AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Managing Complications of Acute Myocardial Infarction

Patients with myocardial infarction often develop complications. This course reviews the management for the patient in cardiogenic shock, with persistent or recurrent chest pain, with dysrhythmia, or with other complications. The latest indications and contraindications for pacemakers, balloon pumps, and pharmacologic therapy are reviewed.

- Describe the complications of acute myocardial infarction and when to anticipate them.
- Discuss how to treat common dysrhythmias, cardiogenic shock, and persistent chest pain.
- Review the indications and appropriateness of various medical interventions and their clinical effects.

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**Wyeth-Ayerst: Speaker*

FACULTY

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COMPLICATIONS OF MYOCARDIAL INFARCTION

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BRIEF DESCRIPTION OF THE COURSE:

Each year approximately 1.5 million Americans suffer an acute myocardial infarction. As many as 500,000 of these patients will die, 250,000 within the first hour. Most of these deaths are the direct result of pathophysiologic changes which occur as a result of the myocardial infarction. Many more patients, who do not die, suffer from complications of myocardial infarction. These patients require prompt recognition of their condition and aggressive management in order to prevent unnecessary morbidity and mortality.

This course will discuss common complications of myocardial infarction. It will describe the clinical presentation and consequences of each complication, as well as the generally accepted pharmacological and nonpharmacologic modalities for managing such complications.

Information will also be presented regarding the use of intra-aortic balloon pump and temporary pacemaker in the setting of acute myocardial infarction.

COMPLICATIONS OF MYOCARDIAL INFARCTION

Jorge A. Martinez, MD, JD

I. VASCULAR

POST-MYOCARDIAL INFARCTION ANGINA:
Facts: **Post-MI angina is result of occlusion of patent branch of coronary vessel, reocclusion of recanalized culprit vessel, or coronary spasm**
◆ **Must be differentiated from pulmonary embolus, pneumonitis, & pericarditis**
◆ **20-30% of MI's develop post-infarction angina without new MI**
◆ **Incidence of post-infarction angina not decreased with thrombolytics, however, lower incidence in patients undergoing PTCA**
◆ **Higher post-MI morbidity and mortality (especially if obvious EKG changes or hemodynamic instability)**
◆ **If recurrent cardiac ischemia causes extension of original infarct or infarction of new myocardial zone it is termed "recurrent myocardial infarction" (RMI)**
◆ **RMI occurs within 6 weeks in 5-20% of post-MI patients**
◆ **RMI more common in obese females, nontransmural MI's, DM, patients with prior MI's, and early peaking CPK-MB's**
◆ **RMI's have higher incidence of in-hospital complications and morbidity because larger mass of compromised myocardium**
◆ **Distinction of RMI from noncardiac chest pain complicated because of EKG changes from original MI. However, new ST elevations or Q waves on EKG suggest RMI. Troponin remains elevated for 7-10 days, therefore not reliable in diagnosing RMI developing within 7 days of original MI. If RMI > 24 hrs after initial MI, CPK-MB usually re-elevates > 50%**
◆ **Distinguish RMI from postinfarction pericarditis by 1) response to NTG, 2) appearance of new ST segment and T wave changes with reciprocal changes, 3) typical pattern of T wave changes in consistent with those in pericarditis**
Management: **1) repeat EKG, if ST elevation-consider thrombolysis; 2) initiate intravenous nitroglycerin and beta blockers (lower heart rate to 60 bpm; 3) consider**

urgent PTCA or CABG with hypotension, CHF, or ventricular arrhythmias; 4) utilize intra-aortic balloon pump to stabilize while awaiting revascularization

II. ELECTRICAL

A. *TACHYARRHYTHMIAS:*

SUPRAVENTRICULAR TACHYCARDIAS

1) *Sinus tachycardia*
★ Occurs in 25% of MI's
★ Due to anxiety, metabolic acidosis, hypokalemia, hypomagnesemia, hypoxia, LV dysfunction, increased sympathomimetic tone
★ Treat underlying cause (best to get HR < 70 bpm; remember: SV x HR = CO)

2) *Atrial fibrillation:*
★ Occurs in 10-15% of MI's, usually within 1st 24 hrs
★ More common in large MI's, anterior MI's, inferior MI's with proximal RCA occlusion and involvement of SA nodal artery, CHF, complex ventricular arrhythmias, advanced AV block, atrial infarction, and pericarditis
★ Early onset=atrial ischemia, late onset=atrial distension from MR
★ Decreased incidence in patients receiving thrombolytic therapy

