIN-RELATED PERICARDIAL DISEASE
A. Drug-induced lupus erythematosus
- Procainamide
- Tocainide
- Hydralazine
- Methyldopa
- Mesalazine
- Reserpine
- Isoniazid
- Hydantoins

B. Hypersensitivity
- Penicillins
- Tryptophan
- Cromolyn sodium

C. Idiosyncratic reaction or hypersensitivity
- Methysergide
- Minoxidil
- Practolol
- Bromocriptine
### C. Idiosyncratic reaction or hypersensitivity

- Psicofuranine
- Polymer fume inhalation (Teflon)
- Cytarabine
- Phenylbutazone
- Amiodarone
- Streptokinase
- p-Aminosalicylic acid
- Thiazides
- Streptomycin
- Thiouracils
- 5-Fluorouracil
- Sulfa drugs
- Cyclophosphamide
- Cyclosporine
- Mesalazine
- Vaccines (Smallpox, Yellow fever)
- GM-CSF
DRUG- AND TOXIN-RELATED PERICARDIAL DISEASE (continued)

D. Anthracyclines
- Doxorubicin
- Daunorubicin

E. Serum sickness
- Foreign antisera (e.g., antitetanus)
- Blood products

F. Venom
- Scorpion fish sting

G. Foreign-substance reactions
- Talc (Mg silicate)
- Silicones
- Tetracycline/other sclerosants
- Asbestos
- Iron in β-thalassemia

H. Haemopericardium
- Anticoagulants
- Thrombolytic agents
DRUG- AND TOXIN-RELATED PERICARDIAL DISEASE (continued)

I. Polymer fume fever – Inhalation of the burning fumes of polytetrafluoroethylene (Teflon)