MI Mechanical Complication Case Based

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Mechanical Complications

- Cardiogenic Shock
- Right Ventricular Infarction
- Papillary Muscle Rupture
- Ventricular Septal Defect
- Free Wall Rupture
- Conduction Abnormalities

Right Ventricular Infarction

Right Ventricular Infarction

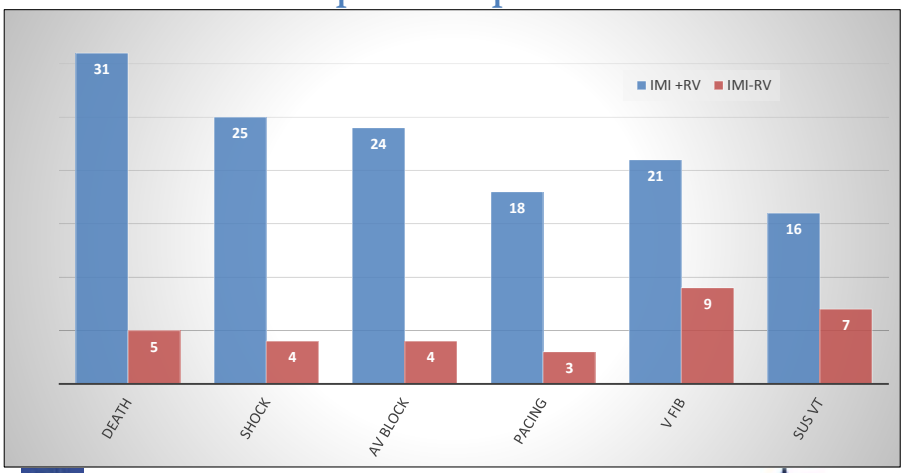
- Pathological studies evidence of RV infarction in 20 - 50% of inferior infarcts
- Significant hypotension or cardiogenic shock resulting from RV infarction occurs in approximately 10% of inferior infarcts

Clinical Findings

- Classic physical findings include: hypotension, elevated JVP, clear lung fields

Right Ventricular Infarction

In-Hospital Complications

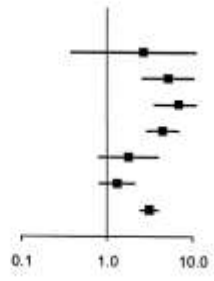


Zehender,NEJM,1993

RV infarct and implications on Mortality

A. Mortality

STUDY	RVMI+	RVMI -	Odds Ratio	95% CI
Berger, 1993	2/58	18/1052	2.67	0.37-19.44
Zehender, 1993	33/107	34090	5.19	2.56-10.52
Bueno, 1997	36/77	11/109	6.90	3.53-13.47
Bueno, 1998	64/296	30/502	4.50	2.88-7.02
Zeymer, 1998	12/169	15/353	1.79	0.78-4.08
Mehta, 2001	35/491	35/638	1.33	0.81-2.16
TOTAL	**184/1198**	**115/2747**	**3.15**	**2.44-4.07**
			Z=8.83	***P*<0.00001**



Mehta, 2001

Diagnosis

- ECG: ST segment elevation in leads V_3or V_4R (most specific test)
- Hemodynamics: elevated right atrial and RVED pressure (>12), normal to low pulmonary pressures, low (<15) PCWP, low C.O.
- Echocardiography: RV enlargement with depressed function *in setting of inferior LV hypokinesis,* ± TR

Differential Diagnosis

- Several equally serious conditions can present with similar findings both on clinical exam and diagnostic tests, especially pulmonary embolus and pericardial tamponade.
- PE, constrictive pericarditis and tamponade may have similar hemodynamics
- PE may have similar echo features
- Remember V_3, V_4 R

Principles of Management

- Stabilization and reperfusion are the hallmarks of treatment. If patients can survive the initial 2 -5 days, RV function typically improves.
- Hemodynamic monitoring and fluid administration to achieve PAWP of 15 -18mm Hg. If patients do not respond with an increase in C.O., dobutamine should be added. IABP may be necessary in some cases

