Case Presentation

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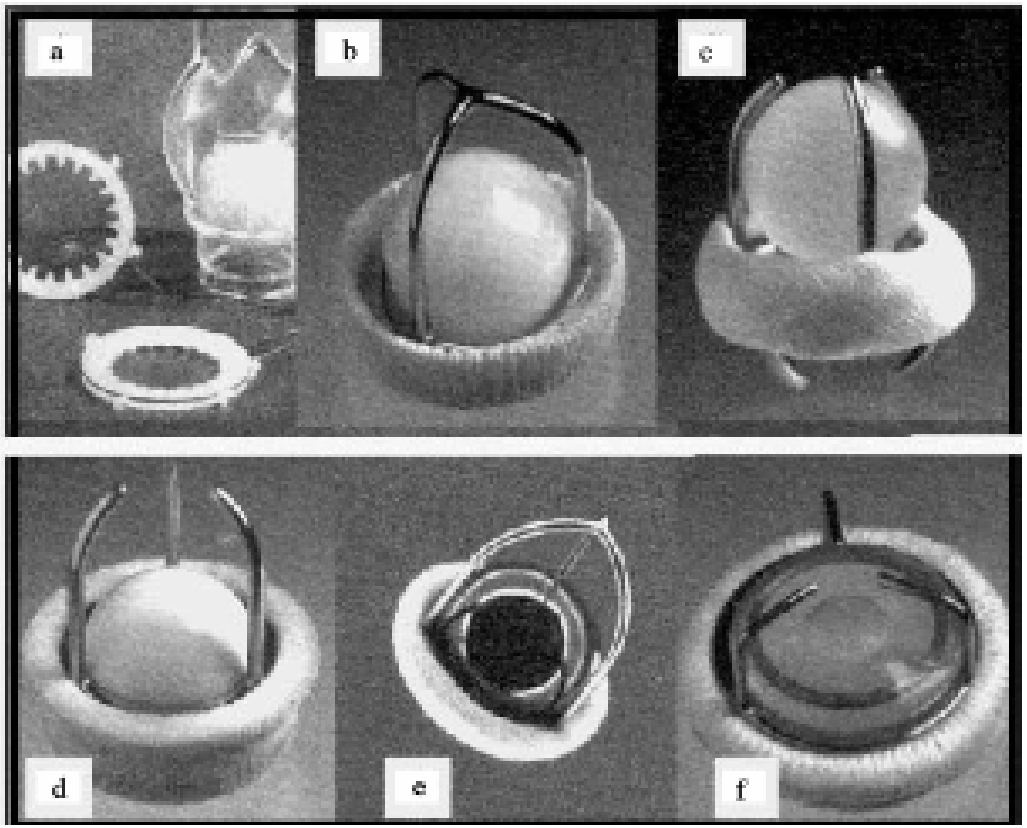
Case #1

- 40 yo WM with hx Aortic Valve replacement for endocarditis
- Hx IVDU
- + Pseudomonas Blood Cx

Caged Ball Valves

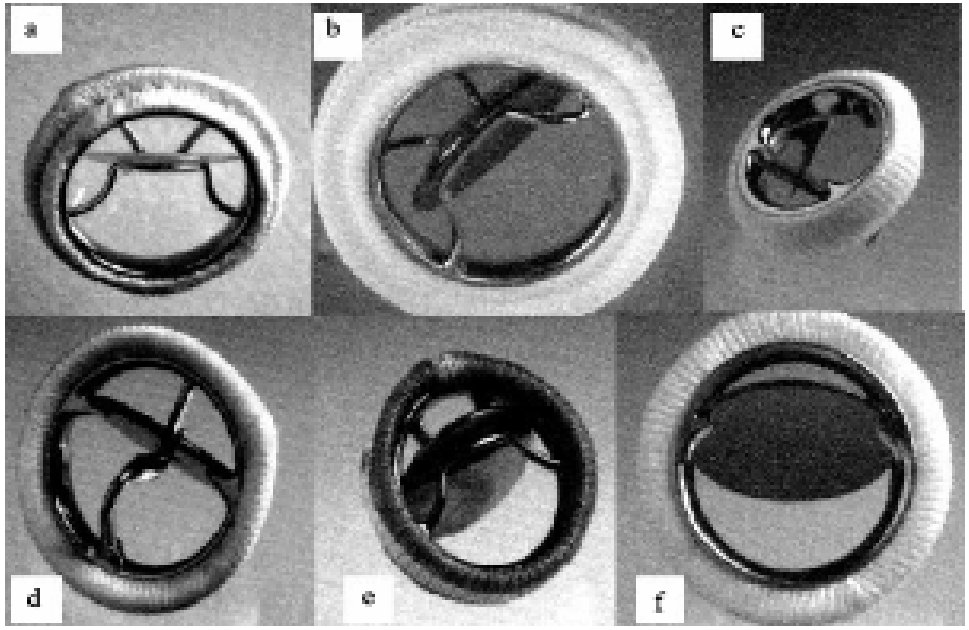
- A – Hufnagel-Lucite
- B – Starr-Edwards
- C – Smeloff-Cutter
- D – McGovern-Cronie
- E – DeBakey-Surgitool
- F – Cross - Jones



