CMC Review Course
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Vascular / CABG / IABP

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Dream
Critical Limb Ischemic (CLI)

- **Definition**
  - Limb pain at rest or night
    - Pain requiring narcotics
    - Pain particularly in forefoot or toes
  - Ulcers or gangrene
    - Painful and tender to touch
    - Pain particularly in forefoot or toes
  - Impending limb loss

- **Risk Factors**
  - ABI < 0.4 in non diabetic patients
  - Diabetic patients
  - Chronic renal failure
  - Low cardiac output state
  - Infection, injury or skin breakdown of affected extremity

**Signs and Symptoms of Critical Limb Ischemia**

- Pain occurring at night or rest
  - Forefoot or toes
- Pain requiring narcotics
- Arterial ulcers or gangrene
  - Painful / tender to touch
  - Open (infection / cellulitis)
    - Specialized wound care
  - Diabetics / immunocompromised
    - Systemic antibiotics
- Impending limb loss due to compromised flow
- Subclinical signs
Other Signs of Chronic Limb Ischemia

- Dependent redness
- Pallor on elevation
- Decreased capillary refill
- Shiny skin / subcutaneous tissue loss
- Trophic skin changes
- Loss of hair
- Calf atrophy
- Potential tissue loss

Critical Limb Ischemia from Atherosclerotic Emboli

- Post endovascular procedure
- Systemic fatigue or systemic muscle discomfort
- Increased creatinine
- Bilateral limb symptoms
- Skin discoloration
  - Red non-blanching network pattern
Treatment for CLI

- Goal: Avoid amputation
- Revascularization evaluation
- Endovascular treatment is first line
- Inflow lesions treated first
- Adequate flow to foot required for healing

- Antiplatelet and anticoagulation therapy
- Intravenous (or intraarterial) prostanoids (vasodilator and platelet aggregation inhibitors)
  - Prostaglandin E1 (PGE1)
  - Prostacyclin (PGI2) (iloprost)
  - Note: Oral iloprost is not effective in reducing the risk of amputation or death.
- Parental pentoxifylline treatment is not useful for CLI
- Maintenance of limb in dependent position
- Treatment of infection to reduce demand

Amputation

- Significant necrosis of the weight bearing portion of foot
- Uncorrectable flexion contracture
- Paresis of the extremity
- Refractory rest pain from ischemia
- Sepsis
- Very limited life expectancy
Acute Limb Ischemia

- **Causes**
  - Thrombosis with plaque rupture
  - Thrombosis of bypass graft
  - Thromboembolization from aneurysm
  - Arterial trauma or dissection
  - Vasospasm
  - Compartment syndrome
  - Hypercoagulability

- **Signs and Symptoms**
  - Pain
  - Paralysis
  - Parathesias
  - Pulselessness
  - Pallor
  - Polar

Treatment for Acute Limb Ischemia

- **Systemic anticoagulation**
- **Catheter based thrombolysis**
  - Urokinase (preferred over streptokinase)
  - Intraarterial
  - Best if ischemia < 14 days
- **Mechanical thrombectomy devices**
  - Option when contraindication to thrombolysis
Venous Thromboembolism

- Virchow Triad
  - Venous stasis
  - Vessel wall injury
  - Hypercoagulability
- Activation of coagulation in areas of reduced flow
- Before thrombus becomes organized and adherent (5-10 days) it can extend proximally or embolize
  - Extension can damage venous valves / can cause venous outflow obstruction
- Chronic venous insufficiency can result from incomplete clot lysis and recanulization
  - Can lead to venous ulceration
Hospitalized patients incidence ranges from 20-70%

- PE from DVT is leading cause of preventable hospital mortality
  - 20% of untreated proximal DVT will result in PE
  - 10-20% of these PE will be fatal
  - Anticoagulation decreases mortality 5 to 10 fold

- Bedside assessment is not sensitive for diagnosis
  - Cannot make accurate diagnosis based on signs and symptoms

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**Signs and Symptoms**

- Signs and symptoms related to degree of venous obstruction and inflammation of vessel wall
- Many patients asymptomatic

- Edema (unilateral)
- Reddish purple hue from venous obstruction
- Tenderness
  - Calf muscles
  - Along deep vein medial thigh
- Homan’s sign
  - Present < 1/3 patients with DVT
  - Present in 50% patient without DVT
  - Non specific
- Venous Distention
  - Patients with thrombophlebitis can have co-existing DVT
- Fever (low grade)
Signs and Symptoms