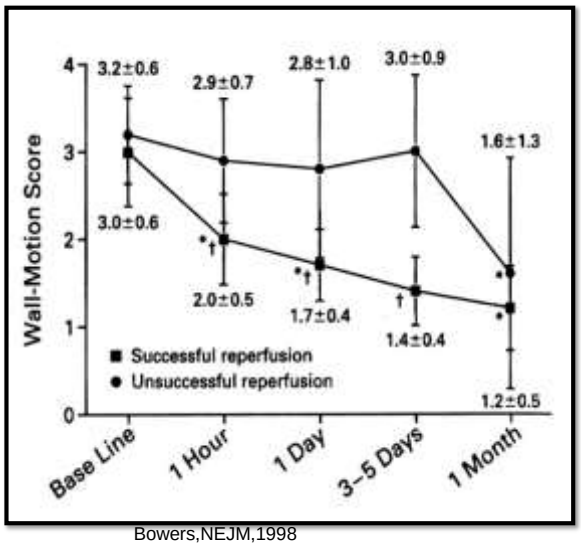
Principles of Management

- Avoid “pushing” fluids beyond above parameters. RV overdistention can RV MVO_2 and actually decrease C.O. by increasing intrapericardial pressure and limiting LV filling
- Maintenance of AV synchrony is important to maintain RV filling.

Principles of Management

- Reperfusion therapy, particularly when patency of RV branches is achieved can dramatically restore RV function and overall hemodynamic stability
- Successful reperfusion has been associated with marked reduction in mortality (2% vs 58% in 1 study) and in-hospital complications

RV Function Following Infarction



Bowers,NEJM,1998

Potential Reasons for Improvement in RV Function

- Favorable O_2 Supply / demand characteristics
- RCA - RA pressure differential
- Thin RV wall - ? direct perfusion from RV
- Low afterload
- • Greater collateral potential
  - – LAD
  - – Conus

Conclusions

- RV infarction significantly increases the morbidity and mortality associated with inferior MI
- Cardiogenic shock from RV infarction has a lower mortality than that of cardiogenic shock from LV infarction
- Reperfusion therapy greatly improves in-hospital morbidity and mortality from RV infarction
- Prognosis of hospital survivors is excellent and RV function generally improves over time

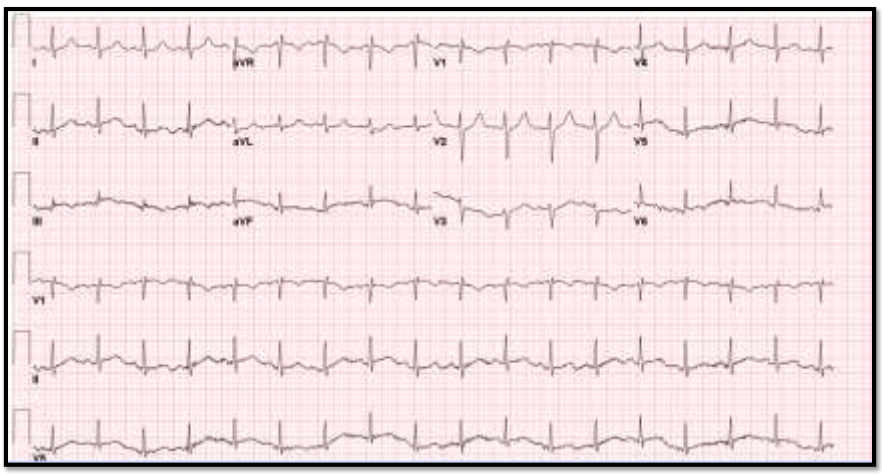
Things to Remember About RV Infarction

- Beware of pseudo RV infarcts (PE, tamponade, constriction)
- V_3/V_4R leads are the most specific for RV infarct
- Do what it takes to support hemodynamics
- Long term survival is good and RV function will improve if patient can get through the first few days

Acute Severe Mitral Regurgitation

MR patient

- 76 M with DM, CVA x2 - last 2012- able to walk with cane, but recently has not been walking due to SOB with minimal exertion for 4- 6 weeks.
- Much more SOB for last few days- even at rest. One episode of mild chest pain for ~ 30 minutes before coming to the ER. NO prior episode of chest pain. Still feels quite SOB.