http://www.ias.ac.in/sadhana/Pdf2003JunAug/Pe1107.pdf

Tilting Disc Valves

- A – Bjork-Shiley Delrin
- B – Bjork-Shiley standard
- C – Lillihei-Kastor
- D – Medtronic-Hall
- E – Zorin
- F - Omniscience



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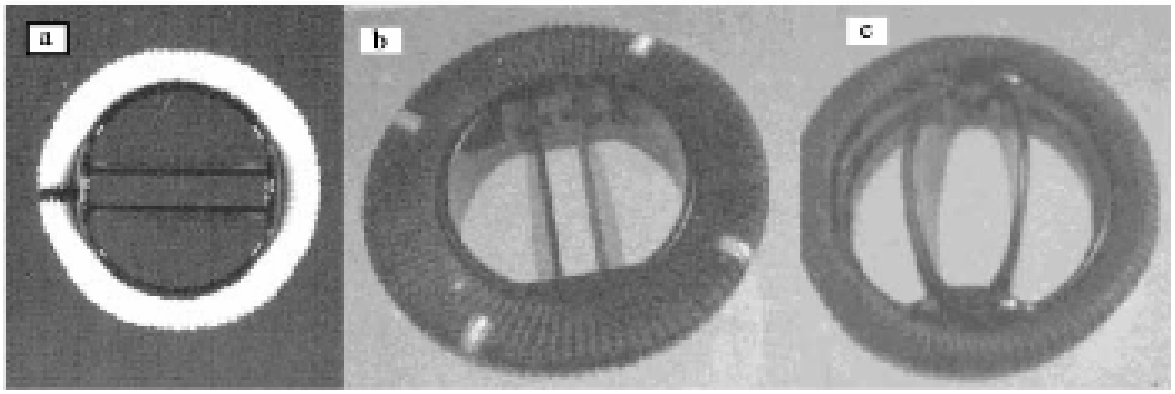
Bileaflet Valves



- A – St Jude
- B – Carbomedics
- C - Duramedics

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Case #2

- 87 yo WF presents with chest pain
- PMH: CAD, HTN
- EKG – LBBB
- CPK 587, index 7, trop I 9.7

- To Cath Lab

Code

- Hypotension
- PEA
- Pt intubated
- Echocardiogram at bedside

Complications of PCI

- Coronary Artery Dissection
  - If small and does not impede antegrade flow may be managed expectantly
- Abrupt Closure (up to 5% with angioplasty alone), occurs within 10 minutes
  - Usually due to dissection with superimposed thrombus/platelet aggregation or vessel spasm

Grossman, 565-571

Complications of PCI

- Myocardial infarction (1%)
  - Abrupt closure or snowplow effect
- Perforation of coronary with stiff guidewire
- Rupture of coronary with oversized balloon
- Ventricular Fibrillation (<1%)
- Balloon Rupture
- Wire fracture
- Misdeployment of stent

Grossman, 565-571

Case #3

- 76 yo AAM with 2 weeks increasing abdominal girth, DOE, increasing LE edema
- Recently changed from demedex bid to lasix qd
- PMH:
  - CHF, EF 20%
  - ? CAD; 2002 stress = fixed inferior wall defect
  - HTN
  - Gout

Meds

- Hyzaar 100/25 1 qd
- Lanoxin 0.125mg qd
- Aldactone 25mg qd
- Lasix 40mg qd
- Allopurinol 100mg qd
- KCl 20 meq bid

Echocardiogram

Echo findings

- Moderate dilitation of all chambers
- LVEF 15-20%
- Mod-Severe MR
- Severe TR
- Moderate AI
- Severe pulm HTN

LV remodeling

- Following MI, Left ventricle undergoes topographic and functional changes in the infarct zone and surrounding areas. Starts minutes after MI, may continue for years.
  - Infarct expansion
  - Subsequent dilatation of uninfarcted myocardium with hypertrophy
  - Interstitial fibrosis and impairment of contraction
  - Assumption of a more spherical shape