- Clinical signs of PE
  - May be primary manifestation in small percent of patients
- Phlegmasia cerulea dolens
  - Cyanosis from massive ileofemoral venous obstruction
- Phlegmasia alba dolens
  - Painful white inflammation from massive ileofemoral venous obstruction with associated arterial vasospasm
  - Pain and white discoloration can occur with acute arterial occlusion
  - Swelling, petechiae, and distended superficial veins can be used to differentiate

Specific Risk Factors

- General
  - Older age
  - Immobility > 3 days
  - Pregnancy / post partum
  - Major surgery within 4 weeks
  - Long plane or car trips (> 4 hours) within 4 weeks
- Trauma
- Vasculitis
- Hematologic factors that result in hypercoagulability

- Drugs
  - IV drug use
  - Oral contraceptives
  - Estrogen HRT
- Medical conditions
  - Cancer
  - Stroke
  - MI
  - HF
  - Sepsis
  - Nephrotic syndrome
  - Ulcerative colitis
  - Previous DVT
Diagnosis

- **D-dimer antibodies**
  - D-dimer assays are not specific to DVT
    - Can be helpful to rule out
    - Cannot be used to rule in
  - Remain elevated in DVT for 7 days
  - There are multiple different D-dimer assays
- A negative D-dimer can be used to rule out in patients with low to moderate risk
- All patients with + D-dimer or patients with moderate to high risk require duplex ultrasound

Diagnosis

- **D-dimer and risk scoring** are used to determine need for further diagnostic testing
- **Objective testing** necessary before anticoagulation
- Noninvasive studies are first line over contrast venography
- Diagnostic criteria with Duplex ultrasound: failure to compress vascular lumen (due to thrombus)
  - Overall specificity 95%
  - Most inaccurate in diagnosis of calf vein thrombosis
Diagnosis

- CT venography
  - Incorporated into CT of chest for PE
  - Can detect venous occlusion proximal to inguinal ligament (ileofemoral DVT)

- Summary:
  - Ambulatory patients: Duplex ultrasound is diagnostic test of choice
  - CT venography: Best for suspected ileofemoral DVT
  - Noninvasive studies, D-dimer and patient risk score are used in determining diagnosis and decision to treat

Goals of Treatment

- Prevent PE
- Prevent postphlebitic syndrome
- Reduce risk of other complications

Note: DVT of calf rarely causes significant PE.
Heparin (UFH / LMWH)

- **Heparin**
  - Prevents extension of thrombus
  - Reduces incidence of fatal and non-fatal PE
  - No effect on pre-existing non adherant thrombus
  - Does not significantly reduce risk of post phlebitic syndrome
    - Primarily caused by initial thrombus

- **LMWH has advantages over UFH**
  - Better bioavailability
  - Less monitoring
  - Lower incidence of HIT
  - Lower incidence of osteoporosis

Full anticoagulation may not be indicated in calf DVT.

Fondaparinux

- **Synthetic Factor Xa inhibitor (Fondaparinux (Arixtra))**
  - Daily SQ injection – fixed dose for most body weights
    - Effective and as safe as BID weight based enoxaprin
  - Also cleared renally (contraindicated in several renal impairment)
    - Creatinine clearance < 30 mL/min
  - No therapeutic monitoring required
  - No reported HIT
  - No specific antidote
Fibrinolytic Therapy

- **Advantage**
  - Prevention of PE
  - Restoration of normal venous circulation
  - Preservation of venous valves
  - Prevention postphlebitic syndrome

- **Limitations**
  - Does not prevent clot propagation, rethrombosis, or potential embolization
  - Not effective once thrombus is adherent and organized
  - If clot is large, the drug may not be able to penetrate entire clot
  - Most patients have contraindications

- **Heparin and oral anticoagulation always follow fibrinolytic therapy**

Only indicated in massive ileofemoral thrombosis with vascular compromise.

