Mechanical Complications of Acute Myocardial Infarction

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Outline

- Introduction
- Rupture of ventricular septum
- Mitral regurgitation from rupture of papillary muscle
- Rupture of ventricular free wall and pseudoaneurysm
- Delayed pericarditis (Dressler's) and tamponade
- Conclusions

Introduction

- Significant focus on management of acute MIs is related to revascularization strategies
- Mechanical complications pose a major threat to recovery in some patients
- Early, aggressive, and judicious treatment of these complications can substantially decrease the morbidity and mortality of acute MI.

Rupture of the Ventricular Septum

Post-MI VSDs

- Infrequent event following transmural MI
- Syndrome ranges from asymptomatic murmur to extensive left-to-right intracardiac shunt
  - Resulting heart failure and shock

Post-MI VSDs

- Initial approaches emphasized delayed repair
  - To allow for fibrosis to occur
  - Enables tissue quality at defect margins to be more substantial
- Salvageable patients deteriorate during period of delay
- Modern approaches → early surgical intervention is accepted treatment

Post-MI VSDs

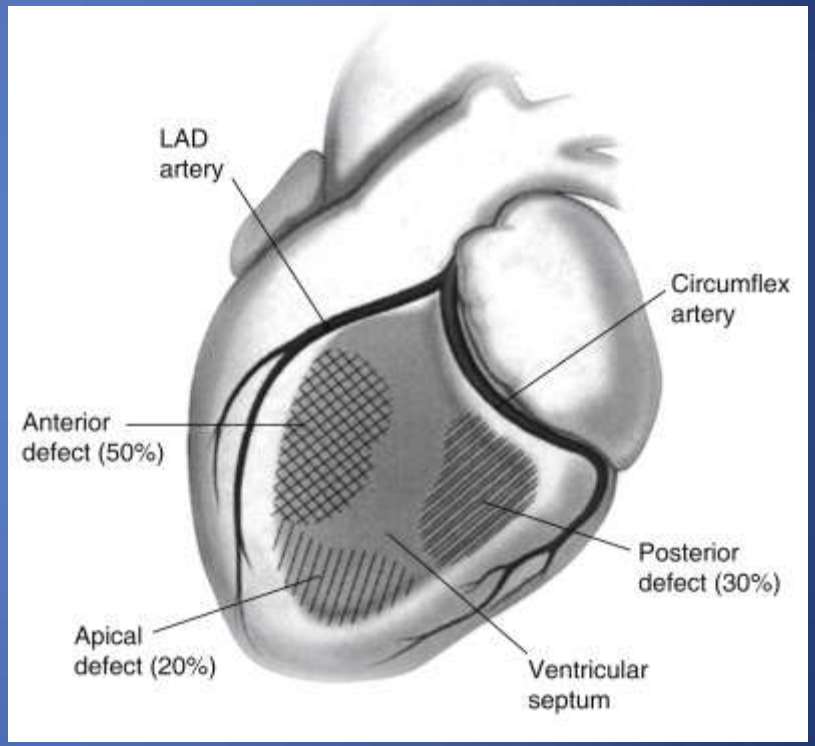
- Incidence
  - Complicates 1-2% of MIs
  - Accounts for 5% of deaths after MIs
  - Declining because of aggressive medical/interventional management of acute MI and better treatment of post-infarction HTN

Post-MI VSDs

- Incidence
  - Post-infarction VSDs more frequent in males (M:F 3:2)
  - Average age is 62 years
  - Septal rupture occurs most often after 1st acute MI

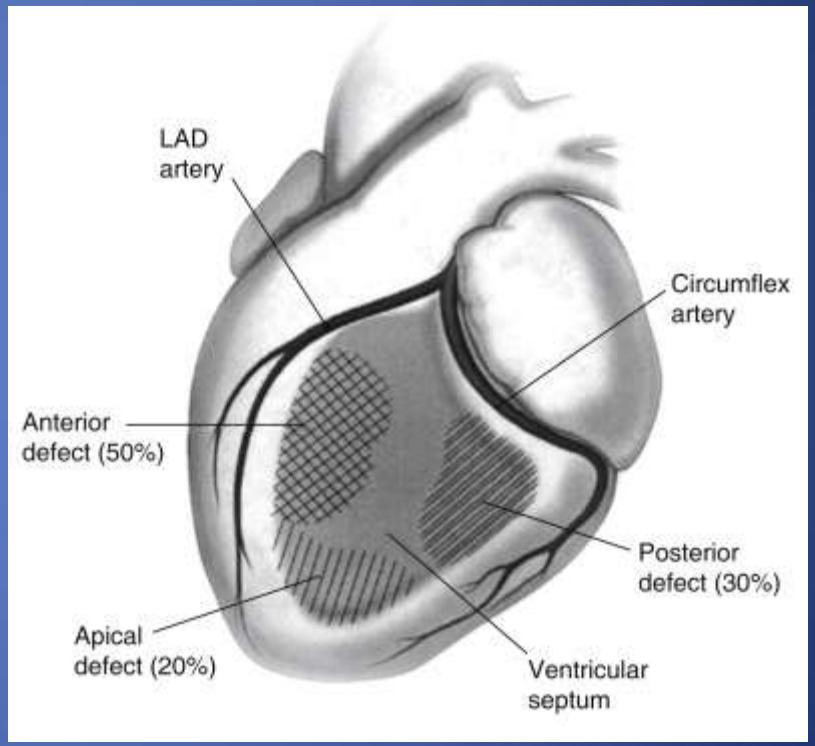
Post-MI VSDs

- Etiology/Pathogenesis
  - Angiography → occluded culprit coronary artery
  - 2/3^{rds} of patients have single vessel disease
  - Located most commonly (~70% of cases) in anteroapical septum
    - Due to full-thickness anterior MI from LAD occlusion



Post-MI VSDs

- Etiology/Pathogenesis
  - 20-40% with VSDs → rupture of posterior septum
    - Due to inferoseptal infarction from occlusion of a dominant right or circumflex coronary artery



Post-MI VSDs

- Etiology/Pathogenesis
  - Simple rupture
    - direct through-and-through defect – more common usually located anteriorly
  - Complex rupture
    - more serpiginous tract
    - less common
    - usually located inferiorly
  - Most patients develop single VSD
    - 5-11% have multiple septal defects

Post-MI VSDs

- Etiology/Pathogenesis
  - Underlying MI for post-MI VSD usually extensive
  - Often involves 26% of free wall on average
    - Compared with 15% in non-complicated acute MIs
  - Develops 2-4 days after acute MI
    - Reported as early as a few hours after MI through up to 2 weeks later

Post-MI VSDs

- Pathophysiology
  - Primary determinant of outcome → development of heart failure
    - Function of both the size of defect and magnitude of MI
    - Left sided heart failure predominates in anterior VSDs
    - Right-sided failure predominates in posterior VSDs

Post-MI VSDs

- Pathophysiology
  - With each defect in septum, proportion of LV ejection is diverted from systemic circulation across septum into right ventricle
    - Compromises forward systemic cardiac output
    - Overloads pulmonary circulation

Post-MI VSDs

- Pathogenesis
  - Resulting cardiogenic shock → end-organ malperfusion and organ failure (irreversible & fatal)
  - Normally compliant RV may develop severe diastolic failure after prolonged L-to-R shunt
    - Results escalation in RV diastolic pressures
    - Can result ultimately in flow reversal across septum (R-to-L), compounding situation further with systemic hypoxia.

