NITIN GARG: No disclosures, except some of these are my cases, unfortunately. So we're going to talk about-- I can't really cover the whole breadth of vascular complications. There are so many complications we have. But I'm going to touch on some of the key things that probably everybody gets to see sometime during their career.

General considerations, acute MI is probably one of the most common things we talk about in our line of work. Anytime we talk to a patient before surgery, any time after surgery, an acute myocardial event is extremely common in our patient population. Not surprising, peripheral arterial disease is actually a surrogate marker for coronary artery disease. All studies will show that at least 90% of patients who undergo vascular surgery-- and I'm talking about peripheral arterial surgery or carotid vascularization have coronary artery disease. So it's kind of the same patient population that you guys heard about from all the cardiology people this morning.

Risk factors, underlying comorbidity, prolonged surgery, blood loss, well, we don't lose much blood in our cases, occasionally. But then poor functional status is another risk factor. So I think this is probably one of the unique things to our patient population when we talk about myocardial events. They don't really present with typical symptoms many, many, many times, and especially if they're diabetic, if they have any kind of renal disease, which is at least half of our patients, if not close to about 70% of our patients.

So if they have nausea as their primary complaint, and there is no other explanation for nausea, we actually do a complete cardiac work-up for just that symptom. And there has been reasonable data to suggest actually nausea is a common symptom in our diabetic patients, especially, often acute myocardial event. So that's probably one of the things if somebody is going out of the hospital, and they have this intractable nausea, get seen by their primary care afterwards. It's probably something to keep in mind.

New onset of arrhythmia is another thing that could be a diagnostic clue that they have a myocardial event. We have a low threshold of obtaining cardiac markers, just because of the population we are dealing with. Their symptoms are not typical, and the fluid shifts we get during some of our big cases, just these are the diagnostic mark-- the troponin levels would be very easy to get. And they're really not expensive. So we have a low threshold of doing that.

So those are primarily the diagnostic modalities we use. EKG is unfortunately not very specific or sensitive because a lot of times these are non-STEMIs. They are mostly what we call demand ischemic events. So EKG really doesn't help. But troponins will pick that up. Patients who have renal disease, a single troponin really doesn't help because it can be artificially elevated. So serial markers are helpful in those cases.

Some of the things we try and do to help with these, peri-operative beta blocker was really, really hot about 7, 10 years ago, I would say. Everybody jumped on that and said, oh, we need to put everybody on beta blockers. That thing has really, really taken a toll in the last two years. Some of the research that was done about 10 years ago has been quite-- I don't know what the proper word for that would be-- but the research has been questioned as not being reliable. And there were some people who so-called fudged the data just to make it look good.

So most of the recent data actually suggests that peri-operative beta blocker does not really help except for decreasing the risk for MI. But mortality risk for those patients is actually higher, especially if the beta blockers are started early on. So if somebody is getting a surgery next week, we don't really start them on beta blockers. If somebody is planning to get a surgery a month or three months from now-- they're hypertensive, they have other indications, like coronary artery disease that we know of-- then yes, it is reasonable to start them on a beta blocker. But if somebody is going to be planning for surgery in the next few days to weeks, it is really not helpful to start them on a beta blocker. It actually increases the mortality.

This, I think, beta blockers have pretty much been replaced by statins. If I see a patient in clinic, if they're not on a statin, it's almost like a 95% chance that they will end up on a statin if I see them. If I don't start them on it, then there's a specific reason if they have liver function problems or they're allergic to it. But even if people who refuse to take statins means it's almost a 15 minute conversation before they leave the clinic.

I think anything we do this day and age, there is pre-operative data, interoperative data, and postoperative data that say that statins actually help your outcomes. So in probably the last year and a half, I would say Lipitor is the drug of choice. It's less expensive than some of the new agents and has better efficacy than we used to use some of the statin before.

Well, I said I don't have any bleeding problems, but I guess I do. So bleeding is definitely a complication that we face. It's not uncommon. Some of these things are from a famous surgeon. And then the rest of them are unknown. So these are some of the famous quotes that you will hear. "Bleeding should never stop a good operation." That is what I tell all my Fellows.

[LAUGHTER]

So keep operating. "Bleeding should never stop a good operation." Again, that's not always true. Bleeding is a problem. But unfortunately, that is really no way we can do a bloodless surgery, especially working on a 2 centimeter aortic vessel which pumps out a blood volume in 30 seconds.

The reason we worry about bleeding is because transfusion is associated with poor outcomes. So patients who get transfusion for any surgery, aortic surgery, lower extremity revascularization, have higher risk for ICU stay, length of stay, high risk for wound infection, and adverse pulmonary events. So all the non-mortal clinical events actually are adversely affected with people who have to have transfusion.