Catheter Directed Thrombolysis

- **Delivers higher drug concentration at the site with lower total dose compared to systemic administration**

- **Indicated in acute ileofemoral DVT**
  - Adequate recannulization less likely in this location with anticoagulation alone
  - Collateral flow is limited
  - Ileofemoral DVT has higher rate of post thrombotic syndrome than other locations

Thrombectomy is an option in massive ileofemoral thrombosis with vascular compromise when thrombolysis is contraindicated.
Wafarin

- Warfarin is initiated at 5 mg daily
- Overlapped with anticoagulation therapy x 5 days until INR is therapeutic
- Treatment of 3 to 6 months for 1st episode
- Treatment of minimum of one year for recurrent episodes

Inferior Vena Cava Filter

- Greenfield filter
  - Enhances thrombolysis of trapped emboli
  - 98% long term patency
  - 4% recurrent PE
  - Can increase later risk of DVT
- Retrievable filters are also available
- Indications
  - Contraindication to anticoagulation
  - Failed anticoagulation
- Filters can be placed under transabdominal or intravascular ultrasound
Other Treatments

Graded Compression Stockings
- Post thrombotic syndrome affects up to 50% of DVT patients after 2 years
- High risk
  - Elderly
  - Recurrent ipsilateral DVT
- Graduated compression stockings (ankle pressure 30-40 mmHg)
  - Reduces risk of post thrombotic syndrome by 50%

Ambulation
- Early ambulation (day 2 after initiation of outpatient anticoagulation) is indicated
- Early ambulation is only indicated when done in conjunction with wearing graduated compression stockings
- Early ambulation results in a decrease in swelling and pain
- May also result in a decrease in post thrombotic syndrome

Inpatient versus Outpatient Therapy
- Most patients with proximal vein DVT can be safely managed with outpatient anticoagulation
- Isolated calf vein DVT may only be treated with NSAIDs or ASA with follow up
- Patients with risk factors need to be re-evaluated at one week.
- Patients with calf DVT (not on anticoagulation) need to re-evaluated in one week to assure DVT has not advanced into the proximal venous system

- Patients needing inpatient treatment
  - Suspected or actual PE
  - Ileofemoral DVT
  - CV or pulmonary comorbidities
  - Familial coagulopathies or bleeding disorders
  - Pregnancy
  - Morbid obesity
  - Renal failure
  - Contraindication to anticoagulation
  - Homeless or other social issues of non compliance
Primary Prevention

- Prophylaxis in all high risk patients
  - One large trial: 40 mg daily enoxaparin achieved 63% reduction in DVT in medical patients (Cohen, et al., 2001)
- Women’s Health Study: Vitamin E supplementation reduced the risk in women

Complications

- PE or other systemic embolization despite anticoagulation
- Soft tissue injury from very large thrombus
- Bleeding complications from anticoagulation
- Chronic venous insufficiency
- Post phlebitic syndrome
  - Pain
  - Edema
  - No new thrombus
Abdominal Aortic Aneurysm

- Aortic diameter 3 cm
- Risk factors for AAA
  - Male
  - Family history
  - Advanced age
  - Cigarette smoking
  - Polycystic kidney disease
  - CAD, CVD, PAD
  - Popliteal aneurysms
  - Marfan syndrome

AAA

Pathophysiology
- Degeneration of media
- Inflammatory process
- “Inflammatory Aneurysms” – smokers
  - White shiny fibrotic material; adhere to structures
  - More symptomatic – increase surgical mortality
- “Infectious Aneurysms”
  - Secondary infection from existing aneurysm

Diagnostic studies
- Most asymptomatic
- Ultrasound
- CTA or MRA for mapping for surgical intervention

Physical exam
- Bruit
- Pulse palpation
- Aneurysmal palpation / pulsatile mass
Symptomatic Aneurysms

- **Back pain or abdominal pain**
  - Pain is most common symptom.
  - Pain is a long lasting steady pain, not generally affected by movement, but some relief may be obtained with knees in a bent position.
  - Abrupt, severe, worsening pain, or pain radiating toward lower extremities is sign of impending rupture.

- **Hypotension**
  - Hemorrhagic shock develops rapidly.

- **Pulsatile abdominal mass**
  - Mass is not pulsatile with significant hypotension.