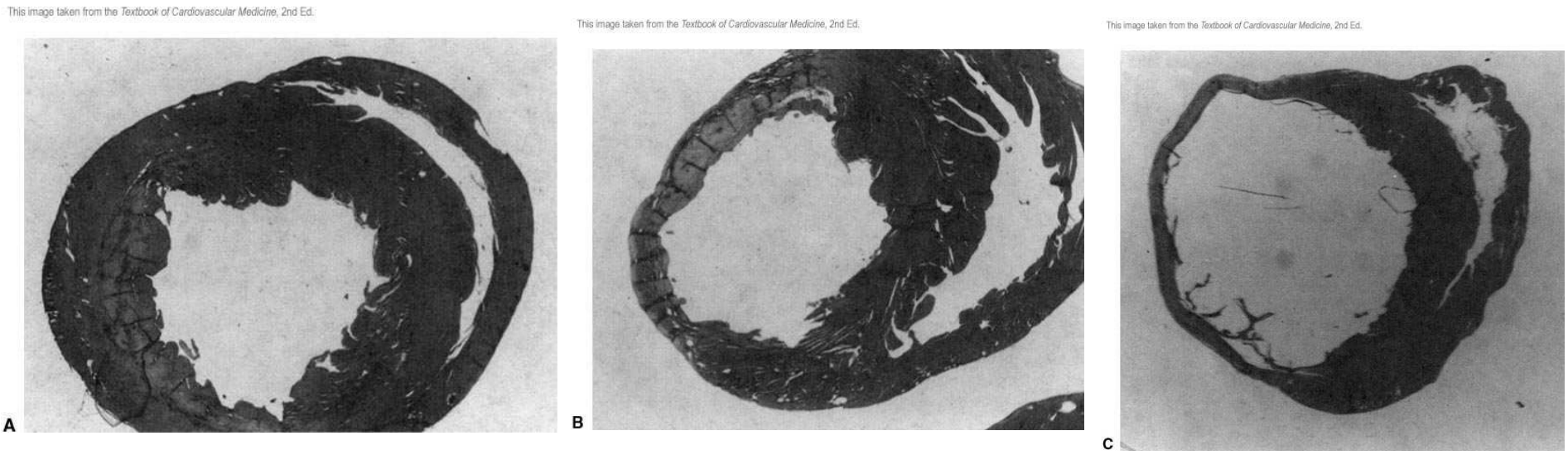
LV Aneurysm

- A discrete bulge of the LV composed of fibrotic tissue occurring when severe infarct expansion persists and scar is laid down on the topographic substrate.

Infarct Expansion

- Starts soon after coronary occlusion, reversible if flow reestablished quickly
- Large infarcts expand more than small ones
- Anterior and apical infarcts are at greatest risk of expansion
- Tansmural infarcts are more likely to expand than nontransmural ones.
- Expansion results in early LV dilitation