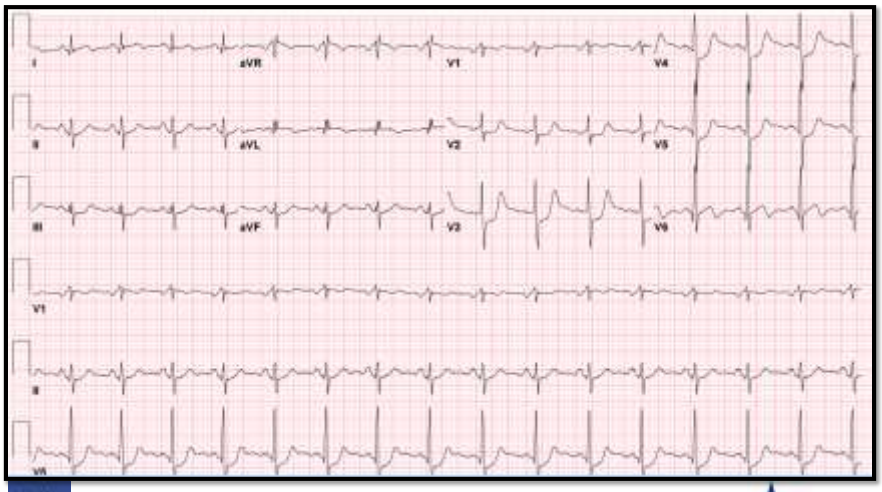


To OR

- Patient went to OR that night with LIMA to LAD and Mitral valve replacement
- Discharged but ended up in a nursing home for rehab

MR patient 2

- 66 WM with acute cardiogenic shock s/p MI
- Pt has history of dm, htn, hld presented with 3 days of CP and SOB. Also left shoulder pain, upper back pain. Presented to the ER and diagnosed with posterior MI, taken urgently to cath lab



Before the Cardiac Cath

- Patient did not look right
- HR 100-110
- SBP 90mmHg
- Pale
- Diaphoretic
- No murmur

Outcome

- Initially IABP, then Tandem Heart
- Stabilized
- Surgery next morning
- Doing well for 3 days
- Sudden drop in BP
- Opened bedside
- Myocardial Rupture

Incidence and Etiology

- Uncommon, <1% of total infarcts, but 5-10% of patients with cardiogenic shock
- Papillary muscle dysfunction
- Partial or complete tear of the papillary muscle
- Almost always associated with inferior infarction (~90%)
- Posterior papillary muscle usually responsible due to its single blood supply from the dominant coronary artery

Papillary Muscle Rupture vs Dysfunction

	Rupture N=31	Dysfunction N=16	p value
Age	67± 7	60±8	<0.005
Male	79%	69%	ns
Hypertension	58%	50%	ns
DM	7%	38%	<0.005
Prior MI or Angina	26%	50%	<0.002
Diagnosis to surgery (days)	14	45	<0.002