So it's not the transfusion, per se, which causes it. I think it's transfusion as a marker that you had probably a problematic surgery that you either lost more blood than you anticipated, or it's just the magnitude of the surgery that the patient had. I don't think there's a whole lot in terms of protection. Hemoglobin gives you an idea about the-- if you had too much bleeding. Tachycardia has a great marker for us to use.

If I gave this talk probably five years ago, I don't think I would have had this slide. This is really something. This is becoming a real concern in our line of work now. Cardiologists probably addressed this a few years back because they used to see-- this is actually a patient who had a cardiac cath done. But there is significant radiation injury. I don't think how this thing points. So radiation injury on the patients after a cardiac cath, and it's directly related to the duration of the procedure or the amount of radiation you use, the location of your CR.

Now, we didn't used to see that kind of a problem 5, 6, 10 years ago because most of our procedures were probably tune of 10 minutes of radiation time or less. Now, our endovascular technology has really, really come a long, long way. We are doing some very complex endovascular aneurysm repairs, which are about four to six hour operations, sometimes even longer than that. And you have a fair bit of radiation dose in that case. So having radiation injury is something which is very, very important for us in this new developing endovascular era.

It can vary all the way from just mild erythema to some severe skin necrosis. I have really not seen any kind of advanced skin injury, apart from erythema, despite some of the longest cases that I have done. Fortunately, we try and move the CM around, and try and do some other things to protect it. But if somebody presents after a long endovascular procedure, they complain of some pain or burning sensation of the back, it's always nice to look at the back and look for something like this. So mostly the cure is supportive. In occasional cases, you have to do some surgical intervention with plastic surgery. But most of the cases resolve with supportive therapy.

Wound infections, well, we can talk about all day. We probably, if I talk about the 26 patients on our service right now, at least a third of them actually have some kind of would infection or the other. Diabetes, obesity, peripheral arterial disease, venous disease, a bigger surgery blood loss, we have it all in one patient, basically. So it's not surprising that we deal with wound infections.

The most problematic-- if I have to pick two most problematic surgeries we do when we deal with wound infections is open bypass surgery and people who come in with the prior foot infections or some kind of diabetic foot ulcer that we're tying to salvage the leg. So there is decreased infection rates. Some of our endovascular cases, occasionally, we see it. But those are the two most common populations. We give them very close follow-up because we know they're the highest risk patients.

People who are obese have diabetes or immunosuppressive agents, undergoing urgent or emergent surgery have been shown to have increased risk for wound infection for obvious reasons. If you have a prosthetic graft, and you get a wound infection, that is a very, very big problem for us, very high risk for limb loss because most of the times you have to replace the graft or take the graft out to treat the wound infection properly. And that is mostly their limb salvage graft, so not surprising they have a high limb loss rate.

If a patient comes to clinic, has some drainage from a skin incision, there is any concern they have underlying graft, we almost treat it as an urgent or emergent problem. We don't send them home, saying, hey, it's OK. Just watch it. It'll stop draining in a few days. If they're going home, they're going home after at least some study to make sure there is no deep-seated infection or an abscess or graft involvement.

CT scan is pretty much our modality of choice for anything we do, because a lot of times clinical exam is extremely unreliable, extremely variable, and really does not give you the three dimensional anatomy that you get with a CT scan. Duplex, if done in the proper facility, is extremely helpful to look for a fluid collection, but really does not show an absence very well. So CT scan really is very extensively used in our line of work.

Long-term graft infections, usually we're talking about delayed graft infections. They're mostly a concern for prosthetic infections. Venous bypasses or venous conduits really do not have any long-term infection risk that we deal with. If you have a peri-operative wound complication, your risk for long-time graft complication is going to be higher. They are an extremely problematic issue, high morbidity, high mortality. In fact, one of my partners is doing an infected aortic graft this morning. Two of my partners are going to have to do it. It's just not possible to do it by yourself.

They tend to present with some non-specific symptoms, unfortunately. You have to have high clinical suspicion to first diagnose it if somebody has a prosthetic graft. Local symptoms can give you a clue, cellulitis, abscess, sinus tract, which is draining, especially sinus tract in a [INAUDIBLE] incision, either in the neck or the leg. They tend to have more generalized symptoms if it is a body graft. So what I mean is a body cavity graft as in thoracic or abdominal cavity graft, they will tend to have more of a malaise fever, just not feeling well, rather than any kind of definitive clinical sign of infection.

About 40 50% of the patients, despite having a bloodstream infection in a way, if their graft isn't the vascular system, they do not have positive cultures. And white counts could be notoriously low in these patients because they just don't mount a systemic response many times. So those are really not very good diagnostic tools to go by. Inflammatory markers are elevated most of the times, but again, they're non-specific, so you really can't depend on them.

I think the most important one in this is herald bleeding. So if somebody has a graft in their groin, for example, they notice some bleeding from the groin. And it stopped, even if it stopped on its own. And that's a very, very ominous marker. That requires immediate attention or something. Just put the patient int the hospital. Let's just see how it happens, because that usually tends to be what we call herald bleeding.

