Symptoms of intervertebral disc herniation

Symptoms usually develop **acutely** with posterolateral disc rupture, but more gradually and chronically with spondylosis (narrowing of spinal canal). The symptoms are **proximal radiating** limb pain, with **numbness** and **paresthesias distally** in the nerve root distribution. Symptoms occur on the root below the named vertebra, i.e. a herniated C3-C4 disc causes C4 pain. In the lumbar spine, the first complaint will normally be nonspecific back pain that becomes more radicular in nature. If there is no loss of sphincter function or pronounced radicular weakness, patients are usually treated conservatively for two weeks, followed by physical therapy. Surgery is for those who do not get better, recur, have significant weakness of a muscle group, or have cauda equine syndrome.

**Cervical radiculopathy**

C2: occipital neuralgia
C3 and C4: nonspecific neck and shoulder pain without weakness
C5 radiculopathy presents with shoulder and deltoid pain with weakness of arm abduction
C6 radicular pain runs distal to the elbow, with paresthesias and sensory loss over the thumb and index finger. Bicep weakness, weakness of wrist extension, and diminution of biceps and brachioradialis muscles are characteristic.
C7 radicular pain runs down the back of arm distal to elbow. Paresthesias occur in the middle finger, index finger, and ring finger. There is minimal sensory loss because of overlap with the C6 and C8 roots. Triceps weakness and weak flexion of the wrist, with absent or diminished triceps reflex are key.
C8 usually present with weakness of intrinsic hand muscles. Radicular pain and sensory changes are in the ulnar distribution.

**Thoracic radiculopathy** tends to be more generalized. Left-sided midthoracic radiculopathy can be mistaken for cardiac disease. Right-sided lower thoracic radiculopathy can mimic gallbladder disease.

**Lumbar radiculopathy**

L1 or L2 radiculopathy can cause hernia-like pain

<table>
<thead>
<tr>
<th>Disc</th>
<th>Nerve Root</th>
<th>Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3-4</td>
<td>L4</td>
<td>Anterior thigh, leg, and medial ankle</td>
</tr>
<tr>
<td>L4-5</td>
<td>L5</td>
<td>Posterior hip and posterolateral thigh and leg</td>
</tr>
<tr>
<td>L5-S1</td>
<td>S1</td>
<td>Hip buttock, and posterior thigh and leg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disc</th>
<th>Nerve Root</th>
<th>Sensory change</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3-4</td>
<td>L4</td>
<td>Anterior leg</td>
</tr>
<tr>
<td>L4-5</td>
<td>L5</td>
<td>medial dorsum of foot and occasionally medial ankle</td>
</tr>
<tr>
<td>L5-S1</td>
<td>S1</td>
<td>lateral foot and ankle</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disc</th>
<th>Motor deficits</th>
<th>Reflex loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3-4</td>
<td>quadriceps</td>
<td>knee jerk</td>
</tr>
<tr>
<td>L4-5</td>
<td>foot&amp;toe extension</td>
<td>none</td>
</tr>
<tr>
<td>L5-S1</td>
<td>plantar flexion</td>
<td>ankle jerk</td>
</tr>
</tbody>
</table>
Discuss the diagnosis of an Anterior Neck Mass

Neck masses can be neoplastic (benign/malignant), congenital, inflammatory, or traumatic. Diagnosis starts with a history and physical. Pertinent aspects of the history include associated symptoms (hoarseness, dysphagia, wt. loss, recent infection, recent trauma), the timing of onset, history of smoking or alcohol, HIV, IVDA, or a history of neck irradiation. On physical exam the scalp, ear and neck skin should be examined as well as the mucous membranes and oral/nasal cavities. The voice and the cranial nerves should be assessed. Other areas of lymphatic drainage should also be assessed, such as the axillae, groins in addition to a complete exam. The location in the neck is important for the differential diagnosis. The anterior triangle is formed by the vertical midline of the neck, inferior border of the mandible and the SCM. The posterior triangle is formed by the SCM, clavicle, and anterior border of the trapezius. The anterior triangle is further divided into the submandibular (submaxillary) triangle, carotid triangle and the submental (suprahyoid) triangle.

The diagnostic workup may include imaging studies (MRI, ultrasound, CT), laboratory tests (i.e. calcitonin, thyroid function tests), and FNA biopsy. Imaging studies can identify benign vs. malignant characteristics. The FNA is the standard method of diagnosis, but is subject to false negative results, sampling error, and variable results based on experience of the cytopathologist. The management depends on the outcome of these studies. The differential diagnosis of anterior neck masses includes lymph node (including reactive, metastatic, lymphadenopathy, lymphoma), dermoid cyst, branchial cleft cyst, carotid body tumor, thyroglossal duct remnant, thyroid nodule, and laryngeal neoplasm.
**DIAGNOSIS OF ACL TEAR**

The ACL is a broad ligament joining the anterior tibial plateau to the posterior femoral intercondylar notch. The tibial attachment is to a facet, in front of, and lateral to the anterior tibial spine. The femoral attachment is high on the posterior aspect of the lateral wall of the intercondylar notch.

The biomechanical function of the ACL is complex for it provides both mechanical stability and proprioceptive feedback to the knee. In its stabilizing role it has four (main) functions: Restains anterior translation of the tibia; Prevents hyperextension of the knee; Acts as a secondary stabilizer to valgus stress, reinforcing the medial collateral ligament; and controls rotation of the tibia on the femur in femoral extensions of 0-30°.

