Contents

1 Arthritis

- Contrast osteoarthritis with rheumatoid arthritis? If both rheumatoid arthritis and osteoarthritis can lead to end-stage destruction of the joint(and both can) why is it important to differentiate between the two?
- Given the typical (degenerative) etiology of osteoarthritis, how then might a 20 year old person have this condition?
- What is the role of body mass vis a vis osteoarthritis? Why might we think that this not be a pure mechanical phenomenon? (hint: Framingham)
- What are the cardinal signs of osteoarthritis of the knee on plain radiographs? How (mechanistically) do they appear?
- Why is osteoarthritis painful?

2 Osteoporosis and metabolic bone disease

- Describe the process of bone remodeling?
- What are the three tasks of bone? How can problems related to the two non-structural tasks lead to fracture?
- Define and contrast osteoporosis and osteomalacia?
- How is osteoporosis diagnosed, prevented and treated?
- Describe the relationship between menopause and hip fracture risk. Describe the relationship between body mass and hip fracture risk.
- What are the three fractures typically associated with osteoporosis? Which is worst? Why is it so bad?
- Heaney wrote, "*Although bone mass is certainly the most extensively studied of the fragility factors, low bone mass is not the whole of the osteoporosis story and may not even be its most important component (despite frequent assertions to the contrary). If one could magically normalize bone mass in everyone, would one eliminate osteoporotic fractures? The best answer that can be given today is 'no.' There would be fewer such fractures, but there would still be many, especially hip fractures.*" (Heaney RP Bone Mass, Bone Loss, and Osteoporosis Prophylaxis. Annals Internal Medicine 15 February 1998 128: 313-314). Explain what Heaney meant. That is, What else besides intrinsic bone problems could cause hip fracture?
- Mr. Smith, a 72 year old with hip pain, had x-rays ordered by his PCP that showed lytic lesions concerning for bone cancer. The radiology report, however, read the films as “Paget’s Disease” to which Mr. Smith exclaimed with delight, “It’s not cancer! That’s great news! What is Pagets disease of the bone? And why might that diagnosis not be "great news"?

3 Trauma

- Describe the differences between primary and secondary bone healing?
- What are the necessary conditions for appropriate bone healing (leading to minimal functional residuals) and how may physicians optimize the chances for healing?
- Plating a fracture clearly disrupts the soft tissue envelope around a fracture. Why, then, is surgical plating ever used?
- How can a femoral shaft fracture be lethal?
- What is compartment syndrome and how is it prevented, diagnosed, and treated? What are the consequences of not treating a compartment syndrome and over-treating a suspected compartment syndrome?
- Define joint dislocation subluxation and reduction
- Why is a traumatic hip dislocation typically worse than a shoulder dislocation? Contrast the mechanisms which prevent the normal shoulder from dislocating with those of the hip joint, and consider what structures must be damaged when the joint comes out of place.
- What is osteonecrosis? (also known as “avascular necrosis” or “AVN”) How does hip dislocation lead to avascular necrosis? How does avascular necrosis lead to end stage arthritis?
- Besides osteonecrosis, what other mechanisms may enable a dislocation to cause arthritis?
- A patient falls on his outstretched hand and has normal appear xrays but tenderness in the “anatomic snuff box” (between extensor pollicis longus and abductor pollicis longus and extensor pollicis brevis). Why might such a patient be placed in a cast despite the normal x-ray?

4 Sports Injuries

- What is a stress fracture? How is a stress fracture treated in a normal person? What are the consequences of a stress fracture which is not treated? Why might a young woman with an eating disorder be at particular risk for stress fracture?
- What is the definitional distinction between grade I, II and III sprains? How would these various grades of injury present distinctly on examination?
- What is the function of the Anterior Cruciate Ligament (ACL) in the knee? How is the ACL torn? Along those lines, why might it be the case (as we suspect) that skiingrelated ACL tears occur disproportionately after 2pm? How is an ACL tear detected on exam?
- Why is it that a tear of the ACL typically requires surgical replacement whereas a grade III sprain tear of the medial collateral ligament of the knee can heal with immobilization and rehab?
- What is the function of the meniscus in the knee? What is the consequence of tearing a meniscus? Why are most symptomatic meniscal tears removed and not repaired?
- What is rotator cuff tendinitis? What are the consequences of labeling it (perhaps incorrectly) as an “---itis"? What are the consequences of labeling rotator cuff tendinitis as “impingement syndrome”?

5 Spine

- Back pain is a common, self-limited condition in many people. Discitis, cancer and cauda equine syndrome are causes of back pain which are not innocent and self-limiting. First, by way of background, define "cauda equina syndrome", discitis and the common cancers are found in the spine. Next, name some questions a physician might ask to help detect the diagnoses?

- A so-called herniated disc may compress a nerve root and cause radicular complaints/findings. What are the classic motor and sensory findings of L4 L5 and S1 compression? Why, given that an MRI can localize the disc herniation, if present, should/must a student know the motor and sensory findings of each nerve root level? Suggest a medical rationale for not obtaining an MRI in a patient with low back pain and no neurological features. Forget about the cost of the MRI itself: why might a physician considering only the best *medical* interests of his or her patient choose to omit an MRI?

- What is neurogenic claudication (and contrast with vascular claudication)?

- What is idiopathic scoliosis and what is its relationship to back pain?

6 Carpal Tunnel Syndrome

- Provide a brief description of Carpal Tunnel Syndrome (including complaints, findings and treatment options)
- Cervical radiculopathy is certainly on the differential diagnosis in a patient with suspected Carpal Tunnel Syndrome. Think of some other entries on that list as well. What complaints would make you think that a patient has one of these, and not Carpal Tunnel Syndrome?
- Tinnel's sign is not a true sign---in what way? Why might that be significant?

7 Infection

- What is septic arthritis? What are its causes?
- How is septic arthritis diagnosed definitively? What are the temporal limitations regarding our ability to diagnose definitively? How do we get around that?
- How is septic arthritis treated? Why must septic arthritis be treated expeditiously?
- Both gout and septic arthritis can cause acute pain and swelling of a joint. How can they be distinguished?
- What is osteomyelitis? What are its causes? Although bone is part of the vascular system (and therefore fractures can cause hemorrhage and metastatic cells can lodge in the skeleton) why might IV antibiotics have trouble reaching areas of infected bone?

8 Pediatric Hip conditions

- What is Developmental Dysplasia of the Hip? How is Developmental Dysplasia of the Hip diagnosed in the neonate and why is it critical to detect this, if present, as soon as possible?
- What is a Slipped Capital Femoral Epiphysis?
- What is Perthes Disease?
- Why might Developmental Dysplasia of the Hip, a Slipped Capital Femoral Epiphysis and Perthes Disease cause arthritis of the adult hip? (ie, later in life). Why might successfully treated Developmental Dysplasia of the Hip have the best prognosis whereas untreated Developmental Dysplasia of the Hip might present the biggest treatment (surgical reconstruction) challenges?

9 Disorders of the foot and ankle

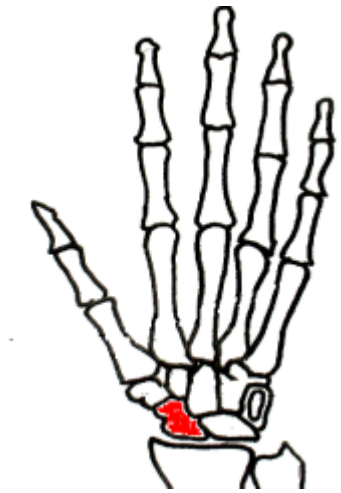
- What is Club foot?
- What is a Charcot joint?
- Why might a 12 year old boy with a swollen ankle and pain on the distal fibula be treated with a cast despite normal xrays?
- Why do 40 year old men rupture their Achilles tendons? What are the biological and mechanical steps leading to tissue failure?
- The phrase "just a sprain" may grossly understate the impairment such an injury imparts. Why might a grade I ankle sprain cause long term impairment such an injury imparts?

10 A patient falls on his outstretched hand and has normal appear xrays

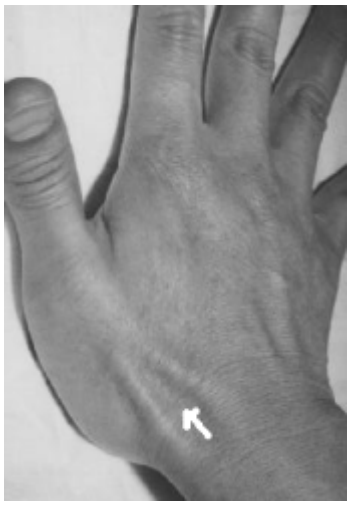
A patient falls on his outstretched hand and has normal appear xrays but tenderness in the "anatomic snuff box" (between extensor pollicis longus and abductor pollicis longus/extensor pollicis brevis).

Why might such a patient be placed in a cast despite the normal x-ray?

Two anatomy pictures to keep in mind: first, the scaphoid bone, shown in red



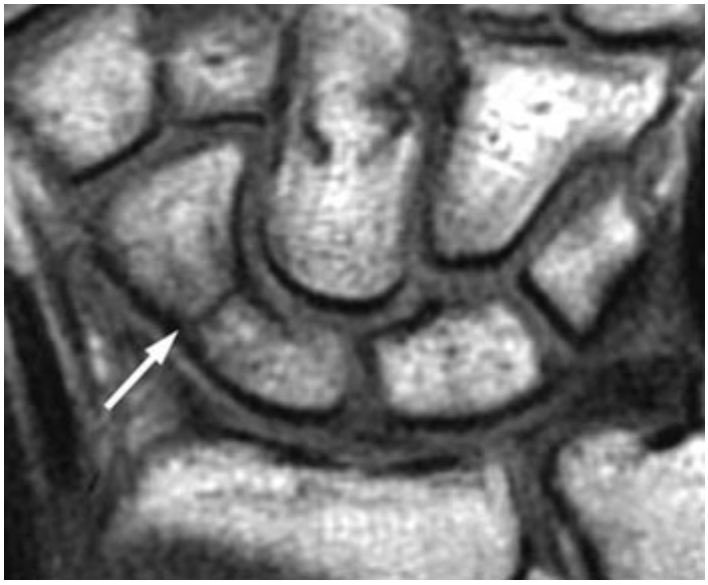
next, the anatomic snuff box denoted by the arrow on the right---the point on the surface at which the scaphoid is palpated.



A fall on an outstretched hand can fracture the scaphoid bone.

Yet (probably owing to the scaphoid's geometry and location), a non-displaced fracture of this bone may not be discernable on a plain x-ray.

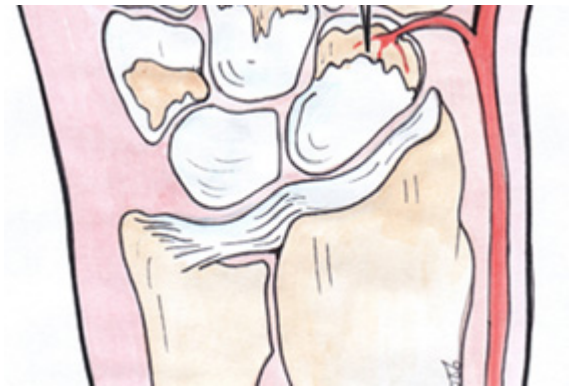
(Below is the MRI of a patient with a normal x-ray)



A patient with a fall and snuff box tenderness may have a non-displaced fracture.

In the event of a suspected scaphoid injury, it is imperative to prevent displacement of the fracture and disruption of the blood supply.

Blood supply to the scaphoid bone is tenuous as most of the scaphoid surface is cartilage--this leaves only a small area for arterial blood supply to enter the bone from the dorsal carpal branch of the radial artery and feed the bone in retrograde fashion.



If the fracture displaces, the blood supply may be interrupted and avascular necrosis of the scaphoid may result.

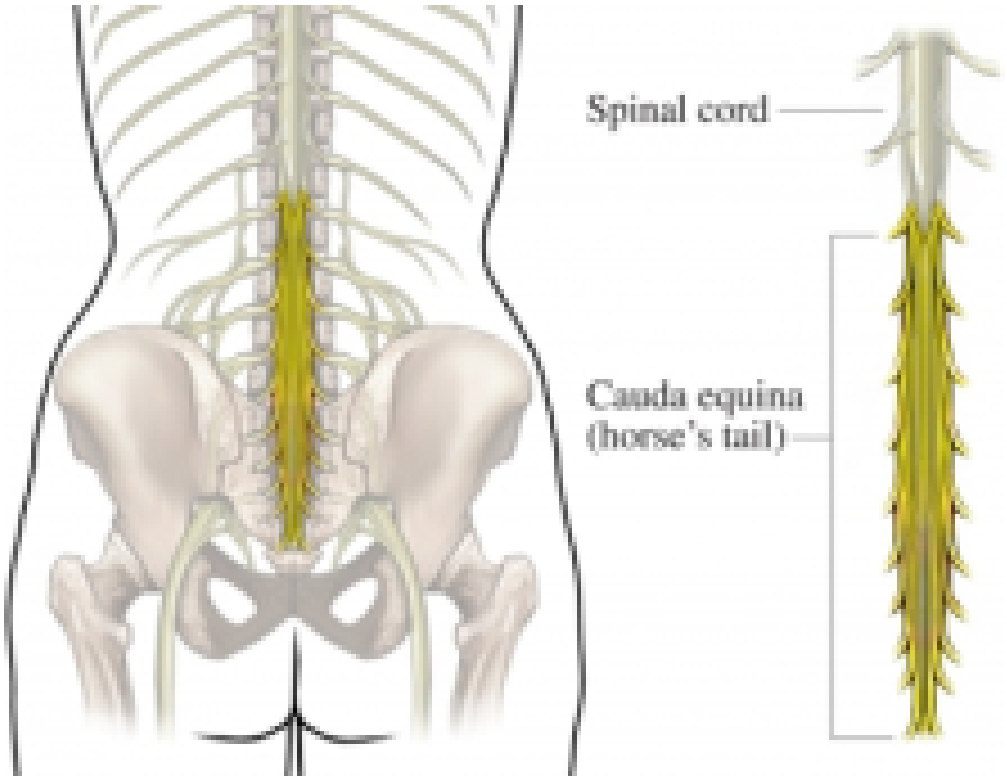
Preventative casting (to prevent fracture displacement) and follow up radiograph imaging is the standard of care following a scaphoid injury. (In the alternative, an MRI may be used to exclude a non-displaced fracture as shown above. This indeed may be cheaper in the long run, to say nothing of kinder: an unnecessary cast is a burden).

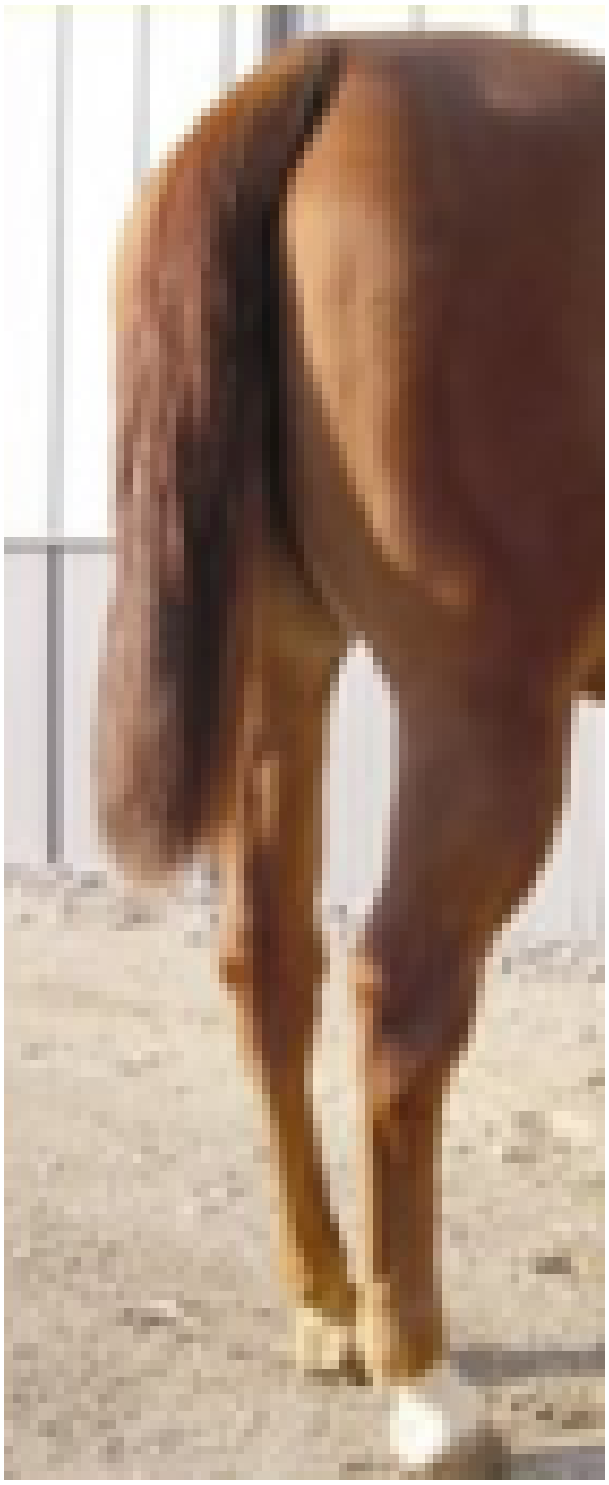
Penn Med Self Study Questions | Back pain

11 Back pain

Back pain is a common, self-limited condition in many people. Discitis, cancer and cauda equina syndrome are causes of back pain which are not innocent and self-limiting. First, by way of background, define "cauda equina syndrome", discitis and the common cancers that are found in the spine. Suggest some questions a physician might ask to help detect these diagnoses.

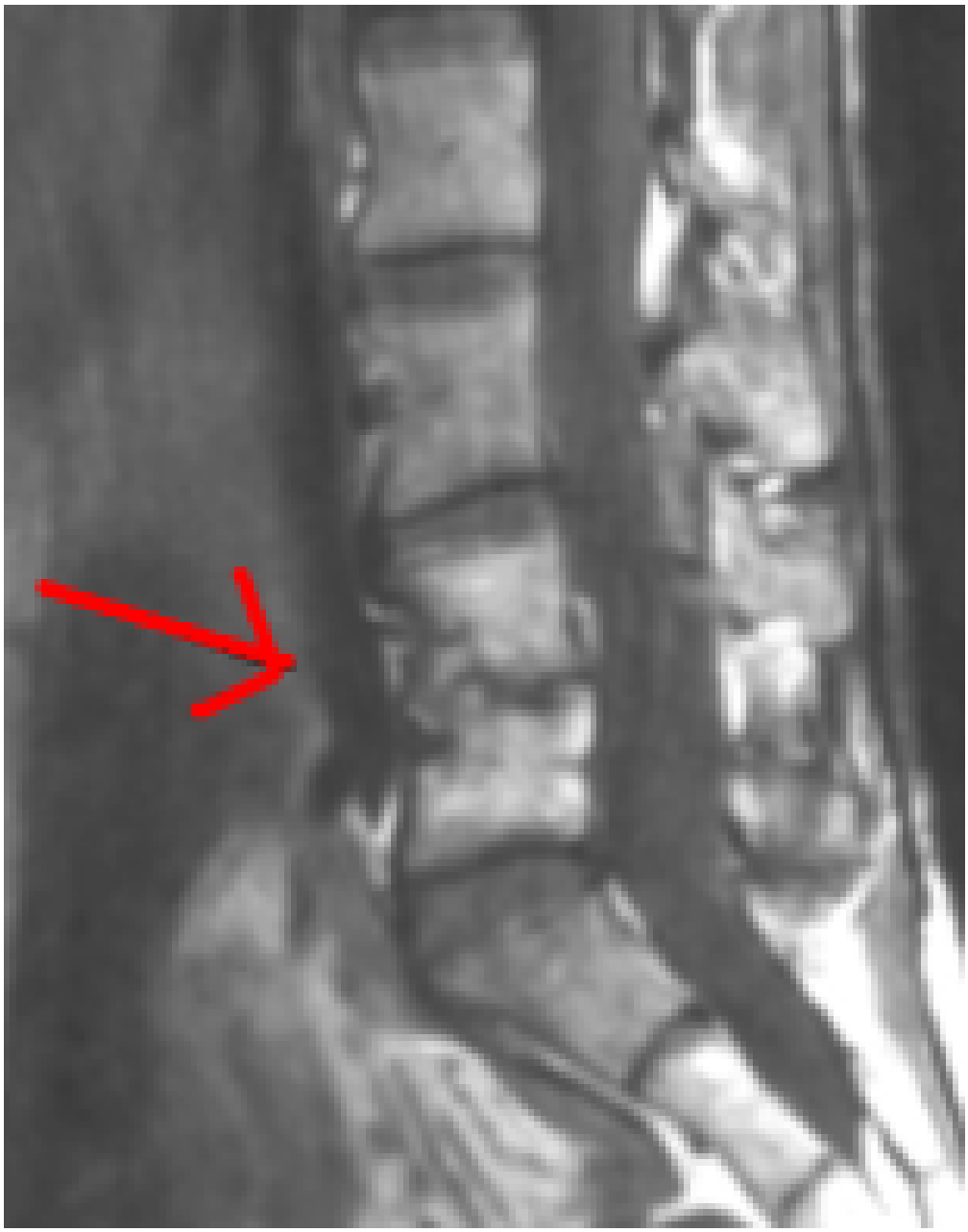
Cauda Equina Syndrome is a serious neurologic condition in which there is acute loss of function of the lumbar plexus. The "cauda equina" is made up of the nerve roots that branch off of the lower end of the spinal canal beneath the termination of the spinal cord (conus). The cauda equina contain the nerve roots from L1-L5 and S1-S5. The nerve roots branching off the spinal canal beneath the termination of the spinal cord is said to resemble a horse's tail, hence the name, but I am not sure. You decide:





Cauda Equina syndrome occurs when the nerves of the cauda equina are compressed by a herniated disc material, tumor, or bone (trauma). Cauda Equina Syndrome presents with weakness of the muscles innervated by the compressed roots, sphincter weaknesses causing urinary retention, and post-void residual incontinence. There may also be decreased anal tone; sexual dysfunction; saddle anesthesia; bilateral leg pain and weakness; and bilateral absence of ankle reflexes. Pain may, however, be entirely absent. The diagnosis is usually confirmed by an MRI scan or CT scan. Treatment typically involves URGENT surgical decompression.

Discitis is an infection in the intervertebral disc space.



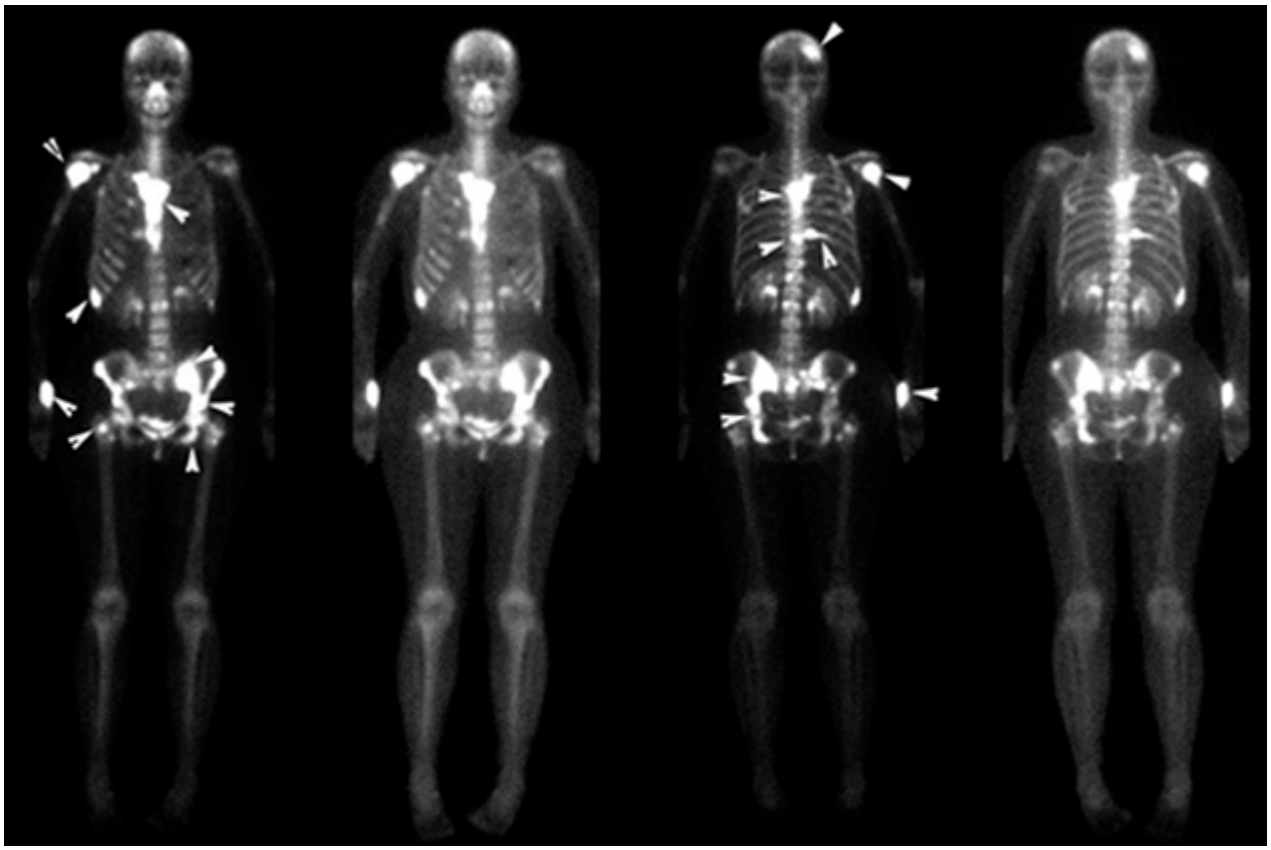
Pathogens can reach the bones of the spine by hematogenous spread from a distant site or focus of infection, direct inoculation from trauma or spinal surgery, or contiguous spread from adjacent soft tissue infection. Staphylococcus Aureus accounts for more than half of cases in developed countries. Symptoms include severe back or neck pain, which often lead to immobility. Fevers have been noted in some patients. The diagnosis is usually confirmed by an MRI scan. Treatment usually includes antibiotics and using a back brace to reduce the mobility of the region.

Primary spinal cancer can happen, but is relatively rare. Metastatic disease is more likely. Spinal metastasis is the third most common site for cancer metastases (behind lung and liver). And note: the patient may not know s/he has a primary tumor!

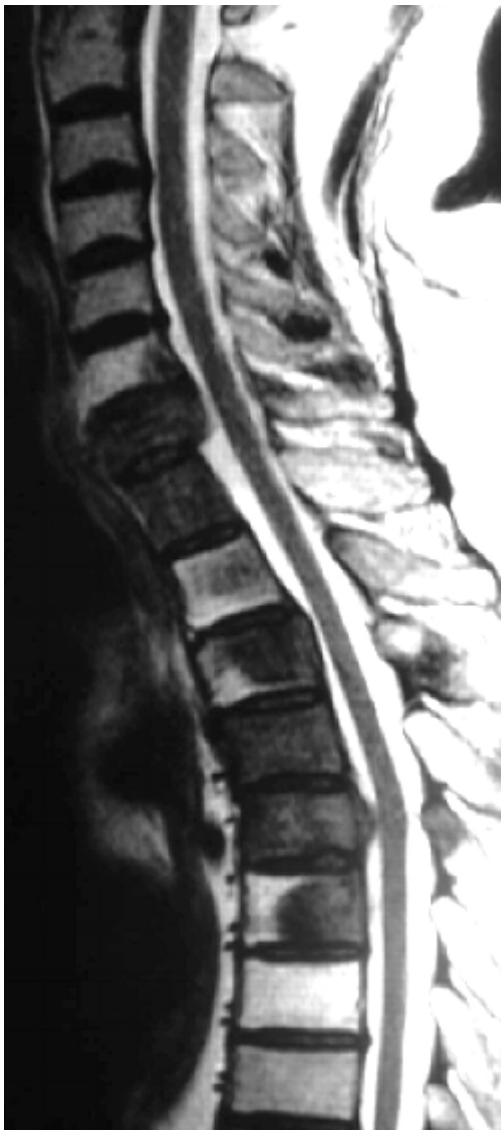
The most common cancers that spread to the spine are breast, lung, thyroid, kidney and prostate.

(mnemonic: **BLT** with a **K**osher **P**ickle)

Shown below is a bone scan with breast cancer metastasis in the spine (and elsewhere)



prostate cancer spread to the spine is shown:



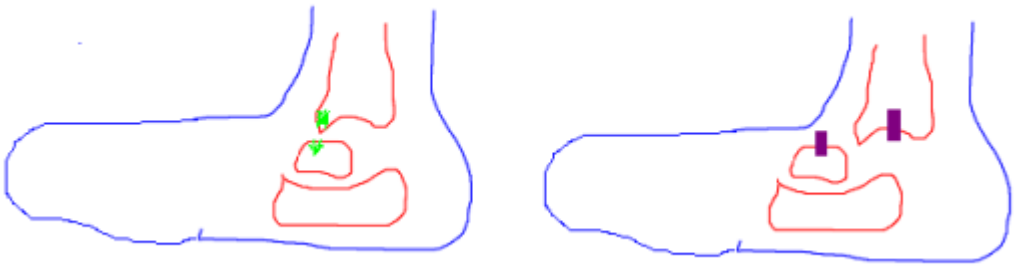
The way to approach back pain, conceptually, is to recall that mechanical back pain (pain after too much activity) is usually benign and self limited; by contrast, visceral pain is much more worrisome. So the first questions should include asking about the timing (and inciting factors) of the pain. Also, inquiring about constitutional symptoms—general wellness, weight loss, fevers, sweat etc—and neurological deficits is helpful.

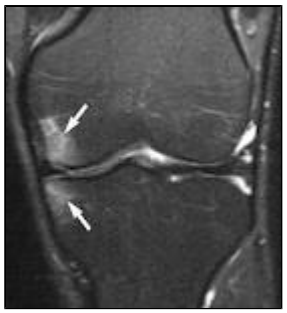
To hone in on the three diagnoses above it may be helpful to concentrate on the risk factors for the conditions. A compromised immune is obviously a risk factor for infection. A risk of primary cancer (eg smoking) is a risk for metastatic disease.

⇦	A patient falls on his outstretched hand and has normal appear xrays	⇧ Penn Med Self Study Questions	Besides osteonecrosis, what other mechanisms may enable a dislocation to cause arthritis	⇨

12 Besides osteonecrosis, what other mechanisms may enable a dislocation to cause arthritis

Besides osteonecrosis, what other mechanisms may enable a dislocation to cause arthritis?
Recall this picture: dislocation can be associated with an impaction injury to the joint surface, and torn or stretched ligaments





The MRI of the knee above shows a so called "kissing contusion"---the two high signal areas were impacted against each other. Although the MRI does not show the articular surface very well, we can infer from the appearance of the bone that there was also an impaction injury to the cartilage, with possible irreparable damage inflicted.
Further, if the ligaments heal loosely, if at all, the joint may be unstable, and an unstable joint may be subject to repetitive "micro-trauma", as the talus (in the example above) rattles around in the joint. (This is the same mechanism by which a loose lug nut on your car may cause your tire to wear out prematurely.) Because of instability the forces in the joint are not applied across a broad area, but rather focally. And because P=F/A, a smaller area for a given force results in higher pressure.

Back pain | Penn Med Self Study Questions | Both gout and septic arthritis can cause acute pain and swelling of a joint

13 Both gout and septic arthritis can cause acute pain and swelling of a joint

Symptoms of both gout and septic arthritis include redness and swelling present in just one joint and pain that is worse with movement.

The key distinction is the results of a culture of the joint fluid obtained by aspiration: a positive culture defines an infection.

One could consider the history (patients with known gout are of course more likely to have gout as the cause, as compared to patients without that history; patients with immune suppression are of course at higher risk for infection).

One could also measure the concentration of white blood cells in the aspirate: the number of cells is higher in cases of infection. The problem is that there is no cut off that perfectly segregates the two.

One can also examine the aspirate under the microscope: crystals suggest gout, and bacteria indicate infection. Yet crystals can be seen in cases of infection as well (where both conditions are present simultaneously).

Because culture results are not known immediately, all of the information listed above should be considered, especially when urgent treatment is needed.