♥♥ Recommendations for Treatment of Atrial Fibrillation (JACC 1996;28(5)@1363)♥♥
***Class I*: 1) Electrical cardioversion in patients with severe hemodynamic compromise or intractable ischemia: begin with 100 J, then 200 to 300 J, then 360 J; 2) Rapid digitalization to slow rapid ventricular response and improve LV function (Dose= 8-15 mcg/kg with ½ dose given initially and ½ after 4 hrs) 3) Intravenous B-blocker to slow rapid ventricular response in patients without clinical LV dysfunction, bronchospastic disease, or AV block**
[B-BLOCKER *AGENTS*: 1) Atenolol:2.5 - 5.0 mg over 2 min, Max dose=10 mg in 10-15 min; 2) Metoprolol: 2.5-5.0 mg q 2 to 5 min, Max dose=15 mg over 10-15 min;

Remember: EKG, BP, HR must be monitored; stop B-blockers if BP falls below 100 mm Hg or HR < 50 bpm)]
4) Heparin should be given
***Class IIa*: 1) Either diltiazem or verapamil intravenously to slow rapid ventricular response if B-blocking agent are contraindicated or ineffective;**
***Intravenous verapamil (5-10 mg [0.075 to 0.15 mg/kg] given over 2 min; repeat dose 30 minutes later;**
***Intravenous diltiazem (20 mg [0.25 mg/kg] over 2 min followed by infusion of 10 mg/min, if no response after 15 minutes give 2nd bolus (25 mg [0.35 mg/kg] over 2 minutes, maintenance infusion of 10-15 mg/hr**
◆Ca channel blockers not recommended as 1st line agents because of intense (-) inotropic effect◆

3) *Atrial flutter*
★ Less common than Afib
★ Rapid conversion with electrical cardioversion
★ Use of B-blockers or Ca channel blockers to slow ventricular rate less effective than in Afib
★ May require atrial overdrive pacer to convert

VENTRICULAR TACHYARRHYTHMIAS

1) *Ventricular premature contractions*(PVC's):
✶ Common in MI within 1st 24-36 hrs
✶ Later occurrence may be sign of ventricular dysfunction or irritability
✶ Only require suppression if symptomatic, > 5/min, or runs of bigeminy or V tach
✶ Prophylactic lidocaine not recommended

2) *Accelerated Idioventricular rhythm (AIVR)*:
✶ Occurs in 40% of MI's, usually within 12 hrs of MI
✶ Commonly known as "reperfusion arrhythmia" when thrombolytics utilized
✶ Considered benign unless rate > 110-120 bpm; [If > 110-120 is considered automatic V tach (NOT BENIGN) = treat with lidocaine]

3) *Ventricular tachycardia (VT)*
★ Occurs in 15% of MI's, usually within 1st 48 hrs
★ Ventricular rate usually 140-200 bpm
★ If sustained (>30 seconds) commonly degenerates to

ventricular fibrillation

AHA/ACC recommendations for treatment of VT (JACC 1996;28(5)@1364-1366)

Class I: **1) Sustained polymorphic VT (> 30 seconds or hemodynamic compromise)-unsynchronized shock of 200 J, second shock @ 200-300 J, third shock @ 360 J**
2) Sustained monomorphic VT associated with angina, pulmonary edema, or hypotension (BP<90 mm Hg)-treat with synchronized electric shock of 100 J initially with increasing energy as needed
3) Sustained monomorphic VT not associated with angina, pulmonary edema, or hypotension (BP<90 mm Hg)-treat with one of the following:

a. Lidocaine: Bolus=1.0-1.5 mg/kg, supplemental boluses of 0.5-0.75 mg/kg q 5-10 minutes, Max dose=3 mg/kg loading dose. Loading followed by intravenous infusion of 2-4 mg/min (30-50 mcg/kg/min) (Lower doses used in elderly, CHF, or liver disease)

b) Procainamide: Bolus=20-30 mg/min loading infusion, up to 12-17 mg/kg; follow with infusion of 1-4 mg/min; *stop administration when* I)obtain desired effect, II) develop hypotension/CHF, III) prolong QRS, IV) 1 gram administered (Use lower doses in renal disease)

c) Amiodarone: Bolus=150 mg over 10 min followed by infusion of 1.0 mg/min for 6 hrs, then maintenance infusion of 0.5 mg/min

d) Synchronized cardioversion starting @ 50 J (with anesthesia)

4) Refractory VT: related to uncontrolled ischemia and increased sympathomimetic tone. *Treatment:* reduce cardiac ischemia with NTG, O2, & Drugs (B-blockers/IV amiodarone) & IABP and revascularization (PTCA/CABG)

4) *Ventricular fibrillation (VF):*
★ Occurs in 8% of MI's; associated with poorer outcome
★ More frequent in large Q wave MI's
★ May occur without previous arrhythmias
★ Primary VF= electrical instability with acute ischemia (3-5% of VF, occurs within 1st 4 hrs of MI), less incidence of occurrence with B-blockers
★ Secondary VF= secondary to CHF, hypoxia, continued cardiac ischemia, metabolic acidosis, electrolyte disturbance