Treatment for Symptomatic Aneurysms

- Symptomatic aneurysms are surgically treated regardless of size
  - An evaluation for urgent surgery is done with the following presentation:
    - Back pain or abdominal pain
      - Pain is most common symptom
      - Pain is a long lasting steady pain not generally affected by movement but some relief may be obtained with knees in a bent position.
      - Abrupt, severe, worsening pain, or pain radiating toward lower extremities is sign of impending rupture.
    - Hypotension
      - Hemorrhagic shock develops rapidly
    - Pulsatile abdominal mass
      - Mass will not be pulsatile with significant hypotension
Treatment for AAA

- **Open Surgical Repair**
  - Requires aortic cross-clamping
  - High rate of mortality with symptomatic CAD
    - Perioperative beta blockers
  - Lowest rate of mortality with infra renal aneurysms

- **Endovascular Repair**
  - Modular stent grafts
  - Transfemoral approach
  - Endograft leaks are common complication

Aortic Dissection Pathophysiology

- Intimal tear
- False channel
- Risk of rupture: outer wall
- Dissection – hematoma – occlusion of vessels

- Ascending aorta
- Descending aorta
- Abdominal aorta
Risk for Dissection

- Hypertension is present in most patients with dissections
- Congenital disorders affecting connective tissue such as Marfan Syndrome or Ehlers-Danlos syndrome; also presence of bicuspid aortic valve
- 3rd trimester of pregnancy
- After procedures where aorta or the aortic branches have been cannulated.

Classification of Dissections

- Acute or chronic
- **Type A Dissections:** Dissections involving the ascending aorta.
- **Type B Dissections:** Dissections involving the descending thoracic aorta. These dissections begin distal to the left subclavian artery.
Complications of Dissection

- Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
- MI from retrograde coronary artery dissection.
- Cardiac tamponade from ascending aorta or aortic arch rupture.
- Intrapleural rupture from descending aortic dissection ruptures into intrapleural space – most commonly left sided.
- Retroperitoneal bleed from rupture of abdominal aorta dissection.
- Stroke from brachial artery compromise.
- Paraplegia, reduced blood flow to kidneys, bowels, and lower extremities from compromise of arterial branches.

Clinical Presentation

Chest or back pain with variation in upper extremity blood pressure is key assessment finding in aortic dissection. Recurrent chest or back pain can indicate extension or rupture. The presence of aortic regurgitation in the setting of chest pain is also suspicious for aortic dissection.
Treatment of Dissections

➢ Stop dissection
  • Control of hypertension
  • Lowest possible pressure without compromising perfusion
  • Velocity of ejection
  • Rate of rise

  • Beta-blocker in combination with sodium nitroprusside

Type A

• Surgery required
• Deep hypothermic circulatory arrest
• Distal false channel not completely eliminated
• Risk for bleeding
• Replacement of aortic valve / reimplantation of coronary arteries
• Hypertension management
• Surgical considerations similar for ascending aortic aneurysms
• Surgical mortality 15-20%

Type B

• Typically treated medically
• Higher surgical risk candidates
• Surgical considerations similar to those for descending thoracic aneurysms
• Surgery limited to complicated dissections:
  • Persistent pain
  • Uncontrolled hypertension
  • Evidence of expansion or rupture
  • Circulatory compromise to visceral, renal, or lower extremity vessels and resultant organ ischemia.
  • 6 to 6.5 cm diameter in chronic dissections

Surgical mortality 15-20%
Hypertensive Crisis

Life threatening elevation in blood pressure requiring emergency treatment (within 1 hour) to prevent end organ damage or death.

End organs:
- Heart
- Brain
- Kidneys

Hypertensive Encephalopathy
- BP > 250/150 mmHg
- Loss of cerebral autoregulation
  - Vasospasm
  - Ischemia
  - Increased capillary pressure
  - Cerebral edema – hemorrhage

Hypertensive Crisis: Etiology

- Uncontrolled hypertension
- Renal dysfunction
- Preeclampsia
- Adrenergic crisis
  - Drug reactions
  - Pheocromocytoma
- Post operative complication
- Pituitary tumor
- Adrenocortical hyperfunction
- Severe Burns
**Signs and Symptoms**

- BP > 250/150 mmHg
- Retinopathy
- Papilledema of optic disc
- Vomiting
- Severe headache
- Altered LOC
- Seizures
- S&S of heart failure