There is a little bit of a rupture of [INAUDIBLE]. It forms a clot, and then it stops. But the next time it's going to bleed, it usually is going to be catastrophic or fatal. We have taken care of some patients who come, unfortunately, who had this presentation. Nobody realized what was going on. And then it becomes a really, really bad problem.

This is one of the patients. I don't know how well it projects, but you can see how many incisions he has. This guy has aneurysms of his venal artery, his aorta, his [INAUDIBLE], all operated in the last probably 10 years. And then he had a graft in his leg. These are two separate patients, by the way. He had a graft in his leg, which was placed about six years ago, and had this chronic draining sinus for the last year. He's immunosuppressed. And he had a prosthetic graft underneath. So even if all the studies will tell you that he has no infection, that doesn't infect your graft unless proven otherwise. So we did an external bypass on him.

The patient on the right side of the screen is a patient who had a thoracic endograft done at some other facility. And what you can see here, if I can get this pointing right, is-- not on the screen-- that's a thoracic endograft. That's the lumen of the aorta. And there's air inside the aortic wall. And then there is air on this paravisceral segment of the aorta.

So there should be no air in your blood vessel. If you have air in a blood vessel, that is a bad problem. So this guy basically had an infected thoracic endograft. Not just the thoracic part of it, he had a paravisceral aortic graft, so all the branches and everything was infected. So this patient pretty much converted to Hospice care, and he passed away within a week. But this, just the kind of problem that we're dealing with, it's a bad one to start to have.

I think one of the probably unique graft infection is an aortoenteric fistula. It's a kind of graft infection. But the specific things you deal with is a herald bleeding is extremely common in that. So the physiologic mechanism for an aortoenteric fistula is you have a prosthetic graft, which was used to the replace the aorta for some reason or other. Either the patient has peripheral arterial disease, required an aortic bifem or had an aneurysm, got a graft placed to repair the aneurysm, emergently, or urgently, or electively, for that matter.

And traditionally, we cover the graft with tissue, so the duodenum is not in direct contact with the graft. But in some cases, either because it was an emergency, or it was not properly done, or it was just erosion over a period of time, the graft just pulsates and eats through the wall of the bowel, basically. And this is what it looks like on endoscopy. You can sometimes see. It's a pretty cool picture to see. Unfortunately, it's a bad problem. Is you can see the graft through the endoscopy.

And a lot of these patients will present with a herald bleed. And they will have an episode of hematemesis. Everything will stop. In fact, you scope them. You don't see any bleeding. You do an angio. You don't see any blood anywhere. You say, oh, life is OK. There is no bleeding. It must have been something that stops on its own. But if the patient has had a prior graft, you have to make sure your gastroenterologist crosses the third portion of the duodenem and into the jejunum because that is where this is located, usually. It's easy to miss if you're not looking for it.

So anybody who has had a prior aortic graft placed, we see them with any kind of hematemesis, this is the first thing we think about. This the first thing that is asked on the board questions for us. So very low threshold for further investigation, checking it out, getting a scan to see if there is any kind of air around the graft, communication, or some contrast outside the graft that's seen here. So those are some general overwhelming things that don't fit into any of these four other categories that I'm going to talk about.

I'm going to just pick on like three or four topics, which are probably very important to most people in the room here, and go through some of them. Carotid endarterectomy and stenting, the most common complication that everybody dreads is a peri-operative stroke. If you talk to any vascular surgeon who has been doing this long enough, everybody has had one. If they have never had one, they've just not done enough carotids. That's what we say. And if somebody says that they can do it without a stroke 100% of the time, that's probably because they don't know what they're doing. So unfortunately, we have all had one of these at some point or other in our career. If not, then I'm sure we will have one.

Patients who are symptomatic to begin with have a higher risk for peri-operative stroke, just because we call them as unstable plaque as compared to patients who are asymptomatic. Although the patients who are asymptomatic, if they have a stroke, that is really, really, really bothersome because as a physician, you basically just destroyed somebody's life who was just fine and healthy before they came to you.

So those are extremely hard on you if that happens to you. Fortunately, I've not had one of those yet. I've definitely had one after symptomatic patient is carotid. Most common time period in the first 24 to 48 hours. Early strokes what we call either technical or because of the prothrombotic surface of endarterectomy side or all the stentic side.

Presentation is very typical. Unless the patient is in the [INAUDIBLE] or the recovery room, or in the operating room when you notice this change in symptoms, this day and age, the CTA with a brain perfusion is the modality of choice to see what's going on. We use interoperative duplex to really see if there is any kind of a technical defect. And a lot of times, it will give you a clue in what's going on.