AHA/ACC Recommendations for treating VF:(JACC 1996;28(5)@ 1364)
***Class I*: Defibrillation with unsynchronized electrical shock=1st shock-200J;2nd shock-200-300J;3rd shock-360J**
Adjunctive measures: 1) epinephrine @1mg IVP, 2) lidocaine @ 1.5 mg/kg bolus, 3) bretylium @ 5-10 mg/kg, 4) IV amiodarone @ 150 mg bolus

B. *BRADYARRHYTHMIAS*
Anatomy:
***SA node=55% from RCA, 45% from Circumflex**
***AV node=90% from RCA, 10% from Circumflex**
***Main Left Bundle = dual supply from RCA and Circumflex**
***Right Bundle & Left Anterior Fascicle of Left Bundle = Septal perforators of LAD**
***Left Posterior Fascicle of Left Bundle=Circumflex**

1) *Sinus bradycardia/sinus pauses*
_ Occur in 30-40% of MI's, especially in IWMI and reperfusion (Bezold-Jarish reflex = increased parasympathetic [vagal] tone); Treat with atropine or temporary pacemaker

***Treatment with Atropine:* (JACC 1996; 28(5) @ 1366)**
Class I: **1) Symptomatic sinus bradycardia (generally, heart rate < 50bpm associated with hypotension, ischemia, or escape ventricular arrhythmia).**
2) Ventricular asystole.
3) Symptomatic AV block occurring at the AV nodal level (second-degree type I or third degree with a narrow-complex escape rhythm).
Class III:* *(no benefit)
1) AV block occurring at an infranodal level (usually associated with anterior MI with a wide-complex escape rhythm).
2) Asymptomatic sinus bradycardia

2) *Atrioventricular node blocks:* The increased mortality associated with AV blocks is usually related to extent of myocardial damage rather than consequence of the AV block

♥ *1st-degree AV block*:* Occurs in 4-13% of MI's; * Transient = observe; * Common in IWMI

♥ *2nd-degree AV block:* * Occurs in 3-10% of MI's, usually within 1st 24 hrs

a) Type I (Weinkebach)[prolonged PR intervals with loss of beat]: occurs most often in IWMI, often responds to atropine (if HR<45 bpm or hypotension), usually narrow QRS complex, observation and avoidance of medications which prolong AV block

b) Type II [intermittent dropped ventricular beats; PR interval remains constant]: more common in anterior MI; implies extensive infranodal involvement, usually associated with BBB, wide QRS complexes common, 35% progress to 3rd degree AV block; temporary transvenous pacemaker suggested

♥ *3rd-degree AV block*

a) Inferior/posterior MI: 3-7% of IWMI's progress to 3rd degree AV block after 2nd degree AV block, usually from transient ischemia to AV node, 2.7x increase in-hospital mortality, responsive to atropine or aminophylline, normal conduction returns within 3-7 days, observe carefully with transcutaneous pacemaker on standby

b) Anterior MI: indicates extensive damage of septum, poor prognosis from pump failure or VF, most patients require temporary transvenous pacemaker

AV conduction disturbances in IW or posterior MI are due to enhanced vagal activity = transient, more responsive to atropine, and less dangerous

AV conduction disturbances in anterior MI = more dangerous because of extensive ischemia to septum and conducting system (remember LAD supplies 2/3 of septum)

3) *Bundle Branch Blocks:*

★ New bundle branch block signifies extensive myocardial damage & higher risk of complications

★ Unifasicular blocks:

LAHB = more common(5% of MI's), usually benign course

LPHB = rare, signifies extensive damage & coronary involvement as the L post fascicle has dual blood supply

★ Bundle branch blocks:

Complete LBBB or RBBB in 10-15% of MI's; 2/3 are RBBB, new onset complete LBBB or RBBB requires temporary pacemaker however most common cause of death=pump failure, VT, or VF

III. MECHANICAL

1) *LV Failure/Cardiogenic Shock*

***Facts*:**

★ Most important predictor of post-MI mortality

★ Most common cause = systolic dysfunction decreased ejection fraction & cardiac output

★ May be caused by papillary muscle dsyfunction[acute MR], VSD, free wall rupture, or RVMI[relative hypovolemia]

***Clinical*: acute SOB, dyspnea, tachycardia (HR x SV=CO), pulmonary rales, S3/S4, JVD, decreases O2 Sats, elevated pCO2/decreased pO2; decreased urinary output**

★ CARDIOGENIC SHOCK = 5-15% of MI's (necrosis of >40% of ventricle)

Definition of cardiogenic shock: Systolic BP<80mm Hg & poor peripheral perfusion (cool extremities, low urinary output [< 30cc/hr], alteration of mental status, metabolic acidosis) & elevated LVED pressures

➢More common in elderly patients, prior MI & new anterior MI; 90% treated medically die

➢Revascularization reduces mortality by 40-60%

Diagnosis and Management:

