CCU: How it came to be

- Prior to 1961, patients with AMI who reached the hospital were treated largely with benign neglect.
- Sedated and placed at bed rest, as far removed physically as possible from nurses’ station.
- The early mortality of AMI patients who reached the hospital exceeded 30%.

"Many cases of cardiac arrest associated with acute myocardial ischaemia could be treated successfully if all medical, nursing and auxiliary staff were trained in closed chest cardiac massage and if the cardiac rhythm of patients with acute myocardial infarction were monitored by an electrocardiogram linked to an alarm system"

- DJ Julian

Treatment of cardiac arrest in acute myocardial ischemia and infarction, Lancet 1961; 2: 302-304

Result: Mt Sinai Study

With institution of coronary care units, the in-hospital mortality of acute myocardial infarction was immediately reduced in half

Within several years soon became a requirement for hospital accreditation

Evolution of the Cardiac Intensive Care Unit

Important to Know/ Likely to Be Tested?

- Basic swan gantz catheter hemodynamics
- Complications of myocardial infarction
- Cardiac tamponade
- Arrhythmia management (basics)
- Aortic dissection
- Cardiac arrest

Swan Ganz Catheters

- Assessment of intracardiac pressures
- Calculation of cardiac output, assessment of systemic and pulmonary resistance
- Evaluation for intracardiac shunts

Cardiac Hemodynamics

Abnormalities in cardiac hemodynamics

Abnormalities in cardiac hemodynamics

<table>
<thead>
<tr>
<th>Primary LV Failure</th>
<th>RV Failure</th>
<th>Vasodilatatio/ryshock (serosa)</th>
<th>Hypovolemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA pressure</td>
<td>normal</td>
<td>increased</td>
<td>reduced</td>
</tr>
<tr>
<td>PA pressure</td>
<td>increased</td>
<td>normal</td>
<td>reduced</td>
</tr>
<tr>
<td>PCWP pressure</td>
<td>increased</td>
<td>normal</td>
<td>reduced</td>
</tr>
<tr>
<td>PA saturation</td>
<td>reduced</td>
<td>increased</td>
<td>reduced</td>
</tr>
<tr>
<td>Cardiac output/index</td>
<td>decreased</td>
<td>increased</td>
<td>reduced</td>
</tr>
</tbody>
</table>

- PA sat and CO are increased.
  - Reduced vascular resistance
  - Peripheral tissue over-perfused
  - Relative decrease in oxygen extraction

- PA sat and CO are low
  - Vascular resistance increases
  - Periphery under-perfused
  - Relative increase in oxygen extraction

Question

74 y/o female presents to Emory 3 days after discharge from OSH. Says she “had a heart attack” and was there for 3 days. Has been taking her medicines. Now has shortness of breath, feels weak and lightheaded.

- ECG: anterior ST elevation, inferior and anterior Q waves.
- Exam: SBP 80, rales to ½, harsh holosystolic murmur
- Emergent cath: 60% LM disease, Diffusely diseased, small RCA, stent in proximal LAD which is a wraparound vessel. RA pressure is 8 mmhg, PCWP is 28 mmhg, and PA Sat is 85%

What is the diagnosis

A) Myocardial infarction and RV infarction
B) Myocardial infarction and severe acute mitral regurgitation
C) Myocardial infarction and ventricular septal defect
D) Myocardial infarction only

Exam for acute VSD vs acute severe MR

<table>
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<tr>
<th>Acute VSD</th>
<th>Acute severe MR</th>
</tr>
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<tbody>
<tr>
<td>Harsh systolic murmur audible over a large area.</td>
<td>Murmur of MR is variable, may be absent</td>
</tr>
<tr>
<td>Apical thrill</td>
<td>Apical thrill is rarely present (unlike VSD)</td>
</tr>
<tr>
<td>Left-to-right shunt</td>
<td>No shunt</td>
</tr>
<tr>
<td>Echo demonstrating flow</td>
<td>Transthoracic echo can sometimes miss</td>
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<tr>
<td>PA catheter with oxygen &quot;step up&quot;</td>
<td>TEE can be necessary</td>
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Acute VSD
- Harsh systolic murmur audible over a large area.
- Apical thrill
- Left-to-right shunt
  - Echo demonstrating flow
  - PA catheter with oxygen "step up"

Acute severe MR
- Murmur of MR is variable, may be absent
- Apical thrill is rarely present (unlike VSD)
- No shunt
- Transthoracic echo can sometimes miss
- TEE can be necessary

VSD
- Most patients with VSD develop shock
  - Typically develop 3-7 days post MI
- Step up in RV saturation characteristic (High PA oxygen saturation, low RA oxygen saturation)
- IABP, Dobutamine/Nitroprusside for stabilization
- Mortality 99.9% without surgery, 75% with surgery
  - Timing of surgery is controversial
  - Evolving percutaneous options

Acute Severe MR treatment
- Patients often require mechanical ventilation
- Intra-aortic balloon or nitroprusside/dobutamine for acute stabilization through afterload reduction
- Mitral Valve repair/replacement plus CABG asap
- Surgical delay = increased mortality
**Free Wall Rupture**

- Occurs in the same time frame as other mechanical complications
- Complete rupture often presents with sudden shock and death
- Incomplete rupture often presents with chest pain (but may be vague symptoms), hypotension.
- Echo and pericardiocentesis can help confirm the diagnosis
- Supportive medical management but key is prompt surgical treatment

**Question 2**

- 55 year old male with PMH smoking presents with CP. The ecg shows inferior ST elevation and his BP drop to 60 mmhg after giving him a nitroglycerin tablet. His lungs are clear, JVD appears elevated. The cath lab is activated but are 30 minutes away
  - a) Give a bolus of IVFs
  - b) Thrombolytic therapy for acute MI
  - c) Start dobutamine and give furosemide
  - d) Start heparin gtt and give furosemide