So this is interoperative duplex. This is the color form. This is just the grayscale. And you can see this is the end point for where the endarterectomy ends. The patch ends here. Head is towards the left of the screen. And that looks like a fairly clean surface. That's gone where the screen on the bottom when you see a free-floating particle right in the middle of your artery. So if you notice something like this, if you don't address in the operating room, you're probably going to have a problem. So we use interoperative duplex pretty much 100% of the times after completion of a carotid, decreases your technical stroke risk.

Unfortunately, it does not make it zero. This is a patient of mine, actually, just about maybe two months ago had recurrent TIAs and one stroke before he came to us. Had an extremely irritated carotid artery. We did a patch on him. Doesn't have like a big problem, but the whole surface just looks very irregular. His artery was just very irregular after I finished his endarterectomy. I was very concerned about him.

I had a problem with the [INAUDIBLE]. Took him back to the operating room, had a little thrombus just sitting on this [INAUDIBLE] surface, really can't do anything about it. It's not a technical problem that you can fix. So we just took a vein from his leg, put that on, and you can see the difference in the surface of a vein graft as compared to his native artery, how smooth versus irregular that is. And that's all you have to do sometimes is just replace the artery completely when it is so diseased.

Hyperperfusion syndrome is really-- I have not seen one. But we probably talk about it at least once a week on our service. The way I explain it to my residents and fellows would be auto-regulation in the brain takes some time to re-accommodate. So a brain is excellent in terms of controlling its own blood pressure. It can handle a mean pressure of 60 to 160 without any problems because of the auto-regulation mechanisms it has.

When you have a high grade carotid stenosis that all regulation is disrupted because it auto-regulates to upsize this perfusion. But after you do an endarterectomy or a stent, you have increased the perfusion, and it takes some time for the brain to really deregulate that upsizing that it had done in the past. So that's pretty much the crux of why hyperperfusion happens.

Risk factors for hyperperfusion syndrome, bilateral [INAUDIBLE] symptomatic patients, peri-operative hypertension, which is very difficult to control, is an extremely high risk factor. Stenting tends to increase the risk for hyperperfusion syndrome in many cases, not always. Some of the papers would say that there's no difference. But many papers would say there's an increased risk.

It can vary in spectrum from all the way to just a headache to some serious visual disturbance, seizures, and unconsciousness, and really bad brain hemorrhage. It's related to the degree of edema that you have in the brain, or the hemorrhage that develops. So if you don't have a whole lot of edema, you just have some hyperperfusion, patient would have some headache. But if you obviously have some bleed, then they're going to be on the far side of the spectrum.

It can actually happen way up to about seven days from discharge. And patients are obviously out of the hospital by that time. Our average length of stay for a carotid surgery is about one day at this time. So they are way far out. We instruct patients to watch for this.

Treatment, unfortunately, is mostly supportive. If they really have a big bleed, people who describe a craniotomy to lead to intercranial pressure. I have fortunately never seen a hyperperfusion syndrome go bad. We probably scan 1 out of 10 patients any time they complain of a headache after a carotid endarterectomy to make sure they don't have it. But it's like talked about more than what we see. We just don't want to miss it. High mortality because of the lack of treatment options, primarily.

Nerve injury is not uncommon. If you look at data, about 15% nerve injury rate if you have independent evaluation. If you talk to vascular surgeons, they will say, oh, no, maybe 2% nerve injury risk. But if you have independent evaluation, we look at the data for independent evaluation. It's about 15% nerve injury rate.

Fortunately, 90% plus of these are transient. Most of them resolve in six months. Risk factors for nerve injury are reduced surgery, radiation, high lesion, which is pretty much most of the patients now. We offer stenting because of the risk of nerve injury. Only seen after a carotid endarterectomy. if you see a nerve injury after a stent placement, you're doing something wrong because you should not be anywhere close to a nerve.

Common nerves, vocal cord injury, obviously, leads to hoarseness. Anytime somebody has hoarseness after carotid surgery, it's good to have an ENT evaluation or a direct laryngoscopy if you can do it yourself. If they have dysphagia, it's another common problem. Speech evaluation is really critical because some of these patients don't get noticed and become chronic aspriators down the line.

Infection after an endarterectomy is extremely uncommon. People present 10, 12 years down the line with either a bulge or draining sinus, which we talked about before. Duplex or CT will pick up the diagnosis. Access-related complications for stenting, we'll go through them as a separate category.

Percutaneous access, we had four patients on our service last week alone with some complication or other with percutaneous access. The mode we are going towards, endovascular therapy, the bigger sheets we put in, the more difficult vessels we access, and we access vessels that people never even though you could access before. Means we are going in the brachial artery, radial artery, axillary artery, subclavian artery, femoral artery, popliteal artery, and the pedal artery. There's really no artery that we leave that we can't access or we don't go for. So obviously, the more heroic measures you take, you're going to have more complications. At least you learn the small problems that come with every single access problem.