**Treatment**

- MAP lowered no more than 25% in first 2 hours or to 160/100 mmHg
- Nitroprusside is gold standard (contraindicated in pregnancy)
  - 0.25 to 0.5 mcg/kg/min – titrate every 5 minutes
  - Maximum dose 8-10 mcg/kg/min
  - Immediate onset – lasts 1-5 minutes
  - Thiocynate toxicity after 48 hours or with renal insufficiency
    - Blurred vision
    - Tinnitus
    - Seizures

- Other agents
  - Fenoldopam (dopaminergic agonist)
  - Alpha blocking agent (or combination alpha and beta blocking agent)
  - ACE inhibitors
Cardiac Tamponade

- Clinical syndrome caused by accumulation of fluid in the pericardial space
- Same causes as pericarditis / pericardial effusion
- Increase capillary permeability due to inflammation may cause fluid leak into pericardial space
  - >120cc can cause tamponade if rapid
  - 2 Liters may not cause tamponade if slow
- Results in reduction in ventricular filling and ultimately hemodynamic compromise

Differentiation between pericardial effusion and tamponade is hemodynamic status.

Signs and Symptoms

- Feeling of impending doom
- Beck’s Triad
  - Hypotension, Distended neck veins, Muffled heart sounds
- Equalization of filling pressures (RAP, PAD, PAOP within 5mm of each other)
- Narrow pulse pressure
- Pulses paradoxus
  - Also observed in constrictive pericarditis, severe obstructive pulmonary disease, restrictive cardiomyopathy, PE, and RV infarct with shock.
Pulsus Paradoxus

- To measure the pulsus paradoxus, patients are often placed in a semirecumbent position; respirations should be normal. The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard only during expiration. At this pressure reading, if the cuff is not further deflated and a pulsus paradoxus is present, the first Korotkoff sound is not audible during inspiration. As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded. If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.

(Yarlagadda, Chakri, 2005 Cardiac Tamponade. Retrieved 3-22-06 from www.emedicine.com)

Tamponade Treatment

- Oxygen
- Volume expansion
- Bedrest with leg elevation
- Dobutamine (increase pump without increasing SVR)
- Avoid positive pressure mechanical ventilation (decreases venous return)
- Pericardiocentesis
  - Percutaneous
  - Surgical window
Cardiac Trauma

- **Blunt**
  - Cardiac Contusion
    - S & S as ischemia / injury
    - ECG changes may be delayed
    - Dysrhythmias
  - Severe
    - Delayed free wall rupture
    - Ventricular septal defect
    - Ventricular aneurysm
  - Cardiac Rupture
    - Right ventricle most common
    - Surgery required
  - Valvular Injury
    - Aortic valve most affected
    - Aortic regurgitation / CHF

- **Blunt / Penetrating**
  - Tamponade

Goals of Revascularization

- Improve Survival
- Minimize Complications of Ischemia
- Relieve Ischemic Symptoms
- Improve Functional Capacity

Difficult to compare revascularization procedures because long term data does not reflect recent advances.
Indications for CABG

- Left main disease or multi vessel disease with impaired LV function
- Multi vessel disease in diabetes
- Left main equivalent disease*
- Proximal LAD with very + stress and abnormal ECG
- Survivors of sudden death with CAD

Greatest Survival Benefit

Contraindications to CABG

- Lack of adequate conduit
- Small (< 1-1.5 mm) coronary arteries distal to stenosis
- Severe atherosclerosis of the aorta
- Severe left ventricular failure and coexisting peripheral vascular, renal and pulmonary disease

Wait time for RV infarcts
Traditional CABG

- Median Sternotomy
- Cardiopulmonary Bypass
  - Clamping of aorta
  - Myocardial protection
    - Cardioplegia
      - Potassium, Magnesium, Procainamide
      - Crystalloid or Crystalloid and Blood Mixtures
    - Antegrade or retrograde (usually both)
    - Hypothermic or normothermic (usually cold)

Graft Material

- Saphenous Vein Graft
  - Harvesting
  - Attached to ascending aorta and distal to stenosis
  - Flow is pressure dependent
  - Graft Failure at 10 years
    - Short term patency
    - Long term patency
Graft Material

- Internal Mammary Arteries
  - Patency
  - Long survival and post op mortality benefits
  - Pedicle graft versus free graft
  - LIMA to LAD
  - Bilateral IMA use
- Radial Artery
  - Advances and disadvantages
- Right Gastroepiploic Artery
  - More extensive surgery

