[MUSIC PLAYING]

IBRAHIM SULTAN: There's no relevant disclosures. So the topics really I wanted to cover today were the basics of acute aortic dissection, or current strategies both locally and what are considered state of the art, and how we do in practice here at UPMC and around the country, and what we think the future of acute aortic dissection treatment is.

So the standard call we get is we have a patient with a type A aortic dissection at UPMC Chautauqua or any of our UPMC or non-UPMC affiliated hospitals. As soon as we get the call, even before I answer via MedCall, a helicopter's already on the way, and STAT MedEvac is picking up the patient to bring the patient over to the operating room.

We're one of the few centers in the country where we have 24/7 neurocerebral monitoring available with EEG and SSEPs. And this is critical, really, for all aortic surgery, specifically aortic surgery requires circulatory arrest such as aortic dissections. A TEE is performed by our anesthesia colleagues to confirm the diagnosis. Central lines and arterial lines are placed when the patient's awake, and we get started.

So before I get into how we-- practices and the basic, I always like to pick a little bit of history. One of the first reports of acute aortic dissection was really George II. And this happened so when he was found to be in the bathroom after his breakfast and unfortunately passed away right after.

So an autopsy actually revealed tamponade physiology once the pericardium was open and acute aortic rupture, which was most likely a consequence of an acute aortic dissection. And so I always find that interesting, because a lot of the major articles started off like that. And that was the first known description, at least several hundred years ago.

So acute aortic dissections, as most of us know, are pretty uncommon. Majority of these are type A, meaning involving the ascending aorta. And they can involve both the ascending and the descending aorta and are still considered type A. About a third of these are type B aortic dissections, which exclusively involve the descending aorta. There's a more nuanced classification that includes non-A, non-B, not necessarily relevant for this talk, but this is an evolving terminology.

We see these more commonly in men, and this is predominantly because of the risk factors that are seen, such as hypertension which happens to be really one of the most common, atherosclerosis, aortic aneurysms. So as you can imagine, based on Laplace's law, and this is why we monitor patients with aortic aneurysms very carefully is that as these grow with time that the risk of tear and rupture does increase.

Familiar aortopathies or connective tissue disorders. These are always a major category. And these patients do tend to tear and rupture at a lower absolute diameter as opposed to patients with non-familiar. Bicuspid aortic valve, which, again, is about 1% to 2% of the patient population, which we see a ton of patients, in our aortic clinic.

However, biscuspid aortic valve is not, even though was formerly thought to be a risk factor for acute aortic dissection, it's not necessarily the case to be so. It is a risk factor for developing acute-- for aortic aneurysm but not for acute aortic dissection. And these are some of the data that have been coming up recently. And prior cardiac surgery, there's something about instrumenting the aorta or having prior cardiac surgery that does increase patients' risk at having acute aortic dissection.

So most of these can actually be seen just based on reviewing CT scans. And so as you can go from left to right, the standard classification we use less than 48 hours is hyperacute based on what IRAD has recommended, because that's typically when we feel that the membrane is the most pliable. And that's where patients are most likely to suffer from malperfusion or rupture.

As time goes on for about two weeks or two months, that is, again, a time where the membrane starts to thicken but still is relatively pliable. But after two to three months, it is considered chronic. And again, I think there are a variety of patients that function differently based on the size of the tear, the number of reentry tears, and really the extent of their dissection.

And so one of the major advances of acute aortic dissection treatment, specifically type A, has been really flying these patients directly to the operating room. And the reason for that is there's a clear historic data that indicated about a 1% mortality every hour on the hour that we wait on these patients.

So if you look at these curves, these are historic data from Japan that show how steep this drop off is where nearly 40% to 50% of the patients are essentially virtually dead with non-operative therapy right away. That curve is obviously a little different in type B aortic dissection, which we'll get into a little later in the talk. But for type A, it is still considered a surgical emergency.

So the results from the '90s, which is when really surgeons started being aggressive with acute aortic dissection, really included quite a significant percentage of mortality, because patients would typically go to the ICU and would wait for the operating room after being seen, examined, et cetera. Now with having remote CT scan viewability and telemedicine available, these obviously these things can be expedited.

It is not uncommon for some of the ER physicians to send one of us a CT scan on our phone as a video for us to look at in case there's a doubt or quite frankly use FaceTime from time to time. And so the 1% mortality per hour on the hour, again, it depends on the extent of dissection, reentry tears, and really the absolute diameter of the aorta. In the operating room, there were really no circulatory arrest adjuncts that were used.

And circulatory arrest, again, for the non-physicians in the audience, really defines when we stop circulation to part of the brain and really the rest of the body after cooling down the patient on cardiopulmonary bypass to do aortic arch reconstructions. In the past, there really were no specific adjuncts being used. There's no neurocerebral monitoring to guide when EEG would be silenced. And so the worldwide results were what we call the 30/30 club, getting 30% risk of in hospital mortality and 30% risk of stroke, which is obviously unacceptably high.

And so these are data from IRAD which depict the modern era, which really show that the mortality, although has come down statistically significant and perhaps clinically significant, but still rather high in the 20% range [INAUDIBLE]. This is from 2013, and it really has not changed over the past five to seven years. And this still continues to be the case. Type B, obviously, is lower as expected, but this is on the rise now because of more aggressive endovascular interventions that are being performed.