Femoral access, probably still the predominant vessel access for any kind of an endovascular therapy, either diagnostic or intervensive. Everybody can star this. The femoral artery, you want to access it between the location where the epigastric arteries are because that's the inguinal ligament here, and above where the bifurcation is. Because if you access these, these always tend to be much more diseased. You want to stay on the femoral head because if you're going to compress the artery, you have to compress it against a bone to help the bleeding stop. You can't compress an artery in air or in muscle. So most of the complications that happen are because people don't realize how critical this access point is.

We can see a whole spectrum of complications from access, just a plain old simple hematoma when people complain that this thigh just looks red and black and blue, to pseudoaneurysm, to AV fistula, to hemodynamic problems related to bleeding. Risk factors, obesity, obviously, is a problem because if you can't see it very well, then you can't access it very well. if you pick a wrong access point, that's a problem.

Lack of inexperience with ultrasound is actually growing to be a much bigger issue than we used to think. When I was in residency, I would say I probably put in like 400 or 500 central lines in my residency and maybe use ultrasound in 10 of those. We just didn't do it. Nobody knew about it.

But then we went to IJ access, and everybody wanted to say use ultrasound, use ultrasound. Unfortunately, the problem with ultrasound is if you don't know what you need to do with ultrasound, it can really get you into some bad trouble. And we see a lot of these recently with IJ access. And everybody is using ultrasound, but it's easy to miss the landmarks with ultrasound, actually.

So the problem that happens with the ultrasound is the probe is only looking at the vessel in a single plane. And if your needle is too shallow of an angle, bu the time your needle tip is going to the vessel, you're beyond your probe value. So this is a picture of when you're looking at the artery. And you think you're going to hit it here. But that is actually your needle, and that's not the tip of your needle. The tip of your needle is far beyond your plane, which is right here. And you have no idea what you're hitting.

So we see all kinds of problems with this. The needle goes through the vein into the artery when they're trying to access the vein. Needle only goes into the artery. Needle goes into the nerve in the artery and the vein. And we have had pick lines into the vein, permacaths into the arteries. We have seen all permutations and combinations you can think about. So the key in ultrasound access is you have to try and keep your needle in the plane of the ultrasound probe where you can see the tip of the needle entering your vessel. That's probably the biggest technical factor that you can use to decrease the risk for injury.

Diagnosis for people with access complications, again, ecchymosis, pain. If they have hypertension, then usually, they have some serious bleeding going on. If they have lost a fair bit of blood, there's skin blistering and leg swelling that could happen because of the tension and the compression on the vein. Leg swelling can also happen if they have a connection between their artery and the vein.

Compartment syndrome is a unique problem to our arm access, especially radial artery access, which is increasingly used for cardiac stuff. So that's something you have to keep in mind for upper extremity access. If you're worried about it, duplex really catches them most of the time. If you really wondered about the retroperitoneal space that you can't see on duplex, CT will catch it without contrast.

Indications for intervention, if there are ongoing hemorrhage, they are unstable, they have skin threat, that is, their skin is so stressed out that it's starting to ulcerate, ongoing pain that is not controlled, infection, if there is a large fistula, which is causing either leg swelling or it's causing heart failure, then those are the indications we would intervene. Rest of them, we will try and watch. Options to treat are multiple. You can either compress them. You can inject them. Or many times, you have to treat them in the open fashion.

This is a patient that I just took care of recently. Unfortunately, the light doesn't light the project very well. Maybe if you see on this screen, the head of the patient is towards the left of the screen. That's the inguinal ligament, which is getting pulled about two inches towards the head. That is if you see a faint sign of a suture, that was a hole in the artery. This is the epigastric and the circumflex artery. So access was about an inch higher than where we were supposed to be. Your access is supposed to be here. And this was access up here.

This patient was an extreme. It was actually an [INAUDIBLE] fell out of his groin. So he had about a six millimeter hole in his femoral artery. And the poor nurse was sitting on this table with her fist like this. I couldn't-- I went to find her back after I fixed this artery to tell her how thankful I was for what she did that day. I think she was the one who saved this patient's life.

In these cases, we tend to go in the retroperitoneal space, So an incision, we go up higher, in the artery up higher because it's very hard to control this artery if you go directly and hit it, because it's essentially in no man's land. So we control the inflow of the artery from up higher, which is kind of a deep hole that we have to work in. That way when you open the space and you release the hematoma, it just does not kill the patient instantly. So the bigger the [INAUDIBLE] we put in, the higher the problem that is going to happen.

This is another patient we just took care of recently. She has had multiple accesses in the groin. And probably was a center line injury about two or three years ago that went unnoticed. This is a CT scan. The obviously-- darn it-- head is towards the side. This is the right carotid artery. And that's right IJ.

As compared to the left IJ, you can see the difference in the size of the vessel and the contrast of pacification. So this IJ is actually getting flow in the arterial phase. And this is not something which should be there. That's some kind of a problem. So there is bleeding going on from this artery, which is feeding through this pseudoaneurysm into the IJ, and this IJ is obviously-- this patient has a high-flow AV fistula.