**RV Infarction**

- Proximal occlusion of the right coronary artery leads to occlusion of the RV marginal branches
- Systemic hypotension, normal left sided filling pressures, and increased right sided pressures
- Echocardiography can play a vital role in diagnosis
  - RV dilatation
  - Abnormal right ventricular motion

**RV Infarction Diagnosis**

- Hemodynamics: elevated RA and RVEDP (>12 mmhg), normal to low PA pressures, low PCWP (<15 mmhg), low CO.
- ECG
  - ST elevation V4R most specific test
- PE: JVP, clear lungs, hypotension

**RV infarction Principles of Management**

- Stabilization and reperfusion are the hallmarks of therapy
- If patient survives the initial 2-5 days, RV function will typically recover
- Hemodynamic monitoring and fluid administration to achieve PCWP of 15-18 mmhg (preload-dependent state). Inotropes can be used
- Maintenance of AV synchrony is important
  - Temporary pacing in AV block
  - Cardioversion of atrial flutter/fibrillation
- **RV infarction significantly increases mortality when associated with inferior wall MI**
57 y/o male presents with sudden onset of chest pain with radiation to the back. Past medical history in not significant.

- Physical exam: Bp 150/90, HR 101, afebrile
- CV exam: No murmurs, lungs are clear
- Ecg illustrates: ST elevation in the inferior leads
- CTA of the chest illustrates:

The best treatment option is
A) Medical therapy with beta-blockers and observation in the CCU
B) Emergent Cardiac Catheterization
C) Emergency surgery for type A aortic dissection
D) Emergency surgery for type B aortic dissection
E) Both A and B

Types of aortic dissection

Most common sites of initial tear in aortic dissection
Aortic Dissection

- Sudden Onset Severe Pain
  - May or may not be catastrophic
  - Rippling, tearing
  - Migratory
  - Never experienced before
  - Restless, sense of doom

- Look for underlying disorder in exam or history (or family history)
  - Bicuspid Valve
  - Marfan's Syndrome (connective tissue disorder)
  - Coarctation of the aorta s/p repair

- Hypertension (especially moderate or severe) or known aortic aneurysm (70-90% have underlying hypertension)

Medical Therapy

- Blood Pressure control
  - IV beta blocker to reduce the HR<60; the associated fall in both blood pressure and the rate of rise in systolic pressure will minimize aortic wall stress
  - Esmolol has advantages in the acute setting, due to its short half-life and ability to titrate to effect
  - Nitroprusside should not be used without first controlling the heart rate with beta blockade

- Watch Renal function!!

Surgical Therapy

- Uncomplicated Type B dissection
- Stable, chronic dissection (>2 weeks)
- Stable, lone, arch dissection

- Acute, Type A Dissection
- Acute, complicated Type B Dissection
  - End-organ damage
  - Rupture
  - Extension into ascending aorta
  - Marfan's
  - AI (when none prior)

Who to take to Surgery?

Medical Therapy

- Acute, Type A Dissection
- Acute, complicated Type B Dissection

Surgical Therapy

- Uncomplicated Type B dissection
- Stable, chronic dissection (>2 weeks)
- Stable, lone, arch dissection

Question 4

- 84 y/o female with history of malignancy presents with acute onset shortness of breath. Sbp 70/40, HR 90-105. Duplex u/s illustrated lower extremity DVT. Currently on dopamine, 12 lead ecg illustrates.

Question 4

- The best treatment option is
  A) Echocardiography and emergent pericardiocentesis
  B) thrombolytics therapy for pulmonary embolism
  C) emergent cardioversion for atrial fibrillation
  D) intravenous fluids and reassessment

Question 4

- The best treatment option is
  A) Echocardiography and emergent pericardiocentesis
  B) CT scan and thrombolytics therapy for pulmonary embolism
  C) emergent cardioversion for atrial fibrillation
  D) intravenous fluids and reassessment
Cardiac Tamponade

- During inspiration, RV volume increases & in tamponade, the RV is unable to expand into the maximally stretched pericardium
- Leftward ward bulging of the interventricular septum
- Decreased L VEDV
- Decreased cardiac output

What is the paradox of pulsus paradoxus?

Intrapericardial pressure (IPP) tracks intrathoracic pressure. Inspiration: negative intrathoracic pressure is transmitted to the pericardial space:
- IPP
- blood return to the right ventricle
- Jugular venous and right atrial pressures
- Right ventricular volume → interventricular septum shifts towards the left ventricle
- Left ventricular volume
- LV stroke volume
- ↓ blood pressure (<10mmHg is normal) during inspiration

Question 5

- 55 yo woman collapses in church. Her husband is a physician and immediately starts CPR. EMS arrives after 10 minutes.
- Initial rhythm is VT, successful ROSC with 1 shock
- Patient arrives in ED unconscious but SBP 110/70
- ECG: ST elevations in inferior leads

Which of the following is correct regarding her subsequent management

A) She should go to the cath lab if she regains consciousness
B) Therapeutic hypothermia should be initiated
C) Therapeutic hypothermia should be avoided due to acute MI
D) Administer oxygen to ensure oxygen saturation of 100%

Therapeutic hypothermia

- One of few therapies to improve survival and good neurologic outcomes
- Initiation as soon as possible
- Can be started simultaneously with cath lab activation
- Cool for 24 hours
- Target temperature of 32-36
- No clear evidence of benefit for higher or lower target (33 vs 36)
- Active temperature management after controlled rewarming to prevent fever
  - Timing not well-defined though temp kept to < 37.5 for 48 hours (total 72 hours) in largest trial
- Avoid prognostication during cooling period