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This is Dr. Heather Anderson with the University of Kansas Medical Center. Today I will be presenting a review of the NBME Neurology Shelf Exam.

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In considering the evaluation of a patient with transient alteration of consciousness, your two primary considerations are that of syncope vs. seizure. Sometimes people say "Why not TIA?" For someone to lose consciousness it would have to be to be either a bi-hemispheric event or something that is affecting the reticular activating system. So, for the most part, strokes, especially not TIAs, are really not involved and should not be included in the consideration of transient alteration of consciousness.

There are characteristics prior to, during, and after the event that help us to distinguish syncope vs. seizure. Prior to the event you might see that the patient is pale, clammy. They might feel that they have the sense that they might pass out. Those would be some examples of pre-syncopal symptoms. Prior to a seizure, a patient might describe an aura: Olfactory hallucinations, auditory hallucinations, sensory symptoms, a rising sensation in the stomach, déjà vu. All of these auras are actually simple partial seizures, so when you are taking the history from a patient with seizures it is critical to ask how often they have auras, because those are counted when we are evaluating how often a patient is having seizures.

During the event, if there are any jerking movements you certainly have to think about seizures, but not all jerking movements are seizures. You can have what is called "convulsive syncope," where, depending on what's going on during the event, during a blood draw or some other factor that would maybe make you think it's a syncopal event, you would certainly have to consider convulsive syncope. Patients with convulsive syncope also do not have the post-ictal confusion and the other features of seizures. It's certainly critical you find out how long the event lasted because the duration of the event can also give you an idea of syncope vs. seizure. And then after the event you certainly want to find out was there any post-ictal confusion, and if so how long, the duration of the post-ictal confusion. You want to find out if there is any urinary incontinence or tongue biting or any sort of injury. We would expect that in an epileptic event, the tongue biting would be on the side of the tongue. If the patient bit their tongue on the tip of the tongue, one of the considerations would be that of a non-epileptic event.

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If you are asked to evaluate a patient that has been found down, you certainly have to consider a syncopal event or a seizure, a cardiac event, or trauma, but you also have to consider opioid overdose and less commonly, something like locked-in syndrome. If the patient is unresponsive and has a non-localizing exam, and there is no evidence of trauma or anything like that but has diminished respirations and hypoxia, you certainly have to consider opioid overdose and treat with naloxone or Narcan. Locked–in syndrome is in the consideration if someone is unresponsive, it is certainly not very common but it can happen. In these situations the patient

may not be able to move their arms or legs; they're not able to withdraw from painful stimulation. They may or may not be able to blink their eyes but they do have maintained awareness, and they do have normal sensation. They can feel you mashing on their nail bed or doing a sternal rub. They can also hear you talking around them, so I am always cautious when talking with the family at the patient's bedside. If the patient appears to be unconscious you just have to be careful. You never know how much the patient can actually hear. In locked-in syndrome, the patient can have preserved vertical but impaired horizontal eye movements. Like I said, they may or may not be able to blink. Locked-in syndrome may be caused by an ischemic or hemorrhagic event involving the basilar artery. A basilar artery stroke would be the most common cause. You also have to consider central pontine myelinolysis, due to rapid correction of the sodium; it can also be seen in trauma, brainstem tumor, toxins, and heroin abuse.

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In the evaluation of a patient with sudden onset of weakness, you have to wonder about a stroke. Strokes are a neurologic emergency and require immediate attention. Our one and only one treatment for acute stroke is TPA, and there is a time window during which we can use TPA. On the shelf exam, the time limit would be 3 hours. In reality you have up to 4½ hours. Or perhaps it could be 6 hours for intra-arterial TPA. There are other treatment options available, but on the shelf exam they pretty much stick to the 3 hour time window. This is 3 hours from the last time you know that they were normal, so if the patient wakes up with a stroke, and it has been more than 3 hours since anyone saw them normal, you have to assume that they are out of the TPA time window. So the duration of the event is certainly critical whether or not you're going to give TPA. Now, you also need to be careful, if the patient had the stroke right there as a witnessed event, don't assume that you have 3 hours to give the patient TPA. It is really critical that you get the brain perfused as quickly as possible. The duration of the event is certainly critical, but before you administer someone TPA, you have to get imaging, ideally a CT scan without contrast to make sure there is no evidence of hemorrhage. When you see a patient and you are evaluating them for a stroke, it is critical that you record an accurate history and examine the patient to determine whether or not it looks like a stroke. Please don't rely on imaging to prove it was a stroke, because actually if you can already see a stroke on the admission CT, either the stroke occurred a while ago, or perhaps it's a very large stroke, either way there is an increased risk of hemorrhage if TPA is administered. The primary reason for getting imaging in a stroke evaluation is to make sure there is not a contraindication to administering TPA.