The diagnosis of ACL tear can be confirmed by three tests: the Lachman test, the dynamic extension test, and the Pivot Jerk test. While the Lachman test and dynamic extension test are helpful in making a diagnosis, particularly in the acute injury, the lateral-pivot jerk test is the most important. The lateral pivot jerk test reproduces the rotatory subluxation that occurs in ACL deficiency. The test is difficult to perform. The test is important because the demonstration of the lateral pivot jerk is the replication of the instability that the patient has. MRI is also helpful.

**Posterior hip dislocation**

Posterior hip dislocation usually occurs when the patient’s hip is flexed, a situation classically occurring in a head-on automobile accident when the driver or front-seat passenger’s knee is driven violently against the dashboard. Clinically, this injury is characterized by shortening, adduction, and internal rotation of the affected limb. Diagnosis requires A/P, transpelvic, and oblique x-rays of the affected hip. Common complications of this injury include acetabular fracture, sciatic nerve injury, and fracture of the head or shaft of the ipsilateral femur. If no acetabular fracture is present (or if the fractured fragment is very small), then closed manipulation and reduction is the treatment of choice. Posterior or superior acetabular fractures are indicative of posterior hip dislocation, even if the hip is not displaced at the time of examination. Large fracture fragments require open fixation. Smaller undisplaced fractures can be treated with bed rest and no full weight bearing for about 8 weeks.

References: Way L, Doherty G. *Current Surgical Diagnosis and Treatment, 11th Ed.* pp 1154-1155.

**Conditions Associated with Varicoceles**

Varicoceles are the result of incompetent valves of the pampiniform plexus or obstruction of the testicular vein, and give the scrotum a “bag of worms” appearance. They usually collapse w/ the patient supine—failure to do so suggests left renal vein occlusion (for left varicoceles) since the left testicular vein empties into the left renal vein. Varicoceles have an estimated incidence of 4% in college-aged males and occur more frequently on the left side. Varicoceles have negative impact on male fertility. New onset of varicocele, particularly left-sided after the age of forty, requires work-up for left renal vein occlusion secondary to renal tumor. Right-sided varicoceles are associated with IVC occlusion. Repair of varicocele is by ligation of the spermatic vein w/ collaterals and removing the varicocele. Indications for operation include pain, large size, and subfertility.

Discuss the treatment of basal skull fx

Basilar fractures are linear fractures at the base of the skull. The fracture usually occurs through the temporal bone, with bleeding into the middle ear producing hemotympanum. Often the fracture has caused a dural tear, which produces a communication between the subarachnoid space, the paranasal sinuses, and the middle ear. This offers a route for the introduction of infection into the cranial cavity and is suggested by a CSF leak. As with linear skull fractures, a basilar fracture is not always associated with significant underlying brain injury; these fractures are the result of considerable impact force, however, and TBI must be ruled out.

Basilar fractures can compress and entrap the cranial nerves that pass through the basal foramina, can dislocate the bones of the auricular chain, and can disrupt the otic canal or cavernous sinuses, with subsequent injury to cranial nerves 3, 4, and 5. Fractures of the sphenoid bone can disrupt the intracavernous internal carotid artery, creating the potential for the formation of pseudoaneurysms or carotid venous fistulas. The diagnosis of a basilar skull fracture is based on associated clinical signs and symptoms.

Skull radiographs do not detect basilar fractures well. All patients with clinical evidence suggesting a basilar skull fracture should have a CT scan, to define the fracture and to rule out concurrent intracranial pathology, and should be admitted for observation. Because the basilar skull fracture may afford an entrance for bacteria, antibiotics are often considered. Most CSF leaks spontaneously resolve with no complications in 1 week. Therefore, in general, antibiotics are not given prophylactically during the first week of CSF rhinorrhea. If a patient with a previously diagnosed CSF leak returns to the ED later with fever, the diagnosis of meningitis should be strongly suspected and appropriate workup and antibiotic treatment initiated immediately.

Clinical Characteristics of Basilar Skull Fractures
Blood in ear canal, Hemotympanum, Rhinorrhea, Otorrhea, Battle’s sign (retroauricular hematoma), Raccoon sign (periorbital ecchymosis), Cranial nerve deficits, Facial paralysis, Decreased auditory acuity, Dizziness, Tinnitus, Nystagmus

Describe the treatment of post-op parotitis

Parotitis should be considered as a cause (after the evaluation for the five W’s) of postoperative fevers. Normal saliva has bacteriostatic function. Therefore, it is unusual to acquire spontaneous salivary gland infections in the absence of salivary duct obstruction, except in elderly or debilitated patients.

Predisposing factors include poor oral hygiene, old age, avitaminosis, malnutrition and malignancy.
Time of onset is usually w/in two weeks.
Initiating factors are dehydration and poor oral hygiene
Microbiology: Usually staphylococcal
Signs/Sx- swollen and tender parotid, pus at the opening of Stenson’s duct, high fever, high wbc count
Tx – culture stenson’s duct, give iv abx
If disease progresses, surgical incision and drainage is Necessary.
MANAGEMENT OF REGIONAL SPREAD CA SIGMOID

a. **Clinical presentation** – advanced carcinoma of the sigmoid colon may present as obstruction, perforation, bleeding, or colovesical/colovaginal fistula. This may sometimes be confused with sigmoid diverticulitis, even at operation. Furthermore, diverticular disease is present in 20% of patients with colon cancer. At operation, liver metastases may be appreciated and may or may not change the operative strategy. Obstruction occurs in approximately 15-20% of all colorectal cancer patients, whereas perforation occurs in about 3-9%. Roughly 2/3 will have localized perforation and 1/3 will have free perforation with generalized peritonitis.

