

SPEAKER 1: Hi, everybody. Thank you for coming. It is my pleasure to introduce Dr. Carolyn Lacey. Dr. Lacey went to medical at Drexel in Philadelphia, did her residency here at Travis, cardiac fellowship at Walter Reed, and has been back at Travis for the last five years. She just joined John Muir this past September. She has an interest in women's cardiovascular. So welcome, Dr. Lacey.

CAROLYN LACEY: Thank you. Hi. Thank you for giving me the opportunity to talk. I'm going to be talking about syncope today, one of my favorite topics. Woops.

SPEAKER 1: [INAUDIBLE].

CAROLYN LACEY: Or not so much, stink-ope. And I think that a lot of people feel this way about--

[SIDE CONVERSATION]

CAROLYN LACEY: So stink-ope, I bet a lot of people feel this way, because it's kind of a nuisance, right? So it's very common. 1% to 3% of annual ER visits are related to syncope. 6% of possible admissions are for syncope, with a median length of stay up to 5 and 1/2 days. And up to 50% of our patient population may experience syncope. And so some of the numbers are skewed, because there's a large enough group of patients who will experience syncope and then never actually come to be evaluated.

And it costs a lot of money. These are from ESC guidelines for 2009, up to \$2.4 billion a year. And that's mainly driven by the hospitalization costs. And these patients may be seen by lots of specialists, up to three specialists. They might have nine diagnostic tests. They may have very disabling symptoms. And it may be their only warning sign. One episode of syncope may be their only warning sign before they have sudden death.

So just to give you a little background on the epidemiology, about 15% of children before the age of 18 will have their first episode of syncope. In the male population, between 40 and 59 years old, it's another 16%, women 40 to 49, 19%. As people age, there's another bump in how many species are experiencing syncope. So in the over 70 population, it will be up to 23% within a 10-year period, over the age of 70. And if someone experiences syncope, they have a third ep-- they have a third chance of having a recurrence within three years.

And the problem with syncope is that syncope is a symptom. It's not really a diagnosis. And there's a lot of different conditions that can cause syncope. And the problem is there's no gold standard for diagnosis. Some of the things that are nice in cardiology is we have gold standard tests. If you have a patient that sounds like they have coronary disease, we can do a cath and say, you have coronary disease, or you don't. But for syncope, that test doesn't exist.

And because there are so many things that can cause symptoms and so many different tests that can be done, what a lot of patients like to call sort is the process of the evaluation. I'm not suggesting that the shock on approach will not go away after hearing my talk, because I think a lot of patients still don't have a good reasoning for why they have syncopes. Or we might not find a true diagnosis for it. So one more time, can you talk a little bit about what sort of test [INAUDIBLE].

SPEAKER 2: [INAUDIBLE] Thank you.

CAROLYN

LACEY:

So just as a little bit of a review, so when people are standing-- so in order to maintain normal attention while patients are standing, when you stand up, 30% of your blood volume, of the central blood volume, can decrease, can go down into the dependent body parts within seconds. And then you get some shifts over the next 30 minutes. And that can further reduce your blood volume by 5%.

And so we have compensatory responses that help prevent that from happening-- so whether it be muscle pump, neurovascular compensation, or even some local vascular compensation mechanisms. And so what we'll talk about is a little bit of an initial evaluation, immediate causes for syncope, some tests, and some therapy. All right.

So and when you're first seeing a patient, and they come in and say, Doc, I passed out, the first thing you want to answer is, is it syncope? Is it truly syncope? And so there are a number of things that can cause loss of consciousness that aren't truly syncope.

And so some of the questions to answer-- did the patient have a complete loss of consciousness? Was the loss of consciousness, was it transient? And did it come on rapidly, and did it last for a short period of time? Did they get better on their own and completely resolved and without any real sequelae. And was it associated with a loss of postural tone? So all of those things together go into it was syncope, for the diagnosis of syncope.

And so once you've said, yes, we've had syncope, then you want to know if you can de-- then your next step is, can you determine an etiology based on your history? And then, based on your history, is there any evidence for high risk of a cardiovascular death? So you're trying to figure out and re-stratify the patient at the same time, just while you're taking a history and physical exam. So the history is so important, and we'll talk about some historical features in a few minutes.

On your exam, when you're doing an exam, it really should include orthostatic blood pressure measurements. That's so important. That's more useful in the patient who had an episode of syncope and now, all of a sudden, is right there. So it may not necessarily be as useful in the office setting, but potentially could be in a patient that has a lot of orthostatic hypotension. And then you want to get an ECG. And based on those things, then that's the next place to help you-- your next branch point to help you determine what other testing you need to do.

So some high risk features that you want to look for when you're seeing a patient is, do they have evidence of structural heart disease? And so we have heart failure. Do they have a history of low EF? Or have they had prior myocardial infarction? And then, when you are looking at their ECG, you want to see if they have evidence of having arrhythmogenic syncope, so some historical features. They were exercising, and they passed out while they were exercising.

So that history is sometimes sort of difficult to tease out, because you want to-- patients will say, I was exercising, and then I passed out. But then, when you talk to them a little bit more in-depth about it, they'll say, well, I was really done. I had gotten-- I had just stepped away and just finished. And then a few minutes later, after I had finished exercising, I passed out.

But if they were running down on the ground, that's a high risk feature. Or if the patient was supine when it happened, that would be very concerning for arrhythmogenic syncope. Did they have palpitations beforehand? Is there a family history of sudden death? So family history is one of those things where-- in cardiology, when we're talking about coronary disease, we only really, really worry about first degree family relatives. But in patients, when we're considering sudden cardiac death, a lot of those, a lot of the arrhythmogenic syncopes, like QT or Brugada or something like that, can have much more delayed penetrants.

And so you ask about the more extended family. Has anyone died suddenly, other common things that go along with dying suddenly? Dying in a car accident, drownings, seizure disorders, those are important broad family history. And then, when you look at their EKG, did they have a bifascicular block? Do they have a lot of sinus bradycardia?

Is there evidence that their WPW with pre-excitation or long QT or Brugada, those are important things to look for. And then you also want to consider other co-morbid conditions. All of a sudden, do they have an anemia or the hemoglobin at 7, whereas three months ago, their hemoglobin was 14, or their electrolytes are very abnormal?

So this is from the American College of Cardiology guidelines. So a lot of my talk is actually from the European Society, because I think the ESC guidelines are very good at talking about the pathophysiology. The American guidelines are like, oh, you have a patient with syncope. Here, follow this, and we'll see if we come to a diagnosis. And so their processes are similar, but the ESC guidelines have a lot more explanation to them than the American guidelines do.

And so when you're seeing a patient for syncope, what the American College of Cardiology recommends is that you do a history, an exam, and an ECG. If, based on this, you have a diagnosis of orthostatic hypotension or vasovagal syncope, you're done. Otherwise, they fall into the explain syncope. And then, based on that, you can consider an echocardiogram, an exercise treadmill, or an ischemic evaluation. And if you find anything obvious, such as they've had syncope and now, all of a sudden, their ejection fraction, you found, is 20%, well, that patient needs an ICD. But if all those things are normal, and you don't find anything, then you can consider what to do from there.

And so there are a lot of diagnostic tests that we use. And so really, the exam is probably going to give you your highest yield. Based on a careful history and physical exam, you will probably be able to diagnose up to 35% of the patients that you're seeing. ECG is going to give you another up to 10%. And then monitoring, whether it be whole monitoring or-- will give you more. And then doing other tests, like a tilt table test or an EP study, it's going to be much, much less, depending on your patient population.

So I'm going to break it down into several cases. This is funny. Some of my slides haven't shown up, actually. I'm just realizing that now. I don't know why that is. All right.

So we'll break it down into a case. This is a 24-year-old female. She comes in, and she says she passed out. She doesn't have any medical problems, no surgeries, no allergies, no family history. She's only taking oral contraceptives every day. She doesn't smoke. She doesn't use any other drugs. She only drinks occasionally. And currently, she's a graduate student.

On exam, her exam's pretty normal. Blood pressure is normal. Heart rate's normal. She doesn't have any orthostatic changes, and her exam is fairly normal. She's got a little systolic murmur, that a lot of patients have. This is her EKG, which is also very normal. There's nothing concerning on this EKG. It's just a normal EKG.

And then, when you start to talk to her a little bit more, it occurred while she was donating blood. And it was the first time she'd ever donated blood. And she tells you she's had some progerone. She felt warm and flush and maybe was a little nauseous and had some tunneling of her vision. She got better very quickly after she passed out. And then really, she felt tired for the rest of the day, but she didn't have any other residual symptoms beyond that.

And so, based on this evaluation, you can reasonably conclude that the patient had probably vasovagal syncope, OK? And really, at this point, you would not need to do anything else. So based on your initial evaluation, you might get almost up to 50%. Usually about 35% of your patients, you'll be able to define a cause of syncope.

And so it's really important to ask questions just prior to the attack. What were they doing before the attack? What were they doing when things started happening? If they remember it, or if there's anyone that has seen them, that actually saw them pass out, if you can ask them questions about how they felt afterwards, and then the other background that we talked about, with family history, prior cardiac disease, any neurologic history, and from that, you might be able to just be done.

And based on some of her historical features-- she had warmth, tunneling of vision, and it was while she was donating blood, which is a known situational trigger, that's diagnostic. And the testing is actually done, if everything else looks pretty normal. So you don't need to do any other evaluation for that patient.

So these patients that have reflux or neurally mediated syncope, for them, that is the most common etiology in any settings. So for a young patient, for an old patient, it's still the most common cause of syncope, is neurally mediated syncope, or reflex syncope, or vasovagal syncope. Patients that have this, with no other findings, they have no increase in their mortality.

But even though they have no increased mortality risk, it can have a lot of impacts on their quality of life. Sometimes patients will have very many, many episodes, where they have a really hard time controlling their symptoms. And there's no really great treatment for this type of syncope either. And so the biggest treatment is to avoid their triggers. So if they know that they shouldn't donate blood, or they can't get a needle or get a blood draw, you have to tell them to try to pre-medicate, if you will, make sure they're very well hydrated going in, look away, sit while you're having it done, keep your feet up, those sort of things, to avoid their triggers as much as they can.

And then you can give them some education on counterpressure maneuvers. So there are a number of counterpressure maneuvers-- I'll show you a picture-- counterpressure maneuvers that patients can do to help get the blood flow coming back to the heart. And then give them reassurance. Because sometimes they will have episodes where they can have several episodes of syncope in a short period of time, but it generally goes into a remissive phase and then stop.

So some of the counterpressure maneuvers, one of them is hand grips. So they can get a ball, like one of those squeegy balls, and squeeze real hard on it. Another one is leg crossing, where patients are standing, but they cross their legs, and they squeeze the muscles as tight as they can. That helps get blood flow back up to the heart; and then arm tensing, this or a hand grip across the chest.

And they can do that wherever, whenever it doesn't mean anything. It's not like they have to be somewhere special to do that. They can do it whenever they-- they should do it as soon as they feel an onset of symptoms, as well.

And then there's the tilt table test. So has anybody ever ordered a tilt table test? You know how many I've done? Like five. We just don't do a lot of tilt table testing anymore. And when I order one, I'm like, oh, I've got to do a tilt table. Because it's sort of a painful procedure to go through. And the problem with the test is that, not only does it take a long time, but it doesn't give you a of great answers either, which is really unsatisfying.

So the indications for doing a tilt table is someone who has recurrent unexplained syncope that you really think is probably vasovagal. Or they have one episode of syncope, but they're in a high risk setting. For instance, the airline pilot, he's going to get a-- he or she is going to have a tilt table test after one episode of syncope, even if it's classic four vasovagal syncope. And you also can use it to demonstrate susceptibility to the patient. So sometimes patients just need a little extra boost in education. And so you can use the tilt table test.

Tilt table tests are sort of difficult. There's a lot of different-- every cardiologist that you talk to will have a different method for how they do tilt table testing. So there's not even a good, standardized way. It's not like the Bruce treadmill, where everybody does a Bruce the same way. Tilt Table test protocol is different for every person. And it's nice when it's positive, but it's not necessarily useful if it's positive or negative, unfortunately.

And so you can consider using this when you're trying to determine, is the patient having vasovagal syncope, or are they just having orthostatic hypotension? For the patient who you talk to, and they say, well, I have jerking motions at the end of my episode of syncope, that can be normal for a patient who's had vasovagal syncope. But sometimes that needs to be differentiated from epilepsy.

For patients who have a lot of recurrent, unexplained falls, and you're not sure if that's syncope or if it's just falling from orthostatic hypotension, that can be another place to use it. And then sometimes, patients that have just a lot of episodes of syncope, and there is something wrong with the story, and it doesn't sound classic really for anything, and you're concerned about psychiatric disease, this might be a test to help you differentiate. Because if they tell you they're passing out all the time, and they get on a tilt table, and they don't pass out, then you might be dealing with psychiatric or psychogenic syncope.

But the problem is that the tilt table test doesn't give you a lot when your pretest probability is high. So if your pretest probability for having vasovagal syncope is high, the Tilt Table doesn't add very much at all. So this is what it looks like. It really is a table. And the patient really is strapped to it, just like this, so they don't fall.

There is a blood pressure cough that's on the patient that takes the blood pressure every one to three minutes, usually every one minute. And then they're also on the ECG monitoring. The protocol can last anywhere from 20 to 45 minutes. I usually will put the-- you can put the tilt table anywhere between up to 70 degrees. A lot of people use 60 to 80 degrees.

And so what I would do, usually, is put them up like this for 20 minutes. If nothing happens, then maybe give some nitroglycerin and see if that triggers anything. It certainly will make their heart rate-- or will make their blood pressure a little bit lower. But see if that's enough to trigger them to actually have syncope.

And so it's reported as a well-tolerated test. But it doesn't look very comfortable. And the patients don't look very comfortable while you're doing it, either. And it gets a little exciting when it's positive. So thankfully, of my five tests, three of them have been positive, which gets exciting. Like, OK, now what do we do? They passed out, OK.

[LAUGHTER]

OK. So any questions on these vagal syncopes? Common, very common. OK. So this is another patient. He's number two. He's 68. He presents with syncope. His past medical history is not as benign as the first patient.

So he had a history two years ago. He had bypass at the time. He's diabetic, hypertensive, has some high cholesterol. He's my typical patient. So this is my typical patient. So he's a good medication regimen for his coronary disease. He's on the [INAUDIBLE]-- and aspirin. And when you see him, his blood pressure is normal. It's 132/68. His heart rate may be a little bradycardic, but nothing to get too excited about.

And when you listen to him, now you're hearing a murmur at the white upper [INAUDIBLE] border. And it's sort of harsh-sounding. And you're like, oh, I don't know. That's sort of new. And on his EKG, he's got a PR interval that's OK. The upper limit's getting up towards the upper limits of normal, but it's still normal.

It looks like he's had an old infarct on this EKG. He's got a right bundle-- it's an abnormal EKG. Now what do you do with this guy? He's got a lot of high risk features, right? So he doesn't-- and I purposely did not give you any other history on his syncope. But based on just looking at him he has a lot of high risk features.

He's had an MI in the past. He's got a right [INAUDIBLE] on his EKG. And now you hear a murmur that's suggestive of aortic stenosis. And so this is the type of patient that, if he were to come to the emergency room right after this happened, he's probably a reasonable admit to the hospital to get everything done. And so the admission is really to expedite the workup, not so much to make sure you find out a reason.

If he comes in and sees you and says, oh yeah, this happened to me four weeks ago, probably not. He probably doesn't need to be admitted to the hospital. A little workup should be done pretty quickly if this happened a month ago. But you want to be able to get the echo right away, because you don't know what his LV function is, and you don't know if he's got severe aortic stenosis. You want to put him on the monitor to see if he has a brady or a tachyarrhythmia.

And so the role of echo in syncope is sort of priceless, actually. It can help you with a lot of things. Even if you have a patient who you know is having vasovagal syncope, a normal echo can be helpful. Sometimes, when I was in the military, very easy to justify an echo any time. A patient passed out. Well, we can't deploy them until we know that their echo is normal. And so we get a lot of normal echos, because it's extremely useful to have a normal echo, because you can reassure the patient, on so many different levels, if their echocardiogram is normal.

But in this patient, you need it. You don't know what his LV function is. You don't know if he's got aortic stenosis. And so any patient that you suspect cardiac disease, they need an echocardiogram, because the LV function is the most important thing.

And when you have a patient with syncope, there are some findings on the echo that are diagnostic. So if you have a patient with severe, critical aortic stenosis, and they've had syncope, that the etiology of their syncope until otherwise-- until something else gets found. And they need to have that aortic stenosis fixed.

If they have any atrial myxoma, that's obstructing blood flow through the mitral valve. And that needs to be resected. And that is etiology of their syncope. The same thing with tamponade [INAUDIBLE] straight away, dissection, or if they have a coronary anomaly. Coronary anomalies are very, very difficult to evaluate with echocardiogram. And this is going to be in your normal patient-- or in your young patient, where you're looking for a coronary anomaly, leading to syncope; but can be useful in a young patient with very good echo windows.

So you echo this guy. And on echo, his LV function looks OK. It's a little reduced. It's 45%, but it's not terrible. His inferior wall doesn't move very well, but he's had an old infarct, so you expect that. He's got some diastolic dysfunction. He really only has mild aortic stenosis.

So you put him on the monitor. He's been on the monitor all night. And unsurprisingly, he hasn't had any events, and his heart rate is ranging anywhere from 50 to 70 beats a minute. But he's not really having any arrhythmia. So now what do you do? What's your next step now?

And so this is where it starts to get a little tricky. What do you do? I would argue that, on this patient, it's completely reasonable to proceed with an EP study. A lot of our patients with syncope, very few of them, at this point, are going to go on a EP study. In fact, less than 2% of the patients now are going on to EP study.

But in the patients where the EP study is going to have your highest yield, are those patients that have evidence of structural heart disease, which he does. He's got a low-ish EF, and he's got evidence of scar. And so those patients, up to 50% of them are going to have an abnormal EP study.

There are a lot of things that diagnose an abnormal EP study. I promise you, I will not go into those details, because, when you're at the level of the EP study, it doesn't matter if there's sinus node read. Their spurt time is over 5,000 seconds, or whatever. That's not important here.

But what is important is, when you're doing the EP study, if they have evidence for having a bradyarrhythmia, then that patient needs a pacemaker. If they have evidence of having tachyarrhythmia, if you can induce VT or VF in the EP lab, that patient needs a defibrillator. The workup is done. You've identified what caused their syncope, and they've been appropriately treated for it. So that's what the EP study is, really to help you decide. And those patients that have syncope related to sustained VT during the EP study, those are the ones that are at your highest risk-- at their highest risk of having sudden death. And that's why they go on to get implanted with a ICD.

But let's say the EP study's negative, because it's only diagnostic in up to 50%, which sounds great. But then you still have 50% that you don't know why they had syncope. And so, if you have a nondiagnostic EP study, what's the next best step? Well, I think that one of things that we've been learning is that, for patients with recurrent syncope or very high risk features with no demonstrable evidence of other syncope, putting in an implantable loop recorder is becoming an easier and easier procedure. And I'm going to show you a picture in just a minute.

But for patients that have had recurrent syncope, and your evaluation hasn't shown anything, and they have no high risk features, that might be reasonable, to put in place a loop in, to make sure you're not missing a bradyarrhythmia, especially if they're likely to have a recurrence within the device battery life. And so now these batteries can last up to three years. You just put the loop. It stays in for three years. You wait until the patient has another episode of syncope.

And so for very rare episodes of syncope, this is a very nice device. Because a Holter monitor only gets you 24 to 48 hours. And event monitor can get you up to 30 days. But that's a small period of time, compared to when they might have symptoms. And then, if they have the high risk features, and you've done everything, and you can't find a reason for them to have syncope, then that would be an appropriate patient to put a loop in.

And sometimes, when we talk, sometimes-- I sort of glazed over the patients with vasovagal syncope who end up with a pacemaker. I would argue that that's a very controversial topic, which is why I'm not talking a whole lot about it. But if you're thinking about a patient that has vasovagal syncope, and you're considering, do they need pacemaker placement? Well, then they probably should have a loop first, to make sure that they are having long pauses that will help their vasovagal syncope.

And this is a registry out of Europe, [INAUDIBLE] site. I've heard some in patients, recurrent syncope. They all have loop recorder placements. And on average, these people had already seen three specialists. They'd undergone nine diagnostic tests. And in one year, 1/3 of the patients had recurrent syncope. And that loop assisted with the diagnosis in almost 80% of patients. We don't have any other tests that will get you up to 80%. Nothing else will do it.

So loops are very, very useful tools. And just so you can see what they look like, they're small. They're small. I do not get paid by Medtronic. This just happens to be a Medtronic slide.

So this is what our loops used to look like. So it's about the size of a thumb drive. It's a surgical procedure to implant, and it goes sort of in the left chest area. You have to make an incision that's about an inch or so. And it's really sort of barbaric. You just sort of shove it underneath the skin, because you want it to be real tight so it doesn't move around a lot, because you'll get a lot of artifacts. And then you just sew it up.

And the risks are pretty small. Patients can have MRIs when they have loops. And the risk of the device is an infection. And so if it gets infected, you say, oh, oh. You just pop it out, give them some antibiotics, and that's all there is to it. I mean, it's really not a big risk procedure. But it does leave a scar. And so that's something to think about in young patients that don't want to have a scar.

Medtronic, last year, came out with this little thing. This little thing is awesome. So it's really small. It's smaller than the size of my pinky. And it's what they call the injectable loop recorder. Orders So they have a tool where you sort of punch it into the skin. And then it really very much looks like a plunger, and you plunge it in right underneath the skin.

The battery life is still three years, so that's a very long battery life. And you have a long opportunity to really diagnose arrhythmia in these patients. The risk is the same. It's small. The risk is infection, but that risk is small.

Here at John Muir, we do them in the holding bay, at [INAUDIBLE]. But you can do it as a bedside procedure. It's almost like putting in a central line. I mean, that's the sterile technique. The operator's wearing sterile equipment. The patient is draped. But it's not a big, long procedure. It really takes about 10 minutes to do. And then they go home with a little stereo strip. And then they get monitored continuously for up to three years.

So it's small and much more-- we think about it a lot more frequently now that we can put that tiny, little thing in, instead of something that's, not terribly big, but bigger. And I anticipate that all comp-- so Medtronic is the only one that has this one that's small. I anticipate probably all of the companies will have their own at some point in time. Or they might even get smaller. It's hard to know where they're going with it. But this is a pretty useful little tool.

And one thing that we talked about a little bit-- and the reason to be aggressive with this with that other patient, case number two, if he underwent a loop, a loop recorder patient, it's because of the mortality. So syncope does have some mortality. So at one year, 18% to 23% in patients that have cardiac syncope, they have a risk of dying, up to 1/5 risk, up to a 20% risk of dying in one year.

And if you have a patient that's gotten vasovagal syncope, their risk is the same as the general population. It's very small. And it's severe-- their likelihood of dying is directly related to the severity of their LV dysfunction. So the LV is so important. We make jokes about how cardiologists only care about the left ventricle. But the left ventricle really is very, very important. And the more severe their LV dysfunction is, the more likely they are to have mortality related to it.

And then case number three, an 84-year-old, she has syncope. She has high blood pressure, low thyroid, diabetes. She's on hydrochlorothiazide, 25 a day; centroid, 112; metformin, 1,000 twice a day. On exams, she's a little hypertensive, not terrible. Heart rate's OK. She's got a little murmur that's been there for years. On her EKG, she's got probably left atrial enlargement, some right bundle branch block, maybe AV a first degree AV block.

And you're seeing her in the office. And you do an orthostatic challenge on her. And tilt table testing, we already talked about. But that's one form of work to static challenge. The other form is active standing. So instead of just doing orthostatic blood pressure measurements, what you can do is take manual measurements. And so they are laying supine for a few minutes, and then you have them stand up. And as soon as they stand up, you start taking their blood pressure each minute, for three minutes.

Don't just wait for three minutes. I think that's how we're all classically taught, is they have to change position, you have to wait three minutes, and then you do it. What the ESC guidelines recommend is start doing it right away. And you keep taking the blood pressure until it's neutered out or until it gets really bad or really low. And then you set them back down so they don't actually pass out. But you want to check it for up to three minutes, because there are a number of types of orthostatic hypotension.

So the causes of orthostatic hypotension, they're all related to autonomic function. So you can have primary autonomic failure either related to something like Parkinson's or [INAUDIBLE], Lewy Body dementia. Or you can have secondary autonomic failure. This is much more common in our patient population. They'll have diabetes. You can have amyloid, urine, spinal cord injuries. All of those lead to secondary autonomic failure.

And then probably the most common is going to be related to drug-induced orthostatic hypotension. And we all know diuretics cause orthostatic hypotension. And then it gets worse as patients get older. And then you want to look for evidence of volume inflation, whether that be from hemorrhage or vomiting, diarrhea, et cetera.

And what happens, especially as we get older, unfortunately, is that sympathetic pathways just become chronically impaired. And so, when you stand up, when your blood volume, when your central blood volume, drops into your legs, you don't have those pathways really to cause the vasal constriction that your body needs, because they become impaired. And so you'll have an abnormal decrease in blood pressure upon standing. And there are several types.

So your classical orthostatic hypotension, the one we learn about, is the blood pressure goes-- systol goes down by 20. The diastolic goes down by 10. And that's within three minutes of standing.

Some groups of patients that we miss, because we don't check the blood pressure until three minutes, is the initial orthostatic hypotension. So these patients, their blood pressure drops precipitously, very, very quickly. But then it responds and comes back to normal. So you might miss that. And so their blood pressure can drop up to 50 millimeters of mercury while they're standing, but it resolves very quickly. And so they might have brief symptoms, but it goes away rapidly.

And then you can have the delayed orthostatic hypotension which is-- this one's tough. It's slow. It progressively decreases while they stand. And there's not really a bradycardia response. But you can have a mixed response, where they have some bradycardia. And this one's going to be a very challenging one to treat.

And then POTS is one of those orthostatic intolerant syndromes. And these are patients that have dizziness, very severe dizziness, when they stand. But they haven't passed out. Their heart rate-- and it's really predominantly related to an increase in heart rate, less so than a decrease in the blood pressure. But their blood pressure can be unstable at the time. So these are the orthostatic syndromes that can lead to syndromes.

And the treatment is hard. Ideally, you want to get rid of the offending agent. But if hydrochlorothiazide has been working for years, or you have a patient on a multi-drug regimen for hypertension, it might be really hard to stop the hydrochlorothiazide. These patients need to have sufficient fluid and salt intake. So they have to drink a lot of water every day, two to three liters a day. In

Your healthy population, what the ESC recommends is sometimes up to 10 grams of salt a day. That sounds terrible. It sounds like a lot of salt. And I think that's even more than the American diet. So it's a lot of salt that they need to help build up their volume.

And then something I read, that I don't know if it's true, but it says it's ingested. If they get some orthostatic symptoms, you can have them drink cold water quickly, when the onset of their symptoms. I don't know if that would work. I'm trying to find a patient that I can try-- tell to try that to.

This is the first that I've heard, for patients that have a lot of orthostatic hypotension-- sleeping with the head of the bed elevated at 10 degrees. I tried to tell a patient that. I'm still waiting to see if that actually works. But it's stated that it prevents some of this polyuria at night. Maybe it maintains a more favorable distribution of your body fluids and might get rid of some of the hypertension, nocturnal hypertension. That's just what the ESC recommends. I thought that was very interesting, and I'm waiting again to find the right patient to offer that to.

And sometimes, a lot of times, patients need compression stockings and abdominal binders. If compression stockings are hard, they can't pull them on, maybe an abdominal binder would be better. But having patients say, come on, you really need to wear your compression stockings. It really is important, because it does help maintain your blood volume to the heart and then to the brain, which is the most important thing.

And there can be medications that you use. So our common medications that we use are Midodrine and Florninef, or fludricortisone. A Midodrine is like an oral-- it's an oral alpha agonist, so it's like an oral vasopressor, sort of. But it will increase the blood pressure in both the supine and the standing positions. And so patients that have severe hypertension at night, Midodrine might be tough to use. But then you can dose the Midodrine so it's several hours. They don't take their next-- they don't take a nighttime dose. You only have them take it during the day, while they're awake. And then At night, they'll be laying down, and they don't take it then.

And then the fludricortison, the Florninef doesn't have as much data for it. Midodrine is definitely better. But fludricortisone is a mineral corticoid. It stimulates sodium and water retention. And so hopefully, that can help the blood volume increase, expand your fluid volume.

For the elderly population, hospitalization goes up as their age goes up. And it's related to orthostatic hypertension. And up to 30% of patients over the age of 75 can be admitted to the hospital. And very commonly, it's related to orthostatic hypotension. You have to be careful of cardiac arrhythmia in these patients because that risk goes up as they age.

And so what you're looking for is, if they have a history of syncope in the morning, that goes along with orthostatic hypotension being an etiology of their syncope. You also want to ask them about their gait and their balance and stability, and if they have evidence of cognitive impairment. All of those things make it very, very difficult to really fully evaluate what's causing syncope in these patients. But it is important, to get a sense for those things.

And sometimes, orthostatic hypotension is not reproducible. When they come to see you at 4 o'clock in the afternoon in the office, they might be pretty well hydrated for that day and not having a lot of orthostatic hypotension. And so oftentimes, the orthostatics need to be repeated multiple times, or do it the first thing in the morning, when they are having their episodes.

In this patient population, CSM stands for Carotid Sinus Massage. That's important. You want to massage on the carotids. And so you massage on one for up to 30 seconds, and then you massage on-- don't obstruct, just massage-- on the other for up to 30 seconds. And you want to see if they have bradycardia or if they have a pause or if they have a change in their blood pressure. The only real contraindication to doing it is if they've had a stroke or TIA within the last three months or if they have a Brewe that hasn't been fully evaluated with an ultrasound.

You can do tilt table testing in the elderly. It's safe for them. 24-hour ambulatory blood pressure is going to be really useful. It's going to give you a lot of useful information, because they take blood pressures every 30 minutes. So you're doing to get a lot of information on what their blood pressure is doing throughout the course of the day and at night. And then these would be the type of patients we would consider early loop recorder implant on.

And so just to review the causes of syncope by age, in a younger patient, it's going to be vasovagal, situational, maybe psychiatric. You want to do an EKG to worry about the bad stuff, to exclude the bad stuff. And then if their EKG is normal, you've excluded the bad stuff. But if they have any abnormalities, that's when a cardiology consultation, an echocardiogram, would be very important.

In an older patient, orthostatic hypotension and neurally mediated syncope is still very common. Your risk of cardiac syncope, whether it be mechanical, obstructed syncope, like aortic stenosis or myxoma, that goes up. But arrhythmogenic syncope also goes up significantly as well, too.

And then driving-- so the DMV doesn't have a lot of good information on it. It lumps syncope in with seizure disorders. And it's very nebulous as to what you need to do. So physicians report, in good faith, patients who cannot drive, for whatever reason. And then you tell that patient that they shouldn't drive.

But types of syncope that would prohibit driving-- so patients that have heart disease, like they have severe aortic stenosis, and they haven't had surgery yet, and they're having syncope, those patients should not drive. Patients who have a low EF, that don't have an ICD or aren't wearing a life vest, those patients should not drive. Patients who have really long pauses up to several seconds on a monitor, and they don't have their pacemaker put in yet, they should not drive.

And then patients that have current syncope, if it happens without [INAUDIBLE], and if it occurs when they're sitting, then those patients probably shouldn't drive either. But once everything is controlled or it's treated, for instance, they get their ICD, or they get their valve fixed, or they get their pacemaker put in, they can go back to driving, assuming that the device is working normally.

And so this is available. I'm not going to go through it. But these are just some diagnostic criteria, talking about-- if you have vasovagal syncope; when you have situational syncope; the arrhythmia syncope, if you've got a lot of bradycardia, or you have heart block on your EKG; if you have VT on your EKG; or if something's wrong with a patient's device, and it's malfunctioning, and they're having syncope, that's diagnostic.

And then if they're having ischemia, then that would-- they come in, they're having an infarct, and they had just had an episode of syncope, that's diagnostic for the etiology of their syncope, as well. And then those patients that have severe [INAUDIBLE] lymphoma, pulmonary embolism, or acute dissection, that's diagnostic for an etiology of their syncope. And then this is just a summary of the tests, all the tests-- well, a lot of the tests that we can do.

So in summary, syncope common, up to 50%, 1/3 of the patients that we see are into-- have vasovagal syncope. 1/3 of the patients that we see, we're not going to have a reason for them to have syncope. We're not going to find a reason. And it can be very frustrating for both the patient and the physician. So you just have to be patient with it. And you tell the patient to be patient with it. And then you make sure you've got a good treatment plan with them.

But the first place to start is your history, orthostatics, and your EKG, and then, from there, recognizing the features to expedite their workup and recognizing diagnostic features, and when you can stop your workup. Those are important things.