Non homologous graft options:
- Cryopreserved saphenous vein grafts or umbilical vein grafts treated with gluteraldehyde

Minimally Invasive Techniques

- MIDCAB
  - Beating heart
  - No sternotomy or ministernotomy
  - Indications
  - Limitations
- OPCAB
  - High risk aortic atherosclerosis
  - Hemodynamic stability

Future: Port Access / Video Assistance / Endovascular Techniques
Preoperative Assessment

- Chest x-ray
- ECG
- Complete blood count, complete chemistry panel, urinalysis, coagulation panel, and type and screen/crossmatch.
- Thorough history and physical
  - Coagulation abnormalities / previous problems with bleeding
  - Previous vein stripping / varicose veins
  - Skin lesions or rash (especially near planned incisions)
  - Recent or current infection.
  - Recent or current anticoagulant or antiplatelet use (particularly clopidogrel)
  - Bilateral arm blood pressure assessment.
  - History of alcohol use.
  - Baseline neurological and functional status for postoperative comparison.
  - Psychosocial, cultural, and educational needs.

Preoperative Medications

- All antianginal, antihypertensive, and heart failure medications should be continued right up until the time of surgery
  - Possible exceptions are ACE inhibitors (ACEI) and angiotensin II receptor blockers
  - Non-steroidal anti-inflammatory medications (NSAIDs) are commonly held preoperatively
- Insulin and oral hypoglycemic agents are held or given in reduced dose the morning of surgery.
- Routine preoperative mupirocin administration is recommended for all patients.
- Preoperative intravenous prophylactic antibiotics are not given until the patient is in the operating suite so the administration can be timed to be 30 to 60 minutes prior to the initial incision.
- Antiplatelet and anticoagulant medications held according to guidelines
  - EPO to decrease risk of bleeding
Post Operative Period

- Blood pressure management
- Fluid management
- Management of hypothermia
- PA catheter / LA catheter
- Cardiac output and delivery of oxygen
- Ventilation and oxygen
- Epicardial pacing wires
- Chest tubes
- Bleeding
- Pain control and sedation
Key Hemodynamic Considerations

- Optimize pre-load (PAOP 18-20 mm Hg) and HR (90-100 BPM) as first line strategies for maintaining adequate cardiac output.
- Use an inotrope if cardiac index is < 2.0 L/min/mm2
  - Epinephrine.
  - Dopamine if decreased SVR (beta 1 and alpha).
  - Dobutamine if elevated SVR (beta 1 with modest beta 2; beta 2 stronger than alpha).
  - Inamrinone / milrinone if elevated SVR (inotrope with venous and arterial vasodilation).
- Use an alpha agent if blood pressure and SVR is low
  - Phenylephrine if cardiac index is satisfactory.
  - Norepinephrine if cardiac index is marginal.
  - Arginine vasopressin or single dose methylene blue when blood pressure not responsive to norepinephrine.
- Use a vasodilator if SVR is elevated.
  - Nitroprusside.
  - Niroglycerin if evidence of ischemia.

CABG Complications

- Bleeding
- Myocardial depression
- Cardiac tamponade
- Perioperative MI
- Atrial Fibrillation
- Pulmonary
  - Pulmonary edema
  - Atelectasis
  - Pneumothorax
- Renal impairment
- GI
- Neuro
  - Type I
  - Type II
- Postpericardiotomy Syndrome
- Wound Infection
- Death
Atrial Fibrillation

- 20 to 40% of post operative patients
- 2nd to 3rd hospital day
- Risk Factors
  - COPD (or right heart disease)
  - Stopping of preop beta-blockers
  - Increased cross-clamp time
  - Advanced Age
- Aggravating Conditions
  - Increased catecholamines
  - Fluid overload
  - Hypoxia
  - Electrolyte Imbalances

Wound Infection

- Cephalosporins
  - Preop timing
  - Post op course
- Deep sternal wound infection and mortality
- Diabetics and bilateral IMAs
Strategies to Decrease Wound Infection

- Meticulous aseptic technique
  - a) Double gloving of OR team
  - b) Reduced OR traffic
- Clipping rather than shaving of hair and avoidance of hair removal
- Shorter perfusion times
- Avoidance of unnecessary electrocaudery
- Proper timing of pre and perioperative antibiotics
- Strict control of blood sugars during and after surgery

Bleeding

Risk Factors for Bleeding

- Advanced age.
- Preoperative anemia.
- Small body size.
- Acquired or congenital coagulation abnormalities.
- Preoperative antiplatelet or antithrombotic medications.
- Multiple comorbidities.
- Combined valve and CABG surgery.
- Urgent or emergent surgery.
- Reoperation.