★ Must be aggressive: Detailed history and physical examination aimed at making diagnosis= Review patient's medications, EKG/cardiac monitor; intravenous line; arterial blood gases, ensure oxygenation & ventilation (intubate if necessary), ECHO, coronary angiography

★ Begin medical treatment aimed at initiating medical stabilization while preparing for revascularization (PTCA/CABG)

★ THE MANAGEMENT OF CARDIOGENIC SHOCK ★

1) Reduce Preload and Afterload

a) REDUCE PRELOAD: Diuretics (reduce myocardial wall stress by decreasing intraventricular volume; reduces pulmonary vascular congestion, augments renal unloading of Na and water)=furosemide (loop diuretic) @ 10-40mg IV q 3-4 hrs (watch for hypovolemia &

hypokalemia)

b) REDUCE AFTERLOAD: Nitroglycerin (reduces myocardial wall stress by enhancing ventricular emptying; beneficial in VSD or acute MR to promote forward output); NTG decreases Preload and Afterload and increases coronary blood flow; *Dose*: begin at 5-10 mcg/min and titrate until improvement in symptoms or BP falls by 10-15%; do not lower systolic BP < 80 mm Hg; ACE inhibitors should be instituted once the patient is stabilized (reduces ventricular remodeling)

2) Control arrhythmias (see Section II)

3) Utilize inotropes as needed:

***A) Cardiac* inotropes:**

I) Dopamine: *Dose*: 2mcg/kgm/min (dopaminergic-increased flow to kidneys and GI tract); 5-10 mcg/kg/min (mainly beta-increased cardiac output)= peripheral dilation, increased HR and SV;
> 10 mcg/kg/min (mainly alpha) =increased afterload [elevates blood pressure but increases impedance to LV ejection]; Max dose=20 mcg/kg/min

ii) Dobutamine: predominately beta=increased SV and HR, peripheral vasodilation
***Dose*: 2.5 mcg/kg/min and increase by increments of 2.5 mcg/kg/min; Max dose=30 mcg/kg/min**

iii) Norepinephrine: predominately alpha=increases SVR and afterload; *Dose* = 0.02-0.04 mcg/kg/min; Cautions: used as temporizing agent; watch for accelerated tachycardia and extravasation into soft tissues

iv) Amrinone/Milrinone: phosphodiesterase inhibitors with inotropic & vasodilator effects; PCWP & SVR;

***Amrinone dose:* 0.75 mg/kg over several minutes; maintenance of 5-10 mcg/kg/min titrated to stabilization; Max dose=10 mg/kg**

***Milrinone dose:* 50 mcg/kg over 10 minutes, maintenance of 0.375-0.75 mcg/kg/min**

(Reduce dose of inotrope if HR>110 bpm, for supraventricular or ventricular arrhythmias, or acute ST changes)

4) Mechanical support (IABP, temporary pacemaker)
Intra-aortic balloon counterpulsion-decreases afterload (enhanced LV emptying); increases diastolic perfusion pressures (enhanced coronary perfusion)[see Section IV]

2) *Free Ventricular Wall Rupture:*
***Facts*:**
- **Occurs in 1.5-8.0% of MI's, 30% within 1st 24 hrs; 90% within 1st 14 days; peak occurence 1st-4th day**
- **Causes 8-24% of post-MI mortality**
- **More common in women, > 70 y/o, hypertension**
- **Most common in 1st transmural anterior or anterolateral MI**
- **Occurs at junction between necrotic and normal myocardium**
- **7x more common in LV than RV**
- **Cause of death=acute pericardial tamponade**

***Clinical*: 1) ACUTE FREE WALL RUPTURE=abrupt onset of chest pain (usually tearing); agitation, confusion, profound shock, JVD, tachycardia, PEA; clinical evidence of pericardial tamponade**
2) PSEUDOANEURYSM (false aneurysm)=the ruptured contents of the LV cavity contained by the pericardium prevents acute pericardial tamponade
- **Pseudoaneurysm is composed of pericardial & epicardial adhesions and enclosed thrombus which form a narrow neck between the LV cavity and the pericardium**
- **Most common in inferior/posterior MI**
- **Patients usually complain of chest pain or discomfort and symptoms of acute CHF, emboli or arrhythmias**
- **CXR = cardiomegaly; EKG = persistent ST elevation; ECHO = >5mm high density pericardial effusion with evidence of pericardial tamponade, defect in myocardium; visualization of pseudoaneurysm; DOPPLER ECHO = turbulent flow through myocardial wall defect; may be seen on CT of chest**