	Besides osteonecrosis, what other mechanisms may enable a dislocation to cause arthritis	Penn Med Self Study Questions	Cervical radiculopathy is certainly on the differential diagnosis	

14 Cervical radiculopathy is certainly on the differential diagnosis

The differential diagnosis for carpal tunnel syndrome includes:

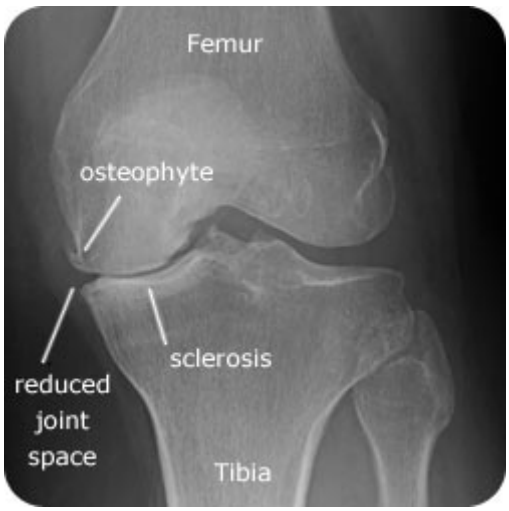
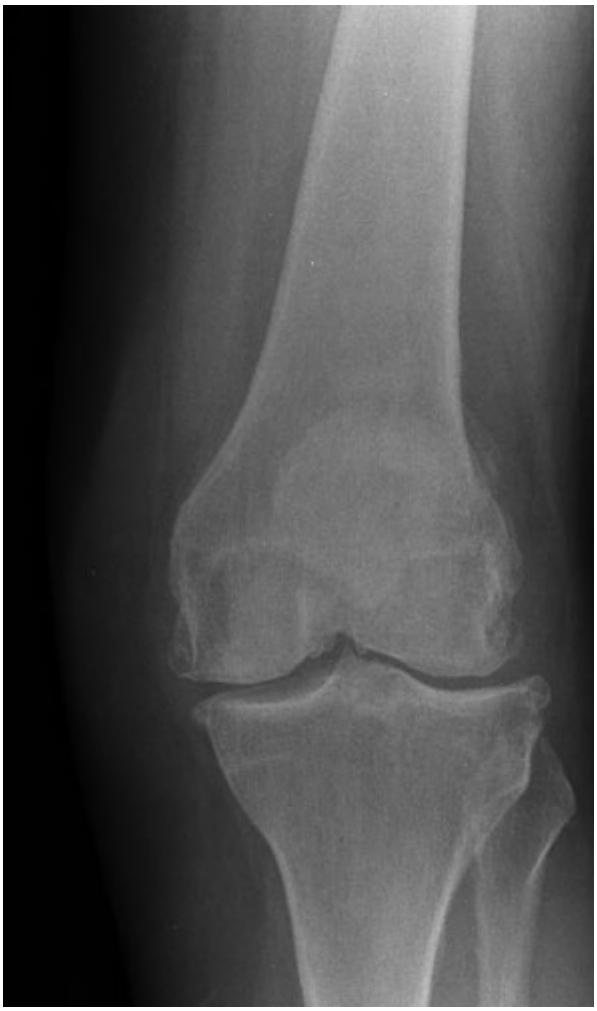
- C6 or C7 radiculopathy. (With radiculopathy expect to see neck pain and pain proximal to the wrist)
- Osteoarthritis. This may exist concurrently occur with CTS, but would see tenderness and crepitus on exam (and EMG would also be normal unless CTS co-exists)
- Ulnar neuropathy Just like CTS but wrong nerve! The sensory symptoms are on the medial aspect of the hand, distal forearm and 4^{th} and 5^{th} fingers (instead of radial 3 ½ finders). No thumb weakness.
- De Quervain tenosynovitis: Extensor tendinitis of the thumb. With this, tend to see pain with movement of thumb along with tenderness near the radial styloid. EMG is normal (unless CTS co-exists)

Both gout and septic arthritis can cause acute pain and swelling of a joint | Penn Med Self Study Questions | Contrast osteoarthritis with rheumatoid arthritis

15 Contrast osteoarthritis with rheumatoid arthritis

Contrast osteoarthritis with rheumatoid arthritis.

	Osteoarthritis	**Rheumatoid arthritis**
Cause	Deterioration of cartilage and overgrowth of bone often due to "wear and tear".	Autoimmune inflammation of the synovial membra to the destruction of the articular cartilage.
Joints	More often affects the larger weight-bearing joints, such as the hips and knees.	More often affects the smaller joints of the hands,
Complaints	Worse pain at the end of the day, when wear and tear builds up.	The stiffness is worse after rest, such as the first tl morning, and often lasts at least 30 minutes or mo
Systemic?	NO	Can be a systemic disease.
Gender?	NONE	Rheumatoid arthritis is three times more common males
Labs?	Lab tests normal	80% of patients are sero-positive (ie positive rheur

X-rays	Bone spurs, sclerosis common. Asymmetric joint space narrowing (where the wearing and tearing took place)  	Osteopenia more common. Symmetric joint space 
Exam:	Effusion, tenderness	Effusion, tenderness but redness/warmth more co

If both rheumatoid arthritis and osteoarthritis can lead to end-stage destruction of the joint -and both can-why is it important to differentiate between the two?
If you believe, as I do and you should, that the doctor has three jobs (identify diagnosis, state prognosis and offer treatment) the RA/OA difference holds three important distinctions:

- Because RA is a systemic disorder than can affect many organs of the body; therefore, it is important to check for, manage, and prevent other manifestations of the disease---ie, DIAGNOSE EXTRA-ARTICULAR disease
- RA has a different prognosis, in terms of overall health as well as joint specific.
- The diagnosis affects treatment: treatment for RA is not just symptomatic, and it also targets the root cause of the disease: immune abnormalities. This is accomplished with biologics and disease-modifying anti-rheumatic drugs (DMARDs) and Biologics. The goal is not just palliation but to STOP DISEASE PROGRESSION!

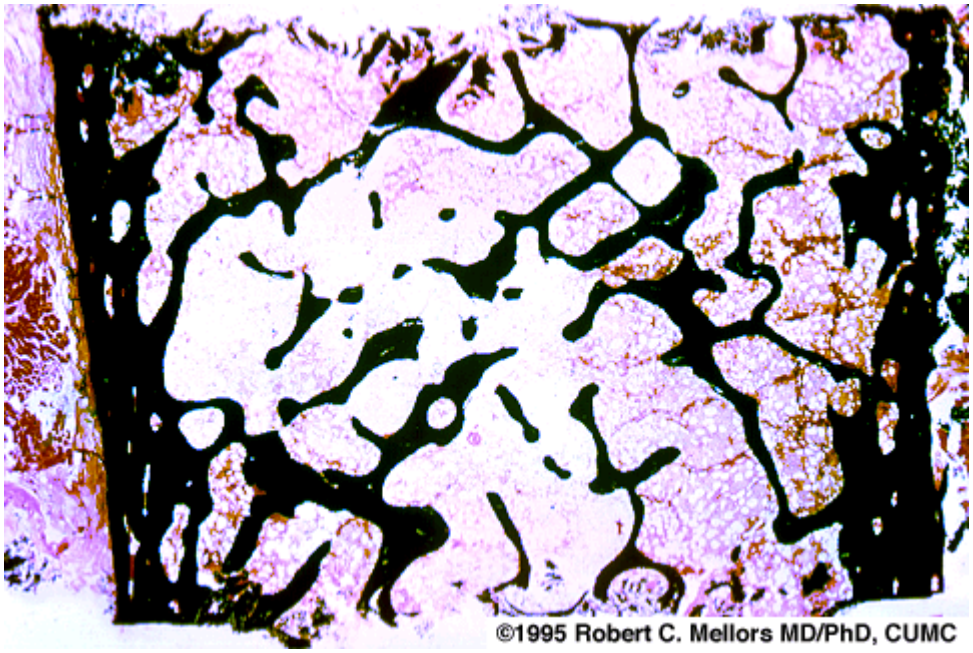
Cervical radiculopathy is certainly on the differential diagnosis | Penn Med Self Study Questions | Define and contrast osteoporosis and osteomalacia

16 Define and contrast osteoporosis and osteomalacia

Define and contrast osteoporosis and osteomalacia.

Both osteoporosis and osteomalacia can cause weak bones.

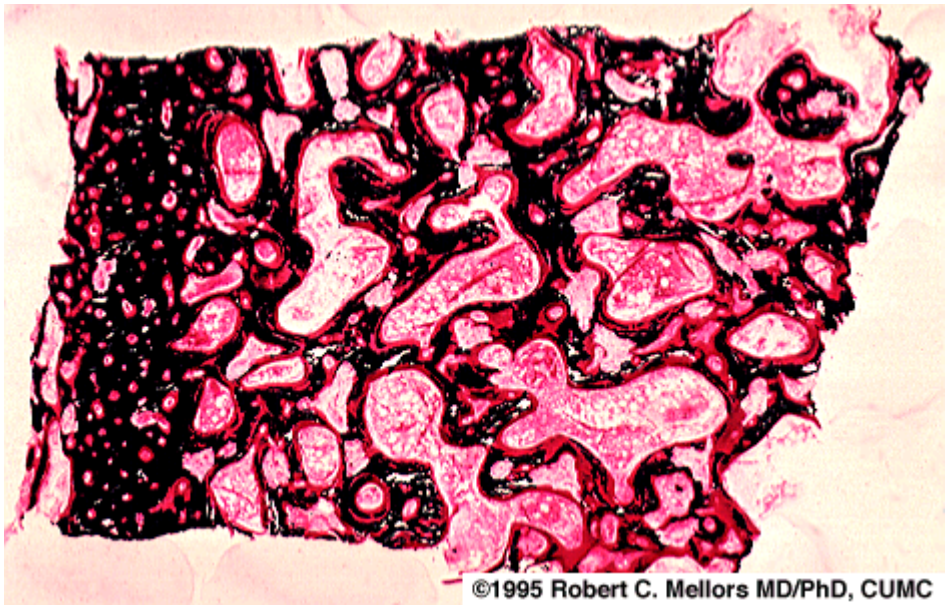
In osteoporosis, there is decreased bone mass with a normal ratio of mineral to matrix.



In this iliac crest biopsy, above, osteoporosis is inferred by the small amount of bone (seen in black)

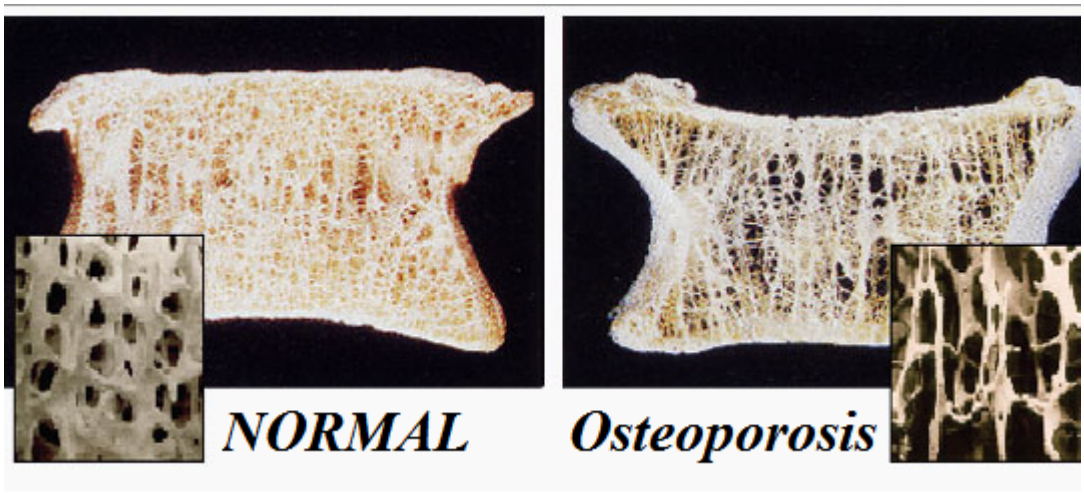
In osteomalacia, the ratio of mineral to matrix is decreased (ie there is too much matrix relative to the amount of bone)

In this iliac crest biopsy, below, osteomalacia is seen. there is lots of OSTEOID (matrix, red) vs bone (again seen in black)

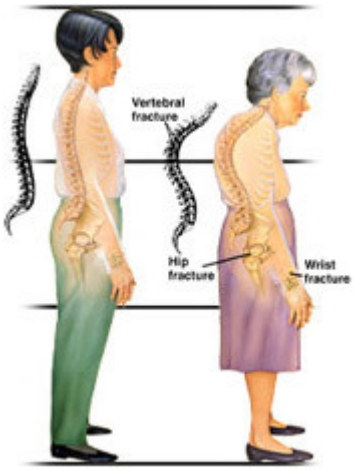


Osteoporosis causes decreased bone mass, with a normal ratio of bone mineral to matrix in addition to altered bone microarchitecture. The catch-phrase of osteoporosis is **"normal-enough bone, but not enough of it!"**

Clinical features of osteoporosis include fractures from minimal trauma, particularly in the thoracic and lumbar spine, wrist and hip.



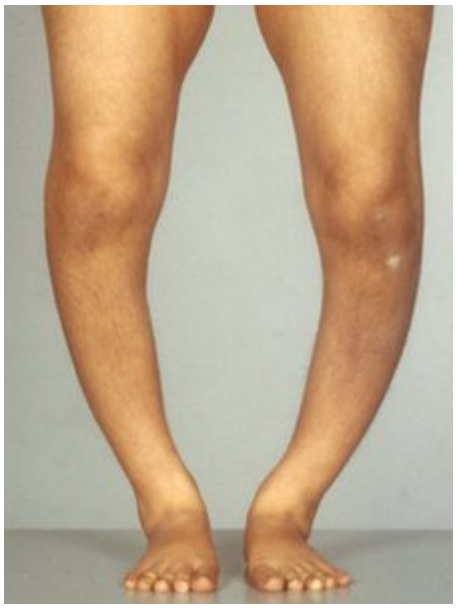
Thoracic vertebral compression fractures can cause dorsal kyphosis (Dowager's hump).



Plain x-rays show decreased bone density; but only once at least 30% of bone is lost. Dual-energy x-ray absorptimoetry (DEXA) is the diagnostic test for osteoporosis; it reports bone density in terms of T scores, representing deviations from the mean of normal individuals). A DEXA > 2.5 is diagnostic of osteoporosis. Lab values of serum calcium, phosphorus and alakaline phosphatase are not diagnostic.

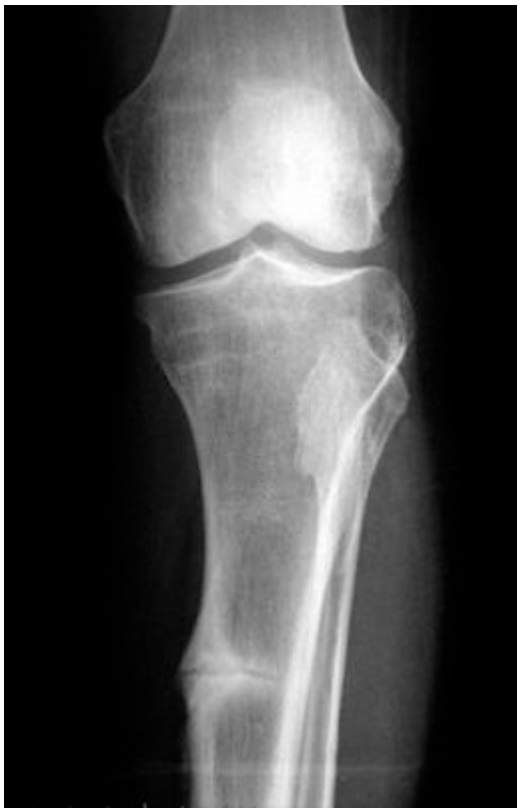
Osteomalacia is characterized by a decreased ratio of bone mineral to matrix.

Osteomalacia when it appears in children is called rickets", seen below



Histologically, the un-mineralized osteoid appears as a thickened layer of matrix. The disease causes characteristic symptoms of diffuse bone pain, tenderness and muscle weakness. X-rays commonly show decreased bone density with thinning of the cortex. Advanced disease can cause concavity of vertebral bodies (codfish vertebrae) and bowed legs.

In addition, fissures/cracks (so-call Looser's zones) may appear. These are incomplete fractures, as shown below, are filled with the un-mineralized osteoid seams.



Lab findings may show low serum and urinary calcium and high serum alkaline phosphate.

Contrast osteoarthritis with rheumatoid arthritis	Penn Med Self Study Questions	Define joint dislocation subluxation and reduction	

17 Define joint dislocation subluxation and reduction

Joint dislocation (also known as luxation) occurs when the joint surfaces become completely disengaged. A dislocation always damages the ligaments.

Joint subluxation is an incomplete/partial dislocation of a joint.

Joint Reduction is the process by which a structure is brought back into its normal anatomic position. (This term also applies to the bones in the case of fracture.)

Reduction can be spontaneous---ie, the joint simply "pops back into place". This process of spontaneous reduction, as you might imagine, is more common in joints that are not particularly stable, such as the patello-femoral joint. (Makes sense: easy out; easy in.)

Consider these drawings of the lateral view of the ankle, showing, left to right, **normal** alignment; a tibio-talar **subluxation** and then a tibio-talar **dislocation**:

And now think about the structural damage: with a dislocation, there could be a bone contusion where there is abnormal contact (shown below as the green "kissing contusion"); and the ligaments can either rip (as shown) or get really stretched.

(A subluxation can cause a bone contusion or stretching of the ligaments; a full tear is less likely.)

⇦	Define and contrast osteoporosis and osteomalacia	Penn Med Self Study Questions	Describe the differences between primary and secondary bone healing	⇨

18 Describe the differences between primary and secondary bone healing

escribe the differences between primary and secondary bone healing .

The two mechanisms of bone healing are primary bone healing and secondary bone healing. (THIS IS JUST LIKE SKIN: you can sew it up or it can scab.)

Primary bone healing involves a direct attempt by the cortex to re-establish itself after interruption without the formation of a fracture callus.

Just like in skin, primary healing only works when the edges are touching exactly. And since touching edges are not common, primary healing is the less commonly seen type of healing.

In fact, this method is employed only after rigid surgical fixation, or in case with a partial crack in the bone, a so-callled "unicortical" fracture, where the remaining bone holds everything rigid.

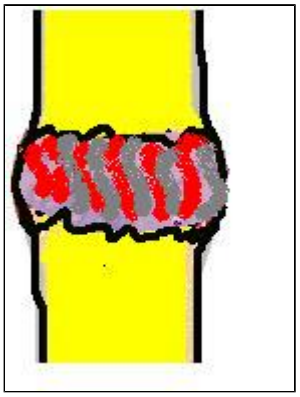
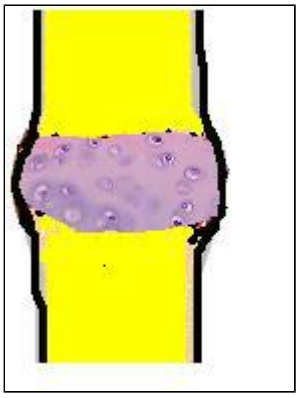
The basic science, in brief: Primary bone healing is lead by the formation of a so-called cutting cone (consisting of osteoclasts at the front of the cone to remove bone and trailing osteoblasts to lay down new bone) across the gaps to form a secondary osteon.

Secondary bone healing involves the classical stages of injury, hemorrhage inflammation, primary soft callus formation, callus mineralization, and callus remodeling. This method of bone healing closely resembles endochondral ossification (which involves a cartilage template being replaced by bone).

This suggests that indirect bone healing results in re-formation of bone with essentially the same mechanical properties as the original bone, if not better.

Key point: healing in a sense recapitulates growth. So fracture healing can lead to completely new bone, not scar. (At the other extreme: cartilage: it heals poorly---it not only forms just scar, the scar is poor mechanical quality. Scientists among you: fix this!)

18.1 SCHEMATIC: SECONDARY BONE HEALING

18.1.1 1. Bone breaks	
18.1.2 2. Hematoma (blood clot) forms at once.	
18.1.3 3. From this hematoma, a primary callus forms **18.1.4 This is composed of granulation tissue** **18.1.5 (fibroblasts and new blood vessels).**	
18.1.6 4. The cells in this soft callus make cartilage .	

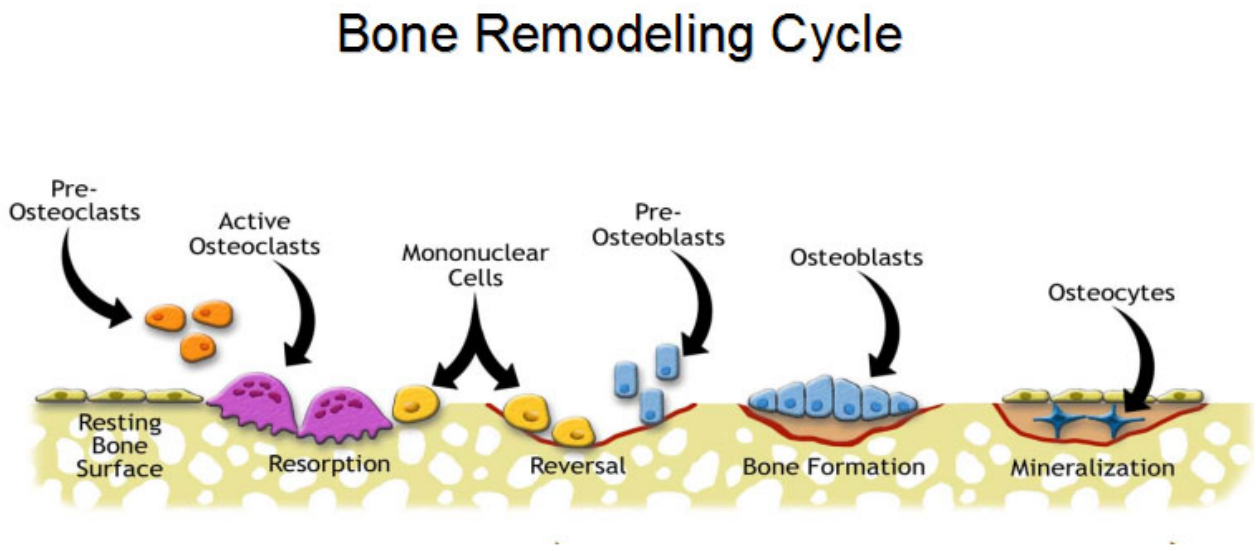
18.1.7 5. The cartilage is then mineralized , **18.1.8 producing "woven" or "lamellar"** **18.1.9 ie disorganized, bone.**	**18.1.10**
18.1.11 5. Last, the woven bone remodels **18.1.12 into normal bone (oriented in direction of load)**	

Define joint dislocation subluxation and reduction | Penn Med Self Study Questions | Describe the process of bone remodeling

19 Describe the process of bone remodeling

Describe the process of bone remodeling. Why does this process exist?

Bone remodeling, in brief, is the process by which osteoclasts eat old bone and stimulate osteoblasts to make new bone.



The activity of osteo**b**lasts is easy to comprehend: make bone where needed. Osteo**c**lasts are bit trickier: why resorb bone?

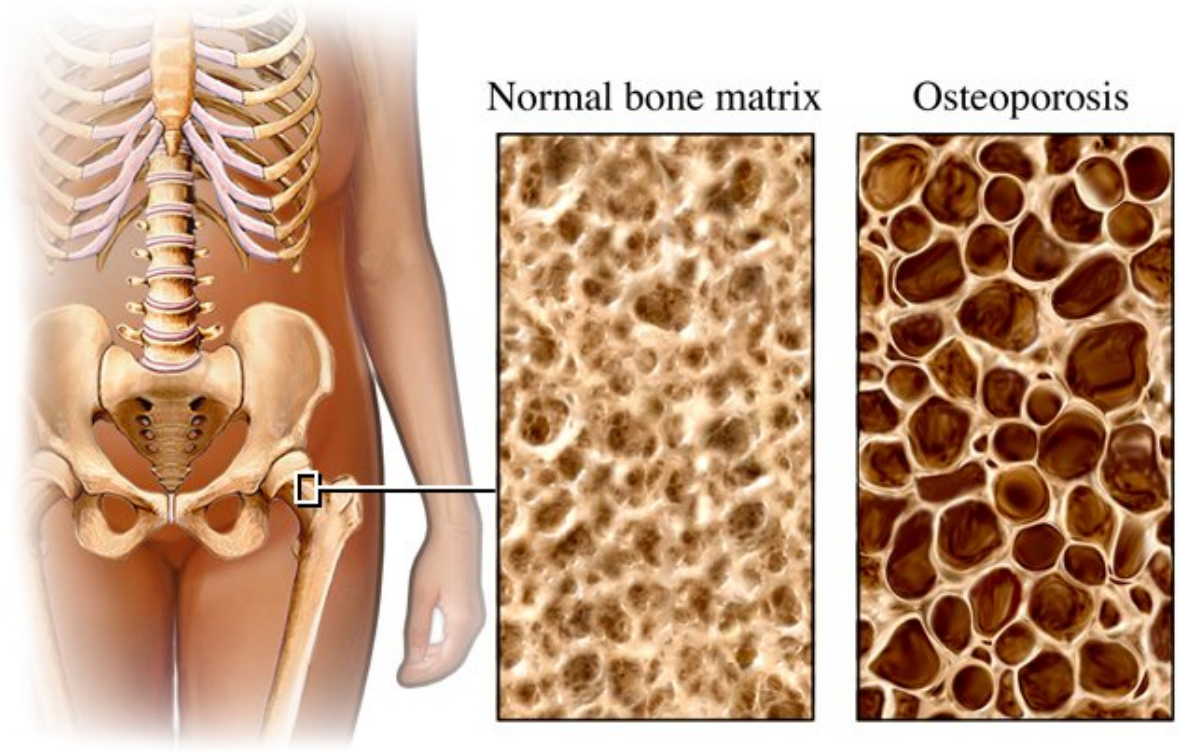
The process of resorption exists for two reasons:

- first, to liberate calcium and other ions; and
- second, to clear out worn out pieces of the skeleton and promote the deposition of newer, better material.

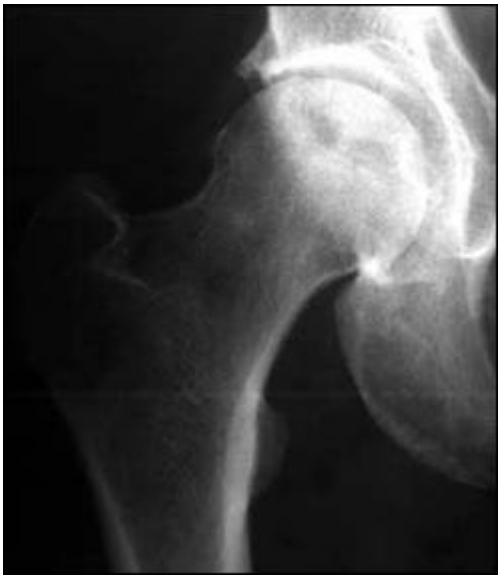
Osteoclastic resorption occurs by secretion of acid and proteolytic enzymes which digest the bone matrix; Ca^{2+} and PO_4^{3-} are then taken up by the osteoclasts and released into the circulation.

Bone formation occurs by osteoblasts secreting an organic matrix (osteoid) and then mineralizing the matrix.

- When the remodeling process is skewed such that, over time, there is more eating than replenishing, you get osteoporosis.

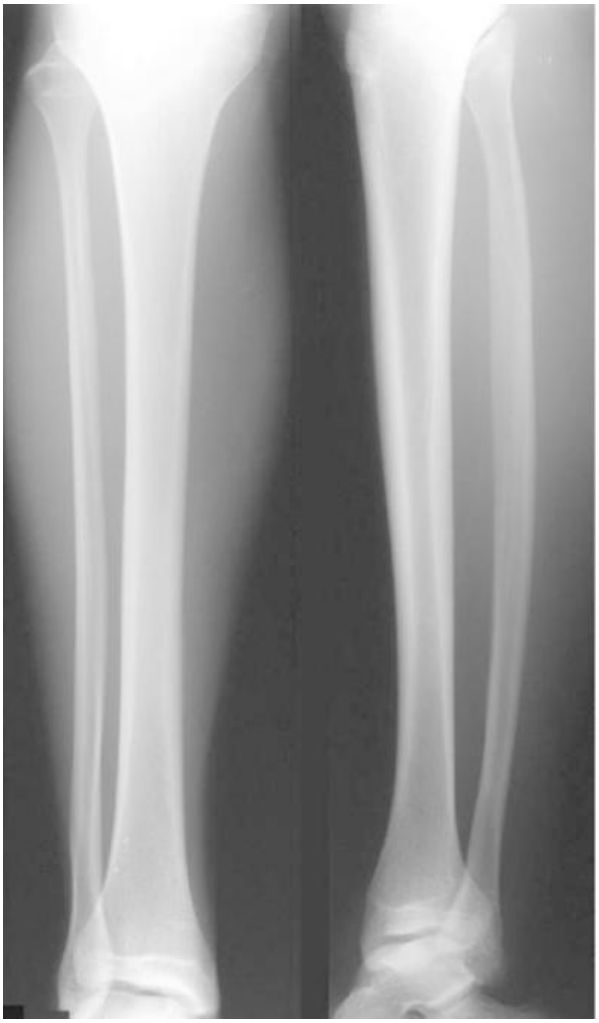


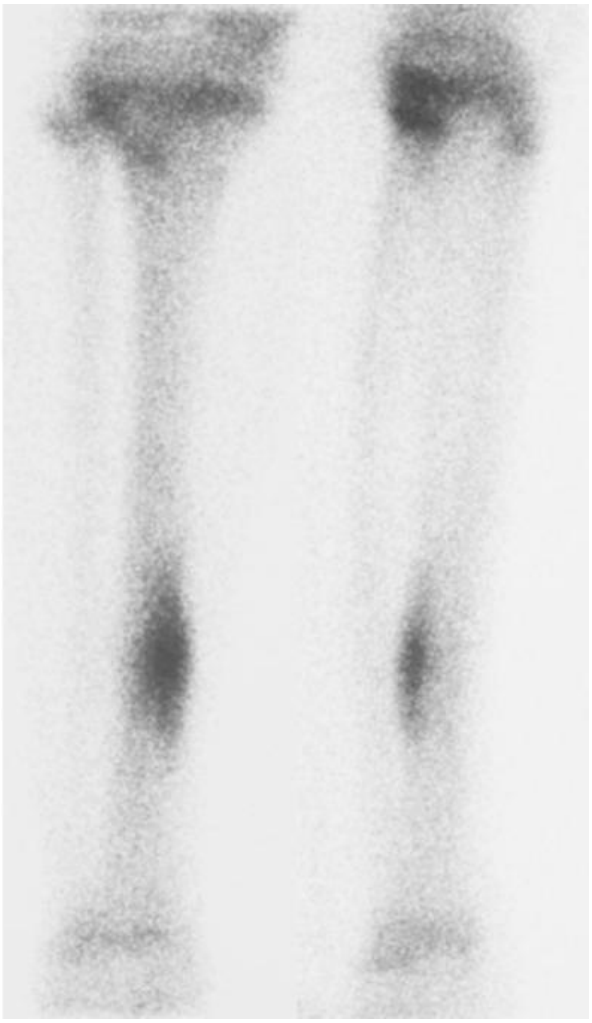
- When the remodeling process is aborted, say in avascular necrosis,



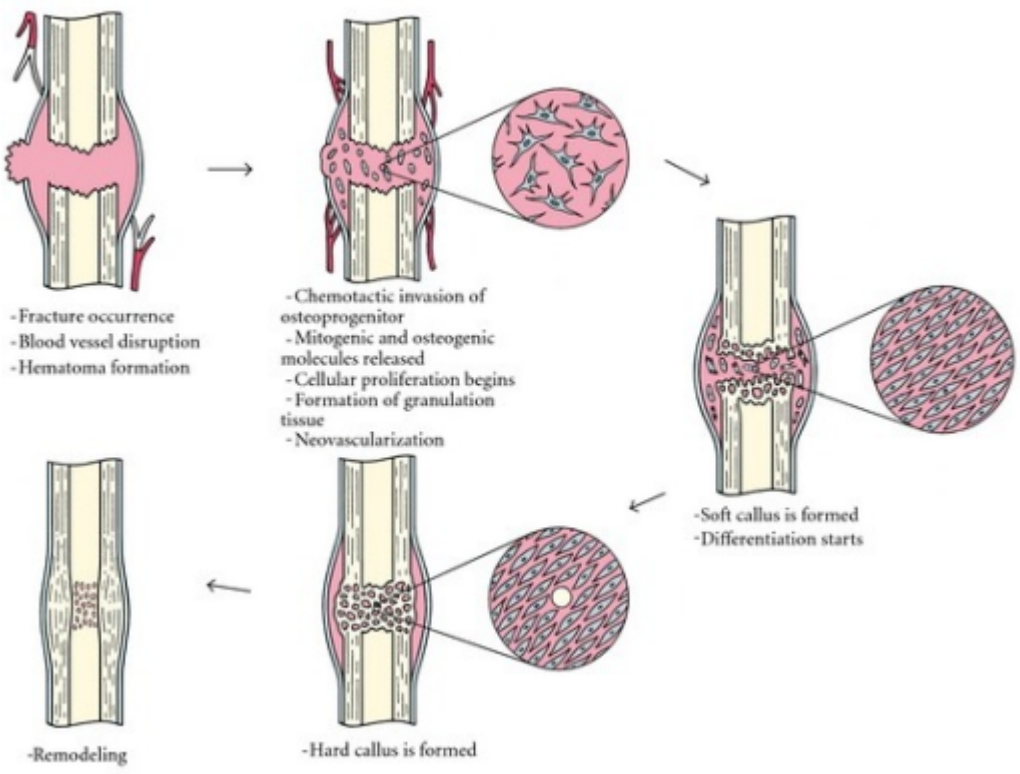
bad bone accumulates. (Seen here as density). This can lead to collapse and failure of the subchondral ("under the cartilage") bone thus can lead to arthritis.

- When the remodeling process just can't keep up with (new) mechanical demands, like over-exercising, you get a stress fracture
  as shown on the bone scan to the right (xrays do not detect this)





- When you get a long bone fracture, bone remodeling kicks in to literally remodel the callus and lay down new bone (not scar). This is the final step of the fracture healing cascade, shown to the lower left



- And to be sure, when the bone needs to liberate calcium and other ions, it employs osteoclasts and invokes the process of bone remodeling; as such the invocation of the bone remodeling program is a key feature of metabolic bone disease (such as hyperparathyroidism).

Bone resorption occurs from osteoclastic breakdown of trabecular bone via the secretion of hydrolytic enzymes. This process occurs throughout life and is tightly regulated by several factors: serum vitamin D, serum calcium, growth hormone, PTH (increase resorption), and calcitonin (increase bone formation) levels, to name a few.

Two things to recall:

1. you cannot "de-mineralize" the bone as you would in this party trick. . You have to "de-bone" the bone, as Dr Fred Kaplan termed it: you cannot simply dissolve out some ions when you need them and replenish the bone later; you must break down the matrix to get the mineral out. Thus, even if the body needs calcium 'only for a minute', it takes a while get the skeleton restored. Think of it as having to get a home equity load if you wanted to borrow even a small amount; it's a much bigger hassle than a credit card overdraft! Implication: if you have a state of high mineral flux, there will be lots of immature bone, as every instance of mineral withdrawal necessitated some 'de-boning'.
2. Metabolic needs trump skeletal needs. This makes sense: calcium is needed for cardiac contractility and nerve transmission. Thus, in metabolic diseases or states of nutritional deficiency, the skeletal system can be harmed.

Describe the differences between primary and secondary bone healing | Penn Med Self Study Questions | Describe the relationship between menopause and hip fracture risk

20 Describe the relationship between menopause and hip fracture risk

Describe the relationship between menopause and hip fracture risk. Describe the relationship between body mass and hip fracture risk.

