CARDIAC AND VASCULAR SURGERY

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PREOPERATIVE ASSESSMENT

HISTORY AND PHYSICAL EXAM

❑ history of present illness
- inquire about symptoms of cardiac disease i.e. chest pain, dyspnea, fatigue, hemoptysis, syncope, palpitations, peripheral edema, cyanosis
- determine the degree of physical disability caused by cardiac symptoms (angina, shortness of breath (SOB), undue fatigue, palpitations) using the New York Heart Association (NYHA) functional classification (see Table 2 Cardiology Chapter)
- determine severity of angina pectoris using the Canadian Cardiovascular Society classification (see Table 1 Cardiology Chapter)

❑ past medical history
- cardiac risk factors: smoking, family history, elevated cholesterol, diabetes mellitus (DM), hypertension (HTN), +/– elevated homocysteine levels
- previous operations: thoracotomy, saphenous vein stripping/ligation, peripheral vascular surgery, carotid endarterectomy
- allergies, medications i.e. anticoagulants, antiarrhythmics, antiplatelet agents, ACE inhibitors, diuretics, etc.

❑ family and social history
- family history of coronary artery disease (CAD), congenital heart disease, Marfan syndrome, malignant hyperthermia and other hereditary disorders should be noted
- consider marital status and living conditions in discharge planning

❑ review of symptoms
- cardiovascular: past cardiac procedures, and investigations
- CNS: previous transient ischemic attack (TIA) or stroke (requires full neurologic work-up)
- respiratory: if chronic obstructive pulmonary disease (COPD) is suspected, obtain spirometry, pulse oximetry and ABG pre-op
- endocrine: DM and its complications should be noted
- hematologic: bleeding disorders, sickle cell screening if African heritage
- renal: impaired renal function and renal dialysis increase the risk of perioperative complications; renal transplant patients should be followed by renal transplant service perioperatively to manage medications
- gastrointestinal (GI): active peptic ulcer disease, active hepatitis, cirrhosis, and other GI problems can seriously affect the outcome of cardiac surgery
- peripheral vascular: venous and arterial disease should be noted; intra-aortic balloon pump insertion through a femoral artery may be difficult with aorta-iliac occlusive disease
- genitourinary (GU): prostate problems may impair Foley catheter insertion
- musculoskeletal (MSK): major skeletal deformities or active arthritic conditions may interfere with airway management, ambulating, and recovery

❑ physical examination
- height, weight, and vital signs
- examine mouth, airway, neck, chest and abdomen
- assess all peripheral pulses and auscultate the carotid and subclavian arteries for bruits
- examine saphenous veins
- perform Allen's test on both hands in case a radial artery is used as a bypass conduit for coronary artery bypass
- infection following cardiac surgery can be disastrous, therefore rule out skin infections and dental caries (especially prior to valve surgery)

CONSENT

❑ risks and benefits of surgery should be clearly outlined to the patient and his/her family (with patient's permission)

❑ serious complications should be explained, such as death, stroke, myocardial infarction (MI), infection, etc.

PREADMISSION TESTS AND ORDERS

❑ CBC, PTT, INR, electrolytes, glucose, BUN, creatinine, and other special blood tests as needed

❑ urinalysis

❑ PA and lateral CXR (+/– CT chest, especially in re-operations to determine relation of sternum to heart and ascending aorta)

❑ ECG to diagnose heart rhythm abnormalities and myocardial ischemia

❑ cross and type for 2 units PRBCs

❑ NPO after midnight

❑ Dulcolax suppository preop

❑ ancef 1 g IV to be given in OR (vancomycin 1 g IV or clindamycin 600 mg IV if penicillin allergic)

PREOPERATIVE MEDICATIONS

- ❑ All regular cardiac medications should be continued to the morning of surgery (including nitro patch), except:
  - amiodarone - should be stopped 4 weeks preop to avoid potential intraoperative problems (resistant bradycardia, hypotension, etc.)
  - ACE inhibitors - small risk of hypotension perioperatively with these drugs, therefore stop 24 hours preop (controversial)
- ❑ anticoagulants
  - warfarin - stop 4-5 days prior to surgery; admit and start on IV heparin if high risk of thrombosis large left atrium, atrial fibrillation (A fib), mitral valve prosthesis
  - heparin - IV heparin should be stopped 2-3 hours preop (unless on intra-aortic balloon pump (IABP) support)
  - ASA/Ticlodipine/NSAIDs - stop 7-10 days preop if possible
- ❑ psychotropic drugs
  - MAO inhibitors - discontinue 3 weeks preop if long acting, 1 week if short acting
- ❑ others
  - steroids and anti-rejection drugs (transplant patients) must be continued

ASSESSING RISK AND BENEFIT OF SURGERY

- ❑ ventricular function
  - the most important determinant of outcome of all heart diseases
  - patients with severe left ventricular (LV) dysfunction usually have a poor prognosis, but surgery can sometimes dramatically improve LV function
  - patients with severe LV dysfunction but good arteries to bypass usually do very well from the operative risk viewpoint, but those with bad ventricles and marginally graftable coronary arteries are usually poor surgical candidates
  - to assess viability of non-functioning myocardial segments, use thallium and sestamibi myocardial imaging, PET scanning or MRI (see Cardiology chapter)
  - surgically correcting volume overloading conditions such as aortic or mitral regurgitation (AR/MR) may not change the prognosis (due to irreversibly damaged myocardium) but symptomatology can be improved
  - depressed ventricular function caused by aortic stenosis almost always improves following relief of obstruction
- ❑ coronary artery disease (CAD)
  - isolated proximal disease in large coronary arteries (>1.0 - 1.5 mm) is ideal for bypass surgery
  - small, diffusely diseased coronary arteries are not suitable for bypass surgery
  - see Cardiology Chapter for discussion of PTCA vs. surgery
- ❑ heart valve disease
  - repair of heart valves is preferable to replacement because the ideal artificial valve has not yet been developed
  - repair is not feasible in all patients with heart valve disease, therefore valve replacement is necessary
- ❑ numerous risk factors for CABG mortality have been identified in several major studies (in decreasing order of significance)
  - urgency of surgery (emergent or urgent)
  - reoperation
  - older age
  - poor ventricular function
  - female gender
  - left main disease
  - others include catastrophic conditions (cardiogenic shock, ventricular septal rupture, ongoing CPR), dialysis-dependent renal failure, end-stage COPD, diabetes, cerebrovascular disease, and peripheral vascular disease
- ❑ commonly identified factors found to increase CABG post-operative morbidity or length of stay (in decreasing order of significance):
  - reoperation
  - emergent procedure
  - preoperative usage of intra-aortic balloon pump (IABP)
  - congestive heart failure (CHF)
  - CABG-valve surgery
  - older age
  - renal dysfunction
  - COPD
  - DM
  - Cerebrovascular disease (CVD)
- ❑ see Table 1 - Cleveland Clinic Clinical Severity Scoring System

Table 1. Cleveland Clinic Clinical Severity Scoring System

Preoperative Factor	Score	Added Score	Morbidity*	Mortality
Emergency Case	6	0-2	4-7%	0-2%
Creatinine 141-167 umol/L	1	3-5	10%	2-4%
Creatinine >168 umol/L	4	6	18%	5%
Severe LV dysfunction	3	7-9	23%	7%
Reoperation	3	10+	> 50%	> 25%
Mitral Regurgitation	3			
Age 65-74	1			
Age 75+	2			
Prior vascular surgery	2			
COPD	2			
Hematocrit <34%	2			
Aortic stenosis	1			
Weight <65 kg	1			
Diabetes	1			
Cerebrovascular disease	1			

*Morbidity defined as myocardial infarction requiring use of IABP, mechanical ventilation for 3+ days, neurological deficit, oliguric or anuric renal failure, or serious infection.
Adapted from Higgins et al. *Stratification of morbidity and mortality outcome by preoperative risk factors in coronary artery bypass patients.* JAMA. 1992:267:234-8.

COMMON POSTOPERATIVE COMPLICATIONS

ARRHYTHMIAS

❑ ventricular ectopy most common
- premature ventricular contractions (PVC's) are usually benign, but may reflect marginal coronary perfusion and ongoing myocardial ischemia
- if frequent (> 6-10/min) or multifocal PVCs, check serum electrolytes, repeat 12 lead ECG and assess hemodynamics
- treatment
  - Lidocaine 100 mg IV bolus (may repeat 50 mg IV bolus), followed by drip at 1-4 mg/min
  - Magnesium sulfate 2-4 g IV, may repeat
  - Amiodarone 150 mg IV slowly over 15-30 minutes, then 900-1,200 mg (in 250 cc D5W) over 24-48 h
  - cardioversion needed if progresses to symptomatic ventricular tachycardia (VT) or if patient develops ventricular fibrillation (V fib)
  - atrial or atrioventricular pacing at a slightly higher rate may suppress ectopy

❑ nodal or junctional rhythm
- treatment may not be necessary (assure no hypotension)
- rule out digoxin toxicity, make certain serum $K^+ > 4.5$, rule out hypomagnesemia
- may require A-V sequential pacing if loss of atrial kick has significant hemodynamic sequelae

❑ supraventricular tachycardia (SVT) - includes atrial fibrillation (A fib) and flutter
- onset may be heralded by multiple premature atrial contractions (PAC's)
- atrial ECG using atrial pacing leads often helpful in distinguishing fibrillation from flutter during rapid rates
- treatment of A fib
  - digoxin used to control rate - 0.5 mg IV once, then 0.25 mg q6h x 2, then 0.125-0.375 mg PO daily depending on body weight and renal function
  - if no asthma/COPD: metoprolol 5 mg IV q15min x 3, then 25-50 mg PO bid
  - if asthma/COPD: diltiazem 0.25 mg/kg IV bolus (further 0.35 mg/kg bolus if inadequate response), then switch to amiodarone 400 mg PO tid x 3-5 days, then 200 mg PO od after loading dose
  - if unstable or Grade IV LV: consider amiodarone 150 mg IV bolus (can repeat), then 900 mg IV over 24 h, then switch to PO amiodarone as above
- treatment of atrial flutter
  - rapid atrial pacing > 400 bpm
  - digitalization followed by IV beta blocker
  - IV verapamil followed by digitalization (calcium channel blockers must be used judiciously as wide complex SVT can mimic VT)
- in both instances, the arrhythmia should be treated with synchronous direct current (DC) cardioversion at 25-50 joules should there be a significant drop in blood pressure (BP) or cardiac output (CO)
- never give IV CCB and B blocker together
- adenosine can be used as a diagnostic and therapeutic intervention (transient bradycardia/asystole to allow interpretation of rhythm, may be therapeutic)

BLEEDING

- ❑ causes include medications, clotting deficits, prolonged operation, emergency surgery, technical factors, deep hypothermia, renal impairment, and transfusion reactions
- ❑ patients at high risk for bleeding complications: endocarditis, aortic dissection, redo cases
- ❑ treatment
  - assure normothermia
  - measure clotting factors stat: INR, PTT, fibrinogen, platelet count, activated clotting time
  - tranexamic acid bolus (50 mg/kg) occasionally given if > 150 cc/h chest tube output
  - correct with fresh frozen plasma, cryoprecipitate, platelets, DDAVP, protamine for continued heparinization
  - transfusion reaction protocol if suspected
- ❑ indications for surgical exploration of post-operative hemorrhage
  - mediastinal tube output > 300 cc/h despite correction of clotting factors
  - 1.5% rate for CABG, 4% rate for valve surgery
  - technical factors found as cause > 50% of time

RENAL FAILURE

- ❑ incidence is 0.3-1%
- ❑ diagnosis - prerenal vs. renal vs. postrenal
- ❑ management
  - optimize volume status and cardiac output
  - discontinue nephrotoxic drugs (indomethacin, aminoglycosides, ACE inhibitors)
  - maintain urine output > 40 cc/h using low-dose dopamine (1-3 ug/kg/h), furosemide 10-300 mg IV bolus +/– 10-20 mg/h drip, or ethacrynic acid (50-100 mg IV bolus) as indicated
  - furosemide/mannitol drips if persistent oliguria
  - dialysis
    - continuous arterial-venous hemodialysis (CAVHD) or continuous venous-venous (CVVHD) approach are most suitable for hemodynamically unstable patients
    - for hemodynamically stable patients, consider intermittent hemodialysis or peritoneal dialysis (peritoneal cavity may communicate with mediastinum and be ineffective)
- ❑ outcome
  - mortality rates 0.3-23% depending on the degree of azotemia
  - if dialysis is required, mortality ranges from 27-53%

