GARY GERSHONY: Good morning, everybody. Welcome to our monthly cardiovascular grand round series. It's my pleasure to introduce our speaker today, Dr. Tanveer Khan, who is one of our esteemed cardiovascular cardiothoracic surgeons. He's had extraordinary training, both on the clinical side and research side.

Amongst his accomplishments and training is doing a cardiovascular research fellowship at Harvard. He's done training at Stanford, at some of our most illustrious institutions, and spent some time, as a faculty member, at UCLA, involved with the transplant service there.

My personal involvement with him is he's my partner on our TAVR program. And we do many cases together. I have the pleasure of working with him as much as I do many other cardiologists.

And he's going to speak to us, today, about another area of interest of his, along with his colleague, Dr. Veeragandham. They have developed a very robust practice treating complex aortic disease. And he will give us an update, today, on the management of disease of the aorta. Thank you, Dr. Khan.

[APPLAUSE]

TANVEER KHAN: Thank you very much, Gary. And thank you to all of you for coming in this morning, bright and early. And I'll be talking about diseases of the aorta and focusing on aneurysms and dissections. Now these are the most common pathology in aortic aneurysms that we see.

So I'll start with what we call AAA or Abdominal Aortic Aneurysms. And again, I think it's important that you know the AAA term is really applied mainly to the abdominal aneurysms, because we also see thoracic aneurysms, as well, very commonly here in our service.

So they're generally considered aneurysms when they're greater than 3 centimeters. And the vast majority of the abdominal aneurysms are infra-renal, so below the renal artery, as you see there. And 40% involve the iliac arteries. So it's pretty common, almost half involve the iliac arteries.

When we're scanning them, we want to scan the entire torso, chest, abdomen, pelvis, that's why we always order those, commonly, because 20% to 25% involve the chest, as well.

So what are the risk factors? And there will be a quiz at the end, so pay attention to some of these points. The risk factors-- so age over 65, and these are older men. They're most often Caucasian and smokers.

Smoking is a big risk factor, not only for having an aneurysms but for enlarging and for rupturing. So smoking is a big risk factor. If you can stop patients from smoking, actually, we can reduce the rate of expansion and the risk of rupture. So it's really important.

Also, family history, of course, this can be familial. A few of the these are related to smoking and hypertension, hypercholesterolemia, and other risk factors, as well.

So how do we diagnose these? So with the common use of imaging, most often, these are diagnosed with ultrasound or CT. Sometimes patients, who have abdominal pain, back pain-- before we did a lot of imaging, in the past, patients that have compressive symptoms, because the aneurysms would press on adjacent organs, that's less common now, because we pick them up before symptoms develop, most often.

Ultrasound is used for screening and usually initially diagnosing it. But it can underestimate it by a few millimeters. Once we have a diagnosis of ultrasound, then we use CT scans to diagnose the AAA. We use 3-D reconstructions not only to assess the size but for planning the repair with that stent graft, so we need really precise measurements.

One of the downsides is the radiation and contrast exposure with CT. So we do these ultrasounds when possible. But we do need CTA. It's really one of the core steps in managing these patients.

Screening, I think, is particularly important. I think this is good for all of us to take a minute to look at what the recommendations are, on screening, to find patients before they rupture. So the Society of Vascular Surgery has made recommendations that men, who are older than age 65, should have a one-time abnormal for screening. And patients who are younger, down to 55 for men, we may take a look at them with ultrasound, if they have a family history, or if they have a very high rate of smoking.

For women, they recommended age 65, if they have family history or if they are smokers. Because aneurysms are much less common in women. And also, their rate for rupture is less common, as well.

So is this covered? Well, Medicare will cover part of this population. If they have a family history, and they're a male, age 65 to 75, and have smoked, Medicare will pay for this, without any other symptoms. But often, there are other symptoms or findings that will be an indication.

So generally, it's almost always covered. But Medicare has actually gotten involved and said they will pay for an asymptomatic screening for this patient population.

Has screening worked? Well, it appears it certainly has. There's a randomized trial that looked and compared to patients in the UK, who were screened versus those who were not.

And they found that the patients that were screened, they identified they had aneurysms, and they prevented rupture. And you could see that the AAA mortality was reduced. And this is up to 13 years, so there's significant reduction, in mortality from AAA, for patients who were screened.

So actually, they identified patients. And they monitored them with ultrasound and the CT and treated them and prevented rupture and death just by the screening program.

So when do we treat? Again, 5.5 centimeters, that's basically been the standard for many years. And that was from early studies that showed the rate of rupture increases dramatically when the aneurysms exceed 5.5 centimeters.

And you ask, well, now, we have stent grafts. And why don't we treat them when they're smaller? Well, they've looked at that. They've looked at studies where the aneurysms are 4 to 5.5 centimeters. There were two major studies.

The patients had stent grafts for aneurysms, in that size, versus patients who were just watched and then treated when they got to 5.5. And they actually found no difference in mortality. So whether you treat them at 4 or you wait till they get to 5.5, there's no difference.

So we still treat patients, all of them, until they get 5.5. One subset is women, because they tend to rupture at a smaller size, so 5 centimeters for women and 5.5 for men.

The operative mortality for endovascular aneurysm repair is very low, less than 1%. It's really a remarkable procedure. Patients can go home the next day or the day after. We had a patient who had a ruptured AAA, who had an endovascular aneurysm repair. I saw him in the office one week later. He's asking me if he could play golf. This is from a ruptured AAA. They used to be in the ICU a week later, when you do them open. So it's pretty remarkable.

