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Welcome to your vascular surgery rotation!

The vascular surgery department is based out of Victoria hospital, where our outpatient clinics, inpatient ward and operating rooms reside. Currently, there are four staff surgeons, two vascular surgery fellows and a number of vascular surgery residents on service at any given time.

The Division of Vascular Surgery is dedicated to excellence and leadership inpatient care, education and research. The clinical program is located at Victoria Hospital where we provide vascular surgery care to the citizens of Southwestern Ontario and beyond, with a catchment area of 1.9M. Over 800 major surgical procedures are performed yearly by our surgeons, including over 330 aortic aneurysm repairs, half of which are treated endovascularly. With close collaboration with our interventional radiology and vascular medicine colleagues, we provide complete patient care including risk reduction, medical management, percutaneous interventions, endovascular and open surgical procedures.

Disclaimer: This manual was designed as an introduction to your vascular surgery rotation, and to be used as a supplemental learning tool. It is not intended as a substitute for physician discretion and clinical judgment.
EXPECTATIONS:

Morning Patient Rounds: We are a very busy service. Mornings begin with inpatient rounds, where we will see all of our inpatients, usually around 25-30. It is expected that all patients are seen by a physician and have a progress note written in SOAP format. With such a high volume of inpatients, it is important to work efficiently. Coming in early to review recent lab values, and chart vital signs can be very helpful. Medical students and junior residents are responsible for:

1. Having inpatient lists printed for all team members
2. Being up to date on recent lab values and vital signs
3. Charting progress notes
4. Undressing and debriding wounds

Morning Teaching Rounds:
Tuesday (0700 – 0800): Endovascular Case of the Week – Library E2
Wednesday (0700 – 0800): Vascular/IR rounds – McLaughlin Room E2
Wednesday (0800 – 0900): Vascular resident teaching – McLaughlin Room E2

Operating Room:
There is at least one vascular OR per day, and at times two cases may be happening simultaneously. It is important to familiarize yourself with the patient’s history prior to the OR as this will facilitate learning and understanding of the procedures. It is also important to introduce yourself to the nursing staff in the operating room.
Radiation Safety
We often use fluoroscopy for endovascular procedures. Radiation safety is very important, and you must wear a leaded apron and thyroid protector for the entirety of the case. These can be found in the central core. Make sure you pick a set that fits properly. Lead is not worn outside of the operating room and you must return it to its place when finished. It is important to avoid creasing the leaded aprons and thyroid protectors as this significantly impairs the ability of the lead to protect the wearer from radiation.

Clinic:
These are also run daily, and can be found in E2. The duties of the medical students and junior residents include:

1. Familiarizing yourself with the patient’s previous history and reason for assessment
2. Taking a thorough history and physical exam
3. Reviewing recent imaging studies
4. Discussing case with staff member

A staff vascular surgeon must evaluate all patients unless otherwise specified.
PALPABLE PULSES:

A pulse examination should be performed in a relatively consistent manner and should always be complete. Comment if the pulse is present, decreased or absent. Examining the contralateral extremity can demonstrate changes that could indicate proximal stenosis or occlusion.


2. Ulnar: Medial wrist. Requires firmer palpation due to its deeper course compared to the radial artery.


4. Carotid: Midneck region anterior to sternocleidomastoid muscle.

5. Femoral: Located at midpoint (or slightly medial) of the inguinal ligament, which runs from the ASIS to pubis.

6. Popliteal: Lateral to popliteal fossa, between the two heads of the gastrocnemius. Best felt with patient sitting with a relaxed leg dangling over the edge of the bed. Firm palpation is required to feel this pulse.

