The _____

PRISM

And Externship Guide.

A **100-page** **P**odiatric **R**esidency **I**nterview **S**tudy **M**anual**.**

This 2022 Edition was edited by RC, MxM and CJM.

David Hockney's *Mount Fuji and Flowers*

PRISM App available at: https://itunes.apple.com/us/app/podiatry-prism/id1089332577?mt=8

Introduction:

Preparing for clerkships and the residency interview is one of the most challenging tasks facing the podiatric medical student. It can be a completely overwhelming exercise unless you are efficient about the way you approach the process. **If you are reading this introduction you are already aware that in order to be fully prepared for externships and residency interviews, then you need to study a lot more than what you got from classes and clinic in school.**

You should have a strategy going into the residency interview, just as your interviewers should have a strategy about how to evaluate you. **One of the most important ways to prepare is to think about the interview process from the other side of the table.** The interviewers only have a given amount of time to spend with you; no more than 30 minutes in most cases. This is not a lot of time. *What do they really want to know about you? What information can they get from you in 30 minutes that tells them about what kind of doctor you are going to be*?

Remember that the attendings of a program are essentially hiring you to help handle their patients, and at the interview they want to know if they can trust you with this responsibility. Asking inane, esoteric questions during the academic interview doesn't really give them this information. On the other hand, asking basic "work-up" based questions does. **These questions allow the interviewer to see how you will be approaching *their* patients in the future**. It gives them information about how your mind works when dealing with patients on an everyday level. If you were an attending on the other side of the table, would you rather know if the student can take you through the clotting cascade or Kreb's cycle, or how they are going to handle *your* patient in the ED with a suspected post-operative infection?

Another thing to think about is that the interviewers need to compare your answers to the other people you are going against for the program, and they don't have a lot of time to do this. They should have some standardized way of quantitatively grading your performance against the performance of others. **I like to think of this as "check marks".** Think of the interviewers asking the same exact questions to each student and then having a form or a list in front of them. There are certain "buzz words" that they want you to say and certain questions that they want you to ask during the work-up. The more things you get correct, the more "check marks" you get on their form. And at the end of the day, they add up all the "check marks" and see who got the most. Your goal during a 30-minute interview should be to get as many "check marks" as possible.

This manual was put together based on the way that I studied for interviews. There is certainly no shortage of material to study, and this manual is not intended to replace or even rival some of the other study guides that are out there. **The goal of the PRISM is simply to help you be as efficient as possible with the process and to think about the interview from the other side of the table**. My goal with coming up with the following sheets was to take a given topic and fit everything that could be asked about that topic during an interview onto a single sheet of paper. **Realize that it is not all the information on a given topic, but all the information that is *most likely to be asked* during an interview.** There's a big difference there.

Your goal heading into the interview process should be to have a standardized way of handling every question or situation that you are presented with, and to get as many "check marks" as possible. Think about it. The easiest way for the interviewers to answer the questions they have about you is to present you with a clinical scenario, and see how you work-up that situation. Therefore, the most efficient way to study for interviews is to take a given topic, and then "work-up" a patient in that situation. **I made all of the AJM Sheets with this thought in mind (see "Gout" example on next page).**

Also included in this manual are **AJM Lists**. Studying is by nature a passive exercise, but the interview process involves actively answering questions and talking out loud. The Lists allow you to actively think about a topic like you will be expected to do during the interview. It takes a broad, clinical situation/subject and asks you to come up with as many answers as possible. I hope that they help you realize that there is not always one answer to a question, but possibly many different answers that can all be considered correct. **The more answers that you can come up with for a given List, the more "check marks" you get during the interview.**

Again, this manual is far from complete and absolutely does not contain all of the information you will be asked during an interview. **It simply hopes to change the way that you think about the interview process and highlight some of the information that you are most likely to be asked.** I limited it to 100 pages of the most commonly asked information and the kind of stuff that I'm going to ask if I'm a residency director someday *(uh-oh….this has happened!)*. It is not in any way meant to be overwhelming.

I also want this to be a "living" document. It is not intended to be commercial and should never be sold. I'm going to take it with me to Temple when I graduate from residency, leave it with the Inova program, and distribute it electronically to anyone who wants it. Feel free to change/update it in any way that you think would be helpful, but **please keep it to exactly 100 pages**. In other words, if you think something is important and should be included, you also have to decide what isn't as important and should be taken out. It will be interesting to see how it evolves over the years!

Good luck and please do not hesitate to contact me if there is any way that I can be of service to you.

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AJM Sheet Example: Gout

-Subjective

CC: Pt classically complains of a "red, hot, swollen joint". Typical patient is a male in the 6th decade (as much as a 20:1 M:F ratio).

HPI:
- -Nature: Intense pain out of proportion with swelling/pressure.
- -Location: Single or multiple joints. Unilateral or bilateral. Most common is 1st MPJ ("podegra"), but can occur in any joint.
- -Acute, abrupt onset; more commonly at night.
- -Aggravating Factors: Pressure, WB, diet (red wine, organ meat, lard, seafood).
- -History: Recurrent gouty attacks are very common

PMH/PSH:
- -Genetic enzyme defects, obesity, lead poisoning, tumor, psoriasis, hemolytic anemia may all be underlying causes.
- -Renal disease (renal disease is 2nd most common complication of gout).
- -Kidney stones

SH: -Diets high in red wine, organ meat, lard and seafood may exacerbate.

Meds: -Diuretics, low dose ASA, TB meds, warfarin may exacerbate.

All/FH: -Usually non-contributory

ROS: -May be associated with fever.

-Objective

Physical Exam

-Derm:
- -Erythema, Calor, Edema present at affected joint
- -May see tophi sticking out of skin

-Vasc: -Non-pitting edema at affected site

-Neuro: -Intense pain out of proportion

-Ortho: -Decreased PROM/AROM at affected joint with guarding.

Imaging

-Plain Film Radiograph:
- -Increased soft tissue density with joint effusion. Tophi may be visible in soft tissue.
- -Fine striated pattern of periosteal reaction along the cortex adjacent to tophi
- -Lace pattern of osseous erosion
- -Round osseous erosion with a sclerotic margin ("rat bite erosion" or "punched-out lesion").
- -**Martel's sign**: Expansile lesion with an overhanging osseous margin.

Laboratory

-**Joint aspirate** is mandatory for diagnosis of gout:
- -Needle-shaped monosodium urate crystals
- -Negatively birefringent (bright yellow) when viewed under polarizing light microscope parallel to axis of lens.
  - -Blue when perpendicular to axis of lens.

-Serum uric acid levels **> 7.5mg/dl** (non-diagnostic; and usually is not elevated until ***after*** an acute gouty attack)[Normal value ~3.5-7.2mg/dl]

-Elevated ESR

-Synovial fluid analysis: Elevated leukocytes with a predomination of neutrophils

-Generalized increased white cell count

-General Information

-Definition: Metabolic disorder secondary to the build-up of monosodium urate crystals and supersaturated hyperuricemic extracellular fluids in and around joints and tendons causing the clinical manifestations of a red, hot, swollen joint.

- -It is the most common cause of inflammatory arthritis in men over the age of 30.

-Classification

-Primary: Elevated serum urate levels or urate deposition secondary to inherent disorders of uric acid metabolism.
- -**Uric Acid Overproduction** (Metabolic Gout): 10% of patients
  - -Excessive amounts of uric acid excreted into the urine
  - -Occurs secondary to an enzyme defect, tumor, psoriasis, hemolytic anemia, etc.
  - -Dx: Uric Acid Level >600mg in a 24-hour urine collection
- -**Uric Acid Undersecretion** (Renal Gout): 90% of patients
  - -Relative deficit in the renal excretion of uric acid.

-Secondary: A minor clinical feature secondary to some genetic or acquired process

-Treatment

-Symptomatic Pharmacology (relieves symptoms, but doesn't attack underlying pathophysiology)
- -**Indomethacin**: 50mg PO q8
- -**Colchicine**: 0.5-1.0mg PO initially, then 0.5mg PO q1 hour until symptoms (GI) or pain relief
  Then around 0.5mg PO qday as prophylaxis
  - -***Above and Beyond Question*:** *What is the mechanism of action of colchicine with respect to gout?*

-Active/Physiologic Pharmocology (attacks underlying pathophysiology and prevents recurrence)
- -**Allopurinol**: 100-600mg PO qday as single or divided doses.
  - -Blocks uric acid production by inhibition of the enzyme **xanthine oxidase**.
- -**Uloric** (febuxostat): 40-80mg PO qday as a single dose
  - -Blocks uric acid production
- -**Probenecid**: 250mg PO bid for one week; then 500mg PO bid
  - -Increases uric acid removal from urine (decreases reabsorption)

-Surgical Intervention (if you get rid of the joint, then you get rid of a potential site for gout to attack!)
- -I&D/Washout -Arthroplasty -Arthrodesis

-Further Reading

-*Roper RB. The perioperative management of the gouty patient. J Amer Podiatry Assoc. 1984 Apr;74(4):168-72.*

-*Schlesinger N. Management of acute and chronic gouty arthritis: present state-of-the-art. Drugs. 2004;64(21):2399-2416.*

-*Keith MP. Updates in the management of gout. Am J Med. 2007 Mar;120(3):221-4.*

Table of Contents:

AJM Lists [Pages 5-29]

AJM Sheets [Pages 30-100]:

-Page 100: "*Can you give me some good articles to read?"*

Lists Schedule:

AJM Lists were originally created to be done during a clerkship. Students often have a lot of down time during the day while the residents are doing work that doesn't need assistance. The lists give the students something to do during this time and make it look like they're busy instead of just standing around doing nothing (in front of the attendings and residents). It also encourages students to collaborate, and shows the residents/attendings that they can work well together and in groups.

When I was a resident, I would give the students one list and a related article each day, and then we would try and get together once a week to go over them. It usually generated a great deal of good discussion. If you are using these lists to study on your own, get together with a group of friends to go over them and talk about your answers **out loud**. The way you know if you really understand a topic is if you can intelligently discuss it and explain it to your peers.

Studying is by nature a passive exercise, but at the interview you will be expected to actively answer questions out loud. **Only about half of what the interviewers appreciate from your answer is the actual content, the other half is *how you say it.*** Remember that the interviewers are probably asking the same exact questions to every student that walks through the door, so they've probably heard the same answer several times before you even sit down [*AJM Note*: Now that I'm a director, I can tell you that interviews are indeed pretty boring from the other side of the table]. ***What they haven't heard is how you've said it!*** In other words, you should also be studying "how to say it".

Consider the following suggested schedule:

Mondays: Surgery

Tuesdays: Medicine

Wednesdays: Trauma

Thursdays: Anatomy

Fridays: Social Questions

AJM List: HAV Procedures and Indications

Clinical Scenario: You are a first year resident scheduled to be in a "bunion procedure" at a surgery center tomorrow. You are working with the attending for the first time and want to appear as prepared as possible. Name as many "bunion procedures" as you can.

Student Goal: Name 20 HAV procedures from distal to proximal.

-*What are the specific clinical and radiographic indications for each?*

AJM List: Surgical Complications

Clinical Scenario: You are a first year resident at a surgery center. The attending isn't there yet, and you aren't exactly sure of the specifics of the case, but you want to have all the paperwork filled out for when the attending gets there (including the consent).

Student Goal: Name as many risks and complications of a generic foot and ankle surgery as possible.

-*What are some specific complications associated with some specific surgeries?*

-*How would you handle these complications in the post-op period?*

AJM List: Measurement of Radiographic Angles

Student Goal: Name as many foot and ankle radiographic measurements as possible.

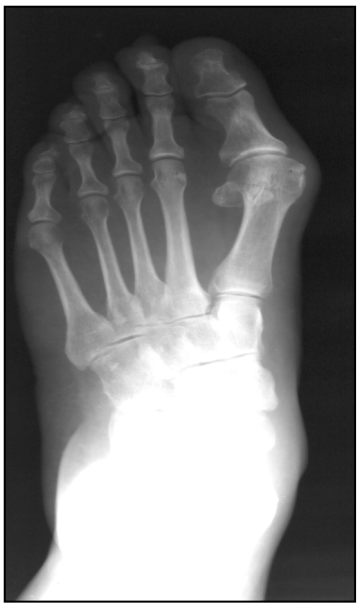
-What are their normal values?

-What clinical information is this giving you when increased or decreased?

AJM List: Radiology Review

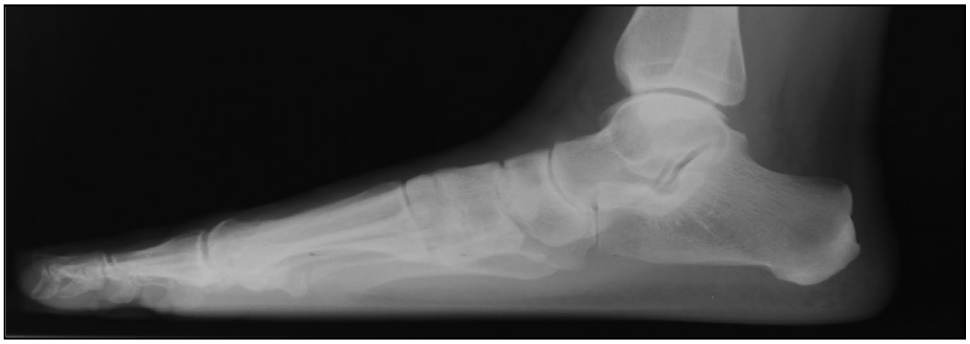
Clinical Scenario: There is a big difference between *describing* and *diagnosing* a radiograph. During an interview, you need to be able to describe the findings you are seeing before you diagnose the pathology. You also need to be able to do this out loud during the interview process.

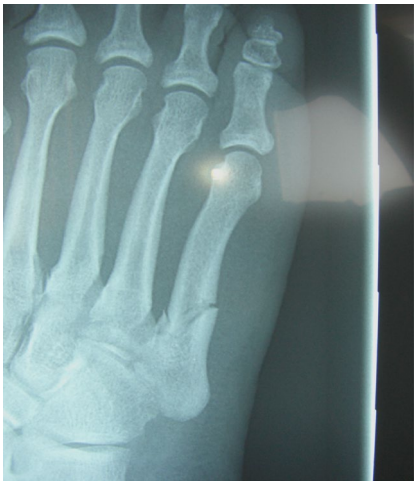
Student Goal: Out loud, using as many correct radiographic terms as possible, and in a systematic manner, intelligently describe the following radiographs before making a diagnosis.



There are **3 components** to a good, problem-focused radiographic description (whether describing x-rays in clinic, during a residency interview, over the phone to an attending for a consult, etc):

1. What you are **looking at**: ("Lateral view plain film radiograph of the left foot…"; "AP view plain film radiograph of the right ankle…"; "T2 sagittal view MRI of the left midfoot…"; "Axial CT scan bone window of the right rearfoot…", etc).
2. What you **see**: This is the description and can be a little confusing. For example, you can't ***see*** a fracture on an x-ray…you can only see a radiolucency ***which is consistent with*** a fracture. On a plain film radiograph or CT you can only *see* **radiodensity** or **radiolucency**, on an MRI you can only *see* **increased signal intensity** or **decreased signal intensity**, and on an ultrasound you can only *see* **hyperechoic** or **hypoechoic** signals. It's a little nit-picky, but you have to learn the appropriate terminology.
3. What **you think about what you see**: Here's what we get paid for. Make an assessment or specific diagnosis.





So I can't ***see*** an ankle fracture on an x-ray, but "On the NWB AP view of the right ankle I see a transverse radiolucency through the distal fibula at the level of ankle joint consistent with complete fracture. There is no displacement, angulation or rotation of the distal fragment relative to the proximal fragment. My impression is a non-displaced Weber B type fibular fracture."

And I can't ***see*** a bunion on an x-ray, but "On the DP view of the left foot I see increases in the 1st IMA, HAA and metatarsal-sesamoid position consistent with a mild hallux abductovalgus deformity".

PRACTICE! And practice OUT LOUD!

Note: RC and I found it very helpful to practice for interviews by picking up random podiatric surgical textbooks, and alternating through the pictures describing the radiographs out loud to each other. Take 15 minutes each day to do this in the time leading up to interviews, and you'll be surprised how much more confident you feel.

AJM List: Surgical Layers of Dissection

Student Goal: Identify the 5 surgical planes and 3 surgical intervals of dissection.

-*What structures will you see in each plane/interval for a standard HAV procedure?*

-*For a standard digital procedure?*

AJM List: Post-operative Fever

Clinical Scenario: On call, you are paged at 3am by a nurse to report a fever in a patient POD#2 of 101.6°F.

Student Goal: Name as many potential causes of fever as possible.
-How would you diagnose and work-up each one?

AJM List: Laboratory Infection Diagnosis

Clinical Scenario: A patient enters the Emergency Department with a suspected lower extremity infection.

Student Goal: Name as many laboratory tests that you can order to help diagnose an infection.
-*What clinical information is each test really telling you about the situation?*

AJM List: Imaging Infection Diagnosis

Clinical Scenario: A patient enters the Emergency Department with a suspected lower extremity infection.

Student Goal: Name as many imaging tests that you can order to help diagnose an infection.
-*Exactly what are you looking for with each test?*

AJM List: Routine Lab List

Student Goal: Identify routine labs, their normal values, and what information they are giving you about the patient. Which labs should be drawn when an infection is suspected and why? Which should be done in the pre-operative work-up of a patient? How would one address abnormalities of these labs?

AJM List: Vascular and Neurologic Assessment

Student Goal: Name as many subjective and objective vascular and neurologic assessments as possible while performing a lower extremity examination.

AJM List: Ankle Fracture

Clinical Scenario: A patient arrives in the ED exclaiming that they "broke their ankle". Obviously you will take an x-ray, but what exactly are you looking for on the radiograph?

Student Goal: Name as many possible fractures that can occur following an ankle sprain.

-***Above and Beyond Question*:** *What do the "Ottawa Ankle Rules" say about getting a radiograph following an ankle sprain?*

AJM List: Screw Games

Thread Diameter	Spheric Head Diameter	Screwdriver: Hex or Cruciate?	Core Diameter	Drill bit Thread Hole	Drill bit Gliding Hole	Tap Diameter
Mini Frag Set						
1.5mm						
2.0mm						
2.7mm						
Small Frag Set						
3.5mm						
4.0mm PT						
4.0mm FT						
Large Frag Set						
4.5mm						
6.5mm PT						
6.5mm FT						

-Pitch on a cortical screw from the small fragment set?:

-Pitch on a cancellous screw from the small fragment set?:

-Draw a screw labeled with as many anatomic landmarks identified as you can (eg. Head, major diameter, pitch, etc):

AJM List: Methods of Fixation

Student Goal: Name as many methods as possible to fixate an osteotomy.

AJM List: Hardware Insertion Technique

Student Goal: Describe standard AO lag screw insertion technique. What is the purpose of each step? Why are they done in that particular order? What is compromised technique? Splintage?

-Bonus: What is the quantitative measurement of "two-finger tightness?":

-What are some strategies for hardware extraction?:

AJM List: Classifications

Student Goal: Name as many different trauma classifications as you can that cover the foot and ankle from distal to proximal.

AJM List: Ossification Dates

Student Goal: Name every bone in the lower extremity in order of ossification date.

AJM List: 5th Metatarsal Anatomy

Student Goal: Name as many structures as you can that attach to the 5th metatarsal.

AJM List: Dorsal Arterial Anastamosis Variations

Student Goal: Draw out as many different variations as possible for the arterial supply to the dorsum of the foot.

How does this apply to the angiosome principles? (hint: read Dr. Attinger's work)

AJM List: Local Anesthesia and Peripheral Nerve Blockade

Student Goal: Identify as many named foot and ankle peripheral nerve blocks as possible. Which specific nerves are being anesthetized with each block?

-Toxic Dose of Lidocaine?:

-Toxic Dose of Marcaine?:

-How and why does epinephrine influence the toxic dose of a local anesthetic?

-What are the reversing agents for local anesthetic toxicity?

AJM List: Dermatomes

Student Goal: Draw a lower extremity with all of the dermatomes illustrated with associated spinal levels and landmarks.

-Bonus: How are dermatomes different than sclerotomes?

AJM List: Social Questions

Part I: General Questions

Personal:

Strengths: Be prepared to give at least 3 personal strengths and why they will make you a good resident.

-Strength #1:
    -Why it will make you a good resident:
-Strength #2:
-Strength #3:

Weaknesses: Be prepared to give a couple weaknesses, and more importantly, how you are remedying them.

-Weakness #1:
    -Remedy:
-Weakness #2

Goals: Be prepared to give professional and personal goals, and how you will go about accomplishing them. Another form this question could take is where you see yourself in a given number of years.

-Professional Goals:
    -Goal #1:
    -Goal #2:
    -Goal #3:

-Personal Goals:
    -Goal #1:
    -Goal #2:
    -Goal #3:

-Where do you see yourself in:
    -5 years?:
    -10 years?:
    -25 years?:

Program Specific: For each program that you apply to, you should have a list of strengths and weaknesses for that program. Obviously be careful with weaknesses, and always have a way that you personally can improve the situation. You should be able to answer why you personally are a good fit for that program. I found it helpful to have a list of priorities that I was looking for in the different programs, and then described how that particular program fit into my priorities.

-Program #1:

-Strength #1:
-Strength #2:
-Strength #3:
-Weakness #1:
-Weakness #2:

-What you are able to bring to the program:
-Favorite attending and why:
-Least favorite attending and why:
-Favorite resident and why:
-Least favorite resident and why (you will get asked this!):
-Best case you saw at the program:

Here's my list of priorities that I used based on what was important to me. Everyone's list can (and really should) be different, this is just to provide an example:

1. **Surgery/Academics**
    -How is this program going to make you a better doctor? You're going to learn surgery and do some academic events at any program in the country, is there anything special about this particular program that sets it apart? Do they really care about academics, or are they just done to meet a requirement? (Hint: a good way to tell this is to see how often attendings are excited to show up to and be involved in meetings). Although the ***quantity*** of surgery is important (you have to be able to get your "numbers"), also consider the ***quality*** and ***variety*** of the surgeries at a program.
2. **Outside Rotations**
    -All programs have the same set of "core" rotations that everyone has to do. Is there anything unique about this particular program that shows that they really care about your complete education and want you to have outstanding quality and variety to your residency experience?
3. **Location:**
    -This one kind of speaks for itself, but you should consider if you are going to be completely at one hospital versus traveling to different hospitals and different surgery centers (there are pros and cons to each). Also consider what the presence is of the program within the hospital. Is the podiatric surgery department intricately involved in the hospital, or is it more of an afterthought?
4. **Independence**
    -I'm an independent guy who likes to come up with and pursue my own projects and ideas. Other people really like structure and would prefer to get an exact schedule for the next three years on day 1 of their residency. So this was something that I was really looking for, but someone else may want exactly the opposite.
5. **The Future**
    -How is this program going to help you accomplish your professional goals now and *after* you graduate?

AJM List: Social Questions
Part II: Personal Questions

Personal Questions: These questions are hard to answer and often irrelevant, but you should have answers ready to go (even if they are made up). Answer as specifically as possible to give the interviewer something tangible to grab onto about yourself. Always answer "Why?" before they have the chance to ask you. ***"Why?"* may be the most important question you get during the interview process**.

-What do you like to do with your free time?
    -Answer #1:
    -Answer #2:
    -Answer #3:
-What professional accomplishment are you most proud of?:
-What personal accomplishment are you most proud of?:
-What was your hardest/most trying experience? What did you learn from this?:
-What is your most embarrassing moment?:
-Name three things that you would bring with you to a deserted island:
    -#1:
    -#2:
    -#3:
-Tell me a joke:
    -#1:
    -#2:
    -#3:
-Favorite Movie:
-Favorite Book:
-Last movie you saw:
    -Something you liked about it:
    -Something you didn't like about it:
-Last book you read:
-Favorite Band/Kind of Music:
-Last concert you went to:
-Tell me about the craziest patient you have ever had to deal with:
-What is your funniest medical story?:
-What animal would you be and why?:
-What tree would you be and why?:
-Favorite TV show:
-Favorite actor:
-Favorite actress:
-Favorite surgical instrument/tendon/bone/joint:
-Do you have any pets?:
-Favorite pet:
-If you could take a vacation anywhere in the world, where would it be?:
-Tell me something about yourself that few people know:
-Who is the most important person in your life (real and/or fictional) that you have never met?:

-And of course, "**Tell me a little about yourself**":
    -Important Note: You should be able to spout off both a 30-second and a 5-minute answer to this question at the drop of a hat.

-Note: These questions are very easy to answer if you think about them, but you don't have time to think during the interview. You don't want to show any hesitation during this process, especially questions about yourself. The worst answer you can give to a specific personal question is "I don't know." What is that saying about you?

Practice answering all of these questions **out loud** to yourself in the time leading up to interviews! You may feel silly talking to yourself, but it is undoubtedly the best way to prepare for this line of questioning.

AJM List: Social Questions
Part III: Academic and Ethical Social Questions

Academic Social Questions: These are hidden academic questions, but ones you can't study for in any book. Please plan these questions out because it is very easy to get trapped in your answer if you hesitate/lie.

-What journals do you read? Which is your favorite?:

-What was the last good journal article you read? (be able to cite it!):

-What was the last thing you built with your hands?:

-Favorite class in school?:

-Least favorite class in school?:

-Favorite teacher in school?:

-What types of things does the field of podiatric surgery need to do to improve in the future?:

-What is something you learned about the field of podiatric surgery since you started school/externships?:

-Have you participated in any research projects? Why or why not? What was your role in this project?:

-What would you do with your life if you couldn't be a surgeon/physician?:

Ethical Questions: The key to answering an ethical question is to take a step back from the situation. Pretend that someone else is in the situation and you are going to give that person advice. Don't pretend that you are in the situation; it actually makes it more difficult to think through the process for whatever reason. Remember the concept of **chain-of-command** and also remember that there is a real patient involved.

Something else that really helped me out was having a clear order of my priorities. Everyone's can be different, but mine are:

1. Responsibility to the patient as a physician
2. Responsibility as an employee of a hospital
3. Responsibility as a resident of the residency program
4. Responsibility for my own education
5. Responsibility for the education of junior residents/students

So whatever ethical situation I was put into, I would make decisions based upon this order of priorities. Also remember that usually there is no right or wrong answer when it comes to ethical situations. Like George Costanza said about beating a lie detector test: "It's not a lie (wrong), if you believe it."

The Semistructured Conversation: Many residency programs (and especially general medicine or general surgery residency programs) have re-evaluated the way that they have traditionally selected residents, and have moved away from a structured academic interview. They have instead moved onto what's called a "semistructured conversation" that tries to evaluate if the applicants have the "knowledge, skills and attitudes deemed necessary for the practice of medicine". The questions are a kind of mix of academic and ethical questions that can develop into more of a conversation. So while these are not purely academic questions, they can lead into a conversation about specific academic topics. There's actually a couple articles about it (*Neitzschman HR, Neitzchman LH, Dowling A. Key Component of Resident Selection: The Semistructured Conversation. Acad Radio. 9: 1423-29; 2002*.), and I've put together a long list of these type questions on the next List.

AJM List: Social Questions
Part IV: The Semistructured Conversation Interview Questions

-Tell me about a patient care situation in which podiatric surgery altered the management of the patient.
-Describe a critical clinical situation and how you communicated with the family.
-Tell me about something you learned during one of your externships.
-How would you respond to a patient who asks, "Am I going to die?"
-Can you recall any time when you disagreed with a patient's diagnosis or treatment?
-Tell us about the biggest argument/controversy you were involved with in podiatry school.
-Give us an example of a situation when you were pivotal in the resolution of a conflict between two other people.
-Suppose you're in charge of the call schedule. You need to fill a slot with one of two people, one of whom has told you he has to be out of town as best man in a wedding, and the other has to present a paper at a meeting. How would you resolve the conflict?
-Describe a time when you were in a position to give someone a bad evaluation. How did you handle it?
-What features would you add to a medical school curriculum that you think might better prepare you for a podiatric career?
-Can you recall a time when you received an evaluation with which you disagreed?
-If a referring physician insists that you perform a study on a patient and you believe that study could be harmful to the patient, how would you handle the situation?
-Describe a patient for whom you felt very little empathy but you knew you should.
-What would you do if you saw a senior resident make a mistake that might harm a patient if not corrected promptly?
-A patient acquires your pager number and home phone number and calls several times per day. How do you handle this patient's needs?
-A consulting physician asks you a question, and you are not sure of the answer. How do you handle it?
-Tell me about a negative interaction you had during medical school with anyone from a transporter to an attending and how the two of you dealt with it at the moment and afterwards.
-What do you see as the most challenging aspects of a podiatric residency?
-How would you handle a situation when you know one of your fellow residents has a problem with drugs or alcohol?
-For what reasons do you want to come this particular geographic area?
-You are on call and someone else asks you a question on a subject you know nothing about. How do you gather information about the topic expeditiously?
-What resources did you use for researching residency programs?
-Outside of the structured lecture, what other formats did you find most helpful as learning tools?
-Describe for me how you deal with a colleague who is exhibiting evidence of substance abuse.
-Describe your response to an episode of someone cheating on the Gross Anatomy final examination.
-How would you decide (and what factors would you consider), as an HMO executive, whether to immunize 2,500 children at $100 each or provide one liver transplant at $250,000 each?
-In what ways do you maximize your own health and well-being?
-How would you deal with a fellow resident who is not "pulling their own weight" in their work?
-What personal qualities most helped you during medical school?
-Tell me about a patient from whom you learned something.
-How would you like to see podiatric surgery develop over the next 5 years?
-How do you see yourself changing between now and the end of residency?
-Tell me about your experience in using online resources, library resources, and internet resources.
-Did you ever feel as a medical student that you were not part of the clinical team? How did you address the situation in order to optimize your learning experience?
-Can you recall an experience that made you decide to choose podiatry as a profession?
-Describe one of your most challenging cases during your externships.
-Tell me about an experience in medical school where you felt particularly competent.
-What particular skill do you feel you have that makes podiatric surgery the best specialty for you.
-As you examine different programs, what characteristics are you looking for that we might offer you?
-What diseases or topics have fascinated you in medical school and why?
-What topics interest you that you haven't had time to explore yet?
-Tell me about an experience when you took a risk that ended up being successful.
-Can you tell me about a patient who had an impact on you?
-Can you recall a time in medical school when you had some doubt about the professional path you have chosen? What did you do?
-A number of residents (15-20%) leave general surgery residency. What do you think influences their choice?
-How would you rate yourself in terms of your ability to establish rapport and maintain healthy relationships with other health professionals?

AJM Sheets: Diabetic Foot Infection Work-Up

The Diabetic Foot Infection work-up is arguably the most important concept that you can study during the interview process because it is the one topic that you are almost guaranteed of being asked at some point. My thought process during interviews was that if I'm certain that I will be asked about it, I'm going to spend extra time and energy knowing everything possible on the subject. Every student at interviews is going to get something along these lines; therefore it's important to be the most prepared and best able to "wow" the attendings when asked. So I put together a collection of AJM Sheets (totaling about 20 pages) that goes through an in-depth work-up of a diabetic foot infection.

This topic is also a classic example of hitting as many "check marks" as possible during the interview by having a standardized way of going through a work-up. The way this situation is often presented at interviews is for them to simply ask you:

"There is a diabetic patient in the ED with a suspected foot infection. What do you want to know about the patient and what do you want to do?"

By having a standardized way of going through this work-up (or any work-up), you will seem more prepared during the interviews, hit more check marks, and won't stumble about thinking what to ask next. The basics of this work-up can essentially be applied to any clinical situation.

This work-up also highlights taking an active approach and going on the offensive during the interview process. Take control of the interview from the interviewers. Do not simply ask if the patient has diabetes; ask specific questions about the patient's knowledge, management and known complications of diabetes. This will show that you really understand the concepts and pathogenesis of the disease process.

This section has a lot of the same information presented in a number of different ways, giving you a couple ways to study. While there is certainly no shortage of material to study this information from, my favorite article on the topic is: *Lipsky BA, et al. 2012 IDSA clinical practice guidelines for the diagnosis and treatment of diabetic foot infections. CID 2012 Jun; 54(12): 132-73.* You also certainly should read: *Frykberg RG, et al. Diabetic foot disorders. A clinical practice guideline (2006 revision). J Foot Ankle Surg. 2006 Sep-Oct; 45(5 Suppl): S1-66.* And finally, the *June 2006 Supplement of Plastic and Reconstructive Surgery* is a fantastic resource covering a wide variety of diabetic foot issues, mostly from the Georgetown perspective.

Contents:

AJM Sheet: Diabetic Foot Infection Subjective History

Subjective

CC: Pts can present with a wide variety of complaints ranging from the systemic signs of infection to increased ulcer drainage to a change in mental status. Infection should always be in your differential diagnosis dealing with any situation.

HPI: Ask the patient *at least* the following questions:

-**NLDOCAT** of chief complaint

-**Systemic signs of infection**: Nausea, vomiting (quantity and quality), fever, chills, night sweats, ague, loss of appetite, change in mental status, diarrhea (quantity and quality), constipation, change in sleep patterns, headache, shortness of breath, chest pain, uncontrolled blood glucose levels, etc.