Open surgical repair, we still do. It's mainly for patients who don't have anatomy suitable for endovascular. And the operative mortality is around 4%.

So how did endovascular compare to open? Well, you can see. This was a trial. If you look at the top curve, survival curve, the probability of death, you see that, endovascular and open, the probability is the same out to eight years.

So we are able to treat these patients at similar survival, with an open or endovascular. So what this shows is that the endovascular repair is as durable. And it gives you a safe result as the open repair with much less morbidity.

Endoleak-- we've all heard about the endoleak. So what are these endoleaks? Well, the Type I is one that we don't want to leave the special procedure suite until we fix this. This is one that's actually leaking from above or below. It's going around the stent.

So if we put a stent in, and they have Type I leak, we haven't really done a good job, because we don't want any leaking coming from above or below, going around the stent.

Type II is not uncommon. You can see it in about 10% to 20% of CTAs post-op. 80% resolved in 6 to 12 months. And this aneurysm still has branches. So these branches, they back-bleed into the sack.

The only time we really need to fix these is if this Type II endoleak is causing the aneurysms to enlarge. Otherwise, we can watch them. And there have been studies that have shown Type II endoleaks, with no aneurysm enlargement, have almost 0 risk of rupture. And they can be watched. They can be watched and monitored as we do all other patients.

A Type III leak and Type IV leak, we don't see very often. This is when there's actually some leakage through the graft by different mechanisms. But it's not a very common. Type III actually can also include going between the pieces or a small perforation. And this is also pretty uncommon. But it does happen. And we treat this by relining the graft with another stent graft inside.

How do we monitor them? We check a CTA at one month. If they have a leak, then we check it at six months. The Type II endoleak, that would be. And at one year, we start to check them annually.

There have been some protocols that have been looked at to avoid contrast and radiation for patients. So some centers, after one year, if there's no endoleak, and the patient has low risk for other possible complications of the graft, such as occlusion of a limb-- Like if one limb becomes a little bit narrowed, by some occlusive disease, and you may be worried that it may clot off, you may monitor them more closely. But if everything looks pretty good, you can transition to ultrasound, as early as one year, in some centers. That'll save radiation and contrast.

So what are the other abdominal aneurysms you hear about? Suprarenal and juxtarenal is when the aneurysm goes up to or above the renal arteries. Thoracoabdominal, it goes from the chest down into the abdomen. Or iliac, I mentioned that 40% involve the iliac arteries.

We have a number of ways to treat these, either open or with very specialized grafts, such as this one that involves the external and internal iliac arteries. This graft here, you got branches going into the renal arteries or an open repair, which we do on occasion. And these are much less common aneurysms. We have both open and endovascular approaches to these aneurysms, as well.

Make sure everybody's awake. So the first question, what is the risk factor for abdominal aortic aneurysm?

AUDIENCE: Male gender.

TANVEER KHAN: Male gender, smoking, age over 65. Those all sound like they would be appropriate. So I would probably put d, but you'll all have a chance to answer that later.

So now, moving into the chest, thoracic aortic aneurysms. So again, these are abnormal dilations. And all aneurysms are related to medial degeneration. The elastic fibers in the middle layer, they degenerate. So these are degenerative aneurysms.

They're not necessarily atherosclerotic aneurysms. We call them, degenerative, because that middle layer degenerates, and that allows the aneurysm to expand. And that they're not as common as abdominal, about 6 per 100,000. Again, these are elderly patients or older patients, with COPD, often smoking, and connective tissue disorders, such as Marfan syndrome. These are risk factors for thoracic aortic aneurysms. Some

These patients are our patients that come in with symptoms a little bit more often than abdominal aortic aneurysms. Because patients tend to have ultrasounds for other reasons, in their abdomen, and CT scans, but not as often of their chest. So sometimes, patients will come in with back pain or chest pain. It's rare these days, again, for aneurysms to compress on adjacent structures.

But before we did a lot of imaging, patients would have hoarseness or have trouble swallowing, because the aneurysm would be pressing on the aerodigestive tract.

Any patients with symptoms, in a thoracic aortic aneurysm, it doesn't matter what the size is, they should be treated. Because the mortality rate of a ruptured thoracic aortic aneurysm is very, very high. It exceeds 90%.

So risk factors of rupture are similar to the risk factors of having the aneurysm. Older patients, if they're increasing in size, and, certainly, if they have pain, that's a sign of impending rupture.

CT, again, is the cornerstone of a diagnosis. We use a chest CT with contrast CT angiography. We use reconstructions, again, to diagnose and plan our procedures.

Now, for these patients, how do we monitor them? So in the chest, we monitor them every six months, if they have a thoracic aortic aneurysm, to detect for growth. And for thoracic aortic aneurysms, they tend to grow at a slightly faster rate than they abdominal aneurysms.

So the thoracic aneurysms will grow almost a half a centimeter per year, abdominal aneurysms a third of a centimeter per year. So what's rapid growth? Well, if they're going closer to a centimeter per year, that's very fast. Anywhere from 6 to 8 millimeters per year, that's considered rapid.

That's also another indication, because patients are of different size, as well. So if you have a very small person, and their aneurysm is only 4 centimeters, but it's gone from 3.2 to 4 in a year, then you'll want to fix that. Even though it hasn't reached 5.5, it's growing rapidly.

