

Day 2, Part 1  
5/19/09

## INFECTIOUS DISEASE

... Gardener, and he gets a nodule which is painless, a painless nodule. The gardener on the exam, it's usually sporotrichosis, unless proved otherwise. It can become like this, and the secretions which show you the septate hyphae which are branching at a right angle. The drug of choice would be itraconazole. Which of the following is not sporotrichosis? This one.

[Laughter] Don't they look alike? [Laughter]

No. 2 is a landscaper, so No. 2 is also sporotrichosis. It begins with a painless papule or nodule followed by similar nodules proximally. They discharge pus. A culture will show the *Sporothrix schenckii*, which is a septate hyphae branching at a right angle, so you have now three questions on Lyme disease.

### [Three questions, one minute]

If you get erythema chronicum migrans then the treatment is with doxycycline, and the reason is that serological testing is not recommended for patients with erythema migrans. Patient was treated because only 30% will have a positive serology after one month. If somebody is coming from, say, New York, New Jersey, Massachusetts area with erythema migrans, you just treat that. Serology does not distinguish between active and inactive infection, and therefore it is most useful in extracutaneous Lyme disease. If serology is positive, you have to confirm with Western blot, and PCR is done for Lyme arthritis. So, those are the facts of Lyme disease on which the questions are based on the exam. A funny thing happened last week only. I got a patient in my office, and he was working outside taking photographs in New Jersey, and he came—he called me that should I go to Emergency Room because I've got a tick in my wound on the right shoulder. He knew all about the tick, so he came to the office. We pulled the tick out and he took a picture of it. He took a photograph of it. [Laughter] I'm thinking I should show it to you, and he e-mailed the picture only three or four days ago. I have it right here. Look at that. I took the tick out of the wound, and he put it out, he took a photograph and e-mailed it to me. This is last week. It's a very good picture. [Laughter] So I just give him doxycycline, although there was no rash, but answer should have been reassurance, [Laughter] but I said New Jersey area, tomorrow I'm coming here, and he could get rash now. You know, I didn't want to take a chance, gave him doxy, because this is the area of Lyme disease all over, this all over area, including Pennsylvania, Wisconsin area, California. So coming from this area and you suspect or you saw the tick, I saw the tick, so might as well treat it.

For Question No. 2, coming from that area, it becomes a question. So Question No. 2 is coming from this area. The tick remains in contact for two hours only, so that's another thing. There's no need to analyze. Treatment is doxy. Question No. 2, the answer is reassurance because it was only there for two hours. It should be there for—attached for at least 24 hours.

Well, you don't write everything, you know all these things. You're writing a textbook of medicine. [Laughter] The only thing you need to know is doxycycline, and that you also know, anyhow, so the rest of it is just for information. It's very strange. People who come to the course, they will write everything down, and then they go home and they never look at it. [Laughter] I've gone to courses in my life. Never came back ever to look at it again, never, but there, sitting down, writing everything.

So the Lyme disease has Stages I, II, and III. Just to give you an idea, that's all, so Question No. 3 is Stage II disease, so when you are finished writing the book, go to three

questions in one minute. Those are questions on Q fever. Can you believe they ask Q fever in your exam. This is the tick. It was a smaller one

than what I showed you, that's baby tick there. [Laughter] This a Bell's palsy, Question No. 3. Looks like he's smiling. [Laughter] One time it was from Vermont in the examination, musculoskeletal symptoms. Musculoskeletal, then you have gone further from the skin, so that's why you will give ceftriaxone.

Okay, Q fever.

**[Three questions in one minute]**

All of these can be transmitted. A, B, C, D, can be transmitted in by aerosol, however Q fever is the most common. Remember in these kinds of questions when you go to the exam, they want the best answer. All the answers could be correct, but what is the best answer? The best answer here is Q fever, although all of them are correct answers, Q fever and *Coxiella burnetii*, which causes pneumonia via aerosol transmission or hepatitis via milk. *Rickettsia typhi*, we call this typhus via rats and fleas. This is in your notes.

The answer to Question No. 2 is infected cattle, sheep, and goats. You just have to know that.

And No. 3, the answer is Q fever, so again Q fever, aerosolized transmission is the most common, *Coxiella burnetii*, which is a gram negative organism via infected cattle, sheep, and goats.

There is a very interesting question on the exam where the patient has been burying—he had nothing else to do, so he was burying a deer. [Laughter] So after that he got a necrotic ulcer on his finger. That's the next question, so when you see on the exam somebody has buried a deer, the answer is tularemia. After you bury the deer, you get this kind of lesion on your body. It becomes an ulcer or glandular tularemia later on. Tularemia is associated with hunters and occurs in Clinton country. [Laughter] President Clinton comes from Arkansas, so his country, the people coming from there in the examination, somebody comes from Arkansas in the examination, think of tularemia. They get purulent conjunctivitis, and they get it through, in the exam question, either squirrels or deer.

And the Board in the examination will try to ask you the differences between tularemia and leptospirosis, so on the next page I give you the table. Tularemia is with squirrels and deer, leptospirosis is with rats. On this side is hunter, on that side is veterinarians. This side Arkansas. You see the differences, and now you do four questions in two minutes.

**[Four questions in two minutes]**

So Question No. 1 is leptospirosis. No. 2 is classical rabbit, Arkansas, purulent conjunctivitis, which is tularemia. No. 3 is—No. 3 is also on the exam. It is blastomycosis. If you see an ulcerative nose lesion which is coming from Arkansas with ulcerative nose lesion, that is on the exam, blastomycosis. This is a picture of leptospirosis. That was for Question number—famous question. Again, leptospirosis is [inaudible], leukocytosis and thrombocytopenia. They get conjunctival suffusion compared with—what was that—purulent conjunctivitis, in the previous one. Here you get conjunctival suffusion.

And with brucellosis you get any human transmission via ingestion of untreated milk or milk products or imported cheese. This is going to be Question No. 4. The answer there is D, tularemia again. We're trying to explain to you the rest of them. Brucellosis, animal to human, leptospirosis is contaminated rat urine, West Nile virus via mosquito bite, tularemia, and Q fever.

Okay, you're going to Baltimore now from here. [Laughter]

**A young female from Maryland presents with fever, faint macular rash on the ankles, palms, and soles, and excruciating headache. The rash fades on pressure but becomes prominent when the temperature is elevated. What is the diagnosis and drug of choice?**

Rocky Mountain occurs in these areas of the United States. In the Board exam, it is Baltimore but can be North Carolina, Virginia, etc. This is a kind of rash which fades away on pressure, compared to syphilis, which does not. This becomes hemorrhagic, and rash is present in all the cases of Rocky Mountain, and it is a vascular rash. It's a vasculitis. That's why they're hemorrhagic. Always on the exam, so usually patient goes to wooded area and comes back with this, with sudden headaches. Sudden headache is a thing with Rocky Mountain. This is the rash of syphilis, which is fixed. This is the rash of Rocky Mountain. Okay, six questions in three minutes. The drug of choice is tetracycline or doxycycline for Rocky Mountain.

**[Six questions in three minutes]**

This strongyloides is very common in the exam questions, and they can come in from outside the country and they can get it. Strongyloides normally causes diarrhea, eosinophilia. It can cause asthma and pneumonia, iron deficiency anemia, pancreatitis, you name it, and phlebitis. They get everything, so they will put strongyloidosis in some form or other in the exam. It can live in the body for decades, so you watch out for strongyloides. It is always there.

This, on the other hand, is—by the way, No. 2 is also strongyloides. Same, same. Iron deficiency, eosinophilia, and all that.

No. 3 is a hog farmer, this one, from Arkansas, CPK is high. That's classical description of trichinosis. With trichinosis you get subconjunctival hemorrhage, and the *Trichinella* antibody is positive with rising titer, so again, just to recapitulate, leptospirosis, you get suffusion, tularemia, conjunctivitis, trichinosis, hemorrhage, but those kinds of words are written in the question and you can tell from that. Again, the examination has become very clinical.

No. 4 is the treatment of malaria, and the answer is chloroquine phosphate plus 5-primaquine, and you will know why if you read the table underneath. It gives the treatment as well as prophylaxis of malaria. By the way, this is a muscle biopsy of trichinosis, muscle biopsy of trichinosis, eosinophilia, muscle pain, high CPK. This is the malaria parasite, ring form, and this is the banana shaped gametocyte of falciparum, falciparum, and there's the other ones, ring form, so you have to read the table to get some idea about what to treat with.

No. 5 is a girl from the mountains who had tick exposure. *Ehrlichia*. *Ehrlichia* looks like this. There are morulae in the peripheral blood smear, and these morulae are in vacuole of a monocyte. A monocyte vacuole and there are morulae inside there. It's a classical picture shown in the exam for *Ehrlichia*. This is babesiosis. Babesiosis is tetrad. Tetrad means four parasites like that. Tetrad of babesiosis.

And Question No. 6 is ehrlichiosis, right? It is tick-borne, ehrlichiosis. There are other tick-borne diseases. I wrote them in the notes.

Next is pasteurella, Question No. 1 on Page 23.

**A 41-year-old patient is bitten by a cat eight hours ago and presents with swollen hand. What is the most possible organism involved?**

*Pasteurella multocida*, always, always on the exam. It can give localized abscess, post-auricular painful lymph nodes, lymph nodes painful, and this is going with Question No. 2. Can you read that?

**A patient comes to you after sustaining a cat scratch. There were two wounds on the left upper arm and left side of the neck with post-auricular painful lymphadenopathy. You send secretions from the wounds for culture and sensitivity. What antibiotics would you now give?**

Now there are two bacteria involved in this because of the scratch, either *Pasteurella multocida* or *Bartonella henselae*. *Pasteurella multocida* is a gram negative coccobacillus, and the treatment is fluoroquinolones. *Bartonella henselae* is a nodular vascular lesion which also responds to antibiotics, usually ciprofloxacin, again a quinolone. In other words, if you have a cat scratch and you have an infection of some kind, a quinolone will be the right choice. It will cover both. There are other antibiotics, don't get me wrong, but I'm just giving you one which is usually on the exam. This *Bartonella*, which is bacillary angiomatosis, can be confused with Kaposi's sarcoma, so they can look alike, so this patient with AIDS had both. This is Kaposi and this is bacillary angiomatosis, so they look alike, so that's why a biopsy has to be done when you see bacillary angiomatosis because it could be Kaposi's sarcoma. In this case the biopsy showed he had both.

The next question is always on the exam, a clinched fist injury, No. 3.

[Inaudible].

No, [inaudible]. Only the *Pasteurella*. *Bartonella* gives vascular nodules, but of course you could get lymphadenopathy, but I don't know about postauricular. [Inaudible].

**So Question 2 [inaudible]?**

Well, [inaudible] the differential.

**Question 3, a 31-year-old man got involved in a fist fight at the bar and punched other people on the mouth. He comes to you with four wounds on the right hand. Which of the following antibiotics is the best one to use?**

The answer is Augmentin. It's always on the exam now. The bacteria is written on the slide and so is the treatment. Now you have five questions in two minutes.

**[Five questions in two minutes]**

So Question No. 1 is osteomyelitis. Therefore the answer is?

[Inaudible].

Was it B or A?

[Inaudible].

Oh, I'm sorry. I'm thinking the next step is MRI of the lumbosacral spine. What do you think?

[Inaudible].

How many people will take D? Yes, I agree that this is a mistake. It's a mistake. The answer is D like in 'doctor.' It's classical osteomyelitis, and the next step—again, always read the last line of the question. The next step is MRI of the spine.

No. 2, when will you use vancomycin? Both. And No. 3 is *Staphylococcus*, and No. 4, clear for surgery, no treatment needed. There are no urinary symptoms, so anytime the urine is just—let him enjoy life. [Laughter] No symptoms, don't bother. If it doesn't bother you, why would you bother them? They will be happy. And No. 5 is immediate catheter removal. That's candidemia. That's the problem.

So the next page gives you immediate catheter removal should be considered in what conditions. They are written for you and the points to remember about catheter infection. You should read those.

We're going to go to new questions. Go ahead.

**Question No. 1, a 21-year-old male patient, a recent visitor from Africa, presents with very painful ulcer on the penis. A culture reveals gram negative bacilli in rows, boxcar appearance. What is the treatment?**

See, I showed you that slide. Really I'm surprised that they ask this question on chancroid. This is chancroid. They have never asked chancroid before for internal medicine. It was always subspecialty question. Now for the first time they put this last year. I don't know whether it was experimental or you're going to get it. If you're going to get it, the answer is A, azithromycin 1gm, PO, single dose is the treatment. This is chancroid due to *Haemophilus ducreyi*, painful ulcers.

No. 2.

**A 31-year-old patient with secondary syphilis is treated with a dose of benzathine penicillin G. What is your next step?**

The next step, that's wrong. What is your next step? A lot of mistakes here. I would check RPR in 12 weeks. A single dose of benzathine penicillin provides low but persistent serum levels of penicillin and is the standard therapy for primary, secondary, or early latent syphilis. All patients should be reexamined clinically and serologically at three, six, and 12 months after treatment, three, six, and 12 months. A fourfold reduction in titer of the nontreponemal antibody test, that is if it is 1:16, now it should be 1:4, is considered evidence of appropriate response. If there is a poor response, you have to treat again and also treat the partners, so I'm taking A for this question on the exam.

No. 3.

**A female IV abuser presents with cough, fever, and neck pain. She is started on azithromycin but without any improvement in three days. She now complains of numbness and tingling of the left upper extremity. What is your next step?**

MRI of the spine to rule out abscess, MRI of the spine to rule out abscess.

No. 4.

**A young female presents from day care center with a sore throat, cervical lymphadenopathy and hepatosplenomegaly. What is the possible diagnosis?**

[Inaudible].

I'm thinking mononucleosis.

**A male patient just returned from Africa and moved to Connecticut. He took mefloquine only for two weeks and then developed a skin rash with central clearing. Diagnosis?**

**Lyme disease. [in unison]**

Lyme disease. He's in Connecticut. See, this is how they try to confuse you.

No. 6.

**A female patient from Connecticut removed a tick attached to her skin for less than a minute. What is your advice?**

**Reassurance.**

Call you if a rash develops.