RESPIRATORY FAILURE

- ❑ mechanical - mucous plugging, malpositioned endotracheal tube, pneumothorax, pre-existing COPD, bronchospasm
- ❑ intrinsic - volume overload, pulmonary edema, atelectasis, pnemonia, pulmonary embolus (uncommon), acute respiratory distress symptom (ARDS)
- ❑ management
  - examine patient and evaluate CXR for correctable causes
  - if intubated: add positive end-expiratory pressure (PEEP) (7.5-10 cm H_2O), increase % oxygen inspired (FiO_2), diuresis, consider bronchoscopy with lavage for sputum, bronchodilators
  - if extubated: pain control, chest physio, diuresis, increase FiO_2, facial continuous positive air pressure (CPAP), bronchodilators
  - if pneumonia: sputum culture and gram stain, bronchoscopy, consider antibiotics early if prosthetic materials in heart

LOW CARDIAC OUTPUT (CO)

- ❑ cardiac index < 2.0 L/min/m^2
- ❑ signs - decreased urine output, acidosis, hypothermia, altered sensorium, cool clammy skin
- ❑ assessment - heart rate and rhythm (ECG: possible acute MI), preload and afterload states (Swan-Ganz catheter readings), measurement of CO
- ❑ treatment
  - stabilize rate and rhythm
  - optimize volume status, systemic vascular resistance (SVR)
  - consider ECHO to rule out tamponade
  - give calcium chloride 1 g IV until more definitive diagnosis reached
  - correct acidosis, hypoxemia if present (CXR for pneumothorax)
  - inotropic agents if necessary - see Table 4
    - Dopamine: increases SVR, protects renal function (increases renal blood flow, GFR and sodium excretion), produces tachycardia only in high doses
    - Dobutamine: increases CO, decreases LV pressure, decreases SVR
    - Epinephrine: increases heart rate, contractility and stroke volume (SV), decreases urine flow
    - Norepinephrine: increases mean arterial pressure (MAP) with less increase in HR compared to epinephrine
    - Milrinone: improves cardiac output and myocardial contractility, decreases systemic and pulmonary vascular resistance without increasing HR, used for right ventricle (RV) failure or high pulmonary artery (PA) pressure
    - Amrinone: similar to milrinone but more prone to cause arrhythmias in high doses
  - persistent low cardiac output despite inotropic support requires placement of IABP

- ❑ intra-aortic balloon pump (IABP)
  - augments cardiac function without increasing oxygen demand
  - inflation during diastole augments coronary perfusion and oxygen delivery
  - deflation during systole reduces afterload, therefore decreasing oxygen demand
  - indications: unable to wean from cardiopulmonary bypass, cardiogenic shock unresponsive to medical therapy, low output syndrome, unstable angina, ventricular tachyarrhythmias caused by ischemia, bridge to transplantation
  - contraindications: aortic regurgitation, aortic dissection, aortic aneurysm (thoracic or abdominal), severe peripheral vascular disease (consider transthoracic route instead), severe blood dyscrasias

Table 2. Adrenergic Catecholamine Receptor Activity

Agent	α1	α2	β1	β2	Dopamine
Dopamine	++	+	+	+	+++
Dobutamine	⊖	⊖	+++	++	⊖
Epinephrine	+++	+++	++	++	⊖
Norepinephrine	+++	+++	+	⊖	⊖

Adapted from Cheng DCH, *David* TE *eds. Perioperative care in cardiac anesthesia and surgery. Austin: Landes Bioscience,* 1999.

CARDIAC TAMPONADE

- ❑ onset - suggested by increased filling pressures (> 20 mmHg) with decreased CO, decreased urine output (U/O), hypotension, poor peripheral perfusion, pulsus paradoxus, quiet and distant heart sounds, absence of chest tube drainage, eventual equalization of right and left-sided atrial pressures
- ❑ high degree of suspicion when coincides with excessive post-operative bleeding
- ❑ echocardiogram if high index of suspicion; CXR may demonstrate wide mediastinum
- ❑ treatment
  - emergent re-exploration is treatment of choice and may be needed at bedside for sudden hemodynamic decompensation
  - transfuse to optimize volume status and inotropic support
  - avoid increased PEEP

PERIOPERATIVE MYOCARDIAL INFARCTION (MI)

- ❑ incidence: 2.4% CABG, 1.3% valve surgery
- ❑ diagnosis: new onset Q waves (or loss of R waves) post-operatively, new ST segment elevation, serial isoenzymes (CK-MB, troponin), segmental wall motion abnormalities by ECHO
- ❑ treatment
  - vasodilation (IV nitroglycerine is preferred to nitroprusside)
  - intra-aortic balloon pump if continued hemodynamic deterioration (unloads the ventricle, and may preserve non-ischemic adjacent myocardium)
- ❑ outcome - associated with increased morbidity and mortality as well as poorer long-term results

HYPERTENSION (HTN)

- ❑ incidence 30-50% following cardiopulmonary bypass
- ❑ predisposes to bleeding, suture line disruption, aortic dissection, increased myocardial oxygen requirements, depressed myocardial performance
- ❑ treat if systolic pressure > 130 mmHg
- ❑ treatment
  - resume preoperative medications when tolerated
  - nitroglycerin 100 mg in 250 cc D5W at 5-50 cc/h (good for ischemia or high filling pressures)
  - sodium nitroprusside 50 mg in 250 cc D5W at 5-50 cc/h (good to reduce afterload)
  - beta-blockers (esmolol 10-20 mg IV bolus, metoprolol 1-5 mg IV bolus, etc.)
  - CCB (nifedipine 10 mg sublingual for arterial spasm, diltiazem 5-10 mg IV q1h)
  - ACE inihibitor if poor LV function and good renal function

POSTOPERATIVE FEVER

- ❑ definition: core body temperature > 38.0ºC
- ❑ common in first 24 h post-operatively
- ❑ etiology unknown but likely due to atelectasis or may be associated with pyrogens introduced during cardiopulmonary bypass
- ❑ treat with acetaminophen, add cooling blankets (associated hypermetabolism and vasodilation may be detrimental to hemodynamic status and increase myocardial work)
- ❑ post-operative patients receive cefazolin 1 g IV q8h (total 3-6 doses), or if pen-allergic then vancomycin 1 g IV q12h (1 dose CABG, 2 doses valve) or clindamycin 600 mg IV q8h (6 doses)
- ❑ if patient febrile beyond 24 hours, culture urine, blood, sputum, and check WBC count

- ❑ consider early antibiotic treatment if patient has prosthetic material
- ❑ arterial and central lines should be changed prophylactically every 7-10 days unless obviously infected
- ❑ inspect sternal wound daily for drainage and stability (sternal wound infections are disastrous and require operative debridement, 0.8% incidence)
- ❑ post-pericardiotomy syndrome
  - characterized by low grade fever, chest pain, malaise and pericardial rub on auscultation
  - usually 2-3 weeks post-op
  - treatment - NSAIDs, rarely steroids are necessary

CNS COMPLICATIONS

- ❑ causes - pre-existing CVD, prolonged cardiopulmonary bypass, intra-operative hypotension, emboli (air or particulate matter)
- ❑ transient neurologic defecit - occurs in up to 12% of patients, with improvement usually within several days
- ❑ permanent deficit - suspect in patients with delayed awakening post-op or with pathologic reflexes present
- ❑ stroke incidence: 1.4% CABG, 2.5% valve surgery, 6.6% aorta surgery
- ❑ CT scan early for suspected localized lesion, EEG in patients with extensive dysfunction
- ❑ treatment - optimize cerebral blood flow, avoid hypercarbia
- ❑ post-cardiotomy psychosis syndrome
  - incidence 10-24%
  - begins post operative day (POD) #2 with anxiety and confusion, progressing to disorientation and hallucinations
  - rule out organic causes of delirium: substance withdrawal, hypoxemia, hypoglycemia, electrolyte abnormalities, etc.
  - treatment - rest and quiet environment, Haloperidol 2-10 mg IV q1h PRN
- ❑ post-op seizures
  - check serum electrolytes (calcium, magnesium, sodium) and glucose
  - treatment: diazepam 2-5 mg IV or lorazepam 1-2 mg IV, then phenytoin 10-15 mg/kg IV load with 3-5 mg/kg/day maintenance

CARDIAC ANESTHESIA

PREOPERATIVE

- ❑ preoperative patient education of the perioperative course is important to reduce anxiety and establish patient's expectations
- ❑ preadmission clinic and same day admission reduce hospital length of stay and reduce delay from last minute abnormal blood tests or suboptimal clinical condition of patients

INTRAOPERATIVE ANESTHESIA

- ❑ premedication: lorazepam 1-3 mg sublingually, 1 h preoperation
- ❑ prophylactic antifibrinolytic treatment with tranexamic acid 50-100 mg/kg IV intraoperatively (reduces perioperative blood loss)
- ❑ induction: propofol (0.5 mg/kg) or thiopental (1 mg/kg), low dose narcotic (fentanyl total 10-15 ug/kg), pancuronium (0.15 mg/kg), midazolam (1-3mg)
- ❑ precardiopulmonary bypass: isoflurane (0.5-2%), midazolam (total 0.07-0.1 mg/kg)
- ❑ a baseline activated clotting time (ACT) is drawn after operation has commenced, and then heparin 3-4 mg/kg (300 units/kg) is administered prior to cannulation of cardiopulmonary bypass to maintain ACT over 480 seconds
  (minimizes activation of coagulation system and formation of fibrin monomers)
- ❑ to reduce risk of myocardial necrosis and ventricular arrhythmias during prebypass period, control myocardial oxygen demand by keeping heart rate less than 90 and systolic pressure less than 130 mmHg (pulse-pressure product < 12,000)
- ❑ maintain stable hemodynamics and aggressively control arrhythmias
  - use fluids and alpha-agents to counteract vasodilation, beta-blockers or additional anesthetic agents for hypertension or tachycardia, and nitroglycerin for ischemia
- ❑ during cardiopulmonary bypass: propofol infusion 2-6 mg/kg/h
- ❑ postcardiopulmonary bypass: postoperative analgesia is essential (indomethacin or diclofenac 50-100 mg PR unless contraindicated) and sedation (propofol) are titrated to allow for early tracheal extubation (within 1-6 hours)

PRINCIPLES OF CARDIOPULMONARY BYPASS

INTRAOPERATIVE MONITORING

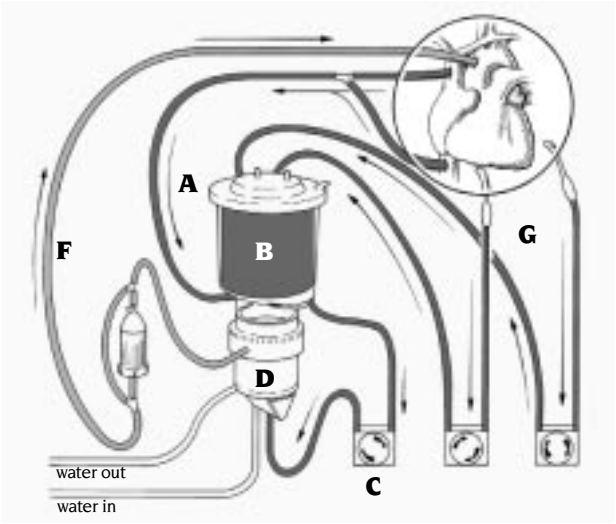
- ❑ standard monitors include:
  - five-lead ECG monitoring with ST segment analyses (leads II, V5)
  - noninvasive blood pressure measurement
  - direct arterial pressure
  - pulse oximetry
  - end tidal gas analysis (capnography)
  - temperature (nasopharyngeal in all patients, also rectal in infants)
  - urine Foley catheter
  - central venous pressure (CVP) (assess filling of right ventricle)
- ❑ commonly used monitors
  - Swan-Ganz catheter
    - monitors central venous pressure (CVP), pulmonary artery pressure,pulmonary capillary wedge pressure (PCWP), CO (as needed), and mixed venous gas measurements (as needed)
    - indications for use: LV dysfunction (EF<40%), poor distal vessels (high risk of poor revascularization), preoperative hemodynamic instability, anticipated long cardiopulmonary bypass time, significant coexistent medical disease (pulmonary, cerebral, renal)
  - transesophageal echocradiography (TEE)
    - indications: significant atheromatous disease of aorta, mitral valve repair, aortic valve repair/replacement, adult/pediatric congenital heart disease, ventricular myomectomy, ventricular remodeling procedures, endocarditis, cardiac tumour resection, heart transplant, perioperative cardiovascular instability or difficult cardiopulmonary bypass separation
    - contraindications: esophageal pathology (stricture, cancer, diverticulae), c-spine instability, lack of informed consent
- ❑ monitors added at conclusion of operation:
  - pacing wires (by convention atrial on right side and ventricular on left)
  - mediastinal and pleural chest tubes

POSTOPERATIVE

- ❑ accelerated weaning and tracheal extubation of patients allows for earlier chest tube removal, mobilization, and oral intake of food on POD #1, facilitating ICU and early hospital discharge
- ❑ mediastinal tubes are discontinued when drainage is less than 200 cc/8 h and no air leak is present (usually POD #1-2)
- ❑ if no arrhythmias, pacing wires are removed at 4 days or the day prior to discharge

PRINCIPLES OF CARDIOPULMONARY BYPASS

- ❑ cardiopulmonary bypass (CPB) and cardioplegia provides a still bloodless heart by diverting blood into a heart-lung machine (extracorpeal circuit) that performs the functions of respiration, circulation and temperature regulation while the heart and lungs are not functioning
- ❑ CPB is required when manipulation of the heart significantly compromises systemic blood pressure and when intracardiac surgery is performed
- ❑ the cardiopulmonary circuit (see Figure 1)
  - venous blood drains by gravity from the right atrium (RA) or vena cavae into a reservoir, passes through an oxygenator/heat exchanger attached to a heating/cooling machine, and is returned to the arterial system through a filter using either a roller or centrifugal pump
  - the membrane oxygenator is ventilated with 100% O_2, run through a blender to control the pO_2 between 100-200 mmHg and pCO_2 between 35-40 mmHg
  - a priming solution (mostly crystalloid) is circulated through the lines to remove air
  - the arterial cannula is usually placed in the ascending aorta or arch
  - occasionally, the arterial cannula is placed in the femoral or axillary artery (e.g. aortic dissection surgery)
  - additional suction lines can be used for intracardiac venting and scavenging of blood from the operative field, and this blood passes through a microporous filter (to remove particulate matter) before returning to the cardiotomy reservoir



Blood drains by gravity through the
(A) venous lines into a
(B) cardiotomy reservoir, is
(C) pumped through the
(D) oxygenator/heat exchanger and arterial line filter back into the
(F) arterial circuit. Additional suction lines
(G) can be used for intracardiac venting and scavenging of blood from the operative field.