So as we know, the dissection is a medial event. This is a nice cross-sectional view of the aorta that shows a compressed true lumen because of stasis that causes thrombus formation in the false lumen, really based on Virchow's triad. And so I think this is really, in my opinion, the most important concept of an acute aortic dissection.

And so not all patients present with malperfusion. About 20% to 30% of patients do present with malperfusion. And the reason for that really is the false lumen compressing on the true lumen. And so these are different cartoons depicting the same. And so we can start in the middle where the larger lumen, or the false lumen, continues to compress in the true lumen, which continues to get squeezed.

And if you can imagine, if this was a coronary artery, the patients would present with a myocardial infarction. If this was the carotid artery, patients would present as a stroke. If this was the common femoral artery, patients would present as a cold limb. Hence, the [INAUDIBLE].

As time goes on, these tend to be either static or dynamic or static completely. And as you continue to see more thrombus formation and once the blood flow gets cut off to that particular organ, patients present with malperfusion. So again, not everyone may present with the classic chest pain symptoms, but they may present with an MI, stroke, et cetera.

Now malperfusion is critical to diagnose, because number one, the mortality immediate and long term is very different if patients present with or without malperfusion. And the reason for that is when you have a patient with an acute aortic dissection who already comes in with stroke, or a cold leg, or dead gut, or presumed MI which typically happens to be inferior wall MI, the cat's already out of the bag. And by the time they get rescued and operated on, it is likely that a lot of the damage has already been done.

Now having said that, we can still save a lot of patients. Our paradigm or concept here is that we really operate on every patient despite with or without malperfusion. A lot of the centers do not-- or surgeons do not operate on patients with malperfusion, again, because these data that show that they do significantly worse upfront. But when we see these patients, many of them are in their 40s to 60s. Many of them have a family history. And most of them are quite functional before they present. So it does add another element for us to consider.

So overall incidence of malperfusion, really, is about 20% to 30%. The most common that we see is iliac femoral malperfusion. Sometimes patients will present with spinal malperfusion, will present with paralysis, or mesenteric, or dead gut, or an AKI. This is, again, very common where patients show up, and it is thought the patient is having an MI, are taken to the cath lab. And that's where the diagnosis is made. Cerebral malperfusion, again, when patients present with stroke. Or multiple malperfusion, which does happen about up to 10% of patients. And this is when mortality and morbidity is incremental as you add things on.

So the key concept in acute aortic dissection management, whether it's A or B, is really to correct the malperfusion. So in type A aortic dissection, we do this by putting the patient on cardiopulmonary bypass, establishing true lumen flow, and really expanding the true lumen. The primary technical objective in an acute aortic dissection is to resect the primary tear in a type A or cover the primary tear in the type B. And then, we address other reentry or secondary tears, because these have consequences to how patients do and how their aortic disease does down the line.

Now, to avoid or minimize risk of stroke and risk of organ damage, we do protect the brain during arch reconstruction. And every time we do arch reconstruction or surgery for acute aortic dissection, we do cool the patient down for about 18 to 20 degrees Celsius. We can keep them as warm as 28 degrees Celsius or even a little warmer. But we perfuse the brain when we do so, either antegrade through bilateral carotid arteries or retrograde through the SVC, which really isn't true brain perfusion but allows for debris and emboli to get flushed out in an open aortic arch.

So the conduct of the operation really is to take care of the brain, heart, and the aorta in the process, because without cerebral and myocardial protection when we're doing these long extensive surgeries, it is unlikely that the patients are going to come out successfully.

This is a good example of a patient with an acute type A aortic dissection, which, again, involves ascending and the descending aorta. The patient here has a large pericardial effusion. Likely it will show up at tamponade. You can see how compressed the true lumen is. We call it the pseudocoarctation. And it's not uncommon that most of these present as what we call DeBakey 1 dissections, where the entirety of the aorta is involved. This particular tear was in the distal arch.

So again, the key concept is dissection versus tears. This particular patient had a dissection that started in the aortic root. And it went all the way to common femoral arteries, but their tear or the primary tear that initiated the dissection was only in a couple spots. Most commonly, about 70% of the time, this is in the ascending aorta. But it does happen to be in the descending aorta or the aortic arch from time to time, or the aortic root. And so the goal, again, is to take out the primary tear but not necessarily replace the entire aorta, because as you can imagine, that would be extremely morbid and would come with significant mortality.

This is an example of what this looks like in the operating room. So head is towards the bottom of the screen. Feet are towards the top. This is obviously an acutely dissected aorta. You can see the flap going up and down. And you can imagine why these patients can rupture and die, really, on transport or in the operating room, as you're seeing them. This is a bruised right ventricle, perhaps a consequence of an inferior wall MI. And the PA right here is pushed away. But again, rather impressive picture that really never gets old when we see these.

And so the reason patients die or the mode of mortality, if you want to refer to it that way, is really most commonly for a few reasons. So acute heart failure because of aortic insufficiency. And this happens when the flap dives into the aortic valve. The aortic cusps themselves or aortic valve cusps themselves are never dissected by definition, nor can dissection go beyond the aortic annulus. But the flap can dive in and out of the aortic valve, thus causing severe aortic insufficiency that is acute and patients presenting with pulmonary edema or acute heart failure