Bone Neoplasms
Chapter 57

What is more common: primary bone tumors or metastatic lesions?
Metastatic lesions. Primary bone tumors are relatively uncommon.

What are the 3 main symptoms of bone tumors?
1. Pain not relieved by rest, and often a deep bony pain at night. Caveat: benign bone tumors may be painless.
2. Presence of a mass
3. Impairment of function including limp, numbness and decreased range of motion.

Benign Bone Tumors

What are the 4 types of benign bone tumors? Which is most common?

1. Osteoma
2. Chondroma
3. Osteochondroma – most common.
4. Osteoclastoma (giant cell tumor)

What are the characteristics of an osteoma?
Small tumors found on long bones, flat bones, and skull. They can be removed or left alone.

What are the characteristics of a chondroma?
Composed of hyaline cartilage, located on the surface or in the medullary canal. These are common in hands and feet. They are removed if they cause pain.

What are the characteristics of an osteochondroma?
This is the most common kind of benign bone tumor. It originates in the epiphyseal cartilage plate and grows only during periods of skeletal growth. May have one or multiple exostoses (affecting several bones).

What is an osteoclastoma and what are it’s characteristics?
An osteoclastoma (giant cell tumor) most frequently affect adults from 20’s to 40’s and are most frequently found in knees, wrists, and shoulders. They are aggressive and behave rather like a metastatic tumors. Starting in the metaphysis of a bone, they erode the bone architecture, replacing it with tumor cells thus leaving the patient prone to pathologic fractures. They tend to
spread to the epiphysis and joint surfaces. Treated with excision/radiation because they commonly recur.

Malignant Bone Tumors

How does a malignant bone tumor differ from a benign one? How do you determine the extent of a malignant bone tumor?
Benign bone tumors tend to show clear and distinct borders on an X-ray image, while metastatic tumors have indistinct borders because they tend to spread beyond the bone and into surrounding tissues. An MRI or CAT scan can help determine the extent of the spread into soft tissue and medullary cavities. Bone scans look for additional sites of metastases and a biopsy provides final diagnosis.

What are 4 types of malignant bone tumors? Which one is a Paget’s patient most likely to have?
1. Osteosarcoma (Paget’s patients get these. Also, they are the most common kind.)
2. Ewing’s Sarcoma
3. Chondrosarcoma
4. Metastatic bone disease (tumor forms metastatically from some other form of cancer, most commonly lung, breast, prostate cancers.)

What is the most prevalent primary malignant bone tumor? What are the characteristics (sites, symptoms, when they are likely to occur, where they spread?)
Osteosarcoma. These are aggressive and highly malignant. They often occur in the knee as well as the proximal area of the humerus, hands, feet, skull and jaw. They are characterized by a deep bone pain with a sudden onset, occurring during times of increased osteoblastic activity (which is why they affect Paget’s patients and young people who are still growing) starting in the metaphysis and spreading to the periosteum and then adjacent tissues. Metastases to the lungs, though this if often asymptomatic, is common.

What predisposes a patient to an osteosarcoma?
1. A young person who is still growing is at risk due to their bony growth processes and increased osteoblastic activity.
2. Paget’s patients are at risk for similar reasons, as these patients have exacerbated osteoclastic and osteoblastic activity.
3. People with prior radiation treatment and bone infarcts are also included in the risk group.
4. There are also 2 genes that have been identified which predispose a person to this, so there is a genetic predisposition factor.

What is the 2nd most common type of primary bone tumor? Discuss the at-risk group, common sites, symptoms, causes, and metastatic activity.
Ewing’s Sarcoma is the 2nd most common type of primary bone tumor, often seen in patients in their teens, usually caused by a translocation on chromosomes 11 and 22. It is most commonly found in the femur, but also in pelvis, pubis, sacrum, humerus, vertebrae, ribs and skull. Symptoms are pain, decreased range of motion, tenderness in the soft tissues above the bone, fever and weightloss, and pathologic fractures. Ewing’s sarcoma metastasizes to lungs, bone marrow and other bones.
What is an chondrosarcoma?
This is a malignant tumor of the cartilage occurring in the medullary cavity and at the proximal sites where muscles attach. These occur mid or late in life (more often in males). These can form from osteochondromas (benign tumors), are often slow growing and metastasize quite late in their development. Early surgical treatment is best as these can transform into mesenchymal chondrosarcoma which is quite aggressive. Radiation is not so effective against chondrosarcomas.

How does metastatic bone disease differ primary bone diseases?
Metastatic bone disease are bone malignancies that arise not on their own, but as a result of another form of cancer in the body. They arise most often due to cancer of:
- Breast
- Lung
- Prostate
- Kidney
- Thyroid
They tend to occur in the bones because the venous blood flow in the skeleton is sluggish, giving the cancer cells more time to attach and flourish.

What are the most common sites for metastatic bone cancers?
- Spine
- Femur
- Pelvis
- Ribs
- Sternum
- Humerus
- Skull
Often occur in several sites with or without other organs being involved.

What are the associated risks of metastatic bone disease?
increased risk for fracture and disability.

What causes the considerable pain associated with metastatic bone disease?
Periosteal stretching and nerve entrapment

What chemical serum levels will be elevated in this disease?
Calcium and alkaline phosphatase (because calcium/phosphates are being released from bone as the cancer displaces the bony tissues).

What are the treatments for metastatic bone disease?
Radiation, surgery, chemo, bisphosphonates (like for osteoporosis); palliative therapy (make patient comfortable while they freakin’ die, man.)
What is the difference between a seropositive arthritis and a seronegative arthritis?
Seropositive just means there’s a blood test for this type of arthritis and seronegative means there’s not.

What does “spondyloarthropathy” mean?
Just another word for arthritis – a group of multi-system inflammatory disorders affecting the axial skeleton.

…so on the test, you might expect to see the terms “seropositive spondyloarthropathy” or “seronegative spondyloarthropathy,” ‘cuz she’s like that…

What are the seropositive arthritis types?
- Rheumatoid arthritis (RA)
- Systemic Lupus Erythematosus (SLE)
- Systemic Sclerosis or Scleroderma – two forms of this disease
  - Diffuse or generalized
  - CREST
- Polymyositis and Dermatomyositis

What is the general definition of ‘arthritis,’ whom does it affect and what is the cure?
Arthritis is a blanket term used to apply to 1) general wear and tear, pain and mobility problems of joints or 2) systemic autoimmune disorders resulting in joint inflammation. No form of arthritis has a cure. Bummer.

What is autoimmune rheumatic disease?
Systemic, chronic disorders with diffuse inflammation and degeneration of connective tissues.
Seropositive spondyloarthropathies

Sero = serum, so seropositive = there’s a blood test. Spondyloarthropathy = arthritis. That said, all of these diseases are autoimmune disorders but not all of them ‘look’ like arthritis as we tend to think of it from the folk definition.