Figure 1. The Cardiopulmonary Circuit

Illustration by Ken Vanderstoep

❑ initiating bypass
- 3-4 mg/kg heparin is administered systemically, and the activated clotting time (ACT) is monitored (a value over 400 is required before bypass is started)
- blood pressure is usually maintained between 55-65 mmHg using vasodilators or vasopressors (cerebral blood flow is usually maintained by autoregulation until the pressure falls below 40 mmHg)
- the lungs are not ventilated during bypass
- pump flows are usually around 2.24 L/min/m2 and is non-pulsatile (pulsatile flow used if significant renal disease)
- the patient may be warmed or cooled depending on the procedure and the surgeon's preference

❑ terminating bypass
- the patient is warmed to normothermia
- air is removed from the LV and aorta with a needle or venting cannula
- the lungs are ventilated and cardiac pacing is initiated as necessary
- the heart is filled by restricting venous return as bypass flow is reduced and turned off
- alpha agents (e.g. dopamine 1-5 ug/kg/min) and calcium chloride (1 g) are often used to improve contractility and systemic blood pressure and facilitate weaning from CPB
- inotropic support is considered for poor cardiac performance
- when the patient is stable, protamine is administered to reverse the heparin effect (1.0 to 1.5 times the original heparin dose), and the cannulas are removed
- blood remaining in the oxygenator and lines at the end of bypass (as well as shed mediastinal blood) can be transfused back to the patient at the conclusion of the operation
- see Table 3 - Postoperative Orders

❑ adverse effects of CPB
- CPB activates numerous cascades (coagulation, complement, fibrinolytic, and kallikrein systems)
- proinflammatory cytokines are released that can cause a systemic inflammatory response and contribute to myocardial reperfusion damage, lung injury, and generalized capillary leak
- CPB can also cause a coagulopathy (dilution of clotting factors and platelets, platelet dysfunction) and renal and splanchnic hypoperfusion

❑ circulatory arrest
- necessary for some operations on the ascending aorta and all aortic arch operations to allow the surgeon to operate without the constraints of vascular clamps and a blood obscured field
- the patient is cooled systemically to 18-20ºC at which the EEG is flat, and the head is packed with ice
- the arterial line is clamped, the CPB machine is turned off, and blood is drained from the circulation (the venous line is intermittently clamped to prevent excessive drainage from the patient)
- after completing the distal anastomosis of the prosthetic graft to the distal ascending/ transverse aorta, the arterial inflow cannula is positioned through the graft, the open end of the prosthetic graft is clamped, and full bypass is resumed before the proximal anastomosis is performed
- the "safe" upper limit for circulatory arrest is 45-60 minutes at 18ºC
- administering blood retrograde into the brain through a cannula in the superior vena cava (SVC) may extend this safe upper limit by providing additional cooling and possibly some oxygen and nutrition to the brain
- retrograde perfusion also maintains cerebral hypothermia and flushes air and debris out of the cerebral vessels

Table 3. Intensive Care Unit (ICU) Post-operative Orders for Cardiac Surgery

1. Vital signs q15min, then q1h when stable
2. Urine output q1h until extubation, then q2h
3. Chest tubes at -20 cm H_2O suction. Record chest tube loss q15min x 1h, then q1h if hemodynamically stable
4. Auto-transfuse chest drainage
5. Cardiac output calculations now and q6h
6. Check peripheral pulses q1h x 4 then q4h
7. Central line IV D5W TKVO
8. K+ replacement - to be decided depending on urine output and last K^+ value
9. Peripheral IV NS TKVO
10. 12 lead ECG now then daily x 3 days
11. Ventilation e.g. VT 700 mL, FiO_2 50%, Rate 12/min, Peep 5 cm/H_2O to keep $PaCO_2$ between 35-45 mmHg
12. Titrate FiO_2 to keep PO_2 > 90 mmHg or O_2 sat > 95%
13. Suction ETT prn and chest care as per assessment
14. Physiotherapy: assessment and treatment
15. Pacemaker connected and checked by MD
16. Morphine sulphate 1-6 mg IV q1h PRN
17. Indomethacin supp 50-100 mg pr q1h x 2 PRN (avoid if diabetic, renal failure, peptic ulcer disease, or age > 75)
18. Gravol 25-50 mg IV/IM q4h PRN x 2 days
19. Cefazolin 1 g IV q8h in 50 cc D5W x 3 doses (Vancomycin 300 mg to 1 g IV q12h if pen-allergic)
20. Propofol 200 mg IV in 100 cc D5W PRN
21. Sodium nitroprusside 50 mg IV in 250 cc D5W to keep SBP < 140 mmHg
22. Nitroglycerin 100 mg IV in 250 cc D5W PRN
23. Dopamine 200 mg IV in 250 cc D5W PRN to keep SBP > 90 mmHg

MYOCARDIAL PROTECTION AND CARDIOPLEGIA

- ❑ nearly all intracardiac procedures and most coronary bypass operations require a quiet, bloodless field that allows for precise surgical techniques
- ❑ cardioplegia is used to arrest the heart (reducing the oxygen demand of the heart nearly 90%), and hypothermia is used to further reduce myocardial metabolism
- ❑ without this reduction in myocardial metabolism by hypothermia or chemical cardiac arrest, crossclamping the aorta for more than 20 minutes would result in myocardial anaerobic metabolism and severe myocardial dysfunction and necrosis
- ❑ blood cardioplegia is used (rather than crystalloid solutions) because it improves postoperative ventricular function by increasing oxygen delivery and preserving myocardial high-energy phosphate stores
- ❑ a wide variety of cardioplegia solutions exist
  - one example is a blood-crystalloid solution mixed at a ratio of 8:1, with a crystalloid composition of 6 meq/L magnesium sulfate, 50 mmol/L dextrose and potassium (30 meq/L KCl for cardiac arrest, 8 meq/L for maintenance) in sterile water
- ❑ cardioplegia can be administered at various temperatures
  - cold cardioplegia (4ºC)
    - advantages: decreases myocardial metabolism, allows for prolonged cardioplegic interruptions to facilitate exposure for complex surgical procedures
    - disadvantages: delays recovery of cardiomyocyte metabolism and function
  - warm cardioplegia (37ºC)
    - advantages: rapid recovery of myocardial metabolism, metabolic enhancement for patients with active ischemia
    - disadvantages: requires near continuous delivery because of the tendency for the heart to resume electrical activity at normothermia, continuos delivery may obscure operative field, questionable increased risk of neurologic injury
  - tepid cardioplegia (29ºC)
    - may optimize myocardial protection

MYOCARDIAL PROTECTION AND CARDIOPLEGIA . . . CONT.

- ❑ cardioplegia can be administered antegrade via the aortic root or coronary ostia, retrograde via the coronary sinus, or combined (antegrade via completed saphenous vein grafts plus retrograde)
    - antegrade
        - advantages: simple to use
        - disadvantages: suboptimal perfusion distal to coronary occlusions, risk of coronary embolization in redo CABG, frequent interruptions (administered intermittently to maintain cardiac arrest), aortic root cardioplegia contraindicated in severe aortic insufficiency (but can administer via coronary ostia)
    - retrograde
        - advantages: allows for near continuous delivery
        - disadvantages: decreased perfusion of right ventricle and posterior interventricular septum, large amount of nonnutritive flow through Thebesian channels, damage to coronary sinus at pressures > 40 mm Hg
    - combined
        - advantages: maximizes myocardial perfusion
        - disadvantages: relatively complex delivery system
- ❑ with near continuous cardioplegic delivery, the large volumes of cardioplegia administered may result in hyperkalemia (consider treating with furosemide 40 mg IV and/or insulin 10 units IV)

CORONARY ARTERY BYPASS GRAFT (CABG) SURGERY

- ❑ the objective of CABG is complete revascularization of the myocardium
- ❑ arteries with severe stenoses (> 50% diameter reduction) are bypassed, except those of small caliber (< 1mm in diameter)
- ❑ indications
    - class 3 or 4 chronic stable angina, either to improve prognosis and/or relieve symptoms
    - unstable angina refractory to medical management
    - acute ischemia or hemodynamic instability post-PTCA (rate of emergency surgery post-PTCA is 3-4%, with 5-6% operative mortality)
    - post-MI angina
    - acute evolving infarction within 4-6 hours of onset
    - ventricular arrhythmias with coronary disease
    - markedly positive stress test before major intra-abdominal or vascular surgery
    - ischemic pulmonary edema
        - usually left main or triple vessel disease, impaired LV function (70% 2 year mortality managed medically)
    - to improve survival in patients (even if asymptomatic)
        - left main stenosis > 50% (annual mortality 10-15%)
        - left main equivalent: > 70% stenosis of proximal left anterior descending (LAD) and proximal circumflex artery (PCA)
        - three vessel disease with ejection fraction (EF)< 50%
        - three vessel disease with EF> 50% but significant inducible ischemia
        - one and two vessel disease with extensive myocardium at risk but lesions not amendable to PTCA
    - other
        - significant coronary lesions accompanying other cardiac lesions requiring surgical correction
        - congenital coronary anomalies
- ❑ approach to reading coronary angiograms
    - **see selected angiograms in Colour Atlas (see Colour Atlas CS1-CS3)**
    - angiographic atherosclerotic stenoses are expressed as a percent reduction of the lumen diameter compared to an area of adjacent normal vessel
    - the catheter usually is a size 6 French catheter (1.8 mm in diameter) - comparing the size of vessels to the size of the catheter can help distinguish graftable from non-graftable (< 1 mm) vessels
    - right coronary artery divides to posterior interventricular (PIV) artery (also known as posterior descending artery) and posterolateral branches (PIV can be identified by the presence of septal perforating branches)
    - left main coronary artery divides to the LAD and circumflex (LAD can be identified by the presence of septal perforating branches and its approach towards the apex)
    - left ventriculogram assesses the global and regional contractile performance of the heart, and presence and degree of MR
- ❑ surgical approach (see Figure 2)
    - right coronary artery - common site of distal anastomosis is just before the bifurcation at the crux, or directly to the posterior descending artery if the bifurcartion is diseased
    - left main coronary artery - not directly grafted because it is inaccessible without dividing the aorta and pulmonary artery (therefore graft LAD and obtuse marginals of circumflex for left main stenosis)
    - left anterior descending (LAD) - distal anastomosis is usually placed from midpoint on; diagonal branches > 1-1.5 mm in diameter are also bypassed especially if they are diseased
    - circumflex coronary artery - difficult to approach since located below great cardiac vein, therefore bypass circumflex artery system by grafting to obtuse marginal branches