b. **Diagnosis** – Lower endoscopy with biopsy, contrast studies of the colon, and CT of abdomen/pelvis are commonly used. Urine analysis will reveal the presence of colovesical fistula with bacterial contamination and hematuria. If perforation is suspected, gastrograffin should be used instead of barium. The entire colon should be inspected if possible for the presence of synchronous lesions. In certain cases, contrast study can examine the rest of the colon even if the lesion does not allow the passage of an endoscope. If the lesion is completely obstructing, care palpation intra-operatively and early post-operative endoscopy should be done. If the CT scan reveals liver metastases, MRI should be ordered to better define these lesions and/or reveal the presence of other smaller mets.

c. **Staging** –

<table>
<thead>
<tr>
<th>TNM</th>
<th>T</th>
<th>N</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>T1, T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage 2</td>
<td>T3, T4</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Any T</td>
<td>N1, N2, N3</td>
<td>M0</td>
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<tr>
<td>Stage 4</td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
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Aster-Coller

<table>
<thead>
<tr>
<th>Stage A</th>
<th>Tis, T1</th>
<th>N0</th>
<th>M0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage B1</td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage B2</td>
<td>T3, T4</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage C1</td>
<td>Any T</td>
<td>N1, N2</td>
<td>M0</td>
</tr>
<tr>
<td>Stage C2</td>
<td>Any T</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td>Stage D</td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
</tr>
</tbody>
</table>

d. **Management** – In high-grade obstruction colon cancers, there are three operations to consider: 1) three stage procedure, 2) Resection with end colostomy and Hartman’s pouch, and 3) subtotal colectomy with ileo-rectal anastomosis. The three-stage procedure consists of a transverse colostomy first, followed by bowel prepping and resection of the tumor with anastomosis, and then the colostomy takedown. This is used only for the poor surgical risk and unstable patients. Resection with end colostomy and Hartman’s pouch or mucus fistula is the most commonly employed option. Subtotal colectomy with ileo-rectal anastomosis avoids the issue of anastamosing un-prepped bowel. It also has the advantage of treating undetected synchronous lesions in the proximal colon as well as being a one-stage procedure. Mortality and morbidity are comparable to that of the two-stage procedure. The disadvantage is the increased operative time and therefore requires a medically stable patient. The loss of colonic absorptive surface can also lead to high stool frequency that requires time to adapt and is therefore more suited to younger and higher-functioning patients.

Perforation can occur at the left colon cancer itself or there can be a right-sided perforation with a left-side colon cancer. Resection of all perforated segments should be done at the first operation. If the patient is stable then reconstruction is considered. Otherwise, a staged
procedure is performed. Subtotal colectomy with ileorectal anastomosis can be considered with right-side perforation secondary to a left-side cancer. Synchronous hepatic lesions are wedge resected at the primary operation if the patient is medically stable and the resection leaves enough liver parenchyma and does not require a formal segmentectomy. Otherwise, major hepatic resection is postponed for 6-9 weeks after careful evaluation of both the number (less than 4) and location of the liver mets. This is indicated only if the primary disease is completely controlled.

References:

Femoral Shaft Fractures

Femoral shaft fractures are common in poly-trauma patients. Careful initial assessment and resuscitation is necessary. Alignment of the fracture and portable traction should be applied before radiographs are taken. Radiographs, antero-posterior and lateral, should include the hip and knee. Simple fractures are isolated femoral fractures with an intact soft tissue envelope and are generally the result of low-energy trauma, although segmental and comminuted fracture patterns are higher energy injuries. Skeletal traction is accomplished through distal femoral or proximal tibial pins connected to balance traction with the knee flexed and the leg supported by slings or a Thomas splint. Early stabilization (<24 hrs) of a femoral fracture decreases respiratory complications, intensive care, hospital stay, and overall cost of care. Ipsilateral fractures of the femoral shaft and the tibia (“floating knee”) are associated with 10% of all femoral fractures, and these are high energy injuries. Mortality and respiratory complications are increased with this combination of fractures, especially when internal fixation is delayed. In general, 20% of femoral and 50% of tibial fractures are open. Ipsilateral hip and femoral shaft fractures occur in 5% of all femoral fractures. Most common are femoral neck fractures, which are often undisplaced and a large percentage are missed on initial examination. Priority is given to treating the hip fracture. A high index of suspicion is necessary to diagnose ipsilateral knee ligament injury. The incidence is approximately 5%. It can go unnoticed during treatment of the femoral fracture. The ideal time to examine for this injury is immediately after femoral shaft stabilization. Many arterial and neurological injuries are associated with knee dislocations. Most patients are treated with a closed, reamed, locked intramedullary nail, which affords better control of length and rotation than an unlocked nail. External fixation should be reserved for grossly contaminated wounds and for severely injured “unstable” patients who have a pulmonary or vascular injury that requires immediate repair after initial stabilization.
Discuss the treatment of low grade acinic cell cancer of the parotid gland

<table>
<thead>
<tr>
<th>Salivary gland tumors—location</th>
<th>Percentage of total salivary gland tumors.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parotid gland</td>
<td>80%</td>
</tr>
<tr>
<td>Submandibular gland</td>
<td>10 – 15%</td>
</tr>
<tr>
<td>Minor salivary glands (including sublingual glands)</td>
<td>5 – 10%</td>
</tr>
</tbody>
</table>

Fine needle aspiration is included in the workup of salivary gland mass; it has a 95% sensitivity. The most common lesion of the major salivary glands is the pleomorphic adenoma (benign mixed tumor). The role FNA is greater in submandibular gland lesions than parotid lesions. The role of CT scan is limited to patients suspicious of malignancy or when distinction between inflammatory and neoplastic disease remains unclear after biopsy. CT can also identify tumor extension into deep parotid lobe and parapharyngeal space.