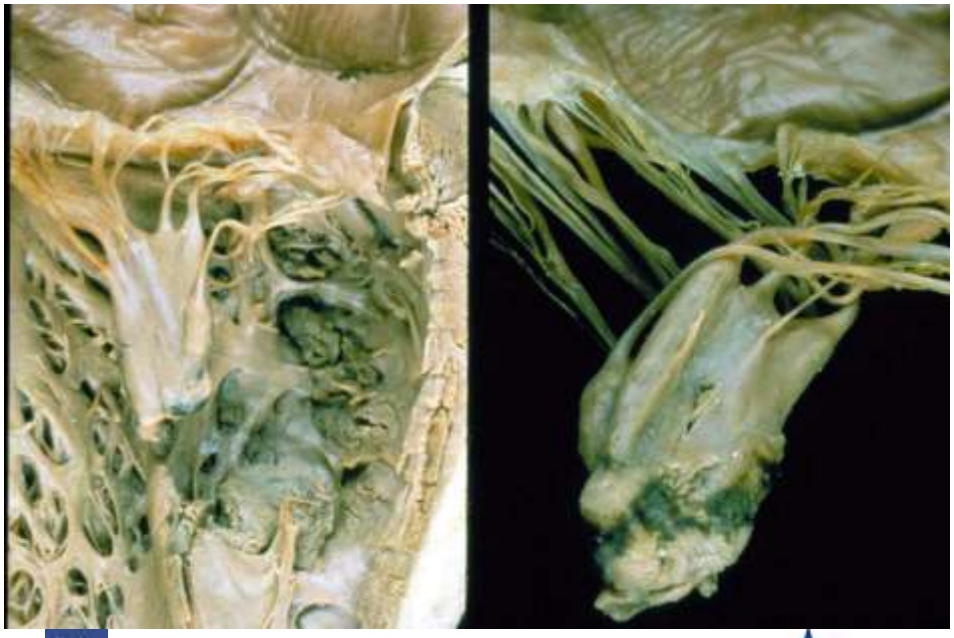
Calvo, EHJ,1997

Acute Mitral Regurgitation

Papillary Muscle Rupture vs Dysfunction

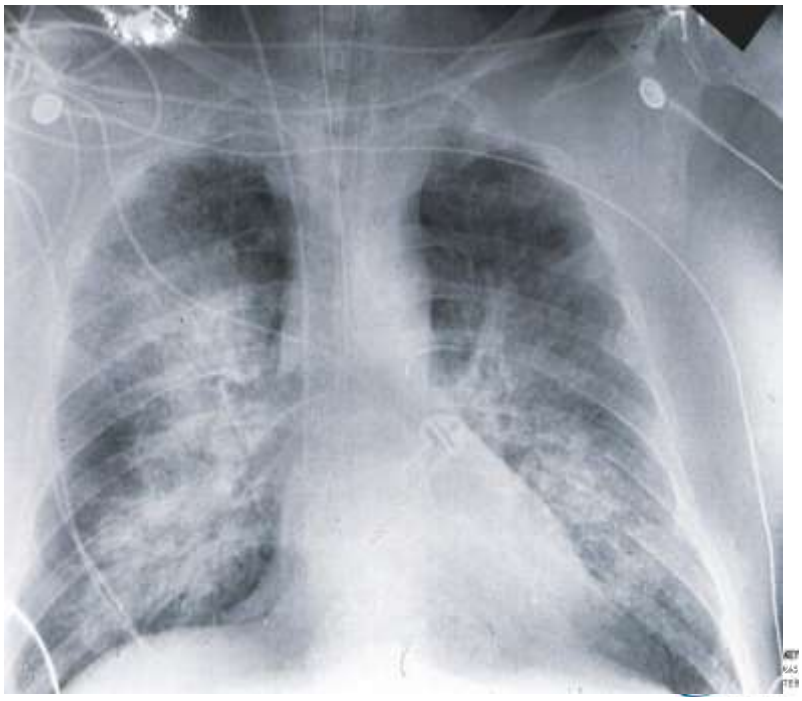
	Rupture	Dysfunction	p value
1 vessel	44%	13%	<0.05
2 vessel	32%	53%	ns
3 vessel	23%	33%	ns
EF	61±14%	46±15%	<0.03
Mortality in hosp	46%	47%	ns
Mortality f/u	15%	13%	ns

Calvo, EHJ,1997



Papillary Muscle Rupture - Clinical Presentation

- Rupture occurs primarily within the first week following infarction (>75% of cases)
- Acute pulmonary edema is the most common presentation. Over 1/2 of patients in Mayo series had cardiogenic shock
- Murmur of MR variable, may be minimal or absent in low output states (18% of Mayo series had no murmur)
- Apical thrill <u>rarely</u> present (in contrast to VSD)