***Management*: usually unsuccessful because of immediate hypotension, pericardial effusion/tamponade and cardiac arrest**
Reverse all anticoagulants (heparin=protamine; coumadin=vitamin K/FFP; thrombolytics= cryoprecipitate)
Perform immediate pericardiocentesis (clotting blood

diagnostic)
Cardiac resuscitation with volume & pressor support; antiarrhythmic treatment; consult CT surgery; IABP
All free wall ruptures and psuedoaneurysms must be surgically corrected. Psuedoaneurysms can wait until patient stabilized=use beta-blockers or ACE inhibitors which reduce heart rate and contractility (reduce ventricular pulsatile forces)

3). *Ventricular Septal Defect*
***Facts*:**
✶ Occurs in 2% of AMI's; 1st 24 hrs 3 weeks(peak: 3-5 days)
✶ More common in large MI's, elderly, hypertension
✶ 60% of VSD's in anterior septum (LAD supplies 2/3 of interventricular septum); 40% of VSD's in posterior septum [worse prognosis]
✶ Most patients have poor collateral circulation, multivessel disease, & major vessels involved
***Clinical*: Pathophysiology = rupture of septum→immediate L to R interventricular shunt→RV overload with increased PA flow and elevated pulmonary pressures; LV volume loss = decreased LV output decreased CO, hypotension, tissue hypoperfusion & shock**
Physical exam: acute chest pain & SOB, sudden tachycardia, hypotension/shock, harsh holosystolic murmur throughout the precordium (90%), systolic thrill (50%), accentuated P2(↑ pulmonary artery flow), JVD
ECHO = ventricular defect, dilated RV, TR; DOPPLER ECHO = increased pulmonary artery flow; L→R shunt at VSD; R HEART CATHETERIZATION = blood from SVC and pulmonary artery reveal 7-10% step-up in O2 Sats
***Management:* Temporary = 1)Treat CHF with O2, diuretics; 2) promote forward ventricular emptying with afterload reduction with nitroprusside, IV NTG, or hydralazine; 3) promote LV emptying with Inotropic agents such as dobutamine, amrinone or milrinone (cardiotonics that cause peripheral vasodilation); 4) IABP in severe hypotension or refractory to medical management. DEFINITIVE TREATMENT = Surgical**

✭ Factors affecting survival in VSD = size of defect, extent of L→R shunt, ability of R and L ventricle to compensate for volume shift

4) *Acute Mitral Regurgitation(papillary muscle rupture)*
***Facts*: occurs in 1-2% of AMI's within 2-7 days of MI**
_ Causes 5% of post-MI deaths
_ Occurs in nontransmural & transmural MI; small & large infarcts
♥♥ PAPILLARY MUSCLES: a) Anterior papillary muscle = supplied by LAD and Circumflex (rarely ruptures); b) Posterior papillary muscle= supplied by RCA (90%) or Circumflex (10%)
_ Partial papillary rupture most common
_ Posterior papillary muscle=75% of all papillary muscle ruptures
_ Complete rupture of papillary muscle = rapid death
***Clinical*: acute SOB, pulmonary edema, tachycardia, hypotension, shock, atrial arrhythmias (LA volume overload), hyperdynamic and displaced LV impulse, holosystolic murmur (may be early ejection or silent), S3 &/or S4, thrill (rare)**
***Diagnosis*: CXR = pulmonary edema (asymmetric pulmonary edema in upper lobes if MR jet streams toward pulmonary veins of R upper lobe of lung; ECHO = flail mitral leaflets, mitral regurgitation, LA enlargement; DOPPLER ECHO = regurgitant jet; R HEART CATHETERIZATION = large V waves in pulmonary artery and PCWP tracing**
***Management*: TEMPORARY = 1) promote forward ventricular emptying = afterload reduction with nitroprusside, IV NTG, or hydralazine; 2) promote LV emptying with inotropes such as dobutamine, amrinone, or milrinone; 3) atrial arrhythmias often refractory to medical management, cardiovert as necessary; 4) IABP in severe hypotension or refractory to medical management**
DEFINITIVE TREATMENT=Surgical

5) *Right Ventricular Wall MI (RVMI)*
***Facts*: seen in 30-40% of inferior MI's**
_ 5-10% with ant MI; 1-5% with no LV involvement
_ More common with proximal R coronary artery occlusion
_ Associated with higher mortality in IWMI

_ MECHANISM: 1ST:RV ischemia causes dilation of RV & increased intrapericardial pressure ALSO reduced RV systolic pressure RESULT: decreased volume to LV (decreased CO); 2ND:increased pooling of blood in

RV=shift of interventricular septum toward LV
3RD: RV diastolic and systolic dysfunction=lower gradient between RA and RV less movement of blood from RA to RV RESULT: decreased RV filling and forward RV emptying into LV
THEREFORE: Anything that _decreases Preload [RA & RV filling pressures] such as hypovolemia, diuretics, NTG, loss of AV synchrony OR _decreases RA contractility (RA infarction) OR _increases RV afterload (LV dysfunction) will decrease LV output