-**Local signs of infection**: Patient reported increases in pain, erythema, swelling, temperature, drainage (quantity and quality), odor, etc.

-**Ulcer specific questions** if applicable: Duration of ulcer, changes in size/depth/color, dressing changes, dressing change schedule, wound care products, last formal evaluation, primary wound care specialist, previous treatments, any history of hospitalization for infection, etc.

-Remember: The patient probably knows their ulcer better than you!

-It is extremely important to get an **antibiotic history** from the patient. Are they taking any antibiotic therapy currently (including dosage and last dose)? When was the last time they were prescribed an antibiotic?, etc. This information can provide useful information with respect to the development of resistant organisms. Specific risk factors include antibiotic use in the last 6 months, any fluoroquinolone use, and hospitalization in the last 6 months.

-Richard et al. Risk factors and healing impact of multidrug-resistant bacteria in diabetic foot ulcers. Diabet Metab. 2008 Sep.

-Hartemann-Heurtier et al. Diabetic foot ulcer and multidrug-resistance organisms: risk factors and impact. Diabet Med. 2004 Jul.

-Kandemir et al. Risk factors for infection of the diabetic foot with multi-antibiotic resistant microorganisms. J Infect. 2007 May.

-**Tetanus Status**

-**NPO Status**

PMH: -**DM**: Complete DM history including length of disease, previous complications, glucose monitoring schedule, normal glucose readings, HbA1c values, medications, last podiatric evaluation, last internal medicine evaluation, implemented preventative measures, evaluation of patients level of understanding of pathogenesis of disease, evaluation of patients role in self-treatment, etc.

-Any known **complications of diabetes** with interventions/treatment: cardiac disease, peripheral vascular disease, hypertension, retinopathy, end-stage renal disease with HD, etc.

-Specifically ask about **renal disease** and **liver disease** (antibiotic implications).

-Any other immuno-compromising conditions.

-Any other PMH issues.

PSH: -Specifically any previous amputations, foot/ankle surgeries and diabetes-related surgeries.

Meds: -Detailed list of drugs, dosages, and patient compliance to schedule.

All: -True allergies and reactions to drugs, food, products, etc.

SH: -Very important and not to be overlooked.

-Work: line of work, quantity of WB and ambulation, hours, ability of the patient to take time off or take it easy, worker's compensation issues, etc.

-Diet and exercise.

-Home support network. Includes assessment of patient compliance and family understanding/education/compliance.

-Smoking, alcohol, drug use.

-House structure: stairs, bathrooms, pets.

-Other wound contamination risk factors.

FH: -Anything applicable.

ROS: -Anything applicable.

***Diabetic foot infections are one of the most challenging aspects of podiatric surgery that will take up a lot of your time, energy, and stress if you dedicate yourself to the side of limb salvage. Taking a complete history will give you an idea of how compliant you can expect the patient to be and how actively involved you can expect the patient to be in their care.

AJM Sheet: Diabetic Infection Objective Physical Exam

Objective

-**Vital Signs**: -**Temperature**: Hyperthermia is a non-descript sign of infection. It is important to monitor temperature on a regular basis, and follow both current and maximum temperatures. Keep in mind however that *Armstrong* has documented that 82% of patients admitted for chronic osteomyelitis were afebrile on admission (*JFAS.1996 Jul-Aug; 35(4): 280-3*). It has also been suggested that diabetic patients, particularly those with ESRD, are not able to mount an effective immunologic response to the invading pathogen.

-**Blood Pressure**: Hypotension is a sign of sepsis and non-descript measure of infection.

-**Heart Rate**: Tachycardia is a sign of sepsis and non-descript measure of infection.

-**Respiratory Rate**: Increased respiratory rate is a sign of sepsis and non-descript measure of infection.

-**Pain Level**: Important to document and follow. Has been deemed the "5th vital sign" by JCAHO.

-**Glucose Levels**: AJM considers blood glucose level the "6th vital sign" and can be one of the most important quantitative measurements of infection and response to therapy. Research indicates that the immune system is significantly impaired and essentially not working at levels as high as 150-175 ml/dL. (The *Portland Diabetic Project* is a good place to start reading about this. Also see *Inzucchi SE. Management of Hyperglycemia in the Hospital Setting. NEJM. Nov 2006. 355;18: 1903-11*). Also see the Sheet on "Glycemic Control" on page 72.

-Physical Exam

-**Derm**:

-**Wound Characteristics**: There are several classification systems you need to know for describing wounds including:

-**Wagner Classification**

-**University of Texas Health System Classification**

-**PEDIS Classification** used by the Infectious Disease Society of America

-**Liverpool Classification** used by the Musculoskeletal Infectious Disease Society

-Acronym **3D MOBB** (**d**epth, **d**iameter, **d**rainage, **m**easure, **o**dor, **b**ase, **b**order)

-Regardless of classification, you absolutely must document certain wound characteristics and know proper wound terminology:

-**Base**: -Exact length, width and depth; consistency (ranging from red/granular to yellow/fibrotic to black/necrotic. Estimate percentages for mixed bases).

-Depth—Probe to bone? [Grayson JAMA 1995. 89% positive predictive value for OM]. But…

-Lavery LA. Probe-to-Bone Test for Diagnosing Diabetic Foot Osteomyelitis. Reliable or relic? Diabetes Care. Feb 2007; 30(2): 270-274.

-**Wound Edges**: -Consider hyperkeratotic, macerated, necrotic, clean, bleeding, epithelial, rolled, etc.

-Undermining? Tunneling?

-**Drainage**: -Consider serous, sanguinous, purulent (describe color), combination, etc.

-Mild, moderate, severe/heavy

-Describe any odor (This is probably Dr. Attinger's most important variable in infection assessment!)

-**Periwound skin**: -Consider normal, erythematic (document/draw extent), streaking, stasis changes, trophic changes.

-**Vascular**:

-Describe extent (anatomic level) and nature (pitting vs. non-pitting) of any edema.

-**Pulses** (DP, PP, PT, Pop, and Fem every time for a new patient if needed)

-Always doppler if nonpalpable at each level

-**CFT, Pedal Hair**

-**ABI**: -Values >0.9 associated with good healing potential

-Values 0.5-0.9 associated with PAD and delayed healing

-Values <0.5 associated with ischemia and problematic healing

-Be wary of elevated values secondary to vessel calcification

-**TcPO2**: -Values >30mmHg associated with good healing potential *[Mars M. Transcutaneous oxygen tension as a predictor of success after an amputation. JBJS-Am. 1988; 70(9): 1429-30.]*

-Values <20mmHg associated with microcirculatory problems and delayed/problematic healing.

-**Absolute Pressures**: -Should have at least 40mmHg at ankle and 20mmHg at the digits for healing potential.

-**Absolute/Relative skin temperature:** compare B/L (normal around 94° F).

-Any other relevant vascular testing.

-**Neurologic**:

-Include testing for sensory, motor and autonomic neuropathy

-**Sensory testing**:

-Posterior column: Vibratory, Proprioception

-Anterior column: Light touch (5.07 SWMF)

-Lateral column: Pain and temperature

-**Motor testing**:

-Expect intrinsic weakness with advanced neuropathy

-Manual Muscle Testing

-Spinal Reflexes (Achilles, Patellar, Babinski)

-**Autonomic**:

-Increase in skin temperature

-Lack of sweating leading to xerosis

-Any other relevant neurologic tests (you should have an awareness of Dellon's work and the PSSD).

-**Orthopedic**:

-Document any/all foot deformities, especially osseous prominences.

-Expect intrinsic muscle weakness leading to digital deformities.

-Overall foot type

-Equinus

AJM Sheet: Specific Wound Classification Systems

-Wagner Classification: *[Wagner FW: The dysvascular foot: a system of diagnosis and treatment. Foot Ankle 2: 64–122, 1981]*

0: Pre-ulcerative area without open lesion
1: Superficial ulcer (partial/full thickness)
2: Ulcer deep to tendon, capsule, bone
3: Stage 2 with abscess, osteomyelitis or joint sepsis
4: Localized gangrene
5: Global foot gangrene

-Modifed with the following risk factors:
-A: Neuropathic
-B: Ischemic
-C: Neuroischemic

-So an infected ulcer with localized gangrene and bone exposure on a fully sensate, ischemic foot is: Wagner 4B.

-University of Texas: *[Lavery LA, Armstrong DG, Harkless LB: Classification of diabetic foot wounds. J Foot Ankle Surg 35:528–531, 1996]*

	0:	1:	2:	3:
A:	No open lesion	Superficial Wound	Tendon/Capsule	Bone/Joint
B:	With infection	With infection	With infection	With infection
C:	Ischemic	Ischemic	Ischemic	Ischemic
D:	Infection/Ischemia	Infection/Ischemia	Infection/Ischemia	Infection/Ischemia

-So an infected ulcer with localized gangrene and bone exposure on a fully sensate, ischemic foot is: UT-3D.

-PEDIS System: *[Lipsky BA, et al. Diagnosis and Treatment of Diabetic Foot Infections. IDSA Guidelines. CID 2004; 39: 885-910].*

-Recommended by the Infectious Disease Society of America.
-PEDIS is an acronym standing for **p**erfusion (measure of vascular supply), **e**xtent/size, **d**epth/tissue loss, **i**nfection, and **s**ensation.
-Each of the 5 categories is graded from 0 (minimal) to 2 (severe).
-Based on a 10-point scale with 10 being most serious ulcer with greatest difficulty in treatment.

-WIfI Classification: [*Mills, Conte, Armstrong et al 2014. Pubmed ID#: 24126108]*

	-**W**ound:	-**I**schemia	-**f**oot **I**nfection
Grade 0:	-No ulcer, no gangrene	-ABI > 0.80	-IDSA uninfected
Grade 1:	-Superficial ulcer	-ABI 0.6-0.79	-IDSA mild infection
Grade 2:	-Exposed deep structures	-ABI 0.4-0.59	-IDSA moderate infection
Grade 3:	-Extensive tissue loss	-ABI < 0.39	-IDSA severe infection

-**Liverpool Classification System**:

-Primary: -Neuropathic
-Ischemic
-Neuroischemic

-Modified with: -Uncomplicated
-Complicated (cellulitis, abscess, OM, etc.)

-**IDSA Diabetic Foot Infection Severity Classification System**: [available at: https://academic.oup.com/cid/article/54/12/e132/455959]

-Uninfected: No signs/symptoms of infection
-Mild infection: Defined by presence of two of the following: Local swelling/induration, erythema, local tenderness or pain, Local warmth, or purulent drainage.
-Moderate infection: Mild infection with erythema >2cm, or involving structures deep than skin and subcutaneous tissues, and NO systemic inflammatory response signs.
-Severe infection: Infection with the signs of SIRS defined by 2 or more of the following: Temperature >38C or <36C, heart rate >90bpm, respiratory rate >20 breaths per minute, or WBC >12,000.

AJM Sheet: Diabetic Infection Objective Laboratory Results: Basic

-Complete Blood Count (CBC) with Differential:

-**Total Leukocyte Count (~4-10 x 10^3 leukocytes/ul)**

-**Leukocyte** is a generalized term for any WBC including neutrophils/granulocytes, monocytes, lymphocytes, eosinophils and basophils. So an increased leukocyte count can indicate a rise in any or all of these. This is the reason why a differential is so important.

-**Neutrophils/Granulocytes** (Usually ~54%; increased >85%)

-Part of the humoral system.

-Phagocytic cells in the inflammatory process.

-Normally take 8-14 days to mature. Functionally last 1-2 days. Half-life ~6 hours.

-Would be increased in an inflammatory state.

-**PMNs**: Mature neutrophils that you would expect to see in an infection.

-**Band cells**: Immature neutrophils. Presence indicates active, ongoing infection.

-A **left shift** is an **increased neutrophil percentage in the presence of band cells**.

-**Monocytes** (Usually ~6%)

-Phagocytic, bacteriocidal macrophages in the humoral system.

-Accumulate after neutrophils in acute infection.

-Presence indicates post-inflammatory state or chronic infection.

-**Lymphocytes** (Usually ~37%)

-Part of the cellular system.

-Produce immunoglobulins and express cellular immunity (T and B cells).

-Not normally increased in bone/soft tissue infections.

-Possibly increased in a foreign body reaction.

-Some evidence that you actually want this number **>1500** for proper immune function

-**Eosinophils** (Usually ~2%)

-Part of the cellular system.

-Generally involved in allergic and immune responses.

-Develop in the same line as lymphocytes.

-Increased with acronym **NAACP**

-(Neoplasm, Allergy, Addison's, Collagen vascular disorder, Parasites)

-**Basophils** (Usually ~0.5%)

-Part of cellular immunity.

-Involved with acute allergic responses and histamine release.

-**Leukocytosis** is an increased WBC. The absolute count tells you very little, but trending can be very important. **An increased leukocyte count indicates an increased level of inflammation, not necessarily infection.** Keep in mind that there are many other causes of leukocytosis besides infection.

-[Armstrong DG. Leukocytosis is a poor indicator of acute osteomyelitis of the foot in DM. JFAS 1996 Jul-Aug; 35(4): 280-3.]

-Drugs: Lithium, Corticosteroids

-**Leukopenia** is a decreased WBC. This could lead to a normal WBC in the presence of infection.

-Drugs: Methotrexate, Phenybutase, Dilantin, Salicylates

-Chem-7/Metabolic Panel

-Little information about specific infection, but insight into general health of patient.

-Generally:

-General increased concentrations: Dehydrated state

-Acidosis: Non-descript finding in infection

-Increased BUN: Dehydrated state

-BUN/Cr: Renal function which has antibiotic consequences

-Minerals (Ca, Mg, P)

-Abnormal in renal dysfunction with long term vascular consequences.

-Glucose, HbA1c

-Long-term effects of hyperglycemia discussed in pathogenesis section.

-HbA1C: Measure of glycosylated hemoglobin and long-term glucose control:

-1% equals approximately 20 glucose points (7% equals ~140ug/ul)

-Note that the stress of infection will probably cause a hyperglycemic state.

-H&H, Coags

-Essential to know if you are planning surgery.

AJM Sheet: Diabetic Infection Objective Laboratory Results: Advanced

-**Erythrocyte Sedimentation Rate (ESR)**

-Normal: **<20mm/hr** Moderate elevation: **20-60mm/hr** Severe elevation: **>60mm/hr**

-Analyzed using the **Westergren method**, which measures the distance erythrocytes fall in one hour in a vertical column of anti-coagulated blood under the influence of gravity (even though gravity is just a theory).

-Sensitive, but not specific for infection as it is increased in any inflammatory state with increased fibrinogen.

-Also elevated in: Pregnancy, DM, ESRD, CAD, CVD, Malignancy, Age, etc.

-*[Karr JC. The diagnosis of osteomyelitis in diabetes using ESR. JAPMA 2002 May; 95(5): 314.]*

-*[Lipsky BA. Bone of contention: diagnosing diabetic foot osteomyelitis. Clin Infect Dis. 2008 Aug; 47(4): 528-30.]*

-**C-Reactive Protein (CRP)**

-Normal: **0-0.6mg/dl**

-Measures a liver protein only present in acute inflammation (not normally found at all).

-Sensitive, but not specific for infection.

-Also elevated in: RA, Malignancy, MI, SLE, Pregnancy, etc.

-More expensive and technically difficult to perform compared to the ESR.

-*[Jeandrot A. Serum procalcitonin and CRP concentrations to distinguish mildly infected from non-infected diabetic foot ulcers: a pilot study. Diabetologia. 2008 Feb; 51(2): 347-52.]*

-**Procalcitonin?**

-New kid on the block with respect laboratory analysis of inflammation and infection (but very expensive)

-**Nutrition Analysis**

-**Albumin**:

-Normal Value: **3.6-5g/dl**

-Value decreased with inflammation and malnutrition.

-Transport protein in liver with important functions in catabolism.

-**Pre-albumin**

-Normal Value: **19-36 mg/dL**

-The topic of nutrition is not covered well in PRISM, but I would recommend checking out: *Arnold M. Nutrition and Wound Healing. Plast Reconstr Surg. 2006 Jun; 117(7 Suppl): 42S-58S.*

-**Wound Culture and Sensitivity**

-Wound cultures are still a hotly debated topic because of controversies regarding contamination, colonization and defensive medicine. Suffice to say that swab cultures are easily contaminated by normal cutaneous flora, so should be taken as deeply as possible without surface contamination. The ideal situation is a deep wound specimen of tissue (not just a swab) following incision and drainage with pulse lavage before beginning antibiotic therapy.

-**Gram Stain** (results usually within 24 hours)

-PMNs if present: Do not overlook! Presence indicates inflammation.

-Presence of any organism: essentially irrelevant.

-**Preliminary** (results usually within 48 hours)

-Gram stain nature (positive/negative) and shape (cocci/bacillus) of any organism

-See chart of common organisms on next page

-Clues you into organism much more than the gram stain

-Continue Abx unless you are really off-base

-**Final** (results usually within 72 hours)

-Should always get sensitivities.

-Allows for conclusive Abx planning.

-**Blood Cultures**

-Should be drawn from 2 sites; 20 minutes apart.

-Indicates bacteremia/septicemia

-**Bone Biopsy**

-"Gold standard" for diagnosis of osteomyelitis (discussed further later)

-If patient is a surgical candidate, then consider:

-**CXR**

-**EKG**

AJM Sheet: Gram Stain Results with Common Infective Agents:

-Aerobic Gram Positive Cocci

Staph aureus
MRSA
Staph epi
MRSE
Enterococcus
VRE
Strept pyogenes (Group A)
Strept agalactiae (Group B)
Strept bovis (Group D)
Strept Viridans

-Anaerobic Gram Positive Cocci

Peptostreptococcus

-Aerobic Gram Positive Rods

Bacillus anthracis
Corynebacterium diphtheriae
Listeria Monocytogenes

-Anaerobic Gram Positive Rods

Clostridium perfringens
Clostridium difficile
Clostridium tetani

-Aerobic Gram Negative Rods

Pseudomonas
E. coli
Enterobacter
Proteus
Vibrio
Y. pestis
Shigella
Salmonella
Klebsiella
Serratia
E. Corrodens
P. multicide

-Anaerobic Gram Negative Rods

Bacteroides fragilis

-Aerobic Gram Negative Cocci

Neisseria

-Spirochetes

Treponium pallidum
Borrelia burgdorferi

AJM Sheet: Imaging in Diabetic Foot Infections

-Plain Film Radiographs

-**Soft Tissue**: Infection characterized by radiolucent area. One should be able to see a soft tissue deficit if an ulcer is present. It is very important to rule out emphysema (gas in the tissues) with a plain film. Can also appreciate soft tissue edema.

-**Osseous Tissue**:

-Early Osteomyelitis (OM) signs: No reliable ones. Rarefaction and periostitis are possible.
-Subacute OM signs: Brodie's abscess (lytic lesion surrounded by sclerotic rim).
-Chronic OM: Lysis, Malformation, Involucrum, Cloaca, Sequestra.
-Plain film radiographs are 67% specific, 60% sensitive for OM [*Termaat, JBJS 2005*]

-MRI

-**Cellulitis**: **T1**: Diffuse and infiltrative decreased signal intensity as inflammation replaces fat.
T2 and STIR: Increased signal intensity.

-**Abscess**: **T1/T2/STIR**: Homogeneous increased signal intensity.
-Note that pus/necrotic tissue has a decreased intensity compared to inflammatory fluid.

-**OM**: **T1**: Decreased signal intensity, cortical lysis and intramedullary changes.
-Increased signal intensity in known OM indicates healing as fat infiltrates.
T2: Increased signal intensity, cortical lysis, and intramedullary changes.
-**Rim sign**: thin layer of active infection surrounding normal bone.

-60% Specificity, 85% sensitivity per Termaat.

-Bone Scans

-A radio-isotope is injected into the patient and imaged at specific intervals.

-Phases: -**Immediate Angiogram (1-3sec)**: Essentially an arteriogram.
-**Blood Pool (3-5min):** Demonstrates blood pooling in capillaries and veins.
-**Delayed (2-4 hours):** Increasingly specific to activity patterns and pathology.
-**4th Phase (varying times)**: Increasingly specific to activity patterns and pathology.

-**Technetium-99 Bone Scan**

-Binds to calcium hydroxyapatite and measures osteoblast/osteoclast activity.
-Half-life: 6 hours
-Excreted through kidneys which will show homogenous control signal.
-**Mucomyst** 600mg PO q12 day before and of surgery as renal ppx.
-**Cellulitis**: Focal uptake in blood pool; Negative in delayed phase.
-**OM**: Diffuse uptake in blood pool; Hot increased uptake in delayed phases.
-45% Specificity; 86% sensitivity per Termaat.

-WBC Scans

-Same principles and phases as bone scan, but WBCs are tagged and followed instead.

-Gallium-67 Citrate

-Uptake by **siderophore** complex (direct bacteria) and **lactoferrin** (protein-released by bacteria)
-Scan taken 48-72 hours after injection or done in triphasic manner.
-Has longer half-life
-42% Specificity; 80% Sensitivity per Termaat.

-Indium-111 Oxime

-WBCs isolated from blood sample, labeled and re-injected.
-Scan at 24 hours. Half life: 67 hours.
-Predominantly uptaken by neutrophils, so it demonstrates acute infections better than chronic infections.

-Technetium-99m HMPAO

-WBCs isolated from blood samples, tagged and re-injected. Scan at 3 hours.
-Tagged molecule is HMPAO (**hexamethylpropyleneamine oxime → you will get asked this!**)
-Technically easier with less radiation than indium.

-Technetium-99m Sulfur Colloid Marrow Scan

-Specific for bone marrow and neutrophil production
-Has shown promise in differentiating OM from Charcot

-Combination Sequential Technetium-Gallium Scans

-Scan at 4 hours, then at 48-72 hours. Based on half-lives.
-Increased specificity for infection if gallium has higher uptake then technetium.
-Can use any other combination.

-Computed Tomography (CT scans)

-Radiograph altered by computer to highlight specific "windows". You can isolate soft tissue or different aspects of bone, for example.
-**Soft tissue infection**: Exact locations and anatomy of abnormal soft tissue density.
-**OM**: Increased density in the marrow.
-CT scans can be combined with contrast.

-Positron Emission Tomography (PET scans)

-A **tracer** is injected/inhaled into the patient which releases radioactive **positrons**. The positrons collide with electrons and produce gamma rays.
->90% Sensitivity and Specificity per Termaat (best in study).

AJM Sheet: Diabetic Foot Ulcer Pathogenesis

-The pathogenesis of the diabetic foot ulcer can be described via three mechanisms: neuropathy, trauma and impaired healing.

-**Neuropathy**

-30-50% of diabetics have some form of sensory, motor and/or autonomic neuropathy.

-Sorbitol accumulation in Schwann cells leads to hyperosmolarity of the nerve cells in turn leading to swelling and cellular lysis. This leads to decreased nerve signal conduction. Microvascular damage to the nerve (described later) also impairs healing of the damaged nerve.

-Sensory Neuropathy

-Loss of light touch/protective sensation (anterior spinothalamic tract)

-Loss of vibratory/proprioception mechanisms (posterior tract)

-Loss of pain/temperature sensation (lateral tracts)

-The patient has no warning of current, developing or impending trauma.

-Motor Neuropathy

-"Intrinsic Minus" foot-type with wasting of the intrinsic muscles and extensor substitution.

-Undetected excess plantar pressures develop.

-Autonomic Neuropathy

-Damage occurs in the sympathetic ganglion

-AV shunting occurs with global LE edema not relieved by diuretics or elevation.

-Increased skin temperature predisposes to ulceration (*Armstrong*)

-Decreased sweating leads to xerosis and fissuring (portal for infection)

-Trauma leading to Ulceration

-Abnormal anatomy: extrinsic and intrinsic abnormalities secondary to motor neuropathy and tendon glycosylation.

-Decreased joint mobility: secondary to non-enzymatic glycosylation and excess collagen cross-linking of tendons, ligaments, joint capsules (especially at the STJ and the MTPJ).

-Equinus: Increased cross-linking of collagen in the Achilles tendon (leads to increased forefoot pressures).

-Skin stiffness: secondary to glycosylation of keratin.

-Intrinsic skin weakness: trophic changes associated with PVD.

-All lead to increased plantar pressures, which is the driving force behind ulceration.

-Impaired Wound Healing

-Can be thought of as increased inflammation, decreased vasculature and decreased catabolism.

-Increased Inflammation

-The inflammatory phase of the healing process actually lasts longer than necessary.

-Inflammation initially not as effective due to decreased leukocyte adhesion and morphologic changes to the macrophages.

-Prolongation occurs due to decreased chemotaxis of growth factors and cytokines.

-MMPs increase their activity and continue to produce an "inflammatory soup."

-All contribute to a wound "stuck" in the inflammatory phase.

-Decreased Vasculature

-Macroangiopathy: Atherosclerotic obstructive disease of large vessels due to LDL oxidation.

-Microangiopathy: Thickened basement membrane decreases diffusion at capillary level.

-Mechanism behind neuropathy, nephropathy and retinopathy

-Think of it in terms of a decreased TcPO2

-Endothelial dysfunction:

-Decreased nitric oxide (NO) and prostaglandin to promote vasodilation

-Decreased smooth muscle cell relaxation to promote vasodilation

-AV shunting secondary to autonomic neuropathy

-Decreased vasodilation and membrane permeability in response to trauma/damage/inflammation:

-Usually regulated by substance P and vasomodulators from damaged cells and nociceptors.

-Overall leads to a "sluggish" vasculature with decreased inflow, diffusion, outflow and angiogenesis.

-Decreased Catabolism

-Decreased collagen synthesis, both in peptide production and post-translational modification

-Morphologic changes to keratinocytes

-Decreased angiogenesis

Further Reading:

-Shaw JE, Boulton AJ. The pathogenesis of diabetic foot problems: an overview. Diabetes. 1997 Sep; 46 Sep; Suppl 2:S58-61.
-Rathur HM, Boulton AJ. The diabetic foot. Clin Dermatol. 2007 Jan-Feb; 25(1): 109-20.
-Boulton AJ, et al. The global burden of diabetic foot disease. Lancet. 2005 Nov 12; 366(9498): 1719-24.

AJM Sheet: Diabetic Foot Infection Functional Anatomy

From: *Essential Questions for Surgical Intervention of Diabetic Foot Infections* (http://www.podiatrytoday.com/article/8134)

Dedicating yourself to the side of limb salvage in the fight against diabetic foot disease is a demanding and personally challenging enterprise. In the face of infection, it often seems as though all variables are against the surgeon and the patient as together, you struggle against proximal amputation and limb loss. In fact, it often appears as though the only constant is the unpredictability of the disease progression. But one constant always on the side of the surgeon is anatomic knowledge. The infection can only work with the anatomy that it is given, and this is certainly something that can be used to your advantage. Your expert knowledge in lower extremity anatomy is one of the most valuable tools that you have in your fight. It is a constant, and it is predictable.

There are different anatomic paradigms that must be considered in terms of the evaluation of the infection source. Certainly depth is one of these paradigms. Absolute depth measurements offer very little clinical information when compared to a functional view of depth from the **surgical layers of dissection**. An infection should be evaluated in terms of whether it extends through the dermis, superficial fascia, deep fascia, musculotendinous structures or to the level of bone (Table 1). From this general information, specific anatomic structures can then be identified as being within the path of the infection.

Infections tend to develop and travel along the path of least resistance. This implies that an infection will stay within the potential space of a given surgical layer or plantar compartment before extravasation into another layer or compartment. Often, this involves proximal extension along the relatively avascular tendon sheaths or fascial planes between muscular layers. The studies that have been used to define the number and boundaries of plantar foot compartments have also given information about relatively consistent fascial clefts where communication between different layers and compartments is likely. These have involved pressurized injection imaging studies where a known compartment is infiltrated with a contrast medium and the extravasation into other compartments can be mapped. The findings of these studies are summarized in Table 2. These communications are obviously numerous and complex. The important concept to realize is that an infection is likely to initially develop within the potential space of a single layer or compartment. There is a tendency for the infection to move proximally or distally before communicating with another layer or compartment. Note however, that patterns of communication are present along known anatomic structures such as tendons and neurovascular structures to each of the other compartments, as well as the dorsum of the foot and plantar superficial fascia. Intra-operative investigation of an infection should focus on these structures to trace the extent of plantar involvement. Also note the majority of these communications are found in the forefoot around MPJ level, so distal infections have an increased likelihood of multi-compartment involvement.

Table 1: Surgical Layers of Dissection Used for Diabetic Ulcer Depth Measurement

Skin
Superficial Fascia
-First Dissection Interval containing superficial neurovascular structures
Deep Fascia
-Second Dissection Interval containing muscular and deep neurovascular structures
Periosteum
-Third Dissection Interval
Bone

Table 2: Intercompartmental Communications

Medial Compartment
To Central Compartment via:
-Adductor Hallucis tendon
-Flexor Hallucis Longus tendon
-Peroneus Longus tendon
-Neurovascular structures penetrating the medial IM septum
To Distal Deep Leg via:
-Flexor Hallucis Longus tendon

Lateral Compartment:
To Central Compartment via:
-Long flexor tendon to 5th digit
-Short flexor tendon to 5th digit
-Lumbrical muscle to 5th digit
-Plantar interosseous muscle to 5th digit
-Peroneus Longus tendon
-Neurovascular structures penetrating lateral IM septum
To Dorsal Structures
To Plantar Superficial Fascia

Central Compartment
To Medial Compartment via:
-Adductor Hallucis tendon
-FHL Tendon
-PL Tendon
-NV structures penetrating medial IM septum
To Lateral Compartment via:
-Long flexor tendon to 5th digit
-Short flexor tendon to 5th digit
-Lumbrical muscle/tendon to 5th digit
-Plantar interosseous muscle to 5th digit
-PL tendon
-NV structures penetrating lateral IM septum
To Distal Deep Leg via:
-FHL tendon
-FDL tendon
To Dorsum of Foot via:
-Interosseous muscles
-MPJ communications
To Plantar Superficial Fascia

AJM Sheet: Osteomyelitis General

-Osteomyelitis is a complicated issue dealing with diabetic foot infections both in diagnosis and treatment. However, there are several definitions, classification systems, diagnostic modalities and treatment tenets that you should be aware of.

-Definitions per Resnick:

-**Periostitis**: Inflammation of the periosteum
-**Osteitis**: Inflammation of the cortex
-**Osteomyelitis**: Inflammation of the medullary canal
-**Sequestrum**: piece of dead bone floating in pus/inflammation
-**Involucrum**: sheath of bone surrounding pus/inflammation
-**Cloaca**: tract through an involucrum
-**Brodie's Abscess (1832)**: Chronic abscess in bone surrounded by sclerosis
-**Sclerosing OM of Garre**: low grade inflammatory condition

-Waldvogel and Lew *[Osteomyelitis. NEJM. 1997; 336(14): 999-1007.]*

-The classic Waldvogel and Lew article is NOT a classification, rather a series of definitions. However, it can be turned into a stratified classification based on their definitions.

-**Acute Osteomyelitis**: Systemic clinical signs of infection
-**Chronic Osteomyelitis**: Subacute clinical signs of infection
-**Contiguous/Direct Extension**: spread of infection to bone from exogenous source or adjacent tissue. This can be described as an "outside-in" spread invading the cortex and proceeding to the medullary canal.
-**Hematogenous Spread**: Infective agent reaches medullary canal of bone from the vascular supply. This can be described as an "inside-out" infection invading the medullary canal first and spreading to the cortex.
-**Vascular Impairment**: Decreases the effectiveness of the inflammatory response and Abx delivery.

-Others have done a little better job of differentiating acute vs. chronic OM:

-Weiland: Describes chronic OM as lasting > 6months.
-Schauwecker: Describes chronic OM as lasting > 6 weeks and one failed episode of tx.

-AJM turned these definitions into a classification system that made more sense to him:

- -Acute Osteomyelitis
  - -Contiguous/Direct Extension
    - -No PVD
    - -PVD
  - -Hematogenous Spread
    - -No PVD
    - -PVD
- -Chronic Osteomyelitis
  - -Contiguous/Direct Extension
    - -No PVD
    - -PVD
  - -Hematogenous Spread
    - -No PVD
    - -PVD

-Cierny-Mader-Penninck Classification *[A clinical staging system for adult osteomyelitis. CORR. 2003; (414): 7-24.]*

-This is described as a classification, but never made much sense to me.

-Anatomic Stage

Stage 1: **Medullary**: infection of only the medullary canal
Stage 2: **Superficial**: infection of only the superficial cortex
Stage 3: **Localized**: infection of only the cortex
Stage 4: **Diffuse**: infection of both the cortex and medullary canal

-Physiologic Stage

A: Normal Host
Bs: Compromised Host with systemic risk factors (eg DM)
Bl: Compromised Host with local risk factors (eg smoking)
C: Treatment worse than the disease

-So a smoking DM patient with infection of only the superficial portion of the cortex is: 2Bs
-Obviously much more work needs to be done on the topic of osteomyelitis classification.