So risks of rupture-- in the thoracic aorta, 6 centimeters has traditionally been the limit of where we considered fixing these. And one of the reasons is repairing a thoracic aortic aneurysm, traditionally, with an open repair, is a very big operation. It involves opening the chest through a thoracotomy, sometimes a thoracoabdominal incision, often using the heart lung machine for support, sometimes even circulatory arrest. So it's a very big operation.

Even abdominal aortic surgery is also a significant procedure. These aneurysms we would treat at slightly bigger, at 6 centimeters. Again, once you get to that 5.5, 6 centimeter range, that's when you see an exponential rise in the risk of rupture. So that's the time when we fix these aneurysms. And again, the mortality rate for thoracic aortic aneurysm rupture is very, very high. It exceeds 90%. It's really, essentially 100%.

So with the thoracic aorta, how we fix them really depends on where the aneurysm is. The aneurysm can be in the root next to the valve. It can be in the ascending, above the valve, but below the blood vessels that go to the brain, brachiocephalic vessels.

It can be in the arch itself, which is complicated, because you still have to fix this, but you still want blood supply to the brain and the arms. And it can be beyond this left subclavian artery in the descending thoracic aorta. It can extend past the diaphragm and, as I referred earlier, thoracoabdominal aneurysm.

So I'll start with the aortic root aneurysm. Again, here, the main thing is that this does involve the valve. So it involves the valve. You either have to replace the valve or repair the valve and re-implant the coronary arteries. So this is a bit more complicated procedure to fix.

Right now, there is no endovascular stent graft approach for this because of the proximity to the heart, the coronary arteries, and the valves. And this is something that may be treated endovascularly in the future. But for now, right now, the standard approach-- really, the only approach is surgery.

The Bentall procedure is the classic approach. You can use either a mechanical valve or a tissue valve. Here, you can see the coronary arteries are re-implanted into the side of the graft. And you replace the ascending aorta.

In some patients, younger patients, you don't want them to have a tissue valve or a mechanical valve. And what we do is a valve-sparing root replacement. And this is a procedure where we actually maintain the patient's native valves.

So we excise the entire aorta. We excise [INAUDIBLE], right down to the aortic annulus, here. We keep the native valves. We re-implant the coronaries, similar to a Bentall operation. But we preserve the native valve, so the patient does not have to be on anticoagulation, for the mechanical valve, or they don't have to have another operation.

Because the tissue valves really only last about 16 to 18 years. So if you have someone who is young-- and we had, a few years ago, in [INAUDIBLE], who was 28, who had an aortic root aneurysm. She had a family history. She had pain.

We did the valve-sparing root replacement on her. And this was her post-operative echo. And you can see that we've preserved the anatomy. You can see the sinuses here and the valve leaflets here. This is her valve after the procedure. So I think it's a great operation for patients who really aren't good candidates for having a tissue mechanical valve.

Although, it's a complicated operation, operative mortality is surprisingly low. It's only 2% to 5%. And you ask, well, what happens with the valve? Does the valve work very well, long-term, if you leave their native valve?

With the valve-sparing root replacement, the freedom from aortic valve replacement-- so their valve is working. You don't have to do an AVR in the future. --at 10 years is 89%. So 89% of the patients do not need any valve replacement in 10 years when you retain their native aortic valve.

So now let's look at the descending thoracic aorta. This is beyond the left subclavian artery. Again, traditionally, the open repair was through left thoracotomy. In some cases, you would clamp between the carotid subclavian. You clamp below.

In other cases, where you really need to see, you put them on bypass and circulatory arrest. You cool them down. You stop the pumps, so you'd be able to see. Because, when you're in the chest, when you're getting up into the arch and down into the diaphragm, sometimes these areas are very difficult to see with clamps on. So you really have to use the heart lung machine, as well.

This, again, is a big operation, significant morbidity. Thoracic Endovascular Aneurysm Repair, also known as TEVAR, this has become the treatment of choice. We see excellent results with this procedure. It has reduced the morbidity and mortality of descending thoracic aneurysms.

And as we see, with the TAVRs and with the TEVARs, we're able to do these percutaneously. So through a sheath, we use a device, such as this device, shown here, which is a perclose. We can use a couple of these. You don't have to make any incisions.

The device will close the access to the artery. So you can actually do this procedure without using an incision at all, just a puncture, and closing the artery with these devices.

So one of the main differences for repairing the descending thoracic aorta is spinal cord ischemia. So we don't see this in ascending aorta, aortic root, or abdominal. This is for the descending thoracic aorta. And paraplegia obviously is a really devastating complication and one that, for patients and their families, is very difficult, similar to stroke.

So again, we will have a quiz later. So what are the risk factors for spinal cord ischemia? So one of the most important ones is hypotension. You're decreasing the blood supply to the spinal cord, so perioperative or post-operative hypotension.

In some cases of delayed paraplegia has been seen up to a week later, when the patients go home or primary care cardiologists restart them on all of their anti-hypotensives. They drop their pressure into the 80s. They become paraplegics. So it's really important that post-operative or perioperative hypotension is avoided.

Extensive coverage of the thoracic aorta, the more you cover with the stent, the more of these arteries, that give blood supply to the spinal cord are covered by the stent graft. And collaterals are important. So yes, well, if we're covering the artery to the spinal cord, how does the spine get blood?