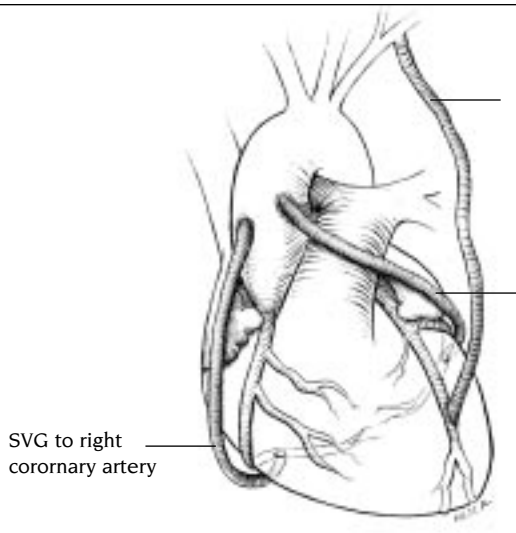


Figure 2. Coronary Artery Bypass Grafting

Illustration by Mi Ji Kim

❑ conduits for coronary bypass surgery
- saphenous vein grafts
  - at 10 years, 50% occluded, 25% stenotic, 25% angiographically normal
- internal thoracic (mammary) artery - anastamosed to LAD
  - at 10 years, 90-95% patency
  - increased 10-year patient survival
  - improved event-free survival (angina, MI)
  - no increase in operative risk
  - contraindications: inadequate mammary blood flow, occluded abdominal aorta, chest wall irradiation, avoid bilateral internal thoracic arteries in diabetics or obese patrients
- right gastroepiploic artery
  - good long term patency
  - usually long enough to bypass any of the 3 vessels
  - concerns: can not use as free graft, laparatomy incision (increased morbidity), arterial graft (therefore vasoreactive postoperatively)
- radial artery
  - time consuming to harvest
  - approximately 90% patency at 5 years
  - prone to severe vasospasm postoperatively ("no touch" technique during harvest of the radial artery pedicle) - avoid with CCB (Adalat XL 20 mg PO OD) for 6 months

❑ redo bypass grafting
- operative mortality 2-3 times higher than first operation
- 10% perioperative MI rate
- reoperation undertaken only in symtomatic patients who have failed medical therapy and in whom angiography has documented progression of the disease
- increased risk with redo-sternotomy
  - adhesions may result in laceration to aorta, RV, LITA and other bypass grafts (consider preop CT chest)
- myocardial protection is very important
  - cardioplegia should be given through the aortic root through the new bypass grafts as constructed and retrograde via the coronary sinus to protect ischemic areas
- distal and proximal incision of old atherosclerotic grafts should be undertaken as early as possible on CPB to prevent atherosclerotic emboli down these grafts
- consider using right or left IMA's, lesser or greater saphenous veins if available, or right gastroepiploic artery
- only arteries that are > 1.5 mm in diameter should be grafted at the second operation

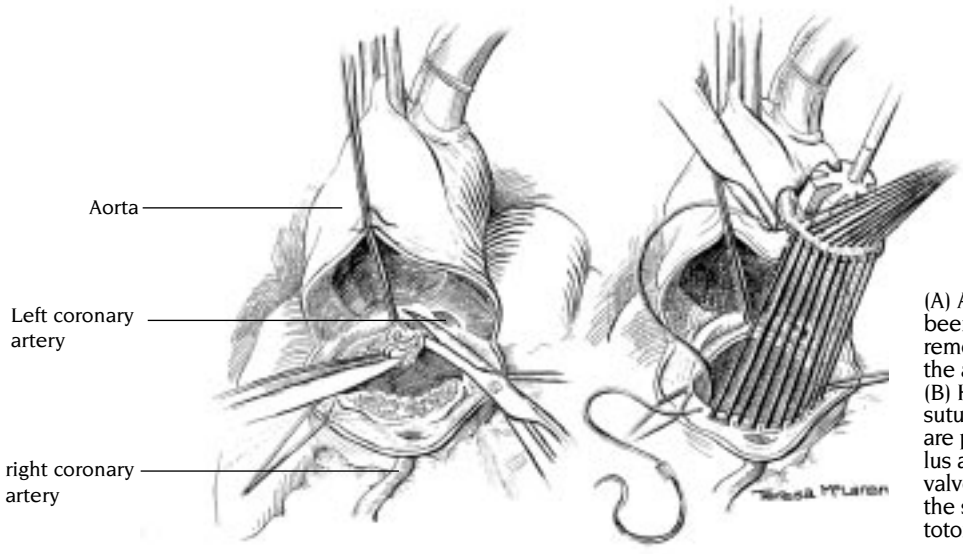
VALVE REPLACEMENT AND REPAIR

❑ see Cardiology Chapter for etiology, symptoms, imaging and medical management of valvular disease

AORTIC STENOSIS (AS)

❑ indications for surgery
- symptomatic patients with valve gradient of > 50 mm Hg or valve area < 0.8 cm^2 (normal 3-4 cm^2)
- asymptomatic patients with significant stenosis and LVH should also be considered
- moderate AS if other cardiac surgery is required (i.e. CABG)

❑ surgical options
- balloon valvuloplasty
  - for critically ill patients with end-stage AS as a "bridge" to aortic valve replacement
  - also considered in pregnancy or if significant comorbidity and high surgical risk
  - 50% recurrence of AS in 6 months
- decalcification/debridement
  - in patients with mild to moderate AS in whom the primaryindication for surgery is coronary artery disease
- commissurotomy
  - useful in a small percentage of patients with aortic rheumatic valve disease with a trileaflet valve and minimal to no calcification
- valve replacement
  - practically all patients with severe AS require aortic valve replacement (see Figure 3)



(A) An aortotomy incision has been made, the valve is removed with scissors, and the annulus is debrided. (B) Horizontal mattress sutures (often with pledgets) are placed through the annulus and the sewing ring of the valve. The valve is seated, the sutures tied, and the aortotomy incision is closed.

Figure 3. Aortic Valve Replacement:

Illustration by Teresa McLaren

AORTIC REGURGITATION (AR)

❑ indications for surgery
- acute AR with CHF
- class III-IV symptoms
- endocarditis with hemodynamic compromise or recurrent emboli
- evidence of LV decompensation in the asymptomatic patient
  - EF <55%
  - end-diastolic dimension > 70 mm
  - end-systolic dimension > 55 mm

❑ surgical options
- valve repair
  - involves resection of portions of the valve leaflets and reapproximation to improve leaflet coaptation (especially for bicuspid valves), often with a suture annuloplasty
  - valuable in younger patients
- valve replacement
  - practically all patients with AR require aortic valve replacement
- Bentall procedure
  - a valved conduit is used if an ascending aortic aneurysm (annuloaortic ectasia) is also present

CHOICE OF AORTIC VALVE PROSTHESIS

- ❑ the aortic valve can be replaced with either a valve (mechanical or porcine bioprosthetic tissue valve) or graft (autograft or homograft)
- ❑ mechanical valve
  - durable valves but require continuous anticoagulation with coumadin (contraindicated if previous bleeding history), requiring patient to take daily medication and have periodic blood tests (to maintain INR 2-3)
  - in carefully anticoagualated patients, the risk of hemorrhage is 1-2% per year, and the risk of thromboembolic events is 1-3% per year
  - preferred valve replacement if long life expectancy or if risk of reoperation is considered high
  - preferred valve replacement if small aortic root (bioprosthetic aortic valve placement in a small aortic orifice may result in obstruction and unacceptably high gradients)
- ❑ bioprosthetic valve
  - low embolic rate in the absence of anticoagulation (1-2% risk of thromboembolic events)
  - less durable than mechanical valves and require reoperation due to degeneration and structural failure
  - however, structural degeneration of bioprosthetic valves is rare in elderly patients
  - preferred valve replacement if life expectancy of patient is shorter than the known durability of the bioprostheses
  - also considered if potential for pregnancy (coumadin is teratogenic)
- ❑ pulmonary autograft
  - Ross procedure: replace the diseased aortic valve with the patient's own pulmonary valve and implant a semilunar valve homograft (e.g. pulmonary valve homograft) in the pulmonary position
  - pulmonary autograft failure is rare in carefully selected patients, but 20% will require reoperation at 10 years because of stenosis of the pulmonary homograft
  - technically demanding operation
  - ideal for children and young adults to avoid anticoagulation
  - contraindicated in patients with dilated aortic root (Bentall procedure recommended instead)
- ❑ aortic homograft
  - particularly suitable for children, women of child-bearing age, and patients with active endocarditis (e.g. aortic root abscess)
  - durability is limited with 20% 10 year reoperation rate (higher in younger patients)
  - procurement a problem: valves from donors older than 40 years of age are often not good

MITRAL STENOSIS (MS)

- ❑ indications for surgery
  - MV area < 1.5 cm^2 (normal is 4-6 cm^2)
  - NYHA classes III-IV
  - NYHA class II when MV area < 1 cm^2 (critical mitral stenosis)
  - history of atrial fibrillation and/or systemic emboli (from left atrial thrombus)
  - worsening pulmonary hypertension
- ❑ surgical options
  - percutaneous balloon mitral valvuloplasty
    - for young rheumatic patients with pure MS and good leaflet pliability, minimal chordal thickening and good subvalvular mechanism
    - also considered in pregnant patients with critical MS in whom CPB should be avoided
    - contraindicated if left atrial thrombus
  - open mitral commisurotomy
    - for patients with mild calcification and mild leaflet/chordal thickening +/– other coexistent diseased valves (e.g. aortic and/or tricuspid)
    - technique involves incision of both commissures, incision/resection of fused chordae, and occasionally incision into papillary muscle to increase mobility (if evidence of chordae shortening from scarring and fibrosis)
    - 50% of patients will require reoperation 8 years following initial commisurotomy due to restenosis
  - valve replacement
    - for moderate to severe calcification with severely scarred valve leaflets or subvalvular apparatus

MITRAL REGURGITATION (MR)

❑ indications for surgery
- acute MR associated with CHF or cardiogenic shock
- acute endocarditis with hemodynamic compromise or recurrent emboli
- NYHA class III-IV
- Class I-II symptoms with onset of atrial fibrillation or evidence of deteriorating LV function
  - EF < 55%
  - end-diastolic dimension > 75 mm
  - end-systolic dimension > 45 mm

❑ surgical options
- valve repair
  - applicable to more than 75% of patients with MR
  - the ideal pathology for mitral valve repair is myxomatous degeneration of the MV
  - several techniques include annuloplasty rings, leaflet repairs, patch repair (for endocarditis), and chordal transfers, shortening or replacement
  - prolapse of the posterior leaflet is usually corrected by rectangular resection of the prolapsing segment and plication of the annulus
  - prolaspe of the anterior leaflet is corrected by transposition of chordae from the posterior leaflet (neo-cordae)
  - chordal enlongation is corrected by invaginating the excess length of chordae into a trench in the papillary muscle
  - a ring annuloplasty or Gortex is often used in MV repair to reshape the annulus to its normal elliptical configuration and to maintain stability
- valve replacement
  - indicated only when satisfactory repair cannot be accomplished
  - most patients with MR due to ischemic heart disease, rheumatic heart disease or advanced myxomatous disease need MV replacement
  - replacement usually required if heavily calcified annulus or if papillary muscle rupture
  - chordal preservation of the posterior leaflet should be strongly considered for all MV replacements (to improve ventricular function and minimize risk of posterior LV wall rupture)

❑ the advantages of repair vs. replacement are the low rate of endocarditis and lack of need for long-term anticoagulation

CHOICE OF MITRAL VALVE PROSTHESIS

❑ the current choice for mitral valve replacements include mechanical prostheses (e.g. ball valve, tilting disc, bileaflet, etc.) and bioprosthetic valves

❑ the main factors affecting choice of prosthesis are anticoagulation, and the attitude of the patient and surgeon regarding reoperation

❑ bioprosthetic valves
- require anticoagulation only for first 3 months
- lower durability and require reoperation due to degeneration and structural failure (20-40% fail by 10 years)
- however, structural degeneration of bioprosthetic valves is rare in elderly patients
- bioprosthetic valves in the mitral position are not as durable as in the aortic position
- preferred valve replacement if life expectancy of patient is shorter than the known durability of the bioprostheses
- also considered if potential for pregnancy (coumadin is teratogenic)

❑ mechanical valves
- require continuous anticoagulation (contraindicated if previous bleeding history)
- in the setting of chronic A fib, patient is already anticoagulated, and therefore mechanical valve used
- preferred valve replacement if long life expectancy or if risk of reoperation is considered high