7. Dorsalis Pedis: Found on the dorsum of the foot between the first and second metatarsal bones.

8. Posterior Tibial: Found in the hollow posterior to medial malleolus.
ARTERIAL ANATOMY: UPPER EXTREMITY

AHC  Anterior Humeral Circumflex  Profunda  Profunda Brachial
BC   Brachiocephalic               R      Radial
Bra  Brachial                      rCC    Right Common Carotid
CCT  Costocervical                 RSA    Right Subclavian Artery
DS   Dorsal Scapular              ST     Superior Thoracic
EC   External Carotid              Sub    Subscapular
IC   Internal Carotid              Supra  Suprascapular
InfTy  Inferior Thyroid           TA     Thoraco-acromial
IT   Internal Thoracic            Tc     Thyrocervical
LT   Lateral Thoracic             Trans Cerv  Transverse Cervical
ltCC Left Common Carotid          U      Ulnar
PHC  Posterior Humeral Circumflex VA    Vertebral Artery
ARTERIAL ANATOMY: ABDOMINAL

CHA Common Hepatic Artery
CIA Common Iliac Artery
CT Celiac Trunk
Cystic Cystic Artery
GDA Gastroduodenal Artery
ICA Ileocolic Artery
IMA Inferior Mesenteric Artery
InA Intestinal Arteries
IP A Inferior Phrenic
LGA Left Gastric Artery
LtC Left Colic
LtHA Left Hepatic Artery
LtR Left Renal
MCA Middle Colic Artery
MSA Median Sacral Artery
PDA Pancreaticoduodenal Artery
RBE Recurrent Branch Esophagus
RGA Right Gastric Artery
RGE Right Gastroepiploic
RtC Right Colic
RtHA Right Hepatic Artery
RtR Right Renal
SA Splenic Artery
SG Short Gastrics
Sig Sigmoid Artery
SMA Superior Mesenteric Artery
SRA Superior Rectal Artery
ARTERIAL ANATOMY: LOWER EXTREMITY

AT  Anterior Tibial
CFA  Common Femoral Artery
CIA  Common Iliac Artery
EIA  External Iliac Artery
IIA  Internal Iliac Artery
PA   Peroneal Artery
POP  Popliteal

Profunda
PT   Posterior Tibial
SEA  Superficial Epigastric Artery
SEP  Superficial External Pudendal
SFA  Superficial Femoral Artery
TPT  Tibial Peroneal Trunk

Profunda Femoris
ARTERIAL ANATOMY: THIGH COMPARTMENTS

**Anterior Compartment Thigh**
- Sar  Sartorius
- RF  Rectus Femoris
- VL  Vastus Lateralis
- VI  Vastus Intermedius
- VM  Vastus Medius
- FA  Femoral Artery
- FV  Femoral Vein

**Medial Compartment Thigh**
- Gr  Gracilis
- AL  Adductor Longus
- AB  Adductor Brevis
- AM  Adductor Magnus

**Posterior Compartment Thigh**
- BF  Biceps Femoris
- ST  Semitendinosus
- SM  Semimembranosus
- Sc  Sciatic Nerve
### Anterior Compartment
- **TA**: Tibialis Anterior
- **EHL**: Extensor Hallicus Longus
- **EDL**: Extensor Digitorum Longus
- **A**: Anterior Tibial Artery/Vein
- **Peroneus Tertius** (not shown)

### Lateral Compartment
- **FL**: Fibularis Longus
- **FB**: Fibularis Brevis

### Deep Posterior Compartment
- **TP**: Tibialis Posterior
- **FHL**: Flexor Hallicus Longus
- **FDL**: Flexor Digitorum Longus
- **P**: Posterior Tibial Artery/Vein
- **Popliteus** (not shown)

### Superficial Posterior Compartment
- **Soleus**
- **LHG**: Lateral Head Gastrocnemius
- **MHG**: Medial Head Gastrocnemius
ANATOMY MNEUMONICS

Branches of External Carotid Artery: Some Attending Physicians Like Freaking Out Potential Medical Students (*Superior thyroid, Ascending Pharyngeal, Lingual, Facial, Occipital, Posterior auricular, Maxillary, Superficial Temporal*)

Branches of Subclavian Artery: Very Tired Individuals Sip Strong Coffee Served Daily (*Vertebral, Thyrocervical trunk (Inferior thyroid, superficial cervical, suprascapular), Costocervical (Superior intercostal, Deep cervical*)