Well there are collaterals that come from above and come from below. So often, you'll see patients. They come to the ICU. They've had a carotid subclavian bypass first, then the stent. So if you have to cover the subclavian artery, we still want blood to get down to the spinal cord. So you do a bypass from the carotid to the subclavian, so that can give blood to the spinal cord.

Also, one of the risk factors I listed here is if they have had aorto-iliac surgery. So you've done a stent graft or open repair or they occlusive disease, so that's covered the blood supply from below. So you're getting less collaterals from below, as well.

So anything that disrupts the blood flow to the spinal cord increases the risk. And what levels? Well, T8 to L2, that's really the main risk. So when you have extensive coverage, your stent goes from the subclavian all the way down into the upper abdomen, you're covering those lower levels, T8 to L2, that's when the ischemia risk is particularly high.

How do we prevent it? During these cases, we do neuromonitoring. We check for any changes. If there's any decrease in the motor potentials in the lower extremities, we increase their blood pressure, with [INAUDIBLE]. We're working together with them.

We also CSF drainage. So what's the perfusion pressure of the spinal cord? Well, it's the Mean Arterial Pressure minus the pressure of the CSF fluid around it. And so, with a spinal tap, you drain the spinal fluid. It's a similar concept. So you drain the fluid around the spinal cord. It increases the amount of blood that can get through the spinal cord.

So what we want is a CSF pressure of at least 10 or less. If our MAP is 70 to 90, that gives a spinal perfusion pressure of 60 to 80. So after these procedures, we usually like to keep MAP 70 to 80, generally. If they're having any signs of weakness, then we bump that MAP to 90 to 100, and we drain their CSF fluid.

So, again you want to avoid hypotension. Oxygen delivery, that's the key. You're depleting the spinal cord of oxygen. So hypotension, hypoxia, and anemia, oxygen delivery, you need red cells, you need oxygen, and you need blood pressure to get to the spinal cord. So you really need to avoid all three of these, particularly if somebody is symptomatic. At

So how does the TEVAR compare to open repair? When Well, you can see. On the right side, mortality and paraplegia rates are significantly lower with TEVAR versus open surgical repair. ICU stays one to two days, hospital stay is reduced, as well.

Endoleak we see in thoracic aortic aneurysm treatment, as well. That's at 5% to 15%. That's something that we follow. We follow them with CT angiograms according to a similar schedule. And the nomenclature for endoleaks for thoracic aorta, basically the same. Type I is from above or below, Type II through the branches, which we follow. Type III and IV are leaks that go through or between the pieces.

So the last type I'll talk about, of the thoracic aneurysms, is the aortic arch aneurysms. These are complicated, of course, because they involve the blood supply to the brain and the arms. So we treat these with open repair. That's been the standard approach.

You can see what we need to do, though, is, in addition to the graft that will usually end here-- or we will do what is called the hemi-arch, sometimes, and bring the graft diagonally across. If the aneurysm really extends completely through the arch, you actually have to re-implant the innominate artery, the left carotid, and the left subclavian.

So this is a complicated procedure. It requires cardiopulm bypass, circulatory arrest. You put the patient on the pump. You cool them down to 18 degrees Celsius. And you stop the circulation. So you stop the pump. Initially, the patient has no circulation. There's no blood flow. They're just cool.

Well, how do they survive? Well, this is similar to the reports of patients that have fallen in a frozen lake for quite a long time. When they recover, they will be revived and survive, because their metabolic rate is so low.

And what you see, here, this actually shows the esophageal temperature. And that's what we follow. So we've cooled them down to 18 degrees Celsius for circulatory arrest. And this is the metabolic rate. So as you see, as you cool the patients down, the metabolic rate comes down, significantly. So at the 18 degrees there, their cerebral metabolic rate is only about 20%.

So why aren't we able to use TEVAR for arch aneurysms? As we saw, they reduce morbidity and mortality in the descending thoracic aorta. Well, you need a neck. You need something to land on. You're not able just to cover these branches up to the brain and the arms.

You need, usually, a 2 centimeter landing zone above and below the aneurysm to create a watertight seal, so to speak, so you don't get the Type I leak. You don't get blood leaking into the aneurysm.

So we can get around this, in some patients, with a hybrid approach. It combines a sternotomy. And we call it debranching, because you're debranching the aortic arch. You're moving the branches to a different location, so that you can cover that aneurysm with the stent graft.

So what we do is we sew graft on to the ascending. Here is the valve. Proximal to the-- can you see the--

AUDIENCE: No.

TANVEER KHAN: Well, since it's be recorded, unfortunately, we can't use a laser pointer. But proximal to where it gets the valve vessels and the aortic arch vessel, we sew grafts.

AUDIENCE: [INAUDIBLE]

TANVEER KHAN: There we go. Great. So we sew a graft here. And this graft has branches, so we can give blood supply to the brain, from the ascending aorta. And we can bring a stent graft up and around, up to this. So blood supply is still getting up to the brain. And we have stent grafts covering the arch.

So with this hybrid approach, combining a bypass, essentially-- we call it debranching in this case-- and a stent graft, we can avoid doing an open operation.

Another type of debranching we do more commonly is just debranching of the subclavian. What we'll do is we'll often bring up a stent just to cover the subclavian. But again, we want the blood supply from the subclavian artery to get down to the spinal cord.

