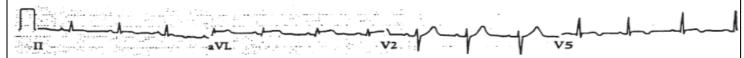
## PERICARDIAL DISEASE

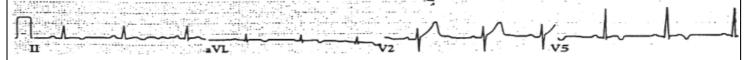
		ANDIAL DIJLAJL	
Table 1. Tests and Treatments for Various Causes of Acute Pericarditis.*			
Condition	Estimated Incidence†	<b>Clinical Indications and Tests</b>	<b>Usual Treatment</b>
Idiopathic	85-90%		Aspirin, NSAIDs
Infectious			
Viral	1–2%	Acute and convalescent viral titers, viral cultures, serologic test for HIV	Aspirin, NSAIDs
Bacterial	1–2%	Fever, elevated white-cell count; exam- ination of pericardial fluid	Antibiotics, drainage of pericardial fluid
Tuberculous	4%	Chest radiography, tuberculin skin test, histologic examination, cul- tures, and measurement of adeno- sine deaminase level in pericardial fluid and tissue	Multidrug antitubercu- lous therapy and prednisone
Acute myocardial infarction	NA (occurs in 5–10% of patients with myocar- dial infarction)	Electrocardiography, serum troponin or creatine kinase, echocardio- gram	Aspirin (avoid NSAIDs)
Aortic dissection	Rare (<1%)	Magnetic resonance imaging, com- puted tomography, transesopha- geal echocardiography	Urgent surgery
Trauma	NA	Clinical history	NSAIDs (avoid aspirin)
Neoplasm	7%	Constitutional symptoms, lymphade- nopathy; chest radiography, examination of pericardial fluid	NSAIDs, glucocorti- coids (by intraperi- cardial instillation)
Chest-wall irradiation	Rare (<1%)	Clinical history	NSAIDs
Uremia	NA (occurs in approxi- mately 5% of patients with chronic renal dis- ease before initiation of dialysis and 13% after initiation of dialysis)	Serum blood urea nitrogen and creati- nine levels	Initiate or intensify dialysis
Cardiotomy or thoracic surgery	Rare (<1%)	Clinical history, evidence of poly- serositis; chest radiography, erythrocyte sedimentation rate	Aspirin, NSAIDs
Autoimmune or inflam- matory disease	3–5%	Rheumatoid factor, complement levels, antinuclear antibodies	Aspirin, NSAIDs, glucocorticoids
Adverse drug reaction‡	Rare (<1%)	Clinical history; eosinophil count	Discontinue drug; aspirin, NSAIDs



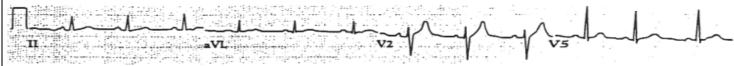
**Stage 1** begins with the onset of chest pain, and lasts several days. Note the concave upward ST elevations seen in the precordial and limb leads. The T waves remain upright in those leads with ST-segment elevation.



Stage 2. The ST segments return to the baseline, T waves become flat, and the PR segment may become depressed.



Stage 3. T-wave inversion may be seen. This may persist for weeks, or even indefinitely.



Stage 4. Normalization.

### **ACUTE PERICARDITIS**

## Physical findings and w/u:

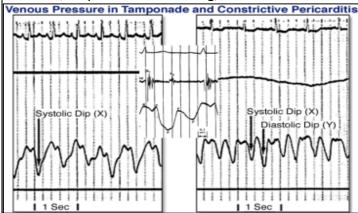
- **1.PE**: friction rub w/ 3 components: A&V-syst,V-diast
- 2.EKG: Diffuse concave ST 1 (except V1 & aVR)
- 3.CXR: CM if sig. per. eff. present
- 4.Lab: ↑WBC & ESR; +/- ↑Trop & CKMB
  - a. Consider: Viral serology/HIV testing; B/C;
  - b. ASO titer; BUN/Cr; ANA; RF; heterophile Ab
- 5.Echo: 60% have per. eff. To R/O WMA