Branches of Axillary Artery: Screw The Lawyers Save A Patient (*Superior thoracic, Thoracoacromial, Lateral thoracic, Subscapular, Anterior circumflex humeral, Posterior circumflex humeral*)

Branches of Internal Iliac Artery: I Love Going Places In My Very Own Underwear (*Ileolumbar, Lateral Sacral, Gluteal (superior and inferior), Pudendal (internal), Inferior vesicle (uterine in females), Middle Rectal, Vaginal, Obturator, Umbilical*)

Branches of Profunda Femoris: Put My Leg Down Please (*Profunda femoris, Medial circumflex, Lateral circumflex, Descending genicular, Perforating arteries*)
SELECTED TOPICS: ANEURYSMS

Aneurysms: Dilation of an artery 1.5 times its original size
True aneurysm: involves all 3 layers of the arterial wall
False aneurysm: presence of blood flow outside the normal layers of the arterial wall (pseudoaneurysm)

1. AAA
   a. Def'n
      i. Dilation of the abdominal aorta to 1.5 times its size (usually 3cm or greater in diameter)
   b. Types
      i. Infrarenal: Aneurysmal below renal arteries
      ii. Suprarenal: Extension of aneurysm above renal arteries
      iii. Juxtarenal: Do not involve renal arteries; however, open surgical repair would require clamping above the renal arteries
   c. Etiology/Risk factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking History</td>
<td>5.1</td>
</tr>
<tr>
<td>Family History</td>
<td>1.9</td>
</tr>
<tr>
<td>Older age (per 7 year interval)</td>
<td>1.7</td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>1.5</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>1.4</td>
</tr>
<tr>
<td>COPD</td>
<td>1.2</td>
</tr>
</tbody>
</table>
d. Symptoms
   i. Majority of AAAs are asymptomatic
   ii. Rarely size will cause compression:
       1. Duodenum (early satiety, N/V)
       2. Ureters (hydronephrosis)
       3. Iliocaval compression (thombosis)
       4. Vertebral compression (back pain)
       5. Peripheral embolic symptoms (as thrombus or atherosclerotic disease breaks from aneurysm sac and travels peripherally)

e. Physical Examination
   i. Pulsatile abdominal mass
   ii. Peripheral pulses may yield peripheral aneurysms (particularly popliteal)

f. Imaging
   i. U/S:
      1. Pros: least expensive and least invasive. Should measure diameter in the anterior-posterior dimension for highest accuracy
      2. Cons: Cannot accurately detect rupture, and often not the upper extent of AAA. May be difficult to visualize due to bowel gas or in obese patients.
   ii. CTA:
      1. Pros: accurate dimensions of aneurysm, and detection of rupture. Used to evaluate patients for potential endovascular repair.
      2. Cons: Patients are exposed to radiation and contrast dye. CT scanner can be a dangerous place for unstable patients.
iii. Screening:
   1. Recommendations are to screen all males above the age of 65 that have ever smoked (defined as 100 lifetime cigarettes), with an abdominal ultrasound. OR if a first-degree relative is diagnosed with AAA.

g. Risk of rupture
   i. Diameter:

<table>
<thead>
<tr>
<th>AAA Diameter (cm)</th>
<th>Rupture Risk (%/yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;4</td>
<td>0</td>
</tr>
<tr>
<td>4-5</td>
<td>0.5 – 5</td>
</tr>
<tr>
<td>5-6</td>
<td>3 – 15</td>
</tr>
<tr>
<td>6-7</td>
<td>10 – 20</td>
</tr>
<tr>
<td>7-8</td>
<td>20 – 40</td>
</tr>
<tr>
<td>&gt;8</td>
<td>30 – 50</td>
</tr>
</tbody>
</table>


   ii. Systemic factors: HTN, current smoking, COPD and female gender are independent risk factors for rupture

h. Medical Management: focuses on reducing concomitant cardiovascular risk
   i. Smoking cessation
   ii. HTN management (Beta-blockers, ACE-inhibitors)
   iii. Cholesterol reduction (statins, diet)
   iv. Exercise therapy
Surgical Management: focuses on preventing rupture. The decision for surgical repair is multi-factorial and weighs the risks and benefits of surgery carefully. Generally a diameter > 5.5cm indicates the risk of rupture is greater than the risks associated with surgical repair.