So what we'll do is we'll bypass. And you see this. We have a small incision in the neck. We bypass from the carotid into the subclavian. And we can bring the stent graft right up to here. We don't want blood going up to the carotid, down to the subclavian, and then back down, and leaking into the aneurysm, so we've blocked this.

And we do this with a vascular plug, so that blood is only going forward. It's not going back down onto the stent graft and getting into the aneurysm. So this is one of the more common bypass procedures that we will see to allow stent grafting.

And with aortic arch outcomes, we see the mortality is similar, because, even though the open repair is a big operation, these patients are pretty sick. And opening their chest, debranching the brachiocephalic vessels, the mortality has been similar. But the stroke rate has been significantly less. And overall, the morbidity for a procedure, where they're not on pump, they're not on circulatory arrest, is much less.

In the future, we have branch grafts that are currently on the way and have been in trials, where we'll be able to have stents coming off the main body, itself. So we'll be able to extend the stents up into the-- here, it shows the innominate artery, as well the subclavian and carotid artery.

And these are similar to the branch grafts that they do in the thoracoabdominal. So again, we have the different types, the root aneurysm, the ascending aneurysm, arch, and descending. The location really determines how we treat these.

So arch aneurysms can be open, but we can do a hybrid approach for greater than 6 centimeters. Descending thoracic aneurysms, again, these are distal to the subclavian. The endovascular approach, TEVARs, have really become the standard of care. And generally, we treat these for over 6 centimeters.

The ascending aorta, open surgical repair is still the standard. And we treat these for over 5.5. And the ascending and the descending, these are the two most common. As you'll see, the ascending is 45%, descending 55%. So 90% of these are either ascending or descending. The arch are pretty rare. Thoracoabdominal are also rare at 10%. And these can be treated with either open or with branch grafts, as you saw before.

So another question for you, for the quiz. So what is the risk factor for paraplegia after thoracic aortic stent grafting? So a is perioperative or postoperative hypotension. b is extensive coverage of the thoracic aorta with stent grants. c is prior abdominal aortic surgery with stent graft. Or d is all of the above.

Certainly hypotension, extensive coverage, as well, you're covering the blood supply. And prior abdominal surgery, you're reducing the collateral coming from below. So I think all these three are significant risk factors for paraplegia. And these are the patients that we're more aggressive with using the CSF drain and maintaining their blood pressure.

Now we move on to aortic dissection. So this is really one of the most catastrophic medical conditions. The overall mortality rate is up to 30% even in the best centers. And it requires really optimal management to prevent serious morbidity and mortality.

So I like history, so I put a little bit of history in here. So this was first described, actually, in 1761, by Morgagni. And what's the pathology? It's somewhat interesting. So this shows the three layers of the aorta. This is in cross-section. This is the inner layer, the intima, the outer layer, the adventitia, and the media.

See here, you can see this is split. So this is the intima here. This is the media. So the media is split. And blood has gotten into the media, so the middle layer. These are blood cells. So it's separated. So the dissection's a tear, but it's a longitudinal tear. So it's going down the barrel, right in the middle layer.

And this is a post-mortem, of course. This aorta is filleted open, actually. And so what you're looking at here is these are the brachiocephalic vessels. And this is a Type B dissection, because this tear is just past the subclavian artery.

So what happens? You get separation of the layers. You get a true and a false lumen. So the true lumen is where the blood normally is. The false lumen is where the blood we saw in the media. That's a false lumen. So you have blood going in two spaces.

What happens with these tears, though, is that the blood will not only go antegrade and down the [INAUDIBLE], but it can also go retrograde. That's always the Type A dissection. It's really a lethal problem, because the tear can go proximally down towards the heart and disrupt the valve. It can tear the coronary arteries. It can cause a rupture, causing pericardial effusion and tamponade.

So the extensions can go proximally and distally. It often happens in a spiral. And the other thing is, once this main tear happens, you get multiple other reentry points. That's how we have to follow these patients, so we can fix the tear, either with a stent, with a Type B, which we'll talk about, or a Type A, you do open surgery.

But since you have the additional tears, the rest of the aorta is at risk for aneurysm or enlargement over time. So you have to follow these patients, because they get multiple, other, little tears.

Because you can imagine, if there's only one entry point, and the blood is flowing into that tear, unless there's a re-entry point for the blood to come back into the lumen, it will basically have a tendency to rupture. Because the blood has to go somewhere. It can't be pumping down a cul-de-sac, a blind end. So that's why you get multiple re-entries.

So classification-- so Type A and Type B, this is the most commonly used when you're outside of Texas, and Texas and some of the other surgeons, still tend to use the DeBakey classification. But out here and most commonly, even in the literature, they use the Stanford A and B.

So Stanford A, this is a surgical emergency. It needs open repair. This is one the tear is in the ascending aorta. Here, you see the tear is just above the aortic root, above the valve. This is the true lumen in red. This is the false lumen, where the tear and the blood propagate.

And as long as the tear is here, this dissection, it can involve the entire aorta, which we see most often going all the way down into the abdomen, or just the ascending. But it's still a Type A, because Type A is not how far the tear goes. See here, this is normal aorta, but this tear goes all the way down here. And the dissection goes all the way down there. But it's where the actual tear is, because that determines what your treatment is.

The treatment for these two are the same, because the tear's in the same place. Even though the dissection goes all the way to the abdomen, you treat them the same way. Because this is what you need to fix.