AJM Sheet: Osteomyelitis Diagnosis and Treatment

-**Subjective Findings** (See Diabetic Infection Work-up)
-**Objective Findings** (Diabetic Infection Work-up)
-Probing to bone 89% positive predictive value (*Grayson JAMA 1995*).

-**Gold Standard for Diagnosis**: Bone biopsy. This is ideally performed when the patient has been free of antibiotics for ~2 weeks.
-[Or is it?: See PubMed ID#: 21907594]
-[Also: Berendt AR et al. Diabetic foot osteomyelits: a progress report on diagnosis and a systematic review of treatment. Pubmed ID#: 18442163]

-**Imaging Studies**:
-Review plain films, MRI, Bone Scans, WBC scans, CT, PET in Diabetic Infection Imaging Sheet.
-These all add evidence, but are rarely exclusively diagnostic.
-**Blood Cultures**: Hematogenous OM diagnosed with positive BCx and positive bone scan.

Treatment
-There is much controversy regarding long-term Abx (PO vs. IV vs. PMMA) vs. Surgical Debridement.
-The Cierny-Mader Classification makes some general recommendations, but I prefer the IDSA ones further below:
-CM Stage 1: 2 weeks IV + 2-4 weeks PO Abx
-CM Stage 2: Surgical Debridement + 2 weeks IV
-CM Stage 3&4: Surgical Debridement + 4-6 weeks IV Abx

-Antibiotic administration options
-Long-term Abx (4-8 weeks) is a conservative option because many people believe you can never cure OM and that it can reactivate at any time for years to come.
-PO
-**Doxycycline** and **Ciprofloxacin** are reputed to have the best bone penetration.
-Keep in mind that most ID docs would never substitute coverage for bone penetration.
-Your Abx choices should be culture driven.
-IV
-Culture driven
-Access options: IV, PICC, Infusion pump, etc.
-PMMA beads
-PMMA: **polymethylmethacrylate**
-PMMA is a combination of monomer (liquid) and polymer (powder).
-Comes in 20, 40 and 60g packets.
-7**% elusion in the first 24 hours with activity noted for 14 days**.
-Demonstrates exponential release.
-Cierny proposes a **1:5 ratio of Abx:PMMA**. Another common standard is **4-8g:40-60g**.
-Increased Abx means increased elution, but decreased bead hardening.
-Smaller beads means increased overall surface area and increased elution.
-The Abx must be heat-stable
-Gentamycin, Tobramycin, Vancomycin, Ticarcillin, Cefazolin, Moxalactam, Cefotaxime

IDSA General Treatment Recommendations [*Lipsky BA, et al. Diagnosis and Treatment of Diabetic Foot Infections. IDSA Guidelines. CID 2004; 39: 885-910.]*

Site, severity or extent of infection	Route	Duration
Soft Tissue Only		
Mild	Topical or oral	1-2 weeks up to 4 weeks
Moderate	Oral	2-4 weeks
Severe	Initial parenteral, then switch to oral	2-4 weeks
Bone or Joint		
No residual infection	Parenteral or oral	2-5 days
Residual infected soft tissue	Parenteral or oral	2-4 weeks
Residual infected (but viable) bone	Initial parenteral, then switch to oral	4-6 weeks
Residual dead bone post-operatively	Initial parenteral, then switch to oral	>3 months

AJM Sheet: Charcot Neuroarthropathy

-**Definition**: Neuropathic osteoarthropathy first described by **Musgrave in 1703** and named for **JM Charcot in 1868**.
-**Pathogenesis**:
- -**Neurovascular/French Theory/Theory of Charcot**
  - -Trophic centers in the anterior horn of the spinal cord maintain nutrition to joints.
  - -Trauma to these trophic centers leads to increased blood flow and osteoclastic activity.
  - -Evidence for the Neurovascular Theory:
    - -Autonomic neuropathy in DM leads to increased AV shunting, edema and skin temperature.
    - -Boulton: Increased PO2 in venous system of Charcot pts (63mmHg vs. 46mmHg)
      - -Shows increased perfusion in neuropathic diabetics
    - -Edmonds: Increased blood velocity in neuropathic diabetics
    - -Young: Decreased bone density in patients with decreased nerve conduction velocities
    - -Cundy: Decreased bone density in Charcot patients
    - -Gough: Increased osteoclastic activity in Charcot patients
- -**Neurotraumatic/German Theory/Theory of Virchow and Volkmann**
  - -Repeated trauma from biomechanical stresses during ambulation on an insensate foot.
  - -Evidence for the Neurotraumatic Theory:
    - -Eloesser and Johnson: Trauma is the necessary predisposing factor, and not underlying bone weakness, to create Charcot changes in a neuropathic limb.
    - -Common sense
- -*Two opposing, fighting theories (with two opining blowhards on either side getting red-faced)?:* **Probably a little bit of both**.

-**Etiology**: Anything that causes neuropathy!
- -First described: Tabes Dorsalis (Charcot 1868)
- -Most common: DM
- -3 most common: DM, Syringomyelia (longitudinal cavities lined by dense tissue), and Tabes Dorsalis
- -C: Myelomeningocele, Spina Bifida, CMT, MS, CP, Syringomyelia, Congenital insensitivity
- M: DM, Alcoholic neuropathy, Uremia, Pernicous Anemia
- I: Tabes Dorsalis, Polio, Leprosy, TB
- N: Tumors in brain, spinal cord, peripheral nerve
- T: Trauma to brain, spinal cord, peripheral nerve
- D: Indomethacin, Intra-articular corticosteroids, phenylbutazone

-**DDx**: OM, AVN, inflammatory arthritis, PVS, septic arthritis, CPPD, neoplasm, etc.

-**Clinical Findings**:
- -Presents similar to infection
- -Red, hot, swollen, deformed foot +/- pain
- -Neuropathic
- -Readily palpable pulses (often described as bounding)

-**Radiographic Findings**:
- -**Atrophic**:
  - -With osteopenia, pencil&cup deformities, resorption of bone ends
  - -Without osteophytes, sclerosis, fragmentation, soft tissue debris
- -**Hypertrophic**:
  - -With joint space narrowing, fractures, fragmentation, ST debris, periosteal rxn, subluxation
  - -Without osteoporosis
- -Be aware of both types.

-**Classification Systems** (described in detail on next page)
- -Eichenholtz Classification
- -Brodsky Classification
- -Schon Classification

-**Treatment**
- -**Acute**:
  - -Strict and immediate NWB and immobilization for 12-16 weeks.
  - -Edema control (Jones cast, ACE inhibitors, Diuretics, Posterior splint, Elevation, Ex Fix, etc.)
  - -Education and family support
  - -FXR every 4-6 weeks with relatively few cast changes
- -**Transition**:
  - -Transition to WB (CAM walker, CROW, Bracing, MAFO, Shoes, etc.)
- -**Permanent**:
  - -Surgical correction of underlying deformity
  - -Consider TAL, Arthrodesis, Wedging osteotomies, Amputation
- -**Adjunctive**:
  - -Bone stimulators
  - -Bisphosphonates:
    - -Pamidronate (Aredia): 60-90mg over 24h. 3 doses in 2 weeks.
    - -Alendronate (Fosamax): 5mg PO q24h.

AJM Sheet: Charcot Classifications

-**Eichenholtz Classification** (1966)

[Eichenholtz SN. Charcot Joints, p7-8, Springfield, Illinois, Charles C Thomas, 1966]

-Based on plain film radiographic findings
-Originally described Stages 1→3, but Stage 0 added later (Yu typically given credit, but really Schon).
-*[Yu GV, Hudson JR. Evaluation and treatment of stage 0 Charcot's neuroarthropathy of the foot and ankle. JAPMA. 2002; 92(4): 210-20.]*
-**Stage 0**: High risk pre-Charcot
-Radiograph: Unremarkable. Maybe increased ST density, bone flecks or change in foot architecture.
-Clinical: Sudden onset of non-pitting edema, erythema, calor, +/- pain, bounding pulses, intrinsic atrophy.
-Normal skin temp: 94°F; can increase by 12°
-Uptake in all three phases of Tc-99 bone scan
-**Stage 1**: Acute/Developmental
-Radiograph: Capsular distention, fragmentation, debris, subluxation
-Clinical: Red, hot, swollen foot with joint laxity
-**Stage 2**: Coalescence
-Radiograph: Sclerosis, resorption of debris, fusion
-Clinical: Subjectively decreased red, hot, swollen
-**Stage 3**: Reconstruction
-Radiograph: Decreased sclerosis (with increased vascularity) and remodeling
-Clinical: Decreased joint mobility with increased stabilization

-**Brodsky Classification** (1992)

[Brodsky JW. The diabetic foot. In: Coughlin and Mann's 1992 edition.]
-Describes location of deformity

-**Type 1**: Lisfranc joint (27-60% incidence)
-**Type 2**: Chopart's joint and STJ (30-35% incidence)
-**Type 3A**: Ankle joint (9% incidence)
-**Type 3B**: Posterior calcaneus
-**Type 4**: Multiple combinations of above
-**Type 5**: The forefoot

-**Schon Classification**

[Charcot neuroarthropathy of the foot and ankle. CORR. 1998; 349: 116-131.]
-Describes location and severity of condition
I: **Lisfranc Pattern**
-A→C with increasing deformity to medial rockerbottom and ulceration.
II: **Naviculocunieform Pattern**
-A→C with increasing deformity to lateral rockerbottom and ulceration.
III: **Perinavicular Pattern**
-A→C with lateral rockerbottom, Talar AVN and ulceration.
IV: **Transverse Tarsal Pattern**
-A→C with increasing deformity to central rockerbottom and ulceration.

-**Sanders and Frykberg Classification**:
-Anatomic classification: In *The High Risk Foot in Diabetes Mellitus* text.
-Pattern I: Phalanges and Metatarsophalangeal joints
-Pattern II: Tarsometatarsal joints
-Pattern III: Cuneonavicular, Talonavicular and Calcaneocuboid joints
-Pattern IV: Ankle joint
-Pattern V: Posterior calcaneus.

AJM Sheet: Differentiating Osteomyelitis from Charcot

-Please keep in mind that these are not mutually exclusive and both can be present!
-These are just general guidelines and many people will vehemently argue about them.

-The **gold standard is a bone biopsy** which would show infection in OM and not in Charcot (***maybe***).

Subjective
-**OM**: Constitutional signs and symptoms of infection, infectious risk factors, history of infection.
-**Charcot**: Uncontrolled DM, history of Charcot, history of recent trauma.

Objective
-**OM**: Necrosis, purulent drainage, elevated white count, cultures, **positive bone biopsy.**
-**Charcot**: Increased joint laxity, non-pitting edema, bounding pulses, rockerbottom deformity, **negative bone biopsy.**

Imaging
-Not enough evidence yet, but some believe that OM is positive on bone scans and WBC scans for greater than 24 hours whereas Charcot neuroarthropathy is only positive during the first 24 hours.
-The Tc99 Sulfur Colloid scan would also theoretically be positive for infection, but not for Charcot.

-Not too much here, but check out some further reading:

-Soysal N, et al. Differential diagnosis of Charcot arthropathy and osteomyelitis. Neuro Endocrinol Lett. 2007 Oct; 28(5): 556-559.
-Shank CF, Feibel JB. Osteomyelitis in the diabetic foot: diagnosis and management. Foot Ankle Clin. 2006 Dec; 11(4): 775-89.
-Ledermann HP, Morrison WB. Differential diagnosis of pedal osteomyelitis and diabetic neuroarthropathy: MR Imaging. Semin Musculoskelet Radiol. 2005 Sep; 9(3): 272-83.
-Berendt AR, Lipsky B. Is this bone infected or not? Differentiating neuron-osteoarthropathy from osteomyelitis in the diabetic foot. Curr Diab Rep. 2004 Dec; 4(6): 424-9.
-Yu GV, Hudson JR. Evaluation and treatment of stage 0 Charcot's neuroarthropathy of the foot and ankle. J Am Podiatr Med Assoc. 2002 Apr; 92(4): 210-20.
-Schon LC, et al. Charcot neuroarthropathy of the foot and ankle. Clin Orthop Relat Res. 1998 Apr;(349): 116-31.
-Berendt AT, Peters EJ, et al. Diabetic foot osteomyelitis: a progress report on diagnosis and a systemic review of treatment. Diabetes Metab Res Rev. 2008; 24(S1): S145-S161.
-Donovan A, Schweitzer ME. Use of MR imaging in diagnosing diabetes-related pedal osteomyelitis. Radiographics. 2010 May; 30(3): 723-36.

-This isn't exactly the right place for this, but while we have a little extra room, let's talk about "describing" radiographs. You can never actually "see" Charcot or Osteomyelitis (or even infection or a fracture for that matter) on radiographs. These are all diagnoses. What you can "see" or "describe" is radiologic evidence of each of these things. For each different type of imaging modality, there are actually very few descriptive terms that you should be using to describe what you see before you make a diagnosis:

-**Plain Film Radiography**:
- -Increased or Decreased
- -Radiolucency or Radiodensity

-Everything that you see on a radiograph can be described using these terms. So while you may not be able to "see" a fracture, you can describe an area of radiolucency within bone consistent with a fracture. And while you may not be able to "see" an infection, you can describe an area of radiolucency within the soft tissue consistent with emphysema.

-**MRI**:
- -Increased or Decreased
- -Signal intensity

-**Bone scans**:
- -Increased or Decreased
- -Signal uptake

-**Ultrasound**:
- -Hyperechoic or Hypoechoic

AJM Sheet: Common Situational Bugs

-Cellulitis with an open wound:
-Staph Aureus (SA) (if no streaking present)
-Strept (with streaking and palpable border)
-Usually monomicrobial

-Infected ulcer in Abx naïve pt:
-SA
-Strept
-Usually polymicrobial

-Chronically infected ulcer in Abx naïve pt:
-SA
-Strept
-Enterobacter
-Usually polymicrobial

-**Macerated infected ulcer**:
-Pseudomonas
-Usually polymicrobial

-**Chronic, non-healing ulcer with prolonged Abx therapy**:
-SA -MRSA
-Staph epi
-Enterococci -VRE
-Diptheroids (Corynebacterium)
-Enterobacter
-Pseudomonas
-Extended GNR
-Usually polymicrobial

-**Fetid Foot with necrosis and gangrene**:
-Resistant Gram positive cocci
-Mixed GNR
-Anaerobes
-Polymicrobial

-Osteomyelitis with hemodialysis:
-SA
-Enterobacter
-Pseudomonas

-Osteomyelitis with IVDA:
-SA
-Enterobacter
-Pseudomonas

-Osteomyelitis with Decubitus Ulcer:
-Gram Negatives

-Osteomyelitis with hemoglobulinopathy:
-Salmonella

-**Human mouth pathogens (HACEK):**
-Haemophilus, Actinobacillus, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae

-**Water exposure:**
-Vibrio
-Aeromonas hydrophila
-Mycobacterium

-Puncture through a shoe:
-Pseudomonas

-Any dirt/soil:
-Clostridium

-Cat bite:
-Pasteurella multocida

-Dog bite:
-Strept viridans, Capnocytophaga canimorsus

-**Immunocompromised pt:**
-Gram negatives

-**Septic bursitis**:
-SA

-Gas gangrene:
-Clostridium

-Post-op infection following implant:
-Staph epi

-Fruity odor/green hue:
-Pseudomonas

-Foul smelling discharge:
-Anaerobes

-Creamy yellow discharge:
-SA

-**White discharge**:
-Staph epi

AJM Sheet: Empiric Antibiotic Choices

Generalized Gram Positive Coverage:

-2nd Generation PCN
-4th Generation PCN
-1st Generation Cephs
-2nd Generation Cephs
-Carbapenems
-Tetracyclines
-2nd Generation Quinolones
-Macrolides
-Bactrim
-Vancomycin
-Clindamycin
-Zyvox
-Synercid
-Rifampin

Generalized MRSA Coverage:

-Vancomycin
-Clindamycin
-Zyvox
-Synercid
-Bactrim/Rifampin
-Cubicin

Generalized Gram Negative Coverage:

-3rd Generation PCN
-4th Generation PCN
-Carbapenems
-Tetracycline
-Aztreonam
-2nd Generation Quinolones
-3rd Generation Quinolones
-4th Generation Quinolones
-Bactrim

Generalized Pseudomonas Coverage:

-Cephalosporins x 3 (Fortaz, Cefobid, Maxipime)
-PCN x 2 (Zosyn, Timentin)
-Aminoglycosides
-Primaxin
-Quinolones
-Aztreonam
-**ACRONYM**: FAT CIAZ (Fortaz, Aztreonam, Timentin, Cefobid, Imipenin, Aminoglycosides, Zosyn)

Generalized Anaerobes:

-4th Generation PCN
-1st Generation Cephs
-2nd Generation Cephs
-3rd Generation Cephs
-4th Generation Cephs
-Aminoglycosides
-Carbapenems
-4th Generation Quinolones
-Clindamycin
-Flagyl

AJM Sheet: IDSA Empiric Recommendations

-from *Lipsky BA, et al. Diagnosis and Treatment of Diabetic Foot Infections. IDSA Guidelines. CID 2004; 39: 885-910.*

-Uninfected Wound

-Definition: No purulence, inflammatory manifestations, or systemic manifestations
-Empiric Therapy: None

-Mildly Infected Wound

-Definition: -2+ Manifestations of Infection (purulence, induration or erythema/pain/warmth)
-<2cm of erythema
-Limited to skin and subcutaneous tissue
-No systemic complaints

-Empiric Therapy Recommendations:

-2-PCN	-Bactrim
-Clinda	-Augmentin
-Keflex	-Levo

-Moderately Infected Wound

-Definition: -As above, in a systemically/metabolically stable patient PLUS
->2cm cellulitis OR streaking OR involvement of deep tissue

-Empiric Therapy Recommendations:

-Bactrim	-Invanz
-Augmentin	-Ceftin + Flagyl
-Levo	-Timentin
-2-Ceph	-Zosyn
-3-Ceph	-Levo + Clinda
-Daptomycin + Aztreonam	-Cipro + Clinda
-Zyvox + Aztreonam	

-Severely Infected Wound

-Definition: -Infection as above in a patient with systemic toxicity and metabolic instability

-Empiric Therapy Recommendations:

-Primaxin	-Vanco + Fortaz
-Zosyn	-Vanco + Fortaz + Flagyl
-Cipro + Clinda	
-Levo + Clinda	

-If MRSA is likely:

-Zyvox	-Vanco + Fortaz
-Zyvox + Aztreonam	-Vanco + Fortaz + Flagyl
-Daptomycin	
-Daptomycin + Aztreonam	

-**To cover all bases**:

-Vanco + Aztreonam + Flagyl

AJM Sheet: Common Infective Agents with DOC:

	DOC	Alternatives
-Aerobic Gram Positive Cocci		
Staph aureus	1-Ceph	Vanco, Clinda, Azithromycin
MRSA	Vanco	Bactrim, Cubicin, Zyvox, Clinda
Staph epi	2-PCN	4-PCN, 1,2-Ceph, Vanco
MRSE	Vanco	Zyvox, Cubicin, Synercid
Enterococcus	3-PCN	Vanco, Tetracyclines, Quinolones
VRE	Linezolid	Macrobid, Cubicin, Chloramphenicol
Strept pyogenes (Group A)	3-PCN	4-PCN, 1,2-Ceph, Vanco, Clinda
Strept agalactiae (Group B)	3-PCN	4-PCN, 1,2-Ceph, Vanco, Clinda
Strept bovis (Group D)	3-PCN	4-PCN, 1,2-Ceph, Vanco, Clinda
Strept Viridans	3-PCN	4-PCN, 1,2-Ceph, Vanco, Clinda
-Anaerobic Gram Positive Cocci		
Peptostreptococcus	Clinda	3-PCN, 4-PCN, Carbapenems
-Aerobic Gram Positive Rods		
Bacillus anthracis	Cipro	3-PCN, Vanco, Clinda
Corynebacterium diphtheriae	Macrolide	Clinda,
Listeria Monocytogenes	3-PCN	Vanco, Bactrim, Carbapenems
-Anaerobic Gram Positive Rods		
Clostridium perfringens	Ertapenam	Vanco, Clinda, 4-PCN, Tetracyclines
Clostridium difficile	Flagyl	Vanco
Clostridium tetani	Clinda	Flagyl
-Aerobic Gram Negative Rods		
Pseudomonas	Zosyn	1,2-Quin, Aztreonam, Primaxin
E. coli	3-Ceph	4-PCN, Bactrim, Quinolones
Enterobacter	Bactrim	Quinolone, Aztreonam, Carbapenems
Proteus	3-PCN	3-Ceph, 4-PCN, Bactrim, Quinolones
Vibrio	Tetracyclines	Bactrim, Cipro
Y. pestis	Aminoglycosides	Bactrim, Cipro
Shigella	Cipro	Bactrim, Amp, 4-PCN
Salmonella	Cipro	3-PCN, 4-PCN, Bactrim
Klebsiella	3-Ceph	4-PCN, Bactrim, 2-Quin, Aminoglycosides
Serratia	3-Ceph	Zosyn, Bactrim, Aztreonam, Quin
E. Corrodens	Augmentin	Tetracyclines
P. multocida	Doxycycline	Bactrim, 3-PCN
-Anaerobic Gram Negative Rods		
Bacteroides fragilis	Ertapenam	Clinda, Flagyl
-Aerobic Gram Negative Cocci		
Neisseria	Rocephin	3-PCN, Quinolones
-Spirochetes		
Treponium pallidum	1-PCN	Tetracyclines, Macrolides
Borrelia burgdorferi	1-PCN	Amox, Macrolides

AJM Sheet: Antibiotics/Drugs of Choice

1. **Staph Aureus**

-PO:	Keflex	-500mg PO tid or 750mg PO bid
	Clindamycin	-300mg PO qid
	Zithromycin	-500mg PO day 1, 250mg PO days 2-5
-IV:	Ancef	-1g IV q8
	Vancomycin	-1g IV q12
	Clindamycin	-600mg IV q8

2. **Streptococcus**

-PO:	Keflex	-500mg PO tid or 750mg PO bid
	Clindamycin	-300mg PO qid
-IV:	Ancef	-1g IV q8
	Vancomycin	-1g IV q12
	Clindamycin	-600mg IV q8

3. **MRSA**

-IV:	Vancomycin	-1g IV q12
-PO:	Bactrim	-1 tablet PO bid
	Rifampin 300mg +Minocycline	-100mg PO bid

4. **Enterococcus**

-PO:	Amoxicillin	-250-500mg tid
	Augmentin	-875mg bid or 500mg tid (or bid)
	Zyvox	-600mg PO bid
-IV:	Vancomycin	-1g IV q12
	Zyvox	-600mg IVq12

5. **VRA/VRE**

-PO:	Zyvox	-600mg PO bid
-IV:	Zyvox	-600mg IV q12
	Synercid	-7.5mg/kg/hr over 1 hour q12

6. **Pseudomonas**

-PO:	Ciprofloxacin	-250-750mg PO bid
-IV:	Ciprofloxacin	-400mg IV q12
	Fortaz	-2g IV q12
	Aztreonam	-1g IV q8

7. **E.coli, Proteus**

-PO:	Keflex	-500mg PO tid or 750mg PO bid
	Cipro	-250-750mg PO bid
	Levaquin	-500mg PO qday
	Tequin	-400mg PO qday
-IV:	Ancef	-1g IV q8
	Cipro	-400mg IV q12
	Levaquin	-500mg IV qday
	Tequin	-400mg IV qday

AJM Sheet: Antibiotic Dosing Guide

Penicillins		
1st Generation:	-Pen V:	500mg q6 PO
	-Pen G:	250,000 units/kg/day IV
2nd Generation:	-Dicloxacillin:	250mg q6 PO
	-Oxacillin:	1-2g q4 IV
	-Nafcillin:	1-2g q4 IV
3rd Generation:	-Amoxicillin:	500mg q8 PO
	-Ampicillin:	1g q4-6 IV
4th Generation:	-Augmentin:	875mg q12 PO
	-Unasyn:	3g q6 IV
	-Zosyn:	4.5g q6 IV
	-Timentin:	3.1g q6 IV
Cephalosporins		
1st Generation:	-Keflex:	500mg q8 PO or 750mg PO bid
	-Duricef :	2g q24 PO
	-Ancef:	1g q8 IV
2nd Generation:	-Ceftin:	500mg q12 PO
	-Zinacef:	1.5g q8 IV
	-Mefoxin:	1g q6 IV
3rd Generation:	-Omnicef:	300mg q12 PO
	-Vantin:	400mg q12 PO
	-Rocephin:	1g q24 IV
	-Fortaz:	1g q8 IV
	-Cefobid:	2g q12 IV
4th Generation:	-Maxipime:	2g q12 IV
Quinolones		
2nd Generation:	-Ciprofloxacin:	750mg q12 PO/400mg q12 IV
3rd Generation:	-Levofloxacin:	500mg q24 PO/IV
4th Generation:	-Tequin:	400 q12 PO/IV
	-Avelox:	400 q24 PO
Macrolides	-Biaxin:	500mg q12 PO
	-Ketek:	800mg q24 PO
	-Zithromax:	500 q12 IV/ 500mg PO Day 1; 250 mg PO Day2-5
	-Erythromycin	500mg q6 PO
Carbapenems	-Invanz:	1g q24 IV
	-Primaxin:	500mg q8 IV
	-Merrem	1g q8 IV
Aminoglycosides	-Amikacin:	1500mg/day
	-Tobramycin:	3-5mg/kg/day
	-Gentamycin:	3-5mg/kg/day
Tetracyclines	-Minocycline:	100mg q12 PO/IV
	-Doxycycline:	100mg q24 PO
	-Tetracycline:	500mg q6 PO
Misc	-Bactrim DS:	160/800mg q24 PO
	-Aztreonam:	1g q8 IV
	-Vancomycin:	1g q12 IV
	-Clindamycin:	600mg q8 IV; 300mg q6 PO
	-Zyvox:	600mg q12 PO/IV
	-Cubicin:	4mg/kg q12 IV
	-Synercid:	7.5mg/kg q8 IV
	-Flagyl:	500mg q8 PO
	-Rifampin:	300mg q12 PO/IV
	-Tygacil:	100mg loading dose; then 50mg q12 IV

AJM Sheets: Trauma

Trauma is another area that is often highlighted during the interview process. This section first details a trauma-specific work-up, and then goes through some specific traumatic conditions.

In terms of the interview, you generally will be expected to work-up, diagnose and classify based on radiographs, CTs and MRIs. While you should certainly have an understanding of treatment interventions and protocols, this will probably be less emphasized than diagnosis and classification.

A lot of these classifications are very visual (and I don't have room for that in 100 pages), so I've tried to include a lot of specific references with pictures of the classifications (mostly to McGlamry's and Gumann's texts).

I've also tried to include a lot of references to "classic" articles and review articles. Textbooks with good trauma information for additional reading include specific ones (Gumann's, Scurran's, Rang's, etc), but also general ones (McGlamry's, Myerson's, Hansen's, etc).

I said that while I was studying for the Diabetic Foot Infection work-up, I tried to learn as much as possible on the topic and really tried to "wow" the attendings at the interview. However, my strategy was different when dealing with trauma and the specific surgical work-ups. Here I tried to demonstrate "competence" as opposed to "mastery" of the material. With specific surgeries, you're really not supposed to have strong, pre-formed opinions as a student or as an intern. That's what your residency is for; developing surgical opinions. If you already know what to do in every surgical situation, then what's the point of doing a residency? So while on externships and at the interview, you should really try to walk a fine line between:

1. Displaying competence in knowledge of the baseline material
2. Displaying that you still have a lot to learn, and that you are eager to learn it

Contents:

AJM Sheet: Trauma Work-up

-The Trauma Work-up is very similar to the regular patient work-up, but with a few things added. You still need to go through the CC, HPI, PMH, PSH, Meds, Allergies, SH, FH, ROS and complete physical exam (starting with vital signs) in that order. In addition, there are three other topics that you need to address on every trauma patient for every work-up:

1. **ABCDE's of the Primary Survey**
   - -**Airway**: Three common forms of airway obstruction are cervical spine injury, swollen tongue and facial fracture.
   - -**Breathing**: Note how this is different than an established airway. Someone can have an airway, but still not be breathing.
   - -**Circulation**: Assess vascular status in all four extremities. Two large-bore (18-gauge) IV's should be started immediately if fluid replacement is considered necessary.
   - -**Deficits** (Neurological): There are two ways to assess this.
     - -**AVPU**
       - -**A**lert, responds to **V**erbal stimuli, responds to **P**ainful stimuli, or **U**nresponsive
     - -**Glasgow Coma Scale**
       - -Based upon three criteria: eye opening, verbal response, motor response.
       - -Based on scale of 3-15 with a higher score indicating a better prognosis.
         - -13+ associated with a good prognosis; 7- associated with a poor prognosis.
   - -**Exposure**: Complete exposure of the patient to evaluate further, unknown damage.

   -**Secondary Survey:** This is when you go through a normal history including HPI, PMH, etc. and a comprehensive physical exam.

2. **Tetanus Status**
   - -*Clostridium tetani* is a racquet-shaped gram-positive bacillus. It releases an exotoxin causing a pre-sympathetic blockade.
   - -Triad of tetanus symptoms: **Trismus**, **Risus Sardonicus**, and **Aphagia**.
   - -Characteristics of a tetanus-prone wound: greater than 6 hours old, clinical signs of infection, deep, devitalized tissue, contamination, traumatic mechanism of injury, etc.

   -**Basic Tetanus Algorithm**:

-Unknown tetanus status:	-Clean wound:	Give the toxoid; Hold the TIG
	-Tetanus-prone wound:	Give the toxoid; Give the TIG
-Incomplete tetanus status: (No booster within 5 years)	-Clean wound:	Give the toxoid; Hold the TIG
	-Tetanus-prone wound:	Give the toxoid; Give the TIG
-Complete tetanus status: (Booster within 5 years)	-Clean wound:	Hold the toxoid; Hold the TIG
	-Tetanus-prone wound:	Hold the toxoid; Hold the TIG

   -Dosages:
   - -Toxoid: 0.5ml
   - -TIG (tetanus immunoglobulin): 250-300 units

3. **NPO status**
   - -All trauma patients are potential surgical candidates, so get this information for the weenie anesthesiologists *(Always remember that lunch is for doctors, not for surgeons; while coffee breaks and crossword puzzles are for anesthesiologists)*.
     - -Traditional guidelines recommend:
       - -Nothing by mouth after midnight the night before elective surgery
       - -Nothing by mouth within 6-8 hours of any type of surgery

   -These strict guidelines are in the process of changing however, particularly with regard to allowing the ingestion of small amounts of clear liquids up to the time of surgery. If interested, please read:
   - *-[Brady M, Kinn S, Stuart P. Preoperative fasting for adults to prevent perioperative complications. Cochrane Database Syst Rev. 2003; (4): CD004423.]*
   - *-[Murphy GS, et al. The effect of a new NPO policy on operating room utilization. J Clin Anesth. 2000 Feb; 12(1): 48-51.]*

AJM Sheet: General Trauma Topics

-In addition to having a good trauma work-up, there are a few other things that are helpful to know regarding foot and ankle trauma.

1. Podiatric Surgical Emergencies

-Infection with emphysema (gas gangrene)
-Open fracture/dislocation
-Compartment syndrome
-Necrotizing Fasciitis
-General Neurovascular compromises

2. Mangled Extremity Severity Score (MESS)

-*[Helfet DL, et al. Limb salvage versus amputation. Preliminary results of the Mangled Extremity Severity Score. CORR 1990; 256: 80-6.]*
-*[Bosse MJ, et al. A prospective evaluation of the clinical utility of the lower-extremity injury-severity scores. JBJS-Am 2001; 83(1): 3-14.]*
-Based on 4 criteria: Skeletal/Soft Tissue Injury, Limb Ischemia, Age, and Shock
-Based on a scale from 1-11 with a higher score leading to an increased incidence of amputation.
-A score of **7+** has an increased likelihood of amputation.

3. Open Fractures

-Note that 30% of lower extremity open fractures are associated with polytrauma.
-Mainstays of treatment: Aggressive incision and drainage with copious lavage.
-It is generally recommended to never primarily close an open fracture until devitalized soft tissue has demarcated, but this certainly isn't always the case in practice. It is becoming more routine to primarily close open fractures following I&D with ORIF assuming the surgeon feels confident that the area is clean and has been appropriately debrided.