i. Endovascular Repair: for those with appropriate anatomy as determined by CT imaging, a bifurcated tube graft is inserted within the aorta to exclude the aneurysm sac from systemic circulation.

ii. Open Repair: for those unfavorable to endovascular repair. Often through an abdominal incision the retroperitoneal aortic aneurysm is exposed, excluded from circulation by proximal and distal vascular clamps, and the aortic sac is opened and replaced by a synthetic tube graft.
SELECTED TOPICS: ANEURYSMS

2. TAA/TAAA
   a. Def'n: Diameter larger than 150% normal
   b. Crawford classification:
      i. Type 1: Distal left subclavian above renal arteries
      ii. Type 2: Distal left subclavian beyond renal arteries
      iii. Type 3: Distal 6\textsuperscript{th} rib beyond renal arteries
      iv. Type 4: Distal 12\textsuperscript{th} rib to iliacs
      v. Type 5: Distal 6\textsuperscript{th} rib above renal arteries
   c. Symptoms:
      i. Majority are asymptomatic
      ii. 20-30\% will have associated AAA
   d. Imaging:
      i. Generation of sagital, coronal, oblique, and 3-D reconstructions
      ii. Allows visualization of disease in branch vessels
   e. Risk of rupture: is difficult to determine, but very high mortality with rupture, generally candidate for repair at 6cm diameter
   f. Endovascular Repair: At times a preferred option with lower morbidity and mortality, however, a number of factors to consider for repair
3. Popliteal aneurysms
   a. Def’n: Often diameter > 2cm (variable sizes of native popliteal arteries (0.5 – 1.1cm) requires a fluctuating definition of the size cutoff)
   b. Epidemiology:
      i. 95% occur in males
      ii. Contralateral popliteal aneurysms found in ~50%
      iii. AAA found in ~40%
   c. Symptoms
      i. Often asymptomatic pulsating mass behind knee
      ii. Minority will experience pain in area from compression
      iii. Acute ischemia may result if aneurysm sac thrombosis or causes peripheral emboli
   d. Physical Examination:
      i. Pulsatile mass in area of popliteal artery
      ii. Required to examine for AAA due to high association
   e. Imaging
      i. CTA or traditional angiography is used to determine size and potential treatment
   f. Risks:
      i. Limb ischemia: 35% risk of thrombosis at 3yrs, with 25% risk of amputation.
      ii. Rupture is rare
   g. Treatment: Often done when aneurysm size reaches 2-3cm
      i. Open Repair:
         1. Bypass and ligation, or interposition grafting
         2. Improved patency rates as compared to endovascular.
      ii. Endovascular Repair:
         1. A feasible approach for those patients too unwell for open repair.
4. Ruptured Aneurysms
   a. Def’n: An acute and often lethal condition. Rupture of the aorta will result in rapid deterioration of a patient unless the bleeding is contained.
   b. Symptoms/PE
      i. Triad for rupture AAA: abdominal pain, shock, pulsatile abdominal mass
   c. Approach:
      i. Establish adequate venous access for resuscitation (2-3 16G)
      ii. Activation of the vascular team without delay
      iii. Activation of transfusion protocol
   d. Imaging can be difficult as these patients quickly deteriorate
      i. If the patient is hemodynamically stable a CTA will establish if an endovascular approach can be offered
   e. Open vs endovascular
      i. Dependent on aneurysm anatomy and stability of patient
      ii. If able, an aortic angiogram can be performed in the OR to determine if there is an endovascular option
SELECTED TOPICS: LOWER LIMB ISCHEMIA