**Rheumatoid Arthritis (RA)**

Note: I’ll abbreviate Rheumatoid Arthritis to RA in the following questions just to make it easier on my fingers (which, knock on wood, are healthy and RA free) unless there is just too much alphabet soup going on in a sentence or question.

**What is RA?**
An abnormal immune response (autoimmune disorder) that causes inflammation of the synovium and the destruction of joints.

**Does RA affect more men or more women? What are the ages at which one can get it?**
RA is 2-3 times more common in women than in men. RA can happen at any age, but is most common in people from 40-60 years of age.

**There is a genetic predisposition for RA. What are the 2 antigens that can be detected with a blood test which indicate this genetic predisposition?**
HLA DR4 and HLA DRB1.

Note: discussed class – HLA is Human Leukocyte Antigen and is a protein on the surface of WBC’s which have to do with the body recognizing self from other.

**List the 3 basic steps in the immune system pathology associated with RA. What happens to cause RA?**
1. Helper T-cells are activated
2. Cytokines, inflammatory enzymes, are released
3. Antibodies are formed

**What is Rheumatoid Factor or RF and how does it affect the body?**
An antibody that reacts to IgG (immunoglobulin G) which forms immune complexes. Basically, RF combines with IgG in the synovial tissues, lodges on the bone and starts eating away the bony tissue.

To make matters worse, neutrophils, macrophages, and lymphocytes are attracted to the sites to dissolve the immune complexes (above), they release inflammatory enzymes and cause more joint destruction.

**How does RF help diagnose Rheumatoid Arthritis?**
RF is present in detectable levels in 70-80% of RA patients.

**What causes the warmth, redness, and swelling associated with RA?**
Vasodilation and synovial hyperplasia

**Define “pannus.”**
Proliferation of blood vessels and inflammatory tissue in the synovium which leads to the destruction of bone and cartilage and the associated reduction in joint movement.
List the characteristics of RA.
- Joint pain (often coarse), warmth, swelling
- Joint instability
- Joint inflammation
- Joint destruction (irreversible)
- Joints involved: few or multiple.
  Commonly, there is symmetric involvement of diarthrodial joints, but not always
- Muscle atrophy (from disuse due to pain of use)
- Ligamentous stretching
  (because synovial capsule swells so much it stretches the ligaments resulting in weaker joints)
- Disease can be progressive or might express with intermittent exacerbations and remissions

What are the general/whole body symptoms of RA?
- Fatigue
- Anorexia and weight loss
- Generalized aches and stiffness, not necessarily in the affected joint either.

What are the joint-specific symptoms of RA?
- Symmetric involvement of any diarthrodial joint is very common … but may not occur that way all the time.
  (note: diarthrosis is a freely moving joint like fingers, toes, hands, wrists, knees, ankles, etc.)
- Polyarticular (multiple joints, not just a single occurrence like in osteoarthritis)
- Pain and stiffness for 30 minutes to several hours, especially in the morning or after exertion.
- Commonly starts in fingers, hands, wrists; feet knees. Can progress to other joints including cervical spine.

When fingers have RA what joints will you most likely see it in? What kind of deviation is common and in what joint?
Mostly seen in MCP (metacarpo-phalangeal), and PIP (proximal interphalangeal) joints, rarely in DIP (distal). The most common deviation is an ulnar deviation in the MCP.

How does RA weaken the joint ligaments? What is the problem this causes?
When the synovial capsule swells and thickens the associated ligaments stretch causing pain and eventually loosening the tension these ligaments should have. This results in a general weakening of the joint due to the stretched ligaments.

What are the 4 types of deformities that are associated with RA?
1. Metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints deviate to the ulnar side.
2. Joint deformities due to muscle imbalances which increases the risk of subluxation (partial dislocation) and dislocation of joints.
4. Boutonniere’s deformity (almost opposite of swan neck deformity): flexion of PIP joint with hyperextension of DIP.
Discuss the knee problems often associated with RA. Include the 4 common problems of RA in the knee.
The knee is commonly affected, resulting in much of the disability associated with RA.
- Joint contractures and instabilities
- Genu vagus (i.e., knock knees)
- Atrophy of quadriceps
- Baker’s cysts: enlargement of the bursa, pulling the wall of the bursa out; happens in popliteal bursa usually.

How does RA affect the movement of the ankle?
Limits range of motion, making it more difficult to walk.

Neck pain associated with RA leads to 3 other problems. What are these?
- Headaches
- Numbness and tingling
- Muscle weakness

What type of blood related symptoms show up with RA?
- Anemia with a resistance to iron therapy
- Leukopenia (low WBC count)
- Elevated ESR (erythrocyte sedimentation rate – indicates inflammation with higher numbers indicating higher levels of inflammation)

What cardiopulmonary and vascular problems are seen in cases of RA?
RA patients often have a small and medium artery vasculitis in the fingers, eyes, and brain resulting in cardiopulmonary complications. This vasculitis also results in ischemia around the nail folds, ulcers, and neuropathy.

What ocular changes might you see in an RA patient?
Episcleritis and scleritis – both terms refer to inflammation of the white of the eye in which the blood vessels become very noticeable. This is due to the vasculitis of the small and medium sized arteries that occurs in the course of RA.

What are rheumatoid nodules?
A symptom associated with RA. These nodules might be tender or not and tend to develop over pressure points.

List the symptoms needed to diagnose a patient with RA.
In order to be diagnosed with RA a patient must present with 4 of the following symptoms:
- Morning stiffness - 1 hour min for 6 weeks
- Swelling of 3 or more joints for 6 weeks.
- Swelling in wrist, MCP, PIP for 6 weeks
- Symmetric joint swelling for 6 weeks
- Rheumatoid nodules
- Positive RH (rheumatoid factor – but remember this is nonspecific and can be positive in 1-5% of people that do not actually have the disease)
- X-ray changes consistent with RA
Treatment of RA

I don’t think we need to know these, but will let you know after the test.

What is the goal of treatment in cases of RA?
No cure, so treatment is palliative. Reduce pain, minimize stiffness and swelling, maintain mobility, educate the patient.

What are the treatment methods in cases of RA?
- Physical and emotional rest
- Therapeutic exercise – swimming, range of motion exercises, non-weightbearing activities
- Heat and cold
- Work on posture and body mechanics to decrease stress on joints – includes supportive shoes
- Aggressive medication management to reduce risks of irreversible damage – NSAIDs and DMARDs (disease-modifying antirheumatic drugs – target immune rsvp, slow disease progression/joint destruction)

Systemic Lupus Erythematosus (SLE)

What is SLE? Who gets it most often and what causes it?
SLE is Systemic Lupus Erythematosus, an autoimmune disorder, a chronic inflammatory disease in which autoantibodies and immune complexes are formed much like in Rheumatoid Arthritis, but which affect organ tissues (any organ).