Post-MI VSDs

- Natural History
  - Without intervention, ¼ of pts die in 24 hours
  - ½ succumb to illness within 1st week
  - 2/3rds die within 2 weeks
  - ¾ die within 1 month
  - Only 7% survive longer than one year

Post-MI VSDs

- Practice of waiting weeks after post-MI rupture of septum
  - Selected out pts with lesser pathology and mild hemodynamic insult
- Deferring early operation in hopes of maintaining hemodynamic stability
  - Deprives patients of chance for successful outcome before irreversible end-organ damage occurs

Post-MI VSDs

- Presentation
  - Harsh new holosystolic murmur, may radiate to axilla
    - Associated with recurrent CP in >50% of pts
  - Signs of right-side heart failure
  - EKG findings → those of the antecedent infarction
  - 1/3rd of pts develop transient AV conduction block that precedes rupture
  - Syndrome may resemble acute mitral regurgitation due to rupture of papillary muscle

Post-MI VSDs

- Diagnosis
  - Right heart catheterization
    - >9% step-up in the oxygen saturation between the RA and PA → diagnostic of VSD
    - Elevated pulmonary-to-systemic flow ratio (ranges from 1.4:1 to 8:1) → VSD (correlates with size)
  - Doppler ECHO
    - Shows size and location of defect
    - Determines ventricular function
    - Assesses pulmonary artery and RV pressures
    - Excludes concomitant mitral valve disease
      - Sensitivity & specificity → 100%

Post-MI VSDs

- Indications for Operation
  - Diagnosis of this entity INDICATES operation
  - If cardiogenic shock → SURGICAL EMERGENCY
  - Patients already in multisystem organ failure → unlikely to survive emergent repair
    - May benefit from mechanical bridge for salvage before operation
    - Pts in intermediate status need operation within 12-24 hours
    - <5% of pts with no clinical compromise can be treated on semi-elective basis

Post-MI VSDs

- Pre-Op Management Considerations
  - Directed toward maintaining hemodynamic stability & preventing end-organ damage
  - GOALS:
    - Reducing systemic vascular resistance (reducing left-to-right shunting)
    - Maintaining cardiac output and peripheral perfusion
    - Maintaining or improving coronary blood flow
    - ➔ IABP or medical Tx

Post-Op VSDs

Table 92–1 -- Preoperative Predictors of Death after Surgical Repair of Postinfarction Ventricular Septal Defect (VSD)

Variable	Predictor of Early Death	Predictor of LateDeath
Need for preoperative catecholamines	$P = .001$	P = NS
Emergent operation	$P < .0001$	P = NS
Anterior VSD	$P = .04$	P = NS
Age > 65 years	$P = .009$	P = NS
Right-side-of-the-heart failure	$P = .01$	$P = .005$
Elevation in blood urea nitrogen	$P = .02$	P = NS
Elevation of serum creatinine	P = NS	$P < .05$
Previous myocardial infarction	P = NS	$P < .05$
Presence of left main coronary disease	P = NS	$P < .05$

NS, not significant.

Operative Management

- Bicaval venous drainage
- Cannulaton of ascending aorta
- Systemic hypothermia
- Antegrade & retrograde cardioplegia
- Revascularization done before opening ventricle (to optimize myocardial protection)

TABLE 28-1A Principles of Infarctectomy Repair of Postinfarction VSD

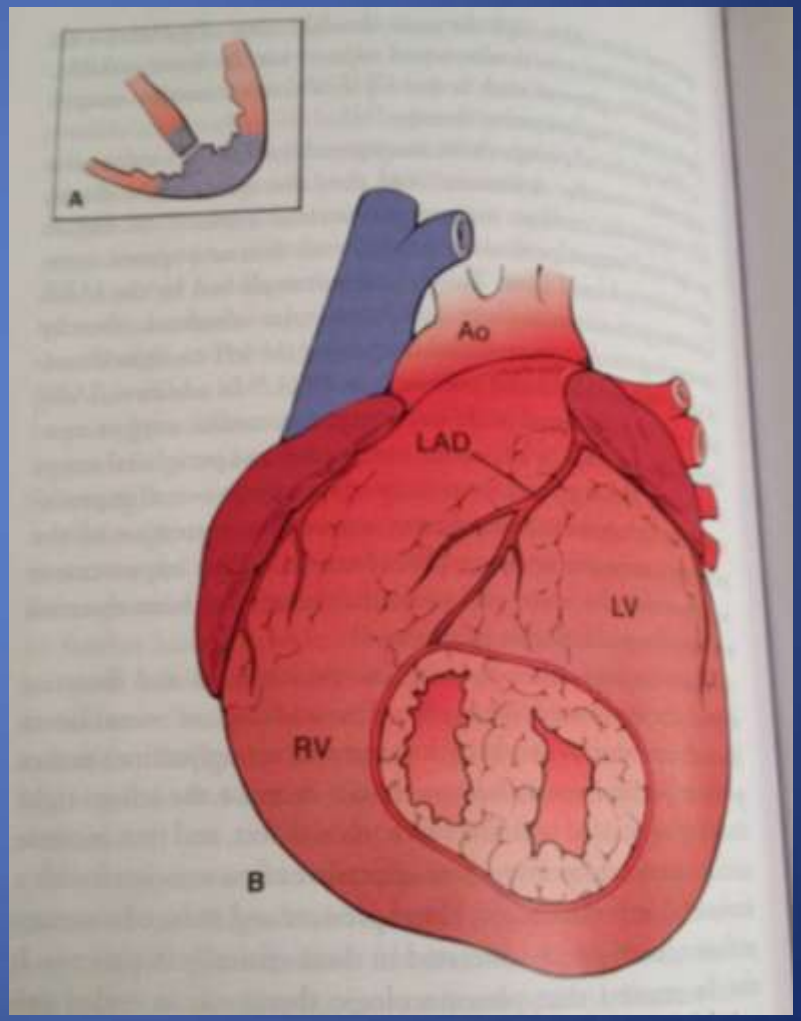
Transinfarct approach to ventricular septal defect

1. Thorough trimming of the left ventricular margins of the infarct back to viable muscle to prevent delayed rupture of the closure
2. Conservative trimming of the right ventricular muscle as required for complete visualization of the margins of the defect
3. Inspection of the left ventricular papillary muscles and concomitant replacement of the mitral valve only if there is frank papillary muscular rupture
4. Closure of the septal defect without tension, which in most instances will require the use of prosthetic material
5. Closure of the infarctectomy without tension with generous use of prosthetic material as indicated, and epicardial placement of the patch to the free wall to avoid strain on the friable endocardial tissue
6. Buttressing of the suture lines with pledgets or strips of Teflon felt or similar material to prevent sutures from cutting through friable muscle

Source: Heitmiller R, Jacobs ML, Daggett WM: Surgical management of postinfarction ventricular septal rupture. Ann Thorac Surg 1986; 41:683.

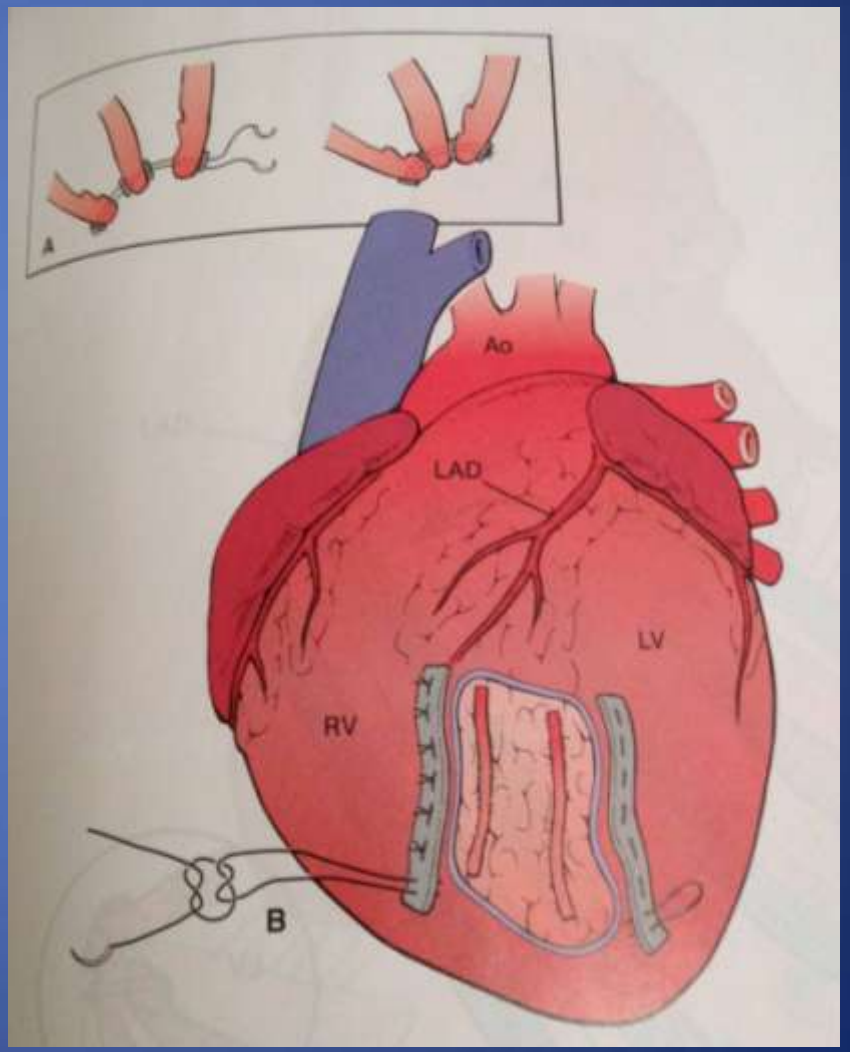
Repair of Apical Septal Rupture

- Apical Amputation
  - Incision through infarcted apex of left ventricle
  - Debridement of necrotic myocardium back to healthy muscle → amputation of apex of heart (including LV, RV, and septum)