No. 7.

**A 23-year-old female is working in a day care center. She has a past history of GERD. She comes with a cough, fever, sore throat for two weeks. Fever and sore throat resolve, but episodic cough persists to the point of vomiting. A kid in day care was diagnosed with pertussis. What is the treatment of your patient?**

**[Inaudible].**

A macrolide.

**Question 8, a patient came back from Nicaragua a month ago and now presents with severe myalgia, abdominal pain, thrombocytopenia, and liver dysfunctions. Diagnosis?**

**[Inaudible].**

I don't know. [Laughter] Myalgia, abdominal pain, thrombocytopenia. Is it Dengue?

**Yes. [in unison]**

Yeah, thrombocytopenia is Dengue, right.

No. 9. C is missing there, I know..

**A butcher from Mexico presents with subconjunctival hemorrhage, periorbital edema, and severe muscle pains. What is the causative organism?**

**[inaudible].**

What is it?

**[Inaudible].**

Human infections are acquired by eating meat containing cysts of *Trichinella*. The most frequent cause of *Trichinella* infection is *Trichinella spiralis*, acquired by consumption of inadequately cooked food from domestic pigs, so the answer is *Trichinella*.

No. 10.

**A case of respiratory failure is presented. A chest X-ray shows wide mediastinum. Patient also develops meningitis. CSF shows large gram positive organisms. What is the organism?**

[Inaudible].

Yeah, that gram positive organism is, boxcar-like, anthrax. The answer is A, anthrax. It causes wide mediastinum, as written in your notes.

No. 11.

**A 41-year-old patient fell on the soil. He developed an ulcer in the arm with lymphatic streak coming down the forearm. Diagnosis?**

[Inaudible].

Sporotrichosis. This is a lymphocutaneous sporotrichosis. You should read about it in your notes. These are all new questions, so again the next one is on West Nile virus, which I don't know anything about, so I write down for you to read later on, all right? West Nile virus was asked, too, last year, so you should know a little bit about these tables so that you pass the exam. After you pass the exam, then you will study [inaudible]. [Laughter] Let's go to Preventive Medicine.

## PREVENTIVE MEDICINE

There are a few questions in the exam on prophylaxis with INH for exposure to tuberculosis, so before you give prophylaxis, the first thing is to rule out tuberculosis and then to give INH for nine months, as is written in Paragraph 2, and if there is a history of BCG, just disregard. MKSAP-13 says so, okay? Just don't worry about it. It has no consequences for you, so do as if this is you and not him.

When you do the PPD, it is positive depending upon the situation. If the situation is a close contact, known HIV, transplanted patient, or a chest X-ray with old, healed tuberculosis then even 5mm is positive. On the other hand, only over 10mm will be positive if you have these conditions written there: 1, 2, 3, 4, 5, 6 of them, which only you have to remember, not me. [Laughter] Under those conditions, you need 10mm for PPD to be positive, and lately, two years ago, they added the last one, which was never there, but they also keep changing. Every two years ago this whole thing is changed, so more than 15mm they want you to treat. It doesn't matter what the age is of the patient. In my time it was over 35 [inaudible], and now you give to everybody. As long as it is more than 15, just don't worry, just give it at all ages, even in a low-risk population. So once you understand these basic facts on chemoprophylaxis then you can help me to do eight questions and see what you come up with.

**[Eight questions in three minutes]**

So Question No. 1.

**An asymptomatic 42-year-old male comes to you with a history that his wife has been found to have active TB. You do his PPD and it is only 5mm. His chest X-ray is normal. Liver functions are normal. What would you do next?**

So it's a family member, a close contact, high-risk group, over 5mm, so give prophylaxis. Next.



**A 71-year-old brittle diabetic man is admitted to a nursing home. His two-stage PPD is negative. Soon after, one of the other patients develops pulmonary TB. Repeated PPD on your patient is now 13mm. His chest X-ray is negative. Liver functions are normal. Would you give prophylactic INH to this patient at age 71?**

**[Inaudible].**

More than 10mm and a recent converter. Prophylaxis is needed even if he was not a diabetic, so the answer is INH for nine months.

**A 50-year-old man known to have a history of intravenous drug abuse has a PPD of 12mm. X-ray of the chest is normal. HIV is negative. Liver functions are normal. Would you give prophylaxis?**

Being a drug abuser plus more than 10mm PPD, he belongs to that risk group where it should be given.

No. 4.

**A 31-year-old male visiting New York from Pakistan is found to have a PPD of 15mm. He is asymptomatic with a normal chest X-ray. What is your next step?**

Fifteen millimeters in a foreign-born person, INH for nine months.

No. 5.

**A healthy 26-year-old female from Pakistan has no risk factors for TB. She had BCG in her childhood. She now works in a cafeteria in New York City. Her PPD is found to be 16mm. Chest X-ray is normal. Would you give INH prophylaxis?**

I have nothing against Pakistan, but these questions are there in the exam like that, okay? [Laughter] So just because you think I am from India, [inaudible]. [Laughter] I don't think like that. This is in the exam just like that, okay? If you see a patient in the exam from Pakistan, just give INH don't worry about it. [Laughter]

No. 6.

**A 42-year-old woman from Laos had immigrated to the U.S. ten years ago. Now her PPD is 8mm. She has no symptoms of cough, fever, or weight loss. Chest X-ray shows apical scarring with pleural retraction and a calcified hilar node. What is your advice?**

Old [inaudible]. INH for 9 months.

**A medical resident is stuck with a needle used on a patient with chronic Hepatitis C. Blood is sent for ALT level and Hepatitis C antibody test. What is the next step?**

There is currently— he is sleeping again on me. [Laughter] Let's send him to Pakistan. [Laughter] There is currently no vaccine available to prevent HCV infection. Immune globulin is not effective as forced exposure prophylaxis and therefore not recommended. There are no data regarding the use of antiviral agents such as interferon following exposure, and this is not recommended unless acute infection develops, therefore the answer is observation.

No. 8.

**Which of the following vaccines cannot be given to an immunocompromised host?**

B like in oral polio because live, attenuated vaccines are normally contraindicated, however live MMR can be given. So far there is no increase in the risk of adverse events that have been noted from live or inactivated vaccine, except from oral polio vaccine, so the answer is oral polio. You have five questions, two minutes.

**[Five questions in two minutes]**

So Question No. 1. The risk of seroconversion following needle stick injury for HIV is 0.3%. Compare that with Hepatitis C. If you get stuck with a needle, 1.8% chance of getting Hepatitis C but 20% for Hepatitis B, and only 0.3% for HIV, thank God. Therefore this one even used for patient having HIV, you have to give—this although is low-risk injury. It appears superficial and occurs from a patient with HIV and viral load of less than 1,500. Although it is low-risk injury—what will be high-risk injury? High-risk injury, it is a needle with the presence of physical blood or exposure from a needle that was in an artery or a vein of the source patient, so although this is a low-risk injury, you should give C, obtain the blood and give prophylaxis using three drugs, and you should repeat the HIV test in six weeks, 12 weeks, and 24 weeks, six weeks, 12 weeks, and 24 weeks.

No. 2, if you are going to the island of Belize to work in the caves, you can catch rabies if you go to the caves, and if you go to the island you take Hepatitis A, so the answer is A. You want to protect yourself from bats and Hepatitis A.

No. 3 is a pregnant nurse who is stuck, acute Hepatitis B. She had three injections of vaccines but the B antibody is negligible, so that means the vaccine did not work, so you have to protect her immediately with a booster of immune globulin and then revaccinate her, so the answer is A, and the details are written underneath for you.

No. 4, a medical student is stuck with a needle, and his workup reveals a positive Hepatitis B antigen and negative antibody. Liver functions are normal, so the antigen went in but he hasn't developed any antibody yet. Also he is not protected yet, so you have to protect him by giving Hepatitis B immune globulin.

**[Inaudible].**

You don't understand?

**Why not A?**

Why not what, D?

**A. [in unison]**

He already has the antigen. Antigen is already there. What is the point of giving more antigen?

**if he had antigen, that means that he has disease already, so why do you give anything? He's probably a hep B patient already, himself.**

But he does not. Liver functions are normal.

**Yeah, but you can have normal liver function with hepatitis.**

You are right, but we don't know all that, so immediately protect him. What you are saying is right. Immediately protect him with the globulin. That's what he needed, protection, nothing else, and the rest will take care of itself. How are you going to protect him? He doesn't have antibody. How are you going to protect this antigen to go into the liver and cause the damage? How? Today. Today he got it, so today you [inaudible], Hepatitis B specific immune globulin. Protect him now. You [inaudible] antibody that day. Now later on hopefully his own antigen takes care of itself.

**The point is with A, when you give the vaccine, that's just giving more antigen. Dr. Arora is saying he already has the antigen.**

Okay, No. 5, a 42-year-old with history of hepatitis vaccination, went to island of Belize again, and as luck would have it, he met a very good-looking prostitute, so he got Hepatitis B, simple. We did this many times.

For endocarditis, that's coming later on, there are things that have changed in the past two years. The cardiac conditions where prophylaxis is indicated are written here, and cardiac conditions where you do not need prophylaxis, including acquired aortic or mitral valve disease. This is a total change from before. Even on hypertrophic cardiomyopathy, that's a total change. Unless obstruction is present, you don't give prophylaxis, so you have to remember these conditions, and the procedures where you give it on the left side and where you don't give on the right side. Again, you have to remember this page, so there will be one or two questions on this page in the exam, but it's a drastic change from before. If you are confused on the exam, simply say no prophylaxis and chances are you will be right. [Laughter]

And on the next page is the area of antibiotics for various procedures and choices are given to you, so again those have to be remembered. They usually don't ask you the doses. They just say antibiotic. They don't ask all that, so don't worry too much about it. Now you can do 11 questions in four minutes.

**[Eleven questions in four minutes]**

Question No. 1 is about going to Colorado to ski and getting all these symptoms of mountain sickness. For mountain sickness, on the next trip you can give him acetazolamide. That occurs because of lack of oxygen, and why do you give acetazolamide then? Acetazolamide is given to shift the oxygen dissociation curve to the left. Acetazolamide shifts the oxygen dissociation curve to the left, and that is why it works. You could also give steroids, but they don't mention that, so the answer here is B like in 'boy.'

Question No. 2, with IgA deficiency, even a trace amount of IgA is present in anything you give, like in intravenous immune globulin, you will sensitize the patient and lead to an anaphylactic reaction, therefore the answer is C. As a matter of fact, pneumococcal vaccine and influenza vaccine should be given to patients with IgA deficiency because they tend to get frequent respiratory infections, so the answer is C. Don't give C, plus A and B are okay.

No. 3, a family has a person in the family with rheumatoid arthritis on Prednisone and methotrexate, so really you cannot give—which of the following should not be used in this family, and the answer is oral polio vaccine, for the reasons we said before.

No. 4, endocarditis prophylaxis is indicated in patients with prosthetic heart valve, right? A. You don't give in bicuspid anymore, ostium secundum, no, and if you're going for TEE, they don't need, even with bioprosthetic.

No. 5, a urine culture, vanco, VRE. What is the answer to 5? B like in 'boy'? Okay.

No. 6, patient with artificial heart valve going for cystoscopy, yes, that's the answer. This question was in the form of a table in the examination.

No. 7, for vaginal delivery you will give it or not?

**No. [in unison]**

Yeah, because somebody in the last year's lecture said, 'Well, why don't you give for vaginal delivery? It's such a dirty area.' I said, who told you it's a dirty area. You came from there. [Laughter] Vaginal delivery is not dirty. It is in people's minds. [Laughter] We all came from there. What are we talking about?

No. 8, while using a knife, an 81-year-old gets a cut on the finger requiring sutures. She does not remember a tetanus vaccination. What's your advice? The answer is tetanus toxoid. Because the wound is clean, the answer is A. If the wound was contaminated or very large, then the answer is B like in 'boy.' In a contaminated or large wound, the answer will be B.

No. 9, how would you best prevent gram negative infection? By hand washing, if there is any place to wash in the hospital these days.

No. 10, how will you prevent aspiration in an intubated patient?

**[Inaudible].**

A, elevating the head end of the bed. Avoidance of acid-blocking medications, decontamination of the oropharynx, patient positioning, and subglottic drainage technique, and silver-coated endotracheal tube, so I'm giving you the way to prevent aspiration in an intubated patient. One is avoiding acid blocking medication, secondly, decontamination of the oropharynx, number three, patient positioning, number four, subglottic drainage techniques, and silver-coated endotracheal tube, but the answer on the exam was A.

No. 11, the answer is to make sure that she understands the risks of continuing smoking. That was a strange answer.

New questions.

**Question No. 1, a 22-year-old patient with HIV has a CD4 of less than 200. He is started on therapy for HIV, and Bactrim for PCP prophylaxis. CD4 improves to greater than 400 and remains so for 12 months. What changes would you order in his management?**

**[Inaudible].**

The patient who experiences immune reconstitution on therapy with a rising CD4 above 200 for more than three months can usually safely discontinue PCP prophylaxis, therefore the answer is B like in 'boy.'

**Question 2, what are the contraindications to the use of herpes zoster vaccination in a 75-year-old patient?**

There are three contraindications, and they're all written here for you. One of them will be on the exam.

**Question No. 3, a 33-year-old patient is found to have HIV on a work screening exam. PPD is 7mm. CD4 is 300. Besides starting on antiviral drugs, what else would you give?**

[Inaudible].

Treat for HIV, and the CD4 count is less than 350, A is for if the CD4 went down to 50 for MAC, and B is for CD4 count less than 200, so the answer here is isoniazid. I will give isoniazid besides the antiviral treatment because PPD is 7.

No. 4.

**A 33-year-old patient from Southeast Asia having active rheumatoid arthritis has been on low-dose steroids and methotrexate. She now needs to begin anti-TNF therapy. A PPD is done and it is 7mm. What is your choice?**

This is a new question, a totally new question. The explanation is given underneath, but the answer is C like in 'cat,' INH for nine months starting now and begin anti-TNF therapy after one month.