1. Vascular Claudication
   a. Def’n: Burning/Cramping/Aching pain occurring consistently with activity, and alleviated by short bouts of rest.
   b. Risk factors:
      i. Advanced age, race (non-Hispanic blacks), male gender, diabetes, dyslipidemia, smoking, hypertension, hypercoaguability, chronic kidney diseases
   c. Physical Exam
      i. Blood pressure in both upper extremities
      ii. Peripheral pulse assessment (presence, strength, and character)
      iii. Lower extremity motor and sensory exam
      iv. Muscle wasting, thin and dry skin, or ulceration may be present
   d. Neurogenic claudication: pain caused by lumbosacral nerve root impingement
      i. Often worse walking downhill, and pain is positional
   e. Noninvasive imaging vs invasive imaging (CTA/angiogram)
      i. Hematologic evaluation: CBC, fasting blood glucose, creatinine, fasting lipid profile, urinalysis
      ii. Ankle Brachial Index (ABI): ratio of blood pressure in the lower legs to the blood pressure in the arms
         1. ABI of >1.2: Calcification of arteries
         2. ABI of 0.9 – 1.2: Normal
         3. ABI of <0.9: Suggests arterial stenosis
   f. Conservative management
      i. Antiplatelet therapy: ASA 75-325mg daily
      ii. Smoking cessation
      iii. Exercise training: develops collateral vessels
      iv. Cholesterol therapy: Statins and diet modification
SELECTED TOPICS: LOWER LIMB ISCHEMIA

2. Acute Limb Ischemia
   a. Def’n: Sudden deterioration in the arterial supply to a limb.
      i. Causes: Thrombosis, embolus, trauma, iatrogenic

   b. Classification

<table>
<thead>
<tr>
<th>Category</th>
<th>Description or Prognosis</th>
<th>Sensory Loss</th>
<th>Motor Weakness</th>
<th>Arterial Doppler</th>
<th>Venous Doppler</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Not immediately threatened</td>
<td>None</td>
<td>None</td>
<td>Audible</td>
<td>Audible</td>
</tr>
<tr>
<td>2a</td>
<td>Threatened – marginally. Salvageable if immediately treated</td>
<td>Minimal (toes) or none</td>
<td>None</td>
<td>Inaudible</td>
<td>Audible</td>
</tr>
<tr>
<td>3</td>
<td>Irreversible – Major tissue loss or permanent nerve damage inevitable</td>
<td>Profound – Anesthetic</td>
<td>Profound – Paralysis</td>
<td>Inaudible</td>
<td>Inaudible</td>
</tr>
</tbody>
</table>

c. Conservative
   i. Anticoagulation: IV Heparin is given initially as a bolus followed by a continuous infusion
      1. Class 1 and 3 benefit from anticoagulation until an OR can be scheduled for definitive treatment
SELECTED TOPICS: LOWER LIMB ISCHEMIA

d. Invasive
   i. Operative embolectomy
      1. Embolus can be removed percutaneously with Fogarty catheter. Subsequent bypass may be required
   ii. Operative thrombectomy
      1. Thrombus removed through open arterial dissection, and usually surgically bypassed
   iii. Catheter directed thrombolysis
      1. Dissolves clot in both large and small arteries
      2. Absolute contraindications: recent stroke or neurosurgery within 2 months, major surgery within 2 weeks, those at significant risk of bleeding

e. Compartment syndrome: Increase in intracompartmental pressure impairing tissue perfusion
   i. Paresthesia, Poikilothermia, Palor, Pulselessness, Paralysis

f. Reperfusion of ischemic tissue presents a high risk of compartment syndrome.
   i. Fasciotomy required for Class 2b or 3 acute ischemia
3. Critical Limb Ischemia  
   a. Def'n: chronic lower extremity PAD and ischemic rest pain or the ischemic changes of non-healing ulcers and gangrene  
      i. Typically symptoms present > 2 weeks  
      ii. ABI < 0.5  
      iii. 1 year outcomes:  
          1. 45% alive with 2 limbs  
          2. 30% amputation  
          3. 25% mortality  
   b. Classification