Type B is distal to the subclavian. The subclavian artery is Type B dissection. Here, you see the dissection extending distally. And those can be treated, initially, medically if there are no complications.

So what are the risk factors? Well, hypertension is probably the most common clinical association. Almost all of these patients, particularly with the Type B dissections, they come in, and their blood pressure is like 180 or 200. And you will see that in the emergency department. Their blood pressure is always high.

Part of this is the pain. I mean they have feeling of intense impending doom. They're having severe chest pain. But they all have severe hypertension. Connective tissue disorders are not common, but they are one of the risk factors, as well bicuspid aortic valve and coarctation of the aorta. These are congenital problems that are risk factors for dissection.

And also, once in a while, we do see dissections of cardiac caths. It's not very often, but it does happen. It's something to think about it if patients have persistent chest pain after an intervention.

What I actually I find important about diagnosing this is that chest X-ray is almost always abnormal. So you don't need the CT scan to really have a high clinical suspicion, if someone has a widened mediastinum, as you see here.

If they have severe pain, it's usually the upper chest radiating and back between the shoulder blades. You have to be worried about aortic dissection. They can have AI, because the tear can disrupt the aortic valve, so they'll have a new murmur. These are things to worry about.

I thought this was also particularly important data. So what are some of the predictors of who does well and who does not? Because we see a lot of dissections here.

In some of our patients, we repair the ascending. They come up. They're looking like a rose. They do great. And they go out of the ICU in a couple of days. And they go home in maybe three to five days. Others with the dissection, they come out, and they're barely hanging on.

And what are some of the things that really determine this? Well, one is how has the dissection affected the blood supply to the critical organs? Has the dissection extended into the carotid arteries, into the kidney, the renal arteries, into the intestines, into the legs?

So this study actually came out of Stanford. I thought it was particularly important as we'll see. Because you would think that a stroke would have a very high mortality. And it does, 14%. However, if you look, the highest mortality is for patients with visceral ischemia.

So if they have abdominal pain, I mean this is a particularly bad sign. And we've seen this. Patients who come in, their main complaint is severe abdominal pain. They don't do well. Because their mesenteric arteries have been dissected. Renal ischemia, as well, because having acute renal injury, often, the mesenteric arteries are involved. And these are particularly lethal dissections.

Pulse deficit-- we do get worried about patients who don't have pulses in their legs. However, mortality is less than patients with visceral ischemia. And most often, patients, once you fix the ascending aorta, when you get rid of that proximal tear, actually, the legs will reperfuse post-op. You'll get the pulses back.

But the gut and the kidneys, they're not as tolerant of ischemia. And those, patients even if you can reestablished flow to the gut and the kidneys, once they've had that initial insult for several hours, it's hard for them to recover and survive.

So diagnoses, again, CAT scan, as we all know, is really the gold standard. And that's how we diagnose and plan these operations. TE is very useful, as well. We use these in some cases where the CT is equivocal.

And we use this in the operating room to help us with looking not only at the dissection but looking at the heart, seeing if there's AI, how much AI there is, if we have to repair the valve. And also, it helps us to look at the true and false lumens.

Because when we put them on the heart lung machine, we want to make sure that the blood flow is going into the true lumen. If we cannulate and the blood flow is going into the false lumen or making this tear worse, then we can end up with a much bigger problem on our hands.

So the data that came out of Stanford looked at the overall survival. This is all patient Type A dissections, whether they were operated on or not. 1-year survival is 67%. At 10 years, it was 37%.

And why do the patients die from Type A dissections? Well, acute AI and left ventricular failure is one, myocardial ischemia that affects the coronary arteries, rupture, of course, and then, like I talked about, branch occlusions. These are the main causes of death in Type A dissections.

It's still a very significant operation. And it's difficult for patients to recover from this, even when they come in timely and have a good repair. The operative mortality, even at the best centers-- the IRAD data looked at that 25 of the busiest aortic centers in the world, and the operative mortality exceeded 20% at these centers.

Interesting, in 2014, a study came out of Duke that looked at how you can improve outcomes for Type A aortic dissection. And what they did, they had a number of surgeons, more than a dozen surgeons doing aortic dissections. They had a limited number of surgeons do the dissections, which increased the volume.

And what they found is that, at centers that have a volume of more than 12, which is considered high volume, which is not that many, and it could reduce their operative mortality down to 5%. And as you see, at our institution, sometimes, we see two or three dissections in a month. So we are actually considered a high volume center. High volume is more than 12 aortic dissections.

And so I think this was encouraging data, that, when you have a lot of experience with these, even though the operative mortality can be very high-- because, at most places, surgeons will do one per year, maybe two per year. At our center, we're seeing a couple a month. So I think that we've seen that in our results, as well. And that's encouraging.

Type B dissection, again, these are dissections that start distal to the left subclavian artery. And the cornerstone of treatment is reducing the pressure and the rate of the rise of the pressure. They call this the dP/dt. To reduce the pressure, you can do that with vasodilators, Nipride or Cardene. When you want to reduce the rate of the rise of the pressure, the beta blockers are the best way to go. And Esmolol is the one we use most often.

So medical treatment-- since we have stent grafts, and the Type B dissection, the tear is beyond the brachiocephalic vessel, so you can ask, well, why not treat everyone, with a Type B dissection, with a stent graft? Because you can cover that tear. And you don't have to interrupt the circulation to the brain.