Now say the patient is not a TPA candidate for whatever reason, one might be interested in trying something like anti-coagulation, putting the patient on heparin. If you use evidence-based-medicine, however, you will see that there is no increased benefit with the anticoagulation, just increased risk of hemorrhage. So the usual order of business is if the patient is not a TPA patient for whatever reason we generally put those on patients on an antiplatelet agent, often aspirin, however an argument could be made for the use of Aggrenox or Plavix. There are indications for anticoagulation in situations like atrial fibrillation, a low ejection fraction, an anterior wall aneurysm, mural thrombus, pulmonary embolus, or DVT. There are times where you would want to anticoagulate a patient but it is not a routine thing in ischemic

stroke. It is also critical you discuss the risks and benefits of anticoagulation with the patient and family because there is a risk of hemorrhage. It is always tricky having a patient with atrial fibrillation with a subtherapeutic INR, and they just had a stroke. You don't want to wait too long to anticoagulate them because you don't want them to have another stroke, but you also don't want to be too aggressive in their anticoagulation because you don't want them to develop a hemorrhage.

The maximum time for edema after a stroke is about 72 hours perhaps up to 5 days. So if the admission CT is negative, and a day or two after admission the patient becomes comatose with dilated pupils and upgoing toes, you have to be concerned with the development of cerebral edema rather than recurrent ischemia.

When seeing a patient with a stroke, it is important you consider the localization of the stroke. If a patient has a sudden onset of symptoms like dysarthria, dysphagia, and diplopia symptoms, we refer to these as bulbar symptoms. The brainstem is like a bulb. If the patient has acute onset of bulbar symptoms you have to think of a posterior circulation or brainstem vascular event. So if you see a patient who has a carotid bruit and acute onset of bulbar symptoms, then that carotid bruit is most likely an asymptomatic carotid bruit because the carotid supply is anterior circulation. They do this all the time on the shelf exam - They throw in a key phrase perhaps intended to throw you off, just to see if you are paying attention. So if you have an acute onset post anterior circulation event with anterior circulation carotid bruit, then that's probably asymptomatic carotid stenosis.

There are some small strokes, lacunar infarcts, that can occur in the posterior limb of the internal capsule, corona radiata, pons, cerebral peduncle, or medulla and some of the classic lacunar infarct syndrome symptoms are pure motor, pure sensory, dysarthria-clumsy hand, or ataxic hemiparesis. The most common cause of lacunar infarcts is hypertension.

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Patients can have vascular events other than strokes. Some of these include temporal arteritis or giant cell arteritis. With this condition, patients (generally elderly) experience unilateral headaches, jaw claudication, and temporal tenderness. They may have polymyalgia rheumatic as well, which is diffuse pain in the back, shoulders, and extremities that worsens with mild exertion. Temporal arteritis can also be associated with amaurosis fugax, which is a symptom of a curtain going over the vision of one eye. Usually the vision loss resolves but the patient can have permanent blindness or visual impairment as a result.

Other vascular events can be hematoma like an epidural hematoma which is, on imagining, a "lEns"- shaped "Epidural" hematoma where the sutures hold the blood into the lens shape. This may be referred to as "dead man walking." This is where the patient experiences head trauma and gets knocked out, and then there is a transient lucid interval followed by decline. Patient can also have a subdural hematoma which is "Sickle-shaped" "Subdural" hematoma that extravasates the entire length of the hemisphere, irrespective of the sutures.