<table>
<thead>
<tr>
<th>Fontaine Grade</th>
<th>Rutherford Category</th>
<th>Clinical Description</th>
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<tr>
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<td>0</td>
<td>Asymptomatic</td>
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<tr>
<td></td>
<td>1</td>
<td>Mild claudication</td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>Moderate claudication</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Severe claudication</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>Ischemic rest pain</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>Minor tissue loss</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Major tissue loss</td>
</tr>
</tbody>
</table>
SELECTED TOPICS: MESENTERIC ISCHEMIA

1. Def’n: Occurs when perfusion of visceral organs fails to meet normal metabolic requirements
   a. Acute: occurs rapidly over hours to days and frequently leads to acute intestinal infarction requiring resection
      i. Embolic:
      ii. Thrombotic:
   b. Chronic: progresses over weeks to months
      i. Most commonly as a result of progressive atherosclerosis

2. Symptoms/PE
   a. Acute:
      i. Sudden onset abdominal pain. Classically the pain is out of proportion to findings on clinical exam
      ii. Late findings of peritonitis, vomiting, bloody stools, and shock are worrisome
   b. Chronic:
      i. Abdominal pain – dull/crampy (intestinal angina) most commonly after meals (post prandial) or when gut is stressed requiring increased blood flow
      ii. Sitophobia (fear of eating)
      iii. Weight loss

3. Imaging
   a. CTA: gives good visualization of splanchnic vasculature, pathology of occlusion, other intrabdominal findings and treatment planning
4. Conservative management
   a. Medical treatment alone is not effective. Lower other cardiovascular risk factors may improve prognosis.

5. Operative management
   a. Endovascular: if anatomically suitable, angioplasty or a stent may be placed in the area of occlusion or stenosis
   b. Open repair: often patients will require resection of ischemic bowel, at this time a surgical bypass may be performed or a hybrid procedure with angioplasty +/- stenting
SELECTED TOPICS: CAROTID DISEASE

1. The primary goal of treatment of cerebrovascular disease is prevention of stroke, most commonly from embolic plaque.

2. TIA: last 1-10 min with complete resolution and can include the following symptoms:
   a. Embolic symptoms:
      i. Transient monocular blindness or field cuts
      ii. Dysarthria (disturbance in articulating)
      iii. Dysphasia (disturbance in communication)
      iv. Aphasia (disturbance in formulation or comprehension of language)
      v. Monoparesis (single limb weakness)
      vi. Hemiparesis (unilateral weakness)
      vii. Hemisensory deficit
   b. Hypoperfusion symptoms
      i. Bright light amaurosis
      ii. Lightheadedness or presyncope associated with any of the above focal deficits

3. Imaging
   a. Ultrasound good initial test that is noninvasive
   b. If unable to obtain ultrasound a CTA or MRA can be performed
## SELECTED TOPICS: CAROTID DISEASE

### 4. Evidence for intervention

<table>
<thead>
<tr>
<th>Study</th>
<th>Stenosis %</th>
<th># of pts</th>
<th>Endpoint</th>
<th>Medical</th>
<th>Surgical</th>
<th>Absolute RR</th>
<th>Relative RR</th>
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<tbody>
<tr>
<td>NASCET</td>
<td>70-99%</td>
<td>659</td>
<td>2-yr ipsilateral stroke</td>
<td>26%</td>
<td>9%</td>
<td>17%</td>
<td>65%</td>
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<tr>
<td>Symptomatic</td>
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<tr>
<td>ECST</td>
<td>80-99%</td>
<td>576</td>
<td>3-yr ipsilateral stroke</td>
<td>20.6%</td>
<td>6.8%</td>
<td>13.8%</td>
<td>67%</td>
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</tr>
<tr>
<td>NASCET</td>
<td>50-69%</td>
<td>858</td>
<td>5-yr ipsilateral stroke</td>
<td>22.2%</td>
<td>15.7%</td>
<td>6.5%</td>
<td>29%</td>
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<tr>
<td>ACAS</td>
<td>60-99%</td>
<td>166</td>
<td>5-yr ipsilateral stroke</td>
<td>11%</td>
<td>5.1%</td>
<td>6.1%</td>
<td>53%</td>
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</tr>
<tr>
<td>ACST</td>
<td>60-99%</td>
<td>312</td>
<td>5-yr any stroke</td>
<td>11.8%</td>
<td>6.4%</td>
<td>5.4%</td>
<td>46%</td>
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<tr>
<td>Asymptomatic</td>
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</table>
SELECTED TOPICS: RISK REDUCTION