AORTA REPLACEMENT AND REPAIR

THORACIC AORTIC DISSECTION

- ❑ see Abdominal Aortic Dissection for more details
- ❑ DeBakey classification
  - Type I - intimal disruption of ascending aorta, which dissects to involve the descending aorta and abdominal aorta
  - Type II - involving the ascending aorta only (stops at the innominate artery)
  - Type III - descending aorta only (distal to left subclavian artery)
- ❑ Stanford classification
  - Type A - any dissection that involves ascending aorta
  - Type B - dissection involves only the descending aorta
- ❑ multiple causes - atherosclerosis, cystic medial necrosis (i.e. Marfan's syndrome), infectious, trauma, coarctation, bicuspid aortic valve, pregnancy
- ❑ diagnosis is usually made by chest CT or echocardiography
- ❑ preoperative control of hypertension with beta blockers (+/– nitroprusside if necessary) is an essential part of management
- ❑ dissection may advance proximally to disrupt coronary blood flow or induce aortic valve incompetence, or distally causing stroke, renal failure, intestinal ischemia or leg ischemia
- ❑ surgical repair
  - operative repair involves replacement of the affected aorta with prosthetic graft
  - CPB is required for repair of Type A dissections, and hypothermic circulatory arrest is often used for transverse arch dissections and ascending aorta repairs
  - aortic valve replacement and coronary reimplantation may be required for Type A aneurysms that involve the aortic root
  - Type B dissections can be medically managed unless expansion, rupture, or compromise of branch arteries develops or HTN becomes refractory
- ❑ post-operative complications include renal failure, intestinal ischemia, stroke, paraplegia, and death

THORACIC AORTIC ANEURYSM

- ❑ etiology: medial degeneration, atherosclerosis, expansion of chronic dissections
- ❑ indications for surgery
  - ascending aortic aneurysms
    - symptomatic, expanding, > 5.5 cm in diameter, or greater than twice the size of the normal aorta
    - aneurysms > 4.5 cm if operation is indicated for aortic regurgitation (annuloaortic ectasia)
    - all acute type A dissections
    - mycotic aneurysms
  - transverse arch aneurysms
    - ascending aortic aneurysms that require replacement that also extend into the arch
    - acute arch dissections
    - aneurysms > 6 cm in diameter
  - descending thoracic aorta aneurysms
    - symptomatic aneurysms
    - aneurysms > 6 cm
    - complicated type B dissections
- ❑ catheterization is indicated for patients > 40 years of age or if history of chest pain (to diagnose coexistent coronary artery disease)
- ❑ surgical options
  - ascending aortic aneurysms
    - supracoronary interposition graft placement is performed if the aneurysm does not involve the sinuses
    - Bentall procedure (valved conduit) for patient's with Marfan's syndrome, if the sinuses are involved or for annuloaortic ectasia
  - transverse arch aneurysms
    - Hemiarch repair if ascending aorta and proximal arch are involved (graft sewn to the undersurface of the aorta to leave the brachiocephalic vessels attached to the native aorta)
    - extended arch repair involves placement of an interposition graft and reimplantation of a brachiocephalic island during a period of circulatory arrest (use retrograde SVC perfusion and selective brachiocephalic perfusion to minimize cerebral complications)
  - descending thoracic aorta aneurysms
    - interposition graft placement
    - to ensure spinal cord and kidney perfusion, consider using femoral-femoral bypass (femoral artery and vein cannulation, with arterial blood pumped retrograde into femoral artery for cephalad aortic blood flow)

TRAUMATIC AORTIC DISRUPTION

- ❑ this injury results from deceleration injury, and usually occurs just distal to the left subclavian artery at the level of the ligamentum arteriosum
- ❑ chest radiograph findings include widened mediastinum, pleural capping, associated first and second rib fractures, loss of the aortic knob, hemothorax, deviation of the trachea or NG tube, and associated thoracic injuries (scapular fracture, clavicular fracture)
- ❑ definitive diagnosis is made by aortagram, but chest CT and TEE also aid in the diagnosis
- ❑ imperative that immediate life-threating injuries (i.e. positive diagnostic peritoneal lavage) be treated prior to repair

CARDIAC TRANSPLANTATION

- ❑ well-accepted therapeutic modality for the patient with end-stage heart disease
- ❑ majority of patients have ischemic (60%) or idiopathic (20%) cardiomyopathy, with the minority having valvular or congenital problems
- ❑ world-wide overall 1 year survival is 79%, with an annual mortality rate of 4%, and a 5 year survival approximately 60%
- ❑ indications for surgery
  - end-stage heart disease (e.g. EF< 20%, nonbypassable CAD) refractory to other surgical or medical management
  - NYHA III-IV symptoms with maximal medical therapy and prognosis for 1 year survival < 75%
  - no other major organ or systemic disease
  - emotionally stable with social support
  - medically compliant and motivated
- ❑ contraindications
  - incurable malignancy
  - major systemic illness
  - irreversible major organ disease (e.g. renal, hepatic)
  - active systemic infection (e.g. Hep C, HIV)
  - emotional instability or acute psychiatric illness
  - age > 70 years
  - obesity
  - irreversible pulmonary hypertension (PVR > 6 Wood units)
  - severe COPD (FEV_1 < 1 L)
  - active drug addiction or alcoholism
- ❑ precardiac transplant assessment
  - consultations: cardiology, cardiovascular surgery, respirology, transplant immunology, psychiatry, psychology, dental surgery, social work, chaplaincy, transplant coordinator
  - labs: group and screen, CBC, ESR, INR, PTT, lytes, BUN, creatinine, uric acid, glucose, cholesterol, triglycerides, LFTs, protein electrophoresis, thyroid tests, stool analysis, urinalysis (24 hour)
  - investigations: right and left heart catheterization, 2D Echo, ECG, PFTs, ABG, CXR, Abdo U/S, antibody screen (HBV, HCV, HIV), HLA typing, anti-HLA antibodies, digoxin levels, antibody titres (CMV, herpes simplex, EBV, toxoplasmosis)
- ❑ patients are optimized medically with diuretics, vasodilators (ACE inhibitors, hydralazine, B-blockers), IABP, inotropes (digoxin), +/– LV assist device
- ❑ donor hearts are considered from patients up to age 50-55
- ❑ contraindications to heart donation
  - major chest trauma
  - known cardiac disease
  - acute or chronic infection
  - prolonged cardiac arrest
  - HIV or Hep B/C positive
  - systemic malignancy
- ❑ matching is according to blood type (ABO match is mandatory to avoid hyperacute rejection), body size and weight (should be within 25%) and HLA tissue matching (if time allows)
- ❑ donor harvest
  - the heart is inspected for evidence of coronary disease
  - the SVC and IVC are clamped, the aorta is crossclamped, and cold crystalloid cardioplegia is administered
  - the heart is excised: as much SVC and IVC are preserved as possible, the pulmonary veins are divided, the aorta is divided proximal to the crossclamp, and the distal main PA is divided
  - the heart is bagged in cold saline and transported at 4-6°C
- ❑ recipient operation
  - the recipient is placed on CPB
  - the diseased heart is excised leaving: a long cuff of SVC, a segment of low right atrium near the IVC, the posterior wall of the left atrium, and the great vessels are incised just above the semilunar valves
  - the donor heart is prepared by removing excess tissue
  - the first suture line is started at the left atrial appendage of the donor heart and the free wall of the left atrium is sewn and then the atrial septum
  - the SVC and IVC are connected, followed by the pulmonary artery and then the aorta
- ❑ immediate postoperative management
  - keep heart rate 75-90 (may need pacing)
  - keep CVP low with diuretics to improve RV function
  - treat high PA pressures with NO, nitroglycerin, milrinone, etc.
  - support the RV with inotropes and afterload reducers for high PA pressures
  - support LV if necessary (IABP, inotropes)
  - treat coagulopathy aggressively
  - monitor for, prevent and treat other organ system failure (renal, respiratory, hepatic, and neurological)
- ❑ immunosuppression
  - the goal of immunosuppression is selective modulation of the recipient's immune response to prevent rejection while maintaining defenses against infection and neoplasia, and minimizing toxicity
  - immunosuppression protocols include rabbit anti-thymocyte serum (RATS), OKT3, azathioprine, solu-medrol, cyclosporine, FK506 (tacrolimus) and mycophenolate mofetil (Cellcept)

CARDIAC TRANSPLANTATION . . . CONT.

- ❑ complications
  - rejection
    - the majority of transplant patients experience some form of rejection, though less than 5% have hemodynamic compromise
    - no noninvasive tests to detect rejection, and the gold standard remains endomyocardial biopsies
    - risk of acute rejection is greatest during the first 3 months after transplant
  - infection
    - bacterial and viral infections predominate, although fungal (C*andida*) and protozoan (PCP, toxoplasmosis) are noted in 10% of patients
    - fevers, rising WBC counts and abnormal CXR's must be aggressively evaluated
  - allograft coronary artery disease
    - approximately 50% develop graft CAD within 5 years of transplantation
    - graft vasculopathy is the most common cause of late death following transplantation
    - may reduce rate of graft CAD with diltiazem and statins
  - malignancy
    - develop in 15% of cardiac transplant recipients
    - second most common cause of late death following transplantation
    - cutaneous neoplams most common, followed by non-Hodgkin's lymphoma and lung cancer
  - immunosuppressive medication side effects
    - include hypertension (cyclosporine), hyperlipidemia (cyclosporine, steroids), nephrotoxicity (cyclosporine), GI problems (steroids), osteopenic bone disease (steroids)

CONGENITAL HEART SURGERY

- ❑ the appropriate management of congenital heart disease is based on the precise identification and pathophysiology of the patient's abnormality
- ❑ the appropriate diagnosis is achieved by
  - clinical assessement (cyanotic or acyanotic, presence of CHF)
  - CXR (increased/normal/decreased pulmonary blood flow)
  - ECG (normal or left axis deviation, RVH, LVH)
  - echocardiography (anatomy, shunts, valvular lesions, defects)
  - cardiac catheterization (O_2 sats, shunt calculations, chamber pressures, septal defects, orientation of great vessels)
- ❑ numerous congenital anomalies have been described (see Table 4)

Table 4. Common Congenital Heart Defects

I. Pure obstructive lesions
1. Pulmonic stenosis
2. Mitral stenosis
3. Aortic stenosis
4. Coarctation of the aorta
5. Interrupted aortic arch

II. Simple left-to-right shunts (acyanotic lesions with increased pulmonary blood flow)
1. Patent ductus arteriosus
2. Atrial septal defect
3. Ventricular septal defect
4. Endocardial cushion defect (AV canal)
5. Aortopulmonary window

III. Right-to-left shunts (cyanotic defects with decreased pulmonary blood flow)
1. Tetralogy of Fallot
2. Pulmonary atresia with ventricular septal defect
3. Pulmonary atresia with intact ventricular septum
4. Tricuspid atresia
5. Ebstein's anomaly

IV. Complex cyanotic defects (mixing defects)
1. Double outlet right ventricle
2. Univentricular heart (double inlet left ventricle)
3. Transposition of the great arteries
4. Total anomalous pulmonary venous connection
5. Truncus arteriosus
6. Hypoplastic left heart syndrome

V. Coronary artery congenital defects
1. Anomalous origin of coronary artery
2. Coronary artery fistula
3. Osteal stenosis

Adapted from Bojar RM. *Manual of perioperative care in cardiac surgery, 3rd edition. Massachusetts: Blackwell Science Inc., 1999.*

PALLIATIVE PROCEDURES

- ❑ a Blalock-Taussig (BT) shunt involves an end-to-side anastomosis of the subclavian artery to the ipsilateral PA and is used to increase pulmonary blood flow
- ❑ a modified Blalock-Taussig shunt (MBTS) uses a polytetrafluoroethylene (PTFE) tube graft as the shunt between the subclavian artery and the ipsilateral PA
- ❑ a "central shunt" uses a short piece of PTFE graft to connect the ascending aorta to the main PA (used to increase pulmonary blood flow)
- ❑ the bidirectional Glenn shunt entails an anastomosis between the SVC and the right PA, thus providing blood flow to both pulmonary arteries (therefore "bidirectional")
- ❑ the Fontan operation is designed to deliver systemic venous blood to the PA without the use of the (single) functioning ventricle
    - the “hemi-Fontan” procedure involves the placement of a prosthetic patch inferior to the SVC-RA junction and an anastomosis between the SVC-RA junction to the right PA (the main PA is transected and oversewn), so that SVC blood flows directly to the lungs (IVC blood continues to the single ventricle)
    - the "modified" Fontan procedure consists of an anastomosis between the RA and the PA (or the IVC and right PA after a Glenn Shunt), either directly or with a nonvalved conduit (the main PA is transected and oversewn)
    - the "lateral tunnel" Fontan procedure consists of a baffle tunnel between the IVC and SVC, division of the SVC proximal to the cavoatrial junction, anastomosis of the proximal SVC to the superior aspect of the right PA, and anastomosis of the distal SVC to the inferior surface of the right PA (the main PA is transected and oversewn)