Patients can also have subarachnoid hemorrhages, which is the most frightening cause of headache seen in the emergency room. This is where the patient has come in with the worst headache of their life, and it is critical to find out how long it took the headache to become the worst headache of their life. Migraines can certainly be uncomfortable for patients but they progress over time, and they grow in intensity as opposed to a subarachnoid hemorrhage where the patient feels the intensity of the headache immediately like a thunder clap. The appropriate imaging of choice for subarachnoid hemorrhage would probably be a CT scan without contrast. Because you are really looking for blood, a very quick diagnostic test such as a CT which is excellent at picking up blood would be the diagnostic test of choice. CTs are very sensitive picking up blood, however if you are concerned about the clinical presentation of the patient, and that they could possibly have had a subarachnoid hemorrhage and the imaging did not show blood, you could to a lumbar puncture. In this situation you would be looking for xanthochromia which evidence of blood that is broken down in the CSF as opposed to the presence red blood cells. Red blood cells could be introduced by the trauma during the lumbar puncture.

Another vascular consideration would be an anterior spinal artery infarct. In this situation the patient would present with acute onset of weakness of the legs, associated with diminished pinprick and proprioception in the legs, sparing vibration and proprioception, because there is sparing of the dorsal columns. This presentation in general would be in a patient with vascular risk factors such as diabetes or hypertension.

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Another vascular condition is subclavian steal syndrome. This is where there is stenosis or occlusion of the subclavian artery with retrograde flow in the vertebral artery due to proximal stenosis. So the arm on that affected side is supplied by blood flow in retrograde direction down the vertebral artery at the expense of the vertebrobasilar circulation. So at rest, the blood pressure is lower in that affected limb, and that limb can be cool to the touch. With use of that extremity the patient experiences light headedness, and prolonged use can develop weakness in the extremity, with resolution of all of the symptoms after 10-15 minutes of rest. On the shelf exam they may ask you about the diagnostic test of choice, and that would be an angiogram, whether that be an MRA, CTA, or angiogram.

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The most emergent and serious headache that you would encounter is the evaluation of a patient with subarachnoid hemorrhage, but a common severe headache would be a migraine headache. Migraine headaches are typically unilateral, although they can be bilateral. They are usually described as throbbing pain. It can be associated with aura, some sort of visual symptom like scintillating scotomata, they can have photophobia and/or phonophobia, nausea, or vomiting. These can be severe headaches but the pain progresses over time and is not usually maximum at onset. As an abortive treatment, the first-line agent would be a triptan such as sumatriptan. On the shelf exam you'll probably choose sumatriptan as an abortive rather than dihydroergotamine which we generally just use to break a headache cycle. You can also use prophylactic medications such as beta blockers, calcium channel blockers, anti-epileptics

(like Topamax), and tricyclic anti-depressants. I would be cautious about using tricyclics with elderly patients, however. So if the patient is only having one or two headaches a month you would probably want to put them on sumatriptan as an abortive treatment. As an aside, I've had students that say "Well, I take Excedrin migraine for my headaches" but on the shelf exam the abortive treatment of choice is the sumatriptan. If the patient is having daily headaches you would probably want to give the patient an abortive as well as a prophylactic to reduce the severity and the frequency of their headaches.

The next headache to be discussed is a tension headache. These are typically more of a pressure type pain or band-like sensation around the temples especially but sometimes involving the back of the neck, occipital head region. Tension headaches typically respond better to NSAIDS than do migraine headaches. It is interesting that when a tension headache grows and escalates perhaps into a daily headache it can take on the throbbing characteristic like a migraine. If you have a patient in whom you are treating for migraines, and they are not responding to the migraines medications, take a step back and find out when the headache begins, does it start out as a pressure or throbbing. It could be that tension headaches are taking on migrainous features. A lot of times in these situations, the patients have tightness in the neck and may have a component of a cervicogenic headache, or headache that is originating from the neck. I often use physical therapy or neck injections for the treatment of those tension headaches.

Another type of headache is a cluster headache. These are usually short-lived and very severe while they last. They are usually less than an hour, often less than 30 minutes. They are most common in men, and they are associated with strange autonomic symptoms like unilateral ptosis and meiosis, scleral injection, and nasal drainage, and other autonomic features just unilaterally. The ptosis and meiosis should raise consideration for something like Horner's syndrome, but the cluster headache would not have the associated anhidrosis. If you have somebody that has pain and symptoms of Horner's syndrome but it is lasting for more than 30 minutes, then you start wondering about something like a carotid aneurysm or something involving the carotid. In a cluster headache, those autonomic symptoms should subside as the headache goes away.