<table>
<thead>
<tr>
<th>Disease</th>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prehypertension</td>
<td>120 – 139</td>
<td>80 – 99</td>
<td>Lifestyle changes</td>
</tr>
<tr>
<td>Stage 1 HTN</td>
<td>140 – 159</td>
<td>90 – 99</td>
<td>Lifestyle + 1 of: Thiazide diuretics, ACEI, BetaBlocker, ARB, CCB</td>
</tr>
<tr>
<td>Stage 2 HTN</td>
<td>&gt;159</td>
<td>&gt;99</td>
<td>Lifestyle + 2 of: Thiazide diuretics, ACEI, BetaBlocker, ARB, CCB</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disease</th>
<th>Goals</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperlipidemia</td>
<td>LDL &lt; 2.59 mmol/L</td>
<td>Lifestyle + statins</td>
</tr>
<tr>
<td></td>
<td>HDL &gt; 1.30 mmol/L</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>HbA1c &lt; 7%</td>
<td>Lifestyle + oral agents, consider insulin</td>
</tr>
<tr>
<td></td>
<td>LDL &lt; 1.8 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>
1. Thrombolytics: Some patients with acute limb ischemia that has an embolic source can be treated with catheter directed thrombolytics
   a. Current recommendations:
      i. Rutherford class I to IIa limb ischemia
      ii. Symptoms of less than 14 days
   b. Absolute contraindications:
      i. Prior ICH or cerebral AVM or intracranial neoplasm
      ii. Ischemic stroke within last 3 months
      iii. Suspected aortic dissection
      iv. Active bleeding
      v. Significant closed head trauma

2. Antithrombotics
   a. Platelet Inhibitors
      i. ASA -
      ii. Mechanism:
         1. Irreversible acetylation of COX1 → blocks synthesis of thromboxaneA₂
         2. Higher doses (1g/d) inhibits COX2 → blocks synthesis of prostacyclin → reduces vasodilation and inflammation
      iii. Indications:
         2. Primary prevention in pop. with risk MI >1%
            a. Pts over 40yrs with 2+ major risk factors
            b. Pts over 50yrs with 1+ major risk factors
iv. Clopidogrel/Ticagrelor –
v. Mechanism:
   1. Selective inhibition of ADP-induced platelet aggregation
   2. Clopidogrel requires CYP450 activation
      a. When given in usual doses takes several days
vi. Indications:
   1. Clopidogrel: Secondary prevention of cardiovascular events in patients with CAD/CVD/PVD → 8.7% reduction when compared to ASA
      a. Clopidogrel + ASA
         i. X6 weeks after bare metal stent for CAD
         ii. Longer for drug eluting stent
         iii. Unstable angina (20% RR compared to ASA)
      b. Bleeding risk 2.7%/yr
   2. Ticlopidine: Used for secondary prevention of cardiovascular events in patients with CAD/CVD/PVD
SELECTED TOPICS: COMMON MEDICATIONS

b. Thrombin inhibitors
   i. Heparin –
      i. Mechanism:
         1. Activates antithrombin and accelerates the rate antithrombin inhibits → Thrombin + factor Xa
            a. Induces conformational change in antithrombin making its reactive site more accessible (2x for Xa)
            b. Binds directly to antithrombin and thrombin bringing reactants closer together
      ii. Pharmacology:
          a. In circulation heparin also binds to endothelium and plasma proteins → dose dependent clearance
          b. Reversible with protamine sulfate: full reversal is 100:1 dose
      iii. Heparin Induced Thombocytopenia (HIT)
          1. Triggered by Ab release against PF4-Heparin complex
             a. Causes release of platelet microparticles
             b. Prothombotic
          2. Stop Heparin → give alternative anticoagulant → No platelet transfusion → No warfarin (acute protein C decrease can lead to necrosis) → Eval for DVT
   ii. LMWH –
      iii. Mechanism:
          1. Activates antithrombin (even more affinity for Xa than heparin)
          2. Not able to bring antithrombin and thrombin together
SELECTED TOPICS: COMMON MEDICATIONS