**Question 6, what is the meaning of two-stage PPD?**

Yeah, this was never asked before. They are trying to ask for the booster phenomenon. I still don't understand it, so what I did was I took it from Up-To-Date and put it here for you to read, booster phenomenon, if they ask it again.

So it is 12:20 now. We will have lunch. You will have a lunch hour wherever you want, and be here at 1:20, when we will start because we have a lot of material to finish. At 1:20 we will start.

[End Day Two, Part 1]

Day 2, Part 2  
5/19/09

## OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

We're going to begin with red eye.

### Ophthalmology.

There are four causes of red eye, and the first one is, of course, conjunctivitis, and in conjunctivitis they ask you what is the most common virus that causes conjunctivitis, and that is adenovirus. Adenovirus is the most common virus causing conjunctivitis. Then they ask you what is the most common bacteria that causes conjunctivitis, and that one is pneumococcus. Pneumococcus is the most common bacteria, and adenovirus is the most common virus causing conjunctivitis.

So the next thing is they want to know keratitis. In keratitis you get pain, photophobia, and no exudates. Exudates occur in conjunctivitis, and if you look at the table, that tells you—under keratitis, write down exudates are absent and symptoms are actual pain with photophobia, and exudates are absent in keratitis. Fungal keratitis occurs for two reasons on the exam. See, my differential is based on the exam questions. When you read the books, it's based on the whole medicine. My differential is based on the exam, so fungal keratitis from the exam, there's only two. One is an agricultural setting, like a gardener, and the second is contact lens wearer who keeps cleaning the lenses. That's true. If you keep cleaning too much, you'll put fungus on it. Nobody washes their hands these days. So those are the differences between the two.

In uveitis, you usually have a systemic disorder to cause uveitis. How do you know its uveitis? Well, they have irregular pupil, besides everything else. Irregular pupil goes with uveitis, and usually it is bilateral because usually there is a systemic disorder like ankylosing spondylitis, Behçet's, inflammatory bowel disease, sarcoidosis, herpes simplex, stuff like that, and treatment of uveitis is with steroids, usually topical drugs or timolol eye drops. In sarcoidosis the pupils are dry because the eye is very dry due to keratoconjunctivitis, and usually in uveitis you have [inaudible] vision, but if the cause is sarcoidosis there is dryness.

In glaucoma, pupils are usually dilated, and glaucoma is of two kinds, open and closed angle. Closed angle has no symptoms—I'm sorry, opposite. Closed angle has a lot of symptoms with a lot of acute pain. The open angle is usually asymptomatic. Open angle is 95% of the time, and you find it out by a routine examination when the pressure is high. In acute glaucoma, which is closed angle, there is a lot of pain, and glaucoma on the fundus examination is a cupping of the disc, secondary optic atrophy. This is optic atrophy. All the optic disc is white, completely white. That means it's atrophic. Cupping means going to the side, one side. It looks like a cup moving to the side, and you will understand more if I show you the normal one. This is a normal disc. See how the disc is divided into two parts, a white part inside and a kind of yellowish—this part [inaudible], and there is no cupping, compared with the previous one, the whole thing is white and has gone to the nasal side, so there will be cupping and optic atrophy in glaucoma. This is acute closure glaucoma, angle closure, very acute, very painful headaches, lacrymation, severe, dilated pupil, irregular pupil, too. The differences are

written in your notes on the table. This is also acute closed angle glaucoma. Look at that. Dilated pupil, face painful. So now you go to seven questions in three minutes.

**[Seven questions in three minutes]**

Question No. 1, sudden onset, dilated pupil, halos around light, fixed pupil. This is classical—sudden onset means closed angle glaucoma, so this is A. Now you agree?

**Yes. [In unison]**

No. 2 is a gardener, so the gardener, pupil is normal, photophobia, so the gardener in the examination, he either has sporotrichosis or fungal keratitis, and this is keratitis. All the symptoms are there, and there's photophobia, but I'm not giving you everything. I don't have everything to give you, and the exam questions will be much more clear than this one. They will be much more. They're usually bigger questions, too, so all I know is the gardener in the exam question is either sporotrichosis or fungal keratitis, so this is keratitis.

No. 3, somebody is cleaning their ocular lenses. They're obsessed about that. They always get Pseudomonas, bacterial keratitis. That's the most common bacteria involved with contact lenses.

And No. 4 is glaucoma again. The patient is relatively—although young, but the consensual light reflex means that side with no direct reflexes where the optic nerve is severely damaged, but because of the other side, optic nerve is okay. There is consensual light reflex, so the symptoms of this one, severe eye pain, conjunctival injection, pain when exposed to light, irregular pupils, dilated, [inaudible].

**[Inaudible].**

Why not what?

**Uveitis? [In unison]**

Uveitis?

**[Inaudible].**

The patient has photophobia, pain, and irregular pupil, the diagnosis is either uveitis or glaucoma. Uveitis is usually bilateral, and the pupil is usually small in uveitis. One, it is bilateral. Number two, usually it is small, not dilated, so if you compare the two, the answer is glaucoma.

No. 5, which of the following is consistent with anterior uveitis with sarcoidosis? The answer is A, the dry eyes.

No. 6, they don't make ophthalmology Board questions without Question No. 6 in the exam. Every year, same story. [Laughter] The moment you read sulfacetamide eye drops, your answer is drug allergy. There is no other question like that in the exam. Sulfacetamide is equal to drug allergy, as far as you're concerned, unless proven

otherwise. They get better, and then next, after a few days, they get delayed allergic reaction



and they get worse. A similar question we'll do in nephrology. You'll give them penicillin or ampicillin for pyelonephritis. They get better, and the third or fourth day they get worse. Allergic reaction to ampicillin. Same question we'll do in nephrology. Those two questions are always on the exam with the delayed—people getting better, then getting worse. Those two questions are always there, one in the eye, one in the kidney.

And then No. 7 is dendritic ulcer is equal to herpes simplex keratitis, so I'm just giving you pointers. I don't know any ophthalmology, I never did any class on ophthalmology. I don't know anything about it, okay, so [inaudible]. [Laughter] If you can pick up a guy from the street here and make him sit here and give these pointers, they'll pass the Board. [Laughter] Nothing to do with medicine. How hard is it to remember dendritic ulcer, whatever it is? I don't know a dendritic ulcer is. I know peptic ulcer. [Laughter] I don't care, and to me [inaudible]. Whatever it is, I don't care.

Then there's the question on retinal detachment. In retinal detachment, it begins with the floaters like I get. I have eye myopia, so I get floaters. I see them floating around. To me everything is floating around. [Laughter] So they float, flashing lights—I didn't get flashing lights. My ophthalmologist told me the day you get flashing of the light, you come to see me, otherwise let them float. [Laughter] So in other words, eye myopia is a cause of retinal detachment. That's one cause, and it begins with floaters, then flashing light, and then scotoma. The risk factors are written in your notes. As you notice, I put myopia there.

On the examination, fundus, it will appear as elevated sheet of retinal tissue with folding, or we call it corrugated appearance. That's what you see on this slide, corrugated appearance, as if there's a separation of retina from the pigment epithelium, so if you read in the question again floaters and then flashing of lights, you should think of this kind of picture. They go together.

Now we show you—after seeing this abnormal fundus, you should see a normal fundus. This is a normal fundus, beautiful discs, blood vessels coming out, arteries and veins. By the way, which is bigger? Is the artery bigger or is the vein bigger?

[Inaudible].

The vein is bigger. The artery is smaller, so optic disc has sharp margins, outer yellow portions about half—50%, inner pale is 50%, and the ratio of arteriolar to venule is 3:4 normally. In hypertension, there are two kinds. One is malignant and the other is arteriosclerotic. In malignant hypertension, by definition of malignant hypertension, you have arteriolar vasoconstriction plus blurred margin of the disc. That's by definition. In other words, marked attenuation of the arterioles is the diagnostic feature of malignant hypertension. I'm showing you these very constricted blood vessels, arterioles. There is this one, this one, this one. All these small, small blood vessels. The big one is the vein. This starburst, that is non-specific, but the main thing is this arteriolar vasoconstriction with the optic disc has disappeared, okay? So that's classical description of malignant hypertension.

Compare that with arteriosclerosis. Arteriosclerosis is of various grading, I, II, III, IV. Nobody will ask you any of this, but just to give you an example, Grade 1 is silver wiring only. Silver wiring, you see? To my understanding, this is silver wiring. [Laughter] I've never asked anybody. I guess that's what it is. Ignore it if it is something

else. Silver lining. [Laughter] In Grade II, there's a nicking. The artery grows across in the vein or the vein crossing the artery. See the nicking? When there is nicking, there is Grade II. Nobody's going to ask the rating, but just to show you, and Grade III, there are hemorrhages, hemorrhages here, hemorrhage here, and cotton wool exudates. Cotton wool exudates and hemorrhage, that's III, and in IV there is papilledema. You don't see the disc. Hemorrhage and papilledema, all of the previous plus more and fibrous arterioles that constrict slowly, compared to the acute hypertension, malignant one, so this is what hypertensive fundus looks like. This is papilledema. Now papilledema has lots of causes besides hypertension, and that's on the Board. Vitamin A toxicity is always on the exam, or carbon dioxide retention causes papilledema. If it is Vitamin A toxicity causing papilledema, in that question on the exam the patient also has bilateral 6<sup>th</sup> cranial nerve palsy. Bilateral 6<sup>th</sup> cranial nerve palsy, you have a problem with looking outside, and that occurs due to Vitamin A toxicity because of the increase in intracranial pressure. High intracranial pressure due to Vitamin A toxicity causes bilateral 6<sup>th</sup> cranial nerve palsy with headaches, with papilledema, and also dry skin, so don't forget all those signs of Vitamin A toxicity that is going to be asked when you go there, and then they will ask you to differentiate that from Vitamin B toxicity because both get hyperglycemia, so remember all those connections, the intricate connections they keep asking.

In diabetes, on the other hand, you get microaneurysms, tiny, tiny, all over, microaneurysms, so many, and these are the small red dots, and then the next step in diabetes is this one, blot and dot hemorrhages, blot and dot hemorrhages scattered, and then from there you go to nonproliferative retinopathy showing hard exudates, hard exudates. See these white ones? These are all hard exudates. The dots and blots are still there, and exudates are much more, so that goes with nonproliferative retinopathy. The next step, which is the worst one, is proliferative. Now you get proliferation of the blood vessels, and when the proliferation of the blood vessels occurs on the disc, that's neovascularization of the disc. That's when you have a problem with the vision, and that's when you need laser photocoagulation, and that's a Board question. When you need laser photocoagulation for neovascularization, you will need that, and it is proliferative retinopathy with neovascularization on the disc. That's when you need it. That's a Board question. This, for example, is a 54-year-old surgeon, 32 years of diabetes; he's got neovascularization on the disc. You can see, so now he needs the laser treatment. And lastly you get vitreous hemorrhage in this condition, which is a complication of proliferative diabetic retinopathy causing blindness. The treatment of this is vitrectomy, vitrectomy, written in your notes.

Now you've got other questions on the fundus. Let's do them together.

**Question No. 1, a 54-year-old patient has well-controlled diabetes for five years. Urine is normal. What would be the findings in the funduscopic exam?**

[Inaudible].

Yeah, if urine is normal in diabetes, fundus must be normal because with the diabetes, microalbuminuria occurs, and then you get dipstick negative proteinuria, and only then you'll get fundal changes. In other words, you first get a urine problem and then you get a fundus problem. If urine is normal, fundus is normal.

No. 2.

**Four photographs of a retina are shown to you during your exam. Which of these needs laser therapy?**

The one with the neovascularization of the optic disc.

In No. 3, ischemic optic neuropathy, the slide is in front of you. Doesn't it look like papilledema?

**Uh-hugh. [Yes] [In unison]**

Yes, it does. It looks like papilledema, but in this condition the vision is also gone. In papilledema, you don't lose vision. In ischemic optic neuropathy, you lose vision, and they look alike on the fundus examination, and the best example of ischemic optic neuropathy for you is to remember temporal arteritis. In temporal arteritis you get unilateral headache and loss of vision. There is loss of vision in temporal arteritis because of ischemic optic neuropathy. There you lose vision, not in papilledema, so you examine the fundus and you'll find this kind of finding. It will look like papilledema, but the patient has no vision, has temporal arteritis. The diagnosis is ischemic optic neuropathy.

The next one, on the other hand, is optic atrophy. I told you that before. The whole disc is white. No differentiation of 50% and 50% pale and white, and this is due to—the best example of this would be a pituitary tumor, like acromegaly or glaucoma, for that matter. Something is pressing and optical atrophy occurs due to pressure, compression of the optic pathway by a tumor.

The favorite fundus examination in the exam is this one, CMV. Record it. They call it a ketchup and mustard appearance, ketchup, and mustard. Hot dog? [Laughter] I used to eat a lot of hot dog and ketchup and mustard, since I saw this slide, no more. [Laughter] Every time I want to eat that, I think of this slide. Forget it. This is CMV. It's in the exam, and then this is in the exam, Roth spots. Roth spots are pale-centered hemorrhages. The center of the hemorrhage is pale. Okay, now you have eight questions in three minutes.

**[Eight questions in three minutes]**

So in the first question they are saying something called Hollenhorst plaques. These atheroembolic little white spots are called Hollenhorst plaques. You see them in atheroembolic disease. It is from cholesterol usually from carotid artery disease, emboli.

No. 2 is a cherry-red spot, which is right here. This is a cherry-red spot, and this indicates a patient has central retinal artery occlusion. The whole fundus is pale, except for one area, which is reddish, and the reason for the cherry-red spot in the macular area is that the fovea, which is the depressed central area of the macula, that's the inner retinal layer so that it remains transparent and the choroid shows through has a cherry-red spot. So in other words, the blood is going in, but less blood going into the whole fundus is pale except for that one particular area, so this is seen in central retinal artery occlusion.