Complications

- Inflammatory response from transfusion
  - Leukocyte depletion

Tamponade
## Preoperative Discontinuation Time Frames for Drugs Impacting Coagulation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Time Frame</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>May be discontinued up to 3 days only in elective cases</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>5 to 7 days (caution post DES)</td>
</tr>
<tr>
<td>Tirofiban and Eptifibatide</td>
<td>4 to 6 hours</td>
</tr>
<tr>
<td>Abciximab</td>
<td>12 to 24 hours</td>
</tr>
<tr>
<td>Warfarin</td>
<td>4 days</td>
</tr>
<tr>
<td>Unfractionated Heparin</td>
<td>Continued up to time of surgery</td>
</tr>
<tr>
<td>Low Molecular Weight Heparin</td>
<td>12 to 24 hours</td>
</tr>
<tr>
<td>Direct Thrombin Inhibitors</td>
<td>Continued up to time of surgery</td>
</tr>
</tbody>
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## IABP: Counterpulsation Therapy

- Intra Aortic Balloon (IAB) is **inflated** during diastole and **deflated** during systole
  - The IAB is a volume displacement device

### IAB Placement
- Descending thoracic aorta
- 1 to 2 cm below the subclavian artery origin
- Above renal and mesenteric arteries
Hemodynamic Impact of IAB Pumping

- Increased diastolic aortic pressure
- Increased coronary blood flow
- Increase cardiac output / ejection fraction / forward flow
- Increased cerebral and renal blood flow
- Increased systemic perfusion
- Increased coronary and systemic oxygen supply
- Increased hemodynamic pulse rate

Hemodynamic Impact of IAB Pumping

- Decreased systolic aortic pressure
- Decreased afterload
  - Decreased MVO2
- Decreased LV wall tension
- Decreased preload
  - Decreased pulmonary congestion
- Decreased HR
### Indications / Contraindications

**Indications**
- Cardiogenic Shock
- Extending MI
- Unstable Angina
- Intractable Ventricular Dysrhythmias
- Support for high risk intervention
- Bridging Device
- Mechanical Defects
- Post operative myocardial dysfunction

**Contraindications**
- Absolute
  - Aortic Valve insufficiency
  - Dissecting Aortic Aneurysm
- Relative

### Goals of Inflation

- Increase coronary perfusion pressure
- Increase systemic perfusion pressure and peripheral oxygen supply
- Increase baroreceptor response and decrease SNS stimulation
  - Decrease SVR
  - Decrease HR
Goals of Deflation

- Decrease afterload
  - Decrease MVO2
  - Decrease assisted peak systolic pressure (APSP)
  - Increase cardiac output and ejection fraction (increase forward flow)

Why Inflation Works:
Inflation of the IAB during diastole increases aortic volume and pressure.

Why Deflation Works:
IAB deflation just prior to systole creates a potential space in the aorta. This reduces aortic volume and pressure.
**Inflation Timing:** The IAB is inflated immediately upon closure of the aortic valve.

**Deflation Timing:** The balloon must be deflated before the full onset of systole.

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**Arterial Pressure Timing**

- IAB action is mechanical so the effect is evaluated by a mechanical event.
- Place the IABP in 1:2 assist mode
- Use the arterial wave form to assess timing
Timing Assessment Points

- **PAEDP**: Patient’s End Diastolic Pressure
  - This point determines afterload and is a major determinate of MVO2

Timing Assessment Points

- **PSP**: Peak Systolic Pressure
  - Pressure generated by the LV during mechanical contraction. No IAB effect with this point.
Timing Assessment Points

- DN or Dicrotic Notch:
  - Signifies the beginning of diastole when the aortic valve closes.

Timing Assessment Points

- PDP or Peak Diastolic Pressure:
  - Pressure generated in the aorta as a result of balloon inflation during diastole. This increases aortic pressure and also increases the mechanical pulse rate.
Timing Assessment Points

- BAEDP or Balloon Assisted End Diastolic Pressure:
  - Lowest aortic pressure produced by the deflation of the IAB during isovolumetric contraction. Results in reduced afterload and preload and therefore decreased MVO2.