PURE OBSTRUCTIVE LESIONS

- ❑ pulmonic stenosis
    - the critically ill infant with severe PS is usually cyanotic, with pulmonary blood flow provided by the PDA
    - indications for surgery: to increase pulmonary blood flow in critically ill infants; severe PS and presence of symptoms in older infants or children
    - surgical procedures: balloon valvotomy, surgical valvotomy, patch enlargement of right ventricular outflow tract
- ❑ aortic stenosis
    - may occur at 3 levels: subaortic (fibrous membrane or tunnel stenosis), valvar (unicuspid or fusion bicuspid valve), or supravalvar (fibrous ridge or hypoplasia of ascending aorta)
    - indications for surgery: infants with critical aortic stenosis (severe low output states, cyanosis by right-to-left ductal shunting), older children with symptoms or peak gradient > 50 mmHg
    - surgical procedures
        - subaortic stenosis relieved by resection of fibrous membrane or myectomy of hypertrophied ventricular septum
        - valvular aortic stenosis relieved by percuateneous balloon valvotomy or valve replacement
        - supravalvular aortic stenosis relieved by patch augmentation of ascending aorta
- ❑ coarctation of the aorta
    - right-to-left shunting through the patent ductus arteriosus (PDA) provides perfusion to the lower half of the body and may produce cyanosis
    - indications for surgery: infants with severe coarctation and LV failure or ductal dependency, older children with resting or exercise-induced hypertension or gradient > 30 mmHg
    - surgical procedures: resection of coarct segment with end-to-end anastomosis, subclavian flap procedure (augment narrowed segment with divided left subclavian artery), interposition tube graft in adolescents and adults
- ❑ interrupted aortic arch
    - systemic flow to distal aorta is dependent on ductal patency (PGE1 may be required to maintain ductal patency)
    - almost always an associated large subaortic ventricular septal defect (VSD)
    - indications for surgery: within the first week of life
    - surgical procedures: descending aorta is anastamosed to the ascending aorta and VSD is closed

SIMPLE LEFT-TO-RIGHT SHUNTS

- ❑ patent ductus arteriosus (PDA)
  - can increase pulmonary blood flow and produce pulmonary vascular congestion and LV volume overload
  - patency may be critical to survival in neonates with complex heart disease by providing pulmonary or systemic blood flow
  - indications for surgery: PDA contributing to respiratory compromise or persisting beyond 3rd month of life
  - surgical procedures: transcatheter closure using intravascular coils, simple ligation, surgical division
- ❑ atrial septal defect (ASD)
  - results in increased pulmonary blood flow and leads to pulmonary vascular congestion and recurrent respiratory infections
  - indications for surgery: should be closed by age 5 to avoid pulmonary hypertension; in adults with evidence of left-to-right shunt
  - surgical procedures: transcatheter closure, direct suture, use of pericardial or Dacron patch
- ❑ ventricular septal defect (VSD)
  - occur most frequently in the membranous septum (80%)
  - the child will present with pulmonary vascular congestion and recurrent respiratory infections due to excessive pulmonary blood flow
  - indications for surgery: repair should be performed by age 1 in all patients with a significant shunt or LV volume overload
  - surgical procedures: pulmonary artery banding (to reduce pulmonary blood flow if other associated complex lesions), direct suture, patch closure, transcatheter closure (for muscular defects only)
- ❑ endocardial cushion defects (AV canal)
  - defects resulting from deficiency of atrioventricular septum (common in Trisomy 21)
  - indications for surgery: should be repaired by age 6 months to prevent development of pulmonary hypertension
  - surgical procedures: division of common AV valve + closure of ASD and VSD + creation of separate mitral and tricuspid valves
- ❑ aortopulmonary window
  - nonrestrictive communication between posterior aspect of ascending aorta and main PA or right PA, causing CHF
  - indications for surgery: soon after diagnosis is made
  - surgical procedures: patch closure

RIGHT-TO-LEFT SHUNTS

- ❑ Tetralogy of Fallot
  - hypoplasia of the right ventricular infundibulum producing a large nonrestrictive anterior VSD, and overriding aorta, RV outflow tract obstruction and RVH
  - hypoxic "tet" spell: intense cyanosis caused by increased right-to-left shunting (infundibular spasm, increase in pulmonary vascular resistance or decrease in systemic vascular resistance) that can lead to syncope, seizures and death
  - indications for surgery: marked cyanosis, "tet" spells, severe RV outflow tract obstruction, usually in first 2 years of life even if no symptoms
  - surgical procedures: pulmonary valvotomy or pericardial patch enlargement of RV outflow tract or RV-PA valved homograft conduit, + closure of VSD with Dacron patch
- ❑ pulmonary atresia with VSD
  - extreme form of tetralogy of Fallot with no RV-PA connection (severe cyanosis)
  - pulmonary blood flow dependent on PDA and from major aortopulmonary collateral arteries
  - indications for surgery: severe cyanosis
  - surgical procedures: systemic-to-pulmonary artery shunt as palliative procedure; definitive correction (when distal pulmonary arteries are adequate size) by VSD closure with Dacron patch + RV-PA homograft valved conduit
- ❑ pulmonary atresia with intact ventricular septum
  - complete obstruction to RV outflow, producing severe cyanosis and survival dependent on pulmonary blood flow through the PDA
  - indications for surgery: indicated in infancy for profound cyanosis
  - surgical procedures:
    - if RV adequate size then MBTS or central shunt + RV outflow tract patch or pulmonary valvotomy
    - if hypoplastic RV, then MBTS or central shunt and then Fontan procedure after 2 years of age
- ❑ tricuspid atresia
  - characterized by complete absence of the tricuspid valve and varying degrees of RV hypoplasia
  - the relative amounts of systemic and pulmonary blood flow and the patient's clinical presentation depend on the orientation of the great vessels, the size of the VSD and the degree of pulmonary stenosis
  - indications for surgery: severe cyanosis in infancy, or progressive cyanosis or polycythemia before age 2
  - surgical procedures: balloon atrial septostomy if restrictive ASD, PA banding to prevent pulmonary vascular disease if large VSD, MBTS if severe cyanosis, bidirectional Glenn shunt to improve pulmonary blood flow, Fontan operation definitive operation performed between ages 2-5

❑ Ebstein's anomaly
- congenital defect of the tricuspid valve in which the septal and posterior leaflets are malformed and displaced into the RV
- the RA is massively enlarged, and an interatrial communication and tricuspid regurgitation usually exist
- indications for surgery: severe cyanosis with polycythemia, progressive CHF, debilitating arrhythmias
- surgical procedures
  - in newborns, consider closure of tricuspid valve + aortopulmonary shunt, or transplantation
  - in older children, tricuspid valve repair or valve replacement + ASD closure

COMPLEX CYANOTIC DEFECTS

❑ double outlet right ventricle
- a complex spectrum of lesions in which one great artery and more than 50% of the other arise from the RV, with LV outflow through a VSD
- classified by the location of the VSD - subpulmonic, subaortic, doubly committed (lies beneath both valves), or noncommitted
- indications for surgery: progressive cyanosis, refractory CHF, palliative procedures to delay definitive correction
- surgical procedures
  - subaortic or doubly committed VSD: VSD enlargement + tunnel patch LV outflow into aorta
  - subpulmonic VSD: VSD enlargement + tunnel patch LV outflow to PA + arterial switch procedure
  - noncommitted VSD: VSD enlargement + tunnel patch LV outflow to aorta or PA +/– arterial switch procedure

❑ univentricular heart (single ventricle)
- spectrum of anomalies in which the heart has only one effective pumping chamber (usually hypoplastic RV)
- both AV valves are committed to the dominant chamber, giving rise to the name "double inlet left ventricle"
- TGA is usually present, so the LV pumps directly into the PA and via the VSD into the aorta
- indications for surgery: palliative procedures for progressive cyanosis and to prevent pulmonary vascular disease
- surgical procedures: PA banding to limit excessive pulmonary blood flow, MBTS for cyanosis and diminished pulmonary blood flow, Fontan procedure after age 2

❑ transposition of the great arteries (TGA)
- characterized by the aorta arising anteriorly from the RV and the PA arising posteriorly from the LV (D-TGA)
- survival depends on mixing by bidirectional shunting through an ASD, VSD, or PDA
- indications for surgery: most infants have severe cyanosis at birth
- surgical procedures
  - initial palliation with balloon septostomy
  - arterial switch operation - definitive repair involving reconnection of the aorta to the LV outflow and the PA to the RV outflow, with translocation of the coronary arteries to the new aorta
  - Mustard procedure - removal of atrial septum and creation of pericardial baffle that directs caval blood behind the baffle through the mitral valve into the LV and eventually the PA
  - Senning operation - involves mobilizing flaps of the atrial free wall and septum to redirect flow in a manner similar to the Mustard procedure
  - arterial switch operation is operation of choice since Mustard and Senning operations are associated with RV dysfunction and atrial dysrhythmias

❑ total anomalous pulmonary venous connection
- characterized by all of the pulmonary veins draining into the right-sided circulation (supracardiac - SVC or innominate vein, infracardiac - hepatic/portal vein or IVC, intracardiac - coronary sinus or RA)
- often associated with obstruction at connection sites
- an ASD must be present to allow blood to shunt into the LA and then to the systemic circulation
- indications for surgery: severe cyanosis or CHF related to pulmonary venous obstruction
- surgical procedures
  - supracardiac and infracardiac - anastomosis of the common pulmonary vein to the posterior wall of the left atrium
  - intracardiac - baffle placed in RA to redirect pulmonary venous flow through the ASD into the LA

❑ truncus arteriosus
- absence of the aortopulmonary septum resulting in a single great vessel arising from the heart which gives rise to the aorta, PA and coronary arteries
- the truncal valve overlies a large VSD
- indications for surgery: repair within the first 6 months of life to prevent development of pulmonary vascular disease
- surgical procedures: patch closure of the VSD + separation of the PA from the aorta/truncus + closure of truncal incision + RV-PA homograft valved conduit

❑ hypoplastic left heart syndrome
- characterized by varying degrees of hypoplasia or atresia of the mitral valve, LV, aortic valve, and aorta
- blood returning from the lungs is shunted through an ASD to the RA due to LA outflow obstruction
- blood flows from the PA through the PDA to the descending aorta to provide systemic flow (therefore maintenance of ductal patency is critical)
- indications for surgery: urgent surgery once the diagnosis is made
- surgical procedures
  - First stage (20-50% mortality): atrial septectomy + Norwood procedure (patch enlargement of the ascending aorta and arch with homograft or pericardium, division of the PA and closure of the distal PA, anastomosis of the enlarged neoaorta to the proximal PA, and placement of a central shunt to provide pulmonary blood flow)
  - Second stage (age 4-8 months): cavopulmonary anastomosis created between the SVC and right PA (Glenn Shunt) + ligation of central shunt
  - Third stage (age 2-3 years): modified Fontan procedure (IVC flow channeled into the right PA)
  - the result is a RV that serves as the systemic ventricle, and pulmonary blood flow is provided directly by systemic venous return
  - heart transplantation is a therapeutic alternative to the Norwood procedure

VASCULAR - ARTERIAL DISEASES

ACUTE ARTERIAL OCCLUSION/INSUFFICIENCY

❑ due to embolus, arterial thrombosis or trauma. Time is of essence, after approximately 6 hours (depending on collaterals), ischemia and myonecrosis is irreversible to limb

Embolus

❑ etiology
- cardiac is the source of 80-90% of embolic episodes; History of MI (< 3 months), rheumatic heart disease, abnormal or prosthetic valves, A fib, MS, cardiomyopathy, endocarditis, atrial myxoma
- arterial source – proximal arterial source such as aneurysm, atheroembolism
- paradoxical embolism with a history of venous embolus passing through intracardiac shunt
- other including a history of medications (oral contraceptives), previous emboli, neurologic / TIAs

❑ presentation
- sudden pain in lower extremity progressing within hours to a feeling of cold numbness, loss of function and sensation
- no history of significant vascular claudication
- pulses are present in contralateral limb
- may have emboli to other locations (cerebral, upper limb, renal)

Arterial Thrombosis

❑ etiology
- it is important to differentiate thrombosis from embolism because the treatment for the two may vary dramatically
- thrombosis usually occurs in a previously diseased (atherosclerotic) artery, congenital anomaly, infection, hematological disorders and low flow rates (CHF)

❑ presentation
- gradual progression of symptoms; but may have an acute-on-chronic event
- progression to loss-of-function and sensory loss may be less profound than with acute embolus
- past history of claudication
- atrophic changes may be present
- contralateral disease may be present

Trauma

❑ etiology
- it is important to determine a history of arterial trauma, arterial catheterization, intra-arterial drug induced injection, aortic dissection, severe venous thrombophlebitis, prolonged immobilization, idiopathic