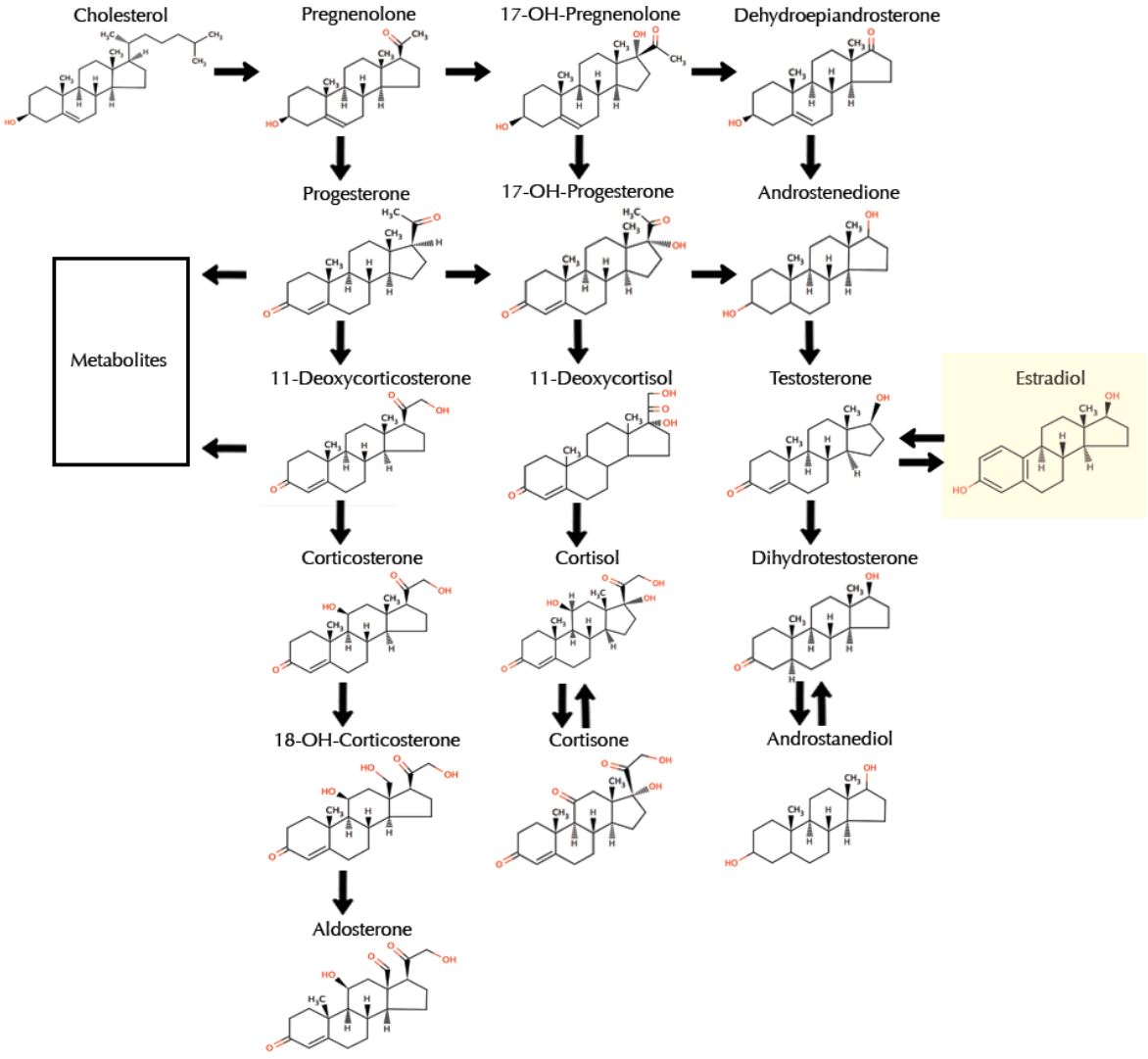
We know with certainty that hip fracture incidence is higher after menopause. The precise mechanism –or rather, the relative contribution of known mechanisms—is debated. (The interested student is pointed to Bernstein, J., Grisso, J.A. and Kaplan, F.S. Body Mass and Fracture Risk. Clinical Orthopedics and Related Research. 364:227-230 1999).

Menopause, with its decreased estrogen production, leads to increased osteoclast activity. Thus, at menopause, women begin to experience a 2% loss in bone mass per year as bone resorption outpaces bone formation.

A low BMI is a risk factor for hip fracture. A BMI of 20 is estimated to have a 2.0 relative risk of hip fracture compared to an individual with a BMI of 25. (Note that a really low BMI is best thought of as cachexia –and that's a sign of general decline, bones included.)

There are two schools of thought why low BMI leads to fracture, both centered on the role of fat.

1. Fat is a substrate for the synthesis of estrogen and thus is an indirect osteoclast inhibitor. More fat = more estrogen = more bone.



(don't memorize this until you start cramming for step I)

2. Fat provides soft tissue padding. The energy absorbed by the bone in a fall is equal to the energy generated by the fall, minus the energy absorbed by other tissues. Hence, More fat = more energy absorbed by soft tissues = less energy absorbed by bone = lower risk of fracture.

What is the practical distinction between the two? Basically, it's the question of whether low energy fractures are an intrinsic bone problem or an extrinsic, medical problem and in turn, whether the best plan to decrease fractures is to optimize the bone or minimize the energy to which the bone is exposed at the time of falling.

	Describe the process of bone remodeling	Penn Med Self Study Questions	Given the typical (degenerative) etiology of osteoarthritis, how then might a 20 year old person have this condition	

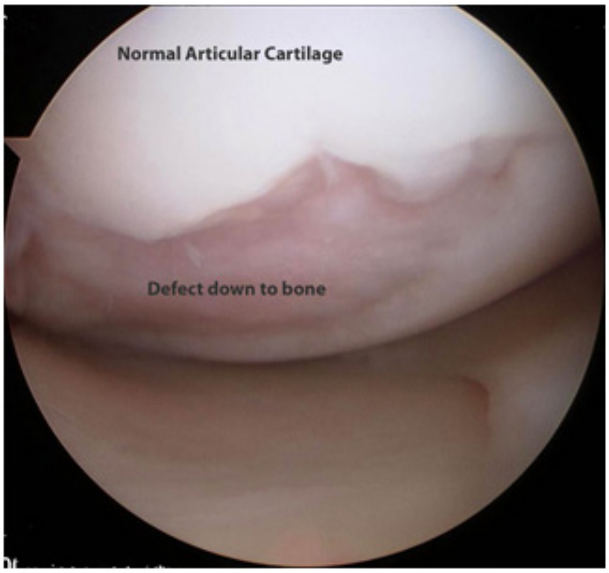
21 Given the typical (degenerative) etiology of osteoarthritis, how then might a 20 year old person have this condition

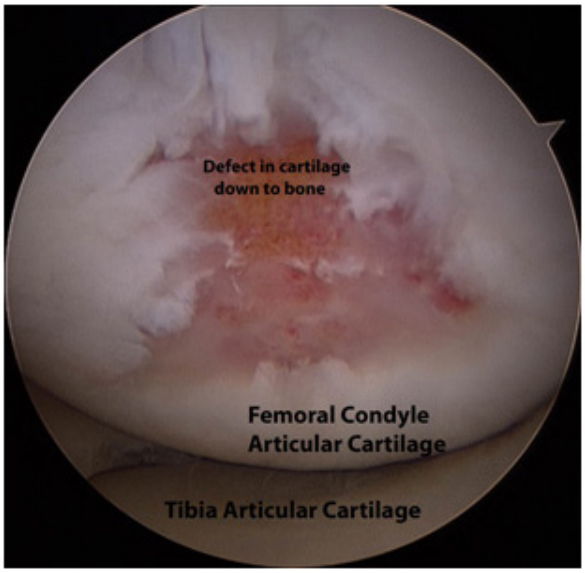
Given the typical (degenerative) etiology of osteoarthritis, how then might a 20 year old person have this condition?

The most likely cause of osteoarthritis in a young person is trauma.

Trauma, in turn, can cause arthritis in 3 ways (at least)

1. DIRECT DAMAGE Damage to the articular surface directly can of course lead to breakdown.

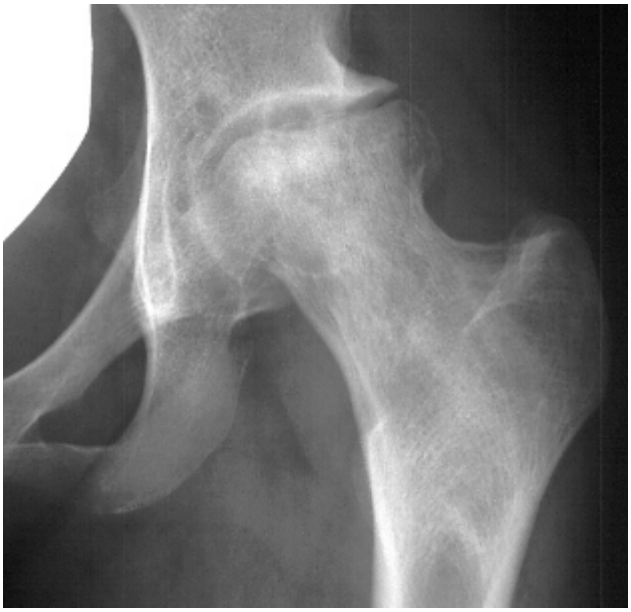




2. Chronic damage inflicted from loose ligaments. In the figure below, you see that (on the left) if the tire is not secured, there will be focal loading on parts of the tire (and tire damage accordingly). Similarly, a loose joint can have eccentric loading on small areas, and in those areas there will be increased pressure (again, recalling P=F/A). This pressure leads to breakdown

3. Trauma can disrupt the blood supply, and the arthritis may be from AVN

   As shown here, dead bone can collapse leading to deformity of the joint surface



The take home point is that DJD in a young person is possible, but not likely, and should be a diagnosis of exclusion.

Describe the relationship between menopause and hip fracture risk | Penn Med Self Study Questions | How can a femoral shaft fracture be lethal

22 How can a femoral shaft fracture be lethal

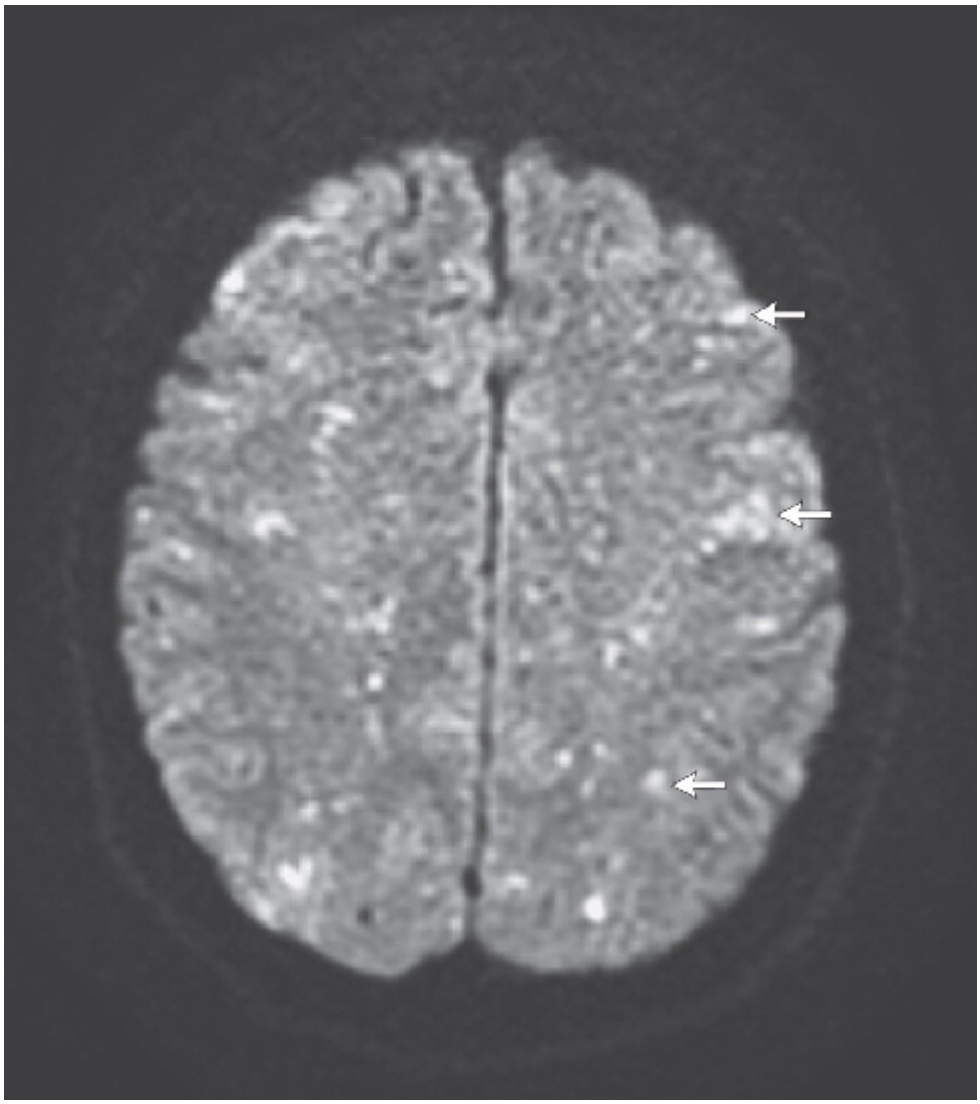
How can a femoral shaft fracture be lethal?

A femoral shaft fracture in isolation should not cause death. Yet a patient with a femoral fracture can die from this injury.

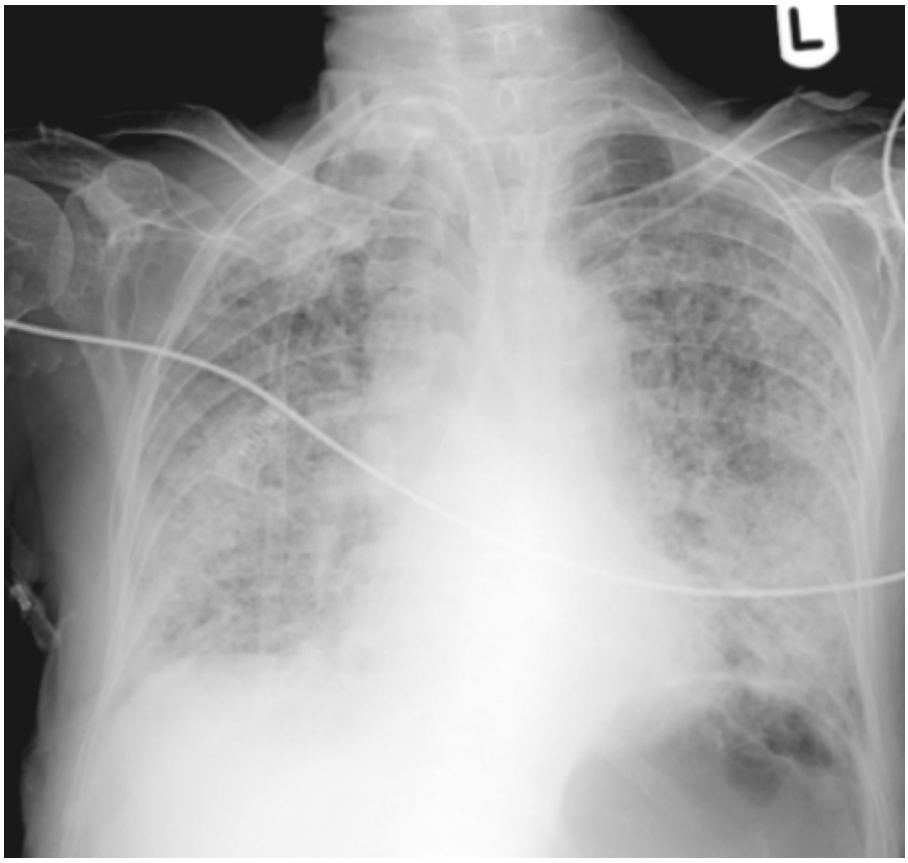
Recall that bone is vascular and fractures let marrow contents (fat especially) out into the circulation.Fat could embolize to the brain or the lungs.

This photo of Pork Neck Bone Soup with Lotus Root with glistening fat droplets on the surface will remind you of this more than an MRI of a brain showing a fat embolism

MRI of a brain showing a fat embolism–see, wasn't I right??

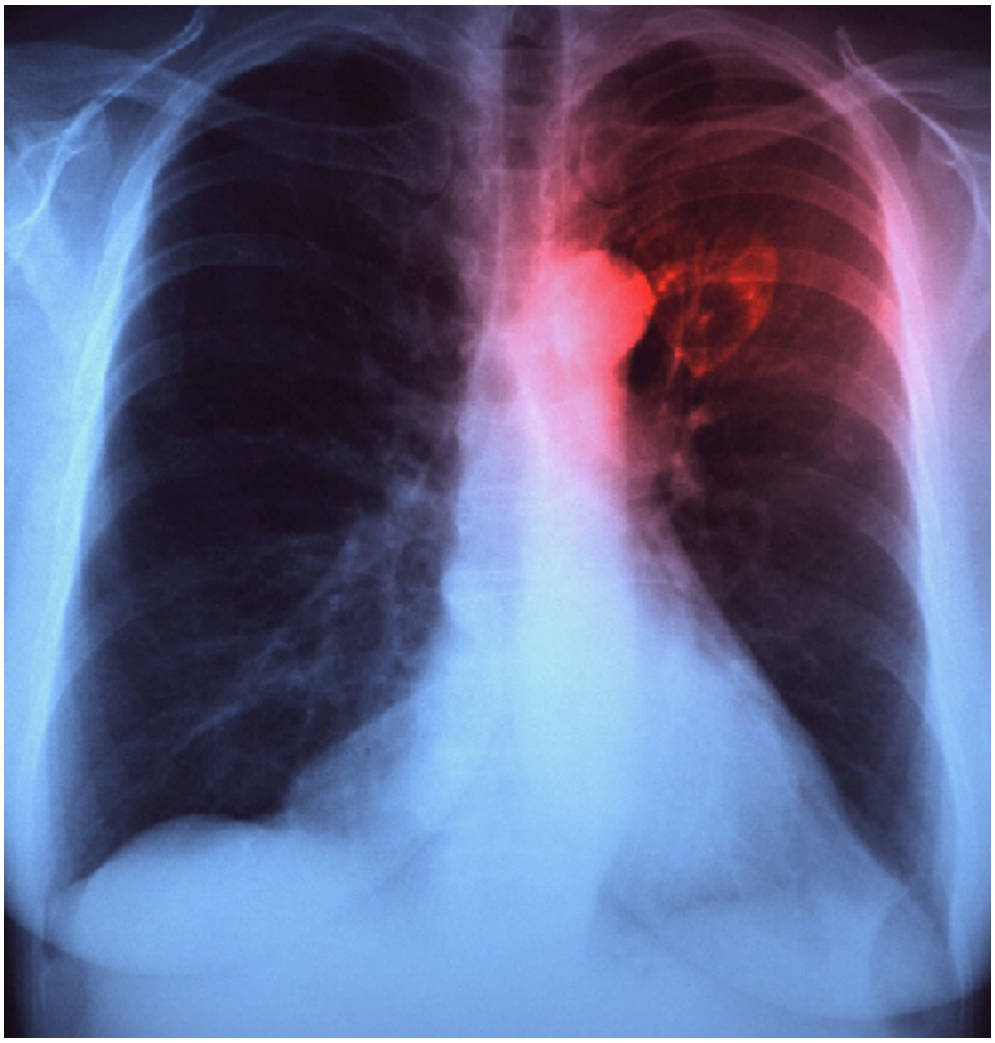


These marrow contents are inflammatory in the lungs, and thus, after a femoral fracture, some patients develop ARDS (adult respiratory distress syndrome).



ARDS

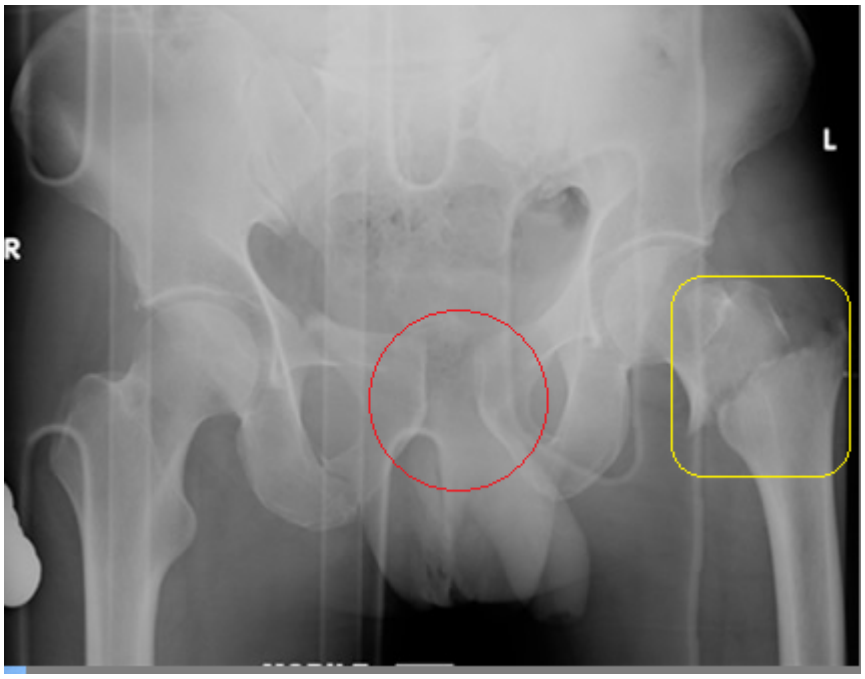
Also, the marrow contents are thrombogenic. Patients with fracture, especially if immobilized can get extremity venous clots, and when a clot embolizes to the pulmonary artery unhappiness is the result.



Other serious co-morbidites of femoral shaft fractures include shock from significant **blood loss** and visceral injuries from the initial injury force that broke the bone.

In this CT angiogram a pseudoaneurysm of medial branch of the profunda femoral artery is seen. Bleeding from this location is not apt to be lethal, but the picture should remind you of the interplay between vascular injury and bone fracture

Below a femur fracture is shown in yellow with a pelvic diastasis (suggestive of a visceral injury) is shown in red



Given the typical (degenerative) etiology of osteoarthritis, how then might a 20 year old person have this condition

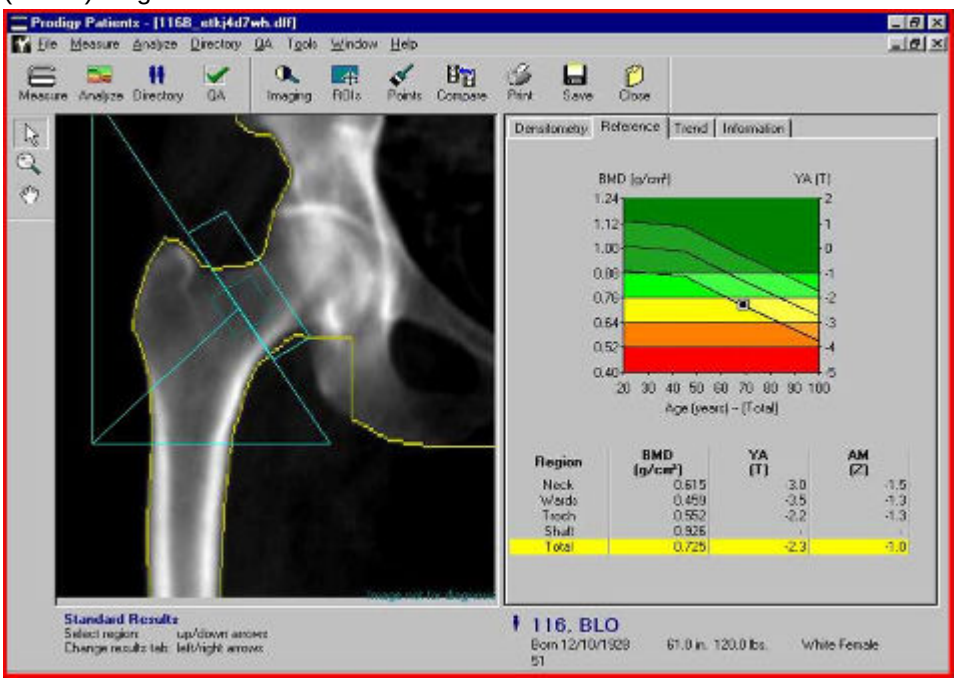
Penn Med Self Study Questions

How is osteoporosis diagnosed, prevented and treated

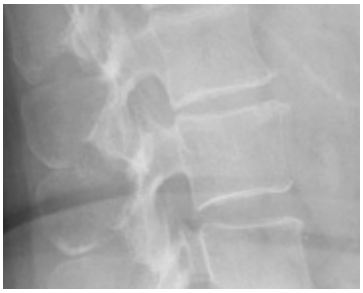
23 How is osteoporosis diagnosed, prevented and treated

How is osteoporosis diagnosed, prevented and treated?
Diagnosis:

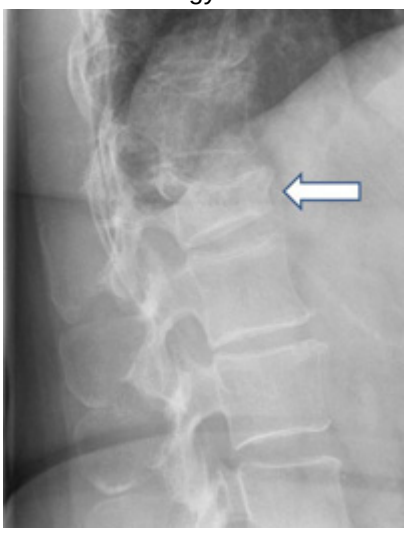
- Good: high index of suspicion in susceptible patients maintained; Dual-energy x-ray absorptiometry (DEXA) diagnostic.



- Bad: faded bones seen on xray (ie advanced osteoporosis as 30% loss of bone does not show up).



- Worst: low energy fracture sustained.



Prevention:

- Primary prevention includes diet supplementation with calcium and vitamin D.
- Pharmaceuticals are typically not used in prevention, but bisphosphonates and raloxifene are approved for preventative use, typically in patients with a DEXA between 2.0 and 2.5.
- Weight bearing exercise also can prevent osteoporosis.
- Avoiding excess alcohol ("excess" defined as "more than I drink") and smoking cessation can also improve bone density.

Treatment:

- Non-Pharmaceutical treatment includes calcium (1500mg daily) and vitamin D supplementation (800 IU daily).
- First-line pharmacologic treatment includes bisphophonates such as alendronate, which inhibit osteoclasts, reducing bone resorption and turnover.
- Estrogen-progestin therapy is now rarely used in postmenopausal women due to cardiovascular side effects.

How can a femoral shaft fracture be lethal | Penn Med Self Study Questions | How is septic arthritis diagnosed definitively

24 How is septic arthritis diagnosed definitively

How is septic arthritis diagnosed definitively? What are the temporal limitations regarding our ability to diagnose definitively? How do we get around that?

Septic arthritis is definitively diagnosed by culturing bacteria from synovial fluid. Yet diagnosis by this method is delayed due to the amount of time it takes to grow out the bacterial culture.

Waiting is bad as the damage begins once the white cells hit the joint.

While waiting for culture results, we can use a clinical decision rule to make a good guess if infection is present: COUNT THE WHITE CELLS AND ASSUME THAT A HIGH COUNT MEANS INFECTION.

(A positive gram stain may help, but it is specific, not sensitive; and even seeing “bugs” does not tell you which bug is present)

(peek into the future here)

How is osteoporosis diagnosed, prevented and treated	Penn Med Self Study Questions	How is septic arthritis treated	

25 How is septic arthritis treated

How is septic arthritis treated? Why must septic arthritis be treated expeditiously?

Septic arthritis is treated removal of the joint fluid and IV antibiotics.

The key consideration is that the body's own response to the infection may inflict horrible damage on the sensitive articular cartilage. Joint fluid with high white counts is toxic! So even if we don't think the patient is at risk for getting systemically sick from a joint infection, we must "wash it out" to save the joint itself. Whether you believe this should be done by serial needle aspirations or by surgery says more about your guild membership (surgeon vs non-surgeon) than about your scientific acumen.

	How is septic arthritis diagnosed definitively	Penn Med Self Study Questions	Plating a fracture clearly disrupts the soft tissue envelope around a fracture

26 Plating a fracture clearly disrupts the soft tissue envelope around a fracture

Plating a fracture clearly disrupts the soft tissue envelope around a fracture. Why, then, is surgical plating ever used?

Here three types of surgical fixation devices seen in the femur

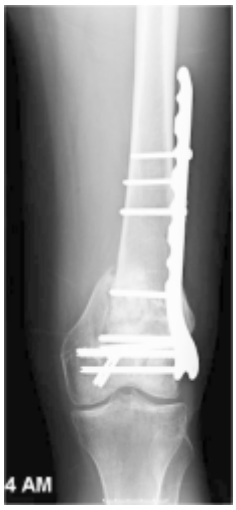
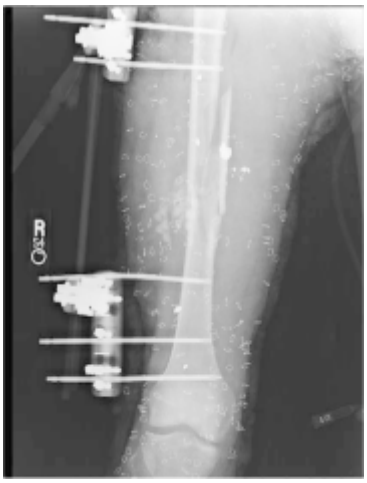
		

Plate	Intramedullary nail (rod)	External fixator

One famous orthopaedic surgeon said "*fractures heal despite internal fixation, not because of it*".

Surgical implantation of bone plates increase risk of non-union. We use them nonetheless, because they likewise decrease the risk of mal-union (healed, but crooked). Plates can be used to restore alignment, especially near the joint line, where even slight deformities are poorly tolerated (ie, if the bone heals but the joint is not aligned, the patient is not helped).

Note: this question applies to **plates** which are a particularly disruptive form of internal fixation. Other means of fixation, such as intra-medullary rods, are less disruptive because they are inserted at a distance and funneled through the center of the bone (medullary canal).

Nails too are invasive of course; it is reasonable, then, for you to ask why they are ever chosen. Simply: because casting the bone in some instances would lead to too much immobilization. The nail seen above, for example, replaces a whole body cast.

The external fixator is probably the least desirable fixation device: it is held by pins, which connect the bone to the (dirty) outside world; and these pins (and the fixator itself) are not very rigid either. The external fixator chosen when no other option exists. In the photo shown, the patient had a gun shot wound with lots of soft tissue and vascular damage. The fixator holds the bone in place while those injuries are addressed. A patient with a gun shot wound with lots of soft tissue and vascular damage may have his fixator removed and replaced by a nail once the soft tissue issues are under control—that is, the fixator may be temporary.

Technical/Biological note: the periosteum, the lining of the bone, is the source of the cells that heal the fracture. Periosteal stripping at the time of surgery may make it easier to get the bone seen and the plate inserted, but will also prevent healing! Surgeons are taught to avoid getting hung up on achieving "perfect" looking xrays, especially at the cost of biological disruption.

	How is septic arthritis treated	Penn Med Self Study Questions	Provide a brief description of Carpal Tunnel Syndrome	

27 Provide a brief description of Carpal Tunnel Syndrome

Provide a brief description of Carpal Tunnel Syndrome, noting the chief complaints, examination findings (including signs, wigns and symptoms); and treatment options.

Carpal Tunnel Syndrome (CTS) is an entrapment neuropathy due to compression of the median nerve as it passes through the carpal tunnel. The tunnel is at the base of the palm, right above the wrist. It has four sides: 3 of which are carpal bones and the 4th side/top of structure being the transverse carpal ligament.

The key clinical point is based on the anatomy: the signs and symptoms must be in the median nerve only; and they need to be related to targets of the median nerve *distal to the tunnel itself*. That is, objective median nerve function in the forearm should not be affected.

Carpal Tunnel Syndrome occurs most commonly in patients between 30-60 years of age; more common in females.

Risk/causative factors: POSITIONAL (this may be why typing a lot causes symptoms); increases in contents of canal (fractures synovitis); neuropathic conditions (DM); inflammatory conditions (RA, gout); alterations of fluid balance (pregnancy, menopause, , thyroid disorders); and external forces (jackhammer?).

The **chief complaints** are numbness and paresthesias in the anatomic distribution of the median nerve: radial 3 ½ fingers (thumb, index, middle, and radial side of ring). Patients may experience pain radiation proximally into the forearm.

A common early complaint is awakening in the night due to numbness or pain in these fingers (night-time worsening). Patients may also complain of swelling in the hands, dry skin, and cold hands (less common symptoms).

Later, patients may report constant numbness, motor disturbances, and decreased strength (tendency to drop objects)

Exam findings: the exam may show: Nothing; Weakness of the thenar intrinsic muscles (clinically tested by abduction of thumb against resistance)**;** Diminished sensation to pin prick in the median nerve distribution; a Positive Phalen test or Tinel's test (not "sign"); or any combination thereof.

EMG would likely show focal slowing of conduction velocity in median nerve across carpal tunnel

(A note to the interested student: the accuracy/sensitivity/specificity of various diagnostic maneuvers or tests is hard to define: What is the reference standard? If all are compared to, say, “positive EMG” we have a problem, as there people without any Carpal Tunnel symptoms who have a positive EMG; likewise there are some with normal EMGs who seem to have the syndrome… Recall, the word “syndrome” means that the condition is somewhat ill-defined. As treating doctors, we don’t necessarily want to know what the patient has, we want to know what to do with them therapeutically (or what to say regarding prognosis) . So the reference question should be “What percentage of patients with a positive Phalen’s test, say, get relief from surgery?” But that is not only an assessment of the test, but also the patients in the study and the surgeon doing the work…)

There are many **treatment options** for individuals with CTS, and the treatment choice depends on the severity of the nerve dysfunction, patient preference, and availability. Non-operative treatment options include: rest, wrist splinting, NSAISDs, and oral steroids or corticosteroid injections.