*Treatment for Salivary Gland Tumors*

Diagnosed by FNA.

Benign

(Includes Pleomorphic Adenoma and Warthin’s Tumor)

Resection of the mass with a margin of normal gland surrounding. This usually involves a superficial parotidectomy (portion of the parotid gland that lies superficial to the facial nerve). If the tumor lies deep to the facial nerve, then a total parotidectomy is indicated.

Benign tumors of the submandibular gland, require total removal of the gland.

Malignant

(Most common: mucoepidermoid carcinoma, adenoid cystic carcinoma, and adenocarcinoma.)

See protocol below.

*Treatment Protocol for Malignant Salivary Gland Tumors*


2. The facial nerve should be sacrificed only for direct tumor invasion or pre-existing facial nerve paralysis.

3. For high grade tumors: elective neck dissection or modified radical neck dissection if a lymph node is palpable.

4. Post-operative radiation therapy is indicated for all high grade tumors; close surgical margins; recurrent disease; skin, bone, nerve, or extraparotid involvement; possible nodes; or unresectable disease.

Surgery: Scientific Principles and Practice. Greenfield, 3rd edition; Chapter 18
Discuss the management of a puncture wound of the fingertip

First, important factors in the patient’s history include the mechanism of injury, the length time since the injury and the tetanus immunization status of the patient.

In regards to the physical exam the damage to the nail and nail bed must be assessed. It must be determined if there is bone involvement, and if there is a foreign body present. To determine if the latter two are present a plain film may be necessary.

Treatment depends on the extent of the injury. If the nail is avulsed, split, or disrupted the nail must be removed and the nail matrix inspected. The nail matrix must be repaired if necessary. If there is a subungual hematoma electrocautery or and 18 guage needle can be used to drain the hematoma. Finally, depending on the status of the patient, tetanus immunization may be indicated.

Describe the diagnosis of a testicular mass

Most cases of testis cancer are brought to clinical attention from self examination. The most common finding is an abnormally enlarged testis or firm nodule. However, any change in previously normal testes, including the presence of a mass, a feeling of heaviness, pain, hardness, or swelling should alert the patient and physician to further evaluation of the testes. Other causes of testicular masses include hydrocele, epididymitis, spermatocele, and orchitis, but to reduce the chances of a delay in diagnosis, any testicular mass should be considered malignant until proven otherwise. Often, scrotal trauma precedes the diagnosis because it prompts either the patient or his physician to examine the testes. Occasionally, the patient may present with orchalgia, but this is more the exception than the rule. The first step is to diagnose is scrotal ultrasonography, which is very helpful in documenting a testicular mass, particularly when a thorough examination is difficult. MRI can sometimes be useful when physical exam and ultrasound results are equivocal. If there is suspicion of a testicular mass, surgical exploration with orchietcomy is indicated. Preoperative evaluation should include CBC, serum tumor markers including lactate dehydrogenase, AFP, and ß-HCG, coagulation profile and CXR.

References:

ECTOPIC PREGNANCY
- causes ~10% of maternal mortality, overall mortality rate of 1-2%, recur in about 10%, 3 out of 100 births
- 96% are tubal in location (most at ampulla - distal 2/3)
- associated with certain risk factors, including: PID (improved treatment leads to scarring but less infertility), surgical correction of fallopian tube abnormalities, previous ectopic, NOT associated with chromosomal abnormalities

Diagnosis
- presents w/ sharp and fleeting abdominal pain (unilateral), irregular bleeding (missed menstruation, period of amenorrhea, vaginal bleeding), 1/2 with palpable mass, hypotension, overt rupture (too late!!!)
- can be detected by 6 weeks' gestation, even as early as 4.5 weeks
- see changes consistent with pregnancy (enlarging uterine corpus, softening of cervix
- GOLD STANDARD - direct vision by laparoscopy
  1. Single measurement of serum progesterone (97.5 sensitivity)
     - indicative of viable pregnancy, production of progesterone from corpus luteum, useful screening test and will prompt further testing
     - 97.5% sensitivity for viable intrauterine pregnancy if levels >25 ng/ml
     - 100% sensitivity for nonviable pregnancies if levels <5 ng/ml, proceed with uterine evacuation
     - if between 5 and 25, need to assess viability by ultrasonography
  2. Serial serum ß-hCG measurements
     - produced by trophoblastic cells, normal doubling of levels every 2 days
     - abnormal intrauterine or ectopic pregnancies have prolonged ß-hCG doubling time
     - can be used to assess viability of pregnancy, also to document effectiveness of diagnostic curettage
  3. Transvaginal ultrasound
     - intrauterine gestation can be visualized if ß-hCG > 1500-2000 mIU/ml (transabdominal, need 6000)
     - if levels are > 1500 and no gestational sac seen, ectopic should be assumed
  4. Uterine curettage
     - trophoblastic villi float in saline and can been in tissue obtained after curettage
     - if no villi, decrease of ß-hCG of >15% 8-12 hours after curettage, complete abortion
     - if no villi, ß-hCG levels plateau/rise, assume ectopic pregnancy
  5. Culdocentesis (large gauge needle perforation of cul-de-sac)
     - can reveal dark blood if there has been pelvic bleeding

