[MUSIC PLAYING]

DOUGLAS LANSKA: The talk today is on what we can learn by looking, a clinical approach to gait disorders. And I'm going to be incorporating modern and archival video footage. Learning objectives, first will be to review the gait cycle, review the classification of gait disorders, recognize some common clinical features of gait disorders, and provide a historical perspective on neurological gait disorders.

So I want you to be aware of this gentleman, Eadweard Muybridge. He was born Edward Muggeridge. He was a British-American photographer. And he spent time at the University of Pennsylvania in 1885 compiling an extraordinary extensive collection of sequential motion series.

So this was before there were actual video cameras. He set up batteries of sequentially triggered single image cameras. And from those series of images, some of the most amazing collections of movement disorders were filmed before in fact, there were motion pictures. So we're going to look at him. And he's kind of a scary looking guy.

While he was off photographing, his younger wife was having an affair. And when he found out about the affair because she wrote the name of the other man on the back of their child's picture-- which he thought was his and wasn't-- he went to the house where this man was holding a party, introduced himself, and shot him dead. And this was in San Francisco at the time. And he was acquitted on the grounds that it was a justifiable homicide.

[LAUGHTER]

So just kind of interesting background on what you're going to see. He was hired by the then governor of California, Leland Stanford, because Stanford had made a bet. Stanford had bet that there was a time in a horse's gallop when all of the hooves were off the ground simultaneously. And at that time, it wasn't clear, because people hadn't imaged this. They all were looking at it visually, and they were all seeing different things.

And so some people believe that there was at least one hoof on the ground all the time, and some people didn't. Muybridge settled that and proved that, in fact, there was a time in the horse's gait when all the hooves were off the ground simultaneously. It was his sequential images which you see for one of Stanford's horses that were in fact the deciding factor.

So this is Muybridge himself in a sort of self-portrait, if you will. And he filmed himself walking up and down stairways. And you can see the recreations of those images and what they would look like as a movie now, or a GIF.

So this is one sequence of him walking on level ground, and we're going to use that sequence. I'm going to move some of that around just to give us a better idea of the gait cycle. So this is what I did, I moved the three last images to the front. And now we're going to look at the gait cycle.

Starting here, there's a period of double support. Then there's a period of single support with the right leg. Again, double support, very brief. And then single support with the left leg.

So the period from double support through single support and double support again with the right leg is the stance phase. It's much longer than the swing phase, which is the part when that leg is propelling itself forward off the ground. So there's two big phases to the gait cycle, the stance phase, the swing phase.

Stance phase is longer. Double support, singles support with that leg, and double support again before that leg moves forward. That whole thing is the gait cycle or one stride.

So let's look at this man's gait cycle. Watch the right leg. Double support stance. Double support stance, double support swing. Double support stance, double support swing.

OK, we're going to use that as we go forward. So we can classify gait disorders. There's different classifications. The one I'm going to use talks about low level, middle level, and highest level. And today, we're going to concentrate on some of the low level and a few of the middle level types of gait disorders.

So in lower level gait disorders, we're talking mostly mechanical aspects. So we have the arthritic and intelligent gaits, which I'm not going to cover today. Then we have gait disorders due to muscle weakness, gait disorders due to peripheral neuropathies, and then the sensory ataxic gait. And I want to show you all of those today.

We'll start with myopathic gaits and related signs. Those related signs include Gower's sign, Trendelenburg's sign, Trendelenburg gait, and the waddling gait. So in 1861, Duchenne described what is now called Duchenne dystrophy. It had been described earlier, but Duchenne is most famous for his description of this.

It affects young boys, so it's an X-linked disease. They develop pseudo hypertrophy of muscles, particularly the calf muscles as shown on your left. And on your right, is what's called Gower's sign, which William Gower's-- British neurologist-- described in 1879.

And Cathy is here, right? Cathy, where are you? Oh, Cathy-- who I was talking to before the talk-- does yoga.

Does anybody else here do yoga? No yogas? Oh my goodness.

Well, if you look at the positions that the child has to go through to stand, he goes through a yoga tabletop position, a yoga down dog position, and then pushes off with his hands to provide enough oomph to stand. So let's watch a patient with Duchenne dystrophy try and stand. So all fours, table top, down dog, and then push off. He uses the same push off to stand from a chair.