Well, they looked at that. And the main indications now, for treating a Type B dissection, beyond medical treatment, is if they have complications. Because they looked at patients who didn't have any complications of the dissection, and they looked at patients who were treated or were treated with medicine or treated with a stent graft. And actually, there was no difference in mortality.

Actually, the mortality, as a trend, it wasn't statistically significant. But there was a trend, early on, for patients who had a stent graft for Type B dissection, without symptoms, to actually have a slightly higher mortality.

What they found is that, when they have these tears, the aorta is very fragile. It's like soft tissue paper. And when you put a stent in, that can actually cause what's called a retrograde dissection. So you can put your stent in, and just the force of the stent can tear the aorta, proximally. The tear will then go retrograde into the arch and back into the ascending aorta. So you convert a Type B into a Type A.

So in cases now, where we have Type B dissections with complications, which is an indication for a TEVAR, now, we try to wait and temporize them as much as we can, to let this aorta heal a little bit, to prevent complications, early on, from the procedure, itself.

So Type B dissections, medical treatment, that's the main stay. That's how we treat them. When we do use stents for Type B dissections? If there's malperfusion, if they have a cold leg or they have intestinal ischemia, if they're having severe pain telling us that this is about to rupture. Or if we're following them, and even in the hospital, we'll get a CT scan three to five days later, and the aorta is even larger than it was when they came in, these are reasons that we need to fix this aorta before it ruptures.

And how do we do that? Again, with the Type B dissection, the tear is here, just past the left subclavian artery. We're able to put the stent grafts off and just past the subclavian. Sometimes we have to cover the subclavian and do the cross-subclavian bypass, which I talked about before.

And the key here is you cover this is this main tear, and you have thrombosis of the false lumen. We often see distal tears, like I've talked about, and so we have to follow the patient. Because these distal tears, they can cause enlargement further down.

But what we see in patients, who have a thrombosed false lumen-- even in patients who don't have a stent, if your false lumen thromboses, spontaneously, your increased rate of expansion of the aorta-- so your risk of having an aneurysm or other problem is much less in patients who have what we call a double barrel.

So when you see patients who are being followed for this, if you see this on the scan, you see a true lumen and a false lumen, open still, that's much more worrisome. Because these patients can have a higher risk for this aorta to enlarge.

If we're following them, even without a stent in place, if you see that this false lumen has thrombosed, these patients have a tendency to do much better, have less risk of aneurysm and enlargement.

So again, this data is from Stanford. When you looked at the overall survival of Type B dissection, these are patients who were treated medically or with stents or with surgery. Overall, the survival was 71% at 1 year, and 35% at 10 years.

If you look at how has TEVAR made a difference in treating patients with Type B dissection with complications, well, medical treatment, early mortality-- so this is in hospital mortality, 30-day mortality-- is still 10% to 20% mortality for patients treated medically with Type B dissections.

If they have complications, their mortality is much higher, 40% to 50%. If you're able to treat a patient with a thoracic stent graft, that reduces their mortality down to level you would see without complications. So their mortality is only 10% to 15% if you can treat their complications with a thoracic stent graft.

So again, we need to follow these patients long-term. And these patients, we follow a little bit more closely at every three to six months for the first year. Because, again, there are these other tears. You can have aneurysm enlargement. And you want to make sure. And this is a very friable, fresh tear. So these patients, sometimes can expand rapidly. You may need to treat them with additional stent, at times.

Also, you want to get an echo annually, to make sure that you don't have any problems with the root of the valve, particularly in patients who have had involvement of their ascending aorta in that repair.

So the last question-- so what is most appropriate treatment for an acute, Type B aortic dissection? So this is acute Type B. This is a tear past the subclavian artery. And there's no complications. There's no kind of malperfusion. They're not ruptured. They don't leak.

So emergency surgical repair, either open, endovascular. Medical treatment with blood pressure control and monitor them with a CTA. Or none of the above. c is right, so everybody is still awake. That's good.

So emergency open repair, it's really an operation that's not done anymore. if someone needs an emergency repair for a Type B dissection, that's done with a stent graft. Emergency endovascular repair, we do. But again, we try to temporize them. Even if they have complications, we try not to do it, right away.

We want that aorta to heal a little bit before we put the stent graft in. And medical treatment, get them on Esmolol, get them on some Nipride, watch them in the ICU. We check a CT in a few days, make sure nothing bad's happening, make sure they're not leaking, it's not expanding. And then you can follow these patients over time. They can always be stented when needed.

Again, thank you, everybody, for attending. It was a pleasure to present at the cardiovascular grand rounds. And I appreciate you all coming to hear me speak.

[APPLAUSE]

GARY GERSHONY: Thank you, Dr. Khan, for an excellent presentation, really helping to simplify a very complicated topic. Are there any questions?

I have one, actually. So the early data, looking at endovascular repair, stent grafts for abdominal aortic aneurysms compared to surgery, suggested an early benefit that was lost later on and actually, potentially worse.

More recent clinical trials, perhaps, with improved operator experience, improved devices, suggested that out to longer follow up, 10 years or beyond, they seem to be comparable. Yet, that still begs the question, since the intraprocedural and in-hospital complications and mortality are probably lower with stent grafts.

Is there still a concern over a late catch up, with endovascular repair, that if we continue to follow the patient down long enough, that they may do worse than surgery? And what is the reason for that catch up, where the curves tend to become more parallel later on?