Females get it more than males by a factor of 10 to 1. The cause is unknown for the most part, but seems to include sex hormones, chemicals and UV light. Also, there does seem to be a genetic predisposition which occurs more commonly in African Americans.

Name 3 antibodies/immune complexes that are formed in SLE.
- Antinuclear antibodies (ANA)
- AntiDNA antibodies
- Antibodies against blood cells which can lead to anemia and thrombocytopenia (low platelet count)

Of the 3 antibodies, which ones can use use as a serum test for SLE?
ANA is the primary test, but since a patient might have an elevated count of ANA in other disease (and in none at all as well), you can also test for Anti-DNA as a secondary test for SLE. A patient might also have an elevated ESR (like in RA – indicating increased inflammation)

What joint/musculo-skeletal problems might you see in SLE patients?
- Arthralgia (pain of the joints) and arthritis, but without joint destruction
- Ligament/joint/tendon inflammation which may lead to deformities
- Tenosynovitis (inflammation of tendon sheath), most common in hand/arm
- Rupture of patellar and Achilles tendons
- Avascular necrosis – often in hip, shoulder, and humerus, but can be anywhere really.

What is the skin rash often seen in SLE?
Malar rash with photosensitivity. This is a butterfly looking rash spanning the zygomatic area and across the nose.

**What happens in the renal system of many SLE patients which is one of the big reasons for the mortality rate seen in this disease?**
50% of SLE patients have renal involvement. This is expressed as either one or both:
- Glomerulonephritis
- Nephrotic syndrome (kidney spills an enormous amt of protein)

**Discuss the 3 pulmonary involvements in SLE.**
- Pleural effusion – accumulation of fluid between the layers of the pleura
- Pleuritis – inflammation of the pleural membranes surrounding the lung
- Hemorrhage – blood leakage in the lungs.

**What are the 4 cardiac complications associated with SLE?**
- Pericarditis – inflammation of the pericardium - membrane surrounding the heart.
- Myocarditis – inflammation of the heart wall muscle tissues
- Hypertension
- Ischemia

**What central nervous system complications do SLE patients experience?**
- Strokes
- Hemorrhage
- Seizures
- Depression
- Psychosis
All generally due to vascular changes and complications.

**How is SLE diagnosed?**
By a combination of medical history, physical examination and lab work.
Blood is tested for:
- ANA – nonspecific, but 95% of untreated SLE patients have a high ANA titer
- Anti-DNA – more specific
- Anemia
- Thrombocytopenia
- Abnormal WBC (too high or too low)

**Discuss the treatment protocols/goals for SLE.**
- Treatment of chronic and acute symptoms
- Prevent organ damage
- Prevent complications of treatment (how typical of the AMA…)
- Drug therapy to include:
  - NSAIDS, hydroxychloroquine, corticosteroids (mostly for renal/cns complications),
  - cyclophosphamide
**Systemic Sclerosis – aka, Scleroderma**

**What is systemic sclerosis or scleroderma? Whom does it affect and what causes it?**
Systemic sclerosis is an autoimmune disorder of the connective tissue in which there is excess collagen deposited into the skin and internal organs. The cause is unknown. Women get it 4 times more frequently than men. It peaks at the ages of 35-50.

**What are the two forms of scleroderma and where do they express?**
1. **Diffuse or generalized form.**
   This is the more severe type of scleroderma, involving organs very early in the disease. Heart, renal and lung involvement are the most commonly involved organs, indicating a very poor prognosis. Diffuse Scleroderma expresses on the trunk and proximal extremeties, especially on the fingers.
2. **CREST**
   This expresses on the hands and face.

**What parts of the body does the diffuse or generalized form of systemic sclerosis affect?**
Mnemonic: S(i)ck Pie
- **S**kin – tight facial skin and lips
- **C**ardiac
  - Pericarditis
  - heart block
  - myocardial fibrosis (cartilage deposits in the muscle causing stiffness and lack of elasticity)
- **K**idneys – vascular problems leading to big time high blood pressure and renal insufficiency (decreased ability of the kidney to rid the body of wastes).
- **P**ulmonary – dyspnea, pulmonary artery hypertension, respiratory failure
- **I**ntestines – malabsorption, atrophy
- **E**sophagus – hypomotility, trouble swallowing

**CREST is the less severe form of systemic sclerosis/scleroderma. What does CREST stand for?**
- **C** = Calcinosis
  hard knots under the skin which are subcutaneous calcium deposits
- **R** = Raynaud’s
  Reversible vasospasm in arteries of the fingers, especially in the cold.
  (A vasospasm is an exaggerated persistent spasm in the blood vessel wall leading to vasoconstriction and restricted blood flow.)
- **E** = Esophageal dysmotility
- **S** = **Sclerodactyly** – scleroderma of the fingers. *This is the only disease that causes this*
- **T** = Telangiectasias. Aka, little spider veins on the skin. Happens in a lot of disease, but this in combination with the others is indicative of scleroderma.

**What is the treatment for systemic sclerosis/scleroderma?**
Treatment is of symptoms, not of the disease. The focus is on avoiding progression of organ involvement.
Polymyositis and Dermatomyositis
Yet another autoimmune disease…

The root of both of these words is “myositis.” What does this mean?
Inflammation of muscle tissues
(myo = muscle, -itis = inflammation … the s inbetween is just a linking consonant to hold the 2 root words together)

What is the difference between polymyositis and dermatomyositis?
Poly = many, Dermato = skin
Both are a chronic inflammation of muscle tissues, the most serious of which is the involvement of cardiac and pulmonary system muscles. The only real difference between the two is that dermatomyositis expresses with a skin rash in addition to other symptoms. (Dermatomyositis often expresses with a “heliotrope” rash on the skin around the eyes/eyelids.

What are the skeletal muscle symptoms of the –myositis club of diseases?
- Systemic weakness of proximal or skeletal muscles. The most often expressed is shoulder muscle weakness so the patient will have trouble ‘doing’ their hair or working overhead.
- Muscle pain and tenderness

What is the treatment for polymyositis and dermatomyositis?
Corticosteroids
Seronegative spondyloarthropathies

Seronegative refers to serum negative or no blood test. Spondyloarthropathies is another term for arthritis. All of the diseases discussed below are some form of arthritis and look more “arthritisy” than some of the autoimmune varieties did.

What is a seronegative spondyloarthropathy?
A group of multi-system inflammatory disorders affecting the axial skeleton, often the spine. It usually begins at tendon and ligament insertion points. Unlike the seropositive types, the RH factor is negative when the blood is tested.