Now watch this child get up. Video's having troubles. So table top, down dog push off with the legs. I am sorry that doesn't show well on the way this computer is processing it.

But because of the weakness, they have to use that very complicated procedure. You can find many patients with other myopathies as children or as adults who will use exactly that process. So that reflects proximal weakness, and they have to use these rest positions from yoga to basically get themselves from the ground to standing.

Another feature of myopathic gaits is the Trendelenburg gait, and reflects weakness of the gluteus medius muscle. What's the gluteus medius muscle do, somebody? No volunteers? I'm sorry?

Hip abductors.

Hip abductor, terrific. So hip abduction. So in the absence of hip abduction, if you're in the stance phase standing on your left leg, what's going to happen to the right side of the hip? If you can't abduct the left hip, and you put your weight on that leg, what's going to happen on the right?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: Who said that? Strong work. So that side will drop. That's a Trendelenburg sign.

So let's first talk about the normal walk. So Friedrich Trendelenburg was a German orthopedic surgeon. And he said in a normal person, the whole body inclines a little alternately to the right and the left, always toward the side of the foot contact. This leaning occurs to bring the center of gravity vertically over the support surface, the sole of the foot of the standing leg, the stance phase. That the pelvis remains horizontal and does not sink on the side of the walking leg is due to the effect of the adductors of the joint, like that gentleman said.

So the Trendelenburg sign is what we were hearing back here by Gigi. The left hip abductor, the gluteus medius weakness, or with congenital hip dislocation, standing on that leg, the pelvis will tilt downward on the opposite side. That's shown on the left. So if your hip abductors are weak, they can't elevate the hip. And so that hip will drop, because the adductors are weak.

On the normal side, the adductors are strong, and the pelvis will not drop. In fact, it may elevate slightly. So let's watch this lady who has a Trendelenburg gait. And there will be an important clinical sign called the arrow sign that will appear momentarily. Putting weight on one hip, and the other side is dropping because the abductors are weak.

There's another way that patients may compensate for that type of proximal weakness. It's called the waddling gait, because Duchenne thought that the gait resembled that of a duck. It's also called the Duchenne-Trendelenburg gait, a Trendelenburg lurch, an abductor lurch or a gluteus medius gait.

So Duchenne said in 1861 a patient with Duchenne dystrophy walks with a lateral inclination of the trunk toward the limb, which is resting on the ground. The word waddling perfectly expresses his mode of walking. So if you are walking and your left abductors were weak, either the right hip would drop, or you could thrust your whole trunk over to keep your balance. And so many patients with myopathic weakness, they'll have hip girdle weakness, shoulders girdle weakness. Instead of dropping that side and doing what I was once told was a Marilyn Monroe walk.

We need somebody else to demonstrate that, trust me. Instead of doing that, they may do this, and thrust their whole trunk over to keep the balance, and try and avoid the issue of that dropping hip and the dragging leg. So Trendelenburg is usually the one who's given credit for this. He described it more than 30 years after Duchenne, but he was smart enough to include a picture in his clinical report.

That's an important tip. So he wrote, the pelvis drops toward the walking side, the swing phase, and the trunk is sharply bent toward the standing side, the stance phase, in order to restore the balance. So watch what this lady does with her trunk. Shift, shift, shift. So that is a waddling gait.

Now let's talk about peripheral neuropathic gaits. If you have a foot drop on one side, you will have a semi steppage gait. If you have a foot drop on both sides, you'll have a full steppage gait. Some people call this a foot slap gait, a drop foot gait, and equine gait, meaning resembling a horse. And we'll talk about back knee in a bit.

A foot drop can occur from various different causes, most common being an L5 radiculopathy, a peroneal neuropathy, or if bilateral, a peripheral neuropathy. So I see this very commonly in diabetics. So watch this man. I want you to notice and tell me which side is abnormal, right or left?

I heard right and I heard left. Watch again. OK, how many for right? How many for left? How many on the right want to change their opinion? OK.

The majority said it was a problem on the left. Can I have somebody tell me why there was a problem? And then we're going to look at it together again. What was wrong with it?