In patients who do not respond to more conservative treatment modalities or in patients with signs of atrophy or muscle weakness, carpal tunnel release--cutting the transverse carpal ligament --can be considered. This surgery is performed to decrease pressure on the median nerve.

Obviously if there is a precipitating cause (like a wrist fracture) that cause should be addressed expeditiously

⇦	Plating a fracture clearly disrupts the soft tissue envelope around a fracture	Penn Med Self Study Questions	the motor and sensory findings of each nerve root level	⇨

28 the motor and sensory findings of each nerve root level

29 Tinnel’s sign is not a true sign

Tinel's test is performed by tapping over the median nerve as it passes through the carpal tunnel in the wrist. A positive response is a sensation of tingling in the median nerve distribution over the hand.

This is *not* a sign because it reports a subjective sensation. Signs are objective. The response to a Tinel's test might better be termed a "wign", defined as a subjective reaction to a provocative examination maneuver deemed to have some valid relationship to the underlying pathology.

This word is pronounced "whine" to remind us it is a spoken response, and its spelling echoes that of sign, reminding us likewise a wign might be more specific that than a complaint.

The distinctions between sign, symptom, and wign are worthy of preservation: treatments offered on the basis of signs can be said to be most rigorously indicated, as symptoms, unlike signs, pass through (and are affected by) the prism of patients' perceptions.

Remaining skeptical about the value of information provided by our patients is in the interest of these patients, as our skepticism might save them from unnecessary treatments and procedures.

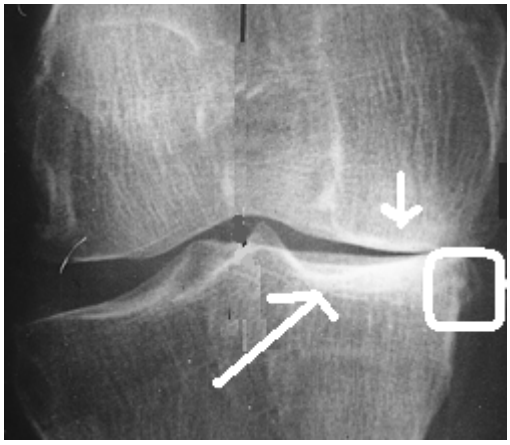
⇦	the motor and sensory findings of each nerve root level	Penn Med Self Study Questions	What are the cardinal signs of osteoarthritis of the knee on plain radiographs	⇨

30 What are the cardinal signs of osteoarthritis of the knee on plain radiographs

What are the cardinal signs of Osteoarthritis of the knee on plain radiographs? How (mechanistically) do they appear?

Cardinal signs of osteoarthritis of the knee on plain radiographs are:

1. **osteophytes** (circled),
2. **asymmetric joint space narrowing** (short arrow)
3. **subchondral sclerosis** (long arrow), and
4. **subchondral cysts** (hard to see here).

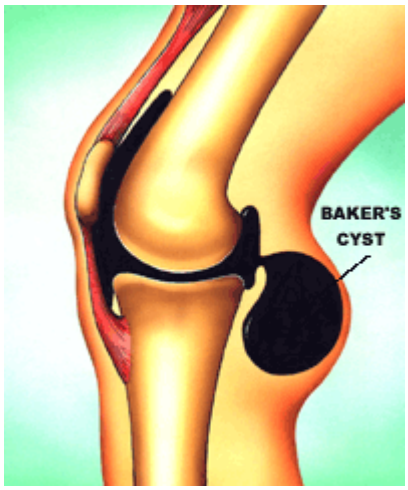


Asymmetric joint space narrowing occurs as articular cartilage is lost is areas of abnormal load. Notice above that the lateral space is wide (though not necessarily disease-free; it may be wide because the joint is tilted to the medial side. That is, if the bones are touching, there is no cartilage; if they are not touching, maybe there is cartilage, maybe there isn't.)

Osteophytes typically develop. Why? Maybe as a (futile/foolish) reparative response. Maybe because of abnormal loads stimulating bone. "Peaking of the tibial spines", ie higher than expected, may be considered a form of osteophytosis.

Subchondral sclerosis is simply the deposition of bone in area under areas of stress—Wolff's law. This makes the bone stiff and less compliant, and more prone to further damage.

Cysts form when joint fluid seeps through the cracks in the cartilage and get into bone. When the fluid escapes into the soft tissue, a BAKERS CYST may develop.



Tinnel's sign is not a true sign | Penn Med Self Study Questions | What are the classic motor and sensory findings of L4 L5 and S1 compression

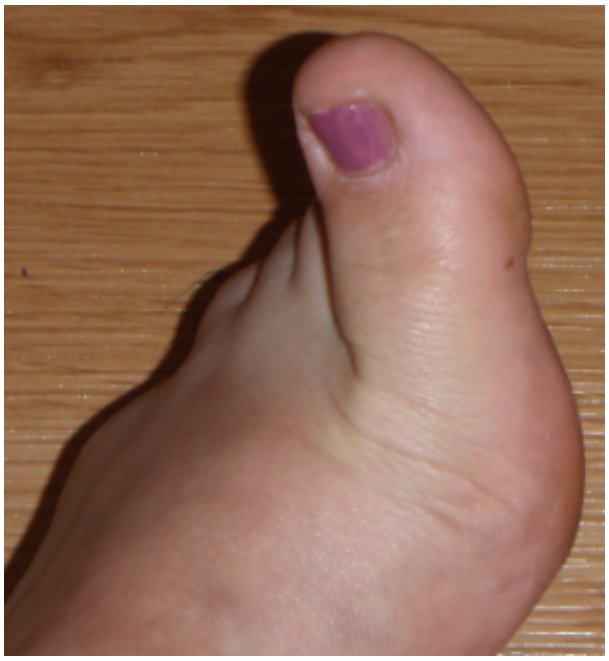
31 What are the classic motor and sensory findings of L4 L5 and S1 compression

A so-called "herniated disc" may compress a nerve root and cause radicular complaints/findings. What are the classic motor and sensory findings of involvement of L4? What are the classic sensory and motor findings of involvement of L5? What are the classic findings for S1?

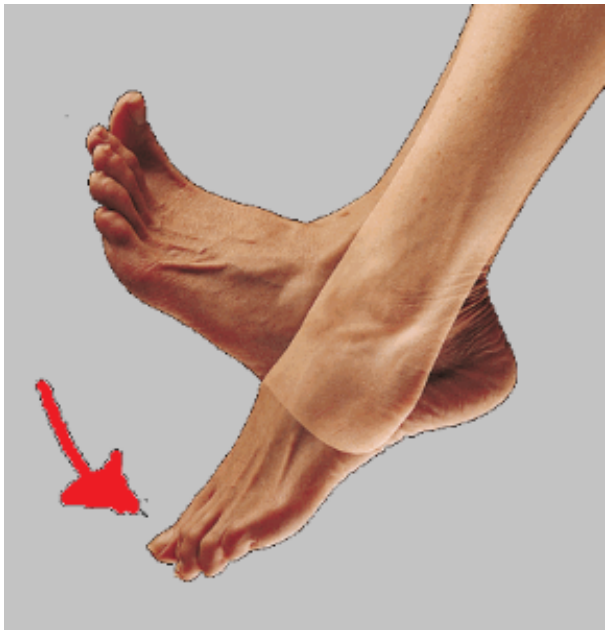
Why, given that an MRI can localize the disc herniation, if present, should/must a student know the motor and sensory findings of each nerve root level?

An L4 disc herniation often presents with quadriceps weakness (if any), medial knee and shin sensory loss and pain distributed down the anterior thigh.

An L5 disc herniation classically presents with weakness in extension of the big toe (EHL), sensory loss in the big toe, and pain distributed down the back of the thigh and lateral calf.



An S1 disc herniation classically presents with weakness of the gastocnemius causing impaired ankle plantar flexion, sensory loss of the lateral foot and pain distributed down the back of the calf. The motor findings are more reliable than the sensory.

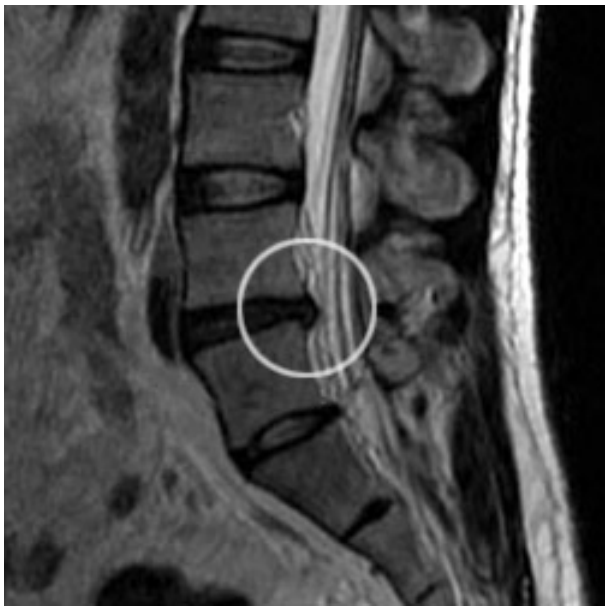


We need to be able to match the likely "positive" findings on MRI with the findings on exam.

A Penn med grad, Scott Boden among others has shown that many asymptomatic people have positive MRIs hence the radiologist will not say that all findings are necessarily pathological; rather he or she will say "clinical correlation suggested". By knowing what each lesion might cause you can make that correlation.

Thus, there is a strong medical rationale for *not* obtaining an MRI in a patient with low back pain and no neurological features: MRIs of healthy patients can lead to false positives and incorrect diagnoses that cause unnecessary stress, psychosocial difficulties and even psychiatric morbidity AND MOST ESPECIALLY OVERTREATMENT.

As noted above, many asymptomatic people have positive MRIs. Accordingly, the purpose of the MRI is not to screen (for it will fail on that account by picking up too much) but rather to plan the next step in treatment.



MRI ABOVE: Does this person have pain? If so, does it match?

MRI is needed primarily as surgical (or injection) planning investigation, a "gateway test", one might say, to many perhaps unnecessary (one also might say) procedures.

If all patients with back pain were to get an MRI there is a great chance that more (unnecessary) back surgery will be done.

The interested student is encouraged to consult the medical literature on the following points: first, the success rates of surgical treatment of back pain; the variability of the rates of back surgery by region of the country (suggesting non-scientific indications, one might say); and the cost of such surgical treatment. The answers are "low", "high" and "high" in case you could not guess…

In the scheme of things, MRI is almost free; that is, the dollar cost of the test itself is a trivial to the cost of the treatment it could invoke. Don't be fooled by the list price (what is charged) but concentrate on what is actually paid.

What are the cardinal signs of osteoarthritis of the knee on plain radiographs	Penn Med Self Study Questions	What are the necessary conditions for appropriate bone healing	

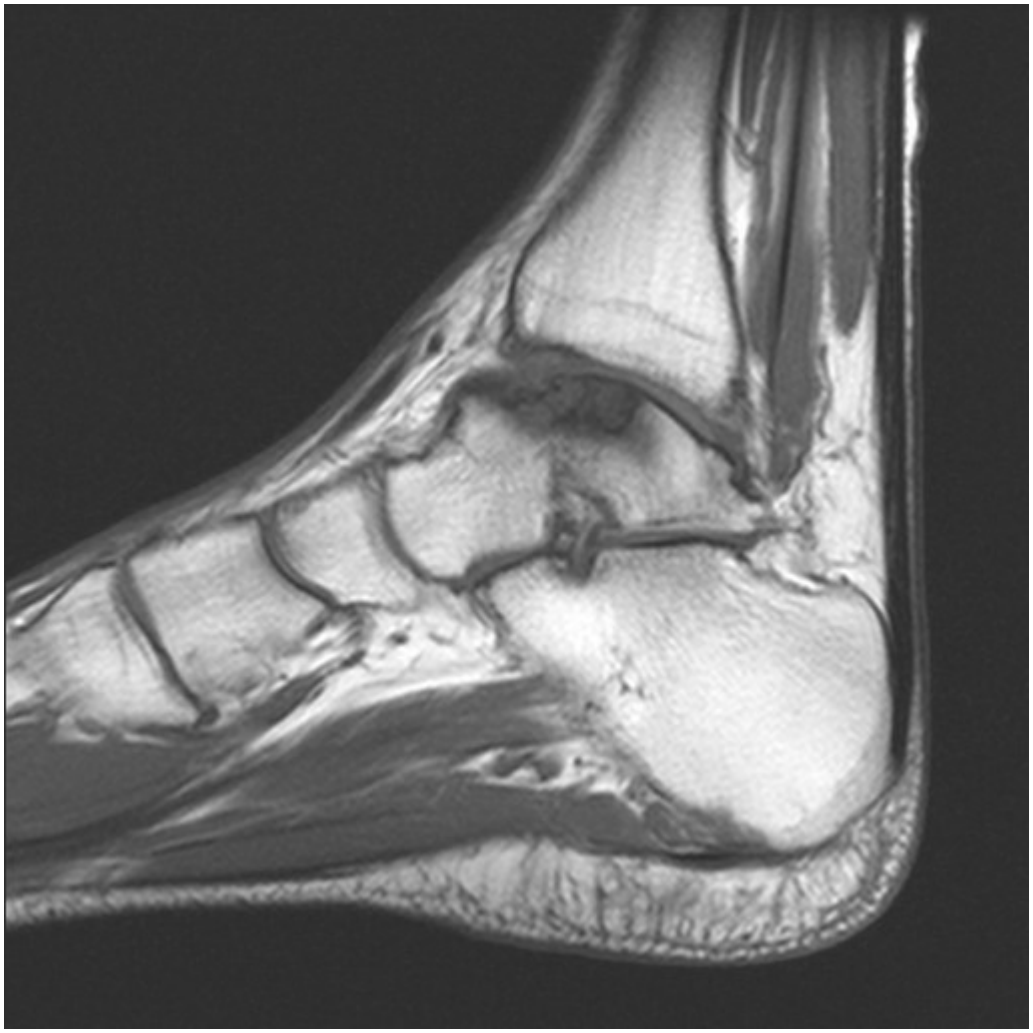
32 What are the necessary conditions for appropriate bone healing

What are the necessary conditions for appropriate bone healing (leading to minimal functional residuals) and how may physicians optimize the chances for healing?

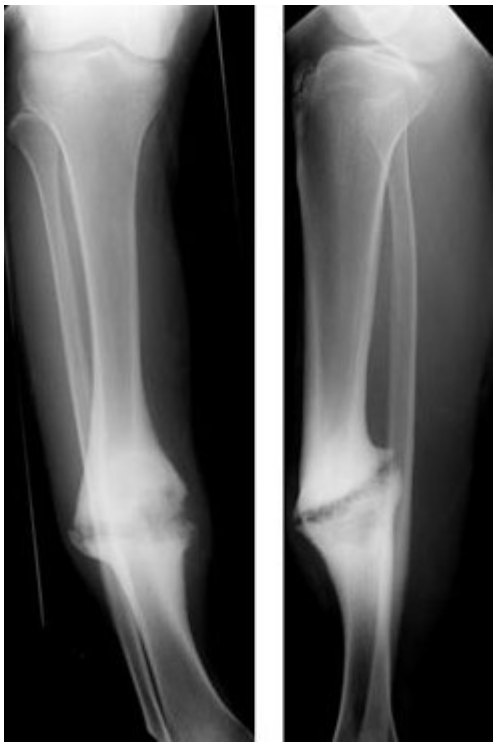
Successful bone healing requires:

- adequate blood supply,
- relative mechanical stability,
- sterility and
- intact surrounding soft tissue.

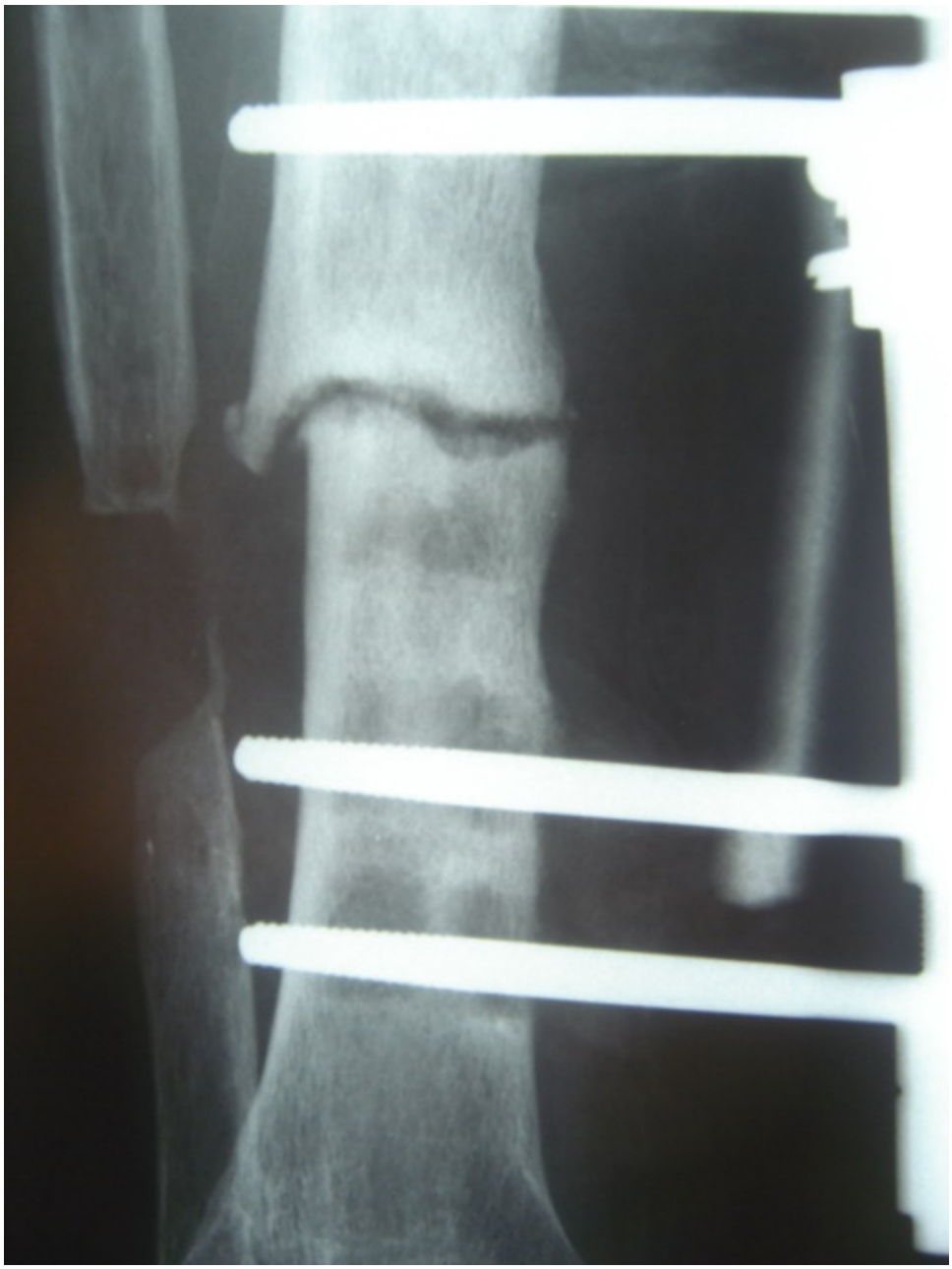
Here is a talus that died because of **inadequate blood supply**



Here is a fracture that did not heal because it was not held rigid

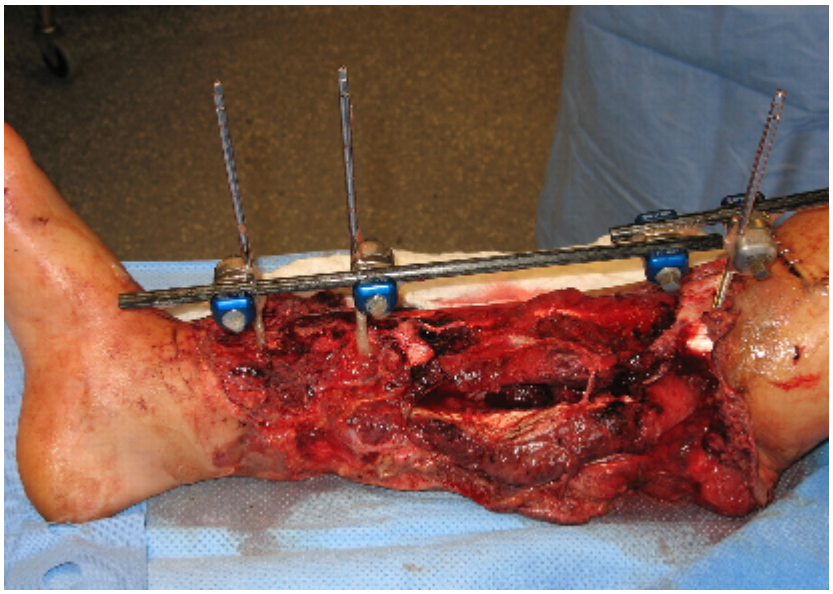


Here is a fracture that did not heal because it got infected (pins are from an external fixator)



Pseudoartrosi settica di tibia

Here is a fracture that is going to have a tough time healing due to a dearth of intact surrounding soft tissue.



Physicians may optimize chances of healing by promoting the proper mechanical and biological environment.
Specific measures include:

- Reducing (aligning) the fracture;
- making sure the blood supply and soft tissue envelope are preserved or restored
- preventing or treating infection;
- minimizing edema (more for pain control and compartment syndrome prevention, but also to promote perfusion) and
- allowing just enough loading on the bone to stimulate bone growth but not so much to ruin the reduction or prevent hardening of the fracture callus. (gross motion at the fracture will lead to a so-called fibrous union: some tissue there, but not hard tissue)

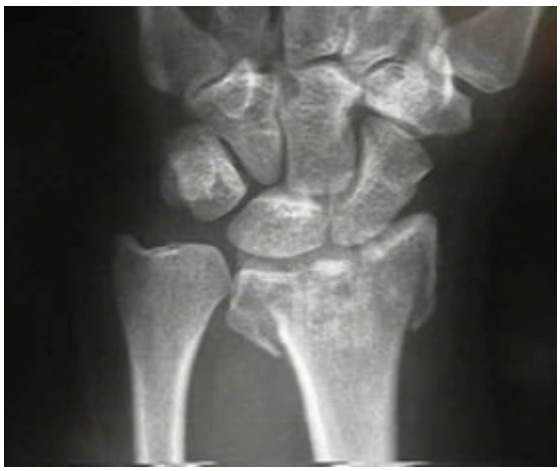
What are the classic motor and sensory findings of L4 L5 and S1 compression | Penn Med Self Study Questions | What are the three fractures typically associated with osteoporosis

33 What are the three fractures typically associated with osteoporosis

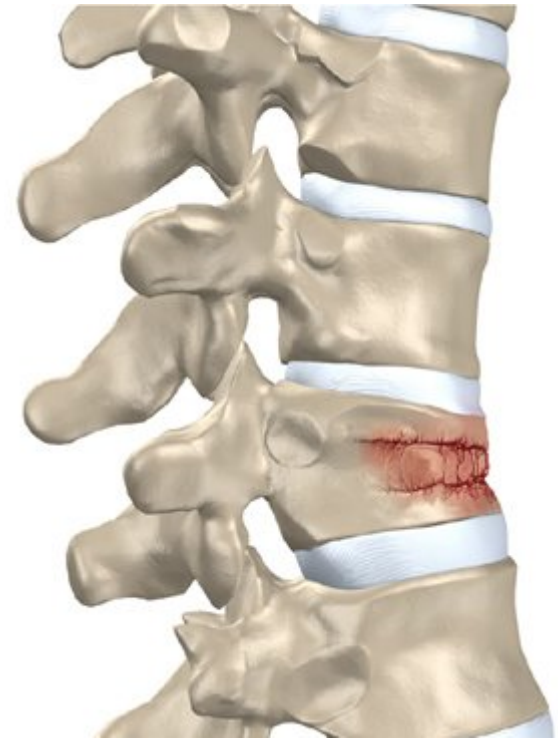
What are the three fractures typically associated with osteoporosis? Which is the worst? Why is this fracture so deadly?

The three areas typically subjected to fragility fracture with osteoporosis are

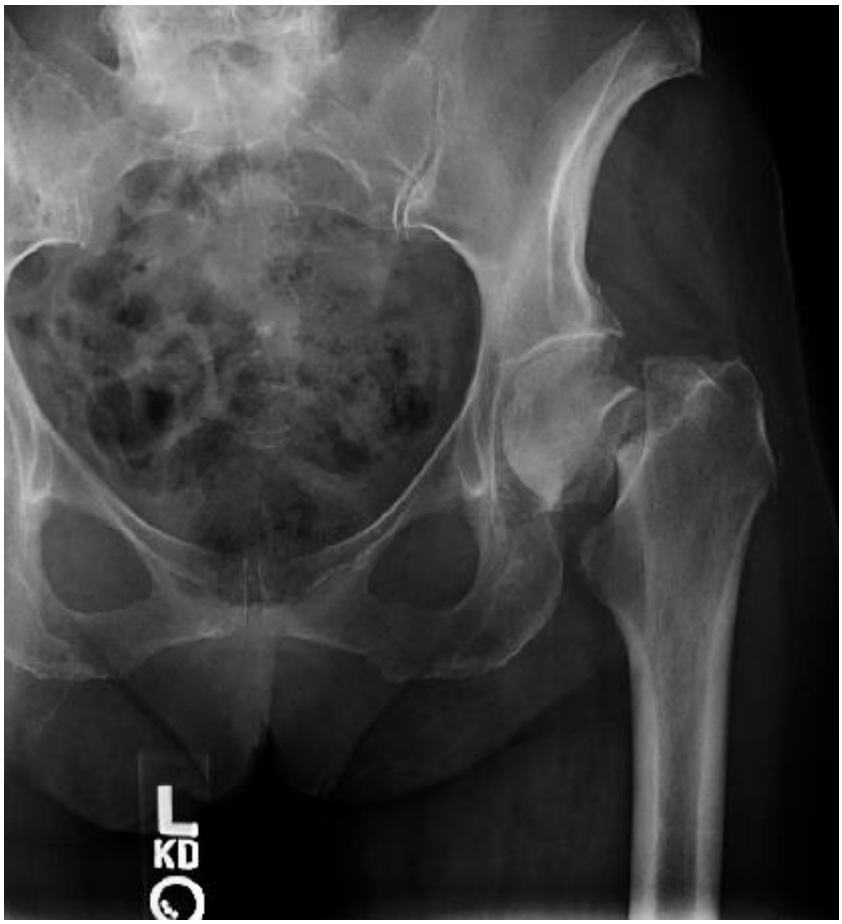
wrist (distal radius)



vertebral body compression



hip (femoral neck shown and intertrochanteric),.



Hip fractures are the worst: there is a ~30% mortality within the first year of fracture. That may be because of its effect on mobility, or maybe because getting a fracture in the first place is a sign of the dwindles…

And don't forget: a low energy wrist fracture is a sign suggestive of underlying osteoporosis—a wrist fracture from a fall should be the initiator of an osteoporosis work up, or empiric treatment.

What are the necessary conditions for appropriate bone healing | Penn Med Self Study Questions | What are the three tasks of bone

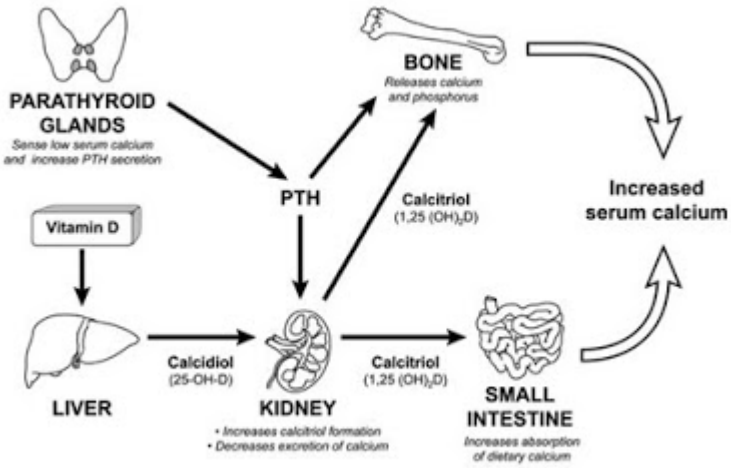
34 What are the three tasks of bone

What are the three tasks of bone? How can problems related to the two non-structural tasks lead to fracture?

The three tasks of bone are: **skeletal homeostasis, mineral homeostasis and hematopoesis.**

Mineral homeostasis involves maintaining the correct serum levels of calcium, phosphate and magnesium and other ions. PTH increases serum calcium levels by increasing GI calcium absorption, renal phosphate and calcium resorption and releasing calcium from the skeleton by de-boning the bone

As shown here (and don't memorize this for this class!) bone is only one of the actors, and plays a relatively subservient role



Hyperparathyroidism, to name one disease of aberrant mineral homeostasis, will increase osteoclast activity and therefore weaken the bone. Vitamin D deficiency in adults, to name another process, can cause defective bone mineralization, and will might lead to pathologic fractures.

In general, if the body needs minerals it will take them from the bones. You need the right level of Calcium to have a heart beat. You need a skeleton (in evolutionary terms) only to get to food and mate(s). Despite what you may recall of the vicissitudes of adolescence, cardiac contractility is more important, at least on a minute to minute basis, than finding food or mates.

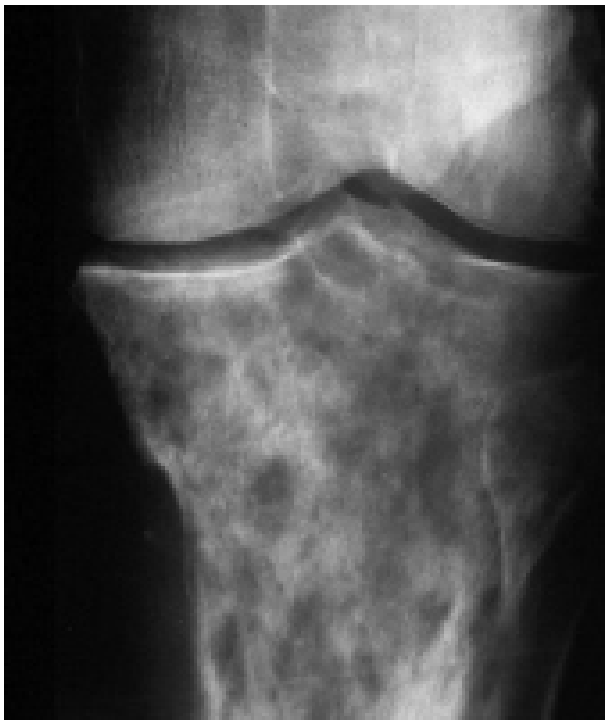
Recall that Calcium is critical for nerve transmission, as shown here

Hematopoesis problems can also lead to fracture - indirectly. The indirect link is that because the blood-making apparatus resides in the bone, blood cell cancers that originate in the bone can cause local bony damage. Also, because the blood-making apparatus is in the bone, the bones are essentially part of

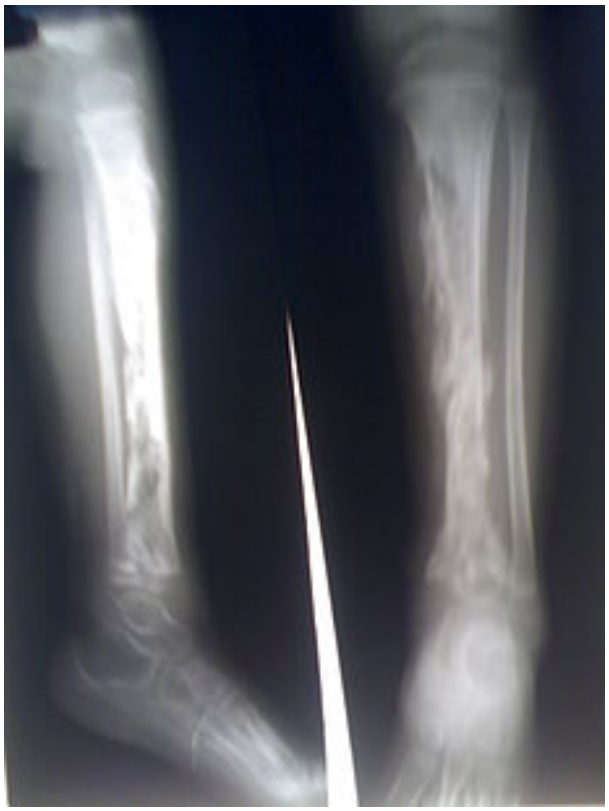
the vascular system. And because bones are part of the vascular system, bad blood cells can get stuck there (for example, sickled red blood cells can muck up the circulation and lead to infarction) as can metastatic cancer cells or infectious microbes.

FIGURES:

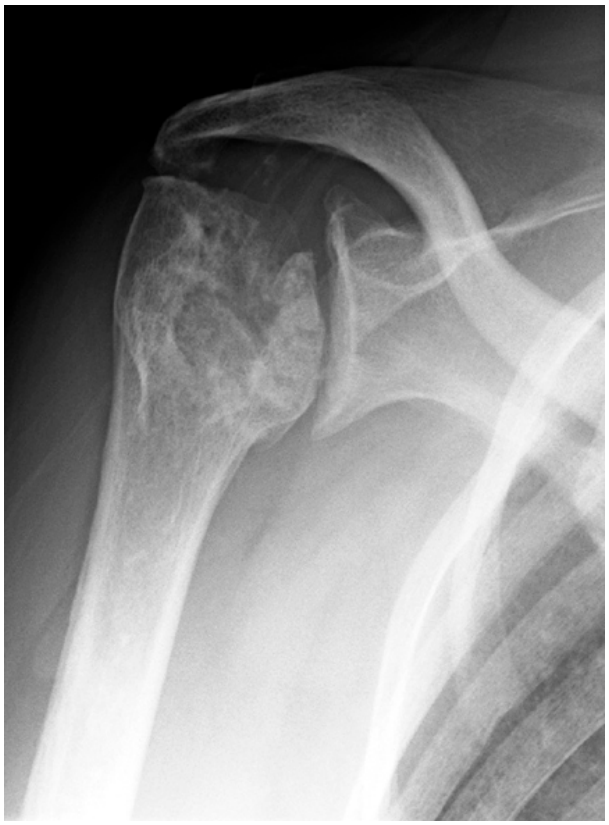
lymphoma of bone



osteomyelitis (infection) of the tibia from circulating bacteria



Infarction of bone from Sickle cell disease



What are the three fractures typically associated with osteoporosis | Penn Med Self Study Questions | What else besides intrinsic bone problems could cause hip fracture

35 What else besides intrinsic bone problems could cause hip fracture

*Heaney wrote, "Although bone mass is certainly the most extensively studied of the fragility factors, low bone mass is not the whole of the osteoporosis story and may not even be its most important component (despite frequent assertions to the contrary). If one could magically normalize bone mass in everyone, would one eliminate osteoporotic fractures? The best answer that can be given today is 'no.' There would be fewer such fractures, but there would still be many, especially hip fractures." (Heaney RP Bone Mass, Bone Loss, and Osteoporosis Prophylaxis. Annals Internal Medicine 15 February 1998 128: 313-314)_

As Heaney implied, What else besides **intrinsic bone problems** could cause hip fracture?