Remember this: seronegative spondyloarthropathies are associated with HLA-B27! HLA = human leukocyte antigen.

Side note for clarity: 90% of patients with ankylosing spondylitis have the HLA-B27 antigen, but only a few people in the known world with the HLA-B27 antigen in their blood work actually get ankylosing spondylitis.

What are the 4 types of seronegative spondyloarthropathies?
1. Ankylosing spondylitis (or AS)
2. Reactive arthritis (which includes Reiter’s syndrome and enteropathic arthritis, actually)
3. Enteropathic arthritis (an inflammatory bowel disease)
4. Psoriatic arthritis

Ankylosing Spondylitis (AS)

Define it. When does it happen and to whom? What antigen is very commonly present in those that have it?
Ankylosing Spondylitis is a systemic chronic inflammatory disease of the axial skeleton, in which the inflammatory response erodes the sites where the tendons and ligaments attach onto the bone. This expresses with stiffness and pain in the spine. Men get the disease most often and it progresses rapidly and severely. It often begins in late adolescence or early adulthood. 90% of AS patients test positive for HLA-B27.

Discuss the characteristics of this disease.
AS is an inflammatory erosion where the tendons and ligaments attach onto the bone. It begins in the sacroiliac joints and moves to the posterior aspect of the spine. Destruction of these small joints causes spinal fusion which progresses upward along the spine. As the joints fuse together they become ‘squared,’ visible on an X-ray, and looking like a stalk of bamboo. Large synovial joints such as hips and knees become involved, but smaller more peripheral joints do not.

What is the hallmark symptom that screams “Anklyosing Spondylitis!”?
Loss of the lumbar lordosis (curve toward the anterior in the lumbar region that is characteristic of a normal spine) with kyphosis of the thoracic spine (“humpback”). So, in English: flat lower back with a humpback in the upper back.
List all of the clinical symptoms associated with AS.

- **Loss of the natural lower back curve (loss of lordosis) with kyphosis (hump in the thoracic region). This is the characteristic symptom of AS**
- Low back pain that gets worse with rest
- Stiffness in the morning and after resting.
- Pain in the butt (heh heh!), hips, and possibly in the thighs.
- Muscle spasms (due to structural changes of the spine)
- Long term progression = spine fused in flexion (flexion = bending, so spine is fused in mid forward bend position)

**What does “bamboo spine” mean again?**
It’s what an X-ray of a spine with AS looks like. There are bony bridges visible between the vertebrae instead of gaps so the affected portions of the spine look like bamboo.

**What are the 6 major complications of ankylosing spondylitis (AS)?**
1. **Constriction of the chest cavity due to the kyphosis. Causes restrictions in the heart and lungs with resulting problems like loss of lung volume and lowered oxygen levels, pneumonia.**
2. **Osteoarthritis due to the abnormal position and modified weight bearing on the hips, knees and shoulders. Hip involvement is the most disabling of these.**
3. **Anterior uveitis. This is an inflammatory disorder of the eye, usually of the iris (that’s the anterior referred to in this term). This is the most common extraskeletal problem with AS (25-30% of AS patients).**
4. **Weight loss**
5. **Fever**
6. **Fatigue**

(WAC OFF is the mnemonic I used for this—crass, I know, but it works.)

**How is AS diagnosed?**
By history and examination of range of motion of the spine plus X-rays. It may not look severe early on and patient may come in with lower back pain and some loss of the lower back curve.

There could also be an elevated ESR (erythrocyte sedimentation rate) and a mild anemia. Again, 90% of patients test positive for HLA-B27, but many people with this antigen don’t have ankylosing spondylitis, so this is not diagnostic.

**How do you treat ankylosing spondylitis?**
Posture education, exercises (low impact are best) and weight maintenance plus NSAIDS and sometimes anti-rheumatic drugs. Eventually the patient might need surgery to fix the spinal fusions, since over the long term the spine fuses so badly the patient can no longer straighten up.
Reactive Arthropathies

What arthropathies does the category of reactive arthropathies encompass? What triggers a reactive form of arthritis?
Reiter’s Syndrome and enteropathic arthropathies. These are usually triggered by infectious agents which can be urinary, intestinal or respiratory in nature. Can occur in AIDS patients.
Some examples: Chlamydia pneumoniae, Pseudomonas, Salmonella, Shigella, Yersinia, Campylobacter, Streptococcus.

What is Reiter’s Syndrome?
Reactive arthritis plus uveitis (eye inflammatory disease), bowel inflammation, and carditis (inflammation of the heart)

What is the antigen that indicates genetic susceptibility for Reiter’s Syndrome?
HLA-B27 again!

What infections seem to trigger Reiter’s Syndrome?
Genitourinary tract: Chlamydia
GI tract: Salmonella, Shigella, Yersinia, Campylobacter

How do you treat Reiter’s?
With immunosuppressants…ain’t that a bitch for the AIDS patients?

What is an enteropathic arthropathy and how is it treated?
A reactive arthritis associated with inflammatory bowel diseases like ulcerative cholitis or Crohn’s disease. The severity of the arthritis does not necessarily correlate to the severity of the IBD. Enteropathic arthropathy is treated by treating the underlying bowel disease.

Note: inflammatory bowel disease is also called IBD. Ulcerative cholitis affects only the colon; Crohn’s can affect any part of the GI tract from mouth to anus.

Don’t confuse IBD with the less serious IBS (irritable bowel syndrome) which is alternating constipation and diarrhea.

Psoriatic Arthritis

Define Psoriatic Arthritis.
Psoriatic Arthritis is a seronegative arthritis of unknown etiology occurring in 5-7% of psoriasis patients which progresses slowly and may present like a spondyloarthritis or like a rheumatoid arthritis.

What is the required symptom/sign for a diagnosis of Psoriatic Arthritis?
Presence of skin or nail changes of the psoriasis. Often there is flakey and dry pitting on the nails. An X-ray will probably show affectations of the distal phalangeal joints - little spikes coming off of these bones when they should be smoothe.

What acidic level changes might you see in the bloodwork of a person with Psoriatic Arthritis?
Might see an elevation in uric acid.

**Osteoarthritis (OA)**

**What is another term for Osteoarthritis?**  
DJD or Degenerative Joint Disease.

**What is OA?**  
A local or generalized degenerative joint disease characterized by a loss of cartilage and synovitis resulting from the inflammation caused by the cartilage attempting to repair itself. As this cycle causes joint pain, stiffness, and limited ROM, this disease is the leading cause of disability in the elderly.

OA can be primary, occurring on its own, or secondary, occurring as the result of joint defects, trauma, metabolic and inflammatory disorders.