❑ symptoms
- symptoms (**6 P's**)
  - **P**ain: absent in 20% of cases because of prompt onset of anesthesia and paralysis
  - **P**allour: replaced by mottled cyanosis within a few hours
  - **P**arasthesia: light touch goes first (small fibers) followed by other sensory modalities (large fibers)
  - **P**aralysis / **P**ower loss: heralds impeding gangrene
  - **P**olar (cold)
  - **P**ulselessness
- do not expect all of the 6 P's to be present and do not rely on pulses
- of the 6 P's the most important are paralysis / power loss
- full cardiac exam including complete bilateral pulse examination
- atrophic skin and nail changes - longstanding arterial insufficiency

- ❑ investigations
  - CXR, ECG, arteriography
- ❑ management
  - immediate heparinization at 5000iu bolus and continuous infusion to maintain PTT > 60
  - in the absence of power and sensation – need emergent re-vascularization: (i) for embolus – embolectomy; (ii) for thrombus – bypass
  - in the presence of power and sensation – need work-up – including angiogram: (i) for embolus – embolectomy; (ii) for thrombus - bypass
  - embolectomy: Fogarty catheter tied to fish embolus out of artery
  - bypass: bypass occlusion allowing blood flow to resume to distal site
  - identify and treat underlying cause
  - continue heparin post-op, start warfarin post-op day 1 for 3 months
  - re-perfusion phenomenon
  - toxic metabolites from ischemic muscle —> renal failure and multi-organ system failure
- ❑ complications
  - beware compartment syndrome with prolonged ischemia; requires fasciotomy
- ❑ treatment of irreversible ischemia is amputation
- ❑ prognosis
  - 12-15% mortality rate
  - 5-40% morbidity rate (amputation)

CHRONIC ARTERIAL OCCLUSION / INSUFFICIENCY

- ❑ predominantly due to atherosclerosis (see Cardiology Chapter)
- ❑ risk factors
  - major: smoking, hypertension, hypercholesterolemia, DM
  - minor: hypertriglyceremia, obesity, sedentary, family history
  - predominantly lower extremities
  - femoropopliteal system > aortoiliac
  - tandem lesions often present
  - prevalence quoted at 1.5% <50 years old, 5% 50-65 years old, and 18% > 65 years old
- ❑ differential diagnosis
  - osteoarthritis (OA) of the hip - worse in the A.M. and P.M. and varies from day-to-day
  - neurogenic claudication – due to spinal stenosis; pain very similar, but relieved by rest (longer than required for intermittent claudication) and requires a postural change for relief
  - varicose veins – localized pain, typically less severe, after exercise and never occurs at rest; related to the presence and site of varices
- ❑ signs and symptoms
  - claudication: 3 components
    1. discomfort with exertion - usually in calves (cramping), but any exercising group
    2. relieved by short rest - 2 to 5 minutes, and no postural changes necessary
    3. reproducible - "claudication distance"
  - 60-80% get better with conservative therapy, 20-30% stay the same, 5-10% get worse
  - pulses: may be absent at some locations (document all pulses)
  - signs of poor perfusion: hair loss, deformed nails, atrophic skin, ulcerations and infections
  - other manifestations of atherosclerosis: CVD, CAD, Impotence
- ❑ investigations
  - non-Invasive
    - ankle-brachial index(ABI): measure brachial pressure bilaterally (use highest pressure) and measure pressure at ankle. An abnormal ABI is defined as an index < 0.90. Rest pain usually appears at an ABI < 0.3. Problem: calcification of the artery may cause overestimation of ABI values. Solution: Trans-cutaneous oxygen studies to measure tissue oxygenation (30 mmHg necessary for primary wound healing). Doppler flow studies and real-time Duplex scanning
  - invasive
    - arteriography: allows you to define site and size of occlusion as well as the status of collateral flow. (Gold standard). Mainly a pre-operative planning tool
    - digital subtraction angiography (DSA): electronically digitalizes x-ray signals and enhances image
- ❑ management
  - conservative
  - 70% of claudicants treated conservatively improve/unchanged, 5-10% develop gangrene
  - modify risk factors
    - exercise program to develop collateral circulation
    - foot care (especially DM) - hygiene, cut nails carefully, treat sore/infection promptly
  - drug treatment: ECASA, Plavix or Solaftizol (not yet available in Canada)
- ❑ surgical
  - indications: claudication interfering with lifestyle, rest pain, pre-gangrene, gangrene
  - endovascular – PTA
  - arterial bypass grafts – aortoiliofemoral, axillofemoral, femoral popliteal, distal arterial. Can use either in situ graft, reversed vein graft, umbilical vein graft or a polytetrafluoroethylene (gortex) graft or dacron graft material
  - amputation – for non-revascularizable limb

CRITICAL ISCHEMIA

- ❑ arterial compromise eventually leading to necrosis
- ❑ signs and symptoms **(see Colour Atlas PL5)**
  - rest pain, night pain
  - ulcerations, gangrene of toes
  - pallor on elevation, dependent rubor, slow capillary refill
  - decreased or absent pulses
  - significant bruits may be heard (at 50% occlusion) – if stenosis severe, no bruit will be heard
  - ABI < 0.5
- ❑ investigations
  - as above
- ❑ management
  - needs immediate surgery due to risk of limb loss
  - initial procedures: transluminal angioplasty, laser, atherectomy and stents
- ❑ operations include
  - inflow procedures for aortoiliac disease
  - endarterectomy
  - reconstructive procedures for superficial femoral artery occlusion
  - profundoplasty
  - femoropopliteal bypass
  - aortoiliac or aortofemoral bypass
  - axillofemoral bypass (uncommon)

ABDOMINAL AORTIC ANEURYSM (AAA)

- ❑ aneurysm: localized dilatation of an artery that is 2x normal diameter
  - true aneurysm: wall is made up of all 3 layers of the artery
  - false aneurysm: defect in arterial with aneurysmal sac composed of fibrous tissue or graft
- ❑ classification
  - etiology: congenital
    - Marfan syndrome, berry aneurysms acquired
    - metabolic / endocrine
    - degenerative
    - inflammation / infection - syphilis
    - neoplastic
    - dissection
    - shape: Fusiform (true aneurysms)
      Saccular (false aneurysms)
  - location: aortic
    peripheral arteries
    splanchnic
    renal
- ❑ structure true or false
- ❑ inflammatory
- ❑ infected
- ❑ 95% of AAA's are infrarenal
- ❑ incidence 4.7 to 31.9 per 100,000 person from 1951-1980
- ❑ the average expansion rate (80% of aneurysms) is 0.2cm/yr for smaller aneurysms (< 4 cm) and 0.3-0.5 cm/yr for larger aneurysms (> 4-5 cm)
- ❑ may be associated with other peripheral aneurysms
- ❑ etiology
  - cystic medial necrosis – likely due to enzymatic abnormalities in the aortic wall
  - atherosclerosis
- ❑ high risk groups
  - 65 years and older
  - male:female = 3.8:1
  - peripheral vascular disease, CAD, CVD
  - family history AAA
- ❑ clinical presentation
  - common
  - 75% asymptomatic (often discovered incidentally)
- ❑ symptoms due to acute expansion or disruption of wall
  - syncope, pain (abdominal, flank, back)
  - uncommon
    - partial bowel obstruction
    - suodenal mucosal hemorrhage —> GI bleed
    - erosion of aortic and duodenal walls —> aortoduodenal fistula
    - erosion into IVC —> aortocaval fistula
  - distal embolization
- ❑ signs
  - hypotension
  - palpable mass felt at/above umbilicus
  - pulsatile mass, in 2 directions
  - bounding femoral pulses
  - distal pulses may be intact

- ❑ investigations
  - U/S (100% sensitive, able to measure up to +/– 0.6 cm accuracy); however, operator dependent, and may not be possible with obese patients, excessive bowel gas or periaortic disease
  - Aortogram (not useful because lumen may not change in size due to thrombus formation)
  - CT (accurate visualization, determines size)
  - MRI (very good imaging, but limited access)
  - Doppler/Duplex (to rule out aneurysmal disease elsewhere in the vascular tree)
- ❑ treatment and prognosis
  - decision to treat is based on weighing the risk of OR to disease complications (such as rupture)
  - risk of rupture depends on
    - size %/yr
    - 4-5 cm – 2-3%
    - 5-6 cm – 5-8%
    - > 7 cm – 25-40%
    - > 10 cm – 100%
    - rate of growth (> 0.4 cm/yr)
  - presence of symptoms, hypertension, COPD
  - consider operate at > 5 cm since risk of rupture greater than or equal to risk of surgery
  - mortality of elective repair = 2-3% (mostly due to MI)
  - consider revascularization for patients with CAD before elective repair of CAD
  - conservative
    - reduce risk factors
    - smoking
    - HTN
    - DM
    - hyperlipidemia
    - exercise
    - watchful waiting if <4-5cm re-U/S q6mo
- ❑ surgery
  - procedure
    - laporotomy performed firm xyphoid process to symphis pubis
    - aorta is dissected out
    - distal clamp is placed onto aorta
    - proximal clamp is placed onto aorta or common iliacs
    - aneurysm opened
    - removal of thrombis
    - graft put into place: tube/bifurcation
    - graft sewn into proximal site
    - graft suture site tested
    - graft sewn into distal site - last stitch not closed until integrity of anastomosis tested
    - aorta wall sewn over graft
  - indications for surgery
    - ruptured
    - symptomatic or rapidly expanding aneurysms
    - asymptomatic aneurysms >5cm
  - contraindications
    - less than 1 year to live
    - terminal underlying condition (cancer)
    - overwhelming medical conditions
    - recent MI, unstable angina, decreased mental acruity, advanced age
  - early post-op complications
    - myocardial ischemia
    - arrhythmias
    - CHF
    - pulmonary insufficiency
    - renal damage
    - bleeding
    - infection
    - cord injury
    - impotence
  - late complications
    - graft infection/thrombosis
    - aortoenteric fistula
    - anastomotic aneurysm
    - infection

- post-op orders
  - bedrest (24-48 hr)
  - NPO
  - NG —> straight drainage, record drainage q12h +/– NG losses 1:1 q shift Ns with 10 mEq KCl/L
  - Foley —> straight drainage
  - +/– PA line
  - +/– arterial line
  - routine post-op vitals q15min until stable then q1h
  - routine CU ICU admission blood work (includes CBC, lytes, BUN, Cr, PLT, PT, PTT, glucose)
  - CXR on CUICU admission and daily until extubated
  - ECG on admission q8h x 24 hrs and PRN after
  - ABI's/pedal pulses q1h x 4 h then q shift
  - chest physiotherapy assessment and treatment
  - weaning protocol as per CUICU portocol
  - titrate FiO_2 to keep PO_2 > 90 mmHg or O_2 sat > 95% c/c
  - ventilation (if required) Vt 700 mL, FiO_2 > 50% c/c, rate 12/min, PEEP 5 cm/H_2) to keep $PaCo_2$ 35-45 mmHg
  - incentive spirometry when extubated
  - epidural proticol if required
  - IV's: N/S or RL at 150 cc/hr x 24 hr; reassess at 24 hrs
  - morphine 2-10 mg IV q1h PRN
  - midazolam 2-4 mg IV q1h PRN max 20 mg/24hr
  - dimenhydrinate 12.5-25 mg IV q4h PRN
  - heparin 5,000U SC q12h for DVT prophylaxis start post-op
  - sulcrote 1 gm NG q4h
  - may require inotropic agents
  - may require antiphypertensive agents

ABDOMINAL AORTIC DISSECTION

- ❑ Stanford Surgical Classification
  - Type A: involves the ascending and aortic arch; requires emergency surgery
  - Type B: involves the aorta distal to subclavian artery; emergency surgery only if complications of dissection (require long-term follow-up to assess aneurysm size)
- ❑ male:female = 3-4:1
- ❑ predominantly older patients
- ❑ etiologic factors
  - hypertension
  - cystic medial necrosis (not atherosclerosis)
- ❑ associated factors
  - Marfan's Syndrome
  - coarctation of aorta
  - congenital bicuspid aortic valve
- ❑ pathogenesis (usually in thoracic aorta)
  - intimal tear —> entry of blood separates media —> false lumen created —> dissection often continues to aortic bifurcation
- ❑ symptoms and signs
  - sudden searing chest pain that radiates to back
  - asymmetric BPs and pulses between arms
  - branch vessel "sheared off" – ischemic syndromes
  - MI with proximal extension to coronary arteries
  - "unseating" of aortic valve cusps
  - new diastolic murmur in 20-30%
  - neurologic injury - stroke (10%), paraplegia (3-5%)
  - renal insufficiency
  - lower limb ischemia
  - cardiac tamponade - false lumen ruptures into pericardium
  - hypertension (75-85% of patients)
- ❑ diagnosis and investigations
  - CXR
    - Pleural cap
    - Widened mediastinum
    - Left pleural effusion with extravasation of blood
  - ECG - most common abnormality is LVH (90%)
  - TEE, CT, aortography
- ❑ management
  - sodium nitroprusside and B-blocker to lower BP and decrease cardiac contractility
  - ascending aortic dissections operated on emergently
  - descending aortic dissections initially managed medically
    - 10-20% require urgent operation for complications