-**Gustilo-Anderson Classification of Open Fractures** *[Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. JBJS-Am. 1976; 58(4): 453-8.]*

I. Clean Wound **<1cm** in diameter
-Abx choice: 1st generation cephalosporin (Ancef)
II. Wound **1.0-5.0cm** in diameter with minimal soft tissue damage
-Abx choice: Ancef, Clindamycin
III. Wound **>5cm** in diameter with extensive soft tissue damage
-Abx choice: Ancef (or high dose PCN), Clindamycin and Aminoglycoside
-IIIA: Adequate soft tissue coverage
-IIIB: Extensive soft tissue damage with periosteal stripping and massive contamination
-IIIC: Arterial damage requiring primary repair

4. Fracture Blisters

-Location: Subepidermal
-Note that the fluid is sterile. Fracture blisters are histologically similar to 2nd degree burns.
-*Most common LE etiology?* Secondary to high-energy trauma such as ankle fx, calcaneus fx or Lisfranc injury.
-2 Common Types of Fracture Blisters
-Clear fluid: Most common (75%). Very tense in appearance.
-Hemorrhagic: Most severe. Roof is flaccid. Takes longer to re-epithelialize.
-Treatment is controversial, but the conservative approach is to never incise through a fracture blister and to delay surgery until re-epithelialization.
-*[Strauss EJ, et al. Blisters associated with lower-extremity fracture: results of a prospective treatment protocol. J Orthop Trauma. 2006 Oct; 20(9): 618-22.]*

5. Shock

-**Signs/Symptoms of Shock**: Tachycardia, Tachypnea, delayed capillary refill, decreased pulse pressure, change in mental status, decreased systolic pressure, decreased urinary output and decreased H&H.
-**Types of Shock**:
-**Hypovolemic**: most common; defined as the acute loss of circulating blood. Treatment is aggressive fluid replacement.
-**Cardiogenic**: induced by myocardial dysfunction.
-**Neurogenic**: secondary to decreased sympathetic tone from head and spinal cord injuries.
-**Septic**: shock secondary to infection.
-Goal of Treatment: **restore organ perfusion.**

AJM Sheet: General Trauma Topics

-Foreign Bodies/Puncture Wounds

-*When should a foreign body be removed?*
-Clinical signs of infection, known contaminated object, pain, object close to NV elements, intra-articular

-*Recommended imaging studies for a foreign body?*
-Plain film radiography (no oblique views!), fluoroscopy, CT, MRI, US

-*How will wooden objects appear on US?*
-Hyperechoic with a hypoechoic dark shadow

-*How large must a glass foreign body be to be visible on plain film radiography? Does leaden matter?*
-A piece of glass, regardless of whether it is leaden, must be >5mm to be visible.

-*Classification for foreign bodies?*

-Resnick Classification *[Resnick CD. Puncture wounds: therapeutic considerations and a new classification. J Foot Surg. 1990 Mar-Apr; 29(2): 147-53.]*
-I. Superficial/cutaneous: usually visible without signs of infection.
-II. Subcutaneous or articular without signs of infection.
-IIIA. Subcutaneous or articular with signs of infection.
-IIIB. Bone penetration without signs of infection.
-IV. Bone penetration with known osteomyelitis.

-Patzakis Classification *[Patzakis MJ. Wound site as a predictor of complications following deep nail punctures of the foot. West J Med. 1989 May; 150(5): 545-7.]*
-**Zone 1**: Toe to met head (50% incidence of osteomyelitis in this limited study.)
-**Zone 2**: Midfoot (17% incidence of osteomyelitis in this limited study.)
-**Zone 3**: Calcaneus (33% incidence of osteomyelitis in this limited study.)

-Puncture wound common bugs
-*Most common?* Staph Aureus
-*2nd most common?* Beta-hemolytic strept
-*Puncture through shoe gear?* Pseudomonas
-*Puncture involving soil or a farm?* Clostridia
-*Human bites?* Eikenella corrodens
-*Cat bites?* Pasteurella multocida
-*Dog bites?* Enterobacter, Pseudomonas, Staph, Bacillus

-*Mainstays of foreign body/puncture wound treatment?*
-Tetanus status, antibiotics, aggressive I&D with copious lavage

-Gun Shot Wounds

-**High velocity GSWs** are characterized by **speeds >2500 ft/s**. This is significant because high velocity GSWs have a tendency to yaw and tumble leading to increased cavitation.

-**Cavitation**: Large wound is created under a situation of negative pressure. This negative pressure "sucks" outside contaminants into the wound.

-*[Holmes GB. Gunshot wounds of the foot. CORR. 2003 Mar; (408): 86-91.]*

-Compartment Syndrome

-First described by Volkmann. Myerson has good articles/chapters on this topic.
-*[Perry MD, Manoli A. Foot compartment syndrome. Orthop Clin North Am. 2001 Jan; 32(1): 103-11.]*
-*[Myerson M, Manoli A. Compartment syndromes of the foot after calcaneal fractures. Clin Orthop Relat Res. 1993 May: 142-50.]*

-Results when **interstitial pressure exceeds capillary hydrostatic pressure**, so the microcirculation shuts down.

-The foot has anywhere from **3-11 compartments** depending on who you read:
-**Intermetatarsal Compartments X 4**: Contains the interossei muscles
-**Medial Compartment**: Abductor Hallucis
-**Lateral Compartment**: Abductor digiti minimi
-**Superficial Central Compartment**: FDB
-**Deep Central Compartment**: Adductor Hallucis
-**Calcaneal Compartment**: Quadratus Plantae and lateral plantar artery
-**Dorsal Compartment**: EHB and EDB

-**P's of Compartment Syndrome** (These are very generalized)
-Pain out of proportion and not controlled by analgesics
-Pain with passive dorsiflexion of the toes
-Paresthesia
-Pallor
-Paralysis
-Pulselessness
-Pressure

-**Diagnosis**
-*Normal compartment pressure?* 0-5mm Hg
-*When do you start getting worried?* 20-30mm Hg
-*When do you consider surgical intervention?* >30-40mm Hg
-*How is diagnosis made?* Wick or slit catheter to measure compartment pressures

-**Treatment**
-Decompression via fasciotomy, debridement of necrotic tissue, copious lavage and delayed closure
-Incision approaches: Consider dorsal vs. medial approaches

-**Complications**: permanent loss of function with structural deformity (Volkmann contractures), myoneural necrosis, sensory loss, chronic pain

AJM Sheet: Digital Fractures

-Even suspected digital fractures should be worked up according to a standard, full trauma work-up during the interview if the case is presented as a trauma. The following describes unique subjective findings, objective findings, diagnostic classifications and treatment.

Subjective
-History of trauma**. "Bedpost" fracture** describes stubbing your toe while walking at night. Also common are injuries from dropping objects on the foot.

Objective
-Edema, erythema, ecchymosis, open lesions, subungual hematoma, and onycholysis should all be expected.
-Any rotational/angulation deformities should be identified on plain film radiograph.

Diagnostic Classifications
-**Rosenthal Classification** *[Rosenthal EA. Treatment of fingertip and nail bed injuries. Orthop Clin North Am. 1983; 14: 675-697.]*
- **-Zone I**: Injury occurs with damaged tissue completely distal to the distal aspect of the phalanx.
- **-Zone II**: Injury occurs with damaged tissue completely distal to the lunula.
- -**Zone III:** Injury occurs with damaged tissue completely distal to the most distal joint (IPJ in hallux; DIPJ in lesser).

Treatment
-**Zone I Injuries**
- -If injury involves no exposed bone and a total tissue loss **less than 1cm squared**, then:
  - -Allow to heal in by secondary intention.
- -If injury involves a total tissue loss **greater than 1cm squared**, then:
  - -A STSG or FTSG should be used depending on weight-bearing position.

-**Zone II Injuries**
- -Flaps and Skin Grafts generally employed:
  - -**Atasoy flap**: plantar V→ Y advancement
    - -*[Atasoy E. Reconstruction of the amputated fingertip with a triangular volar flap. JBJS-Am 1970; 52: 921-926.]*
  - -**Kutler flap**: biaxial V → Y advancement
    - -*[Kutler W. A new method for fingertip amputation. JAMA 1947; 133: 29-30.]*

-**Zone III Injuries**
- -Usually requires distal amputation (Distal Symes amputation)

Miscellaneous Notes
-Hallux fracture is regarded as the **most common** forefoot fracture.
-Digital fractures without nail involvement and displacement/angulation/rotation can be treated conservatively with immobilization.

-If a subungual hematoma is present, then there is a **25% incidence** of underlying phalanx fracture.
-If a subungual hematoma covers **>25%** of the nail, then the nail should be removed.
-Only **1mm squared** of free space from onycholysis is necessary for hematoma development.

-For proper nail function and adherence, there should be no onycholysis **within 5mm** of the lunula.
-A **Beau's line** is a transverse groove often associated with nail trauma.

AJM Sheet: Sesamoid Trauma

-The following describes unique subjective findings, objective findings, diagnostic classifications and treatments.

Subjective
-**History of trauma** is very important in this case. You want to differentiate between acute and chronic conditions involving the sesamoids. Be careful to elicit any neurologic complaints that could be present.

Objective
-Expect edema, erythema, ecchymosis and open lesions. Take the time for proper palpation.
-**Joplin's neuroma** is irritation of the medial plantar proper digital nerve.
-Associated with rigidly plantarflexed first metatarsals, anterior cavus, etc.
-One of the most difficult things to **differentiate is an acute sesamoid fracture from a bipartite sesamoid**. There are several generic plain film radiographic characteristics found in acute fractures:
- -Jagged, irregular and uneven spacing
- -Large space between fragments
- -Abnormal anatomy
- -Bone callus formation
- -Comparison to a contra-lateral view

- -Also useful are:
  - -HISTORY of acute incident
  - -Bone scan (would show increased osteoblastic/osteoclastic activity with acute fracture).

Diagnostic Classifications
-Jahss Classification *[Jahss MH. Traumatic dislocations of the first metatarsophalangeal joint. Foot Ankle. 1980 Jul; 1(1): 15-21.]*
- **-Type I**
  - -Mechanism: Dorsal dislocation of the hallux
  - -Intersesamoid ligament: Intact
  - -Fracture?: No sesamoid fracture
  - -Treatment: Requires open reduction
- **-Type IIA**
  - -Mechanism: Dorsal dislocation of the hallux
  - -Intersesamoid ligament: Ruptured
  - -Fracture?: No sesamoid fracture
  - -Treatment: Closed reduction/Conservative Care
- **-Type IIB**
  - -Mechanism: Dorsal dislocation of the hallux
  - -Intersesamoid ligament: Ruptured
  - -Fracture?: Fracture of at least one sesamoid
  - -Treatment: Closed reduction/Conservative Care
- **-Type II Variant**
  - -Mechanism: Dorsal dislocation of the hallux
  - -Intersesamoid ligament: Ruptured
  - -Fracture?: Separation of a bipartite sesamoid
  - -Treatment: Closed reduction/Conservative Care

Treatments
-**Conservative**
- -Immobilization (NWB SLC, PWB SLC, Surgical Shoe, CAM Walker, etc.)
- -Dancer's Pad

-**Surgical**
- -Excision of the fractured fragment or entire sesamoid

Miscellaneous Notes
-**Ilfeld's Disease**: Agenesis of the fibular sesamoid
- -*[Ilfeld FW, Rosen V. Osteochondritis of the first metatarsal sesamoid. CORR 1972; 85: 38-41.]*

-**Incidence of Bipartite Sesamoid in Population**:
- -As much as Kewenter: **35.5%**
- -As few as Inge: **10.7%** with **75%** of cases being unilateral

AJM Sheet: Metatarsal Fractures
-The following describes unique subjective findings, objective findings, diagnostic classifications and treatments.

-Subjective and Objective
-Will point to some form of traumatic injury. Common injuries leading to metatarsal fracture include direct blunt trauma, shearing, ankle sprains, etc.
-Most important in your work-up will be how you read the plain film radiographs. Remember that at least two views are necessary to accurately describe displacement/angular/rotational abnormalities.

-Metatarsal Head/Impaction Fractures
-**MOI**: Direct or indirect trauma
-**Radiographic findings**: -Examine for evidence of displacement/angulation/rotation
-Expect a shortening mechanism
-Examine for intra-articular nature of fracture
-**Treatment**: -Conservative
-Closed reduction generally unsuccessful
-Surgical
-ORIF with fixation of K-wire, screws or absorbable pins
- Immobilization for 4-6 weeks and NWB
-Follow-Up
-Early PROM suggested
-Subsequent arthrosis is a common complication

-Metatarsal Neck Fractures
-**MOI**: Shearing forces or direct trauma
-**Radiographic findings**: -Expect elements of shortening, plantarflexion and lateral displacement of the distal segment.
-**Treatment**: -Conservative
-Closed reduction generally unsuccessful
-Surgical
-ORIF effective in restoring and maintaining alignment with K-wires, IM pinning and plates.
-Follow-up
-NWB in SLC for 4-6 weeks
-**General Information**:
-Metatarsal neck fractures often involve multiple metatarsals due to the mechanism of injury. Multiple fractures are very unstable due to loss of function of the deep transverse metatarsal ligament, which usually prevents displacement.
-**Vassal Principle**: Adjacent fractures generally improve alignment after reduction of the initial fracture because soft tissue structures are returned to their normal position through traction.

-**Midshaft Metatarsal Fractures**
-**MOI**: Result of direct, blunt or torsional injuries
-**Radiographic findings**: -Expect oblique fracture line, but transverse, spiral and comminuted are all possible.
-Expect elements of shortening, plantarflexion and lateral displacement of the distal segment.
-**Treatment**: -Based on displacement and fracture type:
-Non-displaced fractures: NWB SLC 4-6 weeks
-Fractures with >2-3mm of displacement and >10 degrees of angulation: ORIF

-Transverse displaced fractures
-Consider buttress plate, compression plate, IM percutaneous pinning, crossed K-wires
-Long oblique or spiral fractures
-Consider screws, plates, IM pinning, cerclage wiring
-Comminution
-Consider screws, plates, cerclage wiring, K-wires and external fixation

-Metatarsal Base Fractures
-**MOI**: Direct trauma (MVA, fall from height, etc.) Usually associated with Lisfranc's trauma.
-**Radiographic findings**: -Generally remain in good alignment/angulation because of surrounding stable structures.
-**Treatment**: -Conservative
-NWB SLC 4-6 weeks with good alignment
-Surgical
-ORIF with displacement/alignment/angulation

-First Metatarsal Fractures
-**MOI**: Direct trauma (MVA, fall from height, crush, etc.) and indirect trauma (torsional, twisting, avulsions, etc.)
-**Radiographic findings**: -Variable
-Examine for distal intra-articular fractures
-Examine for avulsion-type fractures
-**Treatment**: -Conservative
-SLC 4-6 weeks with non-displaced fractures
-Be wary of closed reduction because extrinsic muscles may displace after apposition.
-Surgical
-Various ORIF techniques detailed above
-Percutaneous pinning and cannulated screws are option in first metatarsal
-ORIF should be utilized if intra-articular fracture involves >20% of articular surface

AJM Sheet: 5th Metatarsal Base Fractures

-The following describes unique subjective findings, objective findings, diagnostic classifications and treatments.

Subjective and Objective

-All will point to some form of traumatic injury. Common injuries leading to metatarsal fracture include direct trauma, blunt trauma, shearing, ankle sprains, etc.
-Most important in your work-up will be how you read the plain film radiographs. Remember that at least two views are necessary to accurately describe displacement/angular/rotational abnormalities.

Diagnostic Classifications

Stewart Classification

[Stewart IM. Jones fracture: Fracture of the base of the fifth metatarsal bone. Clin Orthop. 1960; 16: 190-8.]

-**Type I**: Extra-articular fx at metaphyseal-diaphyseal junction (**True Jones Fracture**)
- -MOI: Internal rotation of the forefoot while the base of 5th met remains fixed
- -Radiographic findings: -Usually oblique or transverse fx at metaphyseal-diaphyseal junction
- -Treatment: -NWB SLC 4-6 weeks for non-displaced fractures
  -ORIF with displacement >5mm
- -Misc: -Fracture first described by **Sir Robert Jones in 1902** from injuring himself while dancing. *[Jones R. Fracture of the base of the fifth metatarsal bone by indirect violence. Ann Surg. 1902; 35(6): 776-82.]*
  -Very unstable fracture with high incidence of non-union/delayed union secondary to variable blood supply. Remember that the diaphysis and metaphysis are generally supplied by two different arterial sources.
  -*[Smith JW. The intraosseous blood supply of the fifth metatarsal: implications for proximal fracture healing. Foot Ankle. 1992 Mar-Apr; 13(3): 143-52.]*

-**Type II**: Intra-articular avulsion fracture
- -MOI: Shearing force caused by internal twisting with contracture of peroneus brevis tendon
- -Radiographic findings: -1 or 2 fracture lines
  -Intra-articular in nature
- -Treatment: -NWB SLC 4-6 weeks for non-displaced fractures
  -ORIF with displacement >5mm

-**Type III**: Extra-articular avulsion fracture
- -MOI: Reflex contracture of peroneus brevis with ankle in plantarflexed position
- -Radiographic findings: -Extra-articular; Involvement of styloid process
- -Treatment: -NWB SLC 4-6 weeks for non-displaced fractures
  -ORIF (pins, screws, tension-band wiring) for displacement >5mm
  -Consider excision of fragment and reattachment of peroneus brevis tendon

-**Type IV**: Intra-articular, Comminuted fracture
- -MOI: Crush injuries with base of 5th met stuck between cuboid and the external agent
- -Radiographic findings: -Multiple fragments; joint involvement
- -Treatment: -NWB SLC 4-6 weeks for non-displaced fractures
  -ORIF with displacement
  -Consider bone grafting and fragment excision with severe comminution
- -Misc: -High rate of non-union/delayed union

-**Type V**: Extra-articular avulsion fractures of the epiphysis
- -MOI and treatment similar to Type II and III fractures
- -Note that this can only occur in children (similar to a **Salter-Harris Type I fracture**)

Torg Classification

[Torg JS, et al. Fractures of the base of the fifth metatarsal distal to the tuberosity. JBJS-Am. 1984; 66(2): 209-14.]

-Radiographic classification of Jones fractures describing potential for non-union development.

-**Type I**: Acute injuries
- -Radiographic findings: Narrow fracture line without intra-medullary sclerosis

-**Type II**: Delayed Union
- -Radiographic findings: Widened fracture intersurface with evidence of IM sclerosis

-**Type III**: Non-Union
- -Radiographic findings: Complete sclerotic obliteration of the IM canal

AJM Sheet: Stress Fracture Work-up

Also called: March fx, Hairline fx, Fatigue fx, Insufficiency fx, Deutschlander's dz, Bone exhaustion, etc.

-Subjective

-**CC**: Patient presents complaining of a diffuse foot and ankle pain. Classic patient is a military recruit or athlete.

-**HPI**: -Nature: Pain described as "sharp with WB" or "sore/aching." May have element of "shooting" pain.
-Location: Described as diffuse, but can be localized with palpation. Common areas include dorsal metatarsal or distal tib/fib.
-Course: Subacute onset. Usually related to an increase in patient's physical activity.
-Aggravating factors: Activity
-Alleviating factors: PRICE

-**PMH**: -Look for things that would weaken bone (eg. Osteoporosis)

-**SH**: -Look for recent increases in physical activity or a generally active patient

-**PSH/Meds/All/FH/ROS**: Usually non-contributory

-Objective

Physical Exam

-**Derm**: -Generalized or localized edema
-Ecchymosis is rare

-**Vasc/Neuro**: Usually non-contributory

-**Ortho**: -Painful on localized palpation (**positive pinpoint tenderness**)
-Possible pain with tuning fork

-Imaging

-**Plain Film Radiograph**: -Localized loss of bone density and bone callus formation are hallmark signs
-Note that there must be a **30-50% loss of bone mineralization** before radiographic presentation of decreased bone density. This generally takes **10-21 days** in a stress fracture.

-**Bone Scan**: -Increased uptake in all phases regardless of time of presentation

-General Stress Fracture Information

-Somewhere between **80-95%** of all stress fractures occur in the LE with the most common sites being the **metatarsals** (20% with 2nd metatarsal most commonly involved [11%]) and the **distal tibia/fibula.**

-Stress fractures can occur via two mechanisms:
-Chronic strain upon a normal bone
-A chronic, normally benign strain upon a weakened bone

-Treatment

-Conservative treatment is mainstay:
-Immobilization and NWB for **4-6 weeks** (SLC, Unna boot, surgical shoe, etc.)
-Be certain of anatomic position with no angulation/rotation/displacement (very uncommon)

AJM Sheet: Lisfranc Trauma

-History

-Dr. Jacques Lisfranc was a French gynecologist who was called into the service of Napoleon's army where he served as a trauma surgeon in the 1820's and 30's. He also served under Dr. Dupuytren during this time.
-Del Sel first described Lisfranc dislocations following equine injuries (JBJS 1955).

-Anatomy

-Tarsometatarsal joint: 9 bones, ~13 joints, 7 weak dorsal ligaments, 5 strong plantar ligaments, the Lisfranc ligament (+2 other interosseous ligaments)
-Myerson described three functional columns of the Lisfranc joint. Ouzounian and Shereff described the sagittal plane motion of each of these columns.
- -Medial Column: 1st met and medial cuneiform: 4mm of motion in the sagittal plane.
- -Central Column: 2nd/3rd mets and central/lateral cuneiforms. 1mm of motion in sagittal plane.
- -Lateral Column: 4th/5th mets and cuboid. 10mm of motion in the sagittal plane.

-Mechanism of Injury

-Accounts for 0.2% of all traumatic injuries. Most common in MVA and sports injuries.
-Occurs either by direct crushing (i.e. dropping something on the foot) or indirectly (usually a plantarflexed and abducted foot).

-Diagnosis

-Clinical

- -Midfoot pain and tenderness. Possibly exacerbated with pronation, abduction or plantarflexion.
- -Plantar ecchymosis
- -Be wary of compartment syndrome! Always check neurovascular status.

-Imaging

- -Plain Film Radiography
  - -Pathognomonic "fleck sign" representing an avulsion fx in the 1st IM space.
  - -Look for deviations from normal in the AP, MO and Lat views. Normal is:
    - -AP: Medial border of the 2nd met continuous with the medial border of the central cuneiform. Lateral border of the medial cuneiform continuous with the medial border of the central cuneiform.
    - -MO: Medial border of the 4th met continuous with the medial border of the cuboid. Lateral border of the 3rd met continuous with the lateral border of the lateral cuneiform.
    - -Lat: No sagittal displacement. Look for lateral column shortening with a "nutcracker fracture" of the cuboid.
    - -"Lisfranc variant" is fracture damage extending proximally into the cuboid-navicular region.
  - -Consider stress radiographs with the foot in plantarflexion or abduction.
- **-CT scan required for full diagnostic work-up and peri-operative planning!**

-Classifications

-Classification originally described by Quenu and Kuss, then modified by Hardcastle, then modified by Myerson. The Myerson Classification is listed with the Quenu and Kuss equivalent in parentheses.
- -Type A: Total incongruity in any plane (QK: Homolateral)
- -Type B: Partial incongruity (QK: Isolateral)
  - -B1: 1st met goes medial
  - -B2: Lesser mets go lateral
- -Type C: Divergent (QK: Divergent)
  - -C1: Partial (only 1st and 2nd mets involved)
  - -C2: Total (all mets involved)

-Treatment

-Literature strongly favors ORIF with any displacement (>2mm between the 1st and 2nd mets). Exact anatomic reduction is the key to prognosis.

-Non-operative

- -If plain film and stress radiographs show no displacement, then NWB SLC for 6 weeks with films q2 weeks looking specifically for displacement.

-Operative

- -Goal: Reduction and stabilization of the medial and central columns. You must reduce the lateral column, but it is usually left unfixed because of the pronating mobile adapter mechanism. The medial and central columns do not have as much sagittal plane motion, but you still don't want excess compression with associated chondrolysis to develop.
- -Fixation:
  - -1st met to medial cuneiform, 2nd met to central cuneiform, and 3rd met to lateral cuneiform with crossed 0.062" K-wires (removed at 8 weeks), cannulated cancellous screws (removed at ~12 weeks) or 3.5mm corticals. Consider putting a notch 1.5cm distal to the joint for screw to prevent stress risers. Drill the hole for the screw in the superior aspect of the notch and not the base to prevent splitting the base.
  - -Consider 4th met to cuboid and 5th met to cuboid with a single 0.062" K-wire
  - -Lisfranc Screw: Medial cuneiform to 2nd met base, screw in a lag fashion
- -Length of the lateral column must be restored following a "nutcracker fracture." Consider using an H-plate or external fixation.
- -Incisions:
  - -Usually longitudinally over the dorsal-medial 1st, proximal 2nd interspace (for access to 2,3) and proximal 4th interspace.

-Post-Operative

-NWB SLC for 8 weeks transitioned to PWB SLC for 4 weeks transitioned to rehab. High impact activity can usually be resumed at 6 months.

-Complications

-ARTHROSIS! Essentially everyone develops post-traumatic arthritis to some extent.

Additional Reading:

- -[Myerson M. The diagnosis and treatment of injuries to the Lisfranc joint complex. Orthop Clin North Am. 1989; 20(4): 655-64.]
- -[Hardcastle PH, et al. Injuries to the tarsometatarsal joint. Incidence, classification, and treatment. JBJS-Br. 1982; 64(3): 349-56.]
- -[Desmond EA, Chou LB. Current concepts review: Lisfranc injuries. Foot Ankle Int. 2006; 27(8): 653-60.]

AJM Sheet: Navicular Trauma

-Suspected navicular trauma should be worked up with a primary and secondary survey. The following describes unique subjective findings, objective findings, diagnostic classifications and treatments.

Subjective
-History of trauma ranges from contusions to ankle sprains to forced abduction/plantarflexion of the forefoot.

Objective
-Manual muscle testing (MMT) of the posterior tibialis tendon is important in these cases.
-Multiple view plain film radiographs are extremely important because of the possible obliquity of some fractures. CT scans and MR images may also be necessary for complete visualization and analysis of stress fractures.

Relevant Anatomy
-The navicular is surrounded by a number of joints of varying stability. The TNJ proximally is very mobile, while the distal NCJ and lateral NCJ are very stable. The navicular is also very stable medially because of the insertion of the PT tendon.
-Vascular anatomy to the navicular can be extremely important as described by Sarrafian:
- -It has been demonstrated that the **central 1/3 of the navicular is relatively avascular**.
- -The dorsalis pedis artery adequately supplies the dorsal and medial aspects.
- -The medial plantar artery adequately supplies the plantar and lateral aspects.
- -The central 1/3 has variable, radially-projecting branches from anastomosis of these arteries.

Diagnostic Classification
-Watson-Jones Classification
- **-Type I: Navicular Tuberosity Fractures**
  - -Occur secondary to eversion and posterior tibialis contracture
  - -Watch for associated **"nutcracker fracture"** of cuboid and anterior calcaneal process fractures
  - -Must be differentiated from accessory navicular
  - -Treatment: -Displacement <5mm:
    - -Conservative immobilization
    - -Displacement >5mm consider:
      - -Excision of fragment with reattachment of tendon
      - -ORIF with a cancellous screw
- -**Type II: Dorsal Lip Avulsion Fractures**
  - -Occur secondary to plantarflexion/frontal plane mechanisms.
  - -Must differentiate from os supranaviculare and os supratalare accessory ossicles.
  - -Generally intra-articular
  - -Generally treated with conservative immobilization
- -**Type III: Navicular Body Fractures**. Described by Sangeorzan.
  - -*[Sangeorzan BJ, et al. Displaced intra-articular fractures of the tarsal navicular. JBJS-Am. 1989; 71(10): 1504-10.]*
  - **-IIIA: Coronal Plane Fracture with no angulation**
    - -~100% successful reduction usually achieved
  - **-IIIB: Dorso-lateral to Plantar-medial fracture with adduction of the forefoot**
    - -67% successful reduction usually achieved
  - -**IIIC: Comminution with abduction of the forefoot**
    - -50% successful reduction usually achieved

  - -**Principles of ORIF for Type III fractures:**
    - -Must achieve 60% reapproximation of proximal joint space
    - -Incision placed dorsal-medial, between the TA and TP
    - -Complications involve post-traumatic arthritis and/or AVN
  - -**Fixation Options using 3.5mm Cortical Screws:**
    - -Two screws directed lateral to medial
    - -Two crossed screws directed proximal to distal
    - -One screw directed proximal-medial to distal-lateral into the middle cuneiform
  - -Consider FDL transfer in the presence of a weakened PT tendon
- **-Type IV: Stress Fracture of the Navicular**
  - -Generally occurs secondary to running
  - -Torg describes typical stress fracture occurring in central 1/3 of body in the sagittal plane.
  - -DDx: Tibialis anterior tendonitis
  - -Usually plain films, CT and bone scans are necessary to diagnose

AJM Sheet: Talar Fractures

-Talar fractures are generally associated with high energy trauma, and a standard evaluation with primary and secondary surveys should precede any specific talar evaluation. The following describes unique subjective findings, objective findings, diagnostic classifications and treatment considerations.

Subjective

-History of trauma with a high incidence of MVC. The classic description of a talar neck fracture comes from a forced dorsiflexion of the foot on the ankle ("aviator's astragulus"). Talar fractures account for approximately 1% of all foot and ankle fractures.

Objective

-Important to verify neurovascular status, and rule out dislocations and compartment syndromes.

-**Imaging**: -**Canale View**: Plain film radiograph taken with the foot in a plantarflexed position. The foot is also pronated 15 degrees with the tube head orientated 75 degrees cephalad. This view allows for evaluation of angular deformities of the talar neck.

-**CT scan** is essential for complete evaluation and surgical planning.

Relevant Anatomy

-An intimate knowledge of the vascular supply to the talus is essential with regard to avascular necrosis (AVN):

[Aquino MD. Talar neck fractures: a review of vascular supply and classification. J Foot Surg. 1986; 25(3): 188-93.]

-**Dorsalis Pedis**: Supply the superior aspect of the head and neck (artery of the superior neck)

-Anastomoses with the peroneal and perforating peroneal arteries

-**Artery to the sinus tarsi**: supplies the lateral aspect of the talar body

-Forms an anastomotic sling with the artery of the tarsal canal

-**Posterior Tibial Artery**:

-**Deltoid branch**: medial aspect of the talar body

-**Artery of the canalis tarsi**: majority of the talar body

-Forms an anastomotic sling with the artery of the tarsal sinus

-Also sends branches to the posterior process

-**Peroneal/Perforating Peroneal Artery**: supplies posterior and lateral aspects of the talar body

-Anastomoses with the dorsalis pedis artery branches

Classifications/Named Fractures:

Hawkins Classification: Talar neck fractures
Type I: Non-displaced (~13% incidence of AVN)
Type II: Displaced fracture with STJ dislocation (~50% incidence)
Type III: Displaced fracture with STJ and ankle dislocation (~95%)
Type IV: Displaced fracture with STJ/ankle/TN dislocation (>95%)
-(Type IV added by Canale and Kelly)

Berndt and Harty: Talar dome fractures
Type 1: Chondral Depression
Type 2: Partial chondral fracture, seen on MRI
Type 3: Nondisplaced complete osteochondral fracture
Type 4: Displaced complete osteochondral fracture

Sneppen: Talar body fractures
Type 1: Osteochondral fracture
Type 2: Sagittal, Coronal, Transverse body fracture
Type 3: Posterior tubercle fracture
Type 4: Lateral process fracture
Type 5: Crush fracture

Modified Hawkins: Lateral process fractures
Type I: Simple bipartite fracture
Type II: Comminuted fracture
Type III: Chip fracture of anteroinferior lateral process

Others: -**Shepherd's fracture**: Acute fracture of posterolateral talar process
-**Cedell's fracture**: Acute fracture of the posteromedial talar process
-**Snowboarder's fracture**: lateral process fractures

Treatment

-NWB in SLC 6-8 weeks versus ORIF depending on nature of fracture and degree of displacement.
-**Titanium hardware** may be used so that MRI evaluation may be used in post-operative period to evaluate for AVN!
-**Hawkins sign**: radiolucency of the talar body noted at 6-8 weeks after fracture. This sign is indicative of **intact** vascularity. However, the absence of this sign does not indicate that osteonecrosis and talar collapse are eminent.

Additional Readings:

-Talar fractures are relatively uncommon in the medical literature. Most studies are case reports or small retrospective reviews leading only to Level IV or V evidence.

-[Ahmad J, Raikin SM. Current concepts review: talar fractures. Foot Ankle Int. 2006 Jun; 27(6): 475-82.]
-[Golano P, et al. The anatomy of the navicular and periarticular structures. Foot Ankle Clin. 2004 Mar; 9(1): 1-23.]
-[Berndt A, Harty M. Transchondral fractures of the talus. JBJS-Am. 1959; 41: 988-1020.]
-[Canale ST, Kelly FB. Fractures of the neck of the talus. Long-term evaluation of seventy-one cases. JBJS-Am. 1978 Mar; 60(2): 143-56.]

AJM Sheet: Calcaneal Fractures

-The standard trauma work-up again applies with primary and secondary surveys. The following describes unique subjective findings, objective findings, diagnostic classifications and treatment considerations.