Surgical Treatment
- postoperative bleeding, elevated ß-hCG, other symptoms occur in up to 20% of cases, will then need excision of involved oviduct (#1 below) or medical therapy
  1 salpingectomy - offers 100% cure rate
     - laparotomy - if catastrophic hemorrhage or hemodynamic instability
     - otherwise, laparoscopic procedure is more beneficial
  2. linear salpingostomy vs "milking out" ectopic (fimbriae extraction)
     - if ectopic is unruptured and greater than 4 cm
     - incision at antimesentric border, products removed by suction or forceps, incision heal by secondary intention or sutures
  3. segmental resection
     - if ectopic located at isthmus (midportion), need subsequent laparotomy for reanastomosis, otherwise, laparoscopy if do not wish further pregnancy

Medical Treatment (intramuscular Methotrexate)
- eligible if hemodynamically stable, mass < 4 cm by ultrasound
- which inhibits synthesis of purines/pyrimidines (useful in treating gestational trophoblastic disease)
- high doses can cause bone marrow suppression, acute and chronic hepatotoxicity, stomatitis, pulmonary fibrosis, alopecia, and photosensitivity, but treatment of ectopic uses low doses and side effects can be mitigated by leucovorin, little risk of subsequent neoplasia/congenital anomalies in later pregnancies
- commonly get transient pain (3-7 days after initiation of therapy), must distinguish from rupture (which may be accompanied by orthostatic tachycardia, hypotension, falling hematocrit)
- BOTH surgical AND medical treatments need to have weekly monitoring of ß-hCG levels until undetectable

Townsend: Sabiston Textbook of Surgery, 16th ed., Copyright © 2001 W. B. Saunders Company
Discuss the management of melanoma of the thigh

The goals for surgery in patients with melanoma are local control, nodal staging, regional control, and survival benefit. Tumor thickness is the most important prognostic indicator; therefore, proper biopsy is critical. Excisional biopsy is always the most preferred method and should be easily accomplished on the thigh. The specimen should be marked for orientation in case wide local excision is necessary.

Wide local excision for melanoma of the thigh can be carried out by an elliptical incision carried down to the underlying muscle fascia. The excisional margin depends on the tumor thickness:

<table>
<thead>
<tr>
<th>Tumor thickness</th>
<th>Excisional margin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melanoma in situ</td>
<td>0.5 cm</td>
</tr>
<tr>
<td>&lt;1 mm</td>
<td>1 cm</td>
</tr>
<tr>
<td>1-4 mm</td>
<td>2 cm</td>
</tr>
<tr>
<td>&gt;4 mm</td>
<td>&gt; 2 cm</td>
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</table>

Regional lymph nodes are the most common site of metastasis of melanoma. The rate of metastasis is again related to tumor thickness. Melanomas of intermediate thickness (1-4 mm) have a 25% chance of nodal metastasis. Therefore assessing nodal status is of paramount importance. Sentinel lymph node biopsy is effective at determining the presence of nodal metastasis and much less invasive than lymph node dissection.

Lymphadenectomy is indicated for patients with known node positive disease. This includes patients with a positive Sentinel node biopsy or grossly positive nodes. Those with grossly positive nodes should be evaluated for distant metastasis including CT scan. Those with no distant mets should undergo formal lymph node dissection. For melanoma of the thigh this entails a complete ilioinguinal dissection. The goal of this procedure is for regional and overall control of the disease. This dissection involves opening of the femoral canal and removing Cloquet’s node (the lowest node of the iliac chain). Cloquets node should be sent for frozen section, if positive then an iliac and obturator dissection is indicated. If this node is negative the dissection is complete.

Adjuvant therapy has only recently been shown to be effective. IFN-a-2b has been demonstrated to increase median overall survival in patients with stage III disease. This therapy is used following surgical excision.
Describe the conditions associated with peritoneal carcinomatosis

Gastric cancer, small bowel cancer, pancreatic cancer, appendiceal cancer, and colo-rectal cancer all may present with carcinomatosis. Other types of peritoneal cancer include mesothelioma, ovarian cancer, pseudomyxoma peritonei, and sarcomas.

The initial dissemination of cancer occurs via three routes: lymphatic, hematogenous, and peritoneal surfaces. It is possible that spread to peritoneal surfaces may be only superficial contamination and does not always suggest aggressive tumor spread as with lymphatic and hematogenous spread. Prognosis is nevertheless grim (mean survival is only 6 months), though those with more limited disease do have longer survival. Patients will present with pain, debilitating ascites, and starvation.

Patients to undergo treatment are selected based on 1) histopathology of the invasiveness of cancer, 2) CT-scan, 3) peritoneal cancer index (graded based on degree of spread), and 4) completion of cytoreduction (CC) index. Patients receive hyperthermic intraoperative intraperitoneal chemotherapy (HIIC) platinum based chemotherapy. Hyperthermic therapy is beneficial because heat is cytotoxic, there is increased surface penetration of the drug, and the efficacy of chemotherapeutic agents is higher at elevated temperatures. Some patients do achieve disease-free survival.