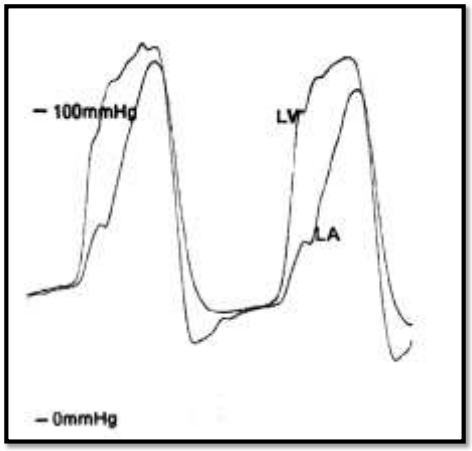


Papillary Muscle Rupture - Diagnosis

- Echocardiography is the diagnostic test of choice. If severe MR is found TEE should be performed to optimally visualize papillary muscles.
- Right heart catheterization typically shows large “V” waves on the PAWP tracing
- Differential diagnosis: VSD, infarct extension with cardiogenic shock

Papillary Muscle Rupture - Diagnosis

LV, LA pressure in PM rupture



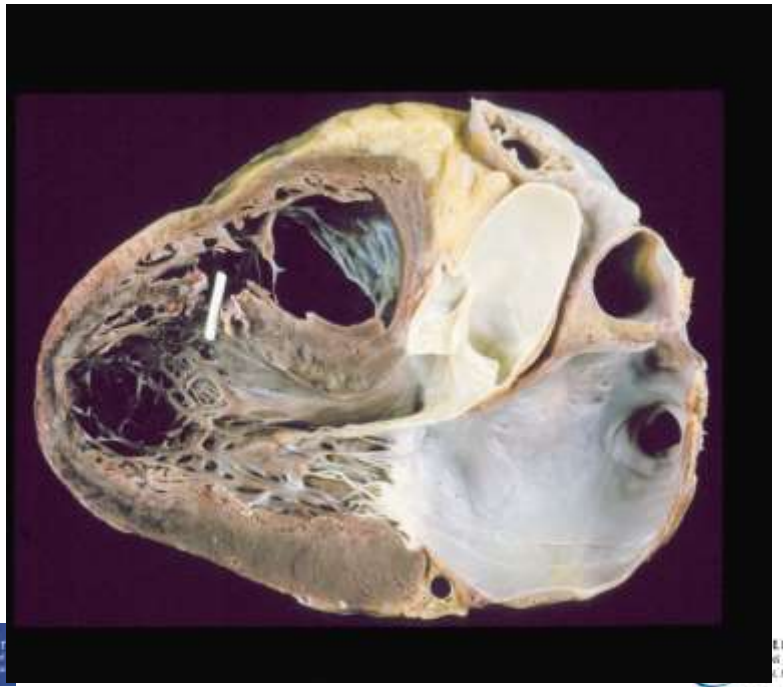
Management

- Majority of patients require mechanical ventilation
- IABP, nitroprusside and / or dobutamine for acute stabilization
- Emergent coronary angiography
- Mitral valve repair / replacement + CABG **ASAP!!** Surgical delay = increased mortality

Things to Remember About Acute, Severe MR

- Associated with inferior infarcts (90%)
- Posteromedial Papillary muscle usually involved
- High % single vessel disease, good EF
- Murmur may not be impressive
- Pulmonary edema, Cardiogenic shock most common presentation
- Echo, TEE best diagnostic test
- Early surgery with CABG
- Excellent long term survival in hospital survivors