***Clinical*: Triad of RVMI = _hypotension _clear lung fields _elevated jugular venous pressure>10mm Hg (JVD)**
Also look for TR and R sided S3
EKG = IWMI and ST segment elevation in V1 suggest RVMI &/or ST elevation in V3R and V4R (higher incidence of major complications and 8x in-hospital mortality) [Remember: pericarditis, pulmonary embolism & ASMI can also cause ST elevation); ECHO = dilated RV with dyskinesis or akinesis, abnormal septal or atrial motion
***Management*: 1) Reperfusion therapy 2) maintain RV preload with volume loading (NS preferred) raise LVEDP to > 12 mm Hg; 3) avoid using nitrates or diuretics; 4) maintain AV synchrony (prompt cardioversion of SVT, atrial fibrillation, atrial flutter; AV sequential pacing for AV block) correct bradycardia; 5) inotropic support (if no rise in BP after fluid resuscitation); DOBUTAMINE BEST = provides inotropic support, decreases pulmonary vascular resistance and reduces RV afterload; 6) reduce left ventricular dysfunction (RV afterload) to promote RV forward output = treat CHF/cardiogenic shock; consider ACE inhibitors**

*** Suspect RVMI in patient with IWMI who becomes hypotensive when diuretics or NTG are administered***

6) *Ventricular Aneurysm*
***Facts*: seen in 5-10% of MI's**
_ More common in transmural anterior MI's
_ Mortality 6x higher than patients without aneurysm
_ Size ranges from 1-8 cm
_ 4x more common in apex and anterior wall than inferior wall
***Aneurysm formation:* Infarcted area composed of**

necrotic and fibrous tissue increased intraventricular tension stretches noncontracting infarcted myocardium dilation of infarcted segment (infarct expansion) causes aneurysm formation aneurysm thinner than myocardium aneurysm bulges with systole=decreases ventricular stroke volume; blood pools in aneurysm pericardium adheres to aneurysm and calcifies (less likely to rupture)

***Clinical*: Consequences of aneurysm formation= decreased cardiac output, hyperkinesis of contracting myocardium, intraventricular mural thrombus formation**

***Symptoms* = post MI chest pain (increased myocardial wall stress), CHF(dyskinetic segment), ventricular tachyarrhythmias, CVA/peripheral embolism, sudden death**

***Diagnosis:* EKG = persistent ST elevation at site of previous MI; ECHO/LV ANGIOGRAPHY = visualize aneurysm, visualize mural thrombosis; CXR = cardiomegaly**

_ Mural thrombi formation in 40% of anterior MI's, 60% of anterior MI's involving the apex

_ Mural thrombus formation within 48-72 hrs of MI have poor prognosis because of size of MI (shock, reinfarction, rupture or tachyarrhythmia) rather than embolic events

_ Systemic embolization in 10% with mural thrombus

***Management*:**

A) 50% reduction in thrombus with anticoagulation.

_ Anticoagulate IF = large anterior MI with or without mural thrombus, mural thrombus, large wall motion abnormalities.

_ Use heparin acutely, maintain PTT @ 1.5 to 2x normal. Then coumadin x 3-6 months (no reduction in systolic embolism noted).

_ Use aspirin in conjunction with coumadin

B) Medical management for CHF & ventricular arrhythmias.

C) If resistant to medical therapy, consider surgical intervention.

7) *Pericarditis (2 types)*

A) Postinfarction pericarditis:

*** Develops within 12 hrs to 10 days of transmural MI; usually in larger MI's**

*** 50% reduction in pericarditis in patients receiving**

thrombolytics
*** Develops in 6-20% of MI patients not receiving thrombolytics**
Clinical: **sharp & persistent chest pain; midsternal pain that radiates to shoulders, interscapular area or trapezius; pain increases with inspiration, relieved with sitting up and leaning forward; low grade fever; pericardial friction rub (within 1st 3 days of MI; may be confused with murmur of MR)**
EKG: re-elevation of ST segments, PR depression (usually in large anterior MI's), upright T waves (in contrast to inverted T waves seen in myocardial ischemia)
*** 28% of postinfarction pericarditis have pericardial effusion (more common with extensive MI)**
*** Higher incidence of complications=CHF, atrial and ventricular tachyarrhythmias**
*** Higher incidence of in-hospital and 1 year mortality**
***Management*: 1) High dose aspirin=650 mg 4-6x/day**
2) Avoid steroids or NSAID's because interfere with myocardial scarring and promote aneurysm formation
3) If receiving anticoagulant therapy-observe for signs of hemorrhagic pericarditis and pericardial tamponade