Another headache to be considered is pseudotumor cerebri. Often these patients are young, obese females, although this is not always the case. What should alert you on their exam would be papilledema which is indicative of increased intracranial pressure. When you are evaluating headache patients it is really important to do a funduscopic exam to make sure there isn't anything emergent like papilledema. Pseudotumor cerebri often have associated transient vision loss, particularly when leaning down and standing back up. On the shelf exam they often use other terminology than to which you are accustomed. Instead of pseudotumor cerebri, they might call it "idiopathic intracranial hypertension". They do that for other diseases as well like for optic neuritis, they might call it retrobulbar neuritis, and for ALS they call it motor neuron disease. So, if you are reading a vignette about a patient and look down at a list of options and don't see the name of the condition you are looking for, look at the rest of the options and see if there is an answer similar to the one you are looking for. In pseudotumor cerebri they have papilledema which indicates increased intracranial pressure so it is really important that you do

a diagnostic procedure to identify the increased pressure prior to doing something like a lumbar puncture. You need to do imaging before to make sure it isn't "tumor cerebri" instead of pseudotumor cerebri. We often get an MRI scan (because it gives more detailed information about the brain, particularly the brainstem and posterior fossa) to make sure that something negative wouldn't happen like herniation if you were to do a lumbar puncture. The normal opening pressure is 10-20 cm of water or 100-200 ml of water so if you have a patient that has evaluated intracranial pressure, you have to be concerned. When you are doing the lumbar puncture, you need to make sure that the patient is lying in the lateral decubitus position because if you check the opening pressure and the patient is upright you will have falsely elevated intracranial pressure. So the diagnostic test of choice for pseudotumor cerebri would be lumbar puncture for opening pressure. There are a lot of causes of pseudotumor cerebri such as metabolic conditions and endocrine issues, but also too much vitamin A (hypervitaminosis A). You can also see this with the use of Acutane (isoretinoid) which increases the vitamin A levels. You can see pseudotumor cerebri in pregnancy and other conditions like polycystic ovary syndrome, venous sinus thrombosis, medications, certain antibiotics, if they discontinue steroids that have been associated with development of pseudotumor cerebri, so it's really important to take careful medical history in such patients.

We talked about temporal arteritis earlier and this would be patients that are elderly, associated with amaurosis fugax, jaw claudication, sensitivity to the scalp, pain combing their hair or wearing glasses, and these patients usually have an elevated sed rate. In 90% of patients that ESR is greater than 50. The diagnostic tool of choice for temporal arteritis would be a temporal artery biopsy. If you see an elderly patient that comes in with headaches you need to consider something like temporal arteritis. If it's a new onset of the headache later in life, it will be less likely to be a migraine. Most migraines occur for the first time before the age of 40.

Another consideration for an elderly patient who comes in with a headache would be a tumor. It is interesting that a lot of our patients are concerned about their severe headache could be because of a tumor, but actually most structures in the brain are not pain sensitive. If there is something involving the meninges, the dura, cranial nerves - those can cause pain, however most brain tumors are actually painless. If you have a patient who has a headache, particularly a new onset headache that's progressive with neurological findings you have to consider a tumor.

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When considering the differential for weakness, weakness can occur anywhere in the neuro axis. So, if there is a problem in the brain, spinal cord, motor neuron, root, plexus, peripheral nerve, neuromuscular junction, or muscle, weakness can happen anywhere along the neuro axis. What helps you distinguish what is causing the weakness is the history as well as the exam which will help you localize. If there are any associated symptoms or associated sensory loss or reflex changes or whatever that will help you distinguish where on the neuro axis it is occurring.

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Anything happening in the brain can certainly cause weakness. It can occur with a stroke, as we discussed earlier, or any disease process like a tumor, abscess, demyelinating lesion like in multiple sclerosis, or trauma.