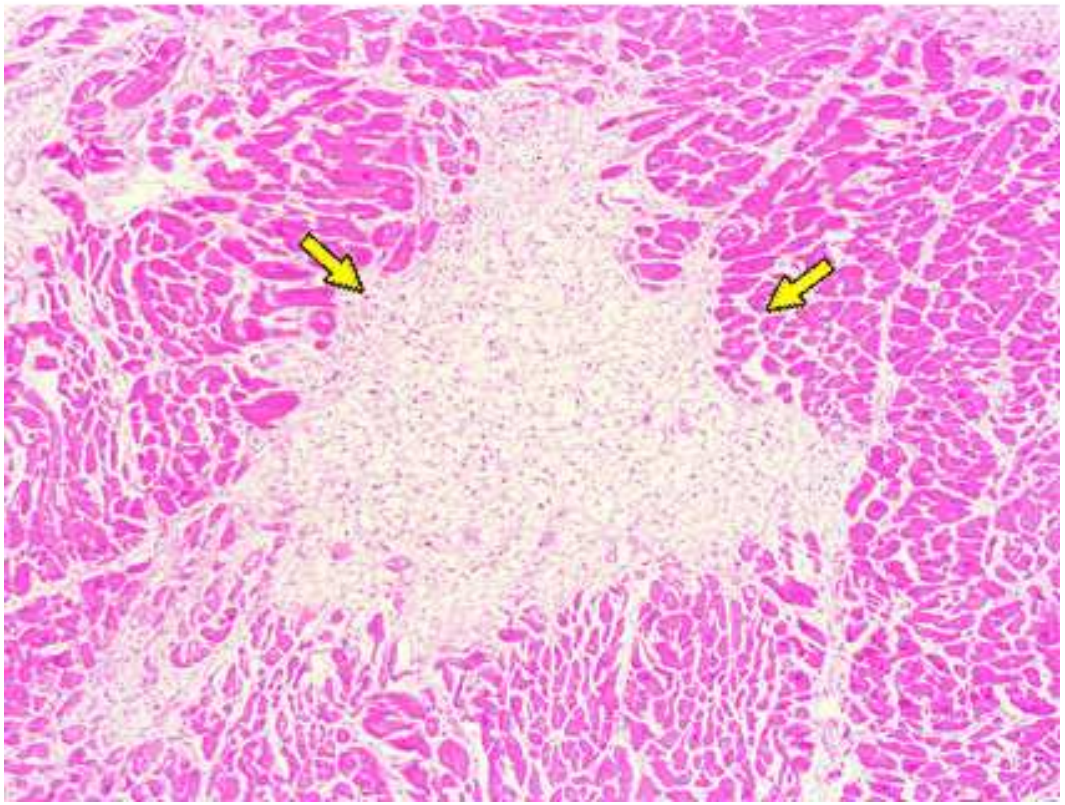
Infarct expansion



- **A:** Subendocardial infarct, early reperfusion, no expansion
- **B:** Late reperfusion, moderate infarct expansion
- **C:** Large infarct, severe expansion; no reperfusion

Early Healing (2 weeks)

This image taken from the *Textbook of Cardiovascular Medicine*, 2nd Ed.



- Removal of the necrotic myocytes
- Early inward growth of vascular tissue and fibroblasts (arrows)

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(True) Aneursym formation

- Due to regional expansion of infarct zone
- Early aneurysms are ‘functional aneurysms’ as they may be reversible by reperfusion
- Chronic aneurysm is a persistent bulge that is not reversed by reperfusion
- Pathologically composed of scar tissue
- Sometimes do contain viable myocardium that is hibernating

Early and Late Artery Hypotheses

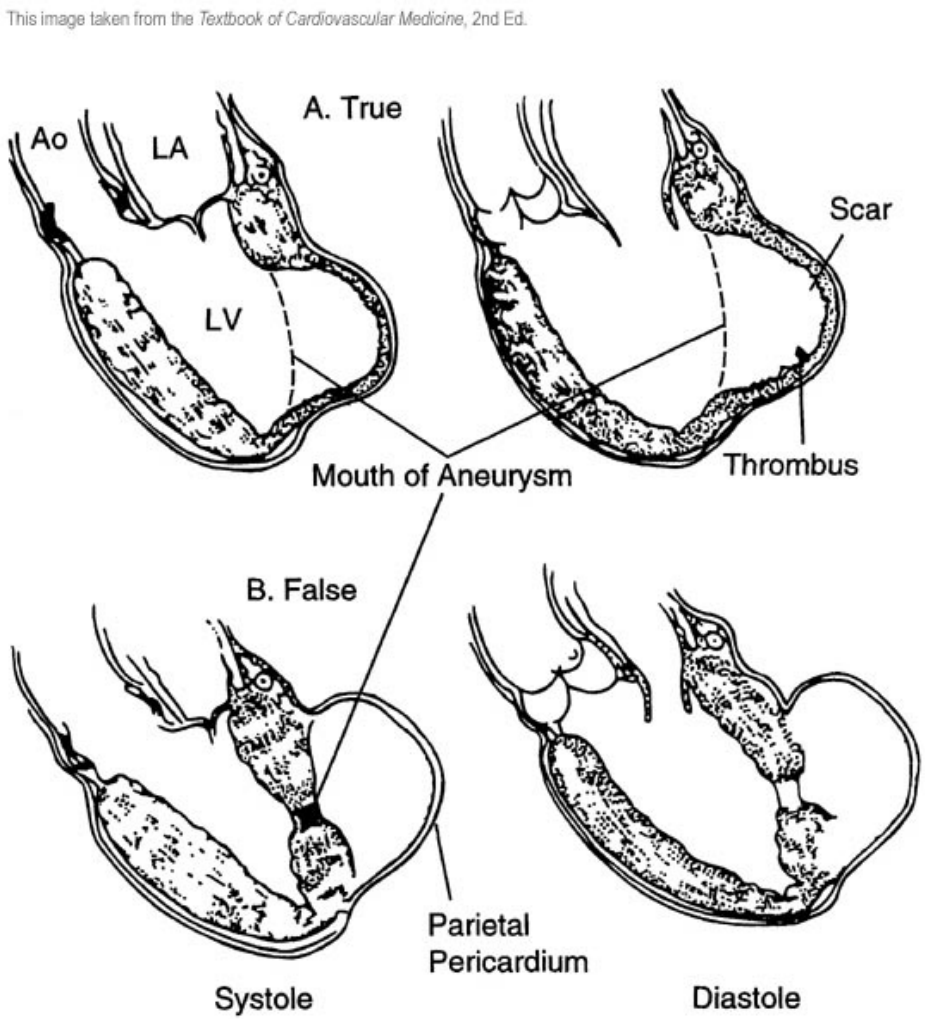
- Early open artery
  - Early reperfusion results in myocardial salvage and inhibits infarct expansion, reducing mortality.
- Late open artery
  - A patent infarct related artery produces a beneficial effect on LV remodeling independent of myocardial salvage