As the figure below shows (with a lot of artistic license), osteoporosis makes the bone intrinsically weak, simply by offering less structural mass

But there is more Old people get the dwindles. They fall more and when they fall, the risk of fracture is higher because they can't catch themselves. A risk of falls (from caused such as bad vision, say, or a neurological disease) is an independent risk factor for a hip fracture.

The key point to know and recall is that the amount of energy needed to break a hip is only a fraction of the energy available from a typical fall. That most falls do not cause fracture is a testament to the normally present energy-absorbing processes (catching yourself, basically).

So if you fall frequently and if you can't catch yourself as you fall, you are going to break bones even if those bones are intrinsically healthy.

A related point: that's why patients falling off the OR table is such a potential disaster: sleeping patients can't catch themselves! (and anecdotally, some of the worst fractures I have seen are in the inebriated.)

What are the three tasks of bone | Penn Med Self Study Questions | What is a Charcot joint

36 What is a Charcot joint

What is a Charcot joint?

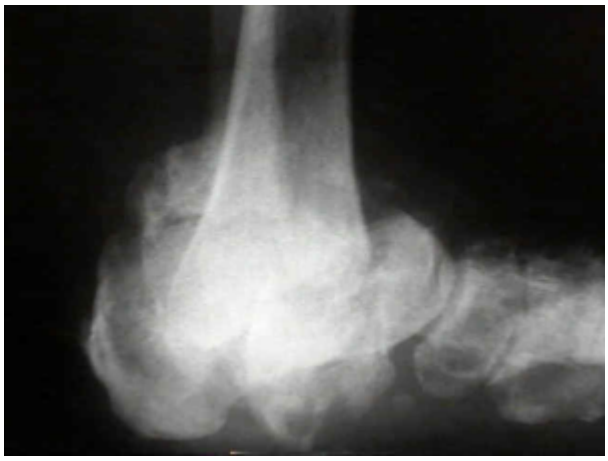
If you don't feel pain, you hand stays in the fire; likewise, if you don't feel overuse in your joints you won't let up and will destroy the joint.

Hence: decreased sensation in cases of peripheral neuropathy leads to JOINT DESTRUCTION (mental image: pimple on one butt cheek, sit on the other side or get a bed sore).

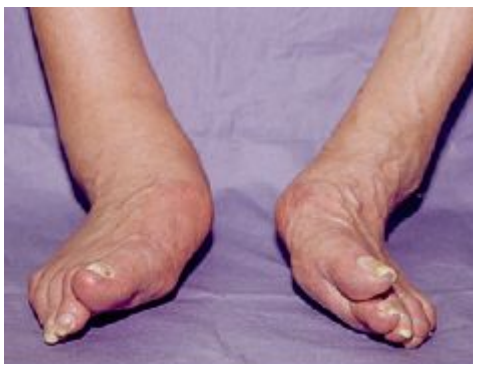
An alternative or complimentary theory: micro-vascular disease of DM leads to altered bone metabolism. The main cause of Charoct today is diabetes. Historically, the #1 cause was syphilis (NB: syphilis would tend to support theory of lost sensation as cause, I say, thinking out loud).

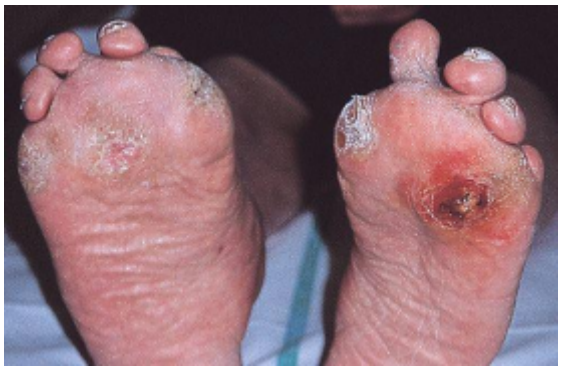
Charcot affects the foot mostly, but can be found in other joints.

The presentation may look like cellulitis/infection (which is more likely, also, in patients with diabetes).



There is a vicious cycle: deformity leads to bony prominences leads to ulcers leads to infection leads to more bony deformity.



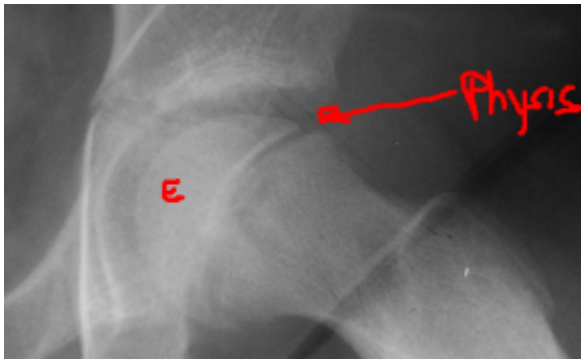


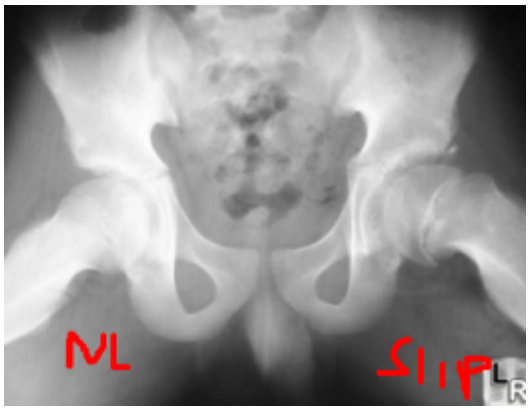
What else besides intrinsic bone problems could cause hip fracture | Penn Med Self Study Questions | What is a Slipped Capital Femoral Epiphysis

37 What is a Slipped Capital Femoral Epiphysis

What is a Slipped Capital Femoral Epiphysis?

First, you have to know that the femoral head (capitus) has a physis (a growth plate). Then you have to know that the bone towards the end, across the physis, is called the epiphysis





Then you have to know that the physis is cartilage-like, and therefore weak. And then you can know/imagine that the epiphysis can slip off, a form of a growth plate fracture, really.

So what else do you need to know?

1. who gets this? 1 in 10,000 kids, 4x more common in blacks, usually during early puberty, when the physis active. Obesity and endocrinopathies are risk factors
2. How do you diagnose? Get an xray---but note that the complaint may be KNEE pain!
3. How do you treat? Pin it in situ
4. Why do you treat? Prevent progression, symptom relief. Most doctors do not attempt to relocate the epiphysis, fearing further damage to the blood supply.

What is a Charcot joint | Penn Med Self Study Questions | What is a stress fracture

38 What is a stress fracture

Define "stress fracture". How is a stress fracture treated in a normal person? What are the consequences of a stress fracture which is not treated? Why might a young woman with an eating disorder be at particular risk for a stress fracture?

A stress fracture occurs when a bone breaks microscopically after being subjected to repeated tensile or compressive stresses, ie cyclical loading, in which not one single load which would be large enough individually to cause the bone to fail. (A "normal host" is defined as a person who is not known to have an underlying disease that would cause abnormal bone fragility.) That is, stress fracture is the granddaddy of "repetitive stress injuries".

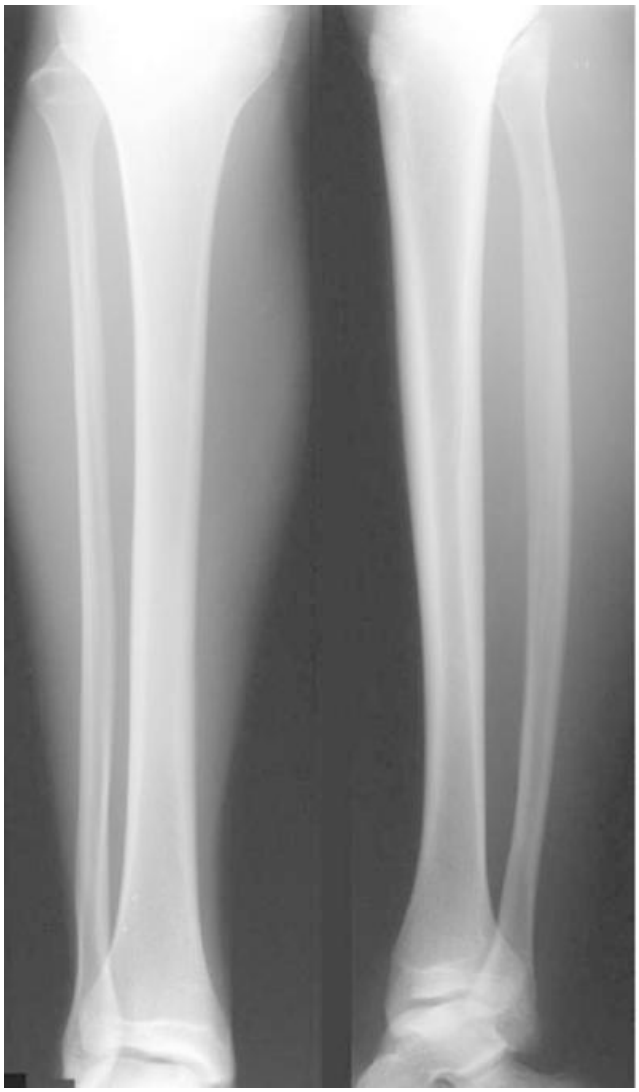
Here is an image to keep in mind

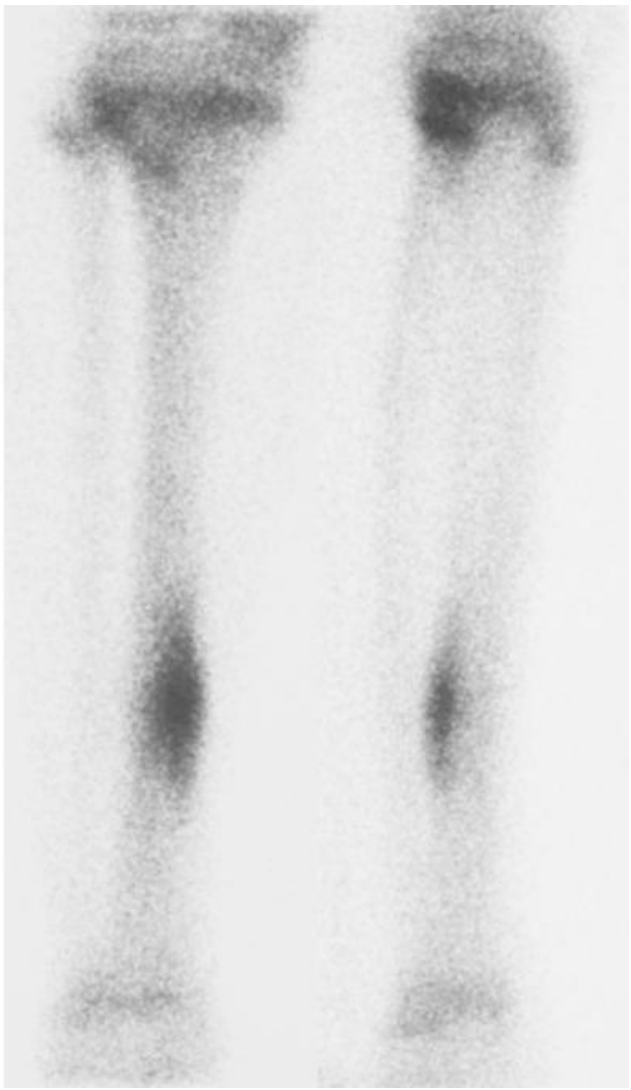
As you know, bending a paperclip too much will break it. If a clip will break with 12 bends, at the 11^{th}, it has a stress fracture.
A stress fracture is believed to develop with abrupt increase in the duration, intensity, or frequency of physical activity without adequate periods of rest.

Important risk factors for developing stress fractures include a history of prior stress fracture, low level of physical fitness, increasing volume and intensity of physical activity, female gender and menstrual irregularity, diet poor in calcium, poor bone health, and poor biomechanics.

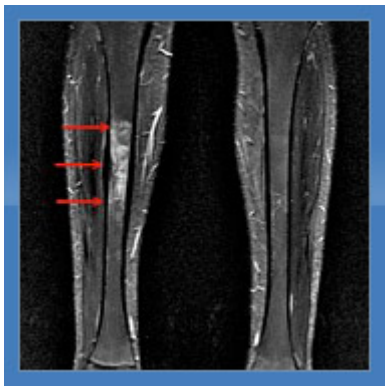
A stress fracture is also called a "march fracture" as taking a 40 mile stroll with military gear on one's back is a nice way to cyclically overload the bones

A bone scan, shown on the right, can help demonstrate the injury, as only an overt break would be seen on plain films



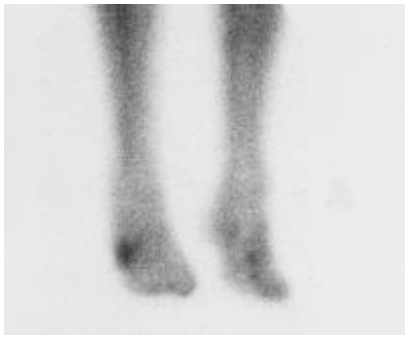


MRI can also make the diagnosis,



Photo

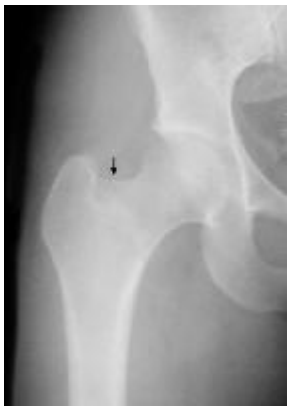
In addition to the tibia, the feet (5th metatarsal especially) are commonly involved



Stress fracture is, fundamentally, an overuse injury and THE TREATMENT OF AN OVERUSE INJURY IS UNDERUSE . (That is, if the patient would only stop beating up the bone and just let it heal all will be well.)

(photo of patient being treated for a stress fracture)

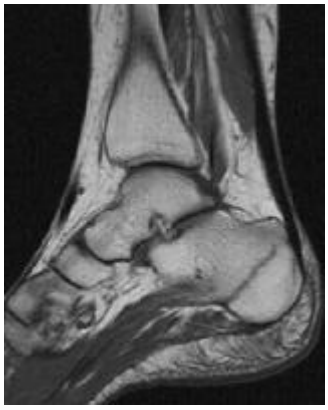
Not listening to the instructions to stop is a sign of not being normal…If they don't listen, a stress fracture can lead to "real" (separated) fracture.



A cortical disruption (arrow), along with a fine l

A stress fracture of the superior femoral neck, shown here, can be a real disaster if not healed, as the fracture can displace and disrupt the blood supply to the femoral head

By contrast, a stress fracture of the calcaneous, if it displaces, will collapse on itself and not separate



T1-weighted sagittal magnetic resonance image from

Women with eating disorders are at particularly high risk for stress fractures because they typically lack adipose (a chemical precusor for many hormones) and thus do not have proper estrogen levels. Because osteoclasts are estrogen sensitive, these women have imbalanced bone remodeling –more eating, less building—and bad bone.

Eating disorders are one third of the so-called female athlete triad (eating disorders, amenorrhea, and osteoporosis).

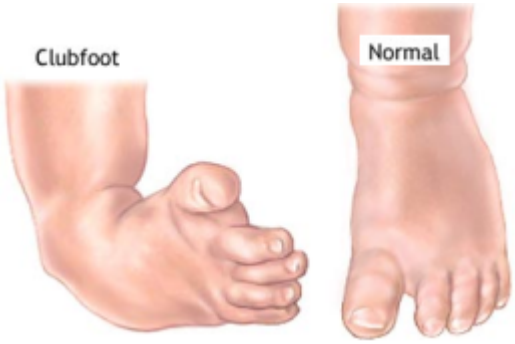
⇦	What is a Slipped Capital Femoral Epiphysis	⇧ Penn Med Self Study Questions	What is Club foot	⇨

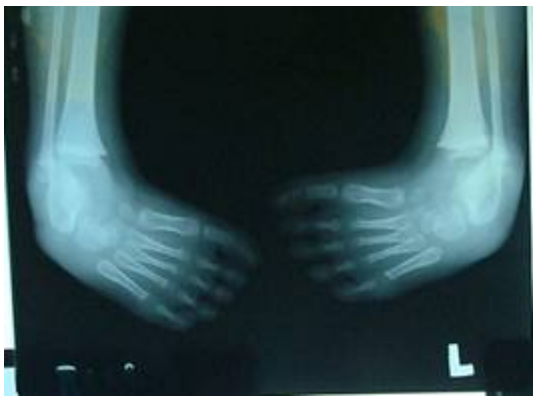
39 What is Club foot

What is "club foot"?

Club foot is technically known as congenital talipes equinovarus (talipes is from Latin talus=ankle + pes=foot; equino-=of or resembling a horse and --varus=turned inward).

Club foot is a developmental deformity of the foot in which one or both feet are excessively plantar flexed, with the forefoot swung medially and the sole facing inward.



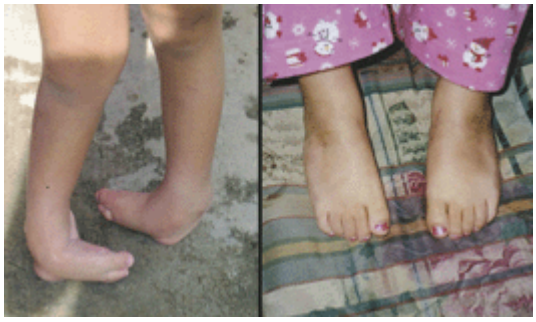


The main anatomic abnormality is in the TALUS but screwing up (over-flexing) a bone in the middle of the action messes up everything else: the talo-calcaneo-navicular joints; the soft tissues on the medial side of the foot; and the gastrocsoleus/Achilles; etc

The forefoot is "normal" but because the hindfoot is bad, the forefoot does not strike the ground normally. Patients with (untreated) club foot often appear to walk on their ankles, or on the sides of their feet. Most cases are idiopathic, but some are associated with chromosomal anomalies.

Dr Ignacio Ponsetti, from Iowa via Spain, 1915-2009 (roughly) and shown below, devised a method that WORKS for manipulating the foot back to normal with serial casts.

One of the few areas where low tech beats high tech so decidedly that even in America we do it this way. Below are before and after Ponsetti treatment pictures from the AAOS website



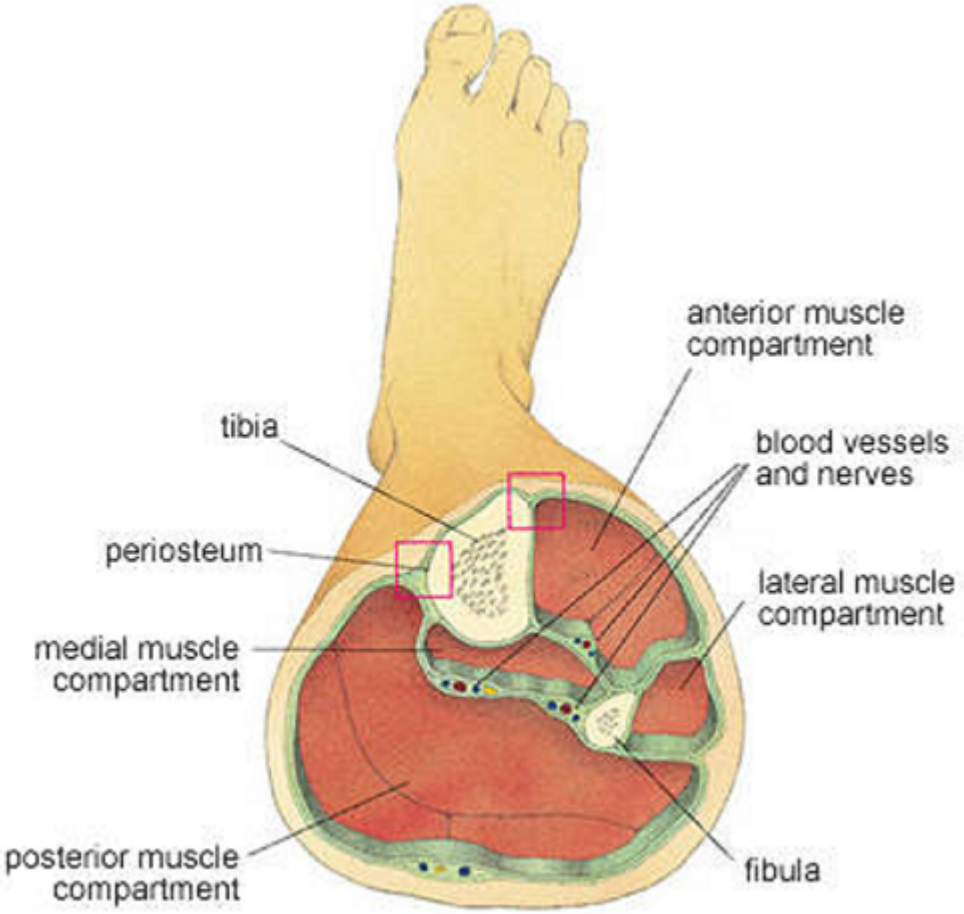
What is a stress fracture | Penn Med Self Study Questions | What is compartment syndrome

40 What is compartment syndrome

What is compartment syndrome and how is it prevented, diagnosed, and treated? What are the consequences of not treating a compartment syndrome and over-treating a (falsely) suspected compartment syndrome?

Compartment syndrome is the clinical condition of increased pressure within an enclosed fascial space, leading to muscle and nerve death from ischemia.

The leg is a typical location because of its well defined (and not very roomy!) compartments, as shown



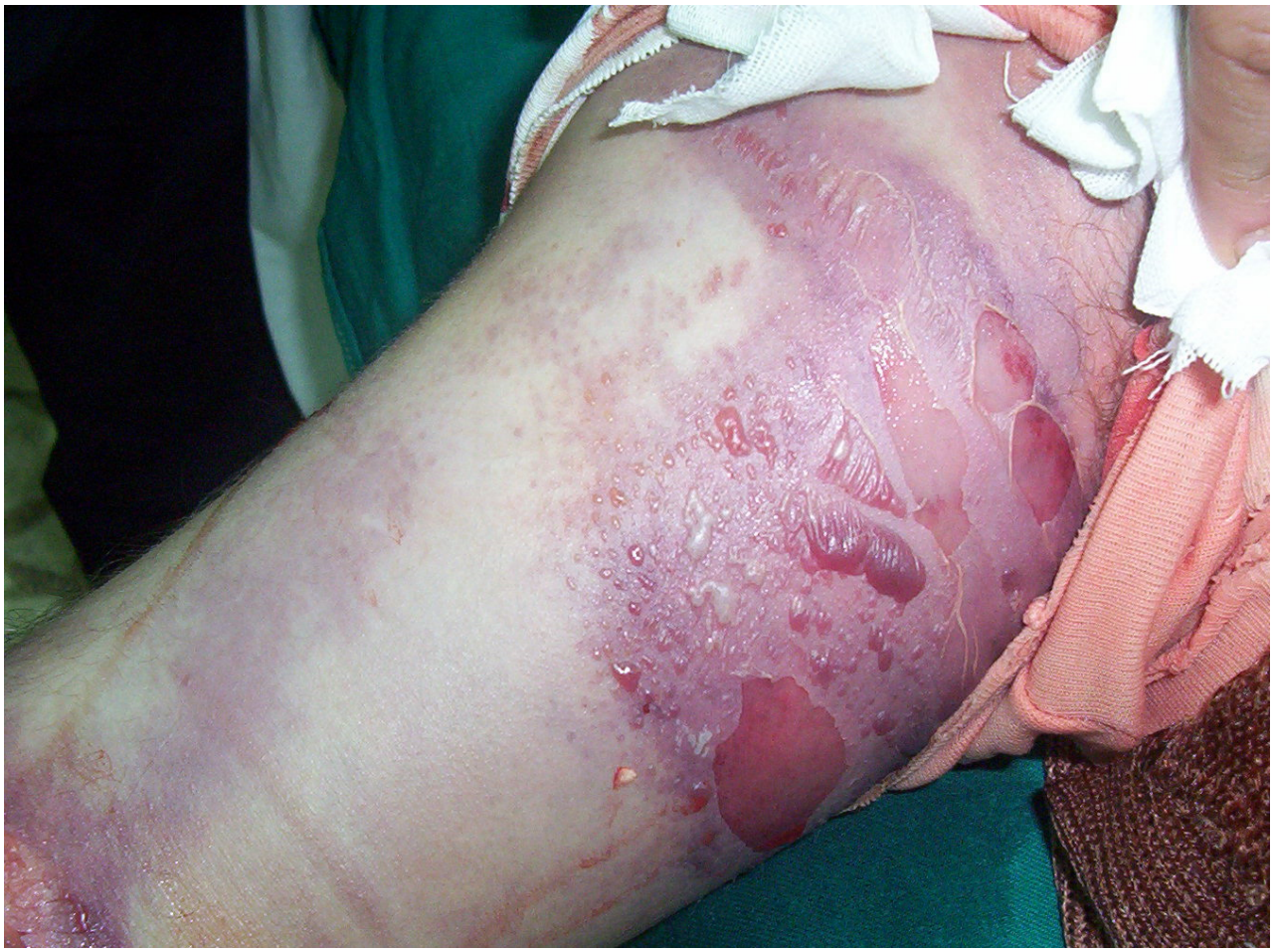
Compartment syndrome could occur from tibia fractures (bleeding), compressive devices (casts, ace wraps), IV infiltration or burns. (Reprofusion after vascular repair is a non-musculoskeletal cause too.)

The hallmark of compartment syndrome is severe pain that is out of proportion to what is expected from the given injury/situation. One clue what is "out of proportion to what is expected" is **pain that increases over time** (by contrast, a patient with a splinted fracture should start hurting less once immobilized).

The patient controlled analgesia machines (by which the pain medicine is self-dosed) can give a clue as to the patient's pain.

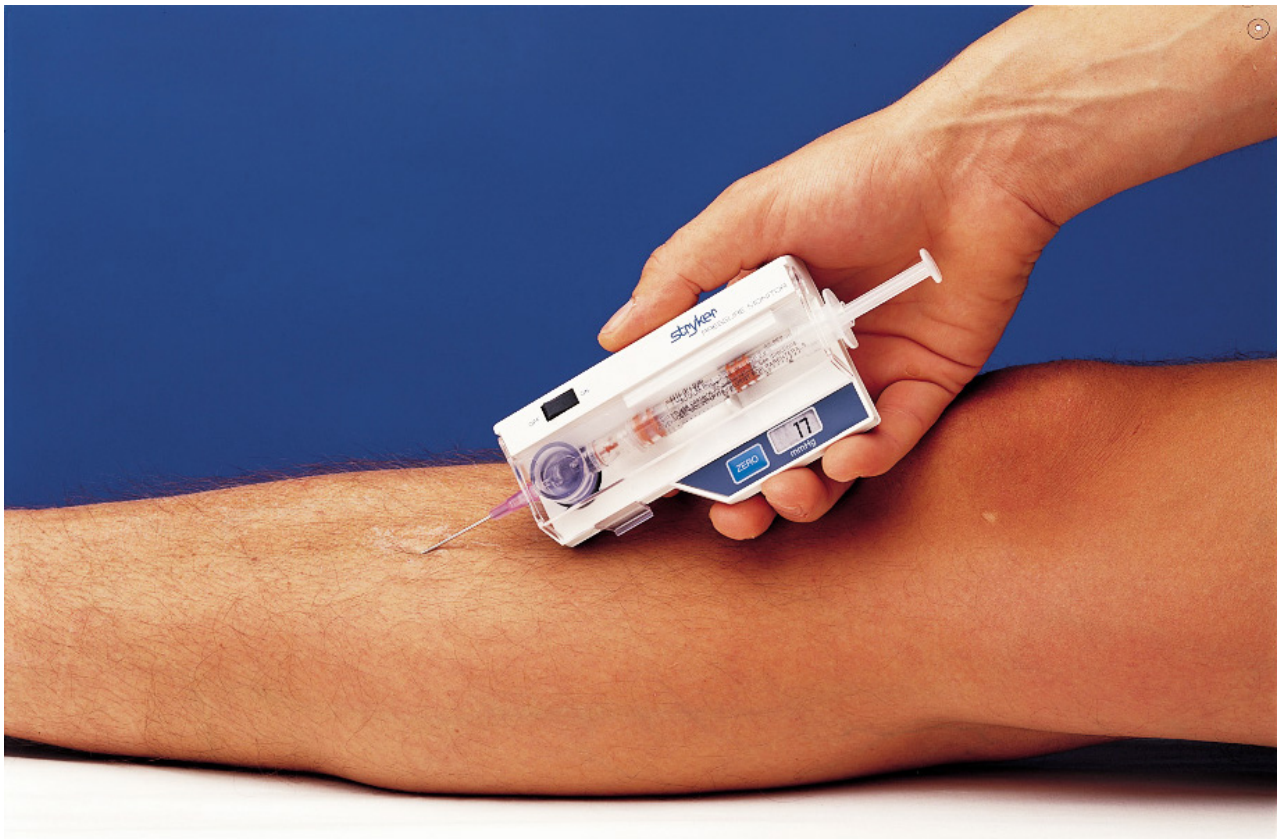
In more advanced cases, symptoms may also include decreased sensation, pale skin, and weakness of the affected area.

Physical exam will reveal tensely swollen and shiny skin, and pain when the compartment is squeezed.

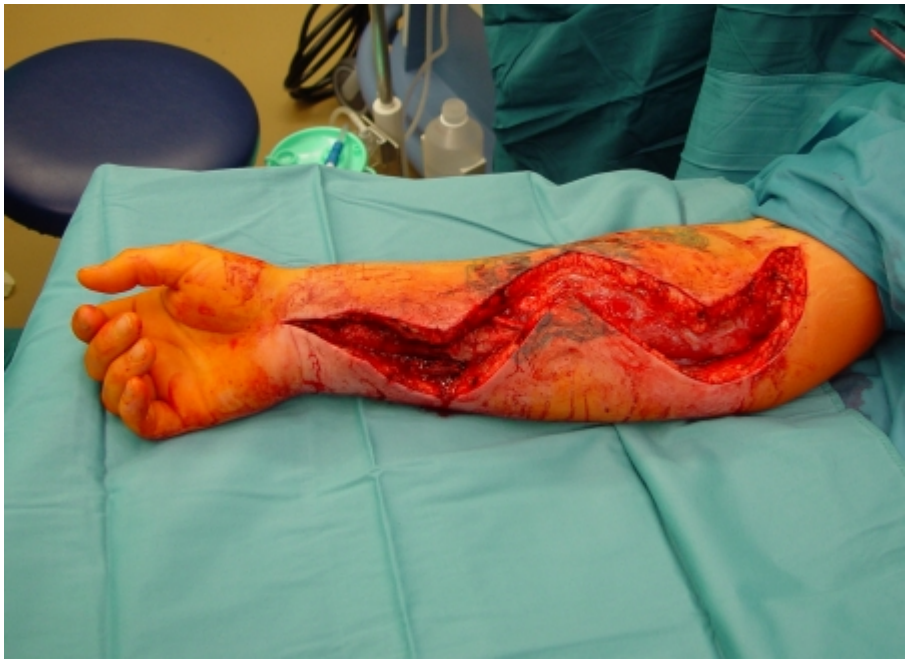


Compartment Syndrome

Confirming the diagnosis of compartment syndrome involves directly measuring the pressure in the compartment, which is done by inserting a needle attached to a pressure meter into the compartment—or treating empirically if needed.



Treatment is a surgical procedure, fasciotomy, where long surgical cuts are made in the fascia to relieve the pressure. The incisions are generally left open to be closed during a second surgery about 48-72 hours later.



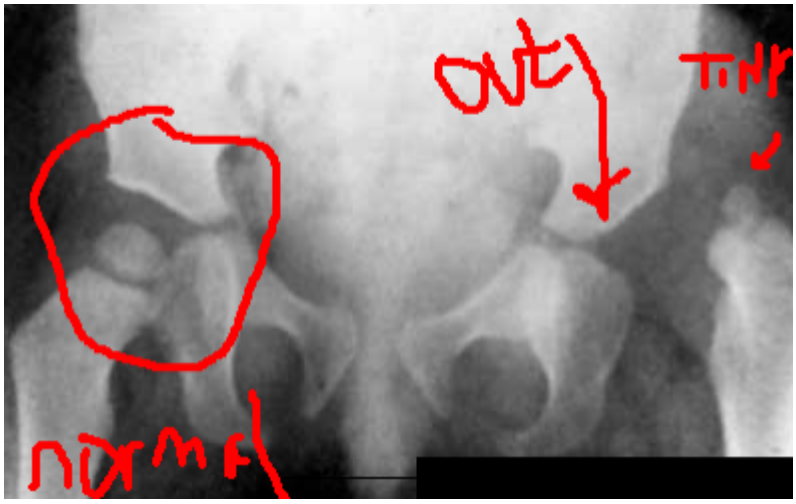
If compartment syndrome is not prevented, permanent nerve injury and loss of muscle function can result and in severe cases amputation may be required. Performing a fasciotomy can potentially increase the risk of infection but overall the risk of not operating is considerably higher…

What is Club foot | Penn Med Self Study Questions | What is Developmental Dysplasia of the Hip

41 What is Developmental Dysplasia of the Hip

What is Developmental Dysplasia of the Hip? & How is Developmental Dysplasia of the Hip diagnosed in the neonate and why is it critical to detect this, if present, as soon as possible? How would it be treated?