**What 2 joint change characteristics are seen in cases of osteoarthritis?**  
1. Loss of cartilage  
2. Synovitis – inflammation of the synovial capsule caused by the body attempting to repair the damaged cartilage.

**What are the 4 symptoms/signs of OA?**  
1. Joint pain  
2. Stiffness  
3. Limited range of motion  
4. Possible instability and deformities

**Are men or women more affected by OA?**  
Men are more affected at earlier ages but my middle age women get it more often.

**What manifestation of OA is hereditary?**  
Osteoarthritis in the distal interphalangeal joint (DIP) of the hand.

**What is a high risk factor for OA of the knee?**  
Aside from previous knee injuries, obesity is the high risk factor for OA in the knees. As a matter of fact, if you have an obese patient with OA and can get them to lose as little as 10 lbs they will get a lot of relief.

**What does an unaffected articular/synovial joint look like?**  
Articular cartilage is smooth and the synovial joint provides a nice slick fluid to lubricate the joint with very little friction. When the joint bears weight the weight is transferred to the springy subchondral bone below the joint.

In a normal joint cartilage consists of chondrocytes and an extracellular matrix of water, proteoglycans (to give both stiffness and elasticity, collagen, and ground substance (a highly hydrated gel).

Articular cartilage undergoes constant turnover of chondrocytes to replace the worn-out matrix.
What’s the difference between the unaffected joint as above and one with osteoarthritis?
Early on in OA, cartilage has more water and fewer proteoglycans (which give an unaffected joint stiffness, elasticity), chondrocytes are reorganized and collagen weakens. Cytokines, inflammatory enzymes, are released which degrade the cartilage and cause edema in the matrix.

As a result, smooth articular surfaces get rough and erode away, forming cracks in the cartilage over time. These fill with synovial fluids which widens the cracks. Eventually the cracks extend to the subchondral bone which hardens and thickens and can no longer absorb the shock of use.

Pieces of bone and cartilage form loose bodies. Any new bone formed is sclerotic and less shock absorbing. Other bone growth problems in OA include bony outgrowths or osteophytes.

What is an osteophyte?
A bony outgrowth resulting from the progression of osteoarthritis.

Which joints are most commonly affected in OA?
Hips, knees, lumbar/cervical vertebrae, PIP and DIP joints in the hands, 1st MTP (metatarsophalangeal) joint in the foot. Joint involvement can be mono or polyarticular (just one or several/many).

What are the clinical manifestations of OA?
- Pain
- Crepitus
- Limited ROM, instability of joints
- Joints feel hard and enlarged.
- Joint involvement:
  - Single joint (monoarticular) or multiple joints (polyarticular)
  - Secondary joints may be affected due to the shift in forces caused by trying to protect the joints with OA

How is OA diagnosed?
By history and physical exam findings, Xrays and exclusion of other diseases by lab work. Labs are typically normal, though the ESR may be slightly elevated.

What X-ray manifestation will you see with OA of the knee?
Narrowing of the medial joint space occurs first. Might also see osteophytes (bone spur or bony outgrowth)

How do you treat OA?
- Rehab to strengthen opposing muscle groups that cushion weight bearing
- Rest affected joints
- Splints
- Heat/cold
- Weight reduction
- Education about proper body mechanics
- Acetominophen orally and NSAIDs
- Intraarticular corticosteroid injections
- Viscosupplementation by injection (especially for knees—synthetic synovial fluids injected)
Surgery to debride, remove loose bodies, or to replace joints.

Gout

What is gout? Include primary and secondary gout in the definition.
Either an acute or recurrent disease in which monosodium urate or uric acid crystals are deposited in the joints, usually in the peripheral joints where body temperatures are cooler. Primary gout accounts for about 90% of the cases occurring mostly in men ranging in ages between their 40’s and 60’s and is hereditary. Secondary gout results from another primary disorder.

What is pseudogout?
Pseudogout, also called chondrocalcinosis, occurs when calcium pyrophosphate dihydrate crystals deposit in the joints.

Gout comes from measurably elevated uric acid levels in the blood. What causes this to happen in cases of primary and secondary gout?
Serum uric acids elevate as a result of purine metabolism. This can be from overproduction of purines, increased breakdown of purines, or decreased urinary excretion. Primary gout, which is hereditary, is probably the result of an enzyme deficiency which causes 1) overproduction and 2) inadequate elimination. Secondary gout is often the result of increased cell turnover such as cancerous tumors lysing during treatment.

What causes an attack of gout? Where is the classic site to get an attack?
Again, elevated uric acid levels. Crystals precipitate into the joint and cause inflammation. Peripheral joints where the body temperatures are cooler are the likely spots to get an attack, but the great toe is the classic spot.

What are the long term risks for chronic repeat attacks of uncontrolled gout?
Chronic arthritis and the formation of large, hard nodules called “tophi.”

What is chronic tophacious gout?
Repeat gout attacks causing tophi, large hard nodules. This is usually polyarticular, affecting more than one joint. (Gout is usually limited to one joint.)

Describe the characteristics for a gout attack.
- Gout attacks are usually monoarticular, classically in the great toe.
- Other common sites: tarsal joints, insteps, ankles, knees, wrists, fingers, elbows.
- Severely painful attacks that happen most often at night
- Redness and swelling of the affected area
  (If there’s redness there won’t be a distinct border, infection will)
- Can last from days to weeks.

What are the triggers to that can precipitate an attack of gout?
- Excessive exercise
- Medications
- Food/diet
Alcohol
(Common to have gout attacks after beer/barbeque/burgers…lots of protein breakdown plus the alcohol)

How is gout diagnosed? What is the “gold standard” test for gout?
Diagnosed by a physical and medical history. Lab tests include a test for hyperuricemia (but a lot of people have this that don’t have gout), 24 hour measure of urine urate excretion (carry around a jug and pee in it for 24 hours…pretty!), synovial fluid analysis.

The gold standard is the synovial fluid analysis.

How is gout treated?
NSAIDs and uric acid lowering meds like colchicine, allopurinol.
(Oh yes, let’s throw some meds at it. Don’t counsel the patient to lay off the beer and barbeque!!!Geez.)

Juvenile Rheumatoid Arthritis (JRA)

How old is a JRA patient likely to be, generally speaking?
16 years of age or less.

10% of JRA patients have systemic symptoms. List these symptoms.
☐ High fever
☐ Rash
☐ Lymphadenopathy
☐ Hepatosplenomegaly (enlarged spleen and liver)
☐ Leukocytosis
☐ Anemia

What are the fatal complications of JRA patients who have systemic symptoms?
Infection, heart disease, and adrenal insufficiency.