c. Oral Anticoagulation
   i. Warfarin –
   ii. Mechanism:
      1. All of the VitK dependent clotting factors (2,7,9,10) require carboxylation, which permits Ca binding
      2. The enzyme responsible is VitK dependent carboxylase
      3. During the reaction VitK is oxidized to VitK-hydroquinone
      4. VitK-hydroquinone is then reduced to VitK epoxide, which is then reduced to VitK by VitK epoxide reductase (VKOR)
      5. Warfarin inhibits VKOR
      6. Dependent on reduction in the functional levels of factor X and prothrombin, which have half lives of 24 and 72hrs respectively → therapeutic levels take time
   iii. Pharmacology:
      1. Rapidly and almost completely absorbed by GI tract
      2. Peak blood levels 90min after ingestion
      3. Racemic mixture of R-S enantiomers, which are each metabolized in the liver via distinct pathways
      4. The more active S-isomer metabolized by CYP2C9
         a. Patients with variance of this allele may require lower doses of warfarin
      5. Reversible with oral (faster) or IV dosed Vit K, or octaplex
POST OPERATIVE ORDERS:

a. AAA
   iii. Open
       1. NPO
       2. Bedrest x24hr then AAT (PT/OT)
       3. Monitored bed
       4. NS @200cc/hr x 24hr (reassess frequently)
       5. Ancef/Vanco x24hr
       6. Daily BW (CBC/lytes/BUN/Cr/INR/PTT)
       7. DVT prophylaxis (Fragmin/Heparin)
       8. Hold antihypertensives
          a. Except BetaBlockers (if SBP>120)
          b. Resume coumadin POD#2 (bridge if valve/stroke/PE)

iv. EVAR
    1. CF – DAT
    2. Bed rest x24hr then AAT (PT/OT)
    3. Monitored bed
    4. NS @125-150cc/hr x 24
    5. Ancef/Vanco x24hr
    6. BW x 1
    7. DVT?
    8. Home medications as per usual
POST OPERATIVE ORDERS:

c. Fem – Pop/Tib
   1. CF - FF
   2. Bedrest x24hr then AAT (PT/OT)
   3. Monitored bed
   4. NS @125cc/hr x 24hr
   5. Ancef/Vanco x24hr
      a. If inf. Pip/Tazo x48hr then Amox-Clav x5 days
   6. Daily BW (CBC/lytes/BUN/Cr/INR/PTT)
   7. DVT prophylaxis (Fragmin/Heparin)
   8. Hold antihypertensives
      b. Except BetaBlockers (if SBP>120)
      c. Resume coumadin POD#2 (bridge if valve/stroke/PE)
      d. Daily ASA

d. AKA/BKA
   1. DAT
   2. Bedrest x48hr
   3. PT/OT/SW
   4. Monitored bed
   5. NS @125cc/hr x24hr
   6. Acef/Vanco x24hr
      a. If inf. Pip/Tazo x48hr then Amox-Clav x5 days
   7. Daily BW (CBC/lytes/BUN/Cr/INR/PTT)
   8. DVT prophylaxis (Fragmin/Heparin)
   9. Hold antihypertensives
      a. Except BetaBlockers (if SBP>120)
      b. Resume coumadin POD#2 (bridge if valve/stroke/PE)
      c. Daily ASA
4. POST OPERATIVE ORDERS:

a. Carotid Endarterectomy
   1. NPO x24hr then DAT
   2. Bedrest x24hr
   3. Monitored bed
   4. NS @125cc/hr
   5. Ancef/Vanco x24hr
   6. BW x 1
   7. DVT? – Encouraged to walk and not generally given
   8. Home meds as usual