Peritoneal carcinomatosis is typically seen with metastatic ovarian cancer. Women will present with increased abdominal girth and pelvic mass. Intraperitoneal washes or biopsy will reveal adenocarcinoma. Elevated CA-125 and presence of psammona bodies in a papillary configuration all suggest metastatic ovarian cancer. Treatment is total abdominal hysterectomy and bilateral salpingo-oopherectomy with debulking of intra-abdominal disease, including omentectomy. Adjuvant cisplatin-based therapy is generally required, and five-year survival at this stage is poor. Only 10% will be disease free at two years.

To review ovarian cancer:
Stage 1: one or both ovaries
Stage 2: extra ovarian involvement in the true pelvis
Stage 3: abdominal involvement outside of pelvis
Stage 4: spread beyond abdomen including the liver
Discuss the management of a compartment syndrome GSW SFA

Prolonged ischemia (typically more than 6 hours) can lead to reperfusion syndrome and the associated compartment syndrome of the extremity. If ischemia is more than 6 hours a prophylactic fasciotomy should be considered at the time of revascularization regardless of compartment pressures. A fasciotomy should be performed immediately upon the diagnosis of compartment syndrome. The diagnosis is based on clinical exam and adjunctive tests. Symptoms include pain (often out of proportion to the apparent severity of injury) and paresthesias. Late findings include those of ischemia including paralysis and pallor. On exam the compartments may be firm, and pain is illicit by passive stretch of the muscles within the compartment. Direct measurement of compartment pressures can be done. Pressures less than 20 mm Hg are normal, 20-30 mm Hg is marginal, and those greater than 30 mm Hg are abnormal and are an indication for urgent release.

The treatment is reduction of compartment pressure by fasciotomy. All affected compartments must be released. The underlying muscle should be assessed for viability. In the case of SFA ischemia and reperfusion, the calf compartments are those at risk for developing compartment syndrome. The calf compartments include anterior, lateral, superficial posterior and deep posterior compartments.
Management of a Non-Healing Burn Wound

Proper acute burn care minimizes the need for burn reconstruction. In most patients, few reconstructive procedures are necessary during the first year after injury. Usual exceptions are any contractures that limit the ability to perform ADL. Any contracture around the mouth or the neck that makes airway access difficult assumes a high priority in early reconstruction. However, waiting until all scars have matured completely for over 2 years prior to embarking on any reconstructive operations may prolong recovery unnecessarily. Most burn reconstructive procedures can be performed using a combination of some basic techniques: incisional release and grafting, excisional release and grafting, Z-plasty, and random flaps. Tissue expansion and free flaps are needed less commonly, but they can be useful in selected patients.

Incisional/excisional release: The contracture is placed under tension, and the release is performed sharply. Adjacent areas of hypertrophic scar can be excised if donor sites are adequate to close the larger wound. Full-thickness skin grafts are less likely to contract than thin split-thickness grafts; the former is the closure of choice in selected circumstances such as flexion contractures of the digits. Full-thickness graft site availability generally is more limited than split-thickness, and thicker split-thickness grafts are adequate in most situations.

Z-plasties: Although simple in concept, properly planned and executed Z-plasties are powerful reconstructive tools. The basic steps involved in constructing a Z-plasty include the following:
- Defining the line(s) of tension that need to be modified
- Planning the central limb of the Z-plasty(s) on this line
- Designing the lateral lines, if possible, so that they fall along natural skin lines (Langer lines) after transposition

Designing the angle between the central and lateral lines of the Z-plasty to be less than 90° with the lateral limbs curved and no longer than the central limb. Within these limits, infinite variety is possible by modifying the blood supply of flaps and local tissue elasticity. A 5-flap Z-plasty can be constructed by placing 2 Z-plasties along the same band, oriented so that they are mirror images of one another. This results in a fifth "dog-ear" flap that can be inset to insert additional elastic tissue into the band. Multiple Z-plasties can be used in series along a band for excellent effect.

Flaps and Tissue Expanders: Have a more limited but important role in burn reconstruction. Thin random flaps can be raised on the chest wall to cover small fourth-degree wounds of the hands in selected cases; the flap is divided at 3 weeks. More commonly used are groin flaps, which have earned an important role in reconstructing defects, particularly volar wrist defects associated with high-voltage electrical injury. Tissue expanders are useful, esp in the head and neck. Perhaps most useful are tissue expanders to correct burn-associated alopecia. Like tissue expanders, free flaps offer an important option in selected, difficult wounds (eg, those associated with high-voltage injury and extensive soft tissue loss of the distal lower extremity).

References:
Emedicine: http://www.emedicine.com/pmr/topic163.htm#target10
http://www.face-doctor.com/scar.htm
Characterize endometriosis

Endometriosis is the presence of endometrial glands outside of the uterus. It is usually asymptomatic, and discovered incidentally.

Epidemiology: Endometriosis is found in 1% of women undergoing gynecologic procedures of all types. It is found in as high a 50% of women with chronic pelvic pain. Delayed pregnancy may be a risk factor. Family history is also a risk factor, as having a first degree relative with endometriosis increases ones risk to 7%.

Theories of pathogenesis:
- implantation theory – retrograde mestuation causing endometrial seeding
- direct transplantation – during surgery
- venous or lymphatic spread
- coelomic metaplasia – peritoneal cavity contains undifferentiated cells which can differentiate into endometrial cells

Altered immune function may also play a role in the development of endometriosis.