What do the terms pauciarticular and polyarticular mean when used in relation to Juvenile Rheumatoid Arthritis?
Pauciarticular means few joints, less than 4 joints affected; this applies to about 50% of JRA patients. Polyarticular means many joints, more than 4, which applies to about 40% of JRA patients.

Juvenile Arthropathies

How is SLE different in children than in adults?
Similar to how the disease expresses in adults, with the degree of renal involvement determining the prognosis for the juvenile.

How is dermatomyositis different in children than in adults?
The big difference is that kids get calcifications over pressure points. There is also a generalized vasculitis in juvenile versions of dermatomyositis.

**Spondyloarthropathies in children are more common when this antigen is present:**
HLA-B27. Again!

### Rheumatic Diseases in the Elderly

**Why are elderly people harder to diagnose with rheumatic diseases?**
Because they have an increased production of autoantibodies anyhow.

**What form of RA is generally mild in elderly people?**
The seronegative kind.

**What about SLE in older people? What might be causing that?**
More likely to be drug induced. (…cuz we’re grossly overdrugging them anyway)

**What is the most common form of arthritis in the elderly?**
OA

**What kind of meds might cause gout?**
Diruretics used for hypertension

**What’s another kind of gout that might be more common in old people?**
Pseudogout due to calcium deposition.

**Name 2 shoulder pain causes that are also common in elderly people.**
Bursitis and tendinitis

**What is PMR?**
Polymyalgia Rheumatica, an inflammatory condition causing aches and morning stiffness in the pelvis and shoulders. The onset of PMR is often abrupt

**Who is most at risk and at what ages?**
Women, of course, usually after age 60 with the risk going up as age increases.

**What is a great risk for patients with PMR?**
Giant cell arteritis which can cause temporal arteritis (inflammation of blood vessels in the temple leading to the eye) which can cause blindness.

**What is needed for a diagnosis of PMR?**
Pain and stiffness for more than a month, with an elevated ESR (erythrocyte sedimentation rate)

**What drug causes the symptoms to resolve quickly?**
Prednisone. Symptoms are usually better within a day or two. Dose is tapered over a year and a half to two.
Alterations in Skin Function/Integrity
Chapter 61

What 6 causes can result in skin disease?
1. Sunlight
2. Systemic disease
3. Insects
4. Infections
5. Physical agents
6. Chemical agents

What factors can make a skin disease difficult to diagnose?
Skin tone of the individual, scratching itches, infections and treatments used (which can unnaturally make things better or worse…)

Name 2 types of flat lesions.
Macules and patches. Can’t feel these because they are flat against the skin. Macule can be a freckle like thingy and a patch could be like a birthmark.

Name 5 types of solid palpable lesions
Papules (small and in the skin tissues)
Plaques (big patches of stuff like you see in psoriasis)
Nodules (a firm bump under the skin)
Tumor
Wheal (a hive)

Name 3 kinds of fluid filled lesions
Vesicle – small blister filled with clear fluid
Bulla – a bigger blister filled with clear fluid, can result from friction
Pustule – filled with pus….and an old X-Files favorite.

Define the following types of skin pathologies
☐ Rash
Temporary eruption of the skin.

☐ Lesion
Traumatic or pathological loss of tissue (in this case skin) continuity, structure or function
Can be a cut, scab, anything disrupting the skin.

☐ Lichenation
Thickened, rough skin due to repeated scratching

☐ Excoriation
Raw, broken skin.
The earlier stage of lichenation before the thick skin builds up to protect itself from the scratching.
Blister
Also called a vesicle. A fluid filled papule caused by disruption of the skin layers due to friction or a bullous (blistery) skin disorder

Callus
Plaque of thick skin due to chronic pressure or friction which causes buildup (hyperplasia) of dead keratinized skin cells. Diffuse border, unlike a corn…

Corn
Small area of hyperkeratosis (thickened skin) with a well-defined border

Pruritus
Itching.

Define the following terms which are descriptive of skin problems
- Blanching – goes white when you press on it.
- Erythema – reddening of the skin
- Hemorrhagic or purpuric – bleeding out into the skin
- Pigmentation – the coloration of a skin problem.
  - Hypopigmentation: lighter than surrounding tissue
  - Hyperpigmentation: darker than surrounding skin

What is “pruritus”?
Itching

In what disorders do you see pruritus?
Many skin disorders as well as in kidney disease and biliary disease

What triggers pruritus?
Warmth, touch, vibration, dry skin. Also, naturally occurring chemicals our body produces such as histamines, bradykinins, substance P, bile salts, and prostaglandins.

What is the reflex response to pruritus and does it solve the problem?
Scratching is the reflex response, but it does not alleviate itching often.

What is Xerosis?
Dry skin or dehydration of the stratus corneum. In the elderly, decreased moisture plus the increase in sebaceous gland secretions increase the level of dryness. The result is often itchy (pruritic), rough, scaly, cracked, wrinkled skin.

What causes skin to be dark and what kind of protection does this give? What two kinds of colorations is dark skin more prone to have?
Darkness of skin is caused by melanin pigmentation. The deeper the melanin pigmentation, the more protection against skin cancer and wrinkles is given.
Dark skin is more likely to have vitiligo (hypopigmentation) and keloiding (hyperpigmentation)

**UV Radiation**

What percentage of solar radiation is UV and what are the two kinds that affect the skin the most?
UV rays account for 5% of the total solar radiation. UVB is responsible for most types of skin responses to sun exposure. UVA also affects skin in the same ways UVB will (though not as much), and will pass through glass.

What skin pigment increases in response to UV rays?
Melanin content increases in the melanocytes, which is why we tan. (And when something in the body is producing more and more, the risk for cancers increases)

Define sunburn. Include symptoms of a severe sunburn.
Sunburn is excessive exposure to UV rays, affecting the dermal and epidermal layers. Symptoms of a severe sunburn include weakness, chills, fevers, malaise, pain, blistering.

What is a drug induced photosensitivity? Name 6 classes of drugs that do this.
Photosensitivity is increased sensitivity to light. Some drugs increase the sensitivity of skin to sunlight unnaturally, increasing the risk for skin damage by UV rays. Some drugs that will do this include:
- Antihistamines (benadryl)
- Antibiotics (sulfa drugs and tetracycline)
- Antipsychotics
- Diuretics
- Hypoglycemics
- NSAIDs

What are 2 types of sunscreens, what the heck is PABA?
1. absorbent - work in the UVB range
2. reflectant – work on all rays. PABA is a reflectant

How can sunscreen be used effectively and what do the SPF numbers mean?
To be effective, sunscreen should be applied 30 min before exposure and a minimum of every 2 hours. SPF is sun protection factor, a term describing the amount of UV rays needed to produce a mild sunburn in protected versus unprotected skin. SPF 15 is probably the most bang for the buck. There is very little increased protection in numbers over 15.