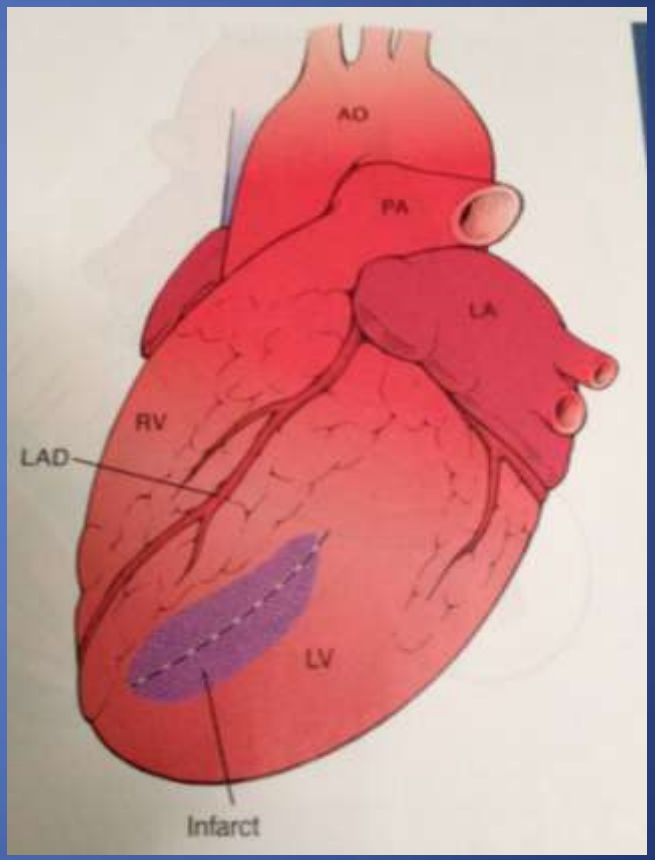
Repair of Apical Septal Rupture

- Apical Amputation
  - Remaining apical portions are reapproximated to apical septum
    - Two interrupted mattress sutures of 0 Tevdek, passed sequentially through buttressing strip of Teflon felt, LV wall, 2nd strip of felt, septum, 3rd strip of felt, RV wall, and 4th strip of felt.
  - After sutures are tied, closure is reinforced with additional running suture.

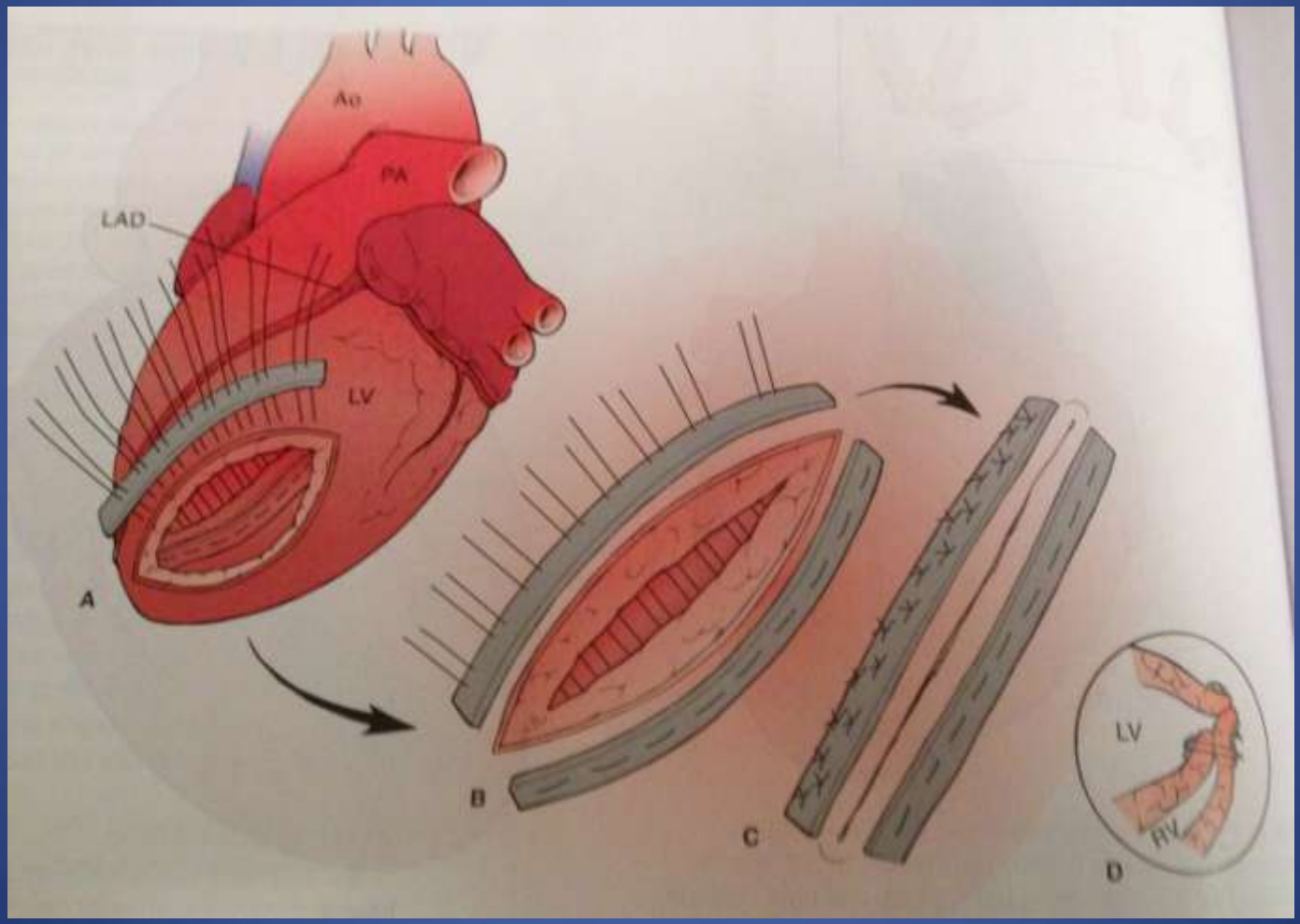


Anterior Repair with Infartectomy

- LV transinfarct incision with infartectomy
- Small defects can be closed by plication
  - Approximation of free anterior edge of septum to RV free wall w/1-0 Tevdek mattress sutures over strips of felt
  - Transinfarct incision closed with 2nd row of mattress sutures buttressed with strips of Teflon felt