AUDIENCE: His foot was slapping down.

DOUGLAS LANSKA: Foot slapping down. OK, excellent. Anything else? Let's watch it again. Oops, and we went too far.

OK. The right foot, the heel hits first. The left foot, which hit part hits first? Which part hits first on the left? Yeah, a lot farther forward, right?

So in a normal gait, the strike should be with the heel, right? So the strike in the left was way far forward. It might have been the ball. It might have been whatever.

And it looked like he was hitting the left foot on what part? The medial part, the lateral part, flat, straight away. Which do you think? Watch again. Flat, medial, or lateral?

Lateral. So what you're observing is he's hitting the foot farther forward than the heel. And he's hitting the outer aspect first, OK? Now what his knees. What can you tell me about his knees?

AUDIENCE: Hyperextends.

DOUGLAS LANSKA: I didn't hear you.

AUDIENCE: Hyperextends.

DOUGLAS LANSKA: Hyperextend is perhaps not the right word. You may mean the right thing, but I don't think you're using the right word.

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: He does straighten it to slap it. You're right about that. There's one more observation I'd like you to make. In the back?

OK, let's try it again. Watch his knees. Which one gets up higher?

AUDIENCE: The left one.

DOUGLAS LANSKA: The left. Why do you think he has to get the left leg knee higher? To compensate for the dropping foot. So the things I wanted you to observe you did very well with.

So he has to lift his left knee higher. He strikes farther forward than the heel. And he strikes with the lateral aspect of the foot. Excellent.

So now let's watch one more guy. And there's another important sign that will appear if this plays right. This is the line sign.

So which knee gets higher? Left. Which foot is striking with the heel and which is not? So which foot is striking with the heel?

AUDIENCE: The right.

DOUGLAS LANSKA: The what?

[LAUGHING]

Somebody just playing with me back there? Which foot is striking with the heel?

AUDIENCE: Right.

DOUGLAS LANSKA: Which foot is striking in front of the heel?

AUDIENCE: Left.

DOUGLAS LANSKA: Which knee gets higher?

AUDIENCE: Left.

DOUGLAS LANSKA: Good. Strong work. All right, so now I want to tell you a very brief history of cinematography, because it will bear on what we see next.

So there was no technology to do multiple sequential images before the 1870s. In the 1870s, pioneering efforts were made-- like you saw with the horse-- to do multiple sequential images to give an impression of sequential motion. Those were all done with arrays or batteries of sequentially triggered single image cameras.

After 1880, we had further developments with celluloid roll film, single cameras able to take multiple sequential images on a single roll of film, and mechanical projectors synchronizing film and shutter motion. So until 1880s, no motion pictures per se were available. And then we had this collaboration in 1885 with the murderer Muybridge and a young, energetic, enthusiastic, fresh out of the box academic neurologist at University of Pennsylvania, Francis Durkheim.

So they employed these arrays to film patients of Durkheim's and his colleagues with various neurological disorders. They filmed 21 subjects. Here's an example plate of a man who'd had probably a what?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: I'm sorry?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: What kind of clinical issue did this gentleman have?

AUDIENCE: Stroke.

DOUGLAS LANSKA: Stroke. Where? Right or left? Right. Left.

So we got half and half. That's not good. So which side is his weakness on?

AUDIENCE: Left.

DOUGLAS LANSKA: Which side is his stroke on?

AUDIENCE: Right.

DOUGLAS LANSKA: There we go. Good. And if we had to bet, we would guess he had a stroke in what vascular territory?

AUDIENCE: [INTERPOSING VOICES]

DOUGLAS LANSKA: OK. So this is one example plate. So he had a series of single image cameras just taking sequential pictures to give us this from the side and from the front. So here's one of those. What's wrong with this man? And I have to double click it, because of this issue here.

What's wrong with him? Which heel strikes first, right or left? I mean, which strikes-- nah, I said that wrong. You know what I was asking.

Watch the heels. The right hits clearly. The left, he's coming down with the toes down. So what has he got?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: A foot drop on the left. Some lady in the back, bless you. Good work. Foot drop on the left.

Which knee went higher?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: Yeah, that's what I want you to see. Bang, did you see? All right.