Does sunscreen totally protect you from skin cancer?
No. Prolonged sun exposure, even with sunscreen, still increases risk of skin cancers.

OK, then. What combination do you have to have for the best protection?
CHESS.
- C = Clothing – sun protective clothing.
- H = Hat with wide brim
- E = eyeglasses that block UV
- S = Sunscreen, minimum of 15 SPF
- S = Shade in midday
Primary Skin Disorders

What are primary skin disorders and what classes of disorders do they include?
Primary skin disorders originate in the skin. They include:

- Pigmentation disorders (like vitiglio and albinism)
- Infectious disorders (fungal, bacterial, viral infections)
- Acne
- Rosacea
- Allergic and drug responses
- Papulosquamous (like psoriasis)
- Arthropod infestations

What is vitiglio? What causes it, when does it occur and why, and how is it treated?
Think Michael Jackson. Vitiglio affects all skin types, but is more prominent in darker skins. Vitiglio is caused by an absence of melanocytes in patches of skin. Large macular patches that sunburn easily are the most common manifestation. About half of them appear before the age of 20 for unknown reasons. It is treated with skin staining, self-tanners, cosmetics, corticosteroids, and in severe cases, with skin grafting.

Define albinism.
A recessive genetic disorder in which there are a normal number of melanocytes but they lack the enzyme (tyrosinase) which makes melanin. The result is a complete or partial absence of pigments in the skin, hair and eyes. This places persons affected at high risk for skin cancers and eye disorders.

What is melasma?
Hyperpigmented macules on the face, more common in women with darker skin. These often occur in preggers and/or with oral contraceptives. Melasma is treated with bleaching and other topical agents.

What are fungi and how do they affect the skin?
Fungi are the collective term for both molds and yeasts. Yeasts are single celled critters while molds grow in filaments called “hyphae.” When they affect the skin they can be either superficial (such as tinea which lodges in the keratinized tissues of skin and hair) or deep, going into the epidermis, dermis, and subcutaneous layers.

What causes a superficial fungal infection and where do you find this on the body? Include diagnosis and treatment
Dermatophytes such as Microsporum, Epidermophyton, and Trichophyton. These can affect just about anyplace on the body – face, neck, scalp, hands, feet, nails… Superficial fungal infections are diagnosed by skin scraping and culturing. They are treated with topical or systemic antifungals.

Tinea
What is tinea? How do you get it most commonly?
Tinea is commonly called ringworm. You can get this most commonly from other infected people, cats, dogs. The terms change, depending on where you get it on the body. Tinea corporis is on the body, tinea pedis is on the foot, tinea capitis is on the head in the hair, etc.
**What is tinea corporis? What does it look like?**
Ringworm on the body or face. It expresses as oval or circular patches on the trunk, back and extremeties with raised red (erythamatous) borders and a clearing in the center. (Also, it usually itches like a Mother F’er)

**What is tinea capitis and what 2 fun variations can it have?**
Tinea capitis is ringworm, but on the head in the hairy zones. It can be inflammatory or not. Both kinds can be scaly, pustular and cause broken hair. Neither are pretty.

The inflammatory kind has a rapid onset, usually is found in just one area and is associated with a hypersensitivity reaction. The non-inflammatory kind expresses as round hairless patches possibly mildly red, with scale.

As a note: this can also lead to secondary bacterial infections creating a painful lesion called a kerion.

**What is tinea pedis, where do you find it and what makes it worse?**
Tinea pedis is commonly called athlete’s foot and is an extremely common fungal infection usually found between the toes, soles and sides of feet. It can express as mild scale or as painful, inflamed lesions. Heat, sweating and exercise make it worse.

**What is tinea manus?**
Same thing, but on the hand.

**Define tinea unguium? Describe the manifestation and treatment in the answer.**
Untreated athlete’s foot can migrate to this, tinea infection of the nails, usually of the toenails. At the onset the nail is white, then turns yellow, brown. Over time it gets very thick and finally separates from the nailbed. Once the infection gets under the nail topical agents do little to nothing. To get rid of it systemic agents (drugs you ingest) are needed. Usually this is a 3 month course of Lamisil taken internally. Even after the fungus is gone it may take 6 months to a year for the nail to look normal again.

**What does “onychomycosis” mean?**
Onycho- has to do with nails. Mycosis is a fungal disease.

**Why are nail infections a special risk for diabetics?**
Because they get so thick and hard and difficult to trim. This leads to more ingrown toenails and more infection.

**What causes tinea versicolor, how does it express, how is it treated?**
Tinea versicolor is caused by a yeast, usually shed from the scalp, which affects upper chest, back and upper arms. It expresses as yellow, brown or pink lesions that will not tan. It is treated with topical creams and selenium sulfide shampoos.

**What is it that causes candida infections? Where do you find it, what are the manifestations and what is the landmark symptom that defines this infection?**
Candida is caused by *Candida albicans* which normally inhabits the GI tract, mouth, and vagina. Usually the infection is superficial, affecting warm moist areas like the areas where the skin folds. Large breasted women (and men too, I suppose!), people with a lot of body fat and rolls, and babies with skin folds are all susceptible as this gives plenty of environment for the candida to thrive.

The landmark symptom that defines a candida skin infection is the erythematous (red) areas in susceptible areas which have distinct borders and satellite lesions close to the main infection. Candida itches and burns and basically drives you to either drink or go get something to kill it!

**Discuss impetigo**

Impetigo is caused by group A streptococcus or staphylococcus. Kids get it most commonly in the summer on their faces, noses, lips. The **hallmark sign of impetigo is a vesicle or pustule which ruptures leaving a honey colored crust.**

**What is the viral cause of verruca and what is verruca in English?**

Verruca is another way to say “wart.” Warts are caused by the human papilloma virus (there about 50 different kinds, so don’t panic) and are common around the age of puberty. Verrucae (the plural) are often found on hands and feet (where they are called plantar’s warts).

They are treated topically with salicylic acid and cryotherapy – the wart virus sits very deeply below the surface of the wart so the idea is to erode the surface and get to the virus. You can also cover them with duct tape of all things in a form of occlusive therapy.

**Herpes**

**What are the two types of herpes viruses and what areas do they affect?**

HSV-1 is usually oral, causing vesicles on lips, face, mouth and nose. HSV-1 may express with a fever or sore throat if it become systemic. It can be triggered by stress, infection, and injury.

HSV-2 is usually genital. It is important to note that the virus can shed from the infected person to others even when the patient is asymptomatic.

Treatment for HSV is usually oral antivirals, though there is no cure.

**What is herpes zoster? How is it treated?**

Herpes Zoster is caused by a reactivation of the varicella-zoster virus (chicken pox). Incidence of Herpes zoster increases with age as T-cell mediated immunity declines.