Now in Question No. 3, the answer is C because a cherry-red spot is seen in the central retinal artery occlusion, and the commonest cause of that is embolus from the

[inaudible] major sclerotic vessels. That is why the retina becomes edematous, loses transparency, fundus appears pale, except for the macular area, which shows up the cherry-red spot, and the disc is also pale. Because the cause is usually in the carotid artery, the patient may also get jaw claudication, which is a typical presenting feature of temporal arteritis, which may also get involved in this condition. This is also a cherry-red spot. See that? The rest of it is pale, so it indicates carotid artery disease, so the answer is C for carotid Doppler ultrasound.

No. 4 is opposite of this, which means the blood goes in but cannot come out. The previous one, blood was not going in. Here you see central retinal vein occlusion. The blood goes in but cannot come out because the vein is occluded, and this you usually see in three conditions, hypertension, diabetes, and glaucoma, so the answer is B like in 'boy.'

No. 5, a patient is on TPN. If somebody is on TPN and gets endophthalmitis—by the way, what is this?

[Inaudible].

Central vein occlusion. Same kind of picture, this one, so remember this. It's going to be there. So Question No. 5, the answer is B like in 'boy' because you get monilia endophthalmitis if you have been taking TPN for a long time. That is a known fact and they will question it.

No. 6 is a retinal detachment, and No. 7 is bad CMV retinitis, and the treatment of that is you can give oral valganciclovir or ganciclovir. Any 'clovir' will be fine. [Laughter] Any 'clovir' for CMV retinitis.

No. 8, the answer on the exam is this. When you see the word 'magnifier' written on any of the answers in the exam, don't read the question. [Laughter] Take the magnifier and thank you very much go to the next question. [Laughter] That will save you two minutes to worry about the next question. All right?

Macular degeneration is nothing but with aging or who knows what, if we knew the cause, there is degeneration of macula, so the stuff that comes out, it causes deposits in there. We call them dry exudates. In time, the blood tries to come there to help you and it becomes wet, so dry exudates become wet exudates in the macular area, and as a result you see this. We call them drusen. If you see drusen in the area of the macula, it's a sign of macular degeneration. It's a quite common disease. Nobody knows the cause. The only therapy is for the wet exudates or wet type with laser photocoagulation and photodynamic therapy. They are trying to make angiogenesis inhibitors these days. The last I heard Lily was making it. I bought a lot of stock waiting for that and nothing has happened so far, [Laughter] but I still have the stock. Hopefully one day they'll find this. This is age-related macular degeneration, so we can now go to miscellaneous questions.

**A severely arteriosclerotic old lady wakes up after hip surgery and is found to have internuclear ophthalmoplegia. During surgery her blood pressure had dropped drastically for a few minutes. What is the cause of her internuclear ophthalmoplegia?**

**[Inaudible].**

Well, the thing is that first of all I don't know what it is, internuclear ophthalmoplegia, okay? [Laughter] So whatever it is, it is written underneath there. You read that. All I know is it has two causes. One is perioperative stroke, and the other is multiple sclerosis. Now you tell me from the history which one it is.

**Perioperative stroke. [In unison]**

Perioperative stroke. Thank you very much. We are done. All right? I went to my friend who is an ophthalmologist. I asked him to explain to me what is internuclear ophthalmoplegia. So he took half an hour of ah, ah, eh, ooh. [Laughter] I said, listen, I'll go and read myself, don't worry. [Laughter]

No. 2.

**A patient with SLE has been on Plaquenil (hydroxychloroquine). What is the most important ophthalmologic complication of this therapy?**

Plaquenil causes retinopathy. We know that. I knew this a long time ago when I was in medical school, and I know that chloroquine causes retinopathy because we used to give a lot of chloroquine for malaria in India. Now they're asking that question on the Board. Okay?

No. 3.

**A 33-year-old obese female with a BMI of 40 complains of pulsatile headaches, transient visual loss, and dizziness. Blood pressure is 160/90. A photograph of the fundus is shown. It is consistent with the finding of papilledema. LP shows very high opening pressure. What is the management?**

**[Inaudible].**

Yeah, this is a young girl who is obese, so she has pseudotumor cerebri and benign intracranial hypertension, which is treated with Diamox, so the answer is acetazolamide.

In acute rhinosinusitis, there are a lot of causes, but the Board is interested from you in only one, which is this one, immunosuppression. This is it. They don't want to know anything more, okay? So immunosuppression is the cause of sinusitis. The best antibiotic for sinusitis, acute sinusitis, is Bactrim or amoxicillin, and all patients with chronic sinusitis have colonization with *Streptococcus aureus* or gram negative bacilli, but know that the treatment is Bactrim or amoxicillin at the first attack. Subsequent attacks you can use Augmentin, etc., but initial attack, the Board wants Bactrim or amoxicillin from you. So let's go to four questions in one minute.

**[Four questions in one minute]**

Somebody getting rhinitis every year, we call it perennial symptoms. The answer is either intranasal glucocorticoids or intranasal cromolyn, so I will take—because they don't give intranasal cromolyn, they're giving oral cromolyn, so I will take A as the

answer. In perennial, either you give intranasal steroids or intranasal cromolyn. If that doesn't help, then you refer him to the allergist.

No. 2 has a post-nasal drip and he is using Claritin and nasal steroids. The answer is B like in 'boy' for vasomotor rhinitis.

No. 3, if I told you cobblestone colon, cobblestone colon was what diagnosis?

[Inaudible].

Remember? What was it? Cobblestone colon.

[Inaudible].

Yesterday I told you; remember cobblestone mucosa, when we do cobblestone nasal mucosa.

[Inaudible].

Cobblestone mucosa. Didn't I tell you yesterday? That psychiatric patient? I never told you? Did you hear of cobblestone mucosa? Wasn't that anything?

[Inaudible].

You forgot already. [Laughter] That was yesterday. The exam is in two months. [Laughter] Yesterday I said there are two 'cobblestones' in the exam. One kind is this. As far as the Board is concerned, nothing else is cobblestone. This is it.

No. 4, what is the treatment of acute sinusitis, allergic to penicillin? Bactrim. It is that.

There are two questions on external otitis in the exam. External otitis is of two kinds, benign and malignant. Malignant doesn't mean that it is a cancer. It is not cancer. First of all, both are inflammations, both are infections, both are due to *Pseudomonas*. The only difference between the two is that the benign one is a young patient who is not immunosuppressed, while in malignant external otitis the patient is immunocompromised, so usually in the exam it's an older person who has diabetes. Actually in 2006 it was a Navajo American Indian woman with diabetes in the exam. Navajo, whatever that means, [Laughter] all right, so the point is that this one I'm showing you now is a young guy who just simply went swimming and came out with the ear looking like that, so this is due to *Pseudomonas* infection in the water or he scratched himself or put too much of cotton applicators inside his ears. Some people do a lot of cotton stuff in their ears. They itch too much inside. It feels good, I guess. [Laughter] So from that they get *Pseudomonas*, so *Pseudomonas* causes external otitis, and the treatment of this condition is to avoid moisture and to use, as written in your notes, ear drops containing aminoglycoside antibiotics and anti-inflammatory steroids every four hours, four drops every four hours. In other words, you use a drop and more frequent, four drops, four times a day. That takes care of this.

And then there is the second question on the exam in which it is much worse, to the point that it causes osteomyelitis, so if you have an immunocompromised host who

| is older person in the exam and has *Pseudomonas* which has gone deep into the bone, so they

get *Pseudomonas* osteomyelitis and you have to do CT scan of the bone. The treatment there is intravenous antibiotics. It can look like this inside. This is inside of the ear, okay? This is not a colonoscopy, [Laughter] although I have to tell you it can look like that. Crohn's disease with stricture formation looks just like that, but treatment of malignant otitis externa is with two anti-*Pseudomonas* antibiotics plus surgical debridement. Now that is a Board question. They don't make ENT questions without these two questions when you go there.

Okay, now let's do Question No. 1.

**A 55-year-old Navajo American Indian woman with a history of diabetes comes with foul smelling discharge from the right ear. Tympanic membrane cannot be visualized because of erythema and edema. What is the best treatment?**

[Inaudible].

E is right. Very good. You are very smart. [Laughter] Next?

**A 15-year-old girl goes for bird-watching in New Jersey. She then develops a painful vesicular rash on her external auditory canal. This is followed by the development of bilateral Bell's palsy, and that is when she decides to come see you. Which of the following is the correct answer?**

[Inaudible].

First of all, nobody told her to go to New Jersey to watch birds. There are no birds in New Jersey. [Laughter] I see a lot of deers in New Jersey. Ramsey Hunt syndrome is due to a DNA virus, so you just have to know it cold. This is a very chronic question on the exam. Every year there. In this condition you get—the next page will tell you what it is. The 7<sup>th</sup> nerve is involved; the 8<sup>th</sup> nerve is involved due to this DNA virus. It is a herpes zoster virus, and therefore the ear is involved because the ear canal, vesicular eruptions on the ear and this palsy. It looks like she's smiling, and that is because of Ramsey Hunt plus bird-watching in New Jersey plus [inaudible] bilateral. It can be bilateral, Bell's palsy. It means DNA virus. Again, very specific. You see an answer DNA virus anywhere, just take it.

Next is hearing loss. In conductive hearing loss, what is better? A bone conduction is better than an air conduction, bone, rather than air in conductive hearing loss. In sensory, air is better than bone, but that is the normal case. Normally air is better than bone, but in sensory air is also better than bone except that your hearing loss is decreased. You can't hear. Like I have that. Everybody over the age of 65, 70 gets hearing loss of sensory hearing, just like it's a part of arthritis, same thing. You have to get used to arthritis, you have to get used to sensory hearing loss, just like you get used to getting used to having got married. Same thing. [Laughter] Yeah, you have to used to being married, just like arthritis and hearing loss. [Laughter] [Inaudible]. [Laughter] So I have hearing loss. It starts with noise—what is that called?

**Tinnitus. [In unison]**

Tinnitus, so at this time sitting here I'm hearing this chu-chu-chu, like birds are in my ears and talking to me. I rather enjoy it because [inaudible]. [Laughter] It doesn't bother



me anymore, until I got to the ear doctor. I told the doctor, I said I'm hearing this. He said let me check. He said you've got non-sensory hearing loss. So I said now what should I do? It will only get worse. [Laughter] So I said, that's okay, I'm prepared for that, but what else? He said I have to tell you this. He said there's one case in this world where the patient was so upset with noises in the ear that he committed suicide. [Laughter] And it's a true story he told me. I was so depressed. [Laughter] But after a while now, I'm used to it. I've started to enjoy it. Now it is like music to me. [Laughter] All these young people put the music volume [inaudible]. [Laughter] I have it naturally. [Laughter] So if you get hearing loss, you put the Weber test on the top of your head if you are bored, which is better. [Laughter] You see which way the vibrating noise is going so that way you can tell if there's any hearing loss, and then you can tell whether it is sensory or conductive by putting it on the side like this, the Rinne test, where you put it on the bone and on the ear to see which is better, bone is better or air is better. [Inaudible] the Rinne test and the Weber test. You can tell whether it is sensory hearing loss or conductive hearing loss, so that's how you do it.

Now presbycusis is sensory hearing loss plus an extra problem, and the extra problem is in the brain, like even if they hear, there is a central auditory processing disorder. They are unable to extract speech from the background noise, so if you are at a party—if I am in a party room, now I really cannot hear. That's why when somebody asks me a question from there back; believe me I don't know what you're saying. [Laughter] I really don't. That's the truth, so this is because I have sensory hearing loss, but she could tell what you were saying, as you can see, because she's younger. [Laughter] One day everybody will get it. [Laughter] Do not be depressed because of that because everybody will get it. That's fine. Everybody will get arthritis, and I'm happy with my arthritis, [Laughter] so this is the second problem in presbycusis. It's old age, and unfortunately there is no treatment. You give them a hearing aid. They don't work because they hear more extraneous sounds than anything else, so all old people will throw them away. They don't like it because you hear more of that extraneous sounds than the people's voices, so they don't like to use hearing aids, and now they say cochlear implant, but I found out there's a major problem with that because you have to relearn everything and it's not that easy, so there's nothing much you can do for presbycusis. You read all this page on presbycusis. There's usually one question on it, but in a person taking aspirin every day, and aspirin can cause tinnitus, and tinnitus can also occur in presbycusis [inaudible].

Okay, next question.

**An 83-year-old woman complains of dizziness and feels her room is spinning when she gets up from her bed in the morning. On exam there is rotary nystagmus. When the patient sits up, another episode of dizziness lasting a few seconds occurs. Dizziness is reproduced when she turns her head to the left while lying flat. There is no hearing loss.**

This is benign positional vertigo with recurrent episodes over time lasting one minute or less. Although individual episodes are brief, these typically recur periodically for many weeks to months. It is due to the calcium deposits within the posterior semicircular canal known as canalithiasis, and episodes can be provoked by the specific types of head movements. They call them Epley maneuver. In the exam, in the question you will read the word 'Epley,' E-P-L-E-Y. When they write Epley maneuver is positive, all you say, the answer is benign positional vertigo, so really you don't need to know the details. If

you want to read about this, you will get dizzy after awhile, [Laughter] so don't read it. Just remember Epley maneuver is positive in benign positional vertigo.

And this is vestibular neuronitis. This is also written in your notes. Vestibular neuronitis occurs in young people and is probably viral. Ménière's disease is a combination of vertigo plus nausea plus unilateral hearing loss and tinnitus. Vertebrobasilar occurs in older people with all the Ds, diplopia, dysphagia, dysarthria, and dizziness. We will do that again in neurology. Acoustic neuroma is one-sided unilateral hearing loss, so for acoustic neuroma you have to do a CAT scan or MRI of the brain, so now you have three questions in one minute.

**[Three questions in one minute]**

All are Bs. The first one is acoustic neuroma because it is unilateral, and No. 2 is a young swimmer, so it has to be benign. The answer is B like in 'boy,' and No. 3 is presbycusis. Aspirin causes tinnitus only. Presbycusis causes sensory hearing loss, plus tinnitus. Acoustic neuroma is unilateral, progressive, and Ménière's disease causes other problems like vertigo, tinnitus, unilateral. Let's go to New Questions.

That's acoustic neuroma. The arrow right there. This is [inaudible]. Unilateral, benign. Because it grows, you have to do operation although it is benign. Okay, Question No. 1.