So when there's quadriceps weakness-- we're going to move on now. When there's quadriceps weakness, what will happen if someone tries to stand on the leg with the quadriceps weakness? What's likely to happen? Help me out.

Whereas the quadriceps? Nobody? [LAUGHTER] All right, quadriceps.

Big muscle in the front of the leg. If it's weak and you're trying to stand on the leg, what's going to happen? OK.

So if someone has quadriceps weakness, what could they do to compensate? They can try and force their leg back and let the ligaments handle the weight. So let the structural elements rather than the muscles handle the weight. That's what they do.

And when they do this repeatedly, they actually remold the knee and cause damage to the knee joint. And they develop what's called genu recurvatum. So hyperextension deformity of the knee, back kneeing. It's a lot easier to say back kneeing.

Let's watch this man. He's got quadriceps weakness. Look at his right knee as he walks. Notice the angle. He's putting his knee backward to help stabilize that leg.

He's doing it on both sides. So that kind of backward curvature of the knee is not normal in somebody walking. Let's watch this child.

What do you think about the right knee? Have courage. What about the right knee?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: Terrific. Do you see it? I mean, I heard you say it, but do you actually see it? That's what I want to make sure.

AUDIENCE: Yes.

DOUGLAS LANSKA: OK. Now, you don't normally see quadrupedal gaits past toddler stage, certainly. This child had polio. And prior to the polio vaccine, 1950s, about 3% of polio survivors wound up requiring-- they were left in a position of disability that required them to ambulate on all fours. So something we don't see commonly anymore, but is seen in other countries with residual of polio and other things.

Let's talk about the Romberg sign. So loss of postural control due to impaired proprioception is often attributed to Maritz Romberg, who in 1851, reported this in his textbook. And he said he had noticed this all the way back to 1840, because he wanted to have priority over somebody he knew who would described it. But he hadn't kind of done the retrospective far enough, because this is actually described in Marshall Hall in 1836. So if you're going to try and claim priority, at least do it in a way that covers the whole thing.

So Marshall Hall was the man who formulated the concept of a spinal reflex arc in his lectures on diseases of the nervous system in 1836. But he also described loss of postural control and darkness in a patient with severely compromised proprioception. Maritz Romberg though used that information to develop a clinical test. And that was to demonstrate the loss of control in darkness in patients with tabes dorsalis. So what's tabes dorsalis?

AUDIENCE: Neurosyphilis.

DOUGLAS LANSKA: I'm sorry? I think I heard neurosyphilis. It's a form of neurosyphilis that we see rarely now.

But it was important, because it demonstrated really to clinicians of that era the intense amount that we rely on the proprioceptive system. Because it affected that particularly. Though also resulted in things called lightning pain, which was very devastating.

So in 1851, this is how he described the Romberg sign. If a patient with tabes dorsalis-- or we will just say proprioceptive impairment-- is told to shut his eyes while in the erect posture, he immediately begins to move from side to side, and the oscillations soon attain such a pitch that unless supported, he falls to the ground. The eyes of such patients are their regulators or feelers. Consequently in the dark, and when amaurosis or blindness supervenes, as was not unfrequently the case, their helplessness is extreme.

So he also described the tabetic gait. And I want you to pay attention to that description and think about it in terms of Romberg sign. The gait of a tabetic begins to be insecure, and the patient attempts to improve it by making a greater effort of the will. As he does not feel the tread to be firm, he puts down his heels with greater force. The insecurity of his gait also exhibits itself more in the dark.

So if we're trying to stand in the dark, or trying to stand steady with our eyes open, that's called station. Station is the relative power to stand steady with eyes open or shut. This can be made numerically accurate by standing the patient in front of a sway meter, like this one.

And you can see this with S Weir Mitchell at the orthopedic hospital in Philadelphia. Standing in front, you can see in the back on the right that sway meter right there. And here's William Osler, and Guy Hinsdale is on the left.

Here's something called an ataxiagraph. So we have a man standing with a pointer on his head under a smoked piece of paper, and he's etching out the path of his sway as he's trying to stand steady. What you see on the right is a negative of such an image. At the top is a normal sway. and on the bottom is the sway of a patient with tabes dorsalis. So markedly impaired postural control due to impaired proprioception.