Postinfarction Ventricular Septal Defect



Ventricular Septal Defect

Incidence

• Uncommon, <1% of total infarcts, but 2-5% of patients with cardiogenic shock

Anatomic Features

• ~ 55% due to inferior infarction, ~ 45% due to anterior infarction
• Inferior infarct VSD's are located in the posterobasal region of the septum, anterior VSD's in the apical septum
• Conduction abnormalities common (~1/3 in 1 series)

Ventricular Septal Defect

Patient Characteristics

- Mean age >65 in virtually all series
- Hypertension present in ≥50% of patients
- Typically first infarct, most patients have no antecedent angina
- High percentage of single vessel disease
- VSD usually occurs within the first week after MI, approximately 50% in the first 48 hours

Ventricular Septal Defect

Clinical Presentation

- Like acute MR most patients develop acute onset of biventricular failure or cardiogenic shock (~ 50/50)
- Classically, patients have a new holosystolic murmur and a precordial thrill
- Magnitude of L → R shunt (and characteristics of murmur) inversely proportional to size of infarct and directly related to residual LV function

Ventricular Septal Defect

Diagnosis and Management

- Echo/Doppler is the best diagnostic tool
- "Step up" in RV saturation characteristic. Large "V" waves often seen on PAWP tracing - can be confused with MR
- IABP, dobutamine, nitroprusside for acute stabilization
- Mortality 100% without surgery
- Timing of surgery remains somewhat controversial

Ventricular Septal Defect

Long Term Outcome

- Hospital survivors have very favorable long term outcome.
- 1 year survival rates are approximately 70%
- 7-10 actuarial survival 60 - 65%
- Most patients NYHA Class 1 or 2

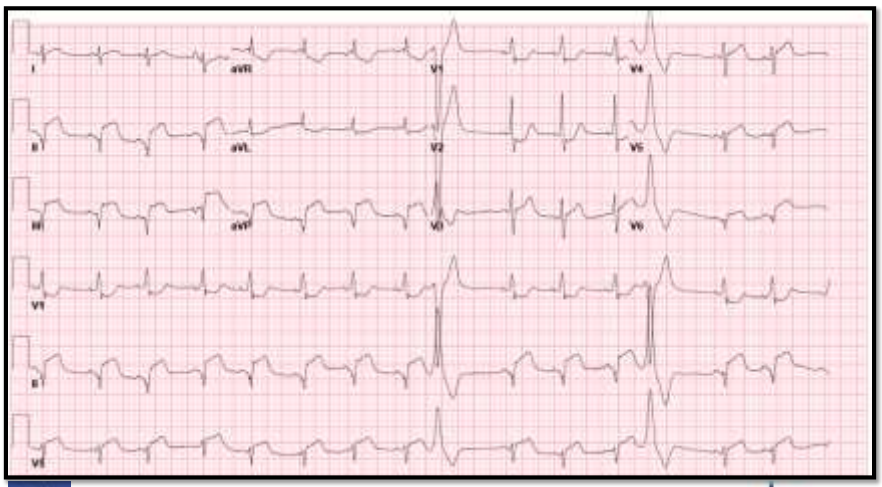
Ventricular Septal Defect

Things to Remember About Postinfarction VSD's

- Approximate equal distribution between anterior and inferior infarcts
- High percentage of single vessel disease, first time infarcts
- Similar presentation to acute MR but pulmonary edema less prominent
- Degree of shunt inversely proportional to size of infarct
- Right heart cath data can be misleading → Echo

Postinfarction Free Wall Rupture

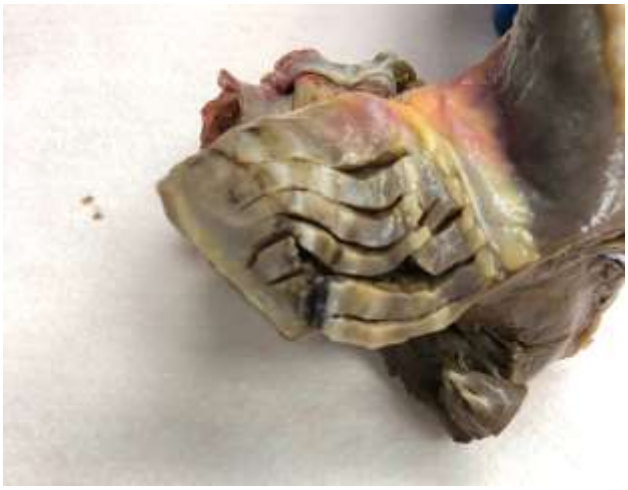
- 68 year old man with history of hypertension, diabetes, smoking who was brought to the ER by the EMS after being found lethargic