**A 51-year-old man presents with ear pain and decreased hearing from the left ear. Pearly white material is coming out of the tympanic membrane.**

So this was asked for the first time. It is called cholesteatoma. Whatever it is, you're going to need this. This is how I learned medicine. The thing about me is that I don't know anything in medicine if it is not on the Board exam. [Laughter] If it is on the Board, then I will know about it. This is a clear-cut example. This was last year. I didn't know anything about cholesteatoma. I probably heard about it. I never knew anything about it, but then somebody told me this question was asked about here, so now I read and find the slide for you to prepare for it, so now I know what it is, although I'm not telling you because I don't remember at this time. [Laughter] I forget, too. I forget more than you do, so the point is that the only way to learn medicine is to get deep into it. If you show it to others, then you'll learn. How many people knew what this is, knew it beforehand? [Inaudible], so about half or one-third. Not bad. Very good. Okay, next slide. [Laughter]

No. 2.

**Red eye with preauricular lymphadenopathy is common in which of the following conditions?**

This occurs in conjunctival infections, adenovirus usually, so the answer is A.

No. 3.

**Viral conjunctivitis in one eye and then the other eye should be prevented by?**

Hand washing.

No. 4.

**A patient had cataract surgery a month ago. Now she presents with visual impairment and hypopyon. Diagnosis?**

[Inaudible].

Hypopyon, layering of the white blood cells in the anterior chamber, like on the slide. This is endophthalmitis due to bacteria.

No. 5.

**A patient has normal bilateral pupil accommodation and convergence. However, the pupils do not constrict when exposed to bright light. They do not 'react' to light. Diagnosis?**

[Inaudible].

This is ARP, Argyll-Robertson pupil. It's seen in neurosyphilis, and you can read all about it on the next page. We will go to Rheumatology.

## RHEUMATOLOGY AND SPORTS MEDICINE

**Question No. 1, the most common risk factor for osteoarthritis of the knee in a 65-year-old man is?**

[Inaudible].

Osteoarthritis of the knee. Usually it is on the medial side of the knee. That is the beginning of old age. The cartilage has worn out. The space has narrowed. The medial meniscus gets damaged, and you start to get bony sclerosis and cyst formations. That's the beginning of the end of the joint, [Laughter] and when that happens, also, not because of aging but more often with obesity and the Board is very much interested in obesity these days. Obesity is a risk factor for knee and hand osteoarthritis. Out of all these risk factors, they will only ask you one, which is obesity. As a matter of fact, there's one more question on the exam on obesity as a risk factor of something else, and that one is—you must remember these two separate questions on the exam. They are totally two separate questions. One, they will ask you a risk factor for osteoarthritis. It's obesity. The other is a risk factor for uterine cancer. It is obesity. If you practice OB in a city, you get obesity. [Laughter] [Inaudible]. All right? So don't forget obesity for these two questions. It's going to be there. Permanently placed questions, okay? No kind of course in the world does like this, the way I'm doing it. No place. This is the way a Board presentation should be. All those they call Board review courses; they're not a Board review course. This is a Board review course, okay? [Laughter]

Now Heberden's nodes.

**Question 2, Heberden's nodes, and Bouchard's nodes are shown in a 71-year-old patient with joint pains. What is the initial treatment for this patient's arthritis?**

[Inaudible].

The answer is— initial treatment is?

[Inaudible].

Tylenol, acetaminophen. Going back all of the way on this slide, they want you to—this is for MKSAP—give medication in that order. I mean, that's not the real practice of medicine, but that's what they want, up to 4g of Tylenol a day.

Okay, we are on Question No. 3 in rheumatology. A 62-year-old, pain in the hip due to osteoarthritis. To which of the following areas would her pain radiate?

[Inaudible].

This is Bouchard's node, by the way, Bouchard's node. That's a Bouchard's node. It can radiate to all those areas mentioned, buttocks, groin, and lateral thigh, but it's supposed to be the best answer. The best answer is it goes to the groin.

The reason I put this second sentence on the slide is there is another question on the exam where they will say the patient has pain in the hip and the pain is much worse on internal rotation. That means it's osteoarthritis of the hip. That is Question No. 4. [Inaudible]. [Laughter] The Board is very predictable. All these things are there, so one by one, I'm giving you all the questions that you will see there. How can you fail? There is no way any one of you can fail this exam. You just have to know this, every part of this, the way I'm doing it, just know it, okay, and I guarantee you that you will pass, but I can't give that in writing because you may not do what I tell you.

This is [inaudible] in spinal stenosis?

[Inaudible].

So this is not—it is positive that it is not spinal stenosis, although both the patient and the doctor are very well dressed. [Laughter]

No. 5.

**A 75-year-old white man says that after walking about 100 yards he must sit down because of severe pain in both calves. Three to six minutes later he can resume walking for about another 75 yards but then must stop again. Leaning backwards aggravates the pain. Leaning forward relieves the pain. He has thus developed a forward stooping posture. The patient underwent bilateral aortofemoral bypass surgery for peripheral vascular disease one year ago. He also has chronic lower back pain for which he underwent laminectomy and fusion of the L3-4 and L4-5 vertebrae 15 years ago. On exam, posterior tibial pulses are easily palpable, straight leg raising is normal, and there are no neurologic findings in the lower extremities. There is loss of hair in the shin area. Which of the following is the most likely cause of this patient's problem.**

[Inaudible].

Spinal stenosis because straight leg raising is normal. The way I'm looking at this picture, the patient has such lovely clothes on, the doctor [inaudible]. It looks like to me that this is his wife and he is just taking the picture and teasing her. [Laughter] It has to be his wife. [Laughter] Next will be in spinal stenosis you get pain in the back when you go downhill. Normally going downhill causes less pain, right? If you get pain going downhill and you're better going uphill, so that's the opposite of normal, so this is spinal stenosis. That's why they say when you are going downhill. [Laughter]

Okay, No. 6.

**A 74-year-old patient complains of pain in the back and legs when walking downhill. The pain improves on stopping or when going uphill. Peripheral pulses are poor. What is the possible diagnosis? What is the test of choice?**

[Inaudible].

MRI.

These are Board-type questions. A 21-year-old man who has pain and swelling of the left heel, swelling of the left heel like this one, pain and swelling of the left heel. When they give you a young man in your examination, a young man having swelling and pain of the left heel, it is Reiter's syndrome, unless proven otherwise, and Reiter's begins after an episode of diarrhea or urinary infection, and they get painless mouth ulcers, balanitis circinata, keratoderma blennorrhagica, and onycholysis. In other words, they get this and this. This is balanitis circinata, crusted lesions which are painless. Same thing on the palms and soles. You can peel this off, no pain. Onycholysis. Here's one picture of all four; painless ulcers in the mouth and uveitis, so they are all there when you go to the exam, Reiter's. There's another name for Reiter's now.

[Inaudible].

When there is Reiter's, you also get back pain, so differential diagnosis of back pain in a young man; you also have to think of Reiter's. It's the next paragraph. Same thing, painless mouth ulcers, and all. Now HLA-B27 may be positive, but you never order that test in the exam, never. HLA-B27 should never be ordered.

And this patient has ankylosing spondylitis, a young adult having low back pain. They have sacroiliitis. This is the sacrum, sacroiliitis right here or here. If you think this is bad, look at the next one. Okay, no more joint left, so sacroiliitis is ankylosing spondylitis, a sclerosis of the joint. They get a bamboo spine due to ossification, deep to the lateral, collateral ligaments. In the neck they get ossification and straightening of the lower cervical spine. There is a bridging of intervertebral discs right here. This is what happens in ankylosing spondylitis, young man. What they want from you, what are the two associated findings in ankylosing spondylitis in the exam. One is in the lungs; the other is in the heart. They get apical fibrosis in the lung. They get aortic regurgitation in the heart. Two questions in half a minute.

**[Two questions in 30 seconds]**

Okay, Question No. 1, the answer is X-ray of the pelvic bones because that's how you're going to see sacroiliitis. How else can you see? Or pelvic CAT scan. They could give you pelvic CAT.

And No. 2, the associated finding is aortic insufficiency.

In rheumatoid arthritis a late complication is vasculitis. That is skin infarctions or bruising of the terminal arteries. They are very painful. Vasculitis becomes large. They get nail-fold infarctions. This is all signs of rheumatoid arthritis. Bad rheumatoid arthritis means rheumatoid nodules. It has a prognostic significance. If you have rheumatoid nodules, that means it is bad disease. It is associated with high titers of rheumatoid factor.

Question 1.

**A 74-year-old female patient has arthralgia in the hands. A photograph is shown with swollen MCPs and PIPs, however rheumatoid factor is negative. Next step?**

[Inaudible].

I would treat this patient, but there is no option for treatment, so I will check the CCP, antibodies to cyclic citrullinated peptides. CCP is the answer, but if they didn't give me—see, this is classical. I don't need CCP with this [inaudible]. The common conditions with a positive rheumatoid factor are in your notes. You know this. Rheumatoid factor is positive in only 80% of the cases of rheumatoid arthritis, yet more than 90% with cryoglobulinemia have positive rheumatoid factor, so really when somebody has rheumatoid factor positive, you should think of first cryoglobulinemia, Sjögren syndrome, and then rheumatoid arthritis, therefore the CCP test is better than rheumatoid factor, and what is known in Up-To-Date on CCP is summarized for you underneath, so read about it.

Question No 2.

**A 33-year-old woman with active and advanced rheumatoid arthritis does not improve with NSAIDs and low-dose oral glucocorticoids. What is your next step?**

[Inaudible].

Disease modifying agents. The current approach is that more than mild disease, you treat aggressively with disease modifying agents, and when starting disease modifying agents most rheumatologists will use antimalarials and methotrexate, both together. They give antimalarial and methotrexate together, and if that doesn't work then they go to—they used to go to immunosuppressives but nowadays they go to anti-cytokine agents, which are in your notes, like infliximab. They are interleukin-1 receptor antagonists, but as far as you are concerned as internists, you only are allowed methotrexate. I don't know whether an internist uses it. In certain areas of the country, yes, but mostly rheumatologists will use methotrexate and antimalarial, but you can use it, but they don't ask a question beyond that of you because it is not in your domain then. These are the late change of rheumatoid arthritis with ulnar deviation deformities. You now have four questions in two minutes.

**[Four questions in two minutes]**



Patient is on low-dose methotrexate and chloroquine and has pain in the right hip, so if you read—this question, by the way, was asked last year and also 2007. The last two years, this question has been there, so according to Up-to-Date, it says there to increase the weekly dose of methotrexate by 2.5mg with a target of 12.5mg to 15mg after one month of therapy. In other words, you give a low dose for one month, like this one, 7.5mg, and now after one month of therapy, if the patient still has the problem of pain, you start increasing the dose by 2.5mg per week, and you can go up to a total of 15mg per week, double that what it is today, slowly, so that's a Board question in 2007, 2008.

Question No. 2, that's what I just showed. No. 2 is going with this Baker's cyst, so you will order ultrasonography of the popliteal space. This is always on the exam. You will know the answer is Baker's cyst if you see Homans' sign is equivocal. There is no other question on the exam where Homans' sign is equivocal, okay? If Homans' sign is equivocal, that means it has to be you have to order ultrasound of the popliteal space.

No. 3 is a salmon-colored rash in a patient who has rheumatoid arthritis and fever, and that's classically with splenomegaly, leukocytosis. This occurs in Still's or Felty's?

[Inaudible].

So what was the answer?

[Inaudible].

Felty's? That was wrong.

[Inaudible].

Three is C. Oh, yeah, I'm sorry. So this is adult Still's disease. This is an ill patient with leukocytosis and salmon rash.

The next one is Felty's syndrome where the disease of rheumatoid arthritis is very severe and deforming rheumatoid arthritis with splenomegaly and leukopenia. The next one is leukopenia. As a matter of fact, the table will help you. In the table I put Still's disease, Felty's, and Reiter's together because they try to confuse you with these three, so if you read the table well, you will know. Another salmon-colored rash is in HIV. This is salmon rash. This is a very severe, chronic rheumatoid arthritis syndrome. This is ulceration that you see in what? Felty's syndrome?

[Inaudible].

Yeah, vasculitis and ulceration of Felty's syndrome. So in Felty's you've got severe disease, high titer, rheumatoid nodules, vasculitis, ulcers, splenomegaly, leukopenia, neutropenia, basically, and therefore you have to use disease modifying agents in Felty's.

Okay, next.

**A 73-year-old patient with known rheumatoid arthritis and frequent occipital headaches develops acute right upper quadrant pain due to acute cholecystitis. You are called to clear her for surgery. What is your next step?**

[Inaudible].

MRI of the cervical spine. This kind of stuff has been asked in two different forms in the examination. The problem with atlantoaxial subluxation, it can occur in rheumatoid arthritis without your knowledge, so you have to be very careful because you can be sued for more than the malpractice insurance you have because this patient is relatively younger and going for cholecystectomy surgery, and you are her internist, you are called to clear the patient, and if you don't know about the rheumatoid arthritis causing atlantoaxial subluxation, you will not check the neck before the anesthesiologists, who are usually very strong people. [Laughter] They will take the neck of the patient and do this to look inside or put the tube in with force, and he can cause subluxation, but he is not responsible. You are responsible because you cleared the patient without telling him, and there's a posterior displacement of the odontoid process. Normally pre-odontoid space is about 8mm. Between A and O on the slide, the space here is—here it's 8mm. Normal is 3mm, so more than 3mm, thank you very much; the patient is going to get paralyzed, so it is very important. In the other area of the exam, same thing will always be there. It's just a different scenario that patient who had difficult intubation and got paralyzed, what is the diagnosis, so please when you clear these patients with rheumatoid arthritis, make sure you do an MRI of the neck. You want to know what is the space between the odontoid process and the posterior margin of the [inaudible]. That space you have to have before you send the patient for surgery of any kind under general anesthesia, so they always have this question on the exam in some form. Look out for it. These are the things you have to look for in the exam. The question should be there. It's a matter of you finding the question. It is there.

Next?