Open artery mechanisms

- Immediate changes in the infarct characteristics with formation of contraction bands and possibly edema and hemorrhage, resulting in a shorter, thicker, and stiffer infarct segment
- Preservation of small residual islands of myofibrils
- Possible preservation of interstitial collagen
- Accelerated healing
- The scaffold effect of a blood-filled vasculature
- Elimination of ischemia in viable dysfunctioning (hibernating) myocardium

True vs Pseudoaneurysm

- True
  - Wide mouth
  - Wall composed of LV fibrous tissue
  - May or may not contain thrombus
  - Almost never ruptures once healed
- False
  - Mouth is small
  - Site of myocardial ruptue
  - Wall is parietal pericardium
  - Almost always contains thrombus
  - Often ruptures



Aneurysm Risks

- Anteroapical, transmural infarction
- Inferoposterior MI may result in posterobasal aneurysm
- Risk higher with delayed or failed reperfusion
- Hypertension
- High ACE levels
- Steroids following MI increase risk due to delayed healing
- NSAIDS have been shown to worsen infarct expansion

Diagnosis - Clinical

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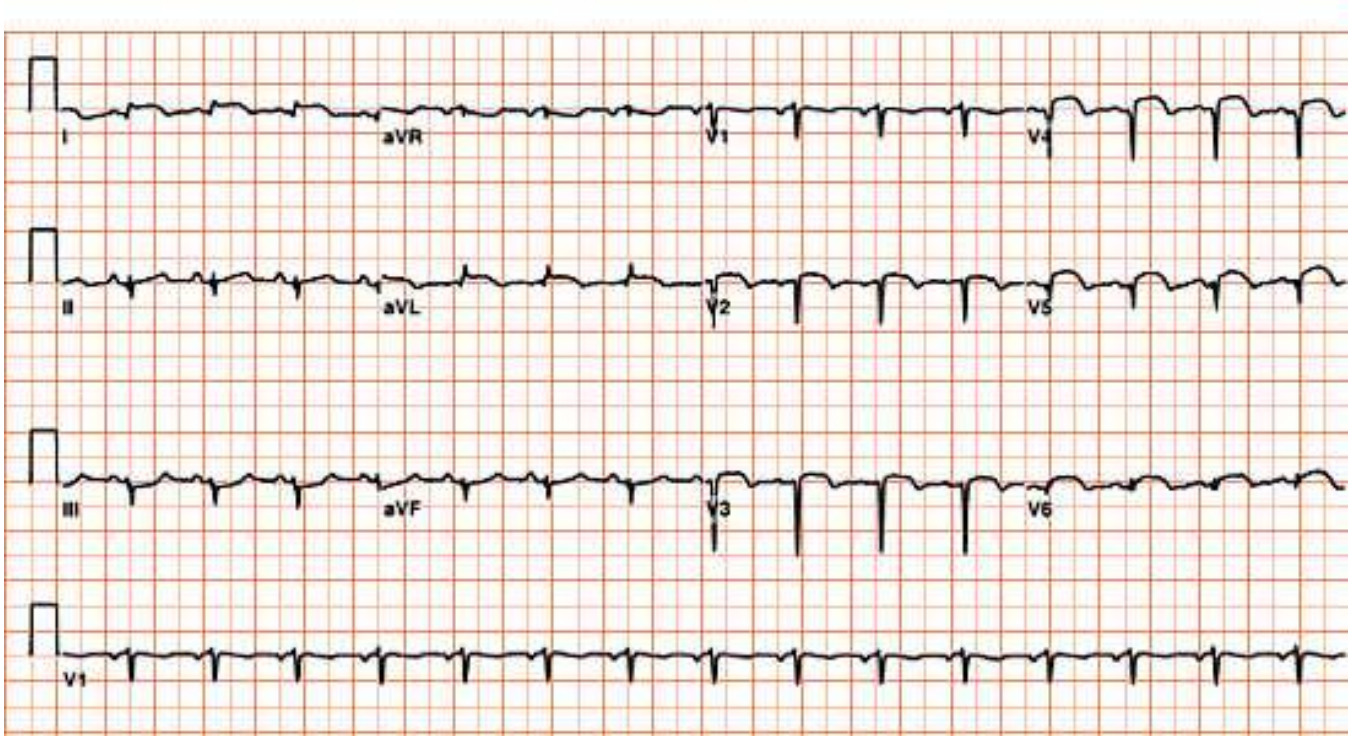