Timing Assessment Points

- ASSP or Assisted Systole: The systole following IAB deflation.
  - This pressure should be lower than PSP reflecting a decrease in LV work as a result of the shortened isovolumetric contraction phase and lower resistance to systolic ejection.
Augmented Diastolic Pressure (Peak Diastolic Pressure)

- Caused by increased pressure (volume) in aorta during diastole
- PDP should be > than PSP
- The amount of augmentation will be affected by the timing of balloon inflation
  - Maximal augmentation: Balloon inflation 40 msec prior to dicrotic notch

Augmented Diastolic Pressure (Peak Diastolic Pressure)

- PDP = additional mechanical pulse
  - Generated without MVO2
  - Has significant impact on regulatory and compensatory systems of the patient
- Additional perfusion event
  - Perfusion is a result of pressure and time
  - Coronary and peripheral circulation
    - Additional O2 and nutrient supply
    - The coronary system receives greatest benefit due to proximity to IAB.
Factors Impacting Augmentation

- Physical
  - Position
  - Volume
  - Diameter
  - Occlusiveness
  - Drive Gas
  - Duration of Inflation
  - Efficiency of System
  - Timing

- Biological
  - Arterial Pressure
  - Aortic Pressure / Volume Relationship

Augmentation

High
- Risks of increased augmentation (or PDP) are mechanical
- In low cardiac output states the benefits outweigh the risks
- High augmentation post cardiac surgery
  - **Benefits**: Maintenance of coronary graft patency
  - **Risks**: Bleeding and disruption of suture sites
  - Use of vasodilators
  - Lower placement

Low
- Weaning states
- Low volume or output states
Early Inflation: Prior to Closure of Aortic Valve

- Increased aortic pressure: regurgitation of blood into the LV
- Rise in aortic pressure will prematurely close the aortic valve:
  - Decreased LV emptying

Hemodynamic and Clinic Implications of Early Inflation

- Increased ESV / ESP
- Increased Preload
- Increased MVO2
- Increased Stroke Work
- Decreased SV
- Decreased CO
- Increased work and oxygen requirements
- Significantly impaired LV function
- Decreased cardiac output and systemic perfusion
Late Inflation: After Closure of Aortic Valve

- PDP (augmentation) is decreased
  - Systolic volume has run off to peripheral circulation

Hemodynamic and Clinic Implications of Late Inflation

- PDP (augmentation) is less than optimal
  - Decreased perfusion pressure and volume to coronary arteries
Principles of IAB Deflation

- Decrease in aortic pressure during isovolumetric contraction
- Creates a potential empty space in aorta
- Allows part of LV stroke volume to be accommodated without resistance
- The decreased afterload results in increased CO and therefore decreased LVEDP

- Results in decreased diastolic pressure and decreased assisted systolic pressure
- Decreased static work

Static and Dynamic Work

- **Static Work**
  - Isometric effort requiring large amount of energy or MVO2
  - Occurs during isovolumetric contraction to develop and maintain ventricular pressure prior to aortic valve opening

- **Dynamic Work**
  - Occurs during ventricular ejection
Factors Impacting Unloading

- **Physical**
  - IAB volume
  - Occlusiveness
  - Duration of Inflation

- **Biological**
  - Arterial pressure
  - Vascular compliance
  - Cardiac reserve

Deflation Timing

- Deflation point is set to achieve two goals:
  - BAEDP < PAEDP
  - APSP < PSP
Early Deflation

- Aortic pressure is allowed to rise to the normal PAEDP
  - Blood fill in the space created by balloon deflation
- Produces a U shaped curve
- APSP = PSP
- No Cardiac Unloading
- No reduction in MVO2

Late Deflation

- Results in:
  - Increased BAEDP
  - Increased workload of LV / increased MVO2
  - Increased isovolumetric contraction time
  - Decreased CO and SV
  - BAEDP is > PAEDP
Discussion of Key Nursing Considerations

- Pressure assessment for optimization of therapy
- Balloon mobility
- Left radial pulse assessment
- Urine output
- Distal pulse assessment
- Groin care
- Platelets
- Other complications

Gratitude