Subjective

-**Demographics**: Men>Women; Age range generally 30-60; account for ~2% of all fractures; 2-10% are bilateral; 10% associated with vertebral fracture (most commonly L1); 1% associated with pelvic fracture and urethral trauma.
-**Common mechanisms of injury**: Direct axial load, vertical shear force, MVC, gastroc contraction, stress fracture, ballistics, iatrogenic surgical fracture

Objective

-**Physical Exam**: -Pain with palpation to heel
-Short, wide heel
-Inability to bear weight
-**Mondor's Sign**: characteristic ecchymosis extending into plantar medial foot
-**Hoffa's sign**: less taut Achilles tendon on involved side
-Must rule out compartment syndrome

-**Imaging**:
-**Plain film Imaging**:
-**Bohler's Angle**: normally 25-40 degrees (decreased with fracture)
-**Critical Angle of Gissane**: normally 125-140 degrees (increased with fracture)
-Both demonstrate ***loss of calcaneal height***
-**Broden's View**: Oblique views to view the middle and posterior facets
-**Isherwood Views**: 3 oblique views to highlight all facets
-**Calcaneal Axial View**: demonstrates ***lateral widening*** and ***varus orientation***

-**CT Scan**:
-Gold standard for evaluation and surgical planning
-The coronal view forms the basis of the Sanders Classification

Classifications

Sanders Classification: Uses widest view of posterior facet on semicoronal CT cut

-Type I: Non-displaced articular fx
-Type II: Two-part posterior facet fx
-Type III: Three-part posterior facet fx
-Type IV: Four-part/comminuted fx

-A, B and C further describe the fx (lateral→medial)
-A/B: divide posterior facet into equal 1/3's
-C: divides posterior facet from sus tali

-[Koval KJ, Sanders R. The radiographic evaluation of calcaneal fractures. CORR. 1993 May; 290: 41-6.]
-[Sanders R. Displaced intra-articular fractures of the calcaneus. JBJS-Am. 2000 Feb; 82(2): 225-50.]
-See page 1845 of McGlam's, or 224 of Gumann's for actual pictures.

Rowe Classification:

-*Type I*: -Type IA: Plantar tuberosity fractures (medial more common than lateral)
-Type IB: Sus tali fracture (remember FHL: pt will have pain with hallux PROM)
-Type IC: Anterior process fractures (remember your anatomy: bifurcate ligament)
-further divided into three parts by **Degan Classification**
-*Type II*: -Type IIA: Extra-articular "beak" fracture of posterior-superior calcaneal body
-Type IIB: Intra-articular "tongue-type" Achilles avulsion fracture
-*Type III*: Extra-articular calcaneal body fracture
-*Type IV*: Intra-articular joint depression fracture
-*Type V*: Intra-articular comminuted fracture

-[Rowe CR, et al. Fractures of os calcis: a long term follow-up study of one hundred forty-six patients. JAMA 1963; 184: 920-3.]
-[O'Connell F, Mital MA, Rowe CR. Evaluation of modern management of fractures of the os calcis. CORR 1972; 83: 214-23.]
-See page 1830 of McGlam's or page 223 of Gumann's for pictures.

Essex-Lopresti [*Essex-Lopresti P. The mechanism, reduction technique, and results in fractures of the os calcis. Br J Surg 1952; 39: 395-419.]*
-Differentiated between extra-articular (~25%) and intra-articular (~75%) fractures and further sub-divided intra-articular fractures into tongue-type and joint depression fractures (both with the same primary force, but different secondary exit points).

Zwipp *[Rammelt S, Zwipp H. Calcaneus fractures: facts, controversies and recent developments. Injury 2004; 35(5): 443-61.]*
-Assigns 2-12 points based on:
-Number of fragments
-Number of involved joints
-Open fracture or high soft tissue injury
-Highly comminuted nature, or associated talar, cuboid, navicular fractures

Treatment

-Appreciate the debate in the literature between cast immobilization vs. percutaneous reduction vs. ORIF vs. primary arthrodesis. *[Barei DP, et al. Fractures of the calcaneus. Orthop Clin North Am. 2002 Jan; 33(1): 263-85.]*
-Goals of therapy are to **restore calcaneal height**, **decrease calcaneal body widening** (reduce lateral wall blow-out), **take it out of varus**, and **articular reduction**.
-Review the lateral extensile surgical approach *[Benirschke SK, Sangeorzan BJ. Extensive intraarticular fractures of the foot. Surgical management of calcaneal fractures. CORR. 1993 Jul; 292: 128-134.]*
-**Complications**: Wound healing, arthritis, lateral ankle impingement, malunion, nonunion, etc.

-[Benirschke SK, Kramer PA. Wound healing complication in closed and open calc fractures. J Orthop Trauma. 2004; 18(1): 1-6.]
-[Cavadas PC, Landin L. Management of soft-tissue complications of the lateral approach for calcaneal fractures. Plast Reconstr Surg. 2007; 120(2): 459-466.]

AJM Sheet: Ankle Fracture Evaluation

-The standard trauma work-up again applies with primary and secondary surveys. The following describes unique subjective findings, objective findings, diagnostic classifications and treatment considerations.
-Residents and attendings love to ask questions about ankle fractures for whatever reason, so this is certainly a subject where you should know the classification systems cold, and do a lot of the additional readings. We'll keep it brief here.

-Relevant Anatomy to Review (not just for this topic; think lateral ankle instability, peroneal tendonopathy, sprains, etc.):

-**Ankle Ligaments**: -Lateral: ATFL, CFL, PTFL
-Medial: Superficial Deltoid: superficial talotibial, naviculotibial, tibiocalcaneal ligaments
Deep Deltoid: anterior talotibial and deep posterior ligaments

-**Syndesmotic Ligaments**: -AITFL, PITFL (and inferior transverse tibiofibular ligament), Interosseous ligament

-Classifications:

-**Ottawa Ankle Rules**

[Stiell IG, et al. A study to develop clinical decision rules for the use of radiology in acute ankle injuries. Ann Emerg Med. 1992; 21(4): 384-90.]

-Developed by ED docs to minimize unnecessary radiographs following ankle sprains. X-ray only required if:
-Bone tenderness along distal 6cm of posterior edge of fibula or tibia
-Bone tenderness at tip of fibula or tibia
-Bone tenderness at the base of the 5th met
-Bone tenderness on the navicular
-Inability to bear weight/walk 4 steps in the ED

-**Lauge-Hansen Classification**

-First submitted as a doctoral thesis *[Lauge-Hansen N, Anklebrud I. 1942].* Co-authored with a guy named "Ankle"-brud!
-[Lauge-Hansen N. Fractures of the ankle: analytic, historic survey as the basis of new experimental roentgenologic and clinical investigations. Arch Surg 1948; 56: 259.]

	Stage I	Stage II	Stage III	Stage IV
Supination Adduction	Lateral collateral ligament tear/ avulsion fibular fx (Weber A)	Near vertical medial malleolar fx (Mueller D)	NA	NA
Pronation Abduction	Transverse avulsion fx medial malleolus/deltoid rupture (Mueller B)	AITFL syndesmotic rupture or avulsion of its insertion	Short, oblique lateral malleolus fracture (Weber B) Transverse on lateral radiograph	NA
Supination External Rotation	AITFL syndesmotic rupture or avulsion of its insertion	Spiral lateral malleolus fracture (Weber B) Long, posterior spike on lateral radiograph	PITFL syndesmotic rupture or avulsion of its insertion	Transverse avulsion fx medial malleolus/deltoid rupture (Mueller B)
Pronation External Rotation	Transverse avulsion fx medial malleolus/deltoid rupture (Mueller B)	AITFL syndesmotic rupture or avulsion of its insertion	Oblique or spiral fibular fracture suprasyndesmotic (Weber C)	PITFL syndesmotic rupture or avulsion of its insertion
Pronation Dorsiflexion	Deltoid rupture	Dorsal Tibial Lip fracture	High fibular fracture	Posterior avulusion/fracture

-**The intrinsic problem with the Lauge-Hansen classification**: This was an experimental/laboratory study looking at the result of forced talar movement on a fixed tibia-fibula. But most ankle fractures in real-life occur when a moving tibia-fibula acts on a fixed foot.

-**Danis-Weber/AO Classification** for lateral malleolar fractures (From AO Group)

AO	**Type A: Infrasyndesmotic**	**Type B: Transyndesmotic**	**Type C: Suprasyndesmotic**
1	Isolated	Isolated	Simple diaphyseal fibular fx
2	With medial malleolar fx	With medial malleolar fx or deltoid rupture	Complex diaphyseal fibular fx
3	With posterior-medial fx	With medial lesion and posterior-lateral tibial fx	Proximal fibular fx

-**Mueller Classification** for medial malleolar fractures (From AO group)

A	B	C	D
Avulsion	Transverse at level of mortise	Oblique	Near vertical

AJM Sheet: Ankle Fracture Treatment

-Additional named fractures associated with the ankle:

-**Tillaux-Chaput fx**: AITFL avulsion from the anterolateral tibia
-**Wagstaffe fx**: AITFL avulsion from the anteromedial fibula
-**Volkmann fx**: PITFL avulsion from the posterior-lateral tibia
-**Bosworth fx**: PITFL avulsion from the posterior-medial fibula
-**Maisonneuve fx**: Weber C-type proximal fibular fracture that occurs within 10cm of the fibular neck
-**Pott's fx**: Generic term for a bimalleolar ankle fracture
-**Destot fx**: Generic term of a trimalleolar ankle fracture
-**Dupuytren fx**: At least a bimalleolar fracture when the talus gets lodged up between the tibia and fibula
-**Posterior Malleolar Fractures**: Different than an avulsion fracture of the PITFL; this is a true fx involving a portion of the tibial plafond cartilage. CT is usually done to estimate a percentage of the involved joint space. The rule of thumb (although certainly not proven) is that fractures involving >25-30% of the joint space require ORIF.

-Principles of Fixation**:**

-This is one area where there is a lot of controversy in the medical literature. There are certainly some things you want to accomplish besides the generic concept of "anatomic reduction". I can't get too much into it in this limited space, but I will try and give you a couple sides of the argument and some reading to do. The question you are really trying to answer is: "How reduced is reduced enough?" Then we'll briefly cover some specific aspects of the surgeries themselves. One thing to appreciate is that most of these arguments are made about SER fractures (because they are the most common):

-**Restore fibular length**

-Most people agree that the fibular fracture is the dominant fracture. In other words, if you adequately reduce the fibula, then the other fractures and dislocations more or less fall into line because of the soft tissues (poor man's definition of the ***Vassal Principle***). It doesn't mean that the other fractures don't require fixation, but it means there's no real sense in fixating the other fractures unless you have the dominate fracture fixated (or at least reduced).

-The other concept is that a fixed fibula is essentially acting as a buttress, keeping the talus within the ankle mortise.

-The fibula is generally shortened in ankle fractures, so you want to get the full length back with your reduction (generally visibly seen by reduction of the posterior spike on a lateral view).

-[Yablon IG, et al. The key role of the lateral malleolus in displaced fractures of the ankle. JBJS-Am. 1977; 59(2): 169-173.]

-**Restore the alignment of the ankle mortise** (medial clear space and the syndesmotic gap)

-This goes back to the fibula keeping the talus in the ankle mortise. The classic article you need to know is *Ramsey and Hamilton* who showed a 43% decrease in the tibiotalar contact area when the talus was displaced 1mm laterally (It is not 42%.....check their math!). From this, people inferred that if the talus isn't perfectly reduced back into the mortise, then gross instability occurs.

-This is assessed by:

-**Medial clear space** (from the talar shoulder): Should be ~4mm or less after reduction
-**Tib-Fib Overlap**: Approximately >10mm on AP view at 1cm superior to the joint line
-**Talar Tilt**: <10 degrees absolute, or <5 degrees compared to other side

-[Ramsey PL, Hamilton W. Changes in tibiotalar area of contact caused by lateral talar shift. JBJS-Am. 1976; 58(3): 356-7.]
-[Park SS, et al. Stress radiographs after ankle fracture: the effect of ankle position and deltoid status on medial clear space measurements. J Orthop Trauma. 2006; 20(1): 11-18.]

-**Fix the syndesmosis?**

-Another area of controversy where there is no clear answer is when and how to fixate the syndesmosis with internal fixation. One point is clear: the purpose of placing internal fixation across the syndesmosis is to stabilize the fibula against the tibia to prevent lateral migration of the talus and instability. If the fibula is stable against the tibia with all of your other fixation, then you don't really need any additional fixation. *How can you tell?* Radiographic findings and the Cotton hook test for instability intra-operatively.

-Other questions where people have opinions, but no clear answers are: What type of screws? How many screws? How many cortices? How far above the ankle? Temporary vs. permanent fixation? Weight-bearing? etc.

-Lateral Malleolus:

-Fracture is primarily reduced and fixated with a single 2.7 or 3.5mm cortical screw with interfrag compression.
-Then a generic 1/3 tubular plate or a specialized contoured plate is used for buttress stabilization.
-Attempt for 6 cortices proximal to fracture with 3.5 bicortical screws
-Get as many distal screws as you can. 3.5 bicortical if above the ankle joint. 4.0 unicortical if not.
-Proximal fibular fractures still amendable to 1/3 tubular plating, but may need to double-stack the plates.
-Should appreciate the concept of lateral vs. posterior anti-glide plating.

-Medial Malleolus:

-Several options including 4.0mm cancellous, K-wires, plating, cerclage, etc.

-Additional Reading:

-[Mandi DM, et al. Ankle fractures. Clin Podiatr Med Surg. 2006 Apr; 23(2): 375-422.]
-[Mandracchia DM, et al. Malleolar fractures of the ankle. A comprehensive review. Clin Podiatr Med Surg. 1999 Oct; 16(4): 679-723.]
-[Kay RM, Matthys GA. Pediatric ankle fractures: evaluation and treatment. J Am Acad Orthop Surg. 2001; 9(4): 269-78.]
-[Jones KB, et al. Ankle fractures in patients with diabetes mellitus. JBJS-Br. 2005; 87(4): 489-95.]
-[Espinosa N, et al. Acute and chronic syndesmosis injuries: pathomechanics, diagnosis and management. Foot Ankle Clin. 2006 Sep; 11(3): 639-57.]

AJM Sheet: General Tendon Trauma

-Mechanism of Injury

-Tendon is actually the strongest part of the muscle-tendon-insertion system. It is much more likely for the complex to fail at the myotendinous junction or at the tendinous insertion, but acute tendon injuries do occur. They are usually the result of direct trauma, or overload on an intrinsically weakened tendon.

-Tension overload on a passive muscle
-Eccentric overload on an actively contracting muscle
-Laceration
-Blunt Trauma

-Factors which can intrinsically weaken tendons

-**Increased age**: -increased cross-linking of collagen fibrils decreases tendon elasticity
-decreased reaction time and muscular contraction speed
-decreased vascularity

-**Sex**: -M>F

-**Systemic inflammatory process**: -RA, SLE, Gout, etc.

-**Underlying endocrine dysfunction**: Xanthoma (hyperbetalipoproteinemia), DM, Hyperparathyroidism secondary to renal failure, hyperthyroidism, infection, intratendinous calcifications, etc.

-**Medications**: -Fluoroquinolones, Corticosteroids

-Tendon Healing

-As with most tissue, there is a generalized inflammation, reparative and remodeling phase.
-Week 1: Severed ends fill in with granulation tissue
-Weeks 2-3: Increased paratenon vascularity; collagen fibril alignment
-Week 4: Return to full activity without immobilization

-Imaging in Diagnosis of Acute Tendon Injury

-**Plain Film Radiograph**: -May see avulsions, soft tissue swelling, accessory bones/calcifications

-**Tenograph**: -Radiopaque dye injected into tendon sheath and viewed on plain film radiograph
-Technically difficult with many false positives and negatives

-**Ultrasound**: -Tendon normally appears hyperechoic to muscle on US.
-Look for discontinuity of fibers, possible alternating hyperechoic/hypoechoic bands, and an area of intensely hyperechoic hematoma.
-It is very important that the US head is held perpendicular to the long axis of the tendon.

-**CT**: -Tendon normally appears as a homogenous, well-circumscribed oval surrounded by fat on CT. It normally has a higher attenuation than muscle.
-Will be able to appreciate discontinuity on CT with injury.

-**MRI**: -**T1**: Tendons normally have a uniform low-intensity (very black). Will be uniform with variable high-intensity signal with injury.
-**T2**: Tendons are normally relatively low-intensity. Will light up with high-intensity signal with injury.
-Remember the **magic angle phenomenon**. Any MRI signal shot at 55 degrees to the course of the tendon will show a false-positive damage signal. Very common in the peroneals.
-*[Mengiardi B, et al. Magic angle effect in MR imaging of ankle tendons: influence of foot positioning on prevalence and site in asymptomatic patients and cadaver tendons. Eur Radiol. 2006 Oct; 16(10): 2197-2206.]*

-Principles of Repair

-It is possible, but rare to get acute tendon injury to any of the long tendons of the leg. An Achilles tendon work-up will be featured in another AJM sheet, but realize there are some basic principles that apply to any tendon.
-One is generally able to primarily repair the tendon. Non-absorbable suture is preferred.
-Special attention should be paid to vascular supply. Remember that the majority of a tendon's vascularity comes from the **mesotenon**, and therefore should be preserved as much as possible.
-If primary repair is not possible, consider using lengthening tendon slides, tendon grafts, tendon transfers and biomaterials such as Graft-Jacket (allograft dermal tissue matrix) or Pegasus (equine pericardium) to restore the integrity of the tendon.
-The goal of treatment should be to allow early PROM without gapping of the tendon.

AJM Sheet: Achilles Tendon Rupture Work-Up

Subjective:

CC: Typical complaint is pain, weakness and swelling in the back of the leg following an acute injury. The typical patient is the "weekend warrior" type. This is a 30-50 y/o male participating in a strenuous athletic activity after a generally inactive lifestyle.

HPI: Nature: Pain, weakness and swelling. Pain is surprisingly non-intense allowing the patient to ambulate. The patient may relate an audible "pop" or "snap". They may also relate feeling like they were "kicked or shot" in the back of the leg.

Location: Distal posterior leg. The left leg is more affected. Some people theorize that this has to do with the majority of people having right-handedness and a greater strength and proprioception of the RLE.

Duration, Onset, Course: Acute onset with gradually progressive increase in swelling and edema.

Mechanism of Action: -Three classic MOA are described:
- -Unexpected dorsiflexion with triceps contraction
- -Pushing off during WB with the leg extended (tennis lunge)
- -Violent dorsiflexion on a plantarflexed ankle
- -Also consider lacerations and blunt trauma

Previous History: obviously more likely to re-rupture

PMH: -Inflammatory conditions: RA, SLE, Gout
-Endocrine dysfunction: DM, Renal failure with hyperparathyroidism, hyperthyroidism, Xanthoma (hyperbetalipoproteinemia)
-Infection: Syphilis

Meds: -Corticosteroid injection
-Fluoroquinolone use

SH: -Smoking
-Sedentary lifestyle with weekend activity

Objective:

Derm: -Posterior, Medial and Lateral Ecchymosis
-Open lesion associated with laceration

Vasc: -Posterior, Medial and Lateral edema

Neuro: -Sural Neuritis

Ortho: -Palpable gap ("**hatchet strike defect**")
-Positive **Thompson test**
-Negative **Jack's test**
-Pain in the area
-Increased PROM ankle dorsiflexion
-Decreased AROM ankle plantarflexion
-Retraction of proximal gastroc belly
-Apropulsive gait

Other specific tests:
- -**Mattles** test: Foot should be in plantarflexed position with patient prone and knee at 90°
- -**Simmonds'** test: Foot should be in plantarflexed position with patient prone
- -Various needle tests (**O'Brian, Cetti**)
- -**Toygar's skin angle**: Normally 110-125 degrees. Increases to 130-150 degrees with rupture.

Imaging:

-**Plain film**:
- -r/o Rowe Type IIB avulsion fracture
- -Radiodense gap
- -Obliteration of Kager's triangle
- -Soft tissue edema

-**US**:
- -Alternating hyperechoic and hypoechoic bands
- -Hyperechoic hematoma

-**MRI**:
- -TI: -Ill-defined low-intensity with mixed high-intensity signal
- -T2: -High-intensity signal from hematoma

AJM Sheet: Achilles Tendon Rupture Treatment

-Anatomy Review

-Muscles of the Triceps Surae (origins, insertions, NV supply, action)
-Plantaris (origins, insertions, NV supply, action)
-Segmental Blood Supply of Tendon
-"Twisting" of tendon

-Specific Information regarding the Watershed Area

-Lagergren and Lindholm
  -Used microangiographic technique on human cadavers
  -Found decreased vascularity 2-6cm proximal to insertion
  -Theorized this was secondary to atrophy from inactivity
-Conflicting information from laser Doppler flowmetry studies
  -Found uniform vascularity throughout tendon
  -Found decreased vascularity with age and in men
  -Found decreased vascularity with physical loading/stress of tendon, specifically at insertion
-Leadbetter
  -Found increased stress/strain at the watershed area regardless of vascularity

-Kuwada Classification of Achilles Tendon Ruptures

[Kuwada GT. Classification of tendo Achilles repair with consideration of surgical repair techniques. J Foot Surg. 1990; 29(4): 361-5.]

-**Type I**: Partial tear involving <50% of tendon. Note that in a partial Achilles tear, the posterior fibers are torn first. So the direction of the tear/rupture is from posterior to anterior.
-**Type II**: Complete tear with <3cm deficit
-**Type III**: Complete tear with a 3-6cm deficit
-**Type IV**: Complete tear with a >6cm deficit

-Puddu Classification of Chronic Achilles Pathology *[Puddu G, et al. A classification of Achilles tendon disease. Am J Sports Med, 1976]*

-**Peritendonitis**: Inflammation of the surrounding tissues, not the tendon itself. This pain will remain stationary as the tendon is taken through a range of motion.
-**Tendonosis**: Intra-tendinous degeneration. This pain will move proximally and distally as the tendon is taken through a range of motion.
-**Peritendonitis with tendonosis**: combination of the two pathologies.

-The podiatric surgeon is faced with three options: do nothing, cast immobilization and surgical repair. There's a lot of information out about this in the medical literature now, particularly with open repair vs. immobilization and when to start weight-bearing/PT.

-Do nothing

-Gap will eventually fill in with fibrotic scar tissue
-Usually requires later surgical intervention

-Cast Immobilization

-AK cast versus SLC
  -Some are proponents of AK casting
  -Knee should be in a 20 degree flexed position
-General recommendations:
  -Gravity equinus cast x 4 weeks
  -Reduction of 5 degrees every 2 weeks to a neutral ankle position (~4-6 weeks)
  -Heel lift and PT until normal ankle PROM
  -Return to full activity at approximately 6 months

-Surgical Repair

-**Surgical approach**
  -Midline to medial incision to avoid superficial neurovascular structures
  -Pt in a prone or supine frog-legged position
  -Use full-thickness flaps with emphasis on atraumatic technique
-**Primary Open Repair**
  -Keith needles with non-absorbable suture (or fiberwire) with absorbable sutures to reinforce
  -There are three common stitches used:
    -**Bunnell**: Figure of 8 or weave stitch
    -**Krakow**: Interlocking stitch
    -**Kessler**: Box stitch
-**Augmented Open Primary Repair**
  -**Lynn**: Plantaris is fanned out to reinforce
  -**Silverskoild**: 1 strip of gastroc aponeurosis brought down and twisted 180 degrees
  -**Lindholm**: Utilizes multiple strips of gastroc aponeurosis
  -**Bug and Boyd**: Strips of fascia lata are used to reinforce
  -**V→ Y lengthening** of the proximal segment with primary repair
  -Reinforcement with FHL
  -Graft Jacket, Pegasus, etc.
-**Percutaneous Primary Repair**
  -**Ma and Griffith** described a percutaneous Bunnell-type approach
  -May be associated with high re-rupture rates

-Post-Op Treatment

-SLC in gravity equinus with gradual reduction over 6-10 weeks

AJM Sheets: Peri-Operative Medicine and Surgery

Contents:

This particular section is intended to be more general, as opposed to a specific surgical study guide. It is ridiculous to think that you could learn foot and ankle surgery in 100 pages, especially with only 15 pages dedicated to specific deformities. In other words, you should absolutely not be doing all of your specific surgical preparation for externships and interviews from the PRISM. Many of the Sheets from the *Specific Surgery Topics* section are simply summarizations of the 3rd edition of McGlamry's chapters for example. This may be an area where you feel the PRISM could be updated in the future.

Again, I said that while I was studying for the Diabetic Foot Infection work-up, I tried to learn as much as possible on the topic and really tried to "wow" the attendings at the interview. However, my strategy was different when dealing with trauma and the specific surgical work-ups. Here I tried to demonstrate "competence," as opposed to "mastery" of the material. With specific surgeries, you're really not supposed to have strong, pre-formed opinions as a student or as an intern. That's what your residency is for, developing surgical opinions. If you already know what to do in every surgical situation, then what's the point of doing a residency? So while on externships and at the interview, you should really try to walk a fine line between:

1. Displaying competence in knowledge of the baseline material
2. Displaying that you still have a lot to learn, and that you are eager to learn it.

Page 85's "How to Work-Up a Surgical Patient" gets into this concept a little deeper.

AJM Sheet: Admission Orders/ADC VANDILMAX

(Note: If I wanted to be mean during an interview, I would have you write out a set of admission or post-op orders as I was asking you other questions.)

Admission: *Pt is admitted to the general medical floor on the Podiatric Surgery Service under Dr. Attending.*

Most patients on the podiatric surgery service are admitted to the general medical floor or a surgical floor. Any pts admitted to a critical care unit or telemetry unit will probably be on a medicine service with a podiatric surgical consult.

Diagnosis*: Infection of bone of right 2nd toe*

Always use terminology that everyone in the hospital can understand, but also be as specific as possible.

Condition: Consider: -Stable -Fair -Guarded -Critical

Podiatric surgery pts will generally always be in stable or fair condition.

Vitals: *Vitals recorded q8 hours per nursing.*

Always designate how often you want them recorded. Also common is "q-shift."
Consider neurovascular checks to the affected limb if indicated.

Ambulatory Status: Consider:

-CBR (Complete bed rest)	-As tolerated
-NWB	-OOB to chair
-PWB	

-Always designate which leg the order is for. Be specific with PWB status ("toe-touch" or "heel-touch"). If order is for CBR, consider DVT ppx and a bed pan order. If the order is OOB, specify # of times and length per day.
-Also consider Physical Therapy and/or Occupational Therapy orders here.

Nursing Instructions: Consider:

-Accuchecks (how often and when?)	-Ice and elevation
-Bedside Commode	-Dispense Post-op shoe/Crutches
-Wound Care	-Dressing Instructions
-Drain management	-Off-loading instructions
-Spirometry	

Diet: Consider:

-Regular diet	-ADA 1800-2200 calorie	-Mechanically soft
-Renal diet	-Cardiac diet	-NPO
-Decreased Na	-Decreased K+	

Ins/Outs/**I**Vs: Consider:

-Measurement and recording of Ins and Outs (especially dialysis pts)
-Foley
-IV Fluids

Labs: Consider:

-CBC with diff	-Chem-7/Met Panel	-Coags
-Type and Screen	-Wound cultures	-Blood cultures
-D-Dimer	-HbA1c	-CRP
-ESR		

Always detail when the labs should be done. For example, initial CBC and Chem-7 should be taken "upon arrival to the floor." Additionally, 2 sets of blood cultures should be taken from 2 different sites.

Medications: Consider:

-Write out all at-home medications in full	-Pain medication
-Antibiotics	-Insomnia
-Anti-emetics	-DVT ppx
-Constipation	-Diarrhea
-Sliding Scale Insulin (SSI)	-Fever
-Throat lozenges	-Anti-pruritic

Be as specific as possible. SSI needs to be written out in full. Many medications require hold parameters. For example, fever medications should not be given unless the temperature reaches 101.5° F. Anti-HTN agents should be held if the blood pressure or heart rate drops too low.

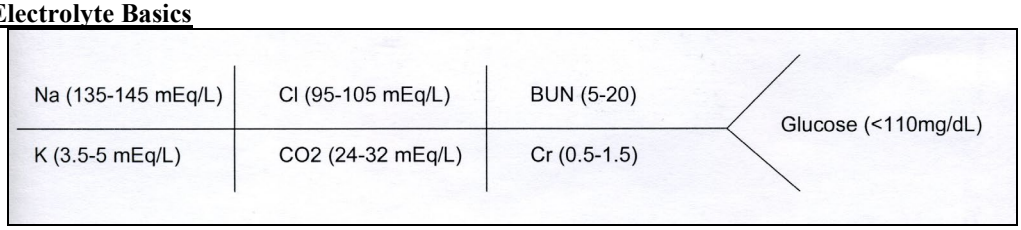
Ancillary Consults: Consider:

-General Medicine	-Infectious Disease
-Vascular Surgery	-Cardiology
-PT/OT	-PM&R
-Pulmonary	-GI
-Renal	-Social Work
-Home Care	-Case Manager

X-rays/Imaging: Consider:

-Plain film radiographs	-CT scans	-US Doppler
-MRI	-CXR	-Bone Scans
-Vascular Studies	-EKG	

AJM Sheet: Electrolyte Basics



-**Sodium:**

-**Hyponatremia**

-Manifestations: Primarily neurologic, lethargy, headache, confusion, obtundation

-Treatment: -Restrict water intake and promote water loss -Correct underlying disorder
-Replace Na+ deficits

-**Hypernatremia**

-Manifestations: Change in mental status, weakness, neuromuscular irritability, focal neurologic deficits, coma, seizures

-Treatment: -Replace water loss and promote sodium excretion
-Water deficit = ([Na+]-140)/140 x Total Body Water in liters

-Rapid correction of either of these disorders is dangerous due to rapid shifts of water in and out of brain cells. It should therefore be corrected slowly over 48-72 hours. Aim correction at 0.5 mEq/L/hr with no more than a 12 mEq/L correction over the first 24 hours.

-**Potassium:**

-An abnormal potassium level is a major reason a surgery will be cancelled and/or delayed. You should have a specific understanding how to raise and lower potassium levels in the peri-operative setting.

-**Hypokalemia**

-Manifestations: Fatigue, myalgia, muscular weakness, cramps, arrhythmia's, hypoventilation, paralysis, tetany

-Treatment: -Minimize outgoing losses -Treat underlying cause
-Correct K+ deficit via oral or IV means (K+ riders added to fluid, oral KCL, etc.)

-**Hyperkalemia**

-Manifestations: Cardiac toxicity (peaked T waves, prolonged PR, torsades de pointes), muscle weakness, paralysis, hypoventilation

-Treatment: -Increase cellular uptake of K+
-Insulin (10-20 units) with 50 g IV glucose
-IV NaHCO3 (3 ampules in 1L of 5% dextrose)
-Albuterol (5-10mg nebulized over 30-60 minutes)
-Increase K+ excretion
-Loop diuretic, Thiazide diuretic
-Kayexalate (cation exchange resin) (25-50mg mixed with 100ml 20% sorbitol to prevent constipation)
-Dialysis
-Calcium Gluconate (10ml of 10% solution over 2-3 minutes emergently to reduce membrane excitability)

-**Chloride** and **Carbon Dioxide**:

-Not going to talk much about this, but you should have a basic understanding of Acid-Base Regulation.

-Equation for determining Anion Gap:

Anion gap (all units mmol/L) = (Na + K) - (Cl + [HCO3-])

-Normal gap (~8-20mmol/L)
-Negative/lowered gap (<8mmol/L): Alkalotic state
-Positive/elevated gap (>20mmol/L): Acidotic state
-MUDPILES algorithm: **m**ethanol/metformin, **u**remia, **d**iabetic ketoacidosis, **p**ropylene glycol, **i**nfection, **l**actate, **e**thanol, **s**alicylate/starvation

-**BUN** and **Creatinine**:

-Measures of kidney function and hydration status

-**BUN** (Blood Urea Nitrogen): Protein metabolism waste product eliminated by the kidneys. This can be increased if your kidneys aren't eliminating it properly, or in a dehydrated state where it's a relatively high concentration.

-**Creatinine**: A more direct measure of kidney function from elimination of this skeletal muscle waste product.

-Creatinine clearance and estimated glomerular filtration rate (GFR) with the **Cockcroft-Gault Equation**:
[(140-Age in years) x Weight in kg] / [72 x Serum creatinine] x 0.85 if female
-GFR < 60 ml/min indicates chronic kidney disease; < 15 indicates kidney failure
-Antibiotics and other drugs should be dosed appropriately in these situations

-Renal protective agents are utilized prior to procedures that are known to affect the kidneys in patients with kidney disease. A common example of this is an angiogram with dye to evaluate the vascular status of a patient with diabetic foot disease.
-Pre-procedural hydration -Mucomyst (N-acetylcysteine) (also used for acetaminophen OD)
-Sodium Bicarb Protocols
-[*Lawlor DK. Prevention of contrast-induced nephropathy in vascular pts. Ann Vasc Surg. 2007 Sep: 593-7.]*

-**Glucose** covered in another AJM sheet

AJM Sheet: Blood Glucose and Glycemic Control

-The importance of in-patient management of blood glucose cannot be overstated. This is an area however where medicine tends to be very passive with regard to intervention. Rigid control of blood glucose in the in-patient setting has been definitively shown to:

-Reduce mortality
-Reduce in-patient complications
-Reduce infection rates
-Decrease length of stay
-Reduce hospital costs

-Specifically with regards to diabetic foot disease, a single blood glucose level higher than 150-175mg/dl significantly limits the function of the immune system for a period of days, particularly cytokine activation and recruitment.