It may be better to think of this condition in terms of its old name "congenital dislocation of the hip" (CDH): the baby is born with the hip out of the socket or unstable.



Being out of socket is not good, of course, yet not so much for present function -~~the baby is not walking on it~~ --but because for the hip joint to grow properly, the (mostly cartilage) head has to be in the socket. If the head is not in the socket, it will be grow to be mal-formed ("dysplastic"). Prompt detection and expeditious treatment allows the hip to remodel and form properly.

DDH can be detected on exam – there are a variety of physical exam maneuvers that can be used for diagnosis, including the Ortolani reduction maneuver (abduction and elevation to feel for reduction) and the Barlow provocation test (adduction/posterior pressure to feel for dislocation)---but in high risk patients (Breech position, female gender, first born children, and a positive family history are risk factors) ultrasound is used.

The goal of early treatment is to maintain reduction of the hip to provide the proper environment for the development of the femoral head and acetabulum, which requires that the cartilaginous surface of the

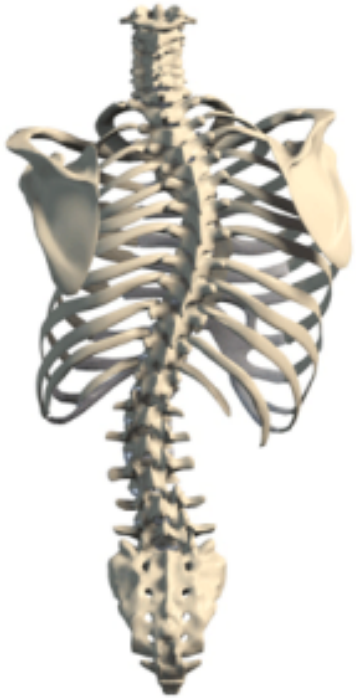
femoral head be in contact with the cartilaginous floor of the acetabulum
Abduction splinting in a Pavlik harness (or even with double diapers for low tech areas) before 6 months of age can usually achieve and maintain hip reduction.
If found late, the hip needs to be reduced, perhaps surgically. Note: even though this put the hip in a "normal" position, it is not "normal" for this baby, and therefore the REDUCTION can damage the blood supply and cause AVN. Untreated DDH should not have AVN
The greater the delay in treatment, the harder it is to treat.

	What is compartment syndrome	Penn Med Self Study Questions	What is idiopathic scoliosis	

42 What is idiopathic scoliosis

What is idiopathic scoliosis and what is the relationship between idiopathic scoliosis to back pain?

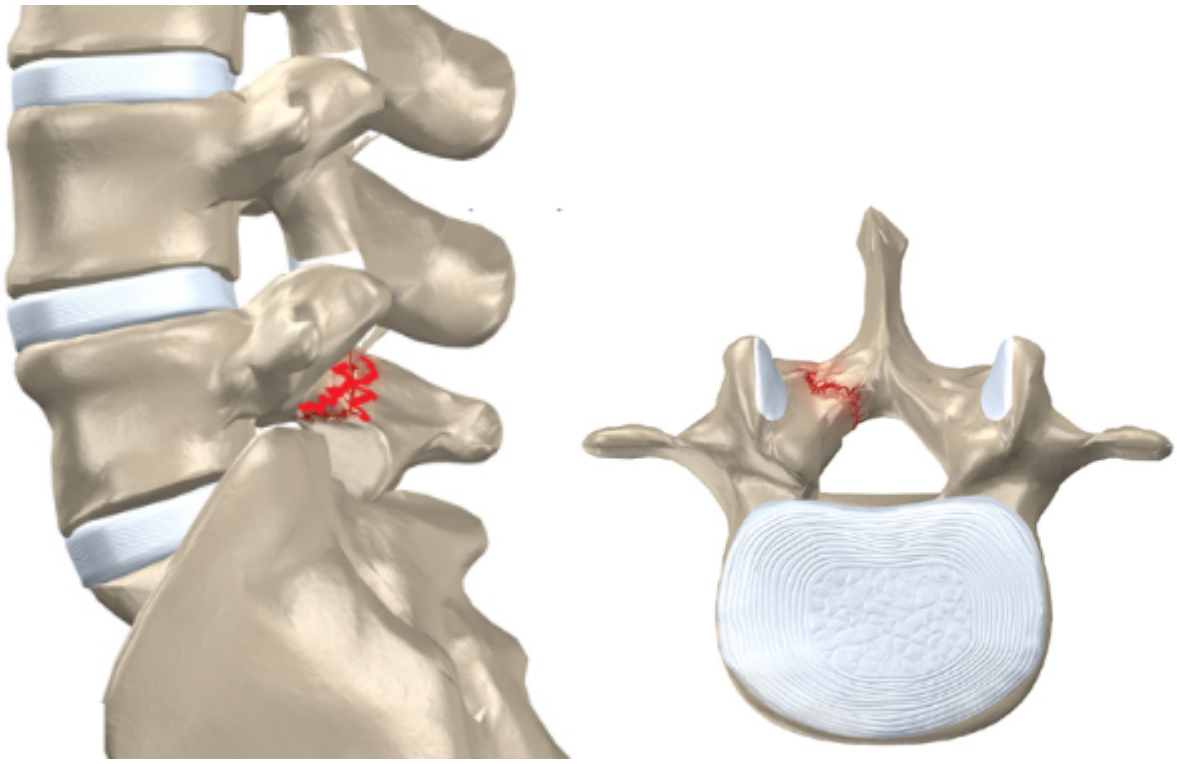
Scoliosis is a lateral curvature of the spine that is usually accompanied by rotation. Idiopathic scoliosis does not have a clear causal agent --" Idiopathic" = "we don't know why you have it".



Labeling a condition idiopathic is a process of exclusion, ie excluding all of the known causes . Adolescents with idiopathic scoliosis ("adolescent type") may present in several ways. In some patients, scoliosis is incidentally found on physical examination. Others have complaints related to the deformity that is caused, such as asymmetry of the shoulders, flank creases, hips, scapulae, or breasts. Still others present because an abnormality was noted during a scoliosis screening at school.

Most patients with adolescent idiopathic scoliosis have little, if any, functional limitation or pain. In practice, the presence of scoliosis not be consider an adequate explanation for acute or severe back pain---at least in the sense that if somebody presents with idiopathic scoliosis and pain, the clinician must not simply attribute the pain to the idiopathic scoliosis.

one not that rare cause of pain that is worth knowing (at least that it exists) is **Spondylolysis**: a stress fracture in one of the pars in the vertebrae



Less common causes include tumor, infection, and in patients with sickle cell disease, sickle crisis.

What is Developmental Dysplasia of the Hip | Penn Med Self Study Questions | What is neurogenic claudication

43 What is neurogenic claudication

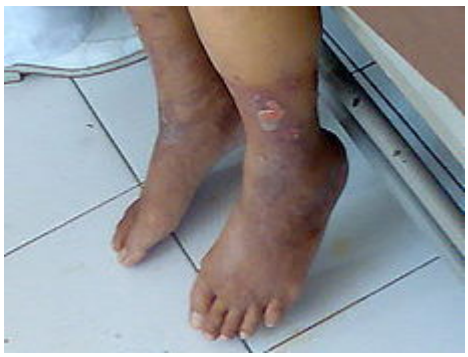
What is neurogenic claudication (contrasted with vascular claudication)?

Basics: nerves don't like pressure. Sit like this too long and bad things happen to your common peroneal nerve

Next: the word "Claudication" (Latin: limp) refers to painful cramping and/or weakness. (The name "Claude" means "lame one"; "Clawed" means "scratched one")

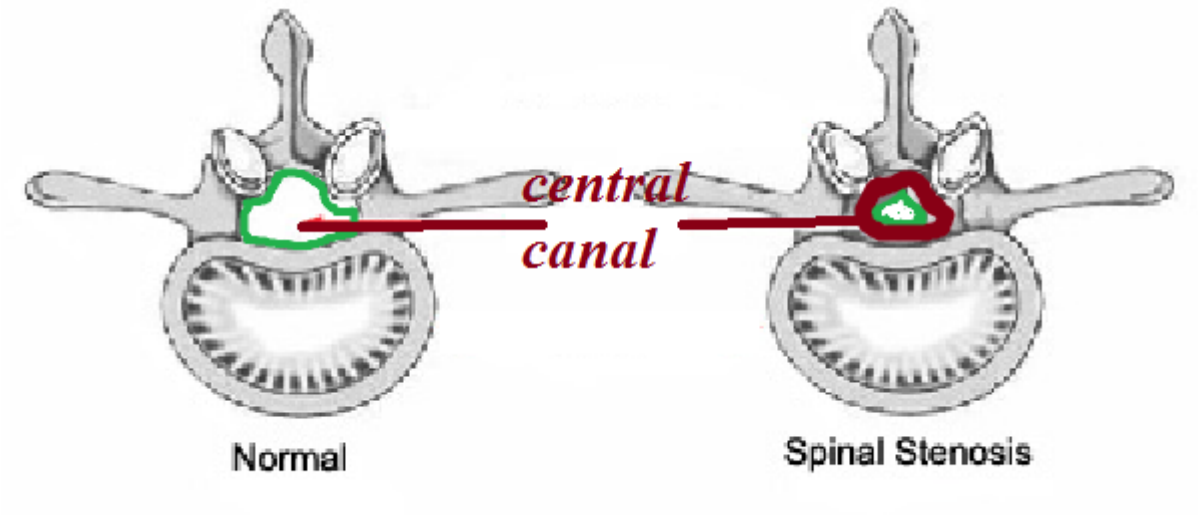
It is important to differentiate neurogenic from vascular claudication. The term "neurogenic" refers to the fact that the problem originates from a problem involving the nerves, but the common theme is ischemia: in classic claudication, the muscles are ischemic; in neurogenic, it is the nerves.

Classic/vascular claudication comes from poor blood supply to the legs



Neurogenic claudication is caused by spinal stenosis (itself caused by bone overgrowth, disc protrusion, facet arthritis, or soft tissue proliferation)

As shown, with spinal stenosis the central canal space is much reduced (putting pressure on the nerves)



Neurogenic claudication can cause bilateral or unilateral lateral calf, buttock, or thigh discomfort, pain, and/or weakness. In some patients, it is precipitated by walking or prolonged standing. The pain is typically relieved by flexion of the waist. The cause is believed to be ischemia of the lumbosacral nerve roots secondary to compression from structures such as hypertrophied facets, ligamentum flavum, bone spurs, scar tissue, and bulging or herniated discs.

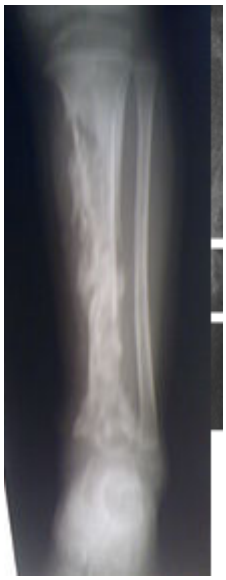
Vascular claudication is due to a circulatory problem. Bad arterial flow leads to ischemia of the calf muscles. Angiography is definitive and measuring the ABI (the Ankle: Brachial Index), ie the ratio of the leg and arm blood pressures) is a good screening measure.
Neurogenic claudication can be differentiated from vascular claudication, by the following features:

- Neurogenic pain is more proximal,
- Neurogenic pain not always uniformly present
- Neurogenic pain can be present at rest
- Neurogenic pain is not dose dependent (ie blocks walked) and
- may be relieved by postural changes (leaning forward to make more space in the spine---eg bike riding is fine whereas walking is painful).

Basically: vascular claudication causes 'dose dependent calf pain'---walk more, hurt more; stop, feel better. All the time, every time.

What is idiopathic scoliosis | Penn Med Self Study Questions | What is osteomyelitis

44 What is osteomyelitis



Osteomyeltis is an infection of bone. Osteomyelitis can be caused by hematogenous spread, contiguous spread from adjacent soft tissues or joints, or direct inoculation (ie: during trauma or surgery)

In addition to bacteria, the focus of osteomyelitis contains dead bone, local inflammation and granulation tissue.

Microbiology most often reveals *Staph aureus* infection, although *Staph epidermis* and gram negative rods are implicated in a minority of cases.

Bone is normally resistant to infection. In order to overcome the normal immune system, bacteria must be present in large number (high inoculum), be able to adhere to the bone surface or bone ischemia must be present to inhibit to a local immune effort. Bone trauma, especially open fracture (thus exposing bone to environmental pathogens), establishes these conditions necessary for chronic infection. The site is most frequently the tibia, as this is the most common site of open fractures. (Non-union of the fractured bone, possibly associated with recurrent infection, is a major complication.)

Typical symptoms include local bone pain, erythema, swelling as well as systemic symptoms such as fever. A classic syndrome of cyclic pain correlating with the progression of bone necrosis has been described, although it is not a sensitive finding.

On exam, signs of local infection may be seen, possibly including small recurrent sinus tracts draining to the skin (actually communicating with underlying infected bone).
Abscesses form within the necrotic bone, expanding contiguously into bone and soft tissue, Sinus tracts may develop, which can lead to the skin and be seen as superficial orifices on exam.

Bone grows over the infection, producing a "sequestrum"--- the island of bone shown below. The infected bone effectively gets walled off from the circulation.

!worddav105cfd12dc27921bf7d46f782bf47e3a.png|align=left!The sequestering of the sequestrum is a positive adaptation in evolution: it keeps that bad segregated from the good. Evolution did not anticipate that

we would have medicine to deliver, and today this wall-it-off strategy is a problem. Antibiotics can't reach their target. As such, surgical debridement is often needed.

Poor nutrition and excess alcohol intake are thought to predispose at-risk patients to developing post-traumatic osteomyelitis. Additionally, patients with diabetes may develop vascular insufficiency, which would also put them at higher risk for developing osteomyelitis in the right setting.

Imaging: Plain films may show characteristic changes after 10-14 days (at which point necrotic changes, including the involucrum may be evident); three phase bone scan is high sensitivity (>90%) but low specificity (<10%); radiolabeled leukocyte imaging is the best nuclear medicine choice for non-vertebral osteo. It too has high sensitivity but higher specificity. MRI shows decreased intensity of marrow on T1 imaging at foci of infection. A bright signal on T2 may be seen, representing granulation tissue.

WBC, ESR, CRP are all nonspecific markers of inflammation or infection. But they may be normal in chronic osteomyelitis. In other words, not much help. Cultures of the debrided tissue are helpful in identifying organisms.

It is of course better to prevent (rather than treat) acute osteomyelitis; and it is better to treat before the bone dies and there is progression to "chronic" disease. Surgical debridement of the involved bone and soft tissue (debrided until normal bleeding is observed) is definitive. However, beyond debridement, the surgeon must decide how to stabilize the bone and fill the resulting "dead space." Some form of rigid fixation had been shown in animals to improve bone union following debridement, and there is evidence supporting both internal and external methods of fixation. At the time of debridement, antibiotic-containing beads may be implanted in this space for up to 30 days (and subsequently removed).

Systemic antibiotics are used as adjuncts to surgery to help prevent infection in the healing of the operative wound and to prevent spread. The antibiotic used is based on culture results.

What is neurogenic claudication | Penn Med Self Study Questions | What is osteonecrosis

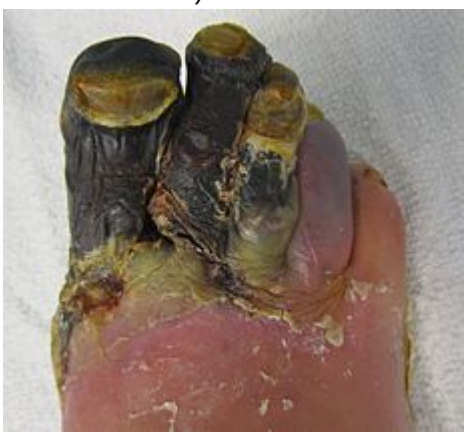
45 What is osteonecrosis

What is osteonecrosis (also known as "avascular necrosis" or "AVN")? How does hip dislocation lead to avascular necrosis? How does avascular necrosis lead to end stage arthritis?

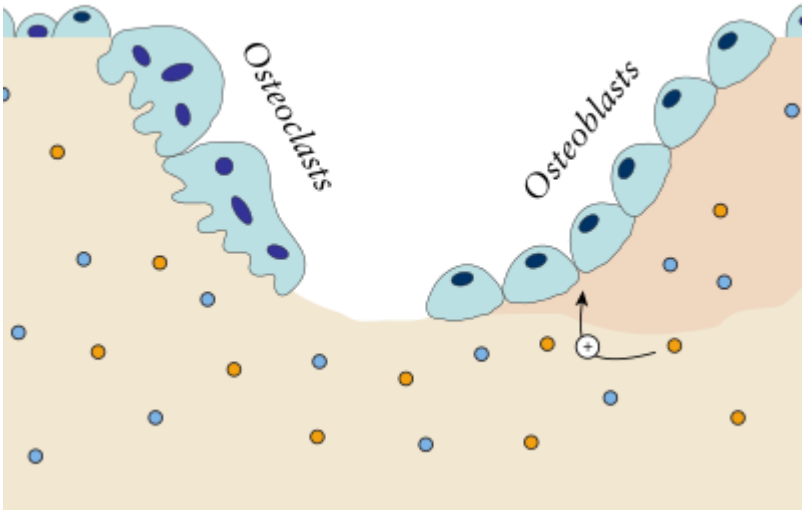
Avascular necrosis is the death of bone, secondary to loss of blood supply and resultant ischemia as seen on the right.

Hip dislocation can cause disruption of the blood supply to the head and thus lead to AVN
Recall that **bone is alive**. Hence:

- If bone (like all living tissue) is deprived of its blood supply, ie gets ischemic, it will die. (A principle illustrated below)



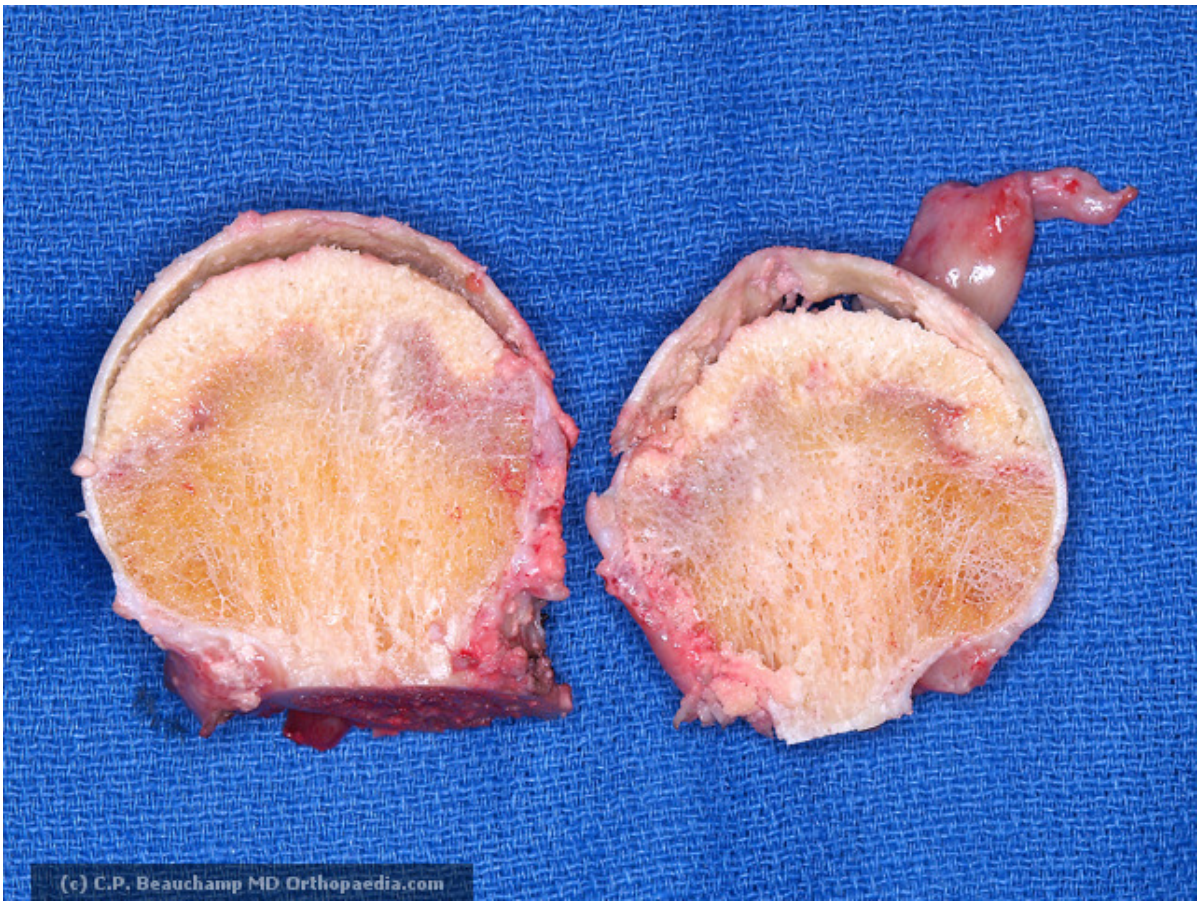
- If bone dies, it does not remodel (you remember this...).



- If bone does not remodel, micro-damage does not get repaired just as a ship that does not get painted with rust.

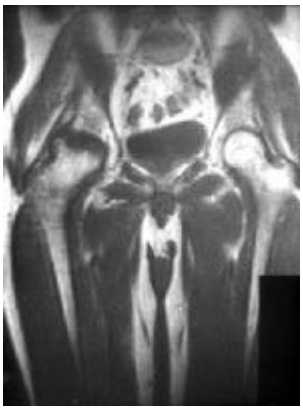
- If enough damage accumulates, the bone loses its normal material properties (strength and compliance). It will break, not bend

- Specifically, the sub-chondral bone will collapse, as shown here.The bone is literally not supporting the surface, which then too will collapse



Osteonecrosis of the Femoral Head with Subchondral Collapse

- If the sub-chondral bone collapses, the joint surface of course becomes irregular and no longer smooth



- If the joint surface is not smooth it will damage its rival surface.

  This is not a histology slide–it's a picture of sandpaper. The effect of a rough surface on a smoother one is to make the smooth rough

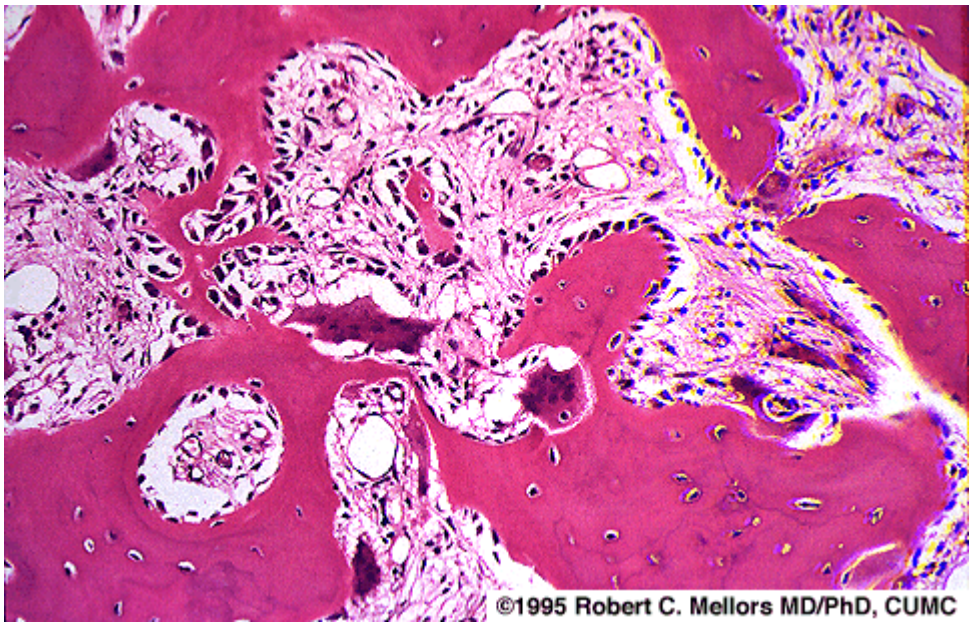
Choosing Sandpaper

Note: although you can detect muscle fairly quickly (within hours after an infarct, certainly), acutely dead bone (within hours after an infarct, say) would look normal if examined histologically. That is because for bone, the blood supply primarily serves maintenance, not sustenance. By (poor) analogy: if you can't get water to drink (sustenance) in three days, you die; whereas if you can't get water to shower (maintenance) in three days, you don't start stinking immediately, but stink you will eventually.

	What is osteomyelitis	Penn Med Self Study Questions	What is Pagets disease of the bone	

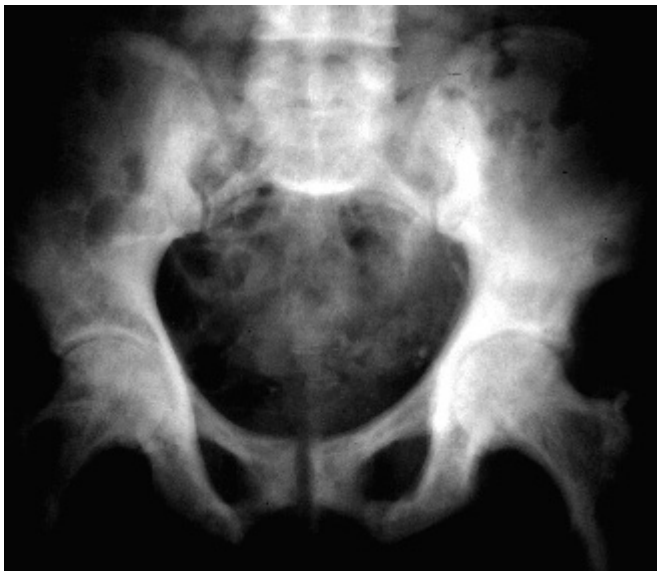
46 What is Pagets disease of the bone

Paget's Disease of the Bone is a disorder where resorption of bone by hyperactive osteoclasts outpaces the ability of osteoblasts to keep up (ie, laying down strong organized bone).

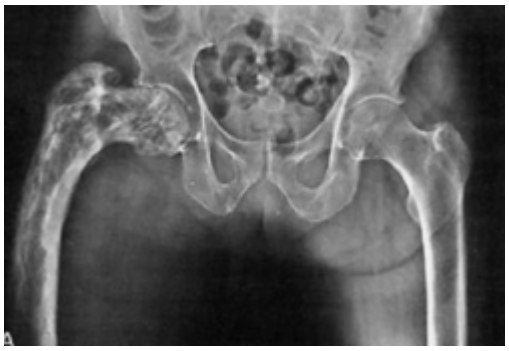


This results in focal lytic lesions affecting one or many bones, as well as areas of proliferation of soft, disorganized bone.

This xray of the pelvis shows earlier signs of Paget's disease: thickening of the femoral neck and diffuse sclerosis of bone.



This xray shows more advance deformity:

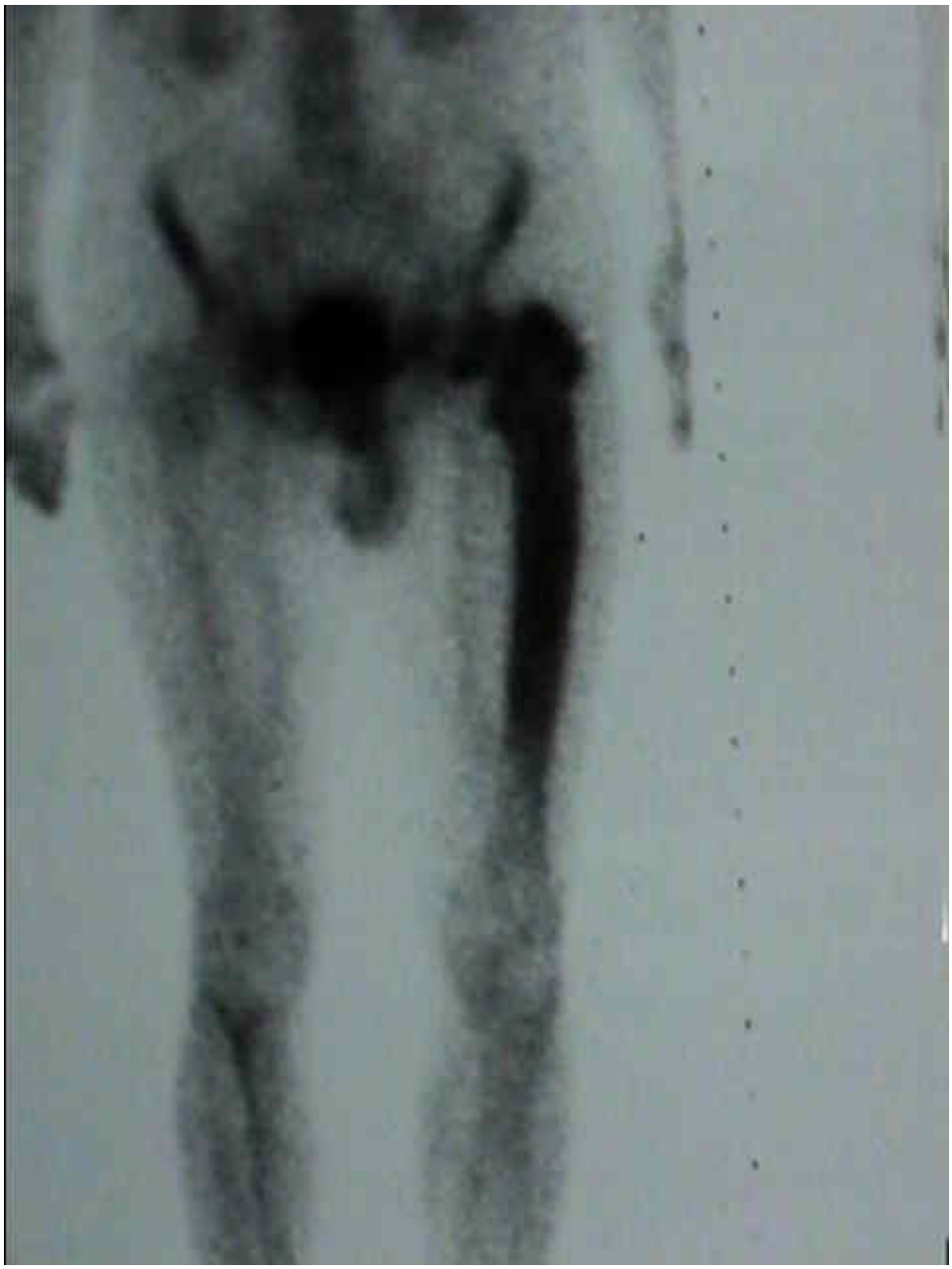


X-ray of Paget disease of proximal femur

The disease runs in families and though the etiology is not entirely clear, it is commonly thought to have both a genetic and a viral component. It is a disease of older patients; those younger than 40 years are rarely affected, with incidence increasing thereafter with age. It is also more common among people of European descent and has a slight predilection for males over females.

Aching bone pain is the most common presenting symptom, but patients are often asymptomatic early in disease, and frequently picked up by incidental radiographic findings or due to blood work that reveals an elevated alkaline phosphatase.

A bone scan will be "hot" because of the increased remodeling activity



Mr. Smith is right to be happy that cancer hasn't been found. (Paget's Disease of the Nipple, a rare form of breast cancer, is unrelated except by name.) Nonetheless, Paget's is associated with increased risk of bone malignancy, affecting about 1% of people with disease and with osteosarcomas of the pelvis, femur, humerus and skull seen most frequently.

Additionally, he should not be dancing for joy (or anything else for that matter!) too aggressively because in addition to accelerated OA, another complication of Paget's is increased fracture risk.

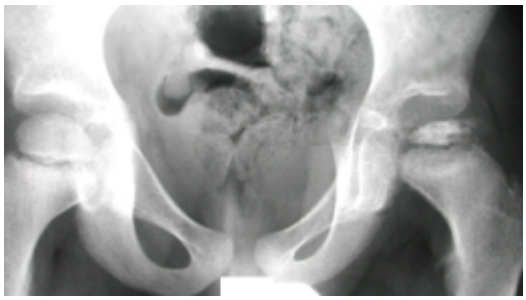
Fortunately for him, remission (understood as normalizing of x-ray findings, serum alk phos and relief of pain–not "cure") can be achieved in more than 90% of patients via IV bisphosphonate therapy. He'll need good follow up and continued monitoring for recurrence or malignancy going forward.

What is osteonecrosis | Penn Med Self Study Questions | What is Perthes

47 What is Perthes

What is Perthes (rhymes with Her'-Sneeze) Disease?

Perthes is AVN of the femoral epiphysis (without a slip) in kids.



The typical patient is a 10 year old boy with a limp.

The cause is not known. The treatment is supportive---non weight bearing to prevent further collapse, maybe, and hope that the epiphysis revascularizes (which can happen).

It is often unilateral.

The older the patient, the worse the prognosis (consider: AVN with collapse in an adult has no cure; the closer one gets to being "adult" the more the disease behaves like the adult form of AVN.)

⇦	What is Pagets disease of the bone	Penn Med Self Study Questions	What is rotator cuff tendinitis	⇨

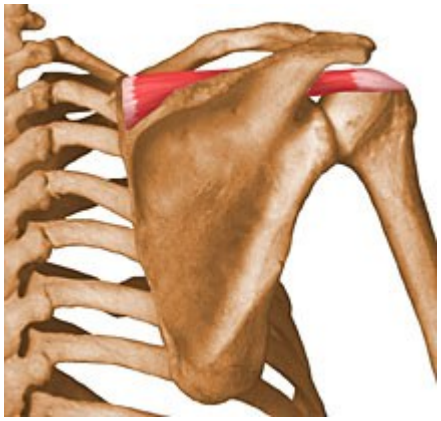
48 What is rotator cuff tendinitis

What is rotator cuff tendinitis? What are the consequences of labeling it (perhaps incorrectly) as an "—itis"? What are the consequences of labeling rotator cuff tendinitis as "impingement syndrome"?