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Anything involving the spinal cord can cause weakness. One of the most common things to consider would be a disc herniation or spinal stenosis. We can also see weakness due to a mass lesion or metastasis to the spinal cord, transverse myelitis, and syringomyelia. In medical school we learn that syringomyelia has a "cape-like distribution" of sensory loss, and this occurs when the syrinx expands in the center of the cord and pressing on anterior commissure, so you affect the decussating spinothalamic fibers causing pain and temperature to be affected, but sparing light touch. These patients might present pain, weakness and numbness in their arms and hands, atrophy in the hands, leg spasticity, and hyperreflexia. This is because the spinothalamic fibers are being affected in the arms causing the numbness in the arms and hands. You get the lower motor neuron findings at that level and upper motor neuron findings in the legs because of the cortical spinal tract involvement. These patients may also have thoracic kyphoscoliosis in syringomyelia. If you have a patient who has metastasis to the spine and it's causing neurologic symptoms it is important to give the patient IV steroids to prevent further neurologic damage. I would recommend that you coordinate the use of the steroids with the neurosurgeon who will perform the surgery because if there does happen to be lymphoma then the lymphoma will melt away with the steroid use.

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ALS can cause weakness, and on the shelf exam they usually call it motor neuron disease instead of ALS. The key factors that will help you distinguish ALS from other causes of weakness would be the associated upper motor neuron and lower motor neuron findings. We often think of patients with upper motor neuron findings as being spastic and hyperreflexive and lower motor neuron having flaccid hyporeflexive, but if they are already spastic and hyperreflexive, then how can they also be hyporeflexive. You have to think outside the box for other lower motor neuron findings such as weakness, atrophy, fasciculation, and muscle cramps. ALS does not usually have sensory involvement. That is not to say that an ALS patient couldn't have a little bit of peripheral neuropathy or a little bit of carpal tunnel syndrome, however the motor symptoms would be out of proportion to any degree of sensory loss. The onset of weakness in ALS patients is typically going to start distally and progress proximally, and it will start out asymmetrically so they may start tripping on their toes in the left foot, and then the left leg will start getting weak, and then they will start dropping things with their right hand, etc. It typically starts out distally and asymmetrically and progresses more proximally.

ALS patients can also have bulbar symptoms, like dysarthria, dysphagia, and dyspnea. ALS patients don't typically have visual symptoms so they don't generally have diplopia or ptosis. It is the respiratory compromise that ultimately leads to death of the ALS patient. It is really important when you are seeing a patient in clinic with ALS that you discuss with them what their

goals of care are because the reality is their respiratory function will likely decline, and if they were put on a ventilator they probably would not be able to get off. It is important to have these discussions before they begin having respiratory compromise because if in the middle of the night the ambulance is called because they are having respiratory distress, they are probably going to get intubated, and that may not have been the desire of the patient. So it is important to have this discussion with the patient and family early. One thing that I have found in clinical practice is that patients and family members often have an easier time dealing with the respiratory distress in an ALS patient with comfort and care measures, supplemental oxygen and use of morphine for air hunger, rather than making the decision of turning off the ventilator. Often times the family members have difficulty turning off the ventilator because they feel like they are killing the patient. It is also important, like with respiratory function, to also talk about nutrition of the patient because they will ultimately have difficulty consuming enough calories orally to sustain their needs. We often discuss with the patient the use of something like a PEG tube. A lot of times patients choose to have the PEG tube placed, but it is important to have the discussion before they develop respiratory compromise because if you decide to have the PEG tube placed when they are having respiratory compromise they may have to be intubated for the procedure. If you have the PEG tube placed before they actually need it, it's really just a maintenance issue. They can continue to consume orally as best they can but as they begin to have difficulty swallowing, they don't have to rely on oral intake for their caloric needs, and they can supplement with a PEG tube.

ALS is not all that common in clinical practice, so if a patient comes in with hyperreflexia in the arms and legs, sometimes doctors consider the possibility of something like cervical stenosis or lumbar stenosis. If you have a patient with hyperreflexia in the knees and ankles, although it could be a high lumbar lesion (because it would have to be above L4 to get the knees and thoracic lesions are very uncommon), the most common cause of hyperreflexia in the legs would be cervical stenosis. It may be that they don't have hyperreflexia in the arms because it's a low cervical lesion. Now, if they have hyperreflexia in the arms and legs, it would also be important to image the cervical spine, because maybe it is a high cervical lesion. It is not uncommon in ALS patients to have the hyperreflexia, weakness, and gait issues, as well as neck and low back pain because of the alteration in function. They can't walk as well, so this puts more strain on the back causing low back pain. So if a primary care physician checks an MRI scan of the lumbar spine, and they find a little bitty disc bulge, and the patient had hyperreflexia in the knees and ankles; one might be tempted to send the patient for surgery for treatment of that little disc bulge. It is really important review the imaging on your patient to look and see if that little bitty disc bulge really explains this progressive neurological dysfunction and hyperreflexia and low motor neuron findings and gait issues that the patient is experiencing, or is that just an incidental finding because the patient would probably not benefit from the surgery procedure.