Clinical Features
- Symptoms- though frequently asymptomatic, can include: pelvic pain, dysmenorrheal, dyspareunia, abnormal menstrual bleeding, and infertility.
- Signs- often none, but pelvic exam may show tenderness on palpation of posterior fornix, tenderness or nodules in cul-de-sac or uterosacral ligaments, adnexal mass, fixation of uterus in retroverted position.

Diagnosis is made by direct visualization with histologic confirmation. Lesions can take on a number of appearances. Usually classified as:
- minimal (isolated implants, no significant adhesions)
- mild (<5cm of total implants, no significant adhesions)
- moderate (multiple implants, may be invasive, can have peritubal adhesions)
- severe (multiple implantsm large endometriomas, dense adhesions)

Treatment
- expectant management – may be used if disease is minimal
- NSAIDs – may be used if pain is mild
- Oral contraceptives – may be used if pain is mild
- Progestins, Danazol, or GnRH analogs – if above medical therapies are insufficient
- Surgery

Surgery is indicated if symptoms are severe, refractory, or if the disease is causing anatomic distortion of organs. Conservative measures include excision, fulgurationor lazer therapy. Definitive surgical therapy requires hysterectomy with or without salpingo-oophorectomy.

Sources
Schenken RS. Pathogenesis, clinical features, and diagnosis of endometriosis. *UpToDate Online*. v11.3. 2003
List the findings of a brachial plexus injury

**Anatomy of the Brachial Plexus**

?? Brachial plexus is formed by the anterior rami of C5, C6, C7, C8 and T1 which unite to form three trunks: upper (C5, C6), middle (C7) and lower (C8, T1)

?? Each of the three trunks divide into anterior and posterior divisions

?? The three posterior divisions unite to form the posterior cord; the anterior divisions of upper and middle trunks form the lateral cord; the anterior division of lower trunk forms the medial cords; the cords are named by their relationship to the axillary artery

---

**Major Branches Cords Divisions Trunks Roots**

<table>
<thead>
<tr>
<th>Musculocutaneous</th>
<th>Lateral</th>
<th>Anterior</th>
<th>Upper</th>
<th>C5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Medial + Lateral</td>
<td>Anterior</td>
<td>Upper</td>
<td>C6</td>
</tr>
<tr>
<td>Ulnar</td>
<td>Medial</td>
<td>Posterior</td>
<td>Middle</td>
<td>C7</td>
</tr>
<tr>
<td>Axillary</td>
<td>Posterior</td>
<td>Posterior</td>
<td>Lower</td>
<td>C8</td>
</tr>
<tr>
<td>Radial</td>
<td>Posterior</td>
<td></td>
<td>Lower</td>
<td>T1</td>
</tr>
</tbody>
</table>

**Brachial Plexus Injuries**

?? **UPPER PLEXUS INJURY** (Erb-Duchenne palsy)

--limb extended at elbow, flaccid at side of trunk, adducted and internally rotated \( \Rightarrow \) waiter’s tip position

--cannot abduct the arm due to paralysis of deltoid and supraspinatus muscles, cannot externally rotate due to paralysis of the infraspinatus and teres minor muscle; cannot flex the elbow due to paralysis of the biceps, brachialis and brachioradialis

--sensation is absent over the deltoid muscle and lateral aspect of the forearm and hand

--most common etiologies: traumatic hyperextension of the head and shoulder (i.e. obstetric injury due to shoulder dystocia, motor vehicle accidents, falls and sports injuries); direct pressure caused by carrying heavy objects (knapsack palsy), idiopathic inflammatory neuritis (Parsonage-Turner syndrome)

?? **LOWER PLEXUS INJURY** (Klumpke palsy)

--involves the segments innervated by C8-T1 (with or without C7 dysfunction)

--dysfunction in intrinsic musculature of hand along w/ paralysis of wrist & finger flexors

--sensory deficit along the medial aspect of the arm, forearm and hand

--associated Horner syndrome (ptosis, miosis and anhydrosis) should alert to the possibility of avulsing injury to the lower brachial plexus

--most common etiologies: Pancoast tumor in apex of the lung, trauma \( \Rightarrow \) humeral head fracture, midshaft clavicular fractures and shoulder dislocations, use of crutches, and iatrogenic causes (shoulder surgery and axillary arteriograms and axillary regional anesthetic blocks)

**References**

Describe the mechanism of collagen degradation

For normal wound healing, collagen must be degraded as well as produced. Breakdown by matrix metalloproteinases, synthesized by inflammatory cells, fibroblasts, and epithelial cells. MMP1, 8 and 13 initiate degradation of the collagen molecule by splitting it into three-quarter and one-quarter fragments termed the TC_A and TC_B fragments. After this initial split, other nonspecific proteases can further degrade into peptides and eventually amino acids. MMPs exist in an inactive or zymogen form that must be activated by other proteases such as plasmin. Once activated, MMPs can be inhibited by complexing the plasma and tissue protein alpha-2 macroglobulin. MMPs can also be inhibited by forming a complex with TIMPS. Tissue inhibitors of metalloproteinases. Pathway for extracellular matrix degradation is complex and controlled.

Major events in wound healing:

<table>
<thead>
<tr>
<th>Injury</th>
<th>3d</th>
<th>7 d</th>
<th>3 weeks</th>
<th>1-2 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clot---growth factors----------collagen deposition------collagen x-link</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Cellular influx:
PMNs-> Macrophages-> Lymphocytes-> Fibroblasts

Most collagens are synthesized by fibroblasts. Basic structural unit is a right-handed triple helix.

Collagenases, gelatinases, and stromelysins are matrix metalloproteinases that degrade ECM components.