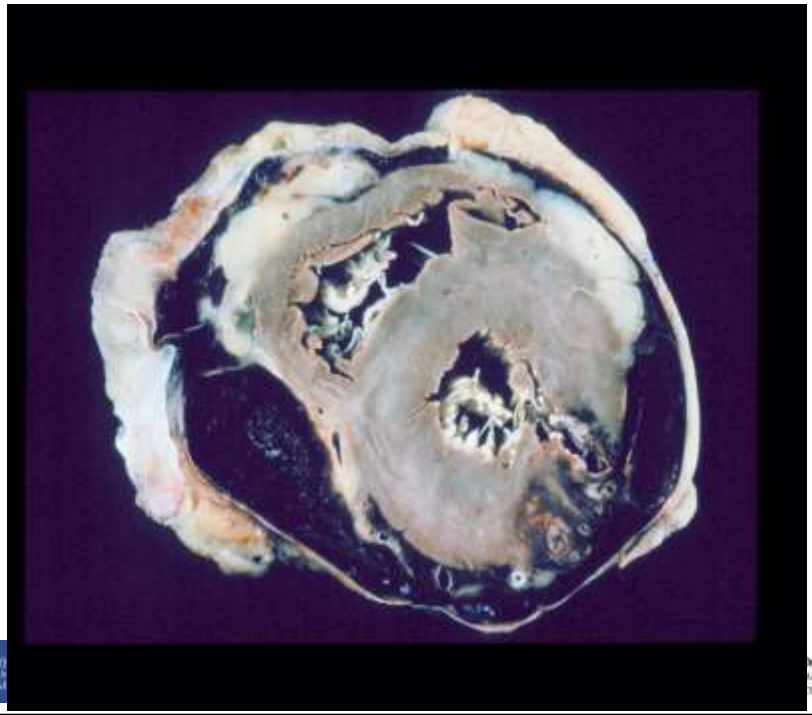


- Called perfusionist
- Placed ECMO
- CV surgery

Autopsy

- **Acute/subacute myocardial infarction of the posterior left ventricle with wall rupture**
- Hemopericardium (150ml) with clot formation
- Cardiomegaly (453g) with biventricular hypertrophy
- Coronary artery atherosclerosis, moderate, s/p stent placement
- Small aneurysms of iliac arteries





Free Wall Rupture

- "Classic" patient: elderly (>70) female with hypertension
- Usually presents as a catastrophic event - EMD due to tamponade. Syncope and shock also common.
- Time course for rupture, prevalence of single vessel disease similar to papillary muscle rupture (PMR) and VSD
- Point of rupture typically at the juncture of normal and infarcted myocardium
- Unlike PMR and VSD, circumflex is often culprit vessel (~ 40% in 1 series)

Free Wall Rupture

- Primary PTCA and early thrombolysis may reduce the incidence of rupture
- Late thrombolysis may increase the incidence of rupture
- *Rarely*, rupture is a subacute process. With prompt diagnosis and surgery these patients may be salvageable
- Echo is the diagnostic modality of choice. Any pericardial effusion in a patient with sudden hemodynamic compromise should suggest the diagnosis. Effusions with echo dense structures (clot) characteristic

Free Wall Rupture

- Mortality in patients who make it to surgery: 33%
- Long term outlook for surgical survivors is good: 13/16 alive (mean f/u 30 mo), 11 NYHA class 1

(Purcaro, et al, AJC, 1997)

Thank you