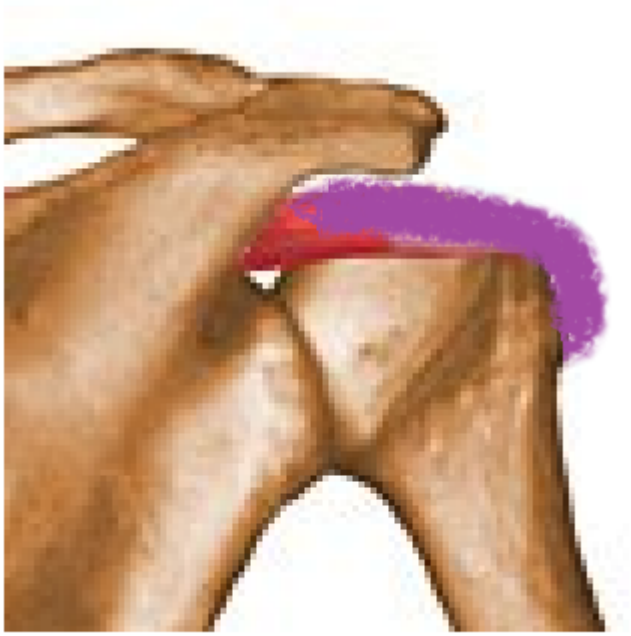
Rotator cuff tendinitis is not an inflammatory condition (as "itis" would suggest); it is a degenerative wearing out of the tendon, owing to repetitive use, poor blood supply and aging. It should be treated with an anti-inflammatory only to the extent that the anti-inflammatory medicine is also a pain reliever.

The supraspinatus tendon, as you see, has to make a bit of a turn before inserting on the humerus, which might impair perfusion to the insertion site

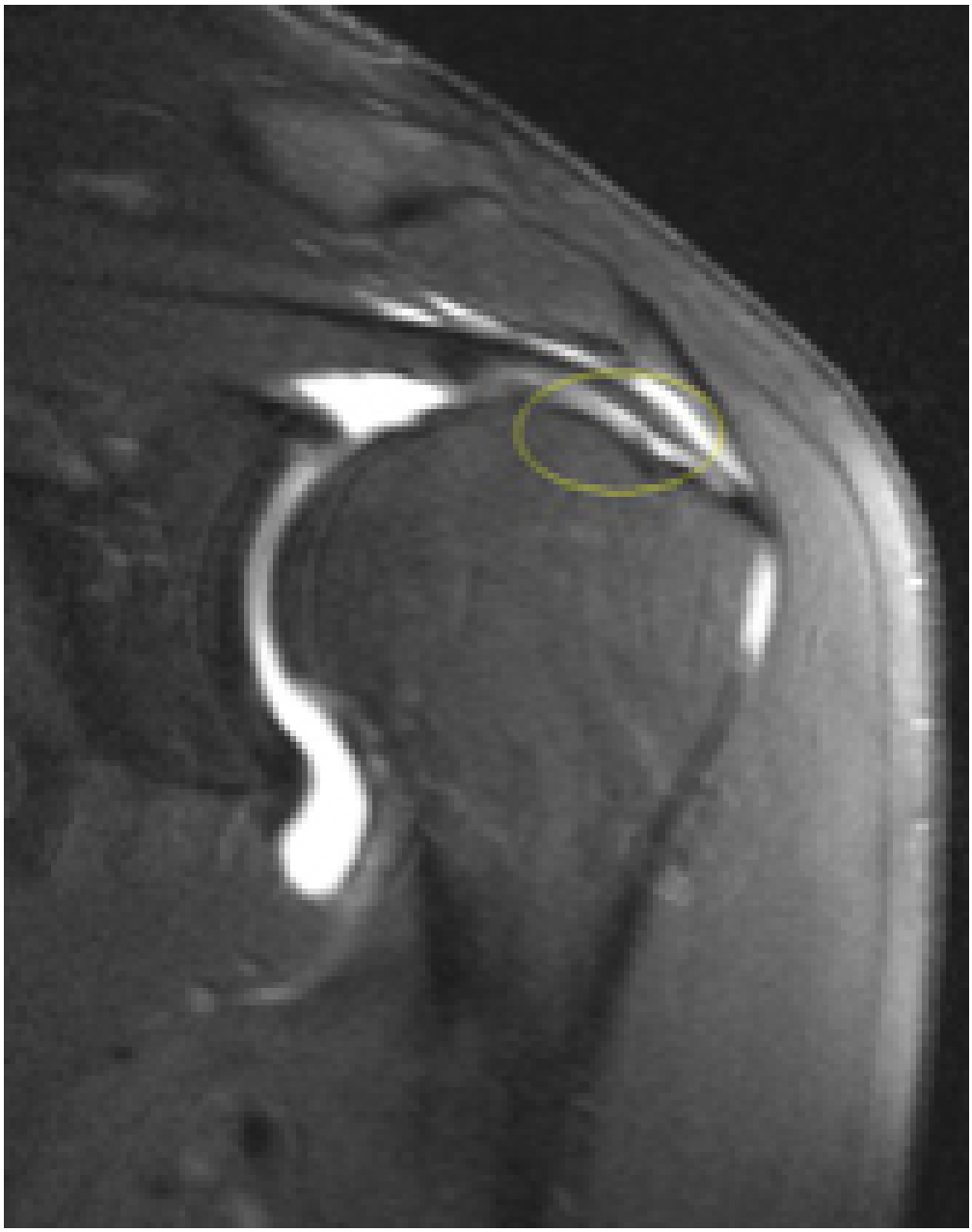
NORMAL SUPRASPINATUS ANATOMY



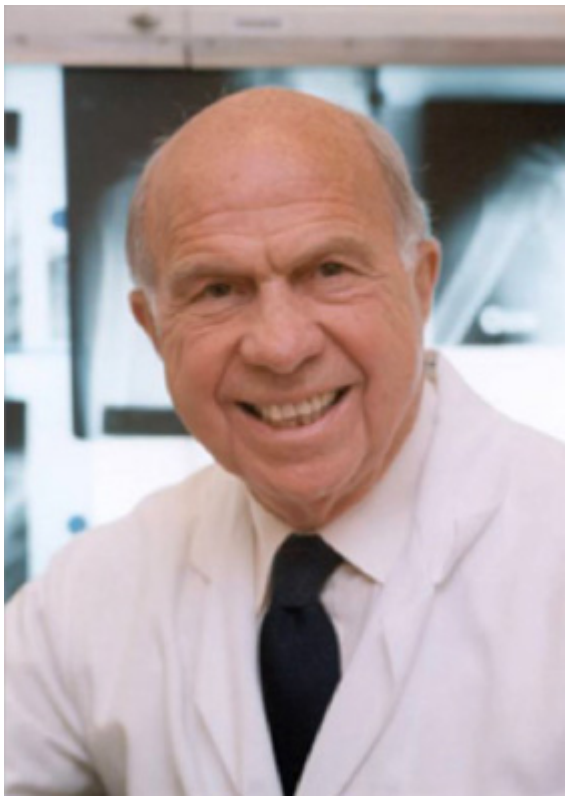
close up of the turn:



MRI VIEW (ok, I exaggerated the turn, but just a bit: it's NOT a straight shot)

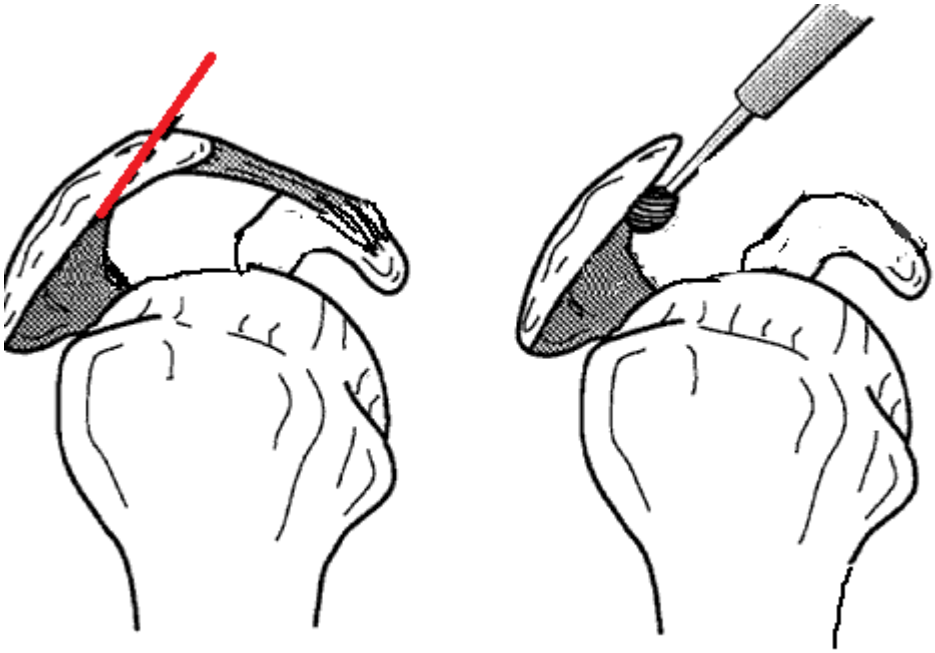


There is no proof that anything is impinging (pressing) on the cuff



(C Neer, Penn Med '42, above, came up with this theory sans evidence and it stuck. By contrasy, a study found that there is no supporting evidence.

Accordingly, there is no evidence that we should surgically remove any "impinging" structures (though acromioplasty, the removal of "impinging" bone the acromion (and shown below), is among the most common operations we do) .



More troubling, perhaps, is that this is a question amenable to a randomized trial, yet such a study has not been done. Case series have shown that patients with no bone removal do as well as historical controls who did have bone resected.

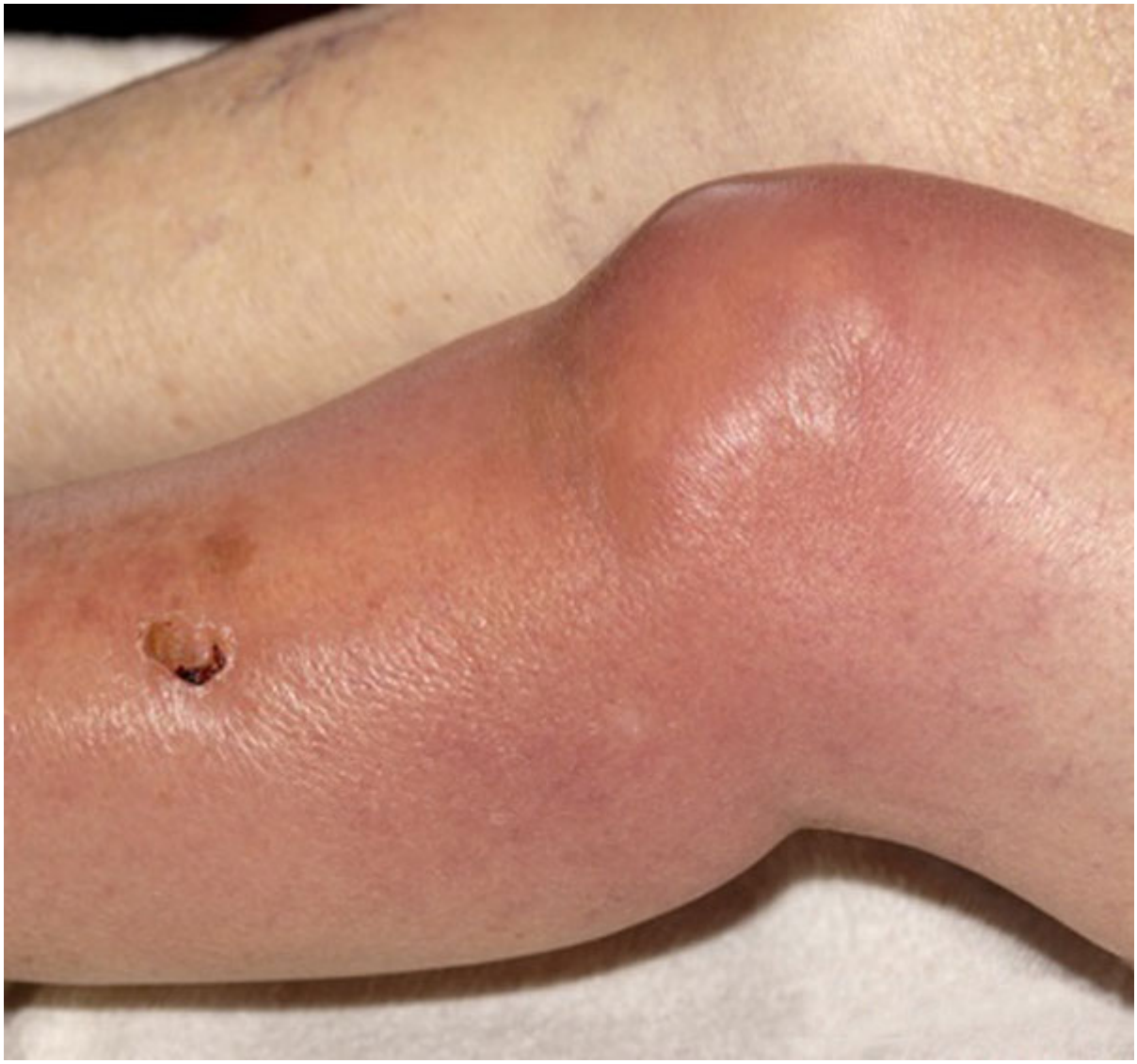
The interested student is pointed to the best (and only; hence worst too) discussion of orthopaedic surgery semiotics and the pernicious effect of mis-naming things

What is Perthes | Penn Med Self Study Questions | What is septic arthritis

49 What is septic arthritis

What is septic arthritis? What are its causes?

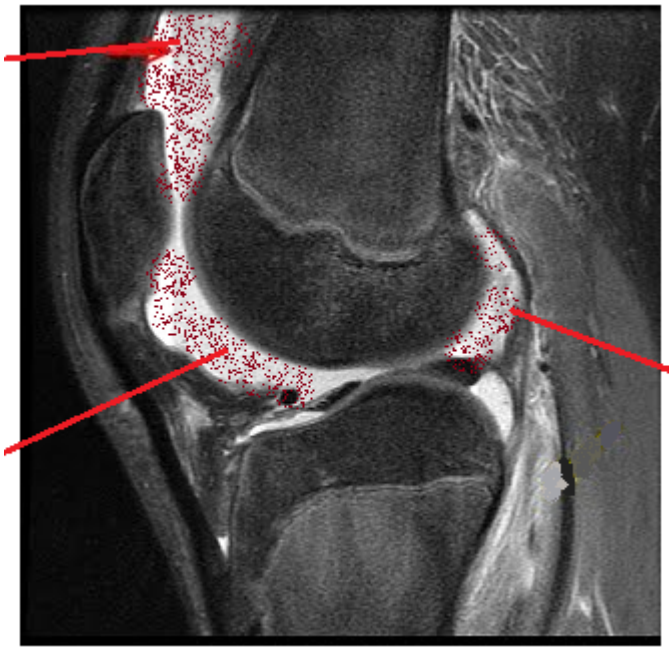
Septic arthritis is a bacterial (and less commonly fungal or mycobacterial) infection of a joint. It can be caused by hematogenous spread (most common), direct inoculation (ie: trauma, during surgery) or contiguous spread from infected periarticular tissue.



Here cellulitis near the knee (from a puncture wound originally) is at risk of spreading into the joint

The most common site is the knee, but the wrist and hip, among others, can be affected.

Note the large space for infected joint fluid in the knee:



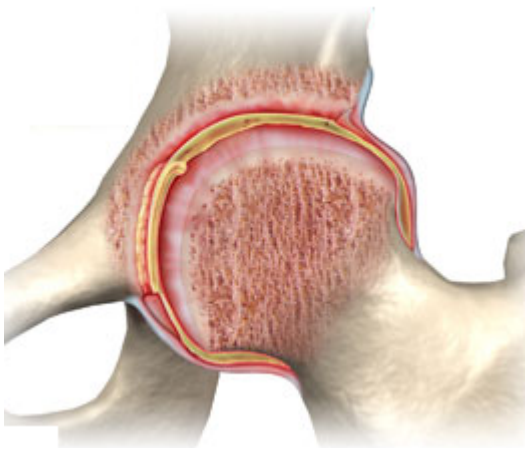
Soft tissue infections of the tendons and discs are considered separately. Also, the condition in pediatrics has a different pattern of presentation (eg the hip is often the site of infection in infants) and predicted course.

Most cases of acute septic arthritis are caused by staphylococcus. Certain sites (foot puncture wounds = Pseudomonas) and certain patients (those with sickle cell disease = salmonella) are susceptible to other characteristic organisms.

Infections can be iatrogenic (as suggested below by a glove-less injector, who probably did not wash his hands either)

Septic arthritis may not cause systemic effects. The patient will have pain especially on motion of the joint

Note that the hip has so little room for joint fluid build up, it may be very painful even without motion



The diagnosis is made by aspirating the fluid and sending it to the lab for cell count and culture / gram stain

Elevated risk for septic arthritis is seen in patients with joint implants, known infection elsewhere, a history of drug abuse, immune suppression and chronic diseases (such as diabetes or rheumatoid arthritis.

It is possible to get an autoimmune arthritis after a systemic infection; but in that case the joint space itself is sterile. This condition is termed "reactive arthritis" and is grouped under the rubric of rheumatological, not infectious, diseases.

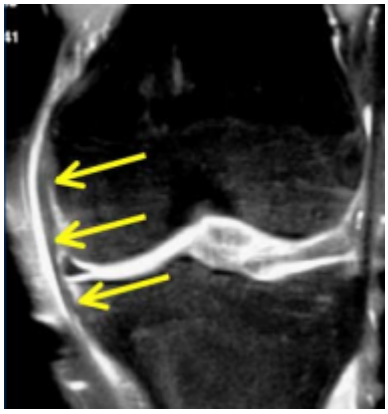
⇦	What is rotator cuff tendinitis	Penn Med Self Study Questions	What is the definitional distinction between grade I, II and III sprains	⇨

50 What is the definitional distinction between grade I, II and III sprains

What is the definitional distinction between grade I, II and III sprains? How would these various grades of injury present distinctly on examination?

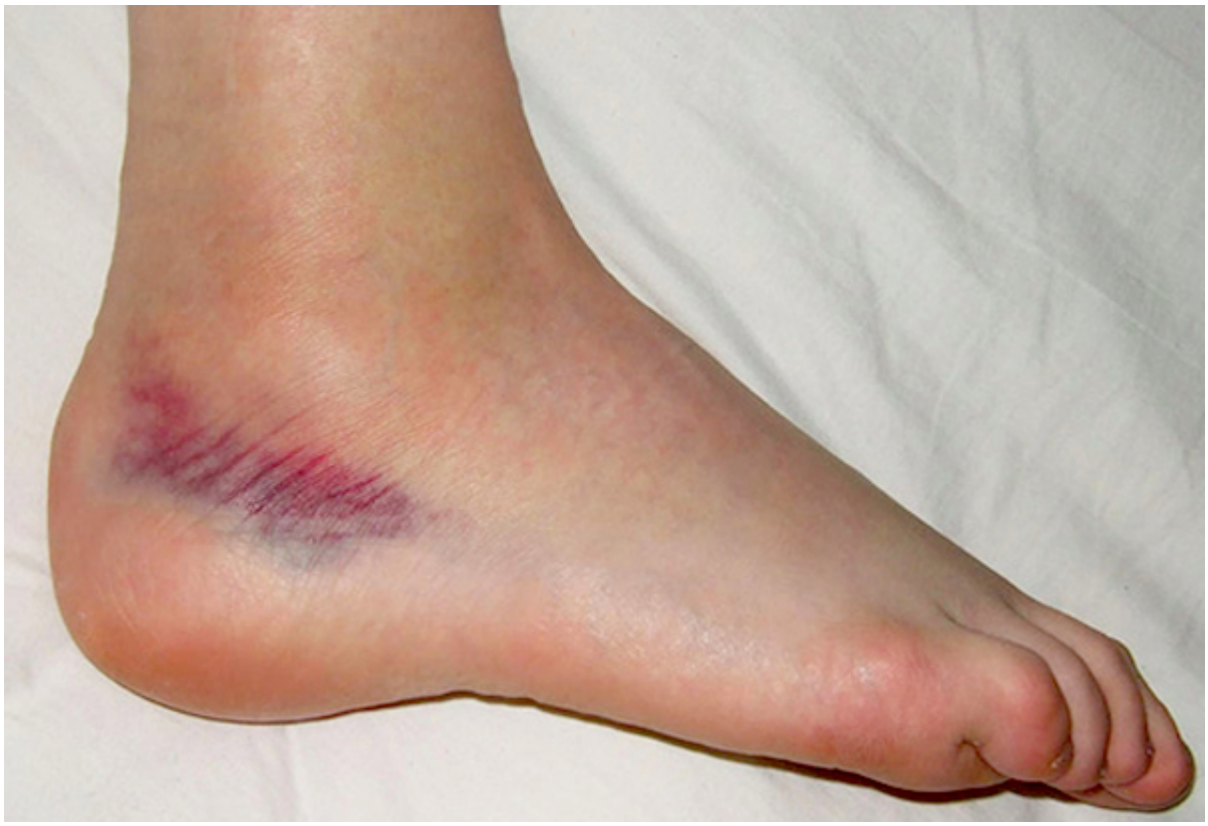
A **grade I sprain** results from mild stretching of a ligament with microscopic tearing only. Patients have mild swelling and tenderness. There is no objective joint instability on examination.

It is often a clinical (subjectively applied) diagnosis: if a patients says "I twisted my ankle and it hurts" and nothing is seen objectively, the diagnosis often applied would be "grade I" sprain. Many such patients have nothing wrong with them, giving rise to the incorrect notion that grade I sprain (when it truly occurs) is completely inconsequential injury.

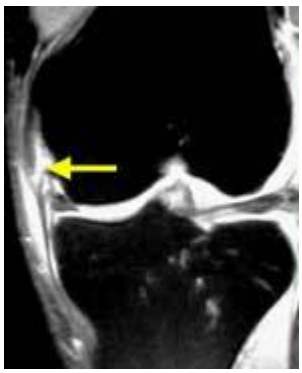
Signal change in the MCL indicates an injury (compare to ITB laterally (which is darker)), but the gross course of the ligament is unchanged 

A **grade II sprain** is a more severe injury involving an incomplete tear/macroscopic stretching of a ligament.

Patients can have moderate pain, swelling, tenderness, and ecchymosis.



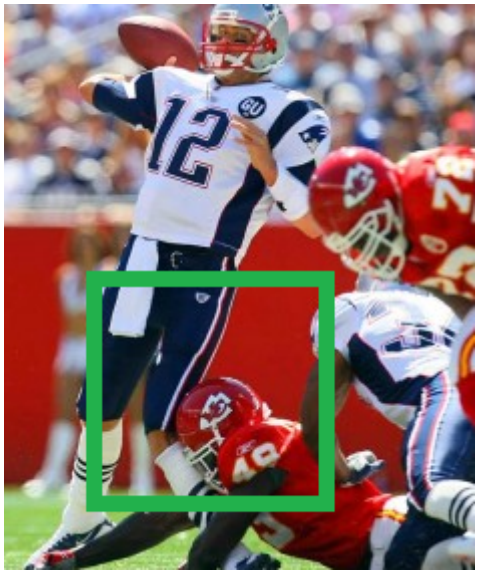
There is mild to moderate joint instability on exam with some restriction of the range of motion and loss of function. If a grade II sprain is found in the leg, weight bearing and ambulation are painful. Here is there is tearing within the ligament, but some fibers are in continuity



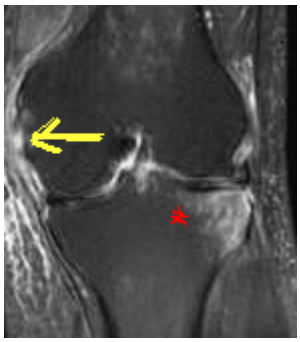
think pulled taffy:

A grade III sprain involves a complete tear of a ligament.

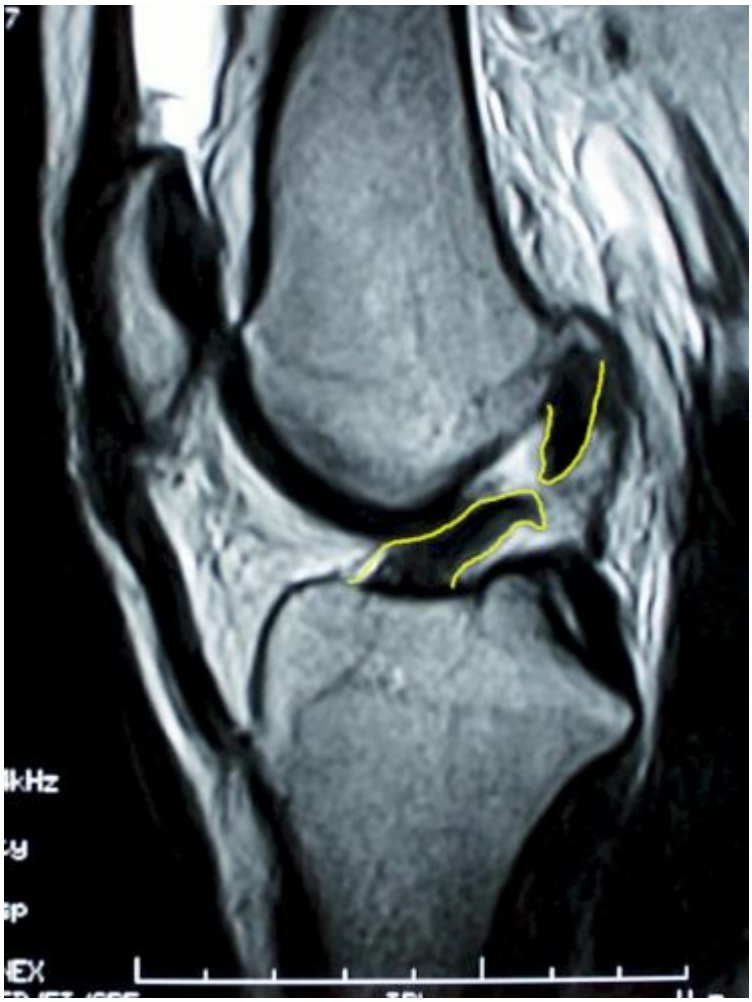
This is often the result of higher energy mechanisms, such as the valgus blow to the knee shown below (likely to injure the MCL)



There is significant laxity perceived by the examiner. Patients might be unable to bear weight or ambulate. *Paradoxically, perhaps, this may hurt less than a grade 2, as once the ligament is torn, it no longer is provoked with every step.*
Here is there is disruption the ligament. Why is there bone edema near the red asterisk ?



Note that a complete tear is the same as a grade III sprain



Here is a RUPTURED anterior cruciate. It is medically correct to call this a grade III sprain – though nobody does....

OTHER POINTS:

There are proprioceptive nerves are within the ligament (informing the brain just how bent the joint is you might say) and therefore even a grade I sprain can cause proprioceptive disruption.

Also, there can be a chondral injury from impaction even with a grade I sprain (that's the edema seen near the red asterisk, above). In all, Grade I injuries may be less benign than they might seem.

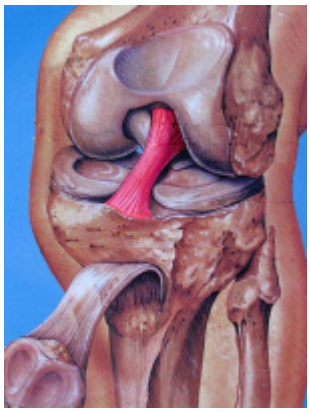
	What is septic arthritis	Penn Med Self Study Questions	What is the function of the Anterior Cruciate Ligament	

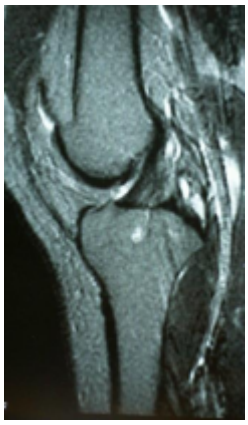
51 What is the function of the Anterior Cruciate Ligament

What is the function of the Anterior Cruciate Ligament (ACL) in the knee? How is the ACL torn? Along those lines, why might it be the case (as we suspect) that skiing-related ACL tears occur disproportionately after 2pm? How is an ACL tear detected on exam?

The main function of the ACL is restraint of anteroposterior translation of the tibia relative to the femur. It also acts as a secondary restraint to tibial rotation and valgus or varus stress.

The ACL courses from the anterior tibia to posterior femur at the knee, as shown:





(This sagittal view points out that the ACL is fairly vertical–not the most advantageous orientation to prevent anterior translation. Yet if the ligament were more horizontal (as I drew it below, with artistic license) the knee would not flex and extend normally)

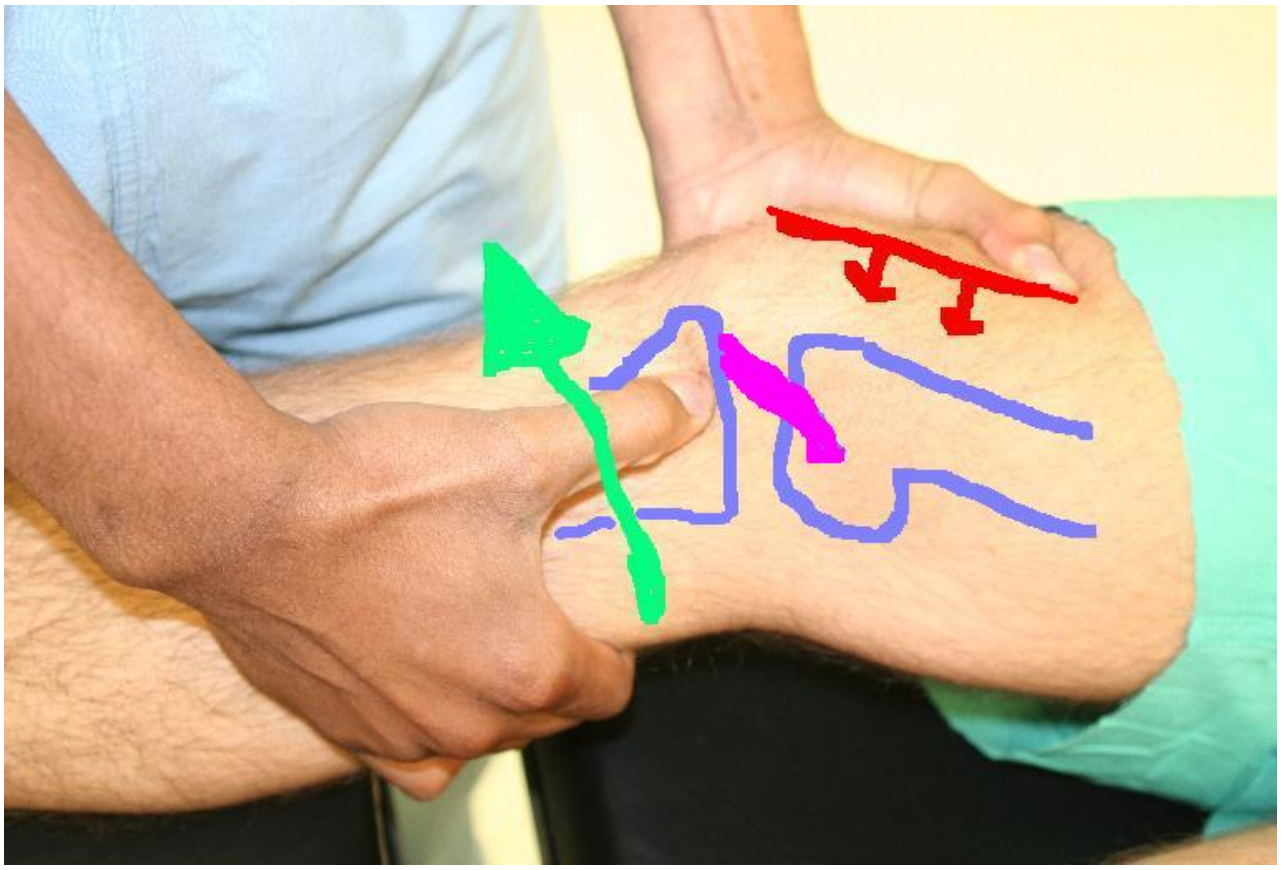
The mechanisms of injury is typically a sudden deceleration or rotational maneuver with a force that sends the tibia one way and femur another (typically because the foot is planted and the body spins).

Not all such forces that exceed the strength of the ligament lead to a tear: often, the secondary restraints (the hamstrings mostly) can help resist a tear. When the secondary restraints are overwhelmed, the ligament can be exposed to forces it cannot bear.

(Why would the hamstrings be overwhelmed? Well, for one thing the knee may be extending at the very second of injury, a phase of motion where the hamstrings do not fire; alternatively, they could be too tired. While skiing, for example, at 10am your still-powerful hamstrings protect the ACL; at 2pm, when the tibia starts to subluxate, the hamstrings just acquiesce and let the bone subluxate, ultimately tearing the ligament.)

An ACL tear is suspected first by history. A "pop" heard by the patient, immediate pain and swelling after a twist are typical features.
To test for an ACL tear on physical exam one can use the anterior drawer and Lachman tests. The Lachman test is the gold standard because it is thought to isolate the ACL and not involve other stabilizing structures.

The Lachman test is performed by p attempting to produce anterior translation of the tibia. An intact ACL limits anterior translation and provides a distinct endpoint. Increased translation compared to the uninjured knee and a vague endpoint suggest ACL injury.