**A 41-year-old patient with rheumatoid arthritis complains of right hip pain. The patient has been on high-dose steroids for rheumatoid arthritis for a long time. The X-ray of the hip is negative. What is the next step for confirming your diagnosis?**

[Inaudible].

This is aseptic necrosis in which the X-ray will be normal. That's the whole idea, so they want you to do an MRI, although in this case the X-ray is showing you a problem. It is not as good as this side, but MRI will give you the diagnosis better. If you suspect aseptic necrosis, don't rely on an X-ray. Order an MRI. The same is true with a chest X-ray. If you think your patient is smoking cigarettes and has cancer of the lung, don't just be happy with a chest X-ray. If the chest X-ray is negative, go for CT scan of the chest because one of my friends got sued for a million dollars. He had to pay a million dollars because he did not order a CAT scan of the chest in a patient who was a smoker. The chest X-ray was normal, so he goes to another doctor, coughing, coughing, coughing. The other guy ordered a CAT scan, and there it was. By that time, three, four, five months were past, and the patient died, and my friend got sued for a million dollars. So simple, so it is not easy to become a doctor [inaudible]. It's a very tough situation. You're dealing with a life here. My plumber came last night. He charged me—I'm not

kidding you. I'll show you the bill. Three thousand dollars to fix some plumbing in my house. So I told him what is this? And he knew I was a doctor. He said, well, I'm like a

doctor. What is wrong with you? [Laughter] He fixed my pipe, but he doesn't know that we are dealing with live people and we cannot open them up to see what is wrong. [Laughter] I cannot sue the plumber. [Inaudible] and call somebody else. [Laughter] It's a different ball game, but every plumber thinks he's a doctor in this country. Every gardener thinks he's a doctor. He has on his van 'garden doctor.' [Laughter] [Inaudible]. [Laughter] I'm a Board doctor, Board of internal medicine doctor.

Okay, 42-year-old female with patches of skin disorder on the elbow, okay? That's psoriasis. The idea in this question is to show you that psoriasis will affect the distal interphalangeal joints to cause psoriatic arthritis. Proximal and distal, but basically distal, and the Board wants to know from you if you know that causes pitting of the nails. Those are the two Board questions, so if they involve the distal joints, this was from *New England Journal* back in 1995, so although disease can be symmetrical, can be bilateral, usually it is asymmetrical, like this one, with narrowing of the interphalangeal joints, distal, with subchondral cysts and new periosteal bone formation is being shown on the slide in a patient with psoriasis. The other side is normal, so the idea is to show you that psoriasis can also be narrowing or minimal telescoping and [inaudible]. Again, distal joint. This is psoriasis, but they want you to connect this picture in the exam with these, okay? This, this, this, and this, they all go together.

All right, next. On the next page compare the psoriatic arthritis with rheumatoid arthritis. In rheumatoid arthritis, you have symmetrical synovitis of the MCP and PIP, metacarpophalangeal and proximal interphalangeal joints. In osteoarthritis you have symmetrical degenerative disease of the articular cartilage. Those are the differences, mainly again of distal interphalangeal joints with Heberden's and Bouchard's nodes. You should see the differences between these different kinds of arthritis.

Another Board-type question.

**A 54-year-old man with hypertension has been on hydrochlorothiazide. He feels fine and comes for a blood pressure checkup. While the blood pressure is normal, routine blood work shows a uric acid of 12.5. What is your next step?**

[Inaudible].

Do you want to treat asymptomatic hyperuricemia?

**No. [In unison]**

It keeps changing in the literature. When I started medicine, they said treat it. Then they said don't treat it. When I started to do Board, I was—that's why I was failing because I was very confused. [Laughter] So until last year, they said don't treat it. Now this year, Up-To-Date is saying maybe we should treat it. This is making us crazy. [Inaudible] we are crazy. Now more and more. On one side they will tell you elevated uric acid level does not cause renal failure and therefore there is no rationale for treating asymptomatic hyperuricemia, but—and the 'but' is in the notes. Three specific circumstances warrant at least consideration for the institution of antihyperuricemic treatment in asymptomatic subjects.

Number one, persistent hyperuricemia in the infrequent patients with sustained serum urate concentrations greater than 13 in men and greater than 10 in women. Now

we have to remember that, too. These high values carry some nephrotoxic risk, perhaps related to the likelihood of some component of uric acid overproduction. Number two, excretion of urinary uric acid in excess of 1,100mg daily is associated with a 50% chance of uric acid calculi, so they should be treated with dietary purine restriction. Allopurinol should be used if dietary restriction does not reduce uric acid excretion to less than 1,000mg a day. The dose should be adjusted to reduce uric acid excretion below 800mg a day. And third, patients about to receive radiotherapy or chemotherapy that is likely to result in extensive tumor cytolysis should be treated to prevent acute uric acid nephropathy.

So now you have three reasons for treating asymptomatic hyperuricemia. One is in men greater than 13, women greater than 10, second is excretion of urinary uric acid more than 1,100, you want to bring that to 800, and, thirdly, if patient is going for chemotherapy, that can increase uric acid, okay? So this is a total change from before, and that is asymptomatic. Now we're going to go to—so this patient, therefore, I will do nothing.

**[Inaudible].**

Oh, yeah, what about A? Hold on. [Inaudible]. Come on, Randy. He has been on hydrochlorothiazide for a long time. That is not causing—first of all, it is [inaudible]. No, no, no. The answer is D, do nothing. He is doing fine. There is no problem in the man. Why do you want to rock the boat? [Laughter] And the blood pressure is normal. [Inaudible] something else? He is fine. The answer is D, do nothing.

Okay, we'll do symptomatic now, and in symptomatic first you give NSAIDs. If you cannot give NSAIDs because of the [inaudible] disease, you give colchicine. If you cannot give colchicine because of diarrhea or neutropenia or bone marrow suppression, then you should give steroids, but definitely you do not give allopurinol. It is contraindicated in acute gout. I'm even afraid to give it in chronic gout because my patient got Stevens-Johnson syndrome. I almost got sued that time, and she almost died, so this is not that easy. It used to be IV colchicine. No more now for you. Only rheumatologists are allowed to give IV colchicine, not internal medicine doctors. So colchicine is contraindicated in bone marrow depression, renal disease, liver disease, and sepsis, and allopurinol is contraindicated in acute gout, okay? So acute gout is treated with NSAIDs, colchicine, or steroids, and if you cannot give by adding by mouth, you can inject the joint with steroids. That will be fine, too.

Now you go to chronic gout on the next page. In chronic gout, the treatment of choice is daily colchicine plus a uricosuric agent or allopurinol. In chronic gout you get tophaceous tophi on the ear, tophi on the fingers. Okay, about the uricosuric agents, you should not give uricosuric agents if the urine uric acid is more than 700 because if it is more than 700 uric acid, it will become 1,000, and you will get uric acid stones, that's why, so in those cases you use allopurinol. In tophaceous gout the treatment of choice is allopurinol. Okay, seven questions, three minutes.

**[Seven questions in three minutes]**

Got it? Have you got the answers? Can I go? So the first question is chalky material coming out. A chalky material comes out of this, and in this you'll find—or in the

tophaceous gout you'll find crystals of monosodium urate crystals which are strongly negatively birefringent, diagnostic of gout.

In Question No. 3 (sic), tophus of the ear, the treatment is allopurinol. Allopurinol shrinks tophi. It allows the resorption of tophi and decreases excessive urate stores.

Now in No. 3, the patient got gout attacks. Symptomatic while on hydrochlorothiazide, so there you'd stop hydrochlorothiazide.

So in Question No. 4, the patient has acute gout. The answer is colchicine, although I would like to discontinue allopurinol, too, but according to Up-To-Date, lowering of the serum urate to less than 6mg/dL results in elimination of recurrent attacks. He is down to 7.6 from 9.6, so you want to bring it to 6. That's why you want to continue allopurinol, and it is really not a very acute gout. With acute gout, I will stop allopurinol. It's just that he's getting better but the pains are still there. Therefore I will add colchicine to it because most older people with obesity and hypertension, they do have gout where the uric acid is overproduced, and that is what's happening in this case, and you have to give allopurinol and then add colchicine to it.

No. 5. The patient is drinking too much beer. Beer causes gout attack. That I know, so beer should be stopped in Question No. 5.

In No. 6, pitting of the nails. That's a Board question, and No. 7, he has right knee pain while he's nothing by mouth following gastric surgery, so this is a classic description of pseudogout that you get after surgery. After any surgery you can get pseudogout. We will discuss pseudogout as soon as we come from the coffee break. Thank you.

### [Coffee break]

Pseudogout is always on the exam because there are certain specific scenarios they have, which the doctors miss all the time the diagnosis of pseudogout even in good places. When my mother-in-law went after her surgery of the right knee and they admitted her after the hospitalization to a rehabilitation center which was very good, well known, and I got a call from her that she had pain in the left knee. Surgery was in the right knee, so immediately I said this is pseudogout, so I went to the doctor there and said, well, what are we going to do with this? He said I don't know. We want to first do X-rays and CAT scan. I said but this is pseudogout. Why don't we just treat pseudogout? He said how do you know without X-rays? In other words, he didn't know [inaudible]. The point is that pseudogout will occur after any surgery in the knee, and also on the exam it occurs after somebody who has hemochromatosis or hyperparathyroidism, or in my case I get it always, pseudogout, from low magnesium. I run low magnesium for reasons I would like to go into but it will take too long. [Laughter] Low magnesium causes pseudogout. It's a very interesting subject [inaudible] because it's very common condition, yet we miss it all the time, and if you did the X-ray, you will get this calcification of the meniscus and the articular cartilage, as you see here in this picture. If you took the crystals out and [inaudible], you will see weakly positive birefringent crystals. That is very favorite of the Board. On the left side is gout, on the right side, pseudogout. Because pseudogout occurs after surgery, on the exam question usually—it will be patient has hyperparathyroidism and therefore going for

| parathyroidectomy, going for parathyroid surgery, and then after that gets pseudogout [inaudible] because there are two reasons for pseudogout. One is

hyperparathyroidism, the other is surgery itself. So this is hemochromatosis. It can cause pseudogout, as you can see the last line, therefore hemochromatosis does it, hyperparathyroidism does it. When you see a patient with pseudogout, you look for iron, ferritin and iron binding capacity, calcium and phosphorus for hyperparathyroidism, and hypothyroidism causes pseudogout, and low magnesium causes pseudogout. Every time I will take magnesium oxide tablet, my knee pain goes away. It's amazing. It is really amazing. You'd have to get it to understand. I get severe pain in my knee, and I don't even do magnesium level anymore because my muscles start to twitch. Twitching of the muscle is a sign of hypomagnesemia. I simply have magnesium chloride with me in my bag. I take one or two tablets, 200mg each. In half an hour I'm fine. The pain is gone, twitching is gone. It's amazing. It's just amazing, and if I had gone to a doctor, you know, you are 70 years old, you must have osteoarthritis. No. I have no osteoarthritis at all. What I have is pseudogout, so it's very important to know these conditions if you are going to practice medicine—otherwise become a lawyer. [Laughter] Four questions, two minutes.

#### **[Four questions in two minutes]**

So Question No. 1, in diabetic, loss of libido, liver dysfunction, so it's classical hemochromatosis, therefore the answer is B like in 'boy,' and on aspiration of the joint you will get crystals due to gout.

On Question No. 2, the patient has parathyroidectomy and the crystals would be calcium pyrophosphate crystals, B.

No. 3 is the patient—oh; this is something you should know. The calcification of the triangular ligament, wherever that is, occurs in arthritis due to hemochromatosis. Arthritis which is associated with hemochromatosis causes calcification of the triangular ligament. That's something they started to ask in internal medicine Board. It's somewhere in the wrist area, so when you read that sentence, calcification of the triangular ligament, you know they are talking about hemochromatosis.

No. 4 is something, acute pain in the left knee after cholecystectomy, BUN and creatinine high, so you cannot give NSAIDs, you cannot give colchicine, and the patient has surgery so you cannot give by mouth anything, so then you give intra-articular steroids. That would be fine for pseudogout.

For the joint analysis, I looked at it very carefully, and I was looking for the easy way to make the diagnosis, so I decided to first do—this is my way of doing it, so you don't have to do it my way, which is you first look at the crystals of the joint. The crystals will tell you whether it is a gout or pseudogout present depending on the crystals. Suppose they have no crystals. Then look for WBC in the joint fluid. If it is less than 2,000, it is osteoarthritis or trauma. Between 5,000 and 75,000, it is rheumatoid arthritis where the sugar, glucose, should be more than 25, and if it the WBC is more 100,000, it is sepsis where the glucose would be less than 25mg/dL. So I devised a very easy way of telling about what is what in the joint analysis, and, as a matter of fact, to make things easier for you, on the next page I give you the table. Fill in the blanks on the table. Under microscopy on osteoarthritis, write down negative. With rheumatoid arthritis under microscopy, it is negative. Gout is needle-like monosodium urate, strongly negative birefringent crystals, and pseudogout is rhomboidal, weakly positive birefringent crystals. Septic arthritis again is negative for any crystals.



Now the next question is always on the exam, no matter when you go, so read it and give me the answer.

**[One question in 30 seconds]**

What is your first answer?

**[Inaudible].**

D like in 'doctor'?

**Yes. [In unison]**

You're right. This is fibrositis syndrome. [Laughter] Don't look at me. I didn't create that one. [Laughter] It is by the Rheumatological Association of the United States of America. [Laughter] They did this. They gave it to me. I guess all of them have fibrositis. This is fibromyalgia. On your Page 13, it says all you need to know including dermatographism. You can play games on their skin. [Laughter] They are very irritable people. [Laughter] They have irritable bowel, they have irritable bladder, they have irritable brain. [Laughter] They are very high-strung people. They get irritated. Their skin is irritated. Everything about them is—actually irritating people. [Laughter] They will have morning stiffness. That's quite irritating. They have psychiatric problems; they have depression, so the point is that—but normal sed rate. Sed rate is normal.