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Weakness can be also be caused by a root lesion, so if you have a patient who has pain in the lower extremities with walking, you have to think about radiculopathy or claudication. Is it vascular or neurogenic? Considering radiculopathy, you have to consider disc herniation. The

majority of disc herniations occur in the lumbar region. 95% occur in the L4-5 region or the L5-S1 regions affecting the L5 and S1 nerve roots. The second most common site for disc herniation would be in the cervical spine. This affects primarily C5-6 and C6-7 affecting the C6 and C7 nerve roots. It is important when you are evaluating a patient with shooting pain from the neck down into the arms or the back down into the legs to consider radiculopathy. Have the patient put the arm in the anatomic position and see if the patient can tell you where exactly the pain goes. If the pain is only over the lateral aspect of the arm think about C5, if it goes into the thumb think C6, middle finger C7, 5th digit think C8, and medial aspect of the forearm think about T1. Sometimes having the patient show you exactly where the pain goes can tell you which nerve root is being affected. The same with the leg; L5 and S1 are the most common roots to be affected, so if the patient tells you the pain goes down the posterolateral aspect of the leg down into the foot, then ask whether the pain goes down into the lateral aspect into the foot (which is S1), or the medial aspect of the foot (which would be L5). The way I think about is that the big toe is bigger than the little toe, and 5 is bigger than 1, so the big toe would be innervated by the L5 nerve root and the lateral aspect of the foot would be innervated by S1. So in radiculopathy something is going to be abnormal on the neurologic exam, whether it be strength, sensation, or reflexes. We have already talked about the sensory distribution - If you have a patient who has an L5 radiculopathy you would expect to see a foot drop on that side where they would have weakness of ankle dorsiflexion and toe extension. If they have an S1 radiculopathy, you would expect the weakness of the gastrocnemius. So, if they stand up on their toes, they have trouble holding their foot up because of the plantar flexion weakness. The ankle jerk is supplied by the S1 nerve root, and the knee jerk is supplied by L4. So, in an L5 radiculopathy you would expect to see sensory loss over the medial aspect of the foot and ankle dorsiflexion weakness. In an S1 nerve root lesion you would expect to see a decreased or absent ankle jerk on that side, weakness of plantar flexion, and numbness over the lateral aspect of the foot.

Same with the arm - With the cervical spine you would expect to see weakness in the distribution of the dermatomal sensory loss as well as the reflex changes. So, if they have shooting pain from the neck down into the arm or back into the leg but the neurologic exam is completely normal, you have to think about joint pathology. If they have pain with palpation over a joint, you have to think about joint pathology such as hip pathology that can cause shooting pain down the leg. If they have pain over the hip or over the sacroiliac joints, or if they have pain with passive range of motion of that joint, if it hurts to lie on that affected side - Those are all indications of joint pathology. If they have a positive straight leg raise you need to think about lumbar radiculopathy.

In vascular claudication, if they have claudication affecting the calf you would expect involvement of the distal femoral artery, and if they have pain and claudication over the thigh and buttock region you would expect more diffuse aortoiliac involvement of the vascular claudication. So, in the evaluation of a patient with vascular claudication, you would primarily start with a Doppler of the lower extremities. I believe that would be the answer on the shelf exam - We certainly use a lot of ankle-brachial indices or ABIs but on the shelf exam the Doppler I believe is the answer.

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There are other back pain issues that arise on the shelf exam including ankylosing spondylitis. In this situation in the vignette they would describe the patient with decreased black flexion, elevated sed rate and decreased joint space in the bilateral SI joints. If they ask about the initial

treatment of choice, it would be an NSAID. Another back pain issue to be considered, in this case a neurologic emergency and neurosurgical emergency, would be cauda equina syndrome. In this situation the patient would have bilateral leg weakness, low back pain, urinary incontinence and retention, saddle anesthesia, and they would be found to have decreased anal sphincter tone.