- Dyskinetic apical impulse
- Anteroapical infarct pattern with t wave inversions

Clinical Consequence

- Acute infarct expansion may cause rupture
  - Chronic aneurysms rarely rupture
- Infarct expansion increases risk of CHF
  - Both acute and chronic
- Recurrent and sustained, monomorphic ventricular tachycardia may occur in acute infarct expansion or in chronic LV aneurysm and may be refractory to antiarrhythmic therapy

LV thrombus

- Inflammation secondary to necrosis can produce thrombus
- Most frequently develops in anterior infarcts with expansion or aneurysm formation involving the apex
- Extensive thrombi (typically in apex) are at risk for embolization
- Mural thrombi more frequent in recently formed LV aneurysms

Mural thrombus

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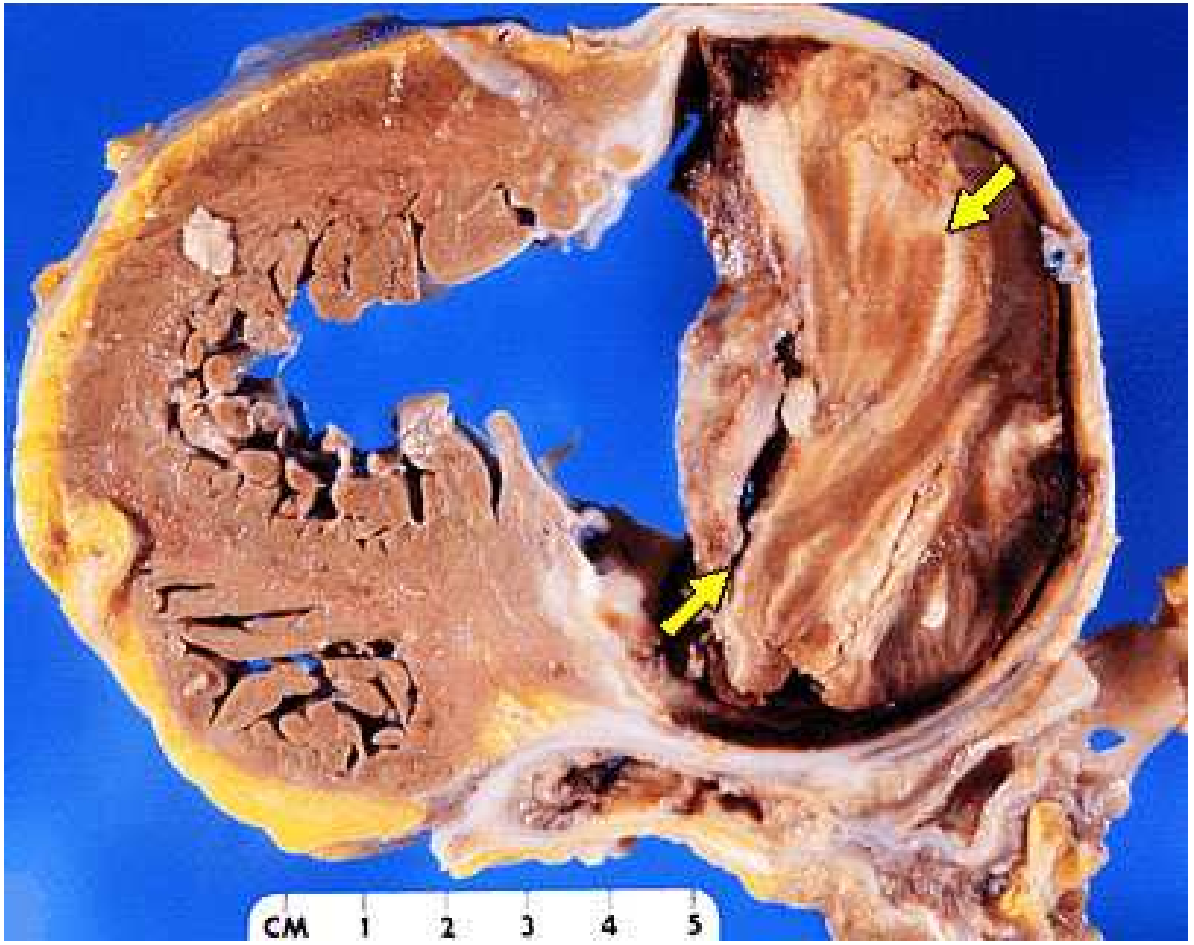