On the other hand, those other three conditions which the Board wants you to differentiate from them are dermatomyositis, polymyalgia rheumatica, and polyarteritis nodosa. All three have a very high sed rate. That's the difference, so that's why the table will help you to decide what is what because the Board loves it. The treatment of dermatomyositis is high-dose steroids, the last part of the table. The treatment of polymyalgia rheumatica is low-dose steroids, unless the patient gets a complication of temporal arteritis, which is a complication of polymyalgia rheumatica. In that case you will give high-dose steroids. Polyarteritis nodosa, the treatment is high-dose steroids and cyclophosphamide. Fibromyalgia is treated with symptomatic treatment, massage therapy, and amitriptyline. Amitriptyline is always on the exam in fibromyalgia.

So this is dermatomyositis. The sheet does not have a [inaudible] here. This is dermatomyositis. Heliotrope rash, yes.

**Okay, I'll repeat the treatment for each column. Under dermatomyositis it's high-dose steroids. Under polymyalgia rheumatica it's low-dose steroids, unless you have the complication of temporal arteritis, then it's high dose. Then under polyarteritis nodosa, high-dose steroids plus cyclophosphamide. And fibromyalgia, massage therapy and amitriptyline.**

This is heliotrope rash of dermatomyositis. This is Gottron papules of dermatomyositis. They are non-violaceous lesions of the knuckles. This is calcification within the muscle tissue under the skin in dermatomyositis. There are nail fold capillaries, which are giant capillaries of dermatomyositis, and this is the biopsy showing you lymphocytic infiltration in a muscle. CPK is high. Dermatomyositis patients tend to get cancer, and the most common cancer is ovary or the lung, ovary or lung cancer in dermatomyositis.

In polymyalgia rheumatica, they have pain in the proximal muscles, so in the exam they will not say pain in the proximal muscles. They will say pain on combing the hair or pain on getting up from the floor, pain in the thighs from getting up from the floor. Compare that with dermatomyositis where it's not the pain, it's the weakness. In dermatomyositis it's weakness in the proximal muscles. In polymyalgia rheumatica, pain in the proximal muscles. This is a polymyalgia rheumatica with pain, and they get a complication of temporal arteritis, and temporal arteritis or giant cell arteritis [inaudible] occurs in 60% of patients, so when I see a patient with possible muscle pain in my office coming to me with unilateral headache or vision problems, I don't think twice. I just give steroids right there in my office intravenously. Even if it is not that disease, I'm not taking a chance of somebody going blind on me, and that's a Board question.

In polyarteritis nodosa you get multisystem disease, and you will know in the exam that the patient has polyarteritis nodosa if you see the word written, 'drop,' foot drop or wrist drop. Wrist drop or foot drop occurs in the question, so that means polyarteritis nodosa. Foot drop occurs either in polyarteritis nodosa or in peroneal nerve palsy, separate, which we'll do in neurology, but if you are having wrist drop, as well as foot drop, then you know the patient has polyarteritis nodosa, and, if you remember, I did yesterday in Hepatitis B. B leads to polyarteritis nodosa with pain in the right upper quadrant, acalculous cholecystitis, and all that scenario of vasculitis I showed you, and that's connected with all of this. These people also tend to get aneurysms of the kidney, hairy cell leukemia. If you're talking about hairy cell leukemia, don't forget people with hairy cell leukemia, they usually die of *Legionella* pneumonia. In hairy cell leukemia, you always look for *Legionella* and give treatment at the first sign of infection of the lungs. So, you know, everything in medicine is connected. We just can't separate the body into different parts. So now you have three questions in 30 seconds.

### **[Three questions in 30 seconds]**

For Question No. 1, you will do a muscle biopsy for the diagnosis of dermatomyositis because the patient has shoulder weakness.

In Question No. 2, you have visual problems, you will give high-dose steroids, and temporal artery biopsy can be negative because it is a segmental disorder, so the biopsy can be negative. As a matter of fact, I was reading about it lately. Now they recommend when you do the biopsy, do bilateral biopsy. Headache is on the one side. Doesn't matter. Biopsy both sides. Hopefully it will come positive on one side.

No. 3, again there's the foot drop. That means—and Hepatitis C in the past, so this is periarteritis nodosa, and talking about periarteritis nodosa, this is what an ulcer will look like in periarteritis nodosa. Because this is a multisystem disease, it causes vasculitis, gangrene of the finger. Periarteritis nodosa biopsy. They will get pericarditis, so all those findings of polyarteritis nodosa are on Page 14. On Page 15 you have five questions in three minutes.

### **[Five questions in three minutes]**

For Question No. 1, you will use low-dose steroids for polymyalgia rheumatica. Usually up to 15mg of Prednisone is enough for these people.

In No. 2, definitely polymyositis, you need a high dose, so 10mg [inaudible] 40mg [inaudible].

And Question No. 3 with jaw claudication and ischemic fundus, this is temporal arteritis, therefore high-dose steroids, like 60mg of Prednisone a day, would be the answer.

And No. 4, we did that question before. You have to do a HIDA scan for diagnosis of cholecystitis.

And No 5 is sinusitis with chronic nasal discharge and saddle nose deformity, cavitary lung lesion and [inaudible]. Diagnosis is made by C-ANCA. These are the lung nodules, which are kind of cavitating in the middle. They get conjunctivitis, uveitis. C-ANCA is very specific for the diagnosis, however you still have to,—because you want to give certain medications, you still have to get a biopsy of the pulmonary tissue to make the diagnosis. You want a biopsy of the pulmonary tissue, a big good piece under direct vision like thoracoscopy because you want to give them Prednisone and high-dose cyclophosphamide, so C-ANCA is okay, but then you need a biopsy before you begin Prednisone and cyclophosphamide. If you did the upper airway biopsy but [inaudible] does not show granuloma. You want to see granuloma of the [inaudible] before you give steroids and cyclophosphamide.

So what do you think of the Board-type question on Page 16? Which tissue will offer the highest diagnostic yield?

**[Inaudible].**

The answer is pulmonary tissue, preferably obtained by open or thoracoscopic lung biopsy, and the treatment is cyclophosphamide and Prednisone. Now they don't make rheumatology questions without this question. This is always there, cyclophosphamide plus Prednisone. Permanently placed question.

In Henoch-Schönlein you can get non-thrombocytopenic purpura, glomerulonephritis, abdominal pain, and arthralgia. A young child getting all that—can you imagine that. The rash is usually purpura usually on the extensor surfaces of the arms and legs. They get abdominal pain because of extensive purpuric hemorrhage of the small bowel. Luckily most patients recover by themselves.

This is Kawasaki, next page, strawberry tongue, fever, desquamation of the skin, congested throat, and lymph nodes. This is vasculitis of unknown cause, a multisystem disease of children. They have cervical adenitis, congestion of the mucous membrane, and desquamation of the skin. They get coronary artery aneurysms in 25% of patients. The disease is self-limited, death rate is 3%, and the treatment is IV gammaglobulin plus aspirin. I have seen one case in my lifetime. Has anybody seen Kawasaki? One, two, three, four, five, six? A good number. Eight or ten people have seen it. So coronary artery aneurysm in 25% of Kawasaki.

Then there is Behçet's syndrome, which is also vasculitis. It is actually venulitis, venulitis with venous thrombosis involving multiple systems, painful ulcers, painful ulcers of genitalia and mouth. You get optic neuritis, retinal vessel occlusions, uveitis, hemorrhage in the sclera, right here, and pathergy test.

Then there is Takayasu's, feeble pulses in the upper extremities. A young girl, young woman with feeble pulses in the upper extremities. This is a granulomatous vasculitis of the subclavian arteries and branches of the aorta. There's claudication and Raynaud's phenomenon in the upper extremities. They even get syncope because of carotid artery involvement, visual changes, and TIAs. TIA. The treatment is steroids because of vasculitis.

What about the feeble pulses? There is another Board question where the pulses are feeble.

[Inaudible].

[Inaudible], so both the questions are there.

**A 34-year-old Asian hairdresser in her first pregnancy comes for numbness and tingling of her left arm. On exam the blood pressure in the left arm is 80/60 and 90/70 in the right arm. Radial pulses are feeble, but pulses are normal in the feet. Examination of the heart reveals a murmur of aortic regurgitation. She complains of joint pains and low-grade fever. Diagnosis?**

That's the classical description of Takayasu's. The next one is a disease of unknown cause.

**A patient gives a history of asymmetric arthritis of large and small joints for months. He now comes to you because of sudden onset of pain, tenderness, and swelling of the cartilaginous portion of his ear. On examination, the skin of the ear is beefy red and tender. This patient has a stuffy nose with rhinorrhea and saddle deformity of his nose. His voice is hoarse. What is the diagnosis?**

[Inaudible].

He looks like President Nixon. [Laughter]

### **Antibodies in rheumatology.**

Antinuclear antibody by itself is not a very sensitive test, however the pattern is somewhat helpful. For example, if they give you peripheral pattern of antinuclear antibody, that means acute lupus, generally, but you have to confirm with double-stranded anti-DNA for lupus. With a diffuse pattern of DNA, which is this one, it's really not good because [inaudible] even in normal people, so this does not help you. The speckled pattern, which looks like this, is again seen in chronic lupus, rheumatoid arthritis, and scleroderma, but the nucleolar pattern, which is this, is usually seen in scleroderma, so I would suggest when you do ANA, if you see peripheral pattern or nucleolar pattern, then only you have some assurance you know what the patient has and go for the diagnosis. With the diffuse and speckled pattern, you don't know, so the table helps you. The table is telling you that anti-double stranded is for lupus, anti-histone is for drug-induced lupus, anti-Smith antibody, which is in the exam every year without fail, is specific for lupus but only occurs in 25% of patients, so when it occurs it is pretty good for lupus, and it is there in the exam. Anti-centromere is for CREST syndrome, anti-RNP is mixed connective tissue disorder, and SSA and SSB (Ro and La) are for Sjögren's syndrome. someone got rhinitis medicamentosa. [Laughter] In fact

his mucosa is cobblestoned. So you have all that in front of you, and now you want to do eight questions in three minutes.

**[Eight questions in three minutes]**

So Question No. 1, the patient has a classical description of lupus, so initially the screening test would be antinuclear antibody.

Question No. 2, the patient has antiphospholipid symptoms, and therefore you will order antiphospholipid antibody. They tend to get livedo reticularis. Livedo reticularis is this one. This came from the *New England Journal of Medicine* last month.

No. 3 is the answer is anti-Smith antibody which goes with the middle part of the slide. Which subclass of ANA would be the most specific? Anti-Smith. The upper one, which subclass of ANA which is most sensitive is?

**DNA. [In unison]**

Which subclass is most sensitive?

**[Inaudible].**

And the last one is drug-induced, [inaudible], but they ask the middle one all the time on the exam. [Inaudible].

And Question No. 4, anti-double stranded DNA. That is A. No. 5 is scleroderma, and that question is every year on the exam, so No. 5 is always on the exam. Interstitial pneumonitis is the answer. Actually there were two questions on this. In one question the answer was interstitial pneumonitis. Another similar question, the answer was pulmonary hypertension, pulmonary hypertension, so in scleroderma you get interstitial pneumonitis and pulmonary hypertension. It depends what they ask and the answers. This is scleroderma, and this is the interstitial pulmonary fibrosis, so No. 6 is hypertension in scleroderma. You give them ACE inhibitors. Actually on the exam it was ramipril. Ramipril.

No. 7 is Raynaud's. She has dysphagia. Raynaud's plus dysphagia is CREST syndrome, and for CREST syndrome you order anti-centromere antibody. In CREST syndrome you have calcinosis, Raynaud's, esophageal dysmotility. What else?

**[Inaudible].**

S is for sclerodactyly. This is sclerodactyly, this is sclerodactyly, this is sclerodactyly. This is telangiectasia, so completes CREST, and also you get disorganization of the capillary beds, [inaudible] of the capillaries in scleroderma, so the answer to Question No. 7 is anti-centromere, and Question No. 8, the answer is changes in the nail bed capillaries. What will increase the likelihood that the patient has a connective tissue disorder? Changes in the nail fold capillaries.

Next is Raynaud's phenomenon. Eighty percent of patients with scleroderma get it, and 20% of patients with lupus get it, therefore I will say if you have a patient with Raynaud's phenomenon, simply order ANA. If it is peripheral type, you know this is lupus. If it is nucleolar type, you know the patient has scleroderma, and the centromere type, you have CREST syndrome, so simply by ordering ANA in a patient with

Raynaud's phenomenon you can get to the diagnosis. If they are not there, then its idiopathic. That is primary, primary or idiopathic. OK, can I go?

Next is dental caries. There are three causes.

**A 33-year-old female develops a cold, ear infection, and parotid swelling. Antibiotics are prescribed and the infection subsides, however the parotid swelling continues. On exam the patient has dry mouth, dental caries, and dry eyes. What is the possible diagnosis?**

[Inaudible].

So Sjögren's syndrome, you get dry mouth, dental caries, xerostomia. Rheumatoid factor is positive in 90% of the cases. Diagnosis is suggested by Schirmer's [inaudible] paper test. It's confirmed by biopsy of the minor salivary gland of the lower lip. That will show lymphocytic infiltration of the labia minor salivary gland.

Can I go? We're going to go to Sports Medicine.

**Question No. 1, a 55-year-old athlete who is a swimmer and tennis player complains of a dull ache in his right shoulder that interferes with his sleep. On exam there is tenderness on the lateral aspect of the humeral head and severe pain when his arm is abducted or externally rotated. What is the diagnosis?**

[Inaudible].

As you can see, the rotator cuff of the shoulder is made up of four muscles, the supraspinatus, the infraspinatus, teres minor, and subscapularis. Out of these, it so happens the supraspinatus is the most important in a way that when injury occurs to the supraspinatus, it's called over-use injury. The patient has difficulty— as in the question—difficult in abduction and external rotation. So it all depends upon if it is inflammation or if it is a rupture of the tendon. If it is a rupture of the tendon, there will be weakness, along with pain, but if it is tendonitis only, it's just pain, so both the questions are in the exam a year ago. For example, in this question, the patient has rotator cuff tendonitis, the repeated action, which is in this case a tennis player and a swimmer, causes overuse injury of the rotator cuff muscles, one of them. The patient has difficulty in abduction and external rotation. Not only that, there is a bursa, which is subacromial bursa, right here, which gets involved with the tendonitis and they get severe pain on abduction and external rotation. This kind of tendonitis is very easily fixed by the internist giving the injection into the joint of inflammation a small amount of steroids, and that takes care of the problem. You are supposed to give NSAIDs, local steroids, and physical therapy without sending the patient to the orthopedic doctor. That's what the Board wants from you in the question on the exam.