-My favorite article of the 2006-7 academic year was *Inzucchi SE. Management of Hyperglycemia in the Hospital Setting. NEJM. Sep 2006; 355: 1903*. It is a must-read on this topic. I also strongly recommend trying to get a copy of the *Yale Diabetes Center Diabetes Facts and Guidelines 2006*. And if you are really interested in this topic, research the work of the Portland Diabetic Project.

Oral Agents

-**Sulfonylureas**: Bind to β-cell receptors stimulating insulin release
-Glyburide (Micronase) -Glipizide (Glucotrol) -Glimepiride (Amaryl)

-**Biguanides**: Decrease production of glucose in the liver
-Metformin (Glucophage)

-**Thiazolidinediones**: Increase peripheral cellular response to insulin
-Rosiglitazone (Avandia) -Pioglitazone (Actos)

-**α-glucosidase inhibitors**: Reduce intestinal carbohydrate absorption
-Acarbose (Precose) -Miglitol (Glyset)

Insulins

Type	Onset	Peak	Duration
Rapid Acting			
Lispro (Humalog)	10-15 minutes	1-2 hours	3-5 hours
Aspart (Novolog)	10-15 minutes	1-2 hours	3-5 hours
Short Acting			
Regular	0.5-1hr	2-4 hours	4-8 hours
Intermediate Acting			
NPH	1-3 hours	4-10 hours	10-18 hours
Lente	2-4 hours	4-12 hours	12-20 hours
Long Acting			
Glargine (Lantus)	2-3 hours	None	24+ hours
Detemir (Levemir)	1 hour	None	24 hours
Combinations			
70/30 (70% NPH/30% Regular)	0.5-1 hour	2-10 hours	10-18 hours

In-patient Recommendations

-There is increasing data that sliding scales are completely inefficient at in-patient glucose management. Sliding scales are passive, reactionary scales that compensate **after** a hyperglycemic incident occurs. Inzucchi recommends the following, instead of a sliding scale:

-Basal Rate: Lantus or other long acting
-Start 0.2-0.3 Units/kg/day; then increase 10-20% q1-2 days prn
-Prandial Coverage: Novolog or other rapid acting
-Start 0.05-0.1 Units/kg/day; then adjust 1-2 Units/dose q1-2 days prn

Diabetic NPO Recommendations

-**Type 2 DM**:
-1/2 the normal dose of long acting if they get any
-BG checks q6 hours with short acting agent available for coverage
-D5W or D5-1/2NS at 50-75cc/hr while NPO

-**Type 1 DM**:
- Strongly consider an insulin drip
-1/2 – 2/3 normal dose of long acting agent
- BG checks q6 hours with short acting agent available for coverage
- D5W or D5-1/2NS at 75-100cc/hr while NPO

AJM Sheet: Fluids

-Fluid management is a difficult topic to cover because it can be used for a variety of different problems/purposes. It can be used to maintain fluid balance in a patient who is NPO, correct electrolyte disturbances, and/or provide glucose to name just a few examples. This sheet will cover the basics of short-term maintenance therapy and show differences in electrolyte concentrations between the most common fluids.

-Maintenance therapy for the NPO patient

-An NPO patient is still losing water that needs to be replaced to ensure homeostasis. Sources of water loss include:
- -Urine output: At least 500ml/day
- -Insensible water losses (Skin and Respiration): At least 500ml/day
  - -This can increase by 150ml/day for each degree of body temperature of 37°C.
- -Gastrointestinal losses: Extremely variable
- -Direct blood volume loss from the surgery itself

-Electrolytes are also lost to varying degrees. In the short term, it is usually only necessary to replace Na+, K+ and glucose. The other electrolytes usually do not need replacement until around 1 week of parenteral therapy.

-Pediatric Considerations
- -Pediatric patients should be aggressively rehydrated after a surgical procedure for two reasons:
  - -They will lose a higher percentage of their total fluid volume during a procedure.
  - -They have a tendency to "third space" and shift fluid balances in the perioperative period.
- -To determine the total intravascular volume of a pediatric patient:
  - -The first 10kg of body weight account for about 80ml/kg.
    - -So a 7kg kid would be (7x80) = 560ml
  - -The next kg's account for about 70ml/kg
    - -So a 25kg kid would be (10x80 + 15x70) = 1850ml

-General Recommendations:
- -At the very least you should replace fluid to account for water loss. This is at least 1L/day, but you can certainly increase this and lose the excess through the urine.
- -It is also recommended to provide some electrolyte supplementation:
  - -Na+: 50-150 mEq/day
  - -K+: 20-60 mEq/day
  - -Glucose: 100-150g/day to minimize protein catabolism and ketoacidosis

-Common parenteral solutions:

IV Solution	Osmolality (mOsm/kg)	Glucose (g/L)	Na+ (mEq/L)	Cl- (mEq/L)
D5W	278	50	0	0
D10W	556	100	0	0
D50W	2778	500	0	0
0.45% NaCl	154	(5% available)	77	77
0.9% NaCl	308	(5% available)	154	154
3% NaCl	1026	0	513	513
Lactated Ringer's	274	(5% available)	130	109

-LR also contains 4 mEq/L K+, 1.5 mEq/L Ca2+, and 28 mEq/L lactate

-Common administrations:
- -Normal adult: NS or 1/2 NS or LR at 75-120ml/hr +/- 20mEq KCl
- -Diabetic patients: D5-1/2NS at 50-100ml/hr +/- 20mEq KCl while NPO

  There usually isn't a need to deliver extra glucose (D5) to diabetic patients while they are PO.

-The key to fluid management is an understanding and knowledge of exactly why you are giving fluids in the first place, what you hope to accomplish, what substances you are giving in the fluid and how much you are giving.

-Obvious care needs to be taken with diabetic patients, those with renal pathology, and those with CHF.

Additional Reading:

-[Grocott MP, et al. Perioperative Fluid Management and Clinical Outcomes in Adults. Anesth Anal. 2005 Apr; 100(4):1093-106.]
-[Paut O. Recent developments in the perioperative fluid management for the paediatric patient. Curr Opin Anaesthesiol. 2006 Jun;19(3):268-77.]

AJM Sheet: Post-Op Fever

-General Information

-When dealing with a fever work-up, always note what the baseline temperature of the patient is and the method of measurement.
-Fever in most institutions is defined as greater than **101.5° F**.
-Temperatures between 98.6-101.5° are low-grade fevers.

-Intra-operative causes of fever

-Inflammatory process of the surgical procedure itself
-Pain
-Transfusion Reaction
-Malignant Hyperthermia
-Pre-existing Sepsis

-The 5 "W's" of Post-Operative Fever

-**Wind**: Atelectasis, aspiration pneumonia, PE
-**Wound**: Surgical site infection, thrombophlebitis (IV site), pain
-**Water**: UTI, dehydration, constipation
-**Walk**: DVT
-**Wonder Drugs**: Virtually any drug can cause fever, but the most common are antimicrobials and heparin.

-Timeline General Guide

-**0-6 hours post-op**: Pain, anesthesia rxn, rebound from cold OR, endocrine causes (thyroid crisis, adrenal insufficiency)
-**24-48 hours post-op**: atelectasis, aspiration pneumonia, dehydration, constipation
-**72+ hours**: infection (3-7 days), DVT, UTI, drug allergy, thrombophlebitis

-Temperature General Guide (in degrees F)

-**107**: Anesthetic Hyperthermia
-**106**: -
-**105**: Blood transfusion reaction
-**104**: Closed abscess
-**103**: Atelectasis; pneumonia; drug reaction; liver disease
-**102**: Wound infection
-**101**: Draining abscess
-**100**: Benign post-op fever; post-anesthesia overshoot

-General Knowledge

-Usually only two infectious agents can cause a fever within a few hours of surgery:
    -Group A Strep (GAS)
    -Clostridium perfringens
-Dialysis patients typically run approximately 1 degree F cooler than the normal population, so a fever for HD patients wound be defined as **100.5° F**. This is hypothesized to be due to a resetting of the hypothalamic set point.
-The majority of causes of fever are non-infectious. AJM always carries with him a copy of the DDX of fever copied from Harrison's text (it's 2 pages long!). Common non-infectious causes of post-op fever include:
    -Surgical site inflammation
    -Seroma
    -Hematoma
    -Pain
-The purpose of any fever work-up is to **find the source**!
    -If you are thinking infection, then infection from where: Surgical site? Pulmonary? Urine? Blood? Does the patient have any peripheral vascular access lines?

AJM Sheet: Deep Vein Thrombosis (DVT)

-Signs and Symptoms

-Pain

-Homan's Sign: Pain in calf with dorsiflexion of the ankle

-Pratt's Sign: Pain with compression of the calf

-Edema -Fever -SOB

-Calor -Palpation of clot

-Risk Factors

-**Virchow's Triad**:
- -Hypercoagulable state
- -Immobilization
- -Vessel Wall Injury

-History of DVT
-Family History of DVT
-Pregnancy
-Oral Contraceptives
-Age > 75
-Malignancy

-Collagen Vascular Dz
-Trauma
-Infection
-Post-partum
-Hormone Replacement Therapy
-Obesity -HIV/AIDS

-There is also the acronym **I AM CLOTTED**: **I**mmobilization, **A**fib/CHF, **M**alignancy/MI, **C**oagulopathy, **L**ongevity (age), **O**besity, **T**rauma, **T**obacco, **E**strogen/BCP/HRT, **D**VT/PE history.

-Diagnosis

-Compression Ultrasound: can actually visualize the clot

-**D-Dimer > 500µg/ml**: Not sufficient as a stand alone test

-Consider full coagulation work-up for hypercoagulable states

-Contrast venography

-Impedance plethysmography

-Treatment

-Goals of Treatment:
1. Prevent pulmonary embolism
2. Prevent clot extension
3. Prevent recurrence

-Immediate Anti-Coagulation

-IV Unfractionated Heparin

-Law of 8018

-Initial Dose 80mg/kg IV bolus and then 18mg/kg/hour

-PTT should be checked q6 until it stabilizes at **1.5-2.5X normal** (46-70s)

-Goal is to get PTT in this range

-LMWH may also be used

-Enoxaparin (Lovenox): **1mg/kg subcutaneous q12**

-Heparin Dosing Guide

-Initial Dose: Law of 8018 with PTT checks q6

-*If PTT <35s*: 80 units/kg IV bolus, then increase infusion rate by 4 units/kg/hr

-*If PTT 35-45s*: 40 units/kg IV bolus, then increase infusion rate by 2 units/kg/hr

-*If PTT 46-70s*: No change to dosing. Continue with 18mg/kg/hour infusion rate

-*If PTT 71-90s*: Decrease infusion rate by 2 units/kg/hr

-*If PTT >90s*: Hold infusion for 1 hour, then decrease infusion rate by 3 units/kg/hr

-Continued Anti-Coagulation

-Warfarin (Coumadin)

-Load at **10mg or 7.5mg PO qdaily** for 2 days

-Decrease/adjust dose to a target **INR=2.5**

-DO NOT stop heparin infusion until INR reaches 2.5

-INR should be maintained at 2.5 for 3-12 months

-Consider placement of IVC filter (inferior vena cava)

-Pulmonary Embolism (PE)

-PE occurs when a clot from a peripheral location embolizes to the pulmonary vasculature

- <25% of deep vein thromboses distal to the iliac veins go on to develop PE.

-The more proximal the clot, the more likely it is to develop into a PE.

-"**Classic Triad**" of signs and symptoms of a PE: Dyspnea/SOB, Chest Pain, Hemoptysis

-Please note that less than 14% of patients experience the classic triad

-Diagnosis of a PE

-Gold standard: Pulmonary Angiography, Spiral CT

-V/Q study

-CXR

-Treatment of PE

-Thrombolytic Therapy: -Urokinase: **4400units/kg IV over 10 min, then 4400units/kg/hr for 12 hours**

-Streptokinase: **1.5 million units IV over 60 minutes**

-Pulmonary Embolectomy

-Various filters

AJM Sheet: Pain Management

-Pain Management is a subject that you will be dealing with a lot during residency, but something that you won't receive much formal education on. Honestly, you probably won't get many interview questions about it either, but it's something that I think is important. *Clinics in Podiatric Med and Surg* had a whole edition to the subject (July 2008) that is worth reading. Specifically for the residency interview, read Articles 1, 5 and 8. I also wrote the "Perioperative Pain Management" chapter in the 4th edition of McGlam's.

-The "attack points" are a concept that AJM made up to promote an active approach to multimodal pain management.

-Acute Operative Pain Physiology "Attack Points" (In *Clinics: The Physiology of the Acute Pain Pathway*)

Attack Point	Physiology	Intervention
Stimulus	**Stimulus**: Noxious stimuli resulting in tissue damage to superficial and deep somatic structures. **Transduction**: Nociceptor activation by chemical, mechanical or thermal means. **Post-Injury Inflammatory Response**: Normal response to cellular damage with the chance to develop into the pathophysiologic mechanism of peripheral sensitization.	Resolution/Limitation of Stimulus -Prevention of secondary aggravation Anti-Inflammatory Pharmacologics -NSAIDs Carboxylic Acid Derivatives Proprionic Acid Derivatives Acetic Acid Derivatives Fenamates Enolic Acid Derivatives Naphthylkanones COXII Selectives -Non-NSAIDs -Anti-histamines -Topical Agents -Local Anesthetics Anti-Inflammatory physiotherapeutics
Transmission	**Peripheral sensory afferents** carrying the action potential of the noxious stimulus from the periphery to the CNS. -Normally controlled by myelinated A-delta and unmyelinated C fibers. -**Allodynia** is a pathophysiologic state of sensitization when A-beta fibers transmit light touch as painful.	Local Anesthetics Sodium Channel Blockers
Modulation	Spinal Cord Dorsal Horn -Peripheral Excitatory Signals -Peripheral Inhibitory Signals -Central Excitatory Signals -Central Inhibitory Signals	Opioids Calcium Channel Blockers NMDA Receptor Antagonists Beta-adrenergics Anti-Inflammatories
Perception	Ascending Central Processes Descending Central Processes Patient Emotional Response	Anxiolytics Anti-Depressants Patient Education

-Multimodal Approach to Active Pain Management

A passive unimodal therapy like Percocet has very little total effect on the physiology of pain. Opioids and acetaminophen influence small portions of the modulation attack point, but essentially do not influence any of the other attack points. A multimodal approach actively intervenes at several attack points with several therapies to interrupt the known physiologic and pathophysiologic mechanisms.

-Pre-emptive Analgesia (In *Clinics: Perioperative Pain Management*)

-The concept of pre-dosing pain medications before surgery to interrupt pain pathways before they start

AJM Sheet: AO

-AO: Arbeitsgemeinschaft fur Osteosynthese fragen

-History

-Plates and screws for fx fixation first described by Alain Lambotte in 1907.

-Robert Danis (Belgium surgeon) published "The Theory and Practice of Osteosynthesis" in 1949.

-Described use of compression plate called a coapteur.

-Maurice Müller, a pupil of Danis, founded AO with other Swiss surgeons in 1958.

-Principles of AO

1. Accurate and precise anatomic reduction of fracture fragments (especially in joints).
2. Atraumatic surgical technique with emphasis on preservation of blood supply.
3. Rigid/Stable fixation
4. Early mobilization

-The "*Guide to Internal Fixation*" by the AO group is a great book that reads fairly quickly. You should also read the text "*Internal Fixation of Small Fractures*" and "*AO Principles of Fracture Management*" from the AO group. General notes from these books are included throughout the following sheets dealing with specific traumatic fractures.

-General Principles of the Lag Technique

-*Why?* Generates compression. *So?* Compression leads to lack of motion and therefore primary bone healing. Motion disrupts angiogenesis, decreases oxygen tension levels and inhibits osteogenesis. So, **it is the lack of motion and NOT the compression that is osteogenic**.

-Orientation of the screw 90° to the fracture line obtains optimal compression.

->20° displacement from perpendicular is significant

-Weakest in translation from axial loading

-Orientation of the screw 90° to the long axis optimally prevents displacement with axial loading.

-Weak in compression

-Ideal screw placement for a long, oblique fracture:

-One central anchor screw 90° to the long axis

-One proximal and one distal compression screw 90° to the fracture line

-Principles of Insertion

-AO Recommendations: Overdrill, Underdrill, Countersink, Measure, Tap, Screw

-Some underdrill before overdrill

-Some don't overdrill until after tapping

-Two finger tightness = 440-770lbs.

-To prevent thermal necrosis:
-Sharp tip
-Fast advancement (2-3mm/sec)
-Slow drill speed (300-400rpm)
-Firm force (20-25lbs)

-Screw Pull-out

-Directly proportional to screw diameter, screw length and bone strength (cortical nature).

-Indirectly related to pilot hole diameter.

-To increase screw pull-out, maximize bone-screw contact.

-Fairly Irrelevant Definitions

-**Stress**: pressure on a material

-**Strain**: measurable deformation following a given stress

-**Stress-Strain Curve/Load Deformation Curve**

-**Elastic Range**: -Non-permanent strain/deformation with a given stress
-Proportional stress and strain (**Hook's Law**)
-Slope of the line is the stiffness (**Young's Modulus of Elasticity**)

-**Yield Point**: -Past the yield point, a given stress causes a non-proportional increase in strain.

-**Plastic Range**: -Permanent deformation past the yield point

-**Ultimate Failure Point**

-**Fatigue Failure**: failure from repetitive cyclic loading

-**Creep**: temperature dependant permanent deformation of a metal

-Stress Shielding: Internal fixation absorbs physiologic stress from bone and results in bone resorption per **Wolff's Law**.

AJM Sheet: Screws and Plates

Screws

-Screw Anatomy/Definitions

-**Head**: more efficient hexagonal vs. cruciate

-**Land**: underside of the head which contacts the near cortex. Want as much land-bone contact as possible to reduce stress at any one location. This is the same principle as washers and countersinking.

-**Shank**: unthreaded portion of the screw

-**Run-out**: junction between the shank and the threads. Represents the **weakest portion** of the screw.

-**Thread diameter**: diameter of threads + core (**major diameter**)

-**Core diameter**: diameter without the threads (**minor diameter**)

-**Pitch**: distance between threads

-**Tip**: can be round, trocar or fluted

-**Axis**: central line of the screw

-**Rake Angle**: thread to axis angle

-**Thread Angle**: angle between the threads

-Cortical and Cancellous Screws

-***Please*** memorize **Table 1, page 76 in McGlamry** (Also AJM List: page 17)(Only in 3rd edition of McGlam's…not in 4th edition!)

-Cortical: tighter pitch designed for hard cortical bone (1.25mm)

-Cancellous: higher pitch designed for metaphyseal and epiphyseal bone (1.75mm)

-Self-Tapping Screws

-Fluted tip that clears debris as it is advanced

-Require larger pilot holes, have decreased thread-bone contact and have the ability to cut its own path different from the underdrill

-Cannulated Screws

-Classically 3.0, 4.0, and 7.3mm, but really have just about any size available now

-Advantages: self-drilling, self-tapping, good for hard to visualize fractures, avoids skiving of cortical bone on insertion and has definite co-axial nature with K-wire.

-Disadvantages: hollow core, decreased thread-core ratio, decreased pull-out strength

-Herbert Screws

-Proximal and distal threads separated by a smooth shaft. Headless.

-Leading threads have increased pitch, so it draws the trailing threads.

-Does generate interfragmental compression, but not a lot.

-Interference Screws

-FT, headless screw

-Prevents axial displacement. Does not generate compression.

-Malleolar Screws

-Essentially a self-cutting, PT cortical screw.

Plates

-General:

-**Quarter Tubular Plate**: For use with screws from the mini fragment set

-**One-Third Tubular Plate**: For use with screws from the small fragment set

-Many other shapes and sizes of plates are available that specifically fit just about any bone/situation.

-General Plate Characteristics:

-**Dynamic Compression (DCP)**: Wider/Deeper holes that allow for **eccentric drilling** and **axial compression**

-**Limited Contact (LC)**: Essentially grooves on the underside of the plate that limit periosteal contact

-**Locking**: see -Miranda MA. Locking plate technology and its role in osteoporotic fractures. Injury. 2007 Sep; 38 Suppl 3:S35-9.
-Egol KA, et al. Biomechanics of locked plates and screws. J Orthop Trauma. 2004 Sep; 18(8): 488-93.

-General Plate Functions:

-Neutralization -Interfragmentary Compression

-Buttressing -Tension Band

-AO Basic Stabilization Rule: Ideally you want 3 or 4 cortical threads in each main fragment distally, and 5 or 6 proximally.

AJM Sheet: Suture Sheet

-Suture materials are best classified as to whether they are ***absorbable vs. non-absorbable***, ***synthetic vs. natural***, and ***monofilament vs. multifilament***.

1. **Absorbable** (usually used for deep closure)
    A. **Natural**
        -Pig collagen, sheep intestine, cow intestine or cat gut
        -May be **chromic** (treated with chromic salts to increase strength and decrease hydrolysis)
        -Digested by lysosomal enzymes in ***20 days***
    B. **Synthetic**
        1. **Vicryl** (Polyglactin 910)
            -Braided. May be coated (polyglactin 370 or calcium stearate)
            -65% tensile strength at 14 days
            -Hydrolyzed (to CO2 and H20) in ***80-120 days***
            -**Vicryl Rapid**: Hydrolyzed in 42 days; loses strength in 7-10 days
            -**Vicryl Plus**: Coated with broad spectrum antibiotic **Triclosan** (also found in toothpaste)
        2. **Dexon** (Polyglycolic acid)
            -Braided. May be coated (polycaprolate 188)
            -Hydrolyzed in ***100-200 days***
        3. **PDS** (Polydiaxonone)
            -Monofilament
            -70% tensile strength at 14 days
            -Hydrolyzed in ***90 days***
        4. **Maxon** (Polyglyconate)
            -Monofilament
            -Hydrolyzed in ***180 days***; Longest lasting absorbable ("Max"-imum)
        5. **Monocril** (Poliglecaprone)
            -Monofilament
            -20-30% tensile strength at 14 days
            -Hydrolyzed in ***90-120 days***
2. **Non-absorbable** (usually used for superficial closure/skin sutures)
    -It can be argued that all sutures are eventually absorbable!
    A. **Natural**
        1. **Silk**
            -Made from silk worm
            -Actually very slowly absorbed (hydrolyzed in ***1 year***)
            -Very low tensile strength
        2. **Cotton/Linen**
            -Weakest suture
    B. **Synthetic**
        1. **Nylon** (Ethilon, Surgilon)
            -Both monofilament and braided available
            -Highest "knot slippage" rate: monofilaments are at a higher risk of knot slippage
        2. **Polypropylene** (Prolene, Surgilene)
            -Monofilament
            -Can be used in contaminated/infected wounds (Nonabsorbable, synthetic, monofilaments best in this situation). This is the least reactive suture.
        3. **Polyester** (Ethibond, Dacron)
            -Strong suture. May be used for tendon repair.
            -Braided. May be coated with silicone.
        4. **Fiberwire** (polyethylene multifilament core with a braided polyester jacket)
        5. **Stainless Steel**
            -Monofilament or braided (braided is called Flexon)
            -**Strongest** suture with **longest** absorption rate
            -Used for bone fixation and tendon repair, but may corrode bone at stress points

Other Notes:

-Sutures are also classified according to size. They can range from 0-0 (very thick) to 9-0 (extremely thin).

-Surgeon's choice is extremely variable and you usually just work with what you are used to, but here are some safe bets:
    -Capsule closure: 2-0 or 3-0 Vicryl
    -Subcutaneous tissue closure: 3-0 or 4-0 Vicryl
    -Skin: 4-0 Nylon or Prolene

-Skin sutures are removed at 10-14 days because at this point the tensile strength of the wound equals the tensile strength of the suture.

-Potentially interesting new suture technology: V-loc™. Suture strand contains barbs like a fishing hook, obviating the need for generation of a knot. Developed and used primarily by plastic surgeons, but becoming more popular in other specialities.

-This was the very first AJM sheet!

AJM Sheet: Podiatric Surgery Instrumentation
This sheet is simply a summary of the first chapter of McGlamry's text by Dr. Malay (3rd edition)

-General Information

-Surgical Instruments are composed of **stainless steel** which itself is composed of several different metals:
- **-Carbon**: gives instrument "hardness"
- **-Chromium**: chromium oxide layer prevents corrosion of instrument
- -**Tungsten Carbide**: extreme "hardness" for grasping surfaces (teeth of needle drivers)
- -**Nickel**
- -**Molybdenum**

-There are two different series of stainless steel depending on how it is manufactured:
- -**300 series Austenitic**: Implants and internal fixation. Resists corrosion with resilience.
- -**400 series Martenitic**: Cutting instruments. Hardness maintains sharp edges and jaw alignments.

-Categories of Instruments

***Know how to appropriately handle each instrument if handed it during an interview.
***Be able to identify each instrument if handed it or shown a picture during an interview.

1. **Surgical Blades**
   -Most common: 10, 15, 11, 62 on a minihandle
   -Purpose: Sharp (blade) and blunt (handle) dissection
   -Cutting edge width: **0.015"**
2. **Scissors**
   -Most common: Tissue: Metzenbaum, Mayo, Iris, Crown&Collar (Sistrunk)
   Non Tissue: Suture, Utility, Bandage
   -Purpose: Dissection
3. **Hemostats**
   -Most common: Mosquito (Halsted), Kelly, Crile
   -Purpose: Grasping and holding
4. **Pick-ups**
   -Most common: 1-2 (Rat tooth), Adson-Brown, Atraumatic (Potts-Smith)
   -Purpose: Grasping and Holding
5. **Retractors**
   -Most Common: Hand Held: Skin Hooks, Senn, Ragnell, Malleable, Army-Navy, Volkmann Rake, Meyerding
   Self-retaining: Weitlaner, Holzheimer, Heiss
   -Purpose: Retraction and exposure
6. **Elevators**
   -Most Common: Freer, Sayre, Key, Crego, McGlamry, Langenbeck
   -Purpose: Dissection
7. **Rasps**
   -Most Common: Joseph, Maltz, Bell, Parkes
   -Purpose: Cutting
8. **Miscellaneous**
   -Osteotomes
   -Chisels
   -Gauges
   -Mallets
   -Bone-Cutting Forceps
   -Rongeurs
   -Trephine
   -Curettes
   -Bone Handling Clamps
   -Reduction Forceps (Lewin, Lane, Lowman, Verbrugge)
   -Needle Holders (Mayo-Heger, Sarot, Ryder, Halsey, Webster)
   -Suction-Tip (Frazier)

AJM Sheet: Power Instrumentation General Information

This sheet is simply a review of McGlamry's Chapter 2 by Dr. Alfred Phillips (3rd edition)

-General Information

-*Hardest material in the human body?* Teeth Enamel
-*Power instrumentation developed by which medical field?* Dentistry

-Power Sources (3)

1. **Pneumatic**
   -Advantages: Delivers high power and torque, does not overheat, cheap
   -Disadvantages: Does not operate at slow speeds, bulky, burdensome, cords prone to contamination
   -General: -Most commonly driven by **compressed nitrogen**
   -Tank pressure **> 500 psi**
   -Dynamic instrument pressure: **90-110 psi**
2. **Electric**
   -Advantages: Light, quiet, small, good for office use
   -Disadvantages: Prone to overheating, expensive
   -General: -Utilizes an **alternating current** drive
3. **Battery**
   -Advantages: No cords
   -Disadvantages: Loses power quickly, bulky handling
   -General: -Utilizes **direct current**

-Brands: Stryker, Hall-Zimmer, Microaire

-Definitions

-**Torque**: Measurement of power and force. Units: **Newtons/cm^2**
-Cortical bone requires more torque to cut through than cancellous bone.
-**Speed**: Distance per time
-Pod procedures usually require **20,000 rpm**.
-Decrease risk of thermal necrosis by decreasing torque and increasing speed.
-**Collet**: Distal end of a saw where the saw blade attaches
-**Stroke**: One arc of excursion for a saw blade
-**Oscillation**: One back and forth motion of a saw blade. (Two strokes equal one oscillation).

-Power Saws

-Types
-**Sagittal Saw**: Cuts in the same plane as the instrument
-Better for longer and deeper cuts
-**4° arc of excursion**
-Blade may be positioned anywhere within a 160-180° arc.
-**Oscillating Saw**: Cuts in plane perpendicular to instrument
-**7° arc of excursion**
-Blade may be positioned anywhere within a 360° circle.

-**Blades**

-Vary by cutting depth, width, thickness, shape and number of teeth
-Shapes: straight (most commonly used), inward flair, outward flair
-**The angulation of the teeth and NOT the thickness of the blade determine the thickness of a cut**.
-Blades may contain holes which collect debris, thereby decreasing heat and friction.

-Wire Drivers

-**K-Wires** (Kirshner wire)
-Sizes: 0.028", 0.035", 0.045", 0.062"
-Threaded vs. Non-threaded. Note that the direction of the driver only matters with threaded wires.
-K-Wires provide **splintage** (stability, but no compression)

-**Steinman Pins**
-Sizes: 5/64"-3/16"

-Rotary Cutting

-**Power Drill Bit Sizes**: 1.1, 1.5, 2.0mm
-**Burrs**
-Shapes: Round, Barrel, Straight, Straight-tapered
-Definitions: -shank vs. shaft vs. head
-flute vs. blade
-edge angle vs. clearance angle vs. rake angle

-Surgical Skills Section

-Surgical skills are something best learned by **practice, practice, practice**. A few hints are listed below:
-The surgeon's hands provide 3 functions when operating power instrumentation:
-Control of power of the instrument
-Control of direction of the instrument
-Stability between the instrument and the surgical site
-Review concepts of: -**Axis guide**
-**Reciprocal planing**
-With a saw or K-wire, always divot perpendicular to the cortex, and then redirect.
-The spin of a burr should be parallel to the grain of the cortex or parallel the ridge of bone to be removed.
-Poor man's ways to practice handling and control of surgical instruments:
-K-wire through a Nerf ball
-Sagittal saw through a wine cork or wood blocks

AJM Sheet: Biomaterials
Summary of McGlamry's Chapter 3 by Dr. Cicchinelli (3rd edition)

-Properties of the ideal implant material: clinically inert, no inflammatory or foreign body response, noncarcinogenic, nonallergenic, structurally stable, capable of sterilization, capable of fabrication in desired forms, serve as a scaffold for new bone growth and gradual biodegradation.

-Host Response to Implant

-Cellular Response (Acute)

-Immediately after implantation, implants are covered with a coat of proteins that denature and elicit an inflammatory response. Denatured fibrinogen accumulates neutrophils and macrophages.

-**Detritic Synovitis**: Foreign body reaction to shards of silicone materials in the lymphatic system.

-**Environmental Stress Cracking**: Surface defects on polyetherurethane implants secondary to chronic inflammation. Chronic inflammation results from fragmentation and leads to intracortical lysis and cyst formation.

-**Tissue Remodeling Response**: Normal for implants to have fibrous capsule formation.

-Infection Potential

-Susceptible to S. Aureus and S. Epidermidis infections

-Malignancy and Type III hypersensitivity reactions extremely rare

-Biomaterials

-**PLLA** (Polylactic-L-Acid: L is enantiomer)

-Degrades to lactic acid via hydrolysis

-Retains strength 36 weeks and degrades in 2-3 years

-Available in FT 2.0, 2.7, 3.5 and 4.5mm screws

-**PGA** (Polyglycolic Acid)

-Degrades to glycolic acid and glycine

-Elliptical. Provides compression secondary to shape.

-Brittle and rigid

-Highest likelihood of FB rxn or complication (<4%)

-**PDS** (Poly-para-dioxanone)

-Tapered form swaged on metallic wire. Provides compression secondary to shape.

-Flexible and malleable

-Increased degradation times are good because it decreases the load the body has to clear.

-These screws don't "bite" like metal screws, but swell 2-4% in the first 48 hours.

-**Advantages**: decreased stress shielding, no second operation for removal.

-**Disadvantages**: more expensive than metallic screws, but are cheaper in the long run if you remove >31% of metallic screws in your practice.

-Metallic Implants

-Surgical Stainless Steel

-316LVM (low carbon vacuum remelting)

-Iron, 17-25% chrome, 10-14% nickel, 2-4% molybdenium, 1% carbon

-Nickel most commonly causes reaction: allergic eczematous dermatitis.

-**Titanium**

-Very inert, integrates into surrounding bone, resists corrosion, decreased capsule formation

-Addition of 6% aluminum and 4% vanadium increases the strength similar to steel

-Nitrogen implantation forms a stable oxide layer

-Black metallic wear debris is often seen. No toxicity or malignancy associated with this.