#### **Management:**

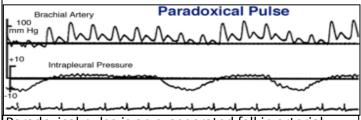
- 1. Hospitalize based on etiology
- 2.Outpt. if uncomplicated
- 3.NSAIDs; w/5-7 d taper
- 4.If CP persists after 2-3 days: 7-10 d steroid taper
- Avoid anticoagulants
- 6.If relapsing: Colchicine PRN & to prevent recurrence
- 7. Pericardiectomy not recommended

## **CONSTRICTIVE PERICARDITIS**

- **1.Anat**.: Thickened, fibrotic & adherent pericardium.
- **2.Phys.**: Restrains diastolic expansion.
- **3.S/Sx**: SOB, fatigue, JVD/edema, hepatomegaly/ascites.
  - a. No pulm congestion. Pericardial knock
  - b. Kussmaul sign († systemic venous pressure w/ insp)
- 4.Etiol: Surgery/viral/XRT/RA in US. TB in Africa
- **5.Dx**: Rapid increase in early diastolic pressure (filling):
  - a. Dip/plateau; sign). CXR- pericardial calcification
  - b. MRI/CT: pericardial thickening/calcification



In cardiac tamponade (left), systolic (X) descent is prominent, and the diastolic (Y) descent is small or absent. The reduction in the Y descent reflects impairment of filling from the beginning of diastole in cardiac tamponade, contrasting with constrictive pericarditis, where impairment is when the ventricle reaches a critical volume at some point in diastole. In constriction (right), X & Y are both prominent, creating a waveform with two dips per cycle.



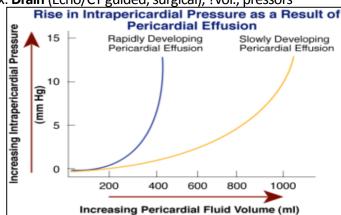
Paradoxical pulse is an exaggerated fall in arterial pressure with inspiration. Systolic pressure falls more than diastolic; the fall in pulse pressure reflects a fall in stroke volume. This occurs in the first beat after inspiration onset (indicated by intrapleural pressure fall)

#### PERICARDIAL EFFUSION

- 1.NI: 50cc. CM on CXR: with ≥ 250cc 2.Echo: to dx, quantify & r/o tamponade
- **3.CT/MR**: to assess pericardium, r/o masses
- **4.EKG**: Low volt; elect. alternans
- **5.Rx**: Pericardiocentesis w/ bx for dx.
- 6.R/O hypothyroidism
- 7.Idiopathic pericardial effusion > 3 mo → drain
- 8. Avoid anticoagulants

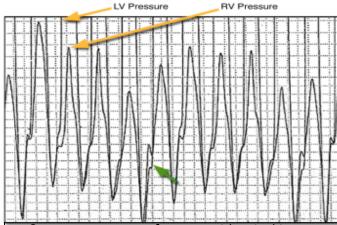
# **CARDIAC TAMPONADE**

- 1. Rapidity of fluid accumulation, not absolute size.
- 2. May be circumferential or loculated
- **3.S/Sx**: Dyspnea, ↓BP, ↑ HR, JVD, ↓ heart sounds
- 4. Pulsus Paradoxus:
  - a. > 10mmHg drop in SBP during normal inspiration.
  - b. May be blunted w/ sig AI, ASD, PH, LVH & RVH
- 5.EKG: Low volt./electrical alternans
- 6.CXR: +/- CM if Acute v.s. sub-acute
- 7.Cath: Diastolic pressure equalization (15-30mmHg)
- 8.Rx: Drain (Echo/CT guided, surgical), ?vol., pressors



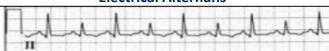
Pericardial pressure rises more steeply in pericardial effusions that develop rapidly than in effusions that develop slowly

# **Diastolic Pressure in Constrictive Pericarditis**



RV & LV pressure waveforms are identical in diastole. During respiratory variation in diastolic pressure, pressures often separate by up to about 5 mm Hg (see top arrows)

#### **Electrical Alternans**



Electrical alternans is caused by the heart swinging from one position to another within the pericardial sac on alternate beats. It disappears when the fluid is removed