Compare that with the second question.

**A 51-year-old construction worker fell off the ladder on his outstretched hand and is now unable to abduct his left arm. During examination, when asked to abduct the shoulder, he shrugs it, however he is able to hold the arm up once 90 degrees of abduction is passively applied by you. X-ray shows mild narrowing of the acromial space. There is no fracture. Diagnosis?**

[Inaudible].



Here there is weakness. There is a weakness on abduction. In other words, he shrugs it. He cannot do it. So when you examine him, you bring his arm up with your hand, and if you let go then he will drop it. It's called positive drop arm sign. That means there's a complete tear of the rotator cuff. The patient has weakness and a positive drop arm sign. This will indicate that you want to refer this patient to the orthopedic surgeon. There's a weakness of abduction and external rotation. In a rupture, unable to initiate abduction, he will shrug. Pain and weakness of abduction and external rotation, positive drop arm sign. Diagnosis by ultrasound or MRI or arthrogram. MRI will show both tendonitis, as well as tear, therefore I will do MRI because it can tell about both, and [inaudible] consultation, orthopedic surgeon. This one, you will not inject steroids. The previous one, yes. Those two questions are always on the exam no matter when you go.

No. 3.

**A 55-year-old lady develops shoulder injury and the shoulder is placed in a sling. After a few weeks when the pain is reduced, the sling is taken off. Now she is unable to move the shoulder. On examination her shoulder is tender with restrictive active and passive movements. An X-ray reveals osteopenia. During an arthrogram only less than 1.5mL of contrast material could be injected into the shoulder joint. What is the diagnosis?**

[Inaudible].

This is adhesive capsulitis or frozen shoulder. It can happen in people who have hemiplegia for whatever reason and they don't move the shoulder for whatever reason. This is the reason for the frozen shoulder, and the treatment of this is early mobilization and physical therapy. Sometimes they need mobilization or manipulation under anesthesia, which is quite painful, that's why under general anesthesia, so that's the third question on the shoulder. Those are the three shoulder questions in the examination.

The next one is you move down from the shoulder to the elbow.

**No. 4, a tennis player develops pain on his lateral epicondyle. He stopped playing tennis and the pain went away, however the same pain returned when later on he lifted a suitcase while at the airport. What is the treatment?**

This is a classical description of tennis elbow. There is pain on the—this is due to the epicondylitis extending on the medial or the lateral side of the elbow. It is precipitated by when you lift a racquet and hit the ball, or when the elbow is extended, so then the pain in the elbow area on either side. Extensor muscles are involved, and the treatment is you give your local injection of steroid you should be carrying in your pocket all the time. [Laughter] You will need a lot of that in sports injuries.

No. 5.

**A patient is unable to extend at the elbow where he has pain and tenderness. What is the probable diagnosis?**

[Inaudible].

So this was tennis elbow—now compare tennis elbow to the next one that she just read, No. 5. This is olecranon bursitis. In olecranon bursitis, the bursa is on the posterior aspect on the triceps insertion, and bursitis olecranon can be traumatic or aseptic, and the traumatic one is called Student's elbow, and it is treated by aspiration, NSAIDs, and steroid injection, but if it keeps coming back, excision is the only choice there, but this one compared to tennis elbow, which is on the lateral or medial side, this is on the posterior side, and they're all on the examination when you go.

There is a question on the exam on carpal tunnel syndrome, No. 6.

**Which of the following is the correct answer regarding initial management of carpal tunnel syndrome?**

[Inaudible].

Carpal tunnel syndrome is median nerve involvement, and therefore you have atrophy of the thenar eminence and you get pain at nighttime in your hand, numbness and pain in the hand, so at night time you will do this, but the most common cause is pregnancy as far as you are concerned for the examination. The only thing they ask is a cause for carpal tunnel syndrome. It's pregnancy. They don't ask others. The answer, the treatment is a wrist splint in the neutral position during nighttime because pain occurs mostly at nighttime, so at nighttime you put the wrist splint, [hard splint, but pregnancy is the cause. Remember that for the examination.

No. 7.

**A female lab technician has pain in the right wrist and hand. Adduction of the thumb across the palm causes pain. The same pain occurs when she grasps a pipette at work. Diagnosis?**

[Inaudible].

The answer is A because you know on the slide. It is tenosynovitis of the abductor pollicis longus and extensor pollicis longus and brevis tendons. There are three tendons in this area, abductor pollicis longus, extensor pollicis longus, and extensor pollicis brevis. Those tendons from overuse of the wrist or the hand, the patient gets pain at the radial site of the wrist during grasping or thumb or wrist movements, and pain radiates to the thumb or up to the volar aspect of the wrist. The patient may complain of difficulty writing something, and this is—what they're showing you in the picture is the Finkelstein test. The Finkelstein test is pain on thumb adduction. On thumb adduction there is pain. It's a positive Finkelstein test, as seen in this tenosynovitis. This was asked last year in the exam. I had never known about it until it came. I have a friend. His last name is Finkelstein, so I called him and asked him what do you think about this tenosynovitis? He knew it right away, the Finkelstein test is positive. [Laughter]

No. 8.

**A 52-year-old patient complains of pain on the upper lateral thigh that worsens while lying on that side. The pain is referred down the lateral aspect of the leg. Examination of the hip is normal. What is the treatment?**

This is trochanteric bursitis, also on the exam. The trochanteric bursa is right here. It's on the lateral aspect, therefore when you lie on the lateral aspect too much, there is nothing else to do, watching TV, lying down, [Laughter] you get the pain going down the leg. That's called trochanteric bursitis. If you have that, somebody has to inject steroid into the trochanteric bursa for you to get better. That was on the exam, too, last year.

**Question 9, a 44-year-old patient has pain in the right knee on repeated climbing of the stairs. On exam there is a point of tenderness 4cm below the knee, inferior and medial to the patella. Diagnosis?**

[Inaudible].

Number 9, they don't make sports medicine questions without No. 9 question. How do you know? The moment they mention 3-4cm below the knee on the medial side. When you see the words '3-4cm below the knee,' they are talking about anserine bursitis, and it's always there. It causes pain while climbing stairs. By now you know injection of steroids is the treatment. [Laughter] That's a classical Board question, anserine bursitis.

**No. 10, a 72-year-old lady with known osteoarthritis complains of one-day history of pain in the right knee that locks and causes a 'give-way' sensation while going down the stairs. On exam there's swelling and tenderness but the anterior Drawer sign is negative. What is the next step?**

[Inaudible].

The answer is B as in 'boy.' MRI is diagnostic for all the tears in up to 90% of the cases. This is known. The problem is there are two conditions which get confused, anterior cruciate ligament tear versus medial meniscus tear, so I decided to help you with it in the next question by giving you a table to decide which of the two. You can tell that in anterior cruciate ligament, it's excruciating pain, okay? On the other hand, a medial meniscus tear, the pain is slowly coming. Like I got injured today so I'll call my orthopedic surgeon tomorrow. I'm not that much in pain, but if I had an anterior cruciate ligament tear, I don't wait til tomorrow. I call the doctor right away, so that's how you will know. Pain is acute, you can't take it. That's excruciating pain, I call it, so it is anterior cruciate ligament tear, okay? Now the other thing to remember is that in anterior cruciate ligament tear, the anterior Drawer sign is positive. Nobody is going to ask you on the exam what is anterior Drawer sign, although if you want to know, it is written underneath for you, but they don't ask that. You just have to somehow remember that in anterior cruciate ligament, anterior Drawer sign is positive, and in medial meniscus tear, McMurray's sign is positive. M goes with M and anterior goes with anterior. All right? And sudden pain, anterior, excruciating pain is anterior cruciate ligament. Slow pain is medial meniscus tear, so if you know those differences, you will be able to answer—there are two questions on the Board on this. You will be able to answer both of them.

The answer to Question No. 10 was MRI. You see the tear. Although it is telling you anterior Drawer sign is negative in Question 10, that means the answer there is medial meniscus tear.

On the next page, No. 11 is the management.

**What's the answer to No. 11?**

What is the diagnosis?

[Inaudible].

McMurray sign is positive. That means medial meniscus tear, and the management of these tears is ligament and meniscal tearing can benefit from arthroscopic management early in the course. Symptomatic treatment is with knee braces. You give them a knee brace, you give them NSAIDs, you give them range of motion exercises, you give them weight-bearing exercises as tolerated, and the treatment of choice, of course, in severe cases is total knee arthroplasty. Total knee arthroplasty is the treatment of choice, if needed. Before that you do NSAIDs, range of motion exercises, physical therapy, and weight-bearing exercises.

**A 31-year-old female has pain in the right sole when she steps out of bed in the morning. The pain is worse in the morning and getting better throughout the day and then starting over again in the morning. What is the possible diagnosis?**

[Inaudible].

Plantar fasciitis. You see the plantar fascia goes like that. It actually comes from the calcaneum and goes to the front of the foot, and it is when you get fasciitis and you are walking, you are really causing a lot of tension in the fascia, so that the fascia pulls on the calcaneum. It pulls on constantly all the time when you are walking. As a result of this chronic pull, after a while you get pain in this area because of the pulling right there, and if you do the x-ray, you will get calcaneal spur from the pull, so you get a calcaneal spur, and the Board will give you this picture, and the Board will keep talking, telling you about there is a calcaneal spur there, there is a spur, there is pain, there is pain. Then finally the question, after all this three-page question they'll ask you, okay, what is the cause of the pain? Well, ladies and gentlemen, the cause of the pain is never the spur. The cause of the pain is plantar fasciitis. They will try to get you to answer as if—they'll pay a lot of attention to the spur, and yet the spur is not the cause of the pain. The cause of the pain is fasciitis, so you don't fall into their trap. They want you to answer spur because of the pain so that you will go there again next year and pay them another \$1000. [Laughter] But I don't want you to give me any money. I don't want you to come back but I raise the price every year. [Laughter] Therefore the answer to this question is plantar fasciitis.

And now to tell you there are a lot of conditions that look similar in the exam, so I decided to give you a table. Its plantar fasciitis, its tarsal tunnel syndrome, Achilles tendinitis, and eosinophilic fasciitis. In eosinophilic fasciitis, you will get pain when you do strenuous exercise. There is marked eosinophilia, so the treatment is steroids. Tarsal tunnel syndrome is just like carpal tunnel syndrome when you get pain and numbness in the feet at the end of each day, especially on walking, and that is due to compression of the posterior tibial nerve, and just like positive Tinel's sign in carpal tunnel syndrome, you get positive Tinel's sign in tarsal tunnel syndrome. Treatment is local steroids. In Achilles tendinitis, pain and swelling in the area of the posterior part of the heel, posterior heel pain, overuse syndrome over the tendon. The tendon can rupture. Treatment is just stop running, start watching TV, [Laughter] take some NSAIDs, and physical therapy. Alright, so read those four causes of pain in the feet and you will be able to do them all very easily. Now you have two minutes and four Board-type questions.

**[Four questions in two minutes]**

Question No. 1 is a classic description of burning of the toes, pressure on the posterior [inaudible] nerve on the medial aspect of the ankle beneath the flexor [inaudible] that occurs in tarsal tunnel syndrome because the nerve is trapped over the posterior—behind the medial malleolus, and it causes tenosynovitis, rheumatoid arthritis, and [inaudible]. The most common cause is pregnancy.

Question No. 2, I did that before. The answer is plantar fasciitis. Question No. 3 is a new question on the exam in the sense that the formation of ANA and antibodies to double-stranded DNA have been important in response to all TNF inhibitors but may be more common with infliximab, so here you get double-stranded DNA. [Inaudible].

[Inaudible].

Okay, let me read the question again. A 41-year-old, facial rash and arthralgia. The finding is related to the development of? Well, because he developed a facial rash and arthralgia, which is lupus-like syndrome.

[Inaudible].

Its drug induced. Its anti-histone.

[Inaudible].

Oh, they are saying why not it is drug induced?

[Inaudible].

Oh, I see, double-stranded DNA antibody. That's what you want to write, anti-double-stranded DNA antibody. You can write that, but if you have [inaudible] the question, really I don't know. I don't know what I'm talking about. [Laughter] [Inaudible]. It says there 'the formation of ANA and antibodies to double-stranded DNA have been reported in response to all TNF inhibitors.' Now this patient got anti-TNF therapy or infliximab. It doesn't matter. Cases of vasculitis and lupus-like syndrome have been reported and associated with TNF inhibitor use. What I'm saying to you is this patient got TNF therapy, so he has developed a syndrome which is lupus-like syndrome with double-stranded DNA antibody. Now if you are talking about anti-histone antibody, I would say what is the drug which causes that?

[Inaudible].

[Overlapping conversation]

Okay, those of you who are wondering or have a question about this can come and talk to me. I will also check it again in the literature to see what is going on. My feeling is that lupus-like syndrome, you would do anti-DNA antibody, so the question could be written wrong by me or whatever, given to me, but this is something in the exam that was never asked before, okay? It seems to be a lupus-like syndrome due to infliximab, so however you want to put it, it doesn't matter.

Question No. 4, somebody falls on their outstretched hand. In the examination there is—you should do a bone scan because falling on the outstretched hand can cause a fracture of the scaphoid bone. Fracture of the scaphoid bone and the non-union

of the fracture is very common. This fracture is rarely seen by X-ray, however when suspected a bone scan should be ordered for diagnosis. MRI is undetermined so far. Studies are underway for MRI for scaphoid fracture. They don't know anything about it yet, so bone scan is the best answer, and actually in the exam last year the treatment was asked for this. The treatment was using a hard splint for two weeks. H-A-R-D. A hard split for two weeks was the treatment of scaphoid fracture. Ladies and gentlemen, except for these questions, read this, I'm done. Thank you very much. See you tomorrow morning.

[End]