MECHANISM OF ALDOSTERONE

Genomic action
- effects are mediated by binding of aldosterone to the mineralocorticoid receptor (forming a complex) in the cytosol of epithelial cells, primarily in renal collecting duct
- transport of ligand-receptor complex into the nucleus where they act as transcription factors which bind to specific hormone response elements on targeted genes leading to altered gene expression
- serum and glucocorticoid-induced kinase appears important
  - increased expression leads to modification of apical Na⁺ channel and basal lateral Na⁺/K⁺-ATPase
  - resulting in increased sodium ion transport across the cell membrane

Nongenomic action
- increasing evidence suggests also a G protein signaling pathway
- seen in both epithelial and nonepithelial cells
- probably modification of the Na⁺/H⁺ exchange activity.

Regulation of activity
- glucocorticoids and mineralocorticoids bind equally to the mineralocorticoid receptor
- tissue specific activity is maintained by presence of a glucocorticoid-degrading enzyme (11ß-hydroxysteroid dehydrogenase)

Functions of aldosterone
1. potassium secretion
   - by principal cells of cortical and medullary collecting tubule
   - early phase
     - greater number of apical Na⁺ channels are in the open state, so that sodium entry into principal cells increases
     - Na⁺/K⁺-ATPase activity increases with Na⁺ entry into these cells
     - electrogenic reabsorption of Na⁺ increases the lumen-negative transepithelial potential
   - late phase
     - more Na⁺/K⁺-ATPase are added to basolateral membrane
     - together, this augments electrochemical gradient for K⁺ secretion
2. sodium reabsorption
   - increasing Na⁺ conductance by the principal cell (open probability increases)
   - Na⁺/K⁺-ATPase increases as a consequence of increased Na⁺ entry into the cell
3. nonclassical effects
   - expression of several collagen genes
   - genes controlling tissue growth factors

Walsh: Campbell's Urology, 8th ed., Copyright © 2002 Elsevier
Larsen: Williams Textbook of Endocrinology, 10th ed., Copyright © 2003 Elsevier
Explain the diagnosis of a burn wound infection

Burn wound infections can be classified on the basis of the causative organism, the depth of invasion, and the tissue response. The entirety of the burn wound must be examined on a daily basis by the attending surgeon. Any change in wound appearance, with or without associated clinical changes, should be evaluated by biopsy. Quantitative cultures of the biopsy sample may identify predominant organisms but alone are not useful for making the diagnosis of invasive burn wound infection. Histologic examination of the biopsy specimen, which permits staging the invasive process, is the only reliable means of differentiating wound colonization from invasive infection.

A change in wound appearance or character provokes the clinician to modify therapy and stimulates an aggressive diagnostic approach. Hemorrhage, rapid eschar separation, or greenish discoloration of eschar or subeschar fat suggests bacterial colonization or invasion of the wound. If bacteremia is documented and other sources are eliminated, urgent surgical intervention is necessary. In the absence of documented bacteremia, signs of sepsis such as hypothermia or hyperthermia, hypotension, decreased urinary output, hyperglycemia, neutropenia or neutrophilia, or thrombocytopenia support early intervention.

Biopsy of the wound has been shown to provide an accurate indication on its status. Studies have reported that quantitative cultures of $10^5$ or more bacteria per gram of tissue or histologic evidence of bacterial invasion of viable tissue correlated with a high (75%) mortality rate. In addition, serial biopsies that indicated advancing wound infection were associated with a mortality rate of 85%, whereas stable or improving wounds were associated with an overall mortality rate of 55%. Routine biopsy of full-thickness burn wounds on an every-other-day schedule has allowed detection of progressive wound infections.

The rapid fixation technique allows histologic diagnosis of invasive infection within 3 hours, whereas quantitative counts and identification of the organism are available within 24 hours. The combined use of histologic and culture techniques provides early diagnosis as well as the identity of the organism and its sensitivity to antimicrobials. However, the only way to conclusively determine burn wound invasion by bacteria or fungi is by histologic methods.

References:
2) Principles and Practice of Infectious Diseases, Mandell, 5th edition, p 3198-3201.
**Strength of intestinal anastomosis**

The strength of intestinal anastomosis was demonstrated by Halsted in 1887 to come from the **submucosa**. This is the only layer to contain any significant amount of collagen. Therefore, any disturbance in collagen synthesis, turn-over, remodeling, will affect the leakage rate of GI anastomosis. Collagen synthesis capacity is relatively uniform throughout the colon, but in the small bowel, synthetic ability is significantly higher in proximal and distal small bowel as compared to the mid-jejunum.

**Hand-sewn vs. stapled** – Stapling causes minimal inflammation and provides immediate strength to the cut surfaces during the weakest phase of healing. Various prospective randomized trials have demonstrated **no differences** in clinical and subclinical leakage rates, length of hospital stay, or overall morbidity, even in adverse conditions.

**Single-layer vs. double-layer** – Pathologic analysis of double-layer anastomoses have revealed increased number of focal areas of necrosis and sloughing in the inner layer. Animals studies have shown that single-layer anastomoses cause less narrowing of the lumen, promote faster vascularization and mucosal healing, and increase the strength of anastomosis in the first few post-operative days. Clinical trials have shown that single-layer anastomoses are associated with a faster postoperative return to normal bowel function (bowel sounds, passage of flatus, return to PO intake). However, they have failed to shown any differences in leakage rates between single- and double-layer anastomoses.

**Reference:**