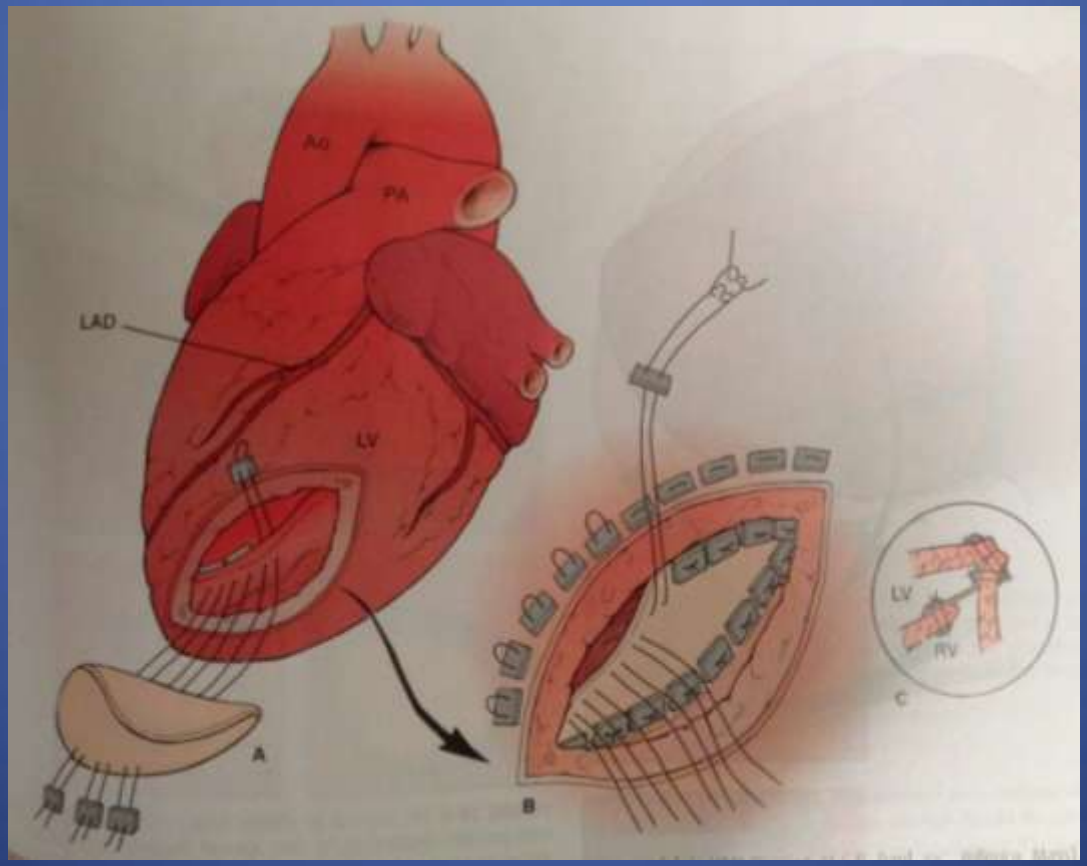


Anterior Repair with Infartectomy



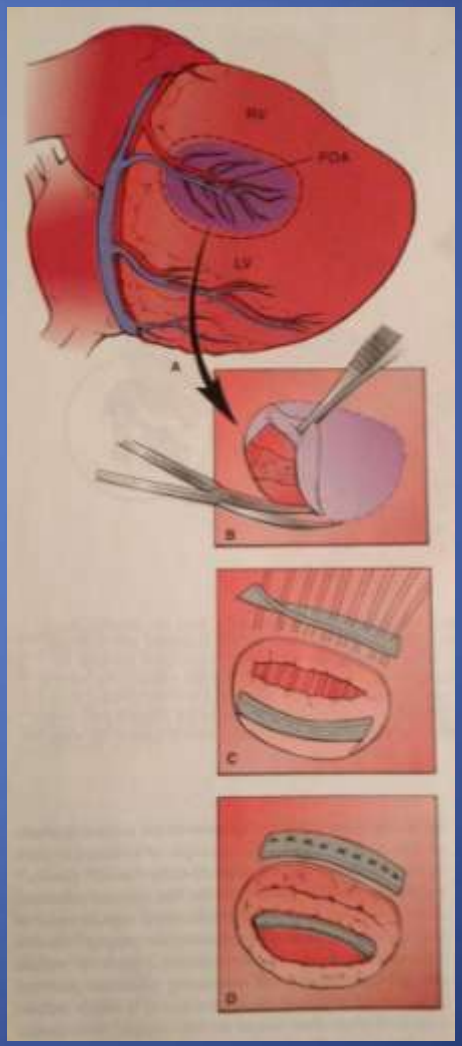
Anterior Repair with Infarctectomy

- Larger anterior defects require closure with a prosthetic patch

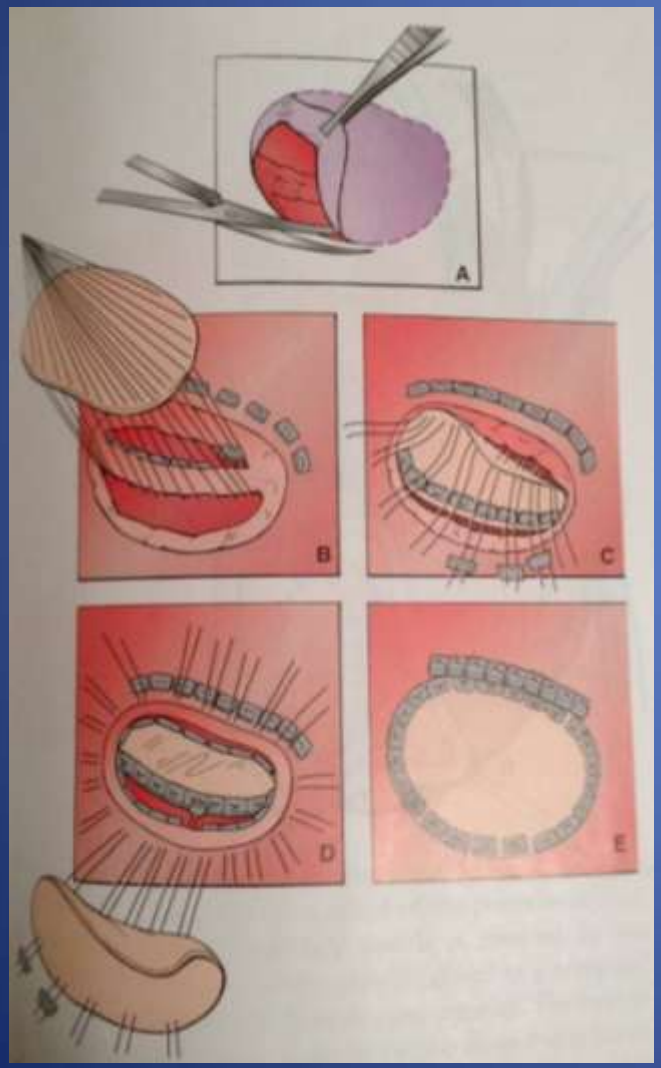


Posterior/Inferior Repair with Infarctectomy: Small Defects

- Inferoposterior defects result form transmural infarction in distribution of PDA
- Difficult to repair, possibly more amenable to exclusion techniques