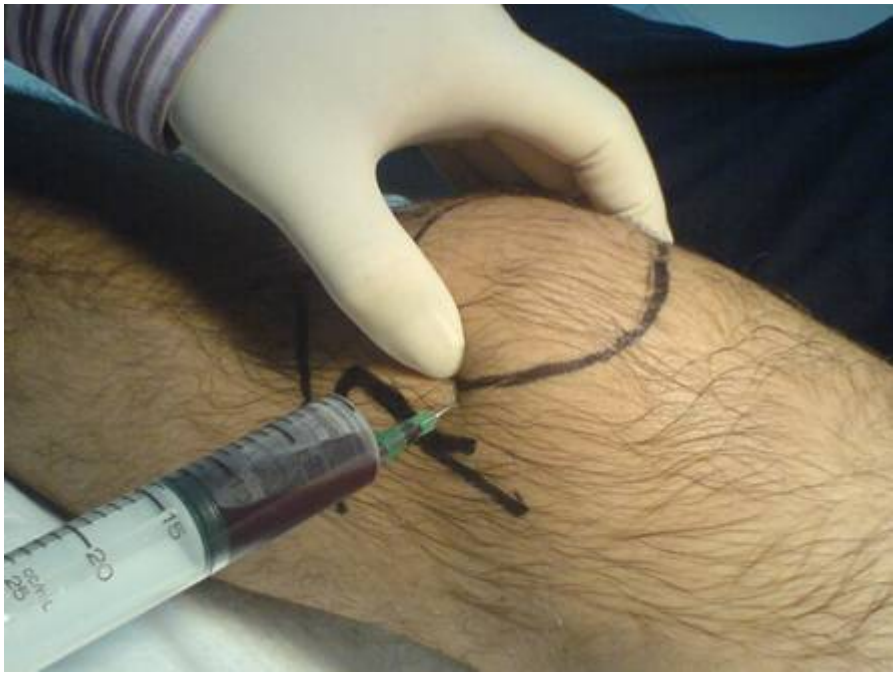
6. The right hand then pulls the tibia foward to place the ACL under tension.

The anterior drawer is performed with the patient lying supine and the knee flexed at 90 degrees. The proximal tibia is gripped with both hands and pulled anteriorly, checking for anterior translation. Often the clinician sits on the foot while performing the test to provide stability. The test is positive if there is anterior translation.

In brief, if there is a good story on history; effusion; what you think is maybe a positive lachman or drawer then get MRI.

NOTE:

Knee aspiration can be a very important step in the initial management:



Here's why:

1. If there is too much fluid you cannot sense laxity–the pressure of the fluid "stabilizes" the knee
2. The pressure of the fluid hurts! It is simply kind and humane to relieve it
3. BLOOD (as seen above) usually indicates a significant injury (MRI worthy to be sure!); if not an ACL then perhaps a chondral fracture, meniscal tear or patellar dislocation.

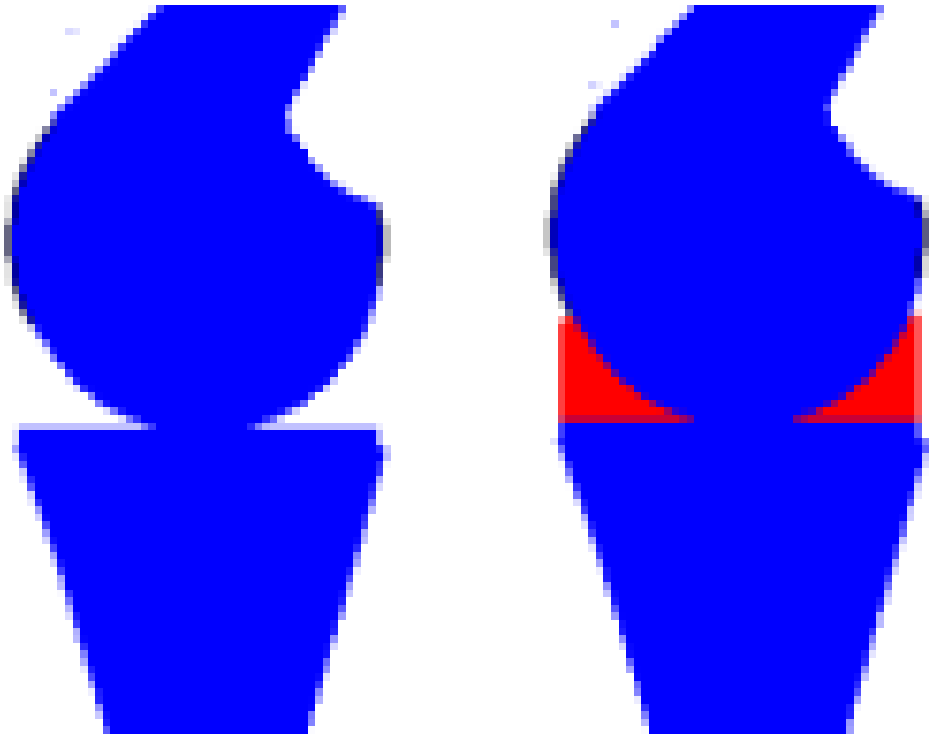
What is the definitional distinction between grade I, II and III sprains	Penn Med Self Study Questions	What is the function of the meniscus in the knee	

52 What is the function of the meniscus in the knee

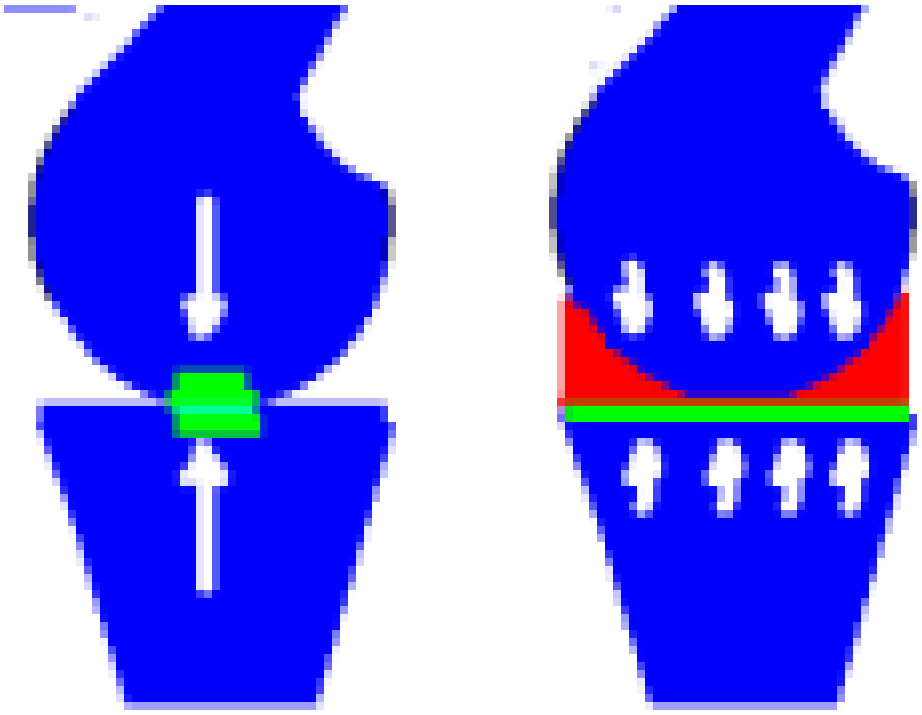
What is the function of the meniscus in the knee? What is the consequence of tearing a meniscus? Why are most symptomatic meniscal tears removed and not repaired?

The medial and lateral meniscus together provide shock absorption, establish a broad base of contact surface and help provide stability to the knee.

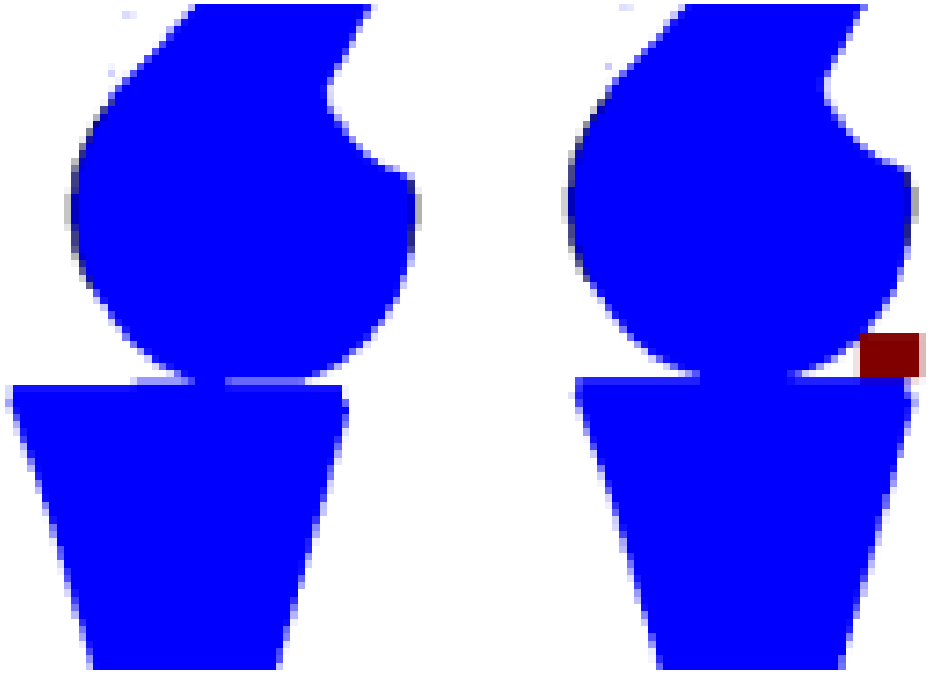
It should be clear, even sans picture, that a wedge of cartilage helps absorb shock:



The meniscus also helps establish a broad base of contact surface. As shown below on the left, without a meniscus there is focal loading and higher pressure at the point of contact



Last, the meniscus is stabilizing – a so called "chock block" effect



Here's how that works:

A lost meniscus (*might)* leads to

- Pain with loading, such as jumping (because of lost shock absorption)
- Less anterior/posterior stability of the knee (because of loss of chock block effect)
- DJD (because lost broad base of contact surface leads to focal load bearing and increased pressure, P = F/A. High pressure stimulates bone formation and in turn a stiff, injury prone subchondral plate.

We remove menisci, in light of the above, only when we have to. And we usually have to remove symptomatic tears, because most tears don't heal. In general, only tears at the periphery have healing potential, as the blood supply enters from the capsule. Also, if the meniscus is macerated, ie torn up, there is no point in repairing it.

The key clinical question (in light of the histological truth that menisci have no pain fibers) is whether the tear is causing the pain, or whether it is just part and parcel of an arthritic picture, or an incidental finding.

	What is the function of the Anterior Cruciate Ligament	Penn Med Self Study Questions	What is the role of body mass vis a vis osteoarthritis

53 What is the role of body mass vis a vis osteoarthritis

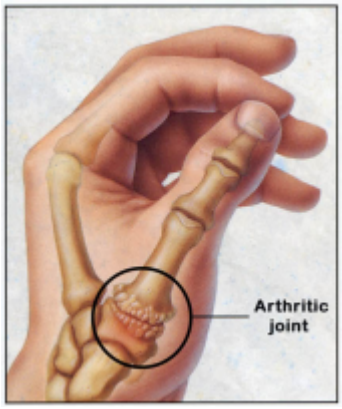
What is the role of body mass vis a vis osteoarthritis? Why might we think that this not be a pure mechanical phenomenon? (hint: Framingham)

Intuitively, excess load should cause excess wear:

Excess body mass increases the load placed on joints which increases stress and accelerates the breakdown of cartilage.

However, OA may not be a pure mechanical phenomenon because being overweight has also been associated with higher rates of hand OA --this suggests that a circulating systemic factor may also play a role.

Basal thumb (C-MC) arthritis is more common among the obese; yet unless these people are doing a lot of handstands, this joint should not be excessively loaded by body mass



What is the function of the meniscus in the knee | Penn Med Self Study Questions | Why do 40 year old men rupture their Achilles tendons

54 Why do 40 year old men rupture their Achilles tendons

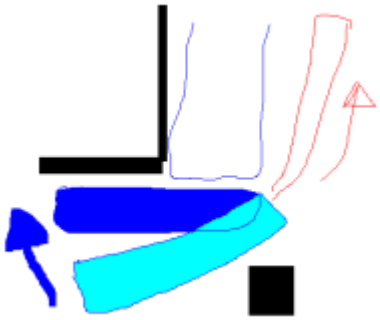
Why do 40 year old men (in particular) rupture their Achilles tendons? What are the biological and mechanical steps leading to tissue failure?

40+ year olds reside in that strange place of a) having accumulated plenty of tissue damage over the years (walking 1 mile a day for 45 years is almost 100,000,000 steps) yet b) have not quite caught on that they are old and decrepit. So they try to do too much. The Achilles tendon not only gets beaten up, it is about as far from the heart as a tendon can get, impeding the blood supply, so wear and tear damage is imperfectly repaired.

So why does the tendon fail? Well, first consider: what does it do? The Achilles powers plantar flexion (left) but also resists dorsiflexion (right) (the light blue signifies the STARTING position) and the blue arrow shows the motion of the foot

The motion of the muscle on the right is called an eccentric contraction---the muscle actually lengthens as it works ("negative" work is how the weight lifters call it) It turns out that ripping the tendon almost always happens with resisting dorsiflexion, eg, when you land.

We can't avoid eccentric contraction, because it happens with every step of gait. You land on the ball of your feet, and the gastroc/soleus decelerates your heel as it "lands" on the ground.
The importance of this is that if you wanted to prevent Achilles injuries in middle aged men, you could do this with an extension block on the shoe or with a soft heel cup: the extension block on the TOP of the shoe prevents excessive dorsi-flexion; the heel cup takes it easy on the gastroc.



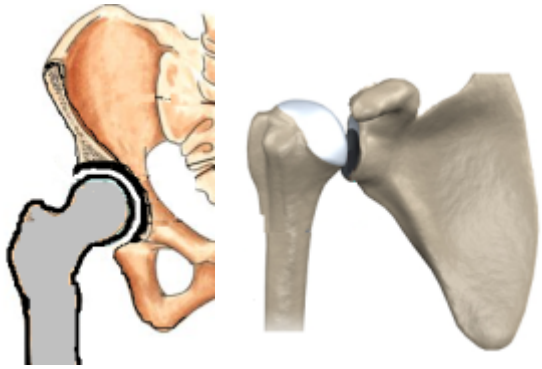
What is the role of body mass vis a vis osteoarthritis | Penn Med Self Study Questions | Why is a traumatic hip dislocation typically worse than a shoulder dislocation

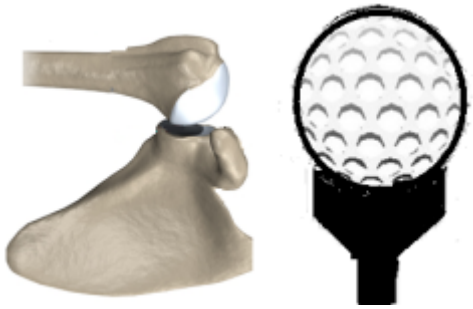
55 Why is a traumatic hip dislocation typically worse than a shoulder dislocation

Why is a traumatic hip dislocation typically worse than a shoulder dislocation? Contrast the mechanisms which prevent the normal shoulder from dislocating with those of the hip joint, and consider what structures must be damaged when the joint comes out of place.

In brief, the shoulder is a loose joint: the humeral head held in place, adjacent to (but not technically "in") the glenoid, by soft tissue, not bone. This arrangement allows for more range of motion yet less inherent stability. The hip is held in place, by contrast, via bony congruity: the femoral head sits within the acetabulum.

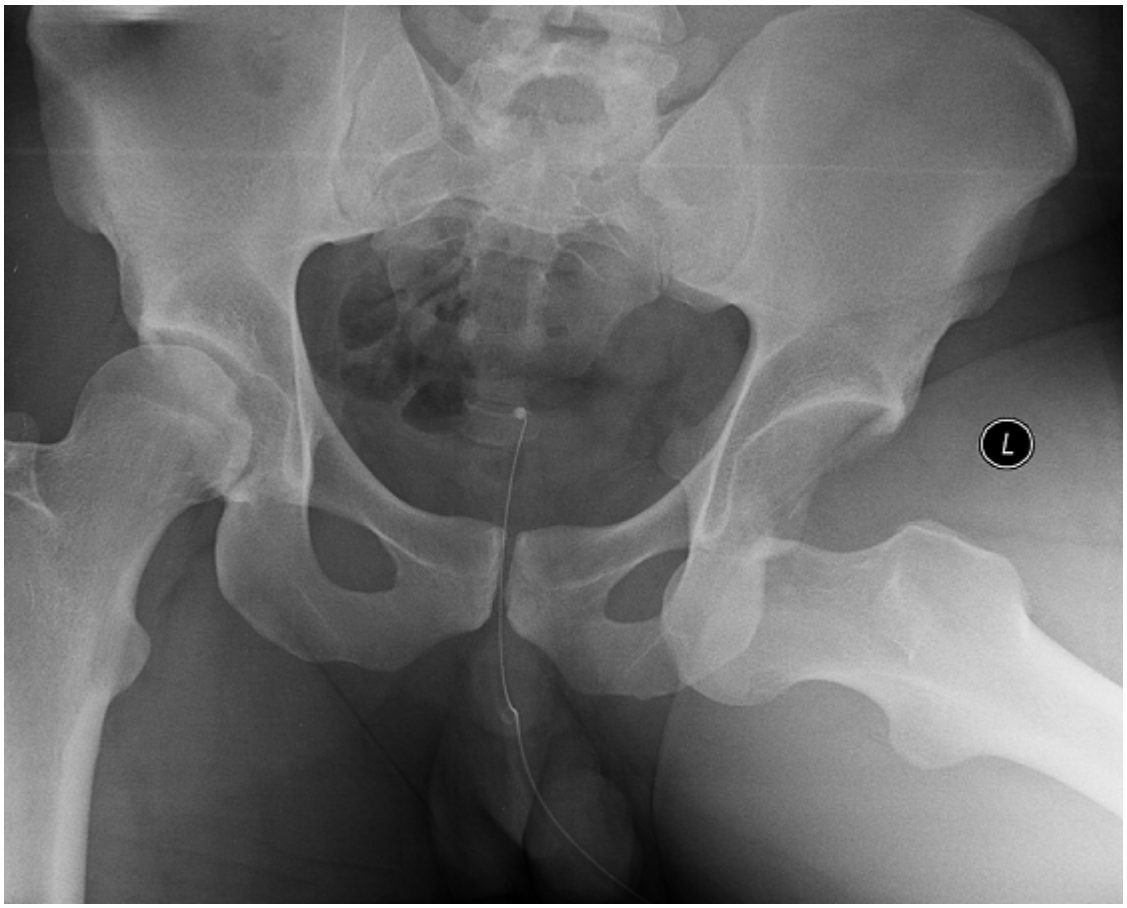
Conceptually, the hip is a "ball in socket" joint whereas the shoulder joint is more like a ball sitting on a golf tee.



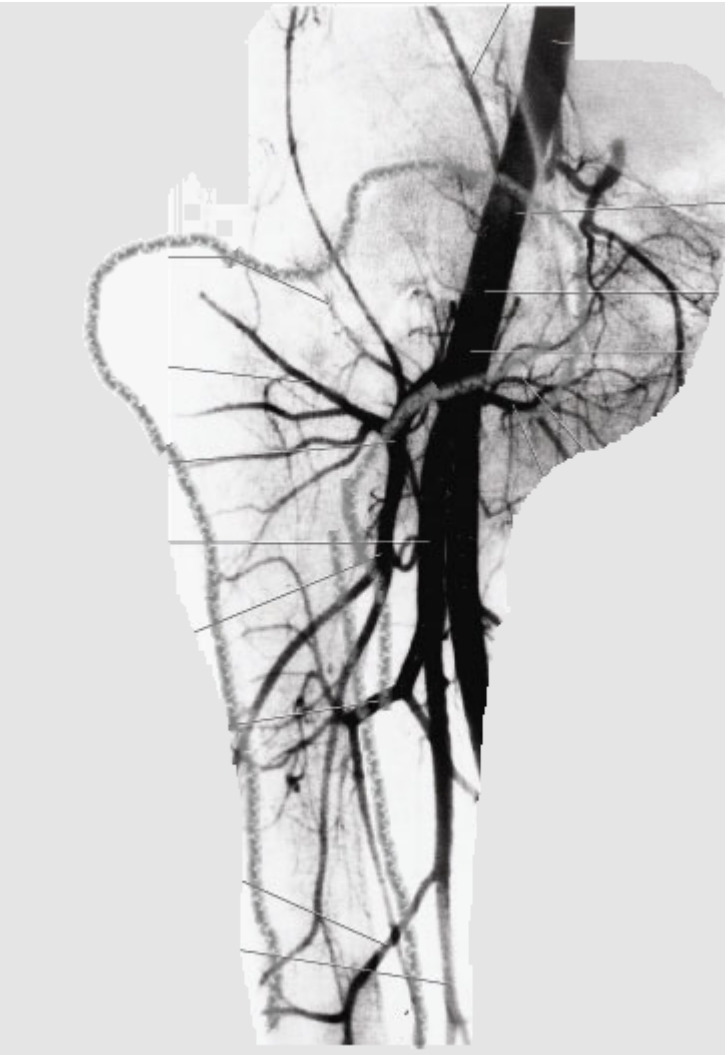


These anatomical differences explain why a traumatic hip dislocation is typically worse than a shoulder dislocation:

- A hip dislocation requires more force to get the joint out of place. And as a higher energy injury, a hip dislocation is more likely to be associated with other structural damage, such as a pelvic fracture or visceral injury (such as a urethral or bowel tear).



- Because the shoulder has so much natural freedom, the soft tissues are not tethered tightly and typically have fairly wide excursion. Specifically, the axillary artery must *naturally* be allowed to move as such motion occurs during normal use of the arm. On the other hand, the femoral circumflex arteries on the femur ordinarily do no move much relative to the pelvis, and when a dislocation "demands" them to move, they are less able to comply.



As such, when the hip is dislocated, it is correspondingly more likely that the blood vessels (and nerves) are apt to be damaged.That said, a shoulder dislocation can, under the right (or should we say "wrong") circumstances be associated with a neurovascular injury as well---it is just that there is more margin for error.

Why do 40 year old men rupture their Achilles tendons | Penn Med Self Study Questions | Why is it that a tear of the ACL typically requires surgical replacement

56 Why is it that a tear of the ACL typically requires surgical replacement

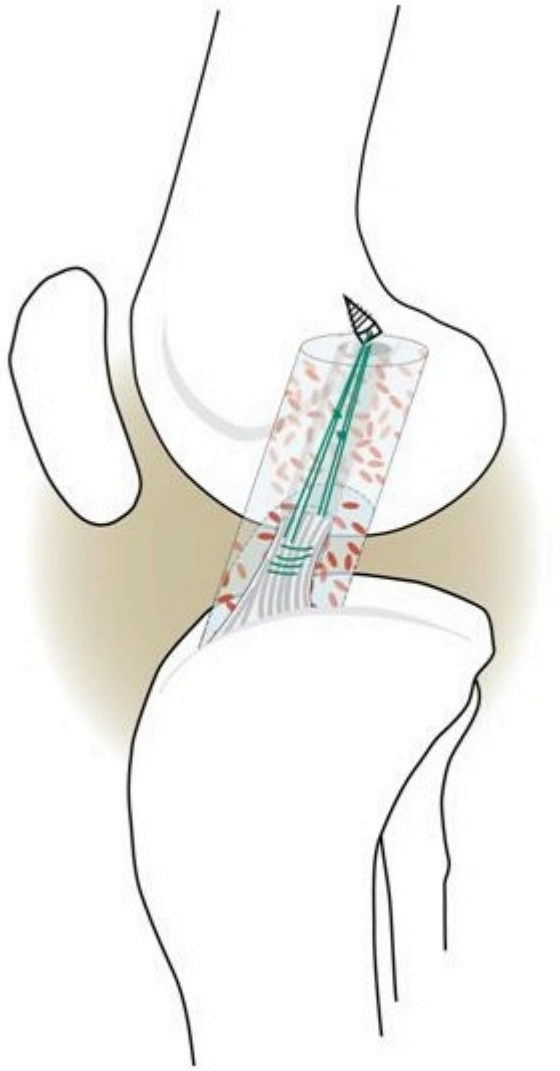
The author has something of a conflict of interest.here, so most of this answer (shown in blue) will be cribbed from an article published in the AAOS newsletter.

It is, in a way, a paean to Martha Murray, who graduated from Penn Med in 1994 and is now a professor at a Harvard

" Suture repair of a torn ACL is generally unsuccessful—with a failure rate of 90 percent. As a result, the current standard treatment for an ACL tear is to remove the ligament and replace it with a tendon graft. "

ACL RECONSTRUCTION WITH GRAFT:

ARTIST'S IMPRESSION OF WHAT ACL REPAIR WOULD LOOK LIKE (The repair is surrounded by an envelope to keep the healing factors in place, an issue discussed below)



" Dr. Murray and her team designed a series of experiments to define key biologic differences in healing between ligaments such as the medial collateral ligament (MCL) that heal and those such as the ACL that don't."

They first compared fibroblasts in the ACL with those in the MCL. They found that cells in both injured ligaments have comparable rates of proliferation, that each ligament was able to revascularize after rupture, and that collagen production in each ligament was comparable up to 1 year after injury. But in the injured MCL and other extra-articular ligaments, a provisional scaffold developed—something that was not seen in the ACL.

The synovial fluid that surrounds the ACL washed away the blood clot that forms as an early bridge between the two torn ends of the ligament. As a result, "there was no structure in place to rejoin the two ends of the ligament, no place for surrounding cells to invade and remodel into a functional scar tissue," said Dr. Murray. The researchers hypothesized that the lack of a provisional scaffold between the two ends of the torn ACL was the key mechanism behind its failure to heal."

	Why is a traumatic hip dislocation typically worse than a shoulder dislocation	Penn Med Self Study Questions	Why is osteoarthritis painful	

57 Why is osteoarthritis painful

Why is osteoarthritis painful?

This is not known with certainty. The key point to recall is that for a given objective presentation, the subjective presentation can be quite variable.

In osteoarthritis, the cartilage may wear away in some areas, greatly decreasing its ability to act as a shock absorber. As the cartilage deteriorates, the joint tips (collapses where cartilage is lost) and soft tissue may stretch (on the tensile side), perhaps causing pain.

Fluid in the knee could be under pressure and also painful by simple distension.

Also, bone edema (seen frequently in DJD) can cause pain, as we know in the cases of "kissing contusions".

The short answer is: we are not sure. Cartilage does not have the receptors to feel pain. Hence, it must be the bone or the synovium that transmits the pain---***but not all "ugly" joints hurt***. You cannot look at an xray and predict necessarily if somebody has pain; more to the point, you cannot look at an xray showing DJD and predict necessarily if the pain is necessarily caused by the DJD (and not, say, bursitis, radiculopathy or vascular disease).

Recall: a dog can have lice and fleas. In the case of arthritis, you can certainly have a second disease present concurrently (after all, it is typically a condition of aging, so other systems may be winding down too!) and you cannot even be sure that the arthritis you see literally in black and white is symptomatic.

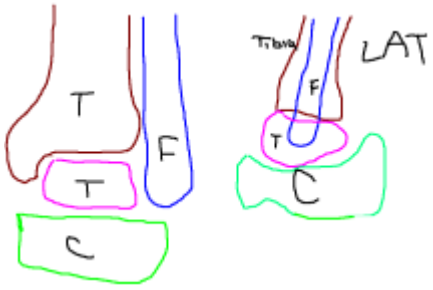
(Note: there is certainly a relationship between objective presentation and subjective presentation. It's just not perfectly correlated. Just as hoof beats suggest horses, not zebras, pain right where there is joint space narrowing with osteophytes suggests that DJD is the cause.)

⇦	Why is it that a tear of the ACL typically requires surgical replacement	⇧ Penn Med Self Study Questions	Why might a 12 year old boy with a swollen ankle and pain on the distal fibula be treated with a cast despite normal xrays	⇨

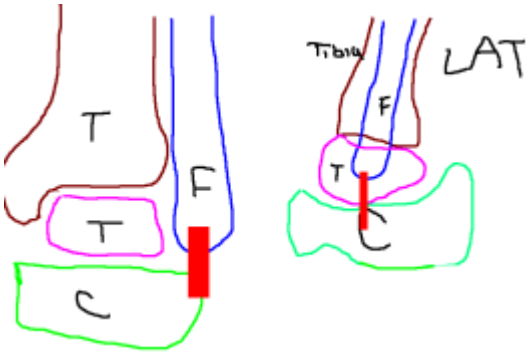
58 Why might a 12 year old boy with a swollen ankle and pain on the distal fibula be treated with a cast despite normal xrays

Why might a 12 year old boy with a swollen ankle and pain on the distal fibula be treated with a cast despite normal xrays?

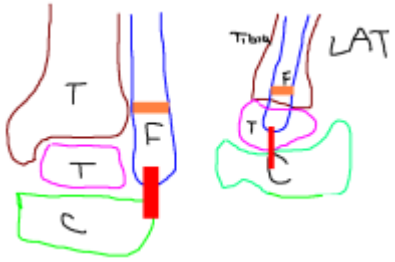
To consider the questions about ankle sprains and Achilles injuries, let's first go over the anatomy, AP view to the left, lateral on the right:



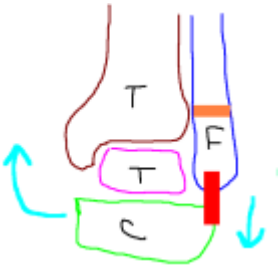
In this next view, I have added the calcaneo-fibular ligament --one of the major supporting structures that is stretched with an inversion sprain (twisting your ankle medial side up, like when you partially land on another player's shoe).



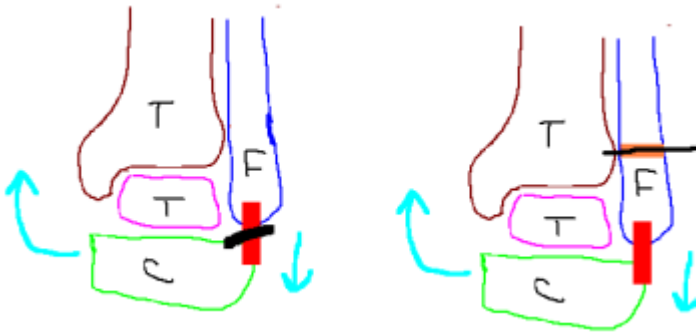
And now I have added the fibular growth plate



Ok, so you are 12 year old and twist your ankle medial side up, by partially landing on another player's shoe The force is shown in blue. This is TENSILE on the lateral side and compressive medially:



Something's got to give:



If you have no growth plate, left panel, you sprain your CF ligament; but in a twelve year old, the growth plate is weaker, so it gets injured.
Now, it there are "normal xrays" then by definition this a non-displaced fracture.
This is an urgent medical situation, however, as we want to keep it non-displaced! It needs a cast, typically.

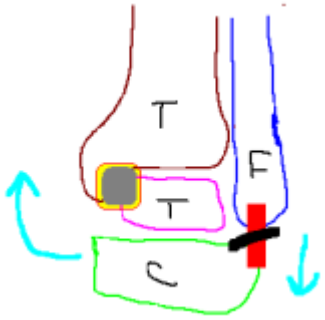
TAKE HOME MESSAGE: 12 year olds don't get sprains. (OK, Maybe they do, but you have to assume that a sprain is a non-displaced growth plate fracture until proven otherwise.

	Why is osteoarthritis painful	Penn Med Self Study Questions	Why might a grade I ankle sprain cause long term impairment

59 Why might a grade I ankle sprain cause long term impairment

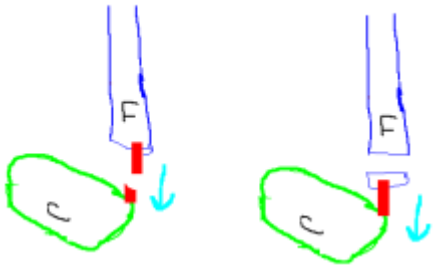
The phrase "just a sprain" may grossly understate the impairment such an injury imparts. Why might a grade ONE ankle sprain cause long term impairment?

Let's revisit that inversion sprain.



There are two injuries here, really. Tension on the CF ligament and compression on the talus.
Even if the CFL is not torn, you can get damage to the proprioceptive nerve fibers in it and get a sense of unsteadiness despite being intact as well as post traumatic arthrosis (arthritis) of the tibio-talar joint

For the interested student only: An inversion force to the ankle can really injure the bone or the ligament. Which gets injured actually depends on the rate of speed of loading, owing to the bone's so-call visco-elastic properties:



Which is worse? And (of course) WHY?

⇦	Why might a 12 year old boy with a swollen ankle and pain on the distal fibula be treated with a cast despite normal xrays	⇧ Penn Med Self Study Questions	Why might Developmental Dysplasia of the Hip, a Slipped Capital Femoral Epiphysis and Perthes Disease cause arthritis of the adult hip	⇨

60 Why might Developmental Dysplasia of the Hip, a Slipped Capital Femoral Epiphysis and Perthes Disease cause arthritis of the adult hip

Developmental Dysplasia of the Hip, a Slipped Capital Femoral Epiphysis and Perthes Disease might cause arthritis of the adult hip (ie, later in life). Why might successfully treated Developmental Dysplasia of the Hip have the best prognosis whereas untreated Developmental Dysplasia of the Hip might present the biggest treatment (surgical reconstruction) challenges?

DDH is truly treatable: relocate the hip before it deforms (or rather, "fails to form correctly"), and that maneuver is tantamount to a cure.

SCFE pinning is basically halting progression: make it better by not allowing it to get worse.

The "Treatment" of Perthes is lots of prayer: that is, the patient may get well even without treatment (or fail despite it).

At the other extreme, untreated DDH is the worst, because not only do you have bad arthritis (as you might with the other two, too-) but you don't even have normal anatomy with which to reconstruct the hip! There is no socket either: the pelvis, as well needs normal anatomy on the other side of the joint, ie femoral head, to form correctly. If either is missing, then the other side of the joint can be marred.

Try to recreate these from memory to underscore your understanding of these conditions. As shown, the acetabulum (the "socket" for the ball of the femoral head) is shallow, possibly to the point of non-existence, in DDH

Why might a grade I ankle sprain cause long term impairment | Penn Med Self Study Questions