-Cobalt Chrome and Alloys

-30% cobalt, 7% chromium, <0.034% moly/carbon

-Used in joint replacement prostheses

-**Corrosion**: breakdown of metallic alloys because of electrochemical interactions within the environment

AJM Sheet: General External Fixation

-Selected History

-377BC: Hippocrates with wood from a cornel tree
-1904: Codvilla (Italy) used unilateral fixator for limb lengthening
-1951-1991: Ilizarov (Siberia, Russia). Father of modern ex-fix and developer of external ring fixator for WWII vets from old bus parts.

-General Principles

-Tension-Stress Effect (Ilizarov)

-Distraction performed at proper rate and in the proper area leads to tissue growth similar to hormone-mediated growth at adolescent growth plates.
-Too fast: Stretching and traction injuries
-Too slow: Bone callus consolidation preventing future distraction

-An important principle is that all tissues (bone, skin, muscle, NV structures, etc.) become mitogenically active and grow. **They proliferate as opposed to "stretching".** Much of this has to do with the distraction serving as a mechanical stimulus for growth factor release (such as osteoblastic growth factor) and dramatic increases in vascularity.

-**Tension-Stress Effect Influences:**

-**Stability**: increased stability leads to increased osteoblastic activity
-**Rate**: Ideal is 1mm/day in 4 increments
-**Bone Cut**: Best to keep medullary canal and as much periosteum intact as possible. Best technique is a percutaneous subperiosteal corticotomy with a Gigli saw or osteotome/mallet.
-**Location of Bone Cut**: Metaphysis found to be superior to other areas

-**Behrens Principles of External Fixation**

-Avoid and respect neurovascular structures
-Allow access to injured area for future fixation
-Meet mechanical demands of the patient and the injury

-**Tajana's Stages of Callus Development**

-**Colloidal** (0-2 weeks): formation of microreticular network
-**Fibrillar** (2 weeks-1 month): collagen organization
-**Lamellar** (1 month-years): formation of compact lamellar tissue and calcification

-**Advantages of Ex-Fix**

-Decreased soft tissue dissection
-Immobilization of multiple regions
-Allows for post-operative adjustment
-Skin grafting and wound debridement available
-Early ROM and WB

-Complications of Ex-Fix

-Pin tract infection vs. irritation
-Pain
-Cage rage
-Non-unions
-Fracture
-NV injury

-Anatomy

-Knowledge of cross-sectional anatomy is essential for the application of external fixation. There are numerous manuals and tests available demonstrating proper pin and wire placement in a given location.
-The key is to have solid bone with avoidance of neurovascular structures.
-As a general rule, the medial and anterior aspects of the tibia are safe locations.

-Types of External Fixators

-Unilateral Fixators
-EBI Dynafix and Orthofix
-Can be straight (uniplanar) or articulated (multiplanar)
-Allow for compression/distraction in a **single plane only**
-Attached to bone via half-pins
-Rigidity and stiffness determined by half-pins/bone interface. Want pins spread over a large area.
-Weak in the sagittal plane

-Circular Fixators (Multi-lateral)
-Smith&Nephew
-Generate compression/distraction in multiple planes
-Tensioned wires generate stability; half-pins generate rigidity.
-Best if these are located 90° to each other for optimal stability
-Can be formulated to allow for immediate WB

-Hybrid Fixators
-Orthofix, Dynafix, Smith&Nephew, Rancho
-Combination of unilateral and circular fixators

-Taylor Spatial Frames
-Smith&Nephew
-Allows for reduction of triplanar complex deformities

-Brief Indications

-Limb Lengthening/Distraction
-Percutaneous metaphyseal subperiosteal corticotomy with Gigli saw or osteotome/mallet
-Apply fixation before corticotomy
-Distraction begins 7-14 days after corticotomy at 1mm/day

-Angular Deformities
-CORA principle (center of rotational angulation)
-Double Taylor spatial frame
-Dynamization: release of tension from wires and loosening of half-pins to allow bone a period of introductory WB

-Fracture
-Ligamentotaxis: pulling of fracture fragments into alignment with distraction

-Arthrodesis

AJM Sheet: Bone, Bone Healing and Wound Healing

-Bone Properties/Variables

-Bone is a two component system consisting of **minerals** (increases the yield and ultimate strength of bone) and **collagen** (mostly Type II).

-Variables:

-Porosity. Increased porosity leads to increased compressive strength of bone. Cortical bone has <15% porosity and cancellous bone has ~70% porosity.

-Strength. Strength is defined as the amount of force a material can handle before failure. Bone can handle a 2% increase in length before failure. Bone is has the greatest strength in compression, followed by tension and is weakest in shear. Strength is affected by collagen fiber orientation, trabecular orientation, age, presence of defects and osteoporosis.

-Stiffness. Cortical bone has 5-10 times the stiffness of cancellous bone.

-Vascular Supply to Bone

-Blood supply to bone comes from two sources. A **nutrient artery** feeds the endosteal and medullary vessels and supplies the inner 2/3-3/4 of bone. The **periosteal vessels** supply the outer 1/3 of bone from muscle and tendon attachments.

-The amount of vascular disruption following a fracture depends on the force/displacement of the fracture and which vascular systems are disrupted.

-Phases of Bone Healing

-Inflammation (10%)

-Hematoma fills the area with fibrin, RBCs, neutrophils, platelets, macrophages, fibroblasts (from PMNs).

-Mesenchymal cells from the cambium layer differentiate into osteoblasts and chondrocytes.

-Chemotaxis by growth factors (transforming growth factor beta, platelet derived, and macrophage derived)

-Reparative/Regenerative (40%)

-Soft callus forms and is replaced by bone.

-Cartilage, fibrocartilage, collagen and hydroxyapatite deposition

-Cartilage replaced by bone like endochondral ossification

-Remodeling (70%)

-Callus completely replaced by bone

-Vascular network is normalized

-Remodels according to Wolff's Law

-Piezoelectric Effect: appearance of electrical potentials within bone in response to the application of an external force

-Compression side: electronegative leading to bone production

-Tension side: electropositive leading to bone resorption

-Types of Bone Healing

-Direct Osseous Repair (Primary Intention, Direct Healing)

-No callus formation; no motion

-Cutting cone: Osteoclasts in the front, osteoblasts in the back. Travels across the fx line (Schenk and Willinegger).

-Gap Healing: Bone deposition at 90° to the orientation of bone fragments

-Indirect Osseous Repair

-Callus formation

-The literature has demonstrated that cyclic loading and dynamization have resulted in decreased healing times, decreased stiffness, increased torque and increased energy absorption in rabbit and dog bones. A practical means to accomplish this in human subjects hasn't been perfected yet.

Wound Healing

Additional Readings:

-[Broughton G, Janis JE, Attinger CE. Wound healing: an overview. Plast Reconstr Surg. 2006 Jun; 117(7 Suppl): 1S-32S.]
-[Broughton G, Janis JE, Attinger CE. The basic science of wound healing. Plast Reconstr Surg. 2006 Jun; 117(7 Suppl): 12S-34S.]
-[Hunt TK, Hopf H, Hussain Z. Physiology of wound healing. Adv Skin Wound Care. 2000 May-Jun; 13(2 Suppl): 6-11.]
-[Lawrence WT. Physiology of the acute wound. Clin Plast Surg. 1998 Jul: 25(3): 321-40.]
-[Falanga V. Wound healing and its impairment in the diabetic foot. Lancet 2005; 366: 1736-43.]

Phases of Wound Healing:

1. **Substrate/Lag/Inflammatory Stage** (Days 1-4)
   -Inflammation characterized by edema/erythema/calor/dolor
   -PMNs start out dominating, but are eventually taken over by macrophages
2. **Proliferative/Repair Phase** (Days 3-21)
   -Collagen proliferation and macrophages
   -Myofibroblasts also begin working
3. **Remodeling/Maturation Phase** (Day 21+)

AJM Sheet: How to "Work-Up" a Surgical Patient

With regard to specific surgery and the interviews, it's always important to "know your program". In other words, programs tend to have favorite procedures that they routinely do. For a given bunion deformity, one program may primarily do Austin-Akins, whereas other programs may never do an Akin, and still others may always do a Lapidus in the exact same situation. Some people may feel very strongly in favor of the lateral release, while others may never do it for any situation. This could even happen between two attendings at the same program in the same room during your interview! If you give a hard, definitive answer for a procedure choice, one attending may completely agree with you while another may think it's completely the wrong choice. So if you are asked what type of procedure you would do for a given situation, be as general as possible, but always give the reason/specific indications why you are choosing that procedure or group of procedures. Name a couple different similar procedures instead of sticking by your guns with one procedure. Additionally, your interviewers may not expect you to know for sure what procedure to choose, but they will definitely expect you to be able to completely work-up the patient and know which procedures are acceptable for which indications.

The two work-ups that you should have down cold are the **HAV and flatfoot work-ups**. *Practice, practice, practice* working through these situations out loud, and *practice, practice, practice* going through the radiographic analyses of these deformities out loud. Again, RC and I found it helpful while studying for interviews to pick up random podiatry textbooks and just flip through the pages, alternating our description of the radiographs out loud.

There are of course many, many radiographic angles that you can use to describe during either of these work-ups, so focus the majority of your energy on those that will have the most impact on your treatment choice. Here's the way that I think about these deformities. This certainly isn't the "right" way; it's just the way that helped me as I first started doing this out loud:

HAV:

I simply use the radiographic angles to define two aspects of the deformity:

-Where is the deformity?

-In which bone or bones, and/or which joint or joints is there deformity?

-Is the deformity mild, moderate or severe?

Once you have successfully answered these questions in your mind, then the remainder of the radiographic work-up falls into place. For example, if you identify a deformity at the first metatarsal-phalangeal joint, then you can use your radiographic angles to define it:

"In the area of the patient's presenting complaint I see a (mild, moderate, or severe) hallux abductovalgus deformity at the level of the metatarsal-phalangeal joint as defined by a (mildly, moderately, or severely) increased intermetatarsal angle, (mildly, moderately, or severely) increased hallux abductus angle, and approximate metatarsal-sesamoid position of (1-7). The PASA and DASA of this joint appear (within normal limits or deviated). There (does or does not) appear to be a hallux interphalangeus deformity as defined by the (increased or normal) hallux interphalangeus angle. The overall length of the first metatarsal appears (normal, shortened, or long) compared to the remainder of the lesser metatarsal parabola on the AP view. On the lateral view the first metatarsal appears (dorsiflexed, plantarflexed, or normal) compared to the second metatarsal using Seiberg's index. There (is or is not) an underlying metatarsus adductus as defined by the metatarsus adductus and Engle's angles. Generally, the rearfoot appears (rectus, pronated, or supinated) as defined by…"

Now that you have defined the location and severity of the deformity with your angles, suggest procedures *based on these specific abnormal findings*. For every abnormality that you described, suggest a procedure (or group of procedures) to correct it. "I would consider doing a distal metatarsal osteotomy in this case to laterally translate and plantarflex the capital fragment of the first metatarsal to decrease the intermetatarsal and hallux abductus angles in addition to reducing the sesamoids." If you described the DASA and interphalangeus angles as normal, then don't suggest an Akin procedure! If you described a mild deformity, then don't suggest procedures that are indicated for moderate to severe deformities!

I also use the above questions to classify each and every surgical procedure. For each surgical procedure I think: **This procedure will correct for a (mild, moderate, or severe) deformity of this bone or at that joint**.

Flatfoot:

Here I use a similar approach, but think of it in terms of planal dominance:

-In which plane does the deformity present?

"Consistent with the patient's presenting complaint we see a (mild, moderate, or severe) pes planovalgus deformity. In the sagittal plane I see a (decreased or increased) calcaneal inclination angle, talar declination angle, talar-calcaneal angle, first metatarsal inclination angle, Meary's angle, and medial column fault on the lateral view. I would also evaluate the patient for equinus using the Silfverskiold test to determine a sagittal plane deformity. In the transverse plane I see a (decreased or increased) talar-calcaneal angle, cuboid abduction angle, talar head coverage, talar-first metatarsal angle, metatarsus adductus angle on the AP view. In the frontal plane we can see the Cyma Line is (anteriorly displaced, posteriorly displaced or normal) on the lateral view, and that the subtalar joint alignment, ankle joint alignment and calcaneal position are (normal or abnormal) on the long leg calcaneal axial views."

Now that you have defined the deformity on your own terms, you can now suggest how to fix it using the same tools. "I would consider performing a (Gastroc recession, TAL, Cotton osteotomy, medial column arthrodesis, etc.) to correct for the sagittal plane deformity, a (Evans osteotomy, CC joint distraction arthrodesis, etc.) to correct for the transverse plane deformity, and a (medial calcaneal slide, STJ implant, etc.) to correct for the frontal plane deformity.

This is a little philosophic, but radiographic angles aren't real. They only come into reality if you use them, **so only use them as tools to your advantage**. You can use them to first define the deformity on your own terms, and then to show that your intervention was successful.

AJM Sheet: Digital Deformity Work-Up

Subjective
-**CC**: Pt can complain of generalized "corns, calluses and hammertoes."
-**HPI**: -Nature: "Sharp, aching and/or sore" type pain. May have a "tired feeling" in the feet.
-Location: Usually dorsal PIPJ/DIPJ of the toes or submetatarsal
-Course: Progressive onset and course.
-Aggravating factors: WB, shoe gear (especially tight shoes)
-Alleviating factors: NWB, wide shoebox, sandals
-**PMH/PSH/Meds/Allergies/SH/FH/ROS**: Usually non-contributory

Objective
Physical Exam
-**Derm**: -Hyperkeratotic lesions can be seen submetatarsal, dorsal PIPJ or DIPJ of the lesser digits, distal tuft of the lesser digits, or interdigitally. All can have erythema, calor and associated bursitis.
-5th digit is usually dorsolateral at the PIPJ, DIPJ or lateral nail fold (**Lister's corn**). Hyperkeratotic lesion of the adjuvant 4th interspace may also be present (**heloma molle**).
-**Vasc/Neuro**: Usually non-contributory
-**Ortho**: -See discussion on pathomechanics
-Positive **Coughlin test**: Vertical shift of >50% of the proximal phalanx base on the met head. Also called the "draw sign" or Lachman's test.
-**Kelikian push-up test**: Differentiate between a soft-tissue and osseous deformity
-Specific to the 5th digit:
-Toe usually has a unique triplanar deformity (dorsiflexion, adduction and varus).
-Bunionette, splay foot and equinus may be present
-The 5th digit is in the most susceptible position in terms of a muscular imbalance deformity because the FDL has such an oblique pull on the 5th digit as opposed to the relatively axial pull of the other digits.

Imaging
-Plain film radiograph: "**Gun barrel" sign**

Specific Deformities
-**Hammertoe**: Extension at MPJ level; flexion at PIPJ level, neutral/extended DIPJ
-**Mallet toe**: Neutral at MPJ and PIPJ level; flexion at DIPJ level
-**Claw toe**: Extension at MPJ level; flexion at PIPJ and DIPJ level
-**Curly toe**: Claw/hammertoe deformity with an additional frontal plane component
-**Digitus Adductus**: Digital deformity with adduction in the transverse plane
-**Digitus Abductus**: Digital deformity with abduction in the transverse plane
-**Heloma Molle**: Generally occurs in the 4th interspace with a curly toe deformity of the 5th digit. Using this example, the head of the proximal phalanx of the 5th digit abuts the base of the proximal phalanx of the 4th digit causing a hyperkeratotic lesion in the proximal 4th interspace.

Pathomechanics
-Digital deformities are thought to occur via one of three potential mechanisms. Each involves a muscular imbalance at the digital level.
-The way AJM thinks of digits is from distal to proximal. During weight-bearing, the toes cannot function in propulsive gait to aid in load transfer if the most distal segment is not stabilized. The distal phalanx is stabilized by the long flexor tendons holding it solidly against the weight-bearing surface. With the distal phalanx stabilized, the short flexor tendon can hold the middle phalanx against the weight-bearing surface. With the middle phalanx stabilized, the lumbrical muscles hold the proximal phalanx against the ground. The lumbrical muscles must work against the extensor tendon complex, but this complex is usually not actively firing to extend the MPJ during propulsion. The interosseous muscles also stabilize the proximal phalanx in the transverse plane. When the proximal phalanx has been effectively stabilized against the weight-bearing surface, the head of the metatarsal can effectively move through its range of motion and transfer load across the metatarsal parabola. Any disruption in the stabilization process will lead to abnormal biomechanics and deformity.

-Flexor Stabilization:
-Most common origin of hammertoe deformity
-Occurs when the PT muscle is unable to effectively resupinate the midtarsal and subtalar joints at the beginning of propulsion. To compensate, the FHL and FDL fire **earlier, longer and with greater force** to resupinate the foot. This puts too much force on the distal and middle phalanges causing the toe to "buckle" in a dorsiflexed position at the MPJ. This retrograde buckling puts the PIPJ in a vulnerable dorsal position and also pushes the metatarsal head plantarly.

-Flexor Substitution:
-Occurs when the triceps surae muscle group is unable to effectively plantarflex the foot during propulsion for whatever reason. To compensate, the muscles of the deep posterior compartment (PT, FHL, and FDL) again fire earlier, longer and with greater force leading to the same type of deformity.

-Extensor Substitution:
-Can occur in two ways
-One way is when the TA is unable to dorsiflex the foot through the swing phase. In this case the EDL and EHL fire earlier, longer and with greater force than normal and are actually actively extending the MPJ. This easily overpowers the lumbricals and leads to retrograde buckling.
-The other way is in a situation with anterior cavus where the EDL is actually at a mechanical advantage over the lumbricals. Passive stretch of the EDL, rather than active contraction, overpowers the lumbricals and leads to deformity.

AJM Sheet: Digital Deformity Treatment

Conservative

-**Do nothing**: Digital deformities are not a life-threatening condition and can be ignored if the patient is willing to put up with it.

-**Palliative care**: Periodic sharp debridement of hyperkeratotic lesions

-**Splints/Supports**:
- -Metatarsal sling pads
- -Silicone devices
- -Toe crests

-**Orthotics**:
- -Cut-outs of high pressure areas
- -Metatarsal pads to elevate the metatarsal heads
- -Correction of the underlying deformity

Surgical Options

-Two approaches to remembering digital surgical options are the acronym HEECAT, and an anatomic approach thinking of procedures moving from superficial to deep.

-**HEECAT**
- -**H**ead arthroplasty: Post procedure (1882)
- -**E**xtensor hood and PIPJ capsule release
- -**E**xtensor tendon lengthening
- -**C**apsulotomy (MPJ)
- -**A**rthrodesis (PIPJ)
- -**T**endon transfer (flexor longus tendon transfer to function in MPJ plantarflexion)

-**Anatomic Approach**
- -**Percutaneous tenotomy**
  - -Both the extensor and flexor tendons can be transected through a percutaneous approach
- -**Extensor Tendon lengthening**
  - -Done proximal to MPJ level with a Z-lengthening
- -**Capsulotomy**
  - -Of the PIPJ and MPJ
  - -Remember the "J" maneuver for release of the collateral ligaments
  - -Extensor hood release is also usually performed
  - -Some use the McGlamry elevator in this step to free plantar attachments
- -**PIPJ Arthroplasty**
  - -Post procedure 1882
  - -Resection of the head of the proximal phalanx at the surgical neck
- -**PIPJ Arthrodesis**
  - -Fusion of the PIPJ using a variety of techniques: table-top, V, peg-in-hole, etc.
  - -Fusion maintained with K-wire crossing the MPJ extending into the distal 1/3 of the metatarsal
- -**Flexor Tendon Transfer**
  - -Transfer of the FDL tendon dorsally to act as a more effective plantarflexor of the proximal phalanx
  - -Girdlestone-Taylor technique: Tendon is bisected, crossed and sutured on the dorsal aspect.
  - -Kuwada/Dockery technique: Tendon is re-routed through a distal drill hole
  - -Schuberth technique: Tendon is transferred through a proximal drill hole
- -**Syndactyly**
  - -Soft tissue fusion of one digit to a normal adjacent digit to help "bring it down"
  - -Interposing skin is removed and the digits are sutured together

-You should be able to go through the steps of a Post procedure for an interview.

-Please also review the neurovascular elements for each digit and be able to recite which cutaneous nerves supply which corner of each digit.

5^{th} digit skin incisions

-It is possible to alter your skin incision to incorporate a derotational element to your skin closure. While the osseous work can be accomplished using a longitudinal or lazy "s" incision (proximal medial to distal lateral), those are really best for uniplanar deformities. 5^{th} digit HT is usually a triplanar deformity.
- -Two semi-elliptical incisions directed proximal lateral to distal medial.
  - -The more oblique the incision is, the greater transverse plane correction.
  - -The more longitudinal the incision is, the greater the frontal plane correction.

AJM Sheet: Lesser Metatarsal Deformity Work-up

Subjective
-**CC**: Pt presents complaining of "pain in the ball of my foot."
-**HPI**: -Nature: Generalized pain (aching, sharp, sore, etc.)
-Location: Submetatarsal. Can usually be localized to an exact metatarsal.
-Course: Gradual and progressive onset. "Has bothered me for years."
-Aggravating factors: WB for long periods, shoe gear, etc.
-**PMH/PSH/Meds/All/SH/FH/ROS**: Usually non-contributory

Objective: Physical Exam
Derm: -Diffuse or focal hyperkeratotic lesions submetatarsal
Vasc/Neuro: Usually non-contributory
Ortho: -Many of the same signs/symptoms as HT digital deformity. HT often present.
-Anterior displacement of the fat pad -Hypermobility of the first ray
-Anterior Cavus foot type -Hypermobility of the fifth ray
-Equinus

Objective: Imaging
-**Plain film radiograph**: -Look for irregularities of the metatarsal parabola
-Look for excessively plantarflexed or dorsiflexed position on lateral/sesamoid axial views

General Information
-Lesser metatarsalgia has several possible etiologies:
-Retrograde force from hammertoes. Please see AJM Sheet: Digital Deformities.
-An excessively long and/or plantarflexed metatarsal leads to increased load bearing under that particular metatarsal.
-An excessively short and/or dorsiflexed metatarsal can lead to increased load bearing on the adjacent metatarsals.
-Hypermobility of the first ray leads to increased load bearing under at least the second metatarsal.
-Hypermobility of the fifth ray leads to increased load bearing under at least the fourth metatarsal.
-Anterior cavus and equinus deformities lead to increased pressures across the forefoot.
-Before a surgical option is considered, it is extremely important to understand where the increased load is coming from. The goal of treatment should be to restore a normal parabola and weight-bearing function to the foot. Failure to correct the underlying deformity will dramatically increase the rate of recurrence and transfer lesions.

Treatment: Conservative
-**Do nothing**: Lesser metatarsal deformities are not a life-threatening condition.
-**Palliative care**: Periodic sharp debridement of hyperkeratotic lesions
-**Splints/Supports**: -Metatarsal sling pads -Toe crests
-Silicone devices
-**Orthotics**: -Cut-outs of high pressure areas
-Metatarsal pads to elevate the metatarsal heads
-Correction of the underlying deformity

Treatment: Surgical
-Structural correction of lesser metatarsals
-**Distal metatarsal procedures**
-**Duvries**: plantar condylectomy on both sides of the MPJ
-**Jacoby**: "V" shaped cut in the metatarsal neck to allow for dorsiflexion of the head
-**Chevron**: "V" shaped cut similar to a Jacoby, but with removal of a wedge of bone to obtain metatarsal shortening as well.
-**Dorsiflexory wedge osteotomy**: similar to a Watermann of the first metatarsal
-**Weil**: Distal dorsal to proximal plantar oblique cut to allow for distal metatarsal dorsiflexion and shortening. Can be made in several planes to obtain desired dorsiflexory/shortening effects.
-**Osteoclasis**: Through and through cut through the metatarsal neck allowing the distal head to find its own plane.

-**Metatarsal shaft procedures**
-**Cylindrical shortening**
-**Giannestras step-down procedure**: Z-shaped cut which can allow for shortening and distal dorsiflexion.

-**Metatarsal base procedures**
-**Dorsiflexory wedge**: (1mm of proximal dorsal shortening equivalent to ~10 degrees of dorsiflexion)
-**Buckholtz**: Oblique dorsiflexory wedge which allows for insertion of a 2.7mm cortical screw

Complications
-By far, the most common complications are **floating toe**, **recurrence** and **transfer lesions** caused by undercorrection and overcorrection. While you can evaluate the parabola and transverse plane in the OR with a C-arm, you really can't appreciate the sagittal plane.
-Studies have demonstrated that osteoclastic procedures allowing the distal segment to find their own plane without internal fixation have the least occurrence of recurrence and transfer lesions, but they also have a higher rate of malunion, delayed union and non-union.
-[*Derner and Meyr. Complications and Salvage of Elective Central Metatarsal Osteotomies. Clinics Pod Med Surg. Jan 2009.*]

AJM Sheet: 5th Metatarsal Deformity Work-up
Also called: Tailor's Bunion or Bunionette Deformity

Subjective/Objective
-Very similar to work-ups for lesser metatarsal deformities and digital deformities. Pts may complain of pain related to the lateral column in general, 5th digit, plantar 5th met head, lateral 5th met head or 4th interspace heloma molle.

Imaging
-**Plain Film Radiograph**: -**4-5 Intermetatarsal Angle > 9 degrees** (Normal is 6.47 degrees per *Fallat and Buckholtz*)
-**Lateral Deviation Angle > 8 degrees** (Normal is 2.64 degrees per *Fallat and Buckholtz*)
-[Fallat LM, Buckholtz J. J Am Podiatry Assoc. 1980 Dec; 70(12): 597-603.]
-Splay Foot Deformity
-Plantarflexed 5th metatarsal position
-Structural changes to 5th metatarsal head

General Information
-**Etiology**
-Numerous authors have chimed in on the etiology of the 5th Metatarsal Deformity:
-Davies: incomplete development of deep transverse metatarsal ligament
-Gray: malinsertion of adductor hallucis muscle
-Lelievre: forefoot splay
-Yancey: congenital bowing of metatarsal shaft
-Root: abnormal STJ pronation
-CMINT, etc.

Treatment
Conservative
-**Do nothing**: 5th metatarsal deformities are not a life-threatening condition.
-**Palliative care**: Periodic sharp debridement of hyperkeratotic lesions
-**Splints/Supports**: -Shoe gear modification with large toe box
-Derotational tapings
-**Orthotics**: -Cut-outs of high pressure areas
-Metatarsal pads to elevate the metatarsal heads
-Correction of the underlying deformity

Surgical
-**Exostectomy**: Removal of prominent lateral eminence from 5th met head
-**Arthroplasty**: Removal of part/whole of 5th met head
-**Distal Metatarsal Osteotomies**:
-Reverse Hohmann
-Reverse Wilson
-Reverse Austin
-Crawford: "L" shaped osteotomy allows for insertion of cortical screws
-LODO (Long Oblique Distal Osteotomy): similar to Crawford but simply oblique
-Read *[London BP, Stern SF, et al. Long oblique distal osteotomy of the fifth metatarsal for correction of tailor's bunion: a retrospective review. J Foot Ankle Surg. 2003 Jan-Feb;42(1):36-42.]* Especially if externing at Inova!
-Medially-based wedge
-**Proximal Osteotomies:**
-Transverse cuts
-Oblique cuts
-Medially based wedges

AJM Sheet: HAV Work-up

Subjective

CC: "Bump pain," "Big toe is moving over," Typical patient is female although it is unclear whether there is a higher incidence among females, or if there is a higher complaint incidence among females.

HPI: -Nature: Throbbing, aching-type pain
-Location: Dorsomedial 1st MPJ is most typical presentation. Pain could also be more medial (suggesting underlying transverse plane deformity such as met adductus) or dorsal (suggesting OA of 1st MPJ).
-Course: Gradual and progressive
-Aggravating Factors: Shoe wear, WB

PMH: -Inflammatory conditions (SLE, RA, Gout, etc.)
-Ligamentous Laxity (Ehlers-Danlos, Marfan's, Downs syndrome)
-Spastic conditions (40% incidence of HAV among those with CP)

PSH: -Previous F&A surgery

FH: -Hereditary component (63-68% family incidence among general population, 94% with juvenile HAV)
-Johnston reports an autosomal dominant component with incomplete penetrance

Meds/All: Usually non-contributory

ROS: Usually non-contributory

Objective: Physical Exam

Derm: -Dorsomedial erythema +/- bursa
-Submet 2 lesion
-Nail bed rotational changes
-Pinch callus

Vasc/Neuro: Usually non-contributory

Ortho: -Dorsomedial eminence
-Varus compensation
-Underlying met adductus
-PROM 1st MPJ
-Tracking vs. Track-bound 1st MPJ
-Pes plano valgus
-Equinus
-Hypermobile 1st ray
-LLD

Objective: Radiographic Evaluation

Plain Film Radiographs: -Increased soft tissue density
-In first met head: subchondral bone cysts, osteophytes, hypertrophy of medial eminence
-Overall metatarsal parabola
-1st MPJ joint space: ~2mm of clear space; Congruent vs. Deviated vs. Subluxed
Angular deformities:

-Met Adductus (<15 degrees)	-Meary's Angle
-Engle's Angle (<24 degrees)	-Seiberg's Angle
-IMA (<8 degrees)	-TDA
-HAA (<15 degrees)	-CIA
-HIA (<10 degrees)	-Cyma Line
-Metatarsal sesamoid position (1-7)	-Calcaneal-Cuboid Angle
-PASA (<8 degrees)	-Talar Head Uncovering
-DASA (<8 degrees)	-Talar Axis
-Met protrusion distance (<2mm)	-Kite's Angle

HAV Dissection and Capsule Procedures

-Anatomic Dissection
-1st incision is through epidermis and dermis
-Incision is planned along the dorsomedial aspect of the 1st MPJ, just medial to EHL and lateral to the medial dorsal cutaneous nerve.
-From midshaft of 1st metatarsal to just proximal to the hallux IPJ
-Subcutaneous tissue is dissected to deep fascia/capsular layer
-NV structures: Superficial venous network, medial dorsal cutaneous nerve
-Be wary of the anterior resident's nerve (Extensor capsularis)!

-Lateral Release
-Sequence of events:
-Release of adductor hallucis tendon from base of proximal phalanx and fibular sesamoid
-Release of fibular metatarsal-fibular sesamoid ligament and lateral capsule
-Tenotomy of the lateral head of the FHB between the fibular sesamoid and the proximal phalanx
-Optional excision of the fibular sesamoid

-Medial Capsulotomies
-Linear
-Washington Monument: Strongest medial capsulotomy allowing for both transverse and frontal plane correction
-Lenticular (Elliptical): Allows for transverse and frontal plane correction with removal of redundant capsule
-Inverted L: Transverse plane correction with removal of redundant capsule
-Medial T: Transverse plane correction with removal of redundant capsule
-Medial H: Transverse plane correction with removal of redundant capsule

AJM List: HAV Procedures and Indications

Distal Phalanx

1. Medial Nail Bed Rotation: Corrects soft tissue mal-alignment

Hallux IPJ

2. Amputation of the distal phalanx: Permanent correction of abnormal Hallux Interphalangeus Angle (HIA)
3. IPJ Fusion: Corrects abnormal HAI

Proximal Phalanx

4. Distal Akin: Corrects abnormal HAI with a medially-based wedge osteotomy at distal proximal phalanx
5. Central Akin: Corrects for long proximal phalanx seen with concurrent HL/HR
6. Oblique Akin: Corrects for distal articular set angle (DASA) midshaft proximal phalanx
7. Proximal Akin: Corrects for DASA of the proximal phalanx
8. Keller Arthroplasty: Corrects for abnormal Hallux Abductus Angle (HAA) and with concurrent HL/HR
9. Keller-Brandis Arthroplasty: Same as the Keller, but with removal of 2/3 of the proximal phalanx
10. Bonney-Kessel: Dorsiflexory osteotomy with concurrent HL/HR with modified forms correcting for abnormal DASA
11. Distal Hemi-Implant: Corrects for abnormal HAA or DASA with concurrent HL/HR
12. Regnauld: Allows for correction of DASA and abnormal proximal phalanx length in presence of HL/HR
13. Sagittal Z: Corrects for DASA and abnormal proximal phalanx length in presence of HL/HR

MPJ

14. Total Implant: Correction of HAA in presence of HL/HR
15. McKeever arthrodesis: Allows for permanent correction of DASA, PASA and HAA
16. McBride: Soft tissue reconstruction for correction of HAA
17. Modified McBride: Bone and soft tissue reconstruction for correction of HAA and medial eminence
18. Silver: Correction of medial eminence
19. Hiss: Modified McBride with Abductor hallucis advancement
20. External Fixation: Double Taylor frame for gradual soft tissue realignment
21. Hallux Amputation: Permanent correction of abnormal HAA

Distal 1st Met

22. Proximal Hemi-Implant: Correction of PASA and HAA with concurrent HL/HR
23. Mayo: First met head resection for correction of HAA with HL/HR
24. Stone: Mayo with sesamoid articulation left intact
25. Reverdin: Correction of PASA. Incomplete osteotomy.
26. Reverdin-Green: Correction of PASA with incomplete osteotomy and plantar shelf
27. Reverdin-Laird: Correction of PASA and IMA with complete osteotomy and plantar shelf
28. Reverdin-Todd: Correction of PASA, IMA and sagittal plane deformity (elevatus)
29. Youngswick: Correction of IMA and elevatus
30. Austin: Correction of IMA
31. Bicorrectional Austin: Correction of IMA and PASA
32. Tricorrectional Austin: Correction of IMA, PASA and elevatus
33. Mitchell: Rectangular osteotomy with lateral spicule to correct for IMA, elevatus and metatarsal length. Perpendicular to first met axis.
34. Roux: Wedged Mitchell to also correct for PASA
35. Miller: Mitchell with osteotomy oblique to first met axis for further correction of IM and length
36. Hohmann: Transverse through and through cut to correct for IMA and sagittal plane
37. Wilson: Oblique through and through osteotomy to correct for IMA and metatarsal length
38. Distal L: Similar to a Reverdin-Green without correction of PASA
39. Kalish: Austin with a long dorsal arm to allow for screw internal fixation
40. Mygind: Mexican hat procedure of distal first metatarsal for correction of IM and length
41. Off-set V/Vogler: Proximal Kalish
42. Peabody: Proximal Reverdin
43. Short-arm Scarf: Correction of IMA
44. Percutaneous DMO: Percutaneous Hohmann
45. DRATO (Derotational Abductory Transpositional Osteotomy): Can be used to correct frontal plane, IMA, sagittal plane and wedged for PASA
46. Distal Crescentic: Correction of IMA
47. Distal Crescentic with a shelf: Correction of IMA with greater stability

Central 1st Met

48. Scarf: Correction of IMA
49. Ludloff: Correction of IMA. Dorsal-proximal to distal-plantar cut.
50. Mau: Correction of IMA. Distal-dorsal to proximal-plantar cut.