TANVEER KHAN: So again, that's a great question. The initial studies showed that the operative mortality and the morbidity was similar for the stent grafts versus open repair. That's in terms of mortality or aneurysm rated mortality. Morbidity was less, for the stent grafts, of course. But the mortality was similar.

However, when they followed the patients further out in the initial trials, the mortality was higher for stent grafts. And a lot of that was due to the secondary interventions that were needed. And what they found was that patients would have endoleaks. The grafts could migrate. They shifted down. Or sometimes, the patient's own anatomy, they had iliac aneurysms that expanded.

So they needed further interventions, which have their own complication rates. And there's some risk rupture down the line, because these stent grafts, they didn't have as good long-term durability.

With the most recent trial that came out of Dartmouth, Dr. Schermerhorn, his group-- and I trained with him, actually, in Boston-- looked at patients out up to eight years. And what they found is that, in the more recent data, that the survival curves no longer diverge. So the survival curves are similar for open and endovascular repair.

And there are a couple of reasons they thought for that. One of the reasons was that now the stent grafts, the technology is better, like we see in TAVR. We're seeing less AI with the valve. Now we're seeing better technology. We're seeing third, fourth, fifth generation stent grafts, less endoleaks.

And I think another reason, which they noted, is overall, in the population, just the prevalence of abdominal retainers is going down. So the size at which they're treated is smaller. So they found that the larger the aortic aneurysms have a higher risk for having grafts being in the wrong place with time, having endoleaks.

But when you treat them smaller, there's less space for the graft to move around. So they tend to be more stable. So they think that's from earlier detection, better smoking cessation, a number of reasons. But the number of aneurysms is going down. They're being treated when they're smaller.

The other thing that contributed to this is that that's how, on the stent graft side, we see better outcomes. The other is that they found that they followed the open repair patients, a little bit more closely, and looked at different end points. And with the open repair patients, they looked at the stent grafts.

But they looked at additional abdominal procedures that needed to be done on patients with open surgical repair, hernias, laxative adhesions for intestinal obstruction, and so on. And when they looked at that and included that, then they found that the difference in mortality or aneurysm or procedure-related mortality was similar.

Because now they're counting the fact that patients, who have open operations, they can have problems not only from an aneurysm rupturing, but they can have problems from an intestinal obstruction or other reasons.

And so when they looked at that data and included that, and, also, the difference in the overall sort of aneurysm population and how they're treated, now we see that the survival is similar between open and endovascular repair. You still get the benefit of having a less invasive procedure, early on, and up front.

GARY GERSHONY: Any other questions? Any questions from the other campus? [INAUDIBLE]

COMPUTER: All guests have been unmuted.

AUDIENCE: [INAUDIBLE]

SPEAKER: Walnut Creek, do we have a question? Any questions? No questions from Walnut Creek at the moment, thank you.

AUDIENCE: [INAUDIBLE]. So does that mean that they're [INAUDIBLE]?

TANVEER KHAN: The radial pulses and the carotid pulses should be the same. So even though you changed where the inflow is into the branches, the runoff will be the same. So in patients that have debranching done, they will have the same carotid and radial pulses.

In some patients, we do cover the left subclavian artery and don't do the carotid subclavian bypass, because it's not necessary to do that in everyone. And you remember, in pediatric cardiac surgery, when the original repair is for coarctation of the aorta, there's a subclavian patch.

Children, when they're born with a congenital narrowing just past the subclavian artery, how they used to repair this is they would use the subclavian artery. They would ligate it and use it to patch, to patch open the narrowing.

So these babies, they would not have direct flow in their subclavian artery anymore. And they grew up just fine. Their arm was fine. Their brain was fine. That's because the body has a lot of collateral circulation. So you can actually cover the subclavian artery. That's pretty well tolerated.

But we like to do the bypass, mainly because we want to preserve the blood flow to the spinal cord. Some patients, so we don't do it. In those patients, you need to know that, because they won't have a radial pulse. Or it'll be very weak.

AUDIENCE: [INAUDIBLE]

TANVEER KHAN: Any other questions?

AUDIENCE: [INAUDIBLE]

TANVEER KHAN: What kind cardiac clearance is needed prior to semi-elective AAA repair? So a question from Mark Nathan, what kind of cardiac clearance is needed prior to semi-elective AAA repair? So Mark, that's a great question. And I think that's one that's evolved over time.

Initially, we would request a cardiac clearance similar to having an open AAA repair, because there's always the question of whether these patients would need some sort of open procedure. And now , as you know, these patients can be done percutaneously, or without any incision. And the risk is much less.

I think that, when you look at the patient population, even though the procedure, itself, is less invasive, I still think having a cardiac clearance and evaluation, similar to what we would do for patients who have any open vascular procedure, is still appropriate, because their risk is so high.

I had a patient who we did a TEVAR on. We did it totally percutaneous. But because this patient population is a high risk for having cardiovascular problems, the patient had a coronary occlusion and acute MI on the table. And actually, Dr. McWhirter came down and stented the RCA, on the table, after we did the TEVAR. Because this patient had an MI.

And fortunately, he did very well. And we were fortunate that, at our institution, we have such great support. But again, I think that, in this population, despite the fact that these procedures have become very low risk, in terms of being minimally invasive and percutaneous, the patient population is still very high risk.

Well, thank you very much.

[APPLAUSE]