The virus lives in the nerves, travels to skin from the nerve ganglia and expresses in a dermatomal pattern. (Remember the dermatome map from A and P). Symptoms include a burning pain, tingling, sensitivity, and itching. This is followed by an eruption of vesicles with an erythematous (red) base for about 3-5 days which then crust and dry over 2-6 weeks.

Herpes zoster is treated with anti-viral agents and vaccinations to prevent outbreak.

**What are the severe complications of herpes zoster?**

1. Eye involvement which can cause blindness
2. Post herpetic neuralgial pain which persists after the rash.
Acne

What is the classic bacteria that is involved in infected acne?
Propionibacterium acnes

Define acne.
A disorder of the pilosebaceous (pilo = hair, sebaceous = sebaceous gland) unit where the hair follicles get clogged with sebum. Acne can express with whiteheads of black heads in the non-inflammatory versions or as cystic acne with pustules and pus-filled nodules.

What causes increased sebaceous activity?
Sebaceous glands are sensitive to hormonal stimulus, especially to androgens which increase sebaceous activity.

What does the term “cystic acne” mean?
The development of pustules or suppurative (pus filled) nodules.

What makes acne worse and how do you treat it?
Stress, pre-period week, anything that rubs on skin and oil based cosmetics can all make it worse. Diet hasn’t been proven to worsen it. Acne is treated:

- topical kerolytics (lyses keratin to keep pores clean)
- antibacterial agents
- systemic antibiotics
- oral contraception
- Accutane, an oral retinoid.

What is rosacea: include cause, manifestations, complications if not controlled, and triggers.
Rosacea is a chronic inflammatory disorder of unknown cause affecting mostly fair skinned middle aged and older adults. It expresses as repeat blushing and then erythema of the nose, cheeks, forehead and/or chin. It may have telangiectasia (broken blood vessels on the skin -- spider veins).

If uncontrolled, rosacea may develop into rhinophyma, an irregular hyperplasia of the nose. Triggers include heat, sunlight, hot or spicy foods and liquids, and alcohol.

What is contact dermatitis, what causes it and what helps with diagnosis?
Contact dermatitis is an inflammation of the skin with may have erythema, edema, and/or vesicles. It is caused by skin contact with an irritant such as wool or cleaning products or by allergens (dyes, perfumes, adhesive, nickel, latex, cheap-ass jewelry, etc.). The location and distribution of the symptoms can help with diagnosis.

What is eczema, how is it different in infants vs adults, how is it treated?
Eczema or atopic dermatitis is a hypersensitive allergic reaction often associated with asthma. Infants often express with vesicles and crusting while adults get dry, leathery hypo or hyperpigmented skin, often in patches. Treatment includes bathing in warm water and following immediately with moisturizer and/or topical corticosteroids.

Define urticaria, including cause and treatment.
Urticaria is also known as hives, raised edematous plaques that itch intensely. Urticaria can be chronic or acute and the cause is unknown. Most patients who have urticaria have elevated IgG antibodies to an
IgE molecule with elevated histamine levels. As a result of the histamine level, treatment is with antihistamines.

**What is psoriasis? Include expression, prevalence, what makes it worse, how to treat.**
Psoriasis is a t-cell mediated autoimmune disease characterized by red, thick plaques which get thicker over time and that are covered with with a silver-white scale (from hyperkeratosis). The prevalence of psoriasis increases with age and there is a hereditary factor to it.

Stress, infection, trauma, xerosis (dry skin) and meds can either cause it or make an existing case worse. Psoriasis is treated with steroids, UV light treatments, and/or emollients to soften the scale and let the topical drugs in.

**Pityriasis Rosea. What is this, what’s the cause, what’s the tip-off symptom?**
Pityriasis Rosea is an outbreak of oval macular or papular rashes with surrounding erythema. The initial lesion and the thing that tips you off to this disorder is called a “herald patch” and appears on the neck or trunk. After the herald patch lesions appear in a “christmas tree” pattern following the skin fold lines. The cause is unknown and it resolves spontaneously in 6-8 weeks.

**Define Lichen Planus**
Lichen planus is an inflammatory eruption which pruritic and papular in nature. The papules are purple with a shiny lacy kind of pattern to them. There is a form called “lichen simplex chronicus” which occurs from repeat scratching.

**What on earth are scabies?**
Scabies refers to an infection by mites in the skin, commonly between fingers, at the wrist, inner elbow, and axilla. They burrow into the skin and lay eggs that hatch in 3-4 days. This cause reddish brown lesions which can blister. It also causes pruritus and excoriation (scratching away the top layer of skin basically). Scabies can live on sheets and in clothing causing reinfection unless you get rid of the pest. Ew.

**Skin –oma’s**

**What is a nevus and a dysplastic nevus?**
A nevus is a mole that can be raised or flat, pigmented or not, hairy or not. Borders on a normal nevus are symmetrical as is the coloring. Dysplastic nevi are flat, slightly raised with irregular borders and vary from one part to another as to the coloring. Dysplastic nevi are cause for concern as they can transform into malignancy.

**What is the ABCD rule?**
A convenient way of determining whether or not a skin manifestation is possibly malignant or premalignant.
A = Asymmetry
B = Border irregularity
C = Color variation
D = Diameter – anything larger than 6mm (about the size of a pencil eraser) should be checked out. Check moles/nevi for changes. Any changes in these areas is cause for concern.
Define malignant melanoma – include expression/manifestation, who is at risk, etc.
Malignant melanoma is a malignant tumor of the melanocytes which are deep in the skin and have more access blood flow which means they can progress rapidly and metastasize. They often express as slightly raised nevi which are brown or black in color with irregular borders and uneven surfaces. They can occur anywhere on the body, but are more likely in areas of sun exposure.

Those most at risk are fair-skinned blonde or red haired individuals, freckled people, those who sunburn easily and/or who have had several severe sunburns in their past. Early diagnosis and treatment substantially increase the survival rate for this disease.

Define basal cell carcinoma
A basal cell carcinoma is a neoplasm (new growth) of non-keratinized cells on the basal layer of the epidermis. This is the most common form of skin cancer among white-skinned people but the good news is that it is usually non-metastasizing. It often starts flat then becomes red, flakey, and may have a waxy, pearly, rolled border.

Define squamous cell carcinoma
This is the 2nd most common form of skin cancer. It is characterized as a red, scaling, keratotic elevated lesion with an irregular border that ulcerates and crusts in the center. It often bleeds and won’t heal. Metastasis is more common in squamous cell carcinomas than in basal cell types. Sun exposure, arsenic, tar, coal, and paraffin all increase the risk for squamous cell carcinoma.