B) Dressler's Syndrome:
*** Usually within 7-11 weeks of MI (range=1-28 weeks)**
*** Occurs in 1-6% of post-MI patients**
*** *CAUSE*: Autoimmune mechanism (antimyocardial antibodies) vs viral infection**
*** Incidence has decreased dramatically with use of thrombolytics in MI; NOT SEEN in patients who are reperfused with thrombolytics [decreased infarct size and production of myocardial antigens]**
*** Diffuse nonspecific inflammatory response over entire myocardium vs pericardial inflammation over area of transmural MI (postinfarction pericarditis)**
***Clinical*: Chest pain (see postinfarction pericarditis); May have pleurisy plus constitutional symptoms=low-grade fever, malaise, weakness, myalgia, arthralgia, anorexia; pericardial friction rub, pleural rub; pericardial effusion rare (unless anticoagulants)**
*** EKG: (see postinfarction pericarditis)**
*** CXR: cardiomegaly, pleural effusion (more common on L), pulmonary infiltrates**
*** Laboratory: elevated ESR and WBC**

*** ECHO: pericardial effusion, cardiomegaly**

***Management*:**

1) Hospitalize and observe for pericardial tamponade
2) ECHO = evaluate extent and duration of pericardial effusion
3) Discontinue anticoagulants if possible
4) *Drug Therapy*: 1st choice = Aspirin @ 650mg 4-6x/day; 2nd choice = Ibuprofen @ 400 mg 4x/day (may interfere with myocardial healing and lead to ventricular remodeling and aneurysm); 3rd choice = Prednisone @ 10-20mg qod for short term (May see recurrence when NSAIDs or steroid used)
5) Perform pericardiocentesis if pericardial tamponade present= *Clinical findings:* tachycardia, distended neck veins/blunted y descent, Beck's triad (hypotension in the face of rising jugular venous pressure & quiet heart sounds); pulsus paradoxicus [drop in peak systolic BP > 20 mm Hg in inspiration], narrow pulse pressure; *FINDINGS ON ECHO* =pericardial effusion, collapsing RA &/or R ventricle during diastole

IV. THERAPEUTIC INTERVENTIONS

A. *Intra-aortic Balloon Pump (IABP)*

The IABP is a devise that is inserted in the aorta which is used to stabilize those patients who are hemodynamically unstable from refractory cardiac ischemia or mechanical complications of MI. It is routinely utilized on patients who do not respond to medical therapy and require surgical intervention (PTCA, CABG, rescue cardiac surgery) to treat the cardiac pathology.

The IABP 1) increases diastolic perfusion pressure increasing coronary blood flow and alleviating myocardial ischemia; 2) decreases afterload thereby promoting foward flow from the LV and decreases LV work & oxygen consumption.

The ideal patient for IABP is _free of aortoiliac disease &/or aortic regurgitation, _suffering their first MI, and _in cardiogenic shock that has failed pharmacologic therapy.

***Description of IABP*: The IABP is a vascular catheter and inflatable balloon which is inserted into the femoral artery to provide circulatory assistance in**

patients with cardiac insufficiency.

The IABP is placed percutaneously through the femoral artery and advanced into the descending aorta under fluoroscopy. The tip of the IABP is seated just below the ostium of the L subclavian artery. The IABP is then attached to a computerized pump, which coordinates inflation and deflation of the balloon with the cardiac cycle.

The balloon deflates at the onset of ventricular systole (isometric contraction-before the aortic valve opens). Deflation of the balloon at this time 1) decreases impedance to LV emptying, and 2) decreases aortic systolic pressure (afterload). The decrease in afterload (systolic BP drops by 10-15%) improves cardiac output by approximately 15%. In addition, decreased resistance to LV ejection decreases myocardial work and oxygen consumption. The LV end-diastolic pressure is also reduced which leads to a reduction in the diastolic perfusion gradient.

The balloon is inflated just after the closure of the aortic valve leaflets. Expansion of the balloon causes increased aortic diastolic pressure which may rise by as much as 70%. This rise in aortic diastolic pressure increases proximal aortic blood flow to the coronary arteries and proximal aortic blood vessels. The increased diastolic blood volume and pressure in the proximal aortic root increases diastolic perfusion pressure. This results in increased blood flow to the coronary vessels. At the same time, arterial end-diastolic pressure and LV end-diastolic pressure fall by 10%. This causes a decrease in the diastolic perfusion gradient thereby enhancing diastolic coronary flow.

***Recommendations for use of IABP*:** **(JACC 1996:28(5)@1362)**

Class I:

1) Cardiogenic shock not quickly reversed with pharmacologic therapy. The IABP is utilized as a stabilizing measure in preparation for angiography and prompt revasuclarization.

2) Acute mitral regurgitation or VSD complicating MI as a stabilizing therapy for angiography and repair/ revascularization.

3) Recurrent intractable ventricular arrhythmias with hemodynamic instability.

4) Refractory post-MI angina as a bridge to angiography and revascularization.
Class IIa:
1) Signs of hemodynamic instability, poor LV function, or persistent ischemia in patients with large areas of myocardium at risk.
2) Class IIb:
1) In patients with successful PTCA after failed thrombolysis or those with three vessel coronary disease-to prevent reocclusion.
2) In patients known to have large areas of myocardium at risk with or without active ischemia.