What's important for us today, 2017, is to know that Romberg sign is not specific. But it's most closely associated with proprioceptive sensory loss. You can either have this as a result of profound loss of proprioception, uncompensated unilateral vestibular dysfunction, or bilateral dysfunction. One of those substrates plus loss of vision, including closing your eyes or darkness, will result in Romberg's sign, and increased falls and sway. Somebody with postural impairment-- particularly with diabetic neuropathies-- will fail that right away.

So let's use that now and look at the sensory ataxic gait. The sensory ataxic gait results from proprioceptive impairment, sometimes called a stopping gait. Patients may lift their legs very high on the ground and stomp them hard to help with the sound to compensate for what they've lost for proprioception. As a result, they may damage their joints. They may develop something called Charcot joints, or neuropathic arthropathy.

So on the left, there's some of those pictures from Muybridge. On the right are some images of Charcot joints in a tabetic patient. And what you can see at the bottom right is what the spinal cord looks like when the posterior columns are damaged as a result of neurosyphilis.

So this man is walking fairly normally. Let's click it again, watch him. This man has neurosyphilis-- tabes forsalis, that form-- and he's walking with his eyes open. Now let's watch him walking with his eyes closed. And if you notice on the right, there's an arm coming, because he's going to drift out of the camera's aim here.

So that marked difference, which you can obviously see, is simply because he closed his eyes. And I see this almost every other day at least in diabetic patients. And when you ask them how they take a shower, they say, doc, it's really hard. I've got to lean into the corner of the wall and kind of wedge myself in, because as soon as they close their eyes to wash their hair, they're going to go down, and they know that. And they've figured out a lot of times long before their doctors will.

So let's look at them side by side, him walking from the side. And on the left is him walking with his eyes open, and on the right with his eyes closed. So that is a sensory ataxic gait. And it's important for you to recognize that. And if you do, you will have gone a long ways in understanding what is wrong with the person.

Now, William Osler, great internist. Spent time at the University of Pennsylvania before he went to England. He had a lot of aphorisms, but he said this one that's relevant to this sensory ataxic gait. The normal man walks by faith, the tabetic by sight. And I daresay you won't see very many tabetic patients anymore, but your diabetic patients this will be true of as well.

So key clinical features of the sensory ataxic gait. Gait ataxia with eyes closed, relatively normal gait with eyes open. Romberg sign will be present. And they will have impaired joint positions sense when you test it.

They may also have some other things that neurologists would find fascinating. Pseudoathetosis and a sensory drift, as opposed to a pronator drift. So if you put the arms out for a pronator drift test, if they have subtle weakness, they'll have flexion and pronation. But a sensory drift, the arm won't do that. It'll just wiggle around or maybe drop.

That's a sensory drift. And when you notice that, don't call it a pronator drift. It's not a sign of weakness. It's a sign of impaired proprioception.

So there's a large differential of a sensory ataxic gait, and it's important to recognize some of these things. Certainly came to attention with neurosyphilis, but B-12 deficiency, copper deficiency, myelopathy, various sensory neuronopathies or ganglionopathies, including toxic ones from cis-platinum and Paradoxine. Peroneal plastic ones with anti-Hu antibodies. Sjogren's syndrome, celiac syndrome, various other strange and rare disorders, and also various peripheral neuropathies. Please notice that and recognize it, because it will help you a lot.

Let's move on now to a middle level gait disorders. We'll talk about those that affect the central nervous system, particularly spastic and Parkinsonian gaits. So spastic diparesis was described in 1853 by a British orthopedic surgeon, William Little, who had himself been plagued by a clubfoot in childhood, and was seriously disabled by that. He's the one who linked prematurity and neonatal asphyxia with motor and mental impairment. And he-- because of his disclosable description-- spastic cerebral palsy was known as Little's disease for many decades.

These are pictures from his monograph. And what I want you to notice on the left is the standing on the toes posture with the knees flexed, the hips flexed. Same on the right, but also the legs are internally rotated. So some of the features of a spastic diparesis are this kind of triple flexion at the ankles, knees, and hips, and internal rotation of the legs as a result of that spasticity.