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Thrombus Formation

- 1985 estimated occurrence by echo 33% for anterior MI and less than 5% to 10% for other locations
- Less common now due to reperfusion
- GISSI-3 overall LV thrombus incidence was 5.1% of anterior MIs and 2.3% of nonanterior Mis
- Higher rates when EF <40%
- Risks include Killip class >I and early IV β-blocker use

Thrombus Embolization

- LV thrombi are associated with increased risk for systemic embolization especially with a protruding appearance
- Overall embolic incidence is low (2-6% with anterior MI in pre-reperfusion era)
- Stroke is the primary manifestation of cardiac emboli (occurring in 85% of cases)
- Atrial fibrillation after MI is additional risk
- Chronic LV aneursym presents low risk of systemic embolization
  - Noncontactile tissue
  - No endocardial inflammation
- **Caveat** – when LVEF is <40% after MI, overall stroke rate is 1.5% / year

Medical Management

- ACE inhibitors reduce early infarct expansion and remodeling
  - However, have not clearly been shown to be beneficial when reperfusion is successful
  - Recommended to start within 24 hours of MI
- ARB
  - Losartan efficacious when added to captopril
  - But higher incidence of hypotension
  - Recommended for ACE intolerance

Medical Management

- Nitrates
  - Reduce infarct expansion, less compelling data than ACE inhibitors
- Anticoagulation (Primary prevention)
  - Heparin and LMWH reduce incidence of LV thrombus
  - Thrombolytics have not shown reduction of incidence
- Anticoagulation (Therapy of LV thrombus)
  - Coumadin for 3 months recommended for LV thrombus, but not much data to support this

Prevention of Embolization

- Survival and Ventricular Enlargement investigators
  - Long-term use of warfarin in patients with EF less than 40% after MI was strongly associated with a reduced 5-year stroke rate (relative risk, 0.19; range, 0.13 to 0.27; $p < .001$).
  - ASA was also associated with a reduced risk (relative risk, 0.49; range, 0.29 to 0.65; $p < .001$)
- Anticoagulants in the Secondary Prevention of Events in Coronary Thrombosis-2 (ASPECT 2)
  - Warfarin alone or in combination with ASA was superior to ASA alone post-MI in preventing stroke (0% versus 0.3% versus 1.5%) as well as MI and death.
- Combined Hemotherapy and Mortality Prevention (CHAMP)
  - Stroke, MI, and death were not reduced by warfarin (Coumadin) plus ASA compared with ASA alone

Warfarin Therapy

- Recommended following anterior transmural infarction
  - Especially with akinetic/dyskinetic apical wall motion
- Recommended following other infarcts with low EF and CHF or Atrial fibrillation
- Target INR 2.0-3.0; 2.5-3.5 for prior systemic emboli
  - Coumadin Aspirin Reinfarction Study showed equivalent outcomes of low intensity (1-3mg coumadin) compared to ASA 160mg
- Duration of therapy questionable
  - 3 months followed by ASA advocated by ASPECT-2 study
  - 6 months for prior embolization
  - ? Indefinitely for patients with EF <30% post MI?

Infarctectomy/Aneursymectomy

- Decreased need due to reperfusion
- LV reduction surgery for CHF
  - Refractory CHF
  - Refractory VT
  - Consider in recurrent systemic thromboembolism