Posterior/Inferior Repair with Infarctectomy: Large Defects



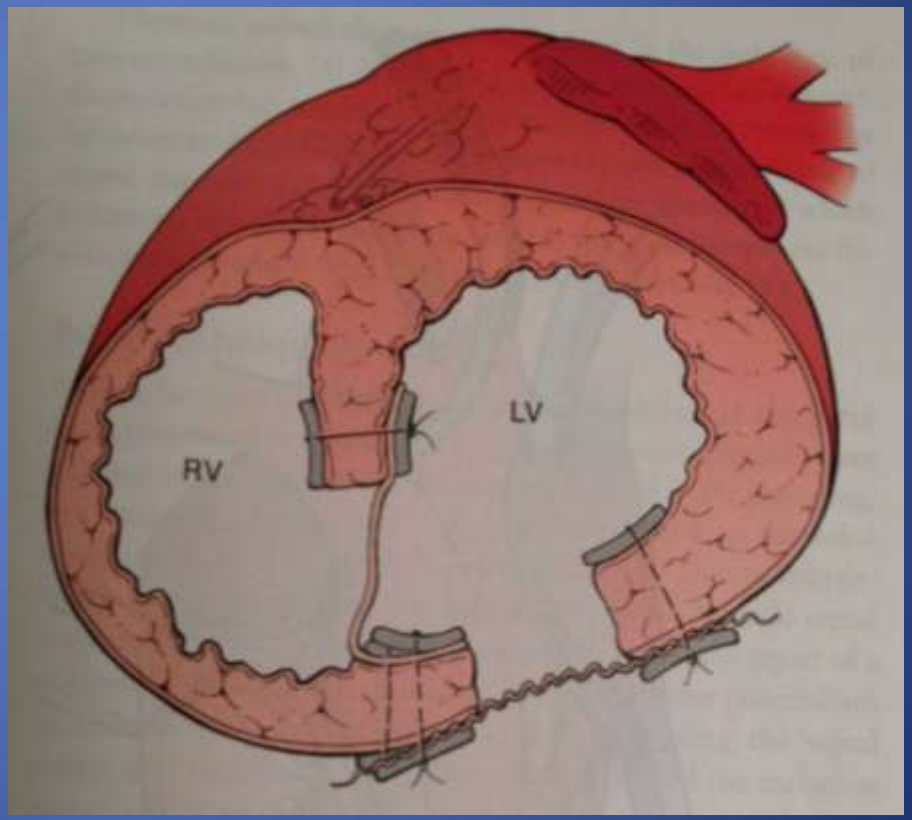


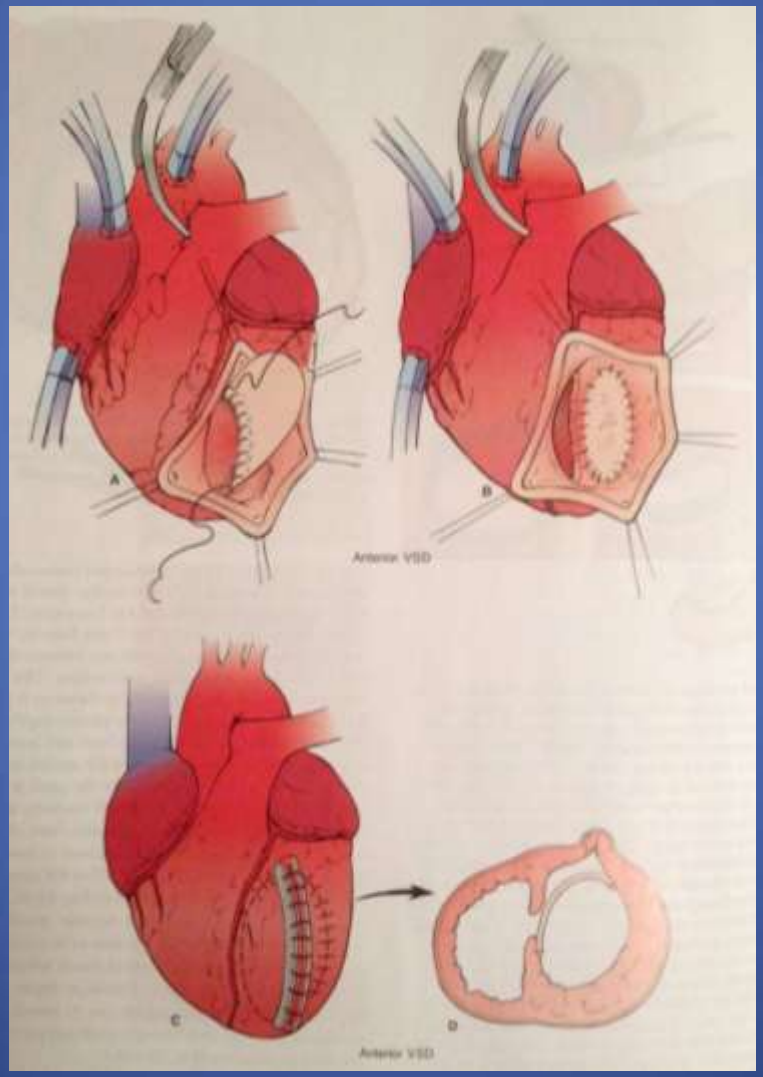
TABLE 28-18 Principles of Exclusion Repair of Postinfarction VSD

Transinfarct approach to ventricular septal defect

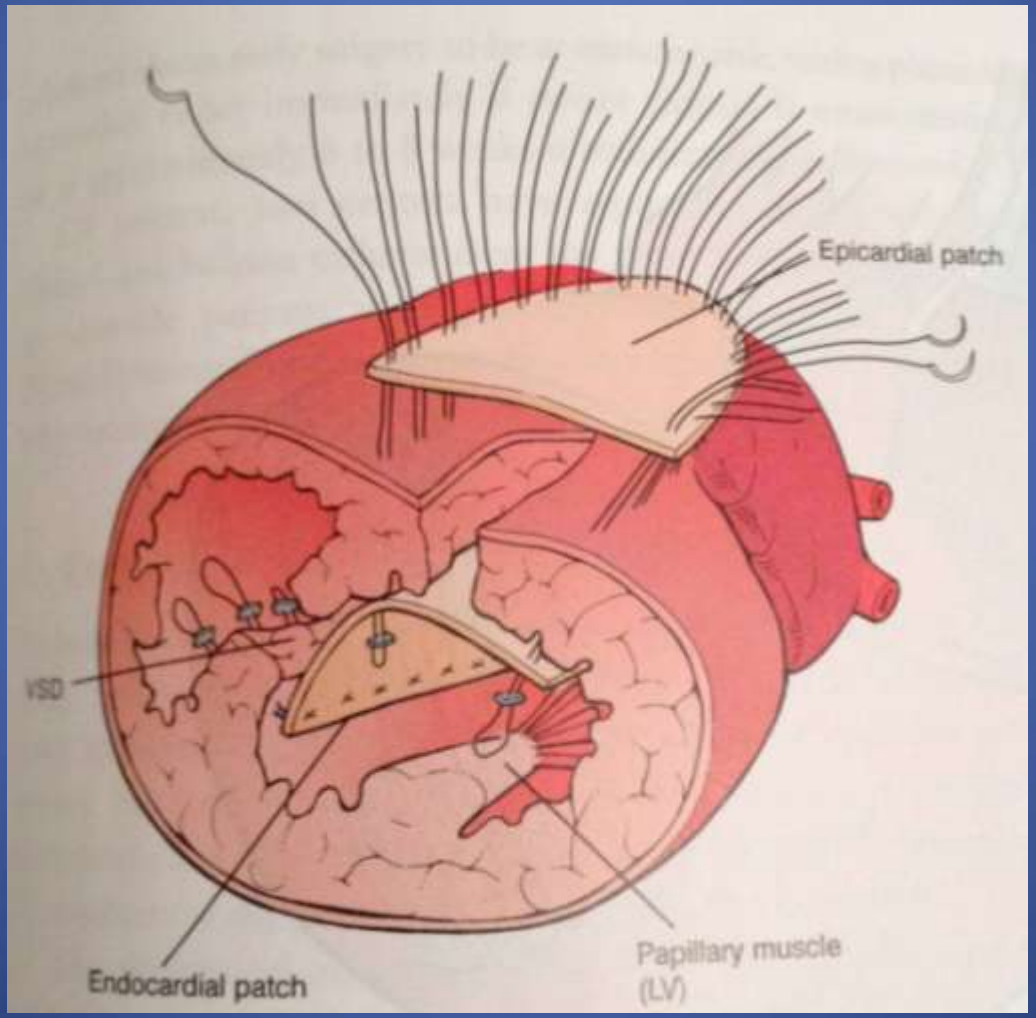
1. No infarctectomy unless necrotic muscle along ventriculotomy is sloughing during closure
2. Bovine pericardial patch in either an oval (anterior defect) or triangular (posterior defect) shape is sutured securely with continuous Prolene around the defect to exclude it from the LV cavity
3. Where necessary, full-thickness bites are taken to the epicardial surface and anchored by strips of pericardium or Teflon (see text for details)
4. An anterior patch is anchored to noninfarcted septum below the defect, then the noninfarcted endocardium of the anterolateral ventricular wall. If the infarct involves the base of the anterior muscle, full thickness anchoring bites are used.
5. A posterior patch is anchored to the mitral annulus, noninfarcted septum, and through the infarcted posterior wall along a line corresponding to the medial margin of the posteromedial papillary muscle (with full thickness anchoring).
6. Closure of the infarctectomy using strips of pericardium or Teflon
7. When possible, infarcted right ventricular free wall is left undisturbed during closure

Source: David TE, Dale L, Sun Z: Postinfarction ventricular septal rupture: repair by endocardial patch with infarct exclusion. J Thorac Cardiovasc Surg 1995; 110:1315.