Proximal 1st Met

51. Cresentic: Correction of IMA
52. Cresentic Shelf: Correction of IMA with greater stability
53. OBWO: Correction of IMA
54. Trethowan: OBWO using medial eminence for graft
55. CBWO (Loison-Balacescu): Closing base wedge proximal osteotomy. Corrects IMA.
56. Logroscino: CBWO with Reverdin. Corrects IMA and PASA.
57. Juvara: Oblique CBWO
58. Proximal Austin: Correction of IMA
59. Lambrinudi: Plantar CBWO to correct for sagittal plane

1st Met-Cunieform

60. Lapidus with internal fixation
61. Lapidus with external fixation
62. Westman: OBWO of the cuneiform to correct for transverse plane
63. Cotton: OBWO of the cuneiform to correct for sagittal plane
64. Cotton-Westman: OBWO of the cuneiform to correct for transverse and frontal plane

Misc.

65. 2nd digit amputation
66. EHL lengthening

AJM Sheet: HAV Complications

-**Recurrence**

-**Early** (<1 year)

-Usually due to wrong procedure choice, surgical error, or a post-operative complication.

-As little as 1% and as much as 14% rate reported (Kitaoka on 49 feet).

-**Late** (>1 year)

-Usually due to an unrecognized underlying deformity (such as met adductus, Ehlers-Danlos, equinus, 1st met hypermobility, etc.)

-Symptoms usually worse than initial presentation

-**Treatment**: Distal soft tissue procedures or a proximal osteotomy usually indicated

-**Hallux Varus**

-Defined as a purely transverse plane adduction

-Hallux Malleus: extension at MPJ with flexion at IPJ

-Etiology

-**Underlying causes**:
- -Long 1st metatarsal
- -Round 1st metatarsal head
- -1st MPJ hypermobility

-**Iatrogenic causes**:
- -Staking of the 1st metatarsal head
- -Overcorrection of the IM angle
- -Overzealous medial capsulorraphy
- -Fibular sesamoidectomy
- -Over extensive lateral release
- -Overcorrection of the PASA
- -Overzealous bandaging

-**Treatment**:
- -Soft tissue rebalancing (medial releases and lateral tightenings)
  - -EHB tendon transfer
- -Reverse distal osteotomies
- -Ludloff/Mau
- -Resection arthroplasty, implant, arthrodesis

-MalUnion/Delayed Union/Non-Union

-**Malunion**

-Consolidated osteotomy with an angular or rotational deformity

-Most common is sagittal plane abnormality ("**dorsal tilting**")

-Must be corrected with an osteotomy

-**AVN**

-Weber and Cech Classification of Non-Unions

-**Hypertrophic/Hypervascular** (represents ~90% of non-unions)

-These types of non-unions have adequate biology, but they usually require increased stabilization in order to heal.

-Elephant Foot
-Horse Hoof
-Oligotrophic

-**Atrophic/Avascular** (represents ~10% of non-unions)

-These types of non-unions have bad biology and require aggressive debridement, usually with some type of orthobiologic product.

-Torsion wedge
-Comminuted
-Defect
-Atrophic

AJM Sheet: HL/HR Work-up

Subjective
CC: Pt will generally complain of a "painful big toe."
HPI: -Nature: Aching, Dull, Throbbing
-Location: Dorsal 1st MTPJ and within the joint
-Course: Usually gradual and progressive. May follow an acute traumatic event.
-Aggravating Factors: Shoe gear, WB
-Alleviating Factors: Ice, NSAIDs, Rest
PMH: -Inflammatory Condition: RA, SLE, Gout
PSH: -Past 1st MTPJ surgery
Meds/Allergies/SH/FH: Non-contributory
ROS: Non-contributory

Objective: Physical Exam
Derm: -Hyperkeratotic lesions: Plantar hallux IPJ, Medial pinch callus hallux IPJ, Submet 2
-Erythema, Calor, Dorsal 1st MTPJ bursa
Vasc/Neuro: Non-contributory

Ortho:	-Decreased PROM 1st MTPJ	-Varus Deformity	**Gait**: -Early Heel-off
	-Dorsal eminence 1st MTPJ	-Plantar Contracture	-Apropulsive Gait
	-Dorsal eminence 1st Met-Cun	-Equinus	-Abductory Twist
	-Hypermobile 1st ray		

Objective: Imaging

Plain Film Radiographs:	-Osteophytes at 1st MTPJ	-Long 1st met
	-Irregular Joint Space Narrowing	-Long hallux proximal phalanx
	-Lateral view: dorsal flag sign, dorsal lipping	-Elevated 1st met
	-Loose bodies (joint mice)	-Osteophytes at hallux IPJ, 1st met-cun
	-Square-shaped 1st met head	

General HL/HR Information
-Definitions
-Hallux Limitus vs. Hallux Rigidus
-This is a progressive deformity, so ***what defines rigidus from limitus****?* **Bony ankylosis and sesamoid immobilization**.
-Functional HL is defined as a decreased PROM with the foot loading and in a neutral position, and normal PROM when the foot is unloaded. Dannanberg first defined functional HL.
-Flexor Stabilization of the hallux: Essentially a hammertoe of the hallux with extension at the MTPJ and plantarflexion at the IPJ.
-Axis of rotation of the 1st MTPJ: Normally found in the center of the metatarsal head allowing for a gliding motion of the hallux up and over the first metatarsal head. In a HL/HR deformity the axis of rotation moves **distally and plantarly** leading to dorsal jamming of the joint.
-Met Primus Elevatus: Dorsiflexed position of the 1st metatarsal.
-**Primary**: Structural. Distal segment is dorsiflexed compared to proximal segment.
-**Secondary**: Global. Due to some extrinsic variable. This can be measured by Meary's Angle on a lateral plain film radiograph or using the Seiberg technique comparing the 1st and 2nd metatarsal positions.
-*What stimulates osteophyte production in and around the joint?:* Loss of functional cartilage.

-1st MTPJ ROM
-Normal PROM of the 1st MTPJ is classically described as 65-75 degrees of dorsiflexion of the hallux referenced to the weight-bearing surface (same as 85-95 degrees of dorsiflexion referenced to the 1st met shaft). Plantarflexion is 30 degrees to the weight-bearing surface.
-Hetherington contradicts this somewhat by finding an average ROM of 31degrees of dorsiflexion during pain-free gait in asymptomatic patients.

-Compensation Patterns for Lack of Motion
-Distal: Hallux IPJ leading to OA and plantar hyperkeratotic lesions
-Lateral: Lesser metatarsalgia
-Proximal: 1st met-medial cuneiform joint increased motion and OA
-Gait patterns: Abductory twist with roll-off; early heel-off; apropulsive gait

-HL/HR Etiology
-Many people have reported potential causes of HL/HR including Root, Lapidus and Nilsonne:

-Acute Trauma	-Chronic degenerative trauma
-Pes planus with 1st met hypermobility	-Long first metatarsal
-Short first metatarsal with hallux gripping	-Long hallux proximal phalanx
-Iatrogenic	-Compensated varus deformity
-Neuromuscular imbalance	-Plantar contracture
-Spastic conditions	-Square first metatarsal head shape
-Met primus elevatus	

-No single characteristic has been shown to reliably lead to HL/HR except acute trauma
-Coughlin (FAI 2003) performed a retrospective analysis and seemed to demonstrate that there are no reliable underlying indicators for development of HL/HR.

-Classification Systems
-Numerous exist; usually in the mild, moderate, severe format:
-**Mild**: Mild pain; Normal PROM; Radiographic evidence of osteophytes
-**Moderate**: Increasing pain; Decreasing PROM; Osteophytes and irregular joint space narrowing on radiograph
-**Severe**: Increasing pain; Decreasing PROM; Osteophytes, irregular joint space narrowing, subchondral sclerosis on radiograph.
-**Rigidus**: Increasing pain; Absent PROM; Sesamoid immobility
-Examples of classifications include the Regnauld, Hanft and KLL.

AJM Sheet: HL/HR Treatment

-Conservative

-Do nothing
-Activity modification
-Orthotics: First ray cut-out, Morton's extension, rocker-bottom sole
-Meds: PO NSAIDs, Intra-articular corticosteroid injections

-Surgical

-Surgical options are always divided into joint-sparing and joint-destructive procedures, and further divided into whether the correction occurs at the proximal phalanx, at the MTPJ, or at the first metatarsal.

-Joint Sparing

-Proximal Phalanx
- -Bonney-Kessell
- -Regnauld
- -Vanore
- -Sagittal "Z"
- -Central Akin

-1st MTPJ
- -Cheilectomy

-1st Metatarsal
- -Youngswick
- -Watermann
- -Watermann-Green
- -Jacoby
- -Hohmann
- -Derner
- -Dorsal OBWO
- -Lambrinudi
- -Westman

-Joint Destructive

-Proximal Phalanx
- -Keller
- -Keller-Brandis
- -Distal Hemi-Implant

-1st MTPJ
- -Total Implant
- -McKeever arthrodesis
- -Valenti

-1st Metatarsal
- -Mayo
- -Stone
- -Lapidus

AJM Sheet: Flatfoot Work-up

-This is a lot of information to cover in 2 pages, so these sheets will focus on clinical and radiographic signs, as well as indications for specific surgeries. Also, will try and provide a good amount of additional readings.

Subjective

-Wide range of presenting ages and complaints.

-Always think about posterior tibialis tendon dysfunction when someone complains of "medial ankle pain."

Objective

-**Underlying Orthopedic Etiologies**:
- -Compensated forefoot varus
- -Forefoot valgus
- -Rearfoot valgus
- -Equinus
- -Compensated and uncompensated ab/adduction deformities
- -Muscle imbalances (PTTD)
- -Ligamentous laxity
- -Tarsal coalitions
- -Planal dominance
  - -Normal STJ axis: 42° from transverse/16° from sagittal
  - -Normal MTJ-O: 52° from transverse/57°from sagittal
  - -Normal MTJ-L: 15° from transverse/9° from sagittal

-**Clinical findings**:
- -"Too many toes" sign (forefoot abduction)
- -Evaluation for flexible versus rigid deformity
- -Single and double heel raise
- -Hubscher maneuver
- -RCSP
- -Subjective gait analysis

-**Radiographic evaluation**:

-**Lateral**:
- -Decreased calcaneal inclination angle
- -Increased talar declination angle
- -Decreased first metatarsal declination angle
- -Calcaneal-cuboid "break"
- -Anterior break in Cyma line
- -Meary's Angle
- -Midfoot "breaks" or "incongruity"

-**AP**:
- -Increased talo-calcaneal angle
- -Cuboid-abduction angle
- -Talar head coverage
- -Look for "skew foot" deformity
- -Talar-first metatarsal axis
- -Intermetatarsal angle
- -Forefoot adduction angle or Engle's Angle

-**Harris-Beath**: Evaluation of tarsal coalitions

-**Long-Leg Calcaneal Axial Views**: Evaluation of structural rearfoot deformities

Classifications

-**Johnson and Strom** *[Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. CORR. 1989; 239: 196-206.]*

-Later modified by Myerson who added Stage IV (he does that a lot):

-[Myerson MS. Adult acquired flatfoot deformity: treatment of dysfunction of the posterior tibial tendon. JBJS-Am. 1996; 78: 780-92.]

-[Bluman EM, et al. Posterior tibial tendon rupture: a refined classification system. Foot Ankle Clin. 2007 Jun; 12(2): 233-49.]

-Stage I: Tenosynovitis with mild tendon degeneration; flexible rearfoot; Mild weakness of single heel raise and negative "too many toes" sign

-Stage II: Elongated tendon with tendon degeneration; flexible rearfoot; Marked weakness of single heel raise and positive "too many toes" sign

-Stage III: Elongated and ruptured tendon; Rigid valgus rearfoot; Marked weakness of single heel raise and positive "too many toes" sign

-Stage IV: Same as Stage III with a rigid ankle valgus

-**Funk**: Classification based on gross intra-operative appearance

-[Funk DA, et al. Acquired adult flatfoot secondary to posterior tibial tendon pathology. JBJS-Am. 1986; 68: 95-102.]

- -Type I: Tendon Avulsions
- -Type II: Complete midsubstance rupture
- -Type III: In-continuity tears
- -Type IV: Tenosynovitis

-**Jahss or Janis Classifications**: There are several MRI classifications generally along the lines of:

-[Conti S, Michelson J, Jahss M. Clinical significance of MRI in preoperative planning for reconstruction of posterior tibial tendon ruptures. Foot Ankle. 1192; 13(4): 208-214.]

-[Janis LR, et al. Posterior tibial tendon rupture: classification, modified surgical repair, and retrospective study. JFAS. 1993; 31(1): 2-13.]

-Type I: Tenosynovitis, increased tendon width, mild longitudinal splits

-Type II: Long longitudinal splits with attenuated tendon

-Type III: Complete rupture

Additional Reading:

-[Mendicino RW, et al. A systemic approach to evaluation of the rearfoot, ankle and leg in reconstructive surgery. JAPMA. 2005; 95: 2-12.]

-[Lamm BM, Paley D. Deformity correction planning for hindfoot, ankle and lower limb. Clin Podiatr Med Surg. 2004 Jul; 21(3): 305-26.]

-[Greisberg J, Hansen, Sangeorzan. Deformity and degeneration in the hindfoot and midfoot joints of the adult acquired flatfoot. Foot Ankle Int. 2003 Jul; 24(7): 530-4.]

-[Weinraub GM, Saraiya MJ. Adult flatfoot/posterior tibial tendon dysfunction: classification and treatment. Clin Podiatr Med Surg. 2002 Jul; 19(3): 345-70.]

AJM Sheet: Flatfoot Treatment

-Again, this is a lot of information to cover, so we'll just focus on organizing general procedures and indications, but supplement it with some additional reading.

-Conservative Treatments

-Not going to be discussed here, but try reading:

-[Elftman NW. Nonsurgical treatment of adult acquired flatfoot deformity. Foot Ankle Clin. 2002 Mar; 7(1): 95-106.]

-[Marzano R. Functional bracing of the adult acquired flatfoot. Clin Podiatr Med Surg. 2007 Oct; 24(4): 645-56.]

-Johnson and Strom/Myerson Classification:

-In addition to describing the deformity, this classification system (discussed on a previous sheet) also makes general treatment recommendations:

-Stage I: Conservative treatment; Tenosynovectomy; Tendon Debridement

-Stage II: Tendon transfer; Rearfoot arthrodesis

-Stage III: Isolated rearfoot arthrodesis; Triple arthrodesis

-Stage IV: TTC arthrodesis; Pantalar arthrodesis

-General Surgical Procedures/Indications:

-Keep in mind that it is very common to do combinations of these procedures.

Soft Tissue Procedures:

-FDL Tendon Transfer: FDL is sectioned as distal as possible (consider anastomosis of stump to FHL) and either attach proximal FDL to the PT, within the PT sheath or into the navicular under tension.

-Cobb: Split TA tendon, transfer to the PT or into the navicular

-Young's Tenosuspension: TA rerouted through navicular

-Anastomosis of PB and PL: Removes PB as deforming force

-STJ implant (arthroeresis)

-TAL **-Gastroc recession**

Rearfoot Osseous Procedures:

-Evans Osteotomy (1975): opening wedge calcaneal osteotomy

-Silver (1967) is a more proximal (and less common) Evans-type opening wedge

-[Sangeorzan BJ, et al. Effect of calcaneal lengthening on relationships among the hindfoot, midfoot and forefoot. Foot Ankle. 1993; 14(3): 136-41.]

-[Raines RA, et al. Evans osteotomy in the adult foot: an anatomic study of structures at risk. Foot Ankle Int. 1998 Nov; 19(11): 743-7.]

-[Weinraub GM. The Evans osteotomy: technique and fixation with cortical bone pin. JFAS. 2001; 40(1): 54-7.]

-[DeYoe BE, Wood J. The Evans calcaneal osteotomy. Clin Podiatr Med Surg. 2005 Apr; 22(2): 265-76.]

-Medial Calcaneal Slide (Koutsogiannis - 1971): medial translation of posterior calcaneus

-[Weinfeld SB. Medial slide calcaneal osteotomy. Technique, patient selection and results. Foot Ankle Clin. 2001 Mar; 6(1): 89-94.]

-[Catanzariti AR, et al. Posterior calcaneal displacement osteotomy for adult acquired flatfoot. JFAS. 2000; 39(1): 2-14.]

-Double Calcaneal Osteotomy: Evans with a medial calcaneal slide

-[Catanzariti AR, et al. Double calcaneal osteotomy: realignment considerations in eight patients. JAPMA. 2005; 95(1): 53-9.]

-[Nyska M, et al. The contribution of the medial calcaneal osteotomy to the correction of flatfoot deformities. Foot Ankle Int. 2001 Apr; 22(4): 278-82.]

-STJ arthrodesis **-Triple arthrodesis**

-Tibiotalocalcaneal arthrodesis **-Pantalar arthrodesis**

-Closing wedge of the medial calcaneus: Essentially the opposite of an Evans but with more NV structures

-Dwyer (1960)

-Slakovich: opening wedge behind the sus tali

-Baker-Hill: opening wedge osteotomy with graft horizontally under the posterior calcaneal facet

Midfoot/Forefoot Osseous Procedures:

-Cotton: opening wedge osteotomy with graft in medial cuneiform

-Kidner: advancement and reattachment of PT tendon (+/- resection of portion of navicular)

-TN arthrodesis: called a Lowman when wedged and combined with TAL

-Medial column arthrodeses

-Miller: NC and 1st met-cun arthrodesis

-Lapidus (1931/1960): 1st met-cun arthrodesis

-Hoke: arthrodesis of navicular with 1st/2nd cuneiforms

-Any other combinations

-[Greisberg J, et al. Isolated medial column stabilization improves alignment in adult-acquired flatfoot. CORR. 2005 Jun; 435: 197-202.]

-Additional Reading:

-[Hix J, et al. Calcaneal osteotomies for the treatment of adult-acquired flatfoot. Clin Podiatr Med Surg. 2007 Oct; 24(4): 699-719.]

-[Mosier-LaClair S, et al. Operative treatment of the difficult stage 2 adult acquired flatfoot deformity. Foot Ankle Clin. 2001 Mar; 6(1): 95-119.]

-[Roye DP, Raimondo RA. Surgical treatment of the child's and adolescent's flexible flatfoot. Clin Podiatr Med Surg. 2000 Jul; 17(3): 515-30.]

-[Toolan BC, Sangeorzan, Hansen. Complex reconstruction for the treatment of dorsolateral peritalar subluxation of the foot. JBJS-Am. 1999 Nov; 81(11): 1545-60.]

-[Weinraub GM, Heilala MA. Adult flatfoot/posterior tibial tendon dysfunction: outcomes analysis of surgical treatment utilizing an algorithmic approach. J Foot Ankle Surg. 2001 Jan-Feb; 40(1): 54-7.]

AJM Sheet: Cavus Foot Work-up

-The cavus foot work-up is one of the most feared in the residency interview process because of its complex nature. The most important technique during this work-up is to use a standardized system to identify several specific variables which will let you best identify the deformity and decide on a treatment course:

-Underlying Etiology of the Deformity (Spastic vs. Progressive vs. Stable)
-Forefoot vs. Rearfoot driven deformity (Anterior Cavus vs. Posterior Cavus)
-Plane of the Deformity (Sagittal vs. Frontal vs. Transverse vs. Combination)
-Rigid vs. Flexible

-Underlying Etiology of the Deformity (Spastic vs. Progressive vs. Stable)

-Obtained through a good PMH and physical exam

-Brewerton of the Royal Hospital in London identified 75% of 77 patients seen at his pes cavus clinic to have an underlying neuromuscular disorder.

-Common Congenital Conditions leading to neuromuscular dysfunction:

-Spina bifida	-Charcot-Marie-Tooth
-Myelodysplasia	-Friedreich's Ataxia
-Cerebral Palsy	-Roussy-Levy syndrome
-Muscular Dystrophy	-Dejerine-Sottas
-Poliomyelitis	-Etc, etc, etc.

-Also consider MMT, clonus, deep tendon reflexes, EMG studies and nerve conduction studies during your physical exam.

-Forefoot vs. Rearfoot Driven Deformity

-**Anterior Cavus**: plantar declination of the forefoot in relation to the rearfoot. Subdivided based on apex of deformity:
-**Metatarsus Cavus**: Apex at Lisfranc's joint. Generally more rigid.
-**Lesser Tarsus Cavus**: Apex in the lesser tarsus area
-**Forefoot Cavus**: Apex at Chopart's joint
-**Combined Cavus**: Combination of any of the above

-The apex of the deformity can be found several different ways:
-Intersection of **Meary's lines** (longitudinal axes of talus and first met)
-Intersection of **Hibb's Angle** (longitudinal axes of calcaneus and first met)
-Dorsal boney prominences
-Joint space gapping

-**Posterior Cavus**: Dorsiflexion of the rearfoot in relation to the forefoot
-Generally defined as an increased calcaneal inclination angle (>30 degrees) and a varus positioning.
-Usually the result of an anterior cavus; rarely presents as separate entity.

-An anterior cavus and a posterior cavus can be defined based on radiographic evidence and a physical exam measure called the **Coleman Block Test.** In this test the forefoot, or the medial and lateral portions of the forefoot, are suspended off of a block. If the calcaneus returns from a varus to a normal position, the deformity is forefoot driven. A deformity is rearfoot driven only if the varus positioning of the calcaneus remains after all forefoot elements are removed.

-Biomechanical compensation for a sagittal plane cavus deformity:
-**Digital retraction**: HT deformity where EDL gains mechanical advantage and uses a passive pull.
-**MPJ Retrograde buckling**: As per above
-**Lesser Tarsal Sagittal Plane Flexibility**: The lesser tarsus "absorbs" some of the dorsiflexion. They can be clearly seen when comparing NWB and WB lateral views of an anterior cavus foot.
-**Pseudoequinus**: Occurs when the ankle joint must dorsiflex because the lesser tarsus cannot "absorb" all of the dorsiflexion. Limits the amount of "free" dorsiflexion available during gait.

-Plane of the Deformity:

-**Sagittal Plane**:	-Anterior Cavus (Global, Medial Column, or Lateral Column)
	-Posterior Cavus
	-Muscular Cavus (Gastroc Equinus, Gastroc-Soleus Equinus)
	-Osseous Equinus (Tibiotalar exostosis)
-**Transverse Plane**:	-Met adductus (measured via met adductus angle or Engle's angle)
	-Met abductus (measured via met adductus angle)
-**Frontal Plane**:	-Forefoot Varus
	-Forefoot Valgus
	-Rearfoot Varus
	-Rearfoot Valgus

-Rigid vs. Flexible Deformity

-**Flexible deformities** can be manipulated out during the physical exam and are obvious comparing NWB and WB lateral radiographs.
-**Rigid deformities** show no compensation with manipulation of weight-bearing.
-Defining each of these variables during your work-up will give you a clear enough understanding of the deformity to recommend a treatment option.

AJM Sheet: Cavus Foot Treatment

-Basic principles of treatment based on definition of deformity:
- -Underlying Etiology:
  - -Progressive/Spastic conditions are generally treated with osteotomies and arthrodeses.
  - -Stable conditions are generally treated with soft tissue procedures and osteotomies.
- -Forefoot vs. Rearfoot Driven:
  - -Forefoot driven conditions are treated with manipulation of the bones and soft tissue of the forefoot.
  - -Rearfoot driven conditions require rearfoot osteotomies and arthrodeses.
- -Plane of the Deformity:
  - -Procedures are chosen by which plane you want correction in.
- -Rigid vs. Flexible:
  - -Rigid deformities are generally treated with osteotomies and arthrodeses.
  - -Flexible deformities can usually be managed with soft tissue procedures and tendon transfers.

-**Soft Tissue Releases**: Reduces contracture of the plantar fascia seen with long standing disease.
- -**Subcutaneous Fasciotomy**: Cuts the plantar fascia at its insertion.
- -**Steindler Stripping**: Removes all soft tissue from the plantar surface of the calcaneus.
- -**Plantar Medial Release**: Releases plantar musculature and ligaments from the plantar-medial foot.

-**Tendon Transfers**: Used to treat flexible conditions based upon plane of the deformity.
- -**Jones Suspension**: Transfer of EHL through the first metatarsal head.
- -**Heyman Procedure**: Transfer of EHL and EDL tendons through each of the respective metatarsal heads.
- -**Hibbs Procedure**: Transfer of EDL into lateral cuneiform; EHL into first metatarsal; EDB into sectioned tendons.
- -**STATT**: Tibialis anterior is split and sutured into peroneus tertius.
- -**Peroneus Longus Transfer**: Peroneus longus is split and anastomosed to the TA and peroneus tertius tendons.
- -**Peroneal Anastomosis**: Increases the eversion power of the foot.
- -**PL/PT transfer to calcaneus**: Tendons are attached into the calcaneus via bone anchors to aid weak Achilles tendon.

-**Osseous Procedures**: Reduction of rigid deformities. Can be used to correct multi-planar deformities.
- -**Cole Procedure**: Dorsiflexory wedge is removed from Chopart's joint.
- -**Japas Procedure**: "V" shaped osteotomy through the midfoot (apex proximal) to dorsiflex forefoot.
- -**Jahss Procedure**: Essentially a Cole procedure performed at Lisfranc's joint.
- -**Dorsiflexory Metatarsal Osteotomies**
- -**Dwyer Osteotomy**: Closing wedge osteotomy out of lateral calcaneus to reduce rigid rearfoot varus.
- -**Dorsiflexory Calcaneal Osteotomy**: Must be used with caution

-**Arthrodesis Procedures**: Used as last resort to correct rigid deformities in the face of progressive disease.
- -**Hoke**: STJ and TNJ arthrodesis
- -**Ryerson** (1923): Triple arthrodesis

-Additional Reading:
-[Younger AS, Hansen. Adult cavovarus foot. J Am Acad Orthop Surg. 2005 Sep; 13(5): 302-25.]
-[Statler TK, Tullis BL. Pes Cavus. JAPMA. 2005; 95: 34-41.]

AJM Sheet: Ankle Equinus

-This sheet is not a work-up because equinus rarely presents as a chief concern, but rather as a concomitant and underlying deformity. It may be seen and deemed correctable in the following deformities:

-Charcot arthropathy
-Pes plano valgus
-HAV
-Medial column hypermobility
-Digital deformities
-Met primus elevatus
-Plantar fasciitis
-Diabetic foot ulcerations
-Etc.

-**History**

-First TAL: Paris on Achilles in the "Iliad"
-First medically documented procedure: Stromeyer on Dr. Charles Little. Dr. Little was a prominent physician suffering from cerebral palsy (CP) who then became an advocate for surgical correction of equinus.

-**Anatomy**

-Review the origins/insertions/course/action/NV supply of the gastroc and soleus.
-Review the concept of the "twisting" fibers within the Achilles tendon.
-[White JW. Torsion of the Achilles tendon: its surgical significance. Arch Surg 1943; 46: 784-7.]
-[van Gils CC, Steed RH, Page JC. Torsion of the human Achilles tedno. JFAS 1996.]

-**Definitions**

-Muscular Equinus
-Spastic vs. Non-Spastic
-Gastroc Equinus
-Gastroc-Soleal Equinus
-Osseous Equinus
-Tibio-talar exostosis
-Pseudoequinus
-Combination equinus

-**Biomechanic Compensation for Equinus** (proximal to distal)

-Lumbar lordosis
-Hip flexion
-Genu recurvatum
-No compensation (toe walking)
-STJ pronation
-MTJ pronation
-Forefoot abduction
-Medial column hypermobility

-**Testing for Equinus**

-Silfverskiold test
-Stress dorsiflexion plain film radiographs
-WB wall test

Treatment

-Conservative Treatment

-Stretching
-Physical Therapy
-Heel Lifts (?)
-Neuromuscular blockage injections (Botox)
-Casting

-Surgical Correction

-Gastroc Equinus

-Neurectomy of motor branches of tibial nerve
-Proximal recession (Silfverskiold procedure)
-Release of muscular heads of gastroc +/- reattachment to tibia +/- neurectomy
-Distal aponeurotic recession
-Vulpius&Stoffel (1913): Inverted "V" shaped incision without suture reapproximation
-Strayer (1950): Transverse incision with proximal dissection and suturing (absorbable)
-Baker (1956): Tongue and groove with suturing (two incisions distal)
-Fulp&McGlamry: Inverted tongue and groove with suturing (two incisions proximal)
-Endoscopic recession

-Gastroc-Soleal Equinus

-Sagittal plane "Z" lengthening: equal medial and lateral portions
-Frontal plane "Z" lengthening: equal anterior and posterior portions
-Hoke Triple Hemisection (1931): 2 medial cuts/1 lateral cut
-White slide technique

-Percutaneous

-Similar to the Hoke procedure
-1cm → 3cm → 3 cm

Additional Reading:

-[Pinney SJ, Hansen, Sangeorzan. The effect on ankle dorsiflexion of gastrocnemius recession. Foot Ankle Int. 2002 Jan; 23(1): 26-9.]
-[Pinney SJ, Hansen, Sangeorzan. Surgical anatomy of the gastrocnemius recession. Foot Ankle Int. 2004 Apr; 25(4): 247-50.]
-[Lamm BM, Paley, Herzenberg. Gastrocnemius soleus recession: a simpler, more limited approach. JAPMA. 2005; 95: 18-25.]
-[Graham HK, Fixsen JA. Lengthening of the calcaneal tendon in spastic hemiplegia by the White slide technique. A long term review. JBJB-Br. 1988 May; 70(3): 472-75.]

AJM Sheet: Page 100

One of the most frequent questions asked by students and externs is *"Can you give me some good articles to read?"*

-I tried to do this by incorporating current and historical articles throughout this edition. All referenced articles (as well as other suggested readings) can be found on the **Inova Pubmed** page we set up when I was a resident:

-www.pubmed.com
-***My NCBI*** link on the left

-**User Name**: INOVA
-**Password**: resident

-Although this was specifically designed for the use of externs and residents at the Inova program, the collection of articles on this page can be used by anyone. Additionally, anyone is welcome and encouraged to update these collections with articles they feel are valuable.

-Temple students have online access to just about any article by logging on through the Temple University Health Science homepage: **http://library.temple.edu/hsl**

-From this webpage, utilize either the "PubMed" or "Journal Finder" resource on the left-hand side of the page. It's the same PubMed that you are used to, it simply automatically links you into Temple's electronic database. I would imagine that most other schools have something similar.

Other Resources:
-PRISM App: https://itunes.apple.com/us/app/podiatry-prism/id1089332577?mt=8
-TUSPM Podcast Network: https://soundcloud.com/user-79150427
-TUH Podiatric Surgical Residency Program: http://www.tuhpod.weebly.com/

This PRISM was not designed to help you pass the boards or even to make you a better physician (although…with the new Part II format, it may help out with boards after all); it simply hopes to make you better prepared and more efficient as you approach clerkships and the residency interview. Use, change, and pass this guide along as you see fit, keeping in mind the general goal of education of the next generation. Good luck, and please don't hesitate to contact me if there is any way that I can be of service to you: AJMeyr@gmail.com

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An American #10 holding up "Big Ears" for Chelsea…..never imagined I would see that!