So what Little wrote was, the soles of the feet are not properly applied to the ground. They're toe walkers. The knees always incline inwardly, and continue bent. In the best recoveries, the gait is shuffling, stiff. Each knee, by forcible spastic rubbing against its fellow, obstructs progression, the so-called scissoring gait.

William Osler, when he was at University of Pennsylvania also described this, and drew a picture of a child walking with a scissoring gait. And you'll notice that the feet are actually crossing the midline. So here are some track patterns that he drew of different patients with a sensory ataxic gait. And for those of you with better vision than mine, I hope you can read the letters. Right foot, left foot, right foot, left foot across the top.

What can you notice about the bottom track? Somebody help me out? So normal or not normal?

AUDIENCE: Starts out crossed, but then crosses left.

DOUGLAS LANSKA: OK. Well, first thing you notice was that the legs actually cross. So you'll see the right foot to the left of the midline, right? That ain't right.

Is the whole foot displaying? Do you see a track pattern of the entire foot? Yes? No? And we got a lot of abstainers.

OK, so there's no heel for most of these. There's no heel. It's the front of the foot.

What else can you notice? I want you to notice one other thing. So we got crossing of the midline. We are predominantly displaying the front of the foot in the track. What one more thing can you notice about those tracks?

AUDIENCE: The angle of them.

DOUGLAS LANSKA: I'm sorry?

AUDIENCE: The angle.

DOUGLAS LANSKA: The angle, excellent. What about the angle?

AUDIENCE: It's not straight.

DOUGLAS LANSKA: It's not straight. Which way is it turned?

AUDIENCE: Looks like it's internal.

DOUGLAS LANSKA: The feet are turned in. That was perfect. Those are the three things I wanted you to notice.

So let's watch this man, young man. Watch on the right, watch his knees. Are the legs straight, internally rotated, or outwardly rotated?

This is multiple choice with three choices. So are they straight, internally rotated, or externally rotated?

AUDIENCE: Internally.

DOUGLAS LANSKA: Internally rotated. Are the legs adducted or abducted?

AUDIENCE: Abducted.

DOUGLAS LANSKA: Abducted. So the knees are just kind of squeezing right around each other as he's trying to walk forward. Are the knees flexed or straight?

AUDIENCE: Flexed.

DOUGLAS LANSKA: Flexed. Are the hips flexed or straight?

AUDIENCE: Flexed.

DOUGLAS LANSKA: Good. Those were perfect. Now, I want one more thing out of you.

What's his diagnosis? Does he look right? I didn't hear you.

AUDIENCE: Looks like he has achondroplasia.

DOUGLAS LANSKA: Well, that's an interesting diagnosis. Not correct, but it's an interesting one. Why do you think he has achondroplasia?

AUDIENCE: [INAUDIBLE]

DOUGLAS LANSKA: OK. Shortish. OK, well, he's flexing. He's a young man.

Is there anything else that doesn't look right about him? What about his head? A funny shape for his head, yes. What do you think is wrong with his head shape?

AUDIENCE: Pagets.

DOUGLAS LANSKA: I didn't hear.

AUDIENCE: Pagets? Pagets?

DOUGLAS LANSKA: No, no, no, not Paget's disease. Don't give me a diagnosis yet. Just give me what's wrong with his head.

You're observing now, he's in your office. And you're observing, and you're trying to decide what am I going to do with this young man who comes to me with this funny gait? And you look at him and you say, my gosh, there's something wrong here. What is wrong?

AUDIENCE: Big crown.

DOUGLAS LANSKA: I'm sorry?

AUDIENCE: Big crown.

DOUGLAS LANSKA: Big, crown that was a great answer. So the top part of his head looks big. The bottom half doesn't. Does that help you with the diagnosis?

AUDIENCE: Hydocephalus?

DOUGLAS LANSKA: Who said that? Stand up and--

[APPLAUSE]

Yeah. So this poor young man had congenital hydrocephalus. So he's got the result of that here. But more importantly, he's got the impaired gait of a spastic diparesis. Excellent job.

So the signs that you can notice with a hemiparetic gait. Of course, we watched a spastic hemiparesis. That posture of middle cerebral artery classic picture that we know, it actually has a name. It's called the Wernicke-Mann paralysis, or Wernicke-Mann posture.

There are other features of the gaits that I'm going to want you to notice, and that's particularly circumduction. But you will also of course find on your examinations hyperreflexia in a Babinski sign. So watch this man. We saw his picture before as a plate. But now I want to watch-- I want you to watch him walk with this recreated sequence.

So we said he's got a hemiparesis on the left. So he probably had a right-sided cerebral stroke. And that stroke, probably in the middle cerebral artery territory. What do we notice about his posture? Is the left arm flexed or straight?

AUDIENCE: Flexed.

DOUGLAS LANSKA: Flexed. Is the left leg straight or flexed? Straight or flexed?

AUDIENCE: Straight.

DOUGLAS LANSKA: Straight. In fact, it's remarkably straight. He doesn't bend the knee as he moves it forward. So since he doesn't bend the knee as he moves it forward, what's he got to do?

AUDIENCE: Swing out.

DOUGLAS LANSKA: Swing it out. Which we call? Circumduction. Very good.

So he's also doing one more thing. So he's got a flex posture of his arm. He's got an extended posture of his leg, and he's got circumduction. And he's using his cane.

Which way is his center of gravity shifting in order to move his left leg forward? He's shifting his center of gravity where? To the right or left?

He's got to bring that left leg forward. It's hyperextended at the foot. Everything's extended. He's swinging it around. He's got to swing his center of gravity to his?

AUDIENCE: Right.

DOUGLAS LANSKA: Right. Very good. Beautiful job. Strong work. Parkinsonian gaits, features that we notice, stooped posture, camptocormia flexion at the hips, shuffling gait, festination, and something called marche a petit pas, gait with little steps.

This is the drawing that Gower's did of a Parkinsonian posture. This is one that a colleague of Charcot's did of a person with progressive Parkinson's disease. And this is a sequence of pictures from Durkheim-- who collaborated with Muybridge-- showing the progression of Parkinson's disease.

Now there's two different things you can notice in different kinds of Parkinsonian gaits. One is on your left, which is that flexed posture, which is characteristic of Parkinson's disease. And on the right, that more extended posture, if you notice that, that will be a Parkinson's-like disease, but that won't be Parkinson's disease. So the so-called Parkinson's plus diseases will have that characteristic posture.

OK, I want you to tell me, which one of these astronauts had a problem? A neurologic problem. So this was from astronauts going up in a shuttle launch. One of them has a neurologic problem that I want you to diagnose. Any guesses?

One more time. OK, here we go. You're going to-- I promise, you're going to do better than the NASA doctors on this one. Second on the right of the parade? Is that what you mean?

Yeah. So what was wrong? Of those several people who recognized the problem, what was wrong with him?

AUDIENCE: He didn't swing the right arm.

DOUGLAS LANSKA: He didn't swing the right arm. Strong work. OK, that was Rich Clifford who went on three space shuttle missions in 1992, 1994, and 1996, and had Parkinson's disease. And he knew he had Parkinson's disease, and he still wanted to go fly. So he tried to hide it from the NASA doctors and was successful.

I heard him talk at the American Academy of neurology meeting. That's my son on the right, my son John. And at the time, my youngest son wanted to be an aeronautical engineer.

So he asked Rich Clifford if he would sign something to the younger one, because he wanted to be an aeronautical engineer. And he wrote, to James, go for launch. Rich Clifford.

So as I mentioned, there are different kinds of small step gaits. The Parkinson's disease small step gait has a stooped stance and a decreased arm swing, like that young lady astutely observed. There's also a different kind with an upright stance, and an often normal arm swing. Those will be Parkinson's-like diseases that you can separate if you just pay attention.

So the last classification, last category-- we're not going to go into it, I just want to let you know what it is. It's a disruption of selection of appropriate postural and locomotor responses, often called the senile gait. So it's supposed to be the higher level of control of the lower central nervous system components that we use to do our normal gait. So with that, I leave you with some little clips of abnormal gaits from 1889. And Gheorghe Marinescu. And I thank you for your kind attention.

[APPLAUSE]