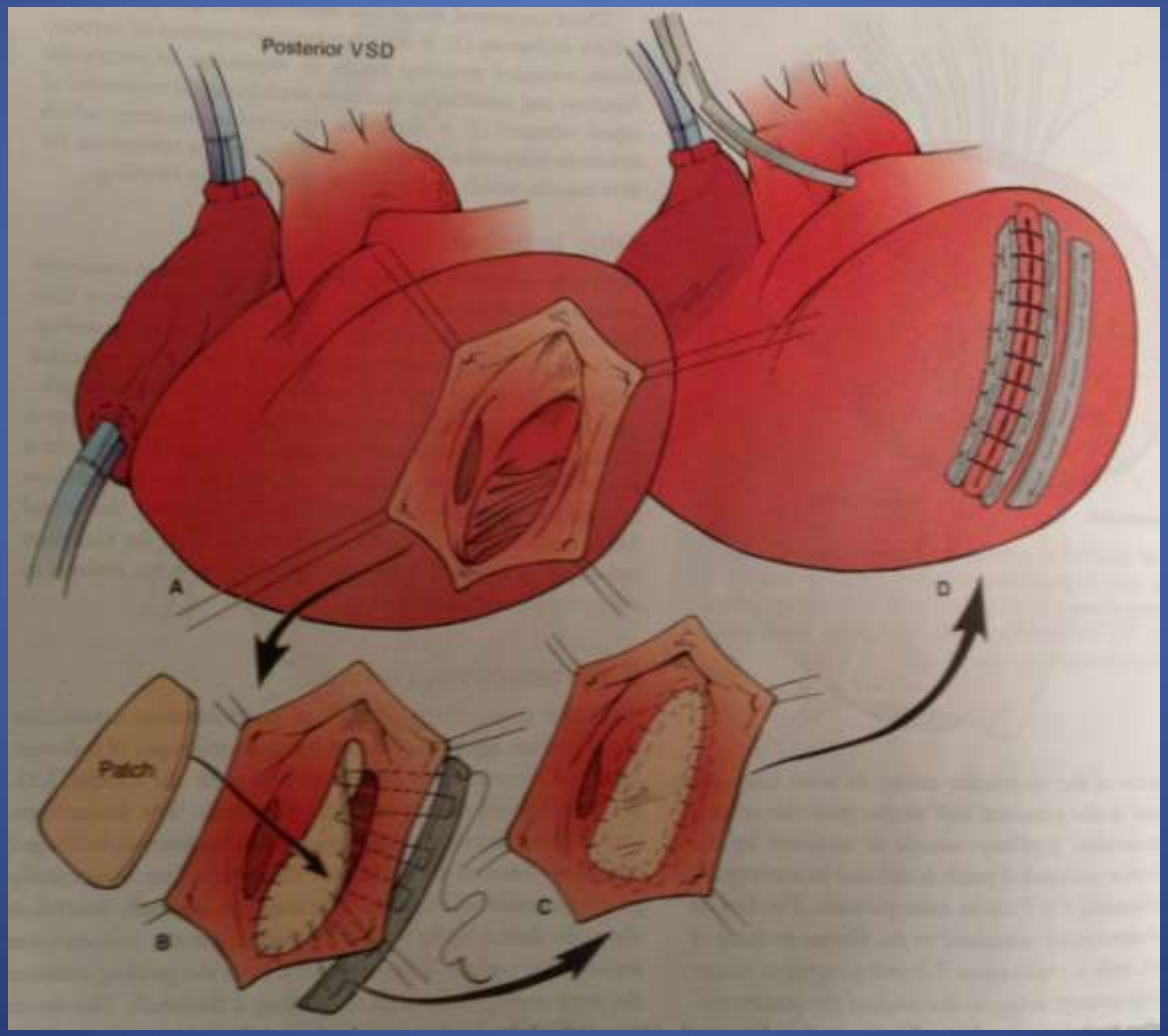
Anterior Infarct Repair by Exclusion



Anterior Infarct Repair by Exclusion



Posterior Infarct Repair by Exclusion



Rupture of the Ventricular Free Wall

Rupture of the Ventricular Free Wall

- History
  - 1st described in 1647
- Incidence
  - 11% of patients after AMI
    - Has been as high as 31% in autopsy studies of anterior MI
  - Ventricular rupture and cardiogenic shock → leading causes of death after AMI
    - Account together for >66% of early deaths in patients suffering their 1st acute infarction

Rupture of the Ventricular Free Wall

- Incidence:
  - More common in elderly women (mean 63 years)
  - In past, 90% of ruptures → within 2 weeks after infarction
    - Peak incidence at 5 days
  - Time to cardiac rupture accelerated by thrombolysis and coronary reperfusion
    - Occurring within hours from onset of MI symptoms

Rupture of the Ventricular Free Wall

- Most common site:
  - Older literature → anterior wall is most frequent site
  - Recent series → lateral and posterior wall ruptures
  - Lateral wall is more likely to rupture than anterior wall, but anterior infarctions are more frequent than lateral infarctions
  - **OVERALL most common site of rupture is anterior wall**

Rupture of the Ventricular Free Wall

- Free ruptures: Simple vs. complex
  - Simple rupture:
    - Straight through-and-through tear perpendicular to endothelial and epicardial surfaces
  - Complex rupture:
    - More serpiginous tear, often oblique to the endocardial and epicardial surfaces

Rupture of the Ventricular Free Wall

- 3 clinicopathologic categories:
  - Acute:
    - Sudden recurrent chest pain, electrical mechanical dissociation, profound shock, and death from massive hemorrhage into pericardium.
    - Not amenable to management

Rupture of the Ventricular Free Wall

- 3 clinicopathologic categories:
  - Subacute:
    - Smaller tear, may be temporarily sealed by clot of fibrinous pericardial adhesions
    - Presents with signs/symptoms of cardiac tamponade and cardiogenic shock
    - Mimics: infarct extension, RV failure
    - Compatible with life for several hours or days

Rupture of the Ventricular Free Wall

- Chronic rupture with false aneurysm formation:
  - Leakage of blood is slow
  - Surrounding pressure on epicardium controls hemorrhage
  - Adhesions form between epicardium and pericardium
    - Reinforce and contain rupture
  - Most common clinical presentation: CHF
  - Angina, syncope, arrhythmias, and thromboembolic complications occur in some pts

Differences between true and false aneurysms

- Wall of false aneurysm contains no myocardial cells
- False aneurysms are more likely to form posteriorly
- False aneurysms usually have a narrow neck
- False aneurysms have great propensity for rupture

Rupture of the Ventricular Free Wall

- Cardiac rupture occurs with transmural infarction
- **Infarct expansion** plays an important role
  - Acute regional thinning and dilatation of infarct zone
    - Due to slippage between muscle bundles → reduction in number of myocytes across the infarcted area
    - Increases size of ventricle, with increase in wall tension (Laplace effect) → endocardial tearing.
  - Seen as early as 24 hours after transmural MI
  - Not related to additional myocardial necrosis

Rupture of the Ventricular Free Wall

- Late [vs. early] thrombolysis might increase likelihood of rupture
  - Converts bland infarct into hemorrhagic infarct

- Rupture of LV wall may occur in isolation or with rupture of the septum, papillary muscles, or RV.

Diagnosis

- Clinical picture → Pericardial tamponade
  - Pulsus paradoxus, distended neck veins, cardiogenic shock
  - ECHO:
    - Effusion thickness > 10mm
    - Echodense masses in effusion
    - Ventricular wall defects
    - Signs of tamponade
      - RA and RV early diastolic collapse
      - Increased respiratory variation in transvalvular blood flow velocities

Diagnosis

- Pericardiocentesis & aspiration of noncoagulated blood → subacute rupture
  - Clear fluid excludes cardiac rupture
  - Also provides short-term circulatory improvement

Symptoms and EKG Criteria Predictive of Cardiac Rupture

TABLE 28-4 Sensitivity, Specificity and Predictive Value of Symptoms and Electrocardiographic Criteria for Cardiac Rupture

	Sensitivity (%)	Specificity (%)	Predictive value (%)
Pericarditis	86	72	68
Repetitive emesis	64	95	90
Restlessness, agitation	55	95	86
Two or more symptoms	84	97	95
ST segment deviations	61	72	58
T wave deviations	94	66	66
ST-T wave deviations	61	68	64

From Oliva PB, Hammill SC, Edwards WD: Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993; 22:720.

Natural History

- Acute rupture is fatal!
- Subacute rupture: Pts can survive hours to days.
- Natural history of false aneurysm of LV not well established
  - Believed to have poor prognosis because of high probability of rupture
  - Outcome potentially alterable by widespread use of ECHO

Preop Management: Subacute Rupture

- Once diagnosis is established, patient should go straight to OR
- No time wasted for coronary angiography
- Inotropy and fluids started in preparation
- Pericardiocentesis for transient hemodynamic improvement
- IABP useful

Preop Management: False Aneursym

- Dependent on age of MI
- False aneursym dx'ed within 2-3 months following MI
  - Surgery following coronary angiography and ventriculography
- False aneurysm dx'ed several months or years after MI
  - Urgency determined by symptoms & severity of CAD, not by risk of rupture

Operative Strategy

- Complete sterile preparation and draping of patient BEFORE induction of anesthesia
- Prepare to rapidly cannulate groin if needed
- Median sternotomy
- Decompression of pericardium → improvement of BP
  - Anticipated, because high BP can worsen ventricular rupture size
- Most cases, rupture is sealed off by clot

Operative Strategy: 5 techniques

- Close rent with large horizontal mattress sutures, buttressed with two strips of Teflon felt
  - Not recommended
  - Sutures are placed into necrotic, friable myocardium that can easily tear
- Results are anecdotal

Operative Strategy: 5 techniques

- Infarctectomy and closure of defect with interrupted, pledgeted sutures or Dacron patch
  - Requires aortic cross clamping
  - Reserved for patients with VSD

Operative Strategy: 5 techniques

- Close defect →
  - Horizontal mattress sutures
  - Buttressed with two strips of Teflon felt
- Cover closed ventricular tear and surrounding infarcted myocardium
  - Teflon patch sutured to healthy epicardium
  - Continuous polypropylene suture