Aneurysm repair

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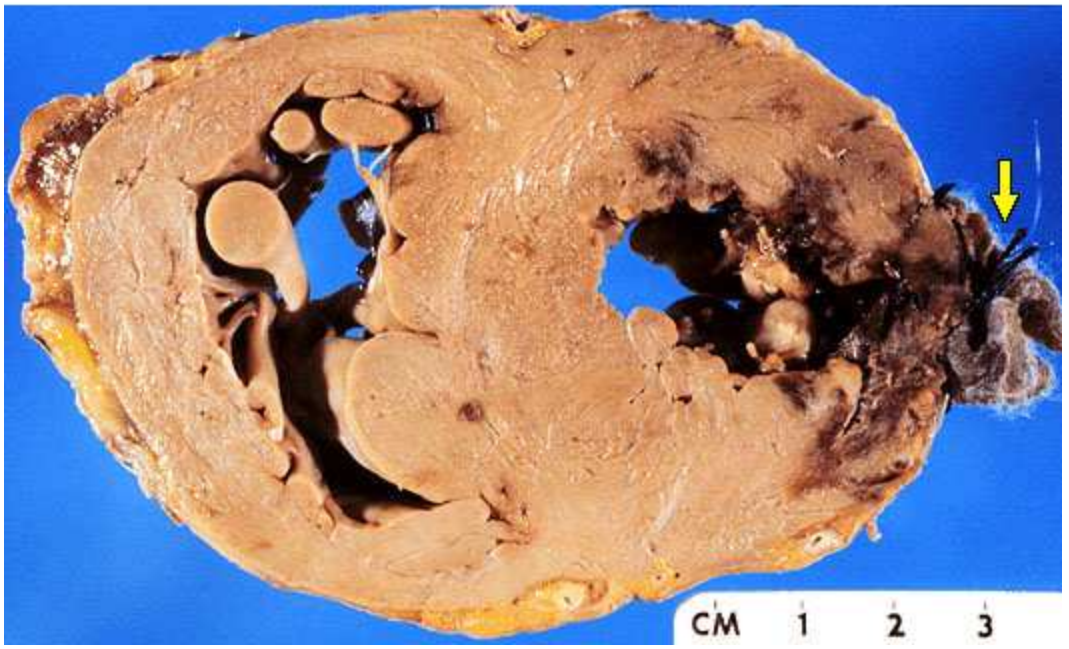

Aneurysm repair in a patient with a recent lateral wall myocardial infarction. "Packing material" is evident overlying the area of repair

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Repair Technique

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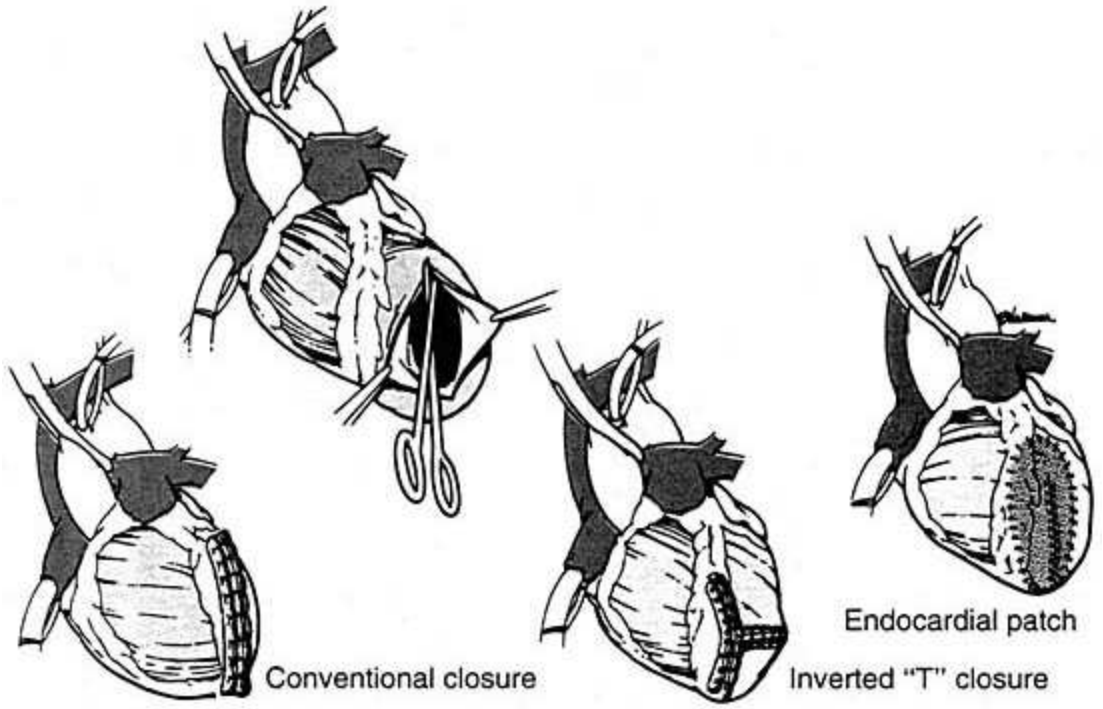

Resection of the ventricular aneurysm enclosure by one of three methods. The conventional closure is illustrated on the left. The T closure and the endocardial patch techniques were developed in an attempt to restore normal left ventricular geometry

Topol

References

- Baim DS, Grossman W: Grossman's Cardiac Catheterization, Angiography, and Intervention, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2000.
- Topol, EJ et al: Textbook of Cardiovascular Medicine, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2002.