RUPTURED ABDOMINAL AORTIC ANEURYSM (RAAA)

- ❑ narrow window of opportunity
- ❑ usually present with classical diagnostic triad (50% cases)
  - sudden abdominal or back pain
  - SHOCKY (Hypotensive, faintness, cool, mottled extremities)
  - pulsatile mass
- ❑ may be confused with renal colic
- ❑ ECG confusing - may show cardiac ischemia
- ❑ diagnosis by history and physical
- ❑ do not waste time in radiology if RAAA strongly suspected
- ❑ if patient stable without classic triad —> consider CT
- ❑ management
  - initial resuscitation including vascular access, notify OR,
  - ensure availability of blood products, invasive monitoring
  - emergency laparotomy as soon as IV and cross-match sent
  - upon opening - gain centre of aorta proximal to rupture with cross clamp
- ❑ prognosis
  - 50% survival for patients who make it to OR
  - 100% mortality if untreated
  - overall mortality 90%

CAROTID SURGERY (see Neurosurgery Chapter)

VASCULAR – VENOUS DISEASE

ANATOMY

- ❑ the venous system is divided into 4 general areas
  - superficial venous system (subcutaneous veins and the greater and lesser saphenous veins)
  - communicating venous system (perforating veins)
  - deep venous system (tibial, popliteal, femoral and iliac veins)
  - venous valves (in all infra-inguinal veins)

DEEP VENOUS THROMBOSIS (DVT) (ACUTE)

- ❑ occlusion of the deep venous system, typically of the lower extremity that can extend up to the right atrium.
- ❑ pathogenesis (Virchow's Triad)
- ❑ flow stasis
- ❑ postulated that stasis protects activated pro-coagulants from circulating inhibitors, fibrinolysis and inactivation in the liver
- ❑ surgery
- ❑ trauma and subsequent immobilization
- ❑ immobilization due to: acute MI, stroke, CHF
- ❑ compression of veins by tumours
- ❑ shock (decreased arterial blood flow)
- ❑ hypercoagulability
- ❑ states that increase coagulability of the blood (ex. Increased fibrinogen or prothrombin) in which there is a deficiency of anti-coagulants (anti-thrombin III, Protein C+S), e.g.
  - pregnancy
  - estrogen use
  - neoplasms: diagnosed, occult, undergoing chemotherapy
  - tissue trauma: activation of coagulation
  - nephrotic syndrome
  - deficiency of anti-thrombin III, protein C or S
  - endothelial damage
- ❑ exposure of the underlying collagen in a breach of the intimal layer of the vessel wall leads to platelet aggregation, degranulation and thrombus formation. There also appears to be a decrease endothelial production of plasminogen and plasminogen activators, e.g.
  - endothelial damage: venulitis, trauma
  - varicose veins
  - previous thrombophlebitis

- ❑ signs and symptoms
  - most frequent site of thrombus formation is calf
  - isolated calf thrombi often asymptomatic
  - 30-50% are asymptomatic or minimal symptoms
  - 20-30% extend proximally and account for most clinically significant emboli
  - classic presentation < 1/3
  - calf/thigh discomfort, edema, venous distension
  - investigations (refer to PIOPED study for details)
  - history and physical
    - calf tenderness (if elicited on ankle dorsiflexion = Homan's sign)
    - wider circumference of affected leg
  - fever POD #7-10
  - clinical assessment incorrect 50% time, therefore must confirm by objective method
  - non-invasive tests
    - duplex doppler U/S
    - 93% sensitive and 98% specific for symptomatic patients, decreased for asymptomatic patients
    - detects proximal thrombi
    - initial negative exam should be repeated 6-7 days later to detect proximal extension
  - invasive testing
    - ascending phlebography (venogram)
    - the gold standard but costly
    - detects distal and proximal thrombi
    - complicated by contrast-induced thrombosis of peripheral veins (2-3%)
- ❑ management
  - goals of treatment
    - prevent formation of additional thrombi
    - inhibit propagation of existing thrombi
    - minimize damage to venous valves
    - prevent pulmonary emboli (PE)
      - 25% develop PE if untreated; 5% if treated
  - aggressive medical management
- ❑ treated as outpatient
- ❑ fragment / low molecular weight heparin (LMWH)
- ❑ coumadin
- ❑ advantage: avoid hospitalization
  - conservative medical management
    - IV heparin, 5,000 U bolus + 1,000 U/hr to keep aPTT 2-2.5x control
    - convert to warfarin 3-7 days after full heparinization; warfarin for 3-6 months
    - risks of therapy - bleeding, heparin-induced thrombocytopenia, warfarin is teratogenic
  - surgical
    - venous thrombectomy - if arterial insufficiency with extensive iliofemoral thrombosis, +/– venous gangrene
    - inferior vena cava (IVC) (Greenfield) filter- inserted percutaneously, indications:
      - recurrent PE despite anticoagulation
      - contraindication to anticoagulation e.g. intra-cranial trauma
      - certain operations for cancer, pulmonary embolectomy
      - septic emboli refractory to combination antibiotic and anticoagulation
      - "free-floating" thrombus loosely adherent to wall of IVC or pelvic veins
      - IVC ligation, surgical clips - increases risk of venous insufficiency; rarely used
- ❑ DVT prophylaxis
  - conservative
    - minimize risk factors
    - early ambulation, passive range of motion
    - anti-embolism stockings
    - pneumatic sequential compression devices
    - elevation of limb
  - medical prophylaxis
    - optimize hydration to prevent hemoconcentration
    - ECASA, warfarin, minidose heparin(5,000 U SC q8-12h) in high risk situations
- ❑ complications
  - pulmonary embolus (PE)
  - varicose veins
  - chronic venous insufficiency
  - venous gangrene
  - phlegmasia cerulea dolens (PCD) - massive DVT with clot extension to iliofemoral system and massive venous obstruction resulting in a cyanotic, immensely swollen, painful and critically ischemic leg
    - risk venous gangrene
  - phlegmasia alba dolens (PAD) - as above with additional reflex arterial spasm resulting in less swelling than PCD
  - cool leg and decreased pulses

SUPERFICIAL THROMBOPHLEBITIS

- ❑ inflammation or thrombosis of any superficial vein
- ❑ etiology
  - trauma
  - association with varicose veins
  - migratory superficial thrombophlebitis
  - Buerger's disease
  - SLE
  - polycythemia
  - thrombocytosis
  - occult malignancy (especially pancreas)
  - idiopathic
- ❑ a pulmonary embolus is rarely present with superficial thrombophlebitis
- ❑ signs and symptoms
  - pain and cord-like swelling along course of involved vein;
  - most commonly involves long saphenous vein or its tributaries
  - red, warm, indurated vein
- ❑ investigations
  - non-invasive tests to exclude associated DVT (5-10%)
- ❑ treatment
  - conservative
    - bed rest and elevation of limb
    - moist heat, compression bandages, mild analgesic, anti-inflammatory and anti-platelet (e.g. ASA), ambulation
  - surgical excision of involved vein indicated if conservative measures fail
    - of suppurative thrombophlebitis - IV antibiotics and excise involved vein
- ❑ complications
  - chronic recurrent superficial thrombophlebitis

VARICOSE VEINS

- ❑ distended torturous superficial veins due to incompetent valves in the deep, superficial or perforator systems
- ❑ often greater saphenous vein with dilated tributaries
- ❑ can also occur in
  - esophagus - esophageal varices
  - anorectum - hemorrhoids
  - scrotum - varicocele
- ❑ etiology
  - primary
    - most common form of venous disorder of lower extremity
    - 10-20% of population
    - inherited structural weakness of vein valves is main factor
  - contributing factors
    - age
    - female
    - oral contraceptive (OCP) use
    - occupations requiring long hours of standing
    - pregnancy
    - obesity
  - secondary
    - result of increased venous pressure from deep-venous valvular insufficiency and incompetent perforating veins
    - malignant pelvic tumours with venous compression
- ❑ congenital anomalies
  - acquired/congenital arteriovenous fistulae
- ❑ signs and symptoms
  - diffuse aching, fullness/tightness, nocturnal cramping
  - aggravated by prolonged standing, end of day, premenstrual
- ❑ investigations
  - patient standing: long, dilated and tortuous superficial veins along thigh and leg
  - if ulceration, hyperpigmentation, indurated appearance think secondary varicose veins
  - Brodie-Trendelenberg test (valvular competence test)
  - while patient is supine, raise leg and compress saphenous vein at thigh; have patient stand; if veins fill quickly from top down then incompetent valves; use multiple tourniquets to localize incompetent veins

- ❑ management
  - majority of symptoms relieved by elevation of leg and/or elastic stockings
  - may require stripping (proceeding high ligation of saphenofemoral junction) or sclerosing of veins if conservative management fails
  - commonly a cosmetic problem
- ❑ prognosis
  - natural history benign, slow with predictable complications
  - almost 100% symptomatic relief if varicosities are primary
  - generally good cosmetic results
  - significant post-operative recurrence if followed long enough
- ❑ complications
  - recurrent superficial thrombophlebitis
  - hemorrhage - externally or into subcutaneous tissues
  - ulceration, eczema, lipodermatosclerosis, hyperpigmentation

CHRONIC DEEP VENOUS INSUFFICIENCY (POST PHLEBITIC SYNDROME, AMBULATORY VENOUS HYPERTENSION)

- ❑ late complication of DVT, often presenting several weeks to years post DVT
- ❑ etiology/pathogenesis
  - recanalization of thrombosed veins with resulting damaged incompetent valves
  - impairment of calf muscle pump, sustained venous hypertension
- ❑ signs and symptoms **(see Colour Atlas PL4)**
  - pain (most common) relieved on recumbency and foot elevation
  - pruritis
  - aching fullness of leg, edema
  - pigmentation - hemosiderin deposits
  - varicose veins
  - venous dermatitis
  - ulceration (stasis dermititis) above medial malleolus
  - venous ulcers are not painful as ischemic arterial ulcers where pain is worse with elevation
  - arterial ulcers are often deep, extending through the fascia, with necrotic base whereas venous ulcers are shallow
  - venous ulcers are weeping (wet) and not well demarcated (opposite is true for arterial ulcers)
  - positive Brodie-Trendelenberg
  - investigations
  - gold standard is ambulatory venous pressure measurement (rare)
  - doppler U/S
  - photoplethysmography
- ❑ management
  - non-operative
  - elastic compression stockings, leg elevation, avoid prolonged sitting/standing
  - ulcers treated with zinc-oxide wraps (unna boot), split-thickness skin grafts, antibiotics, debridement
- ❑ operative
  - if conservative measures fail, or if recurrent/large ulcers
  - surgical ligation of perforators in region of ulcer, strip greater saphenous vein

LYMPHATIC OBSTRUCTION / LYMPHANGITIS

- ❑ inflammation of the lymphatic vessels (lymphangitis) secondary to β-hemolytic *streptococci* or *staphylococci* infection
- ❑ signs and symptoms
  - pain
  - hyperemia along the affected lymphatic vessel
- ❑ management
  - immobilization of affected limb
  - antibiotic treatment
  - should evidence of bacterial seeding be present, drainage may be required
- ❑ complication
  - if the bacterial spread is not terminated at the lymphatic node, septicemia may result

VASCULAR - TRAUMA

PENETRATING (LACERATION)

- ❑ usually mechanism associated with fractures and dislocations that lead to limb-threatening injuries
- ❑ etiology
  - motor vehicle crash (MVC)
  - gunshot wound
- ❑ signs and symptoms
  - loss of or relatively weak pulses
  - expanding hematoma
  - distal cyanosis
  - pulsatile bleeding
  - distal parasthesia
  - polar extremity
  - bleeding not controlled with direct pressure
  - similar to acute arterial insufficiency
- ❑ investigations
  - duplex doppler
  - angiography if patient is stable
  - intra-operative angiography if patient is not stable
- ❑ management
  - vascular shunt
  - repair damaged vessel
  - repair associated orthopaedic injuries
- ❑ complications
  - compartment syndromes – requiring surgical fasciotomy
  - should the above not be performed within 6 hours of an ischemic limb, amputation is inevitable

BLUNT (CONTUSION, SPASM, COMPRESSION)

- ❑ usually mechanism associated with MVC or other direct non-penetrating Injuries
- ❑ signs and symptoms
  - similar to penetrating trauma
- ❑ investigations
  - similar to arterial insufficiency
- ❑ management
  - injuries are treated as in Arterial Insufficiency section

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