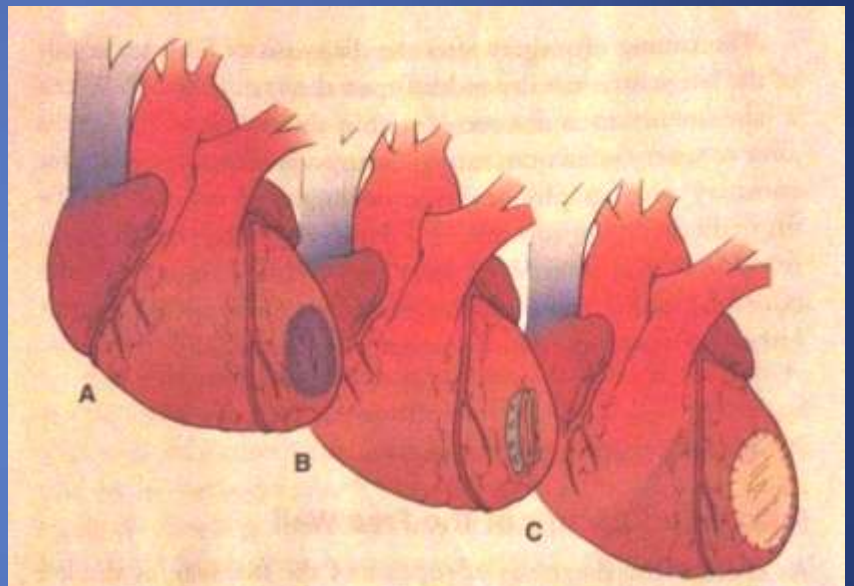


FIGURE 28-17 Technique to repair rupture of the free wall of the left ventricle. (**A**) Left ventricular free wall rupture. (**B**) A limited infarctectomy is closed with horizontal mattress sutures buttressed with two strips of Teflon felt. (**C**) Then the whole area is covered with a Teflon patch sutured to healthy epicardium with a continuous polypropylene suture. Alternatively, the Teflon patch can be glued to the ventricular tear and the infarcted area using a biocompatible glue. (*Adapted with permission from David.*[138])

Operative Strategy: 5 techniques

- Gluing patch of Teflon or bovine pericardium to ventricular tear or infarcted area using fibrin glue
  - Does not require CPB
  - May be useful when ventricle is not actively bleeding
- Results are anecdotal

Operative Strategy: 5 techniques

- For False Aneurysm of Left Ventricle:
  - Best repaired with endocardial patch
  - Chronic anterior false aneurysms can be closed primarily if neck is fibrotic

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  - If primary closure of neck of posterior false aneurysm may exacerbate MR
    - Should be reconstructed with patch of Dacron or bovine pericardium

Mitral Regurgitation after MI

Mitral Regurgitation

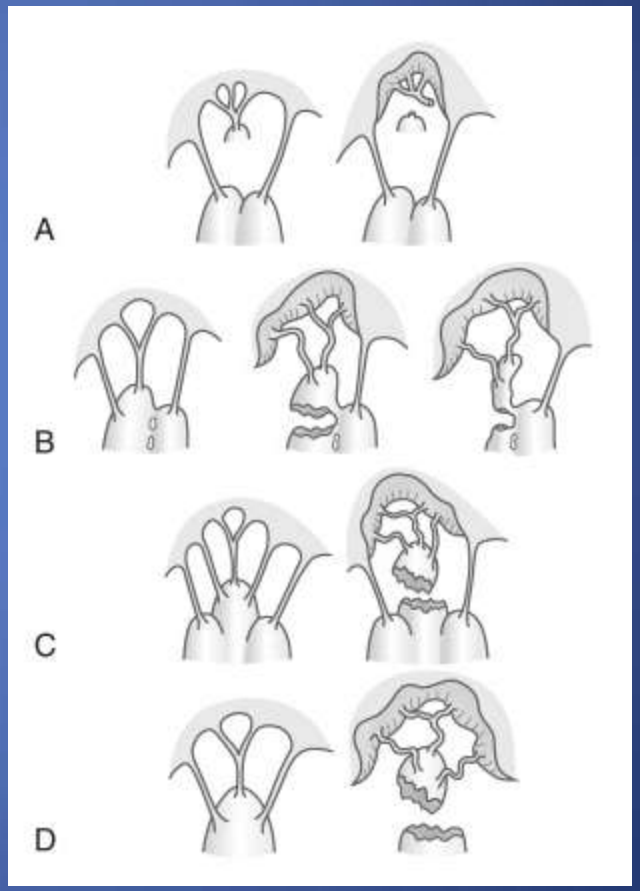
- Two causes of MR after acute MI:
  - Left ventricular dilatation or true aneurysm
  - Papillary muscle or chordal rupture
- In most patients with MR after MI, degree of regurgitation is mild and condition is transient
- In minority of patients, MR is a catastrophic complication
- Rare today due to evolution of medical and interventional reperfusion strategies

Mitral Regurgitation

- In many patients, extent of infarction is limited
  - Shock plus LV failure is the consequence of MR (not primary pump failure).
- Papillary muscle dysfunction arises from:
  - Ischemia or necrosis
  - Underlying LV dysfunction resulting in an abnormal alignment of the papillary muscle and chordae

Mitral Regurgitation

- Rupture of papillary muscle may be:
  - Partial (occurring at one of the muscle heads)
  - Complete
  - Occurs 2-7 days after MI in patients w/small infarcts and single vessel CAD



Mitral Regurgitation

- Rupture of posterior-medial papillary muscle
  - Blood supply from PDA
  - Occurs 6 to 12 times more commonly than rupture of anterolateral muscle
    - Dual blood supply from LAD and LCx arteries

Mitral Regurgitation

- Diagnosis → High index of suspicion:
  - Pulmonary edema in patient with MI (i.e. inferior) and preserved LV function → acute MR
  - Cardiogenic shock & new systolic murmur
    - Systolic murmur (mid, late, or holosystolic) of MR is not usually impressive
      - Early equalization of pressures of LV and LA results in a soft, short, and indistinct murmur.

Mitral Regurgitation

- ECHO
  - Mainstay of diagnosis
  - Differentiates condition from VSD and global myocardial dysfunction
  - Firmly established diagnosis if flail leaflet or belly of papillary muscle is identified by ECHO.
- Lack of a step up in venous oxygen saturation → distinguishes it from VSD

Mitral Regurgitation

- Acute MR due to rupture of papillary muscle (partial or complete) is an emergency!
  - Pts treated medically initially stabilize but rapidly decline

Mitral Regurgitation

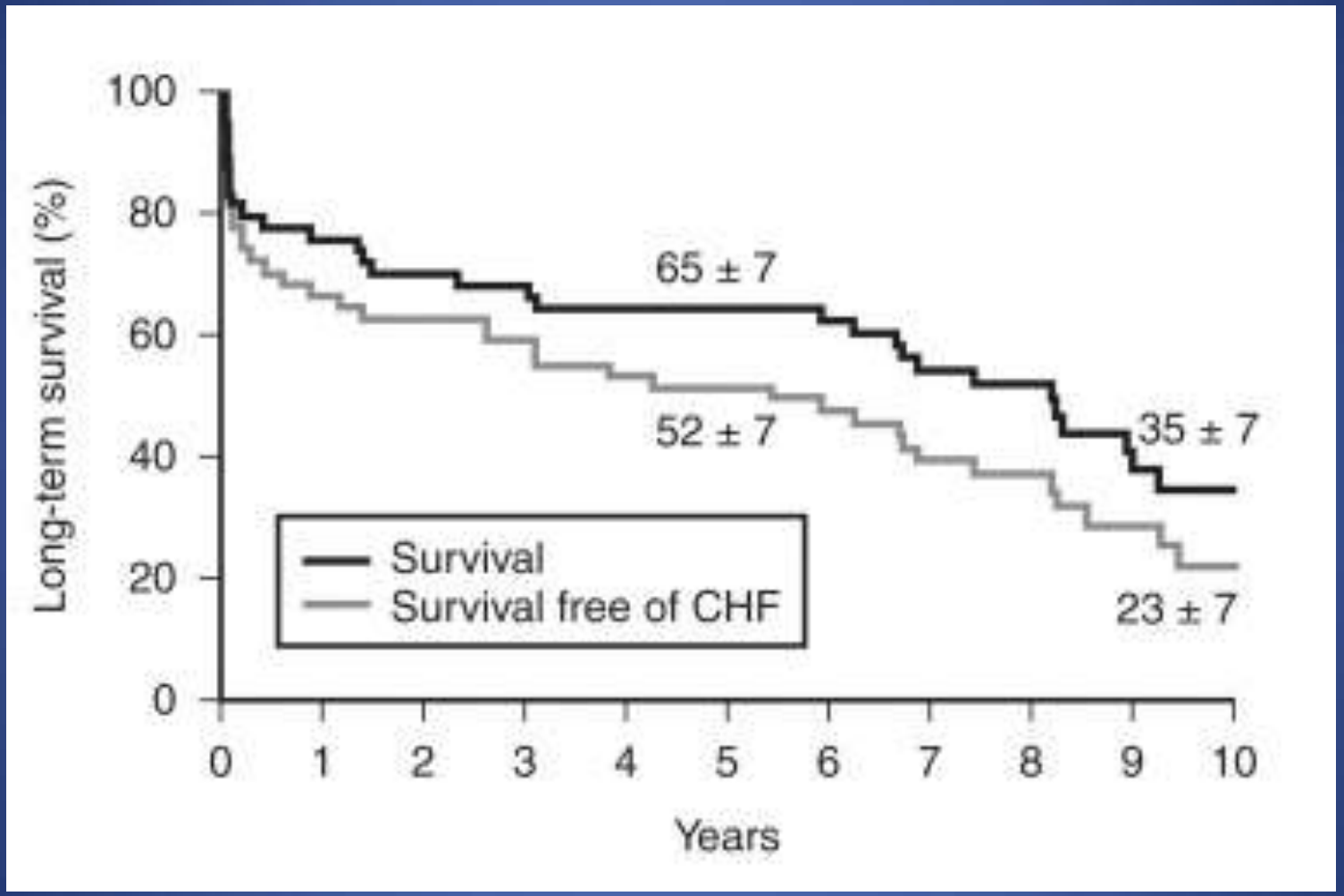
- Treatment
  - IABP for temporary hemodynamic stabilization
  - Diuretics
  - Afterload reduction
  - Maintenance of oxygenation (intubation & mechanical ventilation)
  - OR for mitral valve replacement and cardiac revascularization
    - Delay only for optimizing hemodynamics and invasive monitoring

Mitral Regurgitation

- Surgery options:
  - Mitral valve replacement (and revascularization)
    - Perioperative mortality: 20%
    - Survivors have better long-term survival compared with patients with type IIIb ischemic MR (with long-standing ventricular remodeling who undergo mitral valve surgery)

Mitral Regurgitation

- Surgery options
  - Mitral valve repair with re-implantation of papillary muscle into adjacent non-ischemic head or ventricle
    - Rarely done
    - Infarcted head must be able to hold sutures, placed at insertion of chordae
    - Re-implantation occurs into area of viable tissue


Long-term survival (%)
100
80
60
40
20
0
65 ± 7
52 ± 7
35 ± 7
23 ± 7
Survival
Survival free of CHF
0
1
2
3
4
5
6
7
8
9
10
Years

Pericarditis and Tamponade

Post-MI Pericarditis

- Two syndromes:
  - Early pericarditis
    - Occurs first 72-96 hours after MI
  - Delayed pericarditis (Dressler's syndrome):
    - Manifests weeks later

Early Pericarditis

- Diagnosed in 10% of patients
- (+) Pericardial friction rub on 2nd or 3rd day after admission
- Chest pain extends to back, neck, or shoulders
  - Worsened by movement and respiration
  - Relieved by sitting up and leaning forward
- EKG → elevation of ST segments of two limb leads and most of the precordial leads
  - May be masked by the EKG features of MI.

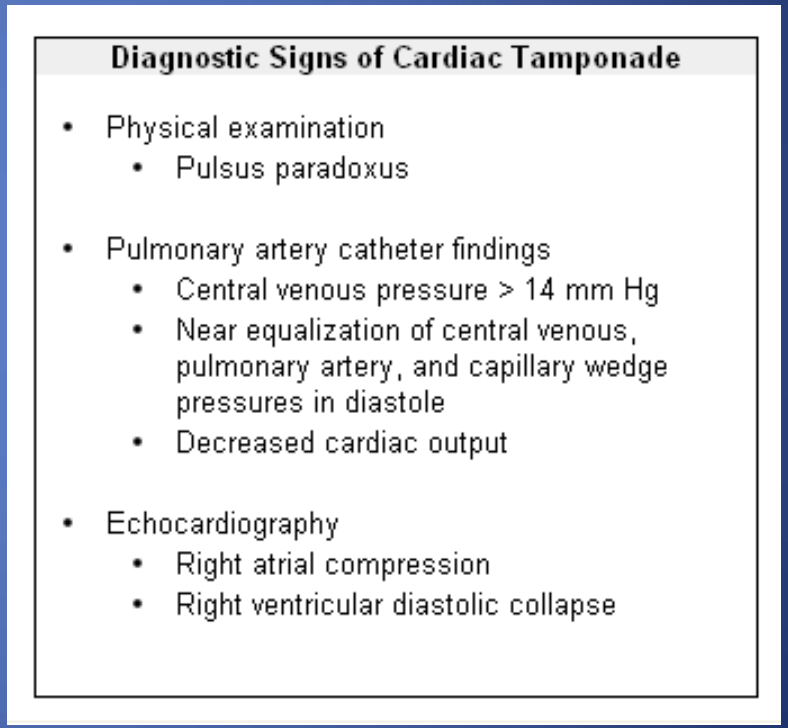
Early Pericarditis

- Treatment → symptomatic
  - NSAIDS (ASA, ibuprofen, indomethacin)

- Surveillance ECHO → evaluate for progression

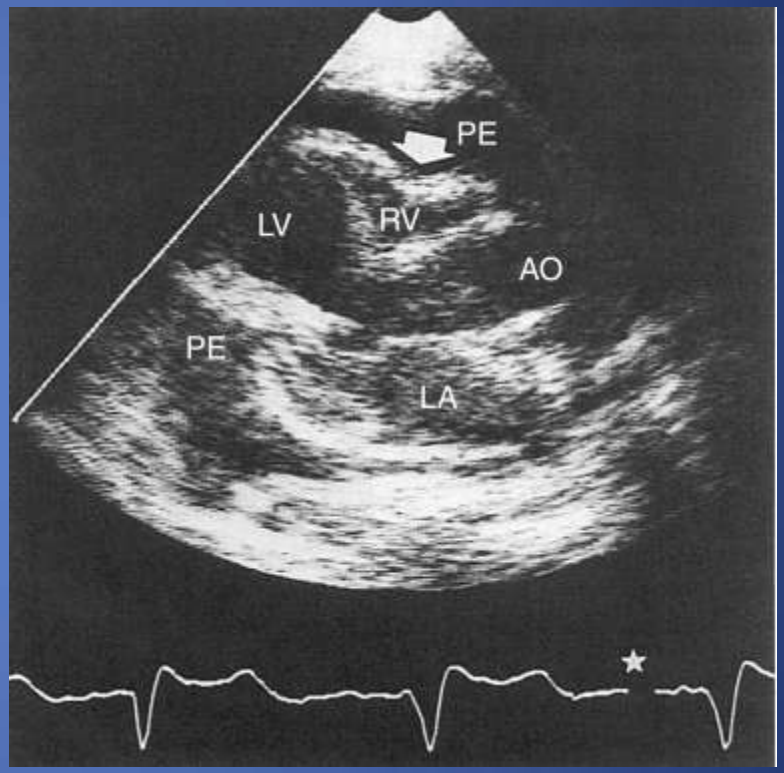
Delayed Pericarditis

- Dressler's syndrome
  - Occurs in 1-3% of patients
  - Moderate-large pericardial effusions more common than in patients with early pericarditis
- Concern exists for development of tamponade



Dressler's Syndrome with Tamponade

- Diagnosis
  - Right heart catheterization
    - CO is reduced
    - Elevation of CVP of at least 14 mm Hg
    - Equalization of CVP, PADP, and PCWP.
  - ECHO
    - Pericardial space distended with fluid
    - Atrial compression or diastolic RV collapse



- 80% of patients have pericardial effusions on ECHO in 1st 3 weeks after heart surgery
- Moderate or large pericardial effusion has been associated with 75% likelihood of developing tamponade

Dressler's Syndrome with Tamponade

- Treatment
  - Emergent pericardiocentesis +/- pigtail catheter drainage for 48 hours
  - Pericardial drainage procedure
    - Subxiphoid pericardial window
      - Ideal because local anesthesia can be used to avoid general anesthesia in the unstable patient
    - Transpleural window
      - Less suitable due to intolerance with one-lung ventilation
      - Initial pericardiocentesis should be performed to alleviate tamponade first

Conclusions

Conclusions

- Mechanical complications of acute MIs are potentially lethal
- Efforts should be made to aggressively identify complications
- Expeditious intervention may lead to improved survival among patients with complicated acute MIs

Mechanical Complications of Acute Myocardial Infarction

Chaitan K. Narsule