Common uses of IABP **(J Crit Ill 1992;7(3)@437)**

1) Unstable angina (common use for IABP)= reduces myocardial work and increases coronary flow

2) Refractory V tach of ischemic etiology = increases coronary flow (augments antiarrhythmics)

3) Acute MI = limit size of MI, (rarely utilized-thrombolytics more effective)

4) Peri-infarction cardiogenic shock = support patient while being prepped for PTCA or CABG; decreases afterload/increases diastolic perfusion pressure/decreases pulmonary wedge pressure/increases cardiac output by decreasing cardiac work and improving LV outflow total

5) AMI with VSD = reduces afterload; augments forward flow through the aorta (CO increased)
Result: L-to-R shunting reduced/decreased LV volume overload and LV end-diastolic pressure/reduces myocardial work & O2 consumption

6) AMI with acute MR = decreases afterload & augments forward flow through the aorta (CO increased)
Result: reduced myocardial work & O2 consumption/ improved coronary flow/reduced afterload promotes LV forward emptying and decreases regurgitant flow/ decreased LV end-diastolic pressure alleviates pulmonary congestion

7) AMI with LV aneurysm = with systole thinned ventricle dilates decreased LV stroke volume & increased LV end-diastolic pressure (further reduces CO & causes pulmonary congestion). IABP reduces afterload & promotes forward flow through aorta/Reduces LV end-diastolic pressure = alleviates pulmonary congestion

Contraindications for IABP:
1) Aortic insufficiency=diastolic balloon inflation increases regurgitant volume & acutely dilates LV
2) Aortic dissection=balloon may be passed into false lumen and rupture aorta
3) Peripheral vascular disease (especially lower extremities)=potential for dissection of vessel, complete occlusion of vessel during inflation and deflation, cautious use in vessels with grafts (relative contraindication)
4) Thoracic or abdominal aortic aneurysm=inflation of balloon may damage aorta further or cause perforation, potential for dissection during insertion
5) Bleeding diathesis=bleeding from insertion site is common complication (20-30%)

Complications of IABP **(higher in women, DM, HBP)**
1) Perforation of iliofemoral artery or aorta
2) Arterial rupture
3) Arterial pseudoaneurysm
4) Arterial occlusion causing limb or organ ischemia
5) Severe bleeding/hematoma at insertion site (5% require surgical intervention)
6) Infection
7) Thrombocytopenia
8) Hemolytic anemia
9) Arterial/aortic thrombosis
10) Embolic phenomenon

B. *Temporary Cardiac Pacemaker* (JACC 1996;28(5)@1367)
*****Temporary pacing has not been shown to reduce mortality associated with AV block or intraventricular conduction delay. Cause of death most commonly due to pump failure or sudden malignant ventricular arrhythmia, rarely bradycardia*****

a) *Transcutaneous Patches With Active Demand Pacing:*
Class I:
1) Sinus bradycardia (rate < 50/min) with symptoms of hypotension (systolic blood pressure < 80mm Hg) unresponsive to drug therapy
2) Mobitz type II second-degree AV block
3) Third-degree heart block
4) Bilateral BBB (alternating BBB, or RBBB and alternating LAFB or LPFB (regardless of time of onset)

5) Newly acquired or age indeterminate LBBB, LBBB and LAFB, RBBB and LPFB
Class IIa:
1) Stable bradycardia (systolic blood pressure >90 mm Hg, no hemodynamic compromise, or compromise responsive to initial drug therapy)
2) Newly acquired or age-indeterminant RBBB
Class IIb: 1) Newly acquired or age indeterminate first-degree AV block
Class III: 1) Uncomplicated acute MI without evidence of conduction system disease

b) *Temporary Transvenous Pacing:*
Class I:
1) Asystole
2) Symptomatic bradycardia (includes sinus bradycardia with hypotension and type I second-degree AV block with hypotension not unresponsive to atropine)
3) Bilateral BBB (alternating BBB or RBBB with alternating LAFB/LPFB) [any age]
4) New or indeterminate age bifasicular block (RBBB with LAFB or LPFB, or LBBB) with first-degree block
5) Mobitz Type II second-degree block
Class IIa:
1) RBBB ad LAFB or LPFB (new or indeterminate)
2) RBBB with first-degree AV block
3) LBBB, new or indeterminate
4) Incessant VT, for atrial or ventricular overdrive pacing
5) Recurrent sinus pauses (> 3 sec), not responsive to atropine
Class IIb:
1) Bifasicular block of indeterminate age
2) New or age-indeterminate isolated RBBB
Class III:
1) First-degree heart block
2) Type I second-degree AV block with normal hemodynamics
3) Accelerated idioventricular rhythm (AIVR)
4) BBB or fascicular block known to exist before acute MI

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