This was actually a pretty cool case. So when he looked at her, her vein is towards that side. So there was a communication between the pseudoaneurysm on the vein, the vein we repaired. Then we got this artery on this side. And that was a pseudoanuerism. So this is the pseudoanuerism stuck to the artery. This is a psuedoanuerism down below. This is definitely one of the problems which could come happen because the ultrasound fools you on IJ access many, many, many times.

I can't really talk about percutaneous access if I don't talk about closure devices. Probably most of the people have heard about closure devices. There's many kinds and shapes and sizes they come in with, Proglide, Angio-Seal, Exo-Seal, mixed device, you name it. I mean, it's like, you can't even-- I don't think even I know how many kinds of closure devices are on the market at this stage. They all work a little bit different.

Most of the time, it's a user preference. What you're comfortable with is what you use. Some of them tend to have a unique problem. This is probably my least favorite device. It's called Angio-Seal. It has an anchor on the bottom, and it puts a thrombin plug or a collagen plug on the top. And if you are not inside the artery, which is the last patient I just took a picture of, the closure device is sitting somewhere here. You think you got it in the artery. You're not holding pressure. And the hole still keeps bleeding the closure devices here. And it just leads to a whole bunch of inflammatory reaction when you have to go and fix it.

The other kinds of problems I've seen with this closure device, particularly, and that's why it's not my favorite is this thing drags through the artery, lifts this plaque up, and closes the artery. So it closes the already here and keeps the hole open here. So you have a clot and bleeding at the same time, which is just awesome to take care of--

[LAUGHTER]

--at 2:00 in the morning.

So I think we're going to talk about endovascular aneurysm repair. This is probably about 70% of our aneurysm modality of choice this day and age. Endoleaks is-- at some point in time, if you have seen a patient who had an endograft placed for an aneurysm, you have heard the term an endoleak. It's like you're getting a call at 2:00 in the morning from some outside ED saying this patient had an endograft placed. He has an endoleak, what should we do?

And endoleaks are different kinds, size, shapes. And this is the categorization we have for it, type I, II, II, IV, V. V is almost kind of nonexistent. People used to put this when they could not find any other reason for endoleak. But most of the five endoleaks are actually in one of these categories above. So we don't really talk about V anymore. Type IV endoleaks used to happen in the good old days when we had the first or second generation grafts. We don't see it anymore. So really, not much of a problem in the last probably seven or eight years since we have the third or fourth generation grafts now.

Type I, II, and III are the type of endoleaks we talk about. Type II are the most common type of endoleaks. They're usually from retrograde flow from branch vessels. So if you are fixing an aneurysm with an endograft, you're not really taking care of the aneurysm sac. And there are lumbar arteries, inferior [INAUDIBLE] artery, which still keep bleeding in many patients. So they keep perfusing the sac.

Most of these leaks are small. They don't really bring systemic pressure to the aorta. And they will tend to seal in six months to one year time period. There is an occasional patient that they don't seal. We have to intervene. But we follow them frequently when if we see them on a follow-up scan.

Type I and III endoleaks are really, really problematic. They are very high flow endoleaks there because they have, essentially, a disjunction of the graft, either with the aorta or the illiac or between the graft components itself. So it's a big wide space that is going to constantly perfuse the aneurysm.

Some people think, and I am definitely one of those believers, that having a type I endoleak with a large aneurysm is probably worse than having just an aneurysm, because in a type I endoleak, you have an inflow into the aneurysm sac. So you're just getting hit by the hurricane force wind here, but you don't have an out. The blood has no way to go out. So the pressure in the sac probably is much larger than if you just had no endograft, because you have laminar flow through the aortic aneurysm, in those cases.

Type I and III endoleaks require attention, fortunately, still not urgent unless the patient has a rupture associated with them. Most of these are caught either during the first phase when we're doing the surgery or CTA on follow-up studies. And if it's a type I or III, they usually will get scheduled for the intervention.

This is a picture of an outpatients with a type III endoleak. So basically, the proximal part of the graft is completely disjointed from the distal part of the graft. So all this blood is going into the graft, but also, going into the aneurysm sac here. So that's a-- excuse me-- large type III endoleak. And you can see the proximal part of the graft towards the head-- head is towards this side-- and the distal part of the graft somewhere here buried in thrombus. So we just explained it to the graft and put a prosthetic graft to fix this one. Sometimes you can repair this endovascularly. Sometimes, you just have to do an open conversion.

Endograft limb occlusion is not an infrequent problem we see with the endovascular aneurysm repair, about a 4% risk if you look at all comers. Risk factor is related to tortuously, prior calcific or occlusive disease. The really difficult part of this, as many times these patients present with very atypical symptoms. They come in. They get all kinds of work up before they really noted to have graft occlusion. They will complain of weakness in their leg, pain in their back or their hip, whichever side the graft is occluded radiating down their leg. And after a few hours, the pain would go away.

Most people would ascribe it to some kind of a spine problem because that's obviously the most common reason for those kind of symptoms. They get an MRI. They get a neurology evaluation, all kinds of things because their foot doesn't look bad. Their foot still is warm. And The collusion tends to be proximal. They collateralize very well.

They just do not get noticed until somebody really says, well, you had an endograft placed. Maybe you should get a follow-up CT. And that's when they get noticed. And then you realize that they have an occlusion. ABIs can really pick even occult disease that is not obvious on clinical exam, if you feel pulses, if you do ABIs on them, you can pick up a disease which is not obvious on clinical exam.

This is another patient on our service a few I think about a year and 1/2 ago who had a graft occlusion, limb occlusion. So on the CT scan, this is the parent limb. So it has contrast in it. This is the limb that is occluded. This is the sagittal section, so you can see the parent limb and the occluded limb on the CT scan very well.

This is probably the topic which is closer to my heart than most vascular surgeons you will talk to. Hopefully, we'll get some time to spend on it. I think we're OK. So venous thromboembolic disease and IVC filters, I think this is a very big problem that is extremely under-noted and under-recognised in the community for us. And that is because there is lack of interest in it, lack of research in it, and frankly, lack of experience with this. There's not a lot of people who can tell you they have taken care of 10 or 15 occluded IVCs in their career. So just the location I trained in, the people I trained under, I think this is something is close to my personal clinical practice and my heart.

Acute filter occlusion, it's a really difficult problem to deal with. Again, people get all kinds of work up because their complaint is my legs are swollen, and I just can't walk. My legs are heavy. I cannot stand up and walk. Neurological work up, neurological outpatient evaluation, MRI, and then somebody realizes actually they have an IVC occlusion, because their filter is occluded. They have swelling because of that. And they have phlegmasia.

Phlegmasia is a term which is more commonly used than you actually see. Phlegmasia just means that they have a blue, cold purple leg. And what that is, that happens is because of their outflow problem. It's not an arterial problem. It's a venous problem. So they're not pale like you usually see with arterial occlusion. They are more in the high-color range, rather than low-color range. But the weakness usually throws people away and puts them in a completely different workup.

Duplex is a gold standard, but unfortunately, it can miss proximal obstruction unless somebody is really looking for it. So if the patient has an IVC in leg vein occlusion, they tend to have distal occlusion along with it. But if they don't, then duplex is going to miss it unless somebody really looks for this. If somebody has any concerns for this, if they have a filter in place, a CT will pick it up. CT usually gets done, actually, for a PE evaluation, and it picks up the bottom of the IVC when they go too far down on their abdomen. And that's when usually people will pick up the problem.

This is a patient we just took care of recently, had a PE six months ago. Had an IVC filter replaced before his urologic surgery, head, feet, right, left. This is an IVC filter. This is aorta. You see contrast in it. In fact, you can see contrast in the IVC at this level. This is where the left venal vein usually comes in. All this dark is thrombus. This vein is about twice the size of normal vein because of acute thrombus. It dilates.

This is his venal gland. So I just put the pictures together because we can't get everything in one frame. So, again, this is the right side, and this is the left side. And this is his illiac vein, completely occluded. This is left illiac vein, all occluded. The groin is about at this level. Now, IVC is completely occluded here.

This is his femoral [INAUDIBLE] vein in the thigh, which is also occluded. So unfortunately, he was like three weeks out from a prostate surgery. And this guy was miserable. This guy had not walked in a week and a half before he got transferred because he just could not get up. His legs were so weak.

His bleeding risk was extremely high. We started lysing him. And we probably lysed him for like 16 hours. We put thrombolic therapy, TPA into his legs both sides. And he started profusely bleeding from his Foley catheter. Unfortunately, when you have an IVC occlusion, it is not uncommon for people to actually have hematuria, or pelvic pain in women. And that's because your internal illiac veins and all your pelvic veins drain through your IVC. So if people complain of lot of hematuria and they're young-- they don't have any other reason to have hematuria-- you have to think about an IVC occlusion in those patients, especially if they have some kind of a mechanical filter in place.

So after we lysed him, his legs looked OK. The segment in the thighs cleaned out pretty nicely. But he still had a lot of thrombus in his illiac veins. We couldn't lyse him any more because of the ongoing bleeding. So we just aspirated the thrombus, angioplastied. We left a little bit of a chunk here right by the filter. But, enemy of good is better as what the-- sorry for that. [INAUDIBLE] was, and we finished up. The guy actually did amazingly well. I just saw him in the clinic last Thursday. But this is an extremely common problem because I think our filter use really went crazy in the last about three to four years for unfortunate reasons.

Chronic iliocaval occlusion is the other spectrum of the same problem. They either have a congenital abnormality that they're born with an atretic IVC. If that is the case, they usually do very well because they collateralize extremely well. Unfortunately, with some inciting event-- either they got dehydrated, had too much to drink one night, or had a long travel, or have some kind of a pro-thrombotic state-- they will occlude their collaterals. And all of a sudden, as I explain to patients is that I-40 was blocked before, and now 52 gets blocked. And there's a lot of traffic.

So their legs are swollen. They can't walk. They chronic skin changes, veins all over. And the first thing they say, is, oh, you've got varicose veins. You've got to treat that. So that's unfortunately really a problem if you treat the superficial veins, because their superficial veins is what is keeping their legs on. If you treat their superficial veins, they really have a bad, bad outcome, because that's like just losing everything they have flowing.

So anybody who has had a prior DVT, family history of DVT, young age, that they shouldn't have these symptoms that we talked about, had a prior IVC filter, recurrent or non-healing wounds in the legs, or venous claudication, which is very easily confused with arterial claudication, feeling heavy after walking a few minutes. If they put their legs up, it actually feels better, which is contrary to arterial problems. Again, duplex can very easily miss proximal obstruction. If it is a chronic illiac vein obstruction, duplex will miss it about 80% of the times.

Clinical exam is probably more specific than anything else. Unfortunately, we don't see skinny patients like this very often. So think about looking at this in a 300 pound patient. You can never see any of this. So if you have a clinical suspicion, you have to either tell your vascular techs to look for it and do a duplex of the illiac veins or the IVC. Or you have to have a venogram or a CT scan to really look for it or an MR venogram. You have to address this prior to any superficial venous treatment because that is, as I said, is a big disadvantage to a patient if you treat their superficial veins.

This is one of my patients who had a chronic-- IVC patient, 35-year-old male, had a filter put in while he was in the military after a DVT. Again, people get filters for some reasons, which are still unclear to me, when all they need is anti-coagulation. This is his groin. And these are not iliac veins. These are collaterals. His leg veins should be in this segment here. You can probably see the wire going from the left side that we crossed an illiac vein.

This was his old filter. This is a large collateral that was all that was draining his leg. This is intervascular arterial [INAUDIBLE] imaging. We use it frequently for treating venous disease because that's pretty much the only way you can size these properly to treat them, and really know where the disease ends and starts. So this is the level of his renal veins. You can see the vein is patent. But beyond that, the vein is essentially occluded. So I think this is probably my record in terms of the number of stints I have put in patient. I think I put 13 stints in this patient. We gave him a new IVC and gave him a new illiac veins.

This guy lost 20 pounds in the first week after surgery. He told me, he came to my clinic two weeks later. He was like this is the first time I've seen my ankles in 20 years. I've never seen my ankles in 20 years, because they just retain so much fluid, and they think there's the way of life for them. And the problem with these patients, they are usually very young, means 35 is probably the median age for the patients I treat with this problem. And they are miserable for many, many, many years in their life. So this is, as I said, close to my personal feelings and my practice patterns.

Paradoxical embolus, we have seen probably four or five patients the last year. I can't tell you if I saw four or five patients in my entire training in seven years. But I don't know if there's just a difference in what's happening, or it's the filters that are causing traumas up higher, which are going. But paradoxical embolus can present like an arterial occlusion. But the actual problem is that you have a venous thrombos that vent through your atrial septum.

The key is that an acute venous thrombolic event, if it's a large thrombus burden, can really occlude your pulmonary circulation to a point that you have a high pressure in your right heart. And that high pressure in the right heart can open up a shunt, even if you did not have a shunt existing, even if you've never had an ESD, never had a PFO. If you had a massive PE, it can actually increase your right heart pressure enough that you will open a shunt. And once the shunt opens, the thrombus has a free way to go into the arterial side on the left atrium and left ventricle and can go any place it wants.

This is the patient we took care of about eight months ago. Lady came in, was sent to us with the diagnosis that she has severe pulmonary hypertension and acute left leg ischemia. So her pulmonary hypertension was just labeled as idiopathic. She got her leg treated because she had an acute occlusion. So we took care of the arterial clot. And the next day, I came around. I was like this just doesn't make sense. Something is wrong.

So I called the cardiologist who was on call with me that day. We got an echo on her, like this just doesn't look right. The right heart pressure is high, but we have no reason why it is. Scanned her chest, this is her PE. That's her main pulmonary artery, and these are her right and left pulmonary arteries. This thing was about 80% occluded with pulmonary artery. She had a large infarct on the right lung already because this probably was going on for a week or two, and she was labeled as a pulmonary hypertension problem.

So we lysed her, got a pretty good result, actually. She had a shunt which was wide open. And once we lysed her, she came off flow line, came off the vent in six hours. And her shunt actually closed on its own once her right heart pressure was normalized. So some unique things that I don't know we see more of them because we're getting smarter, or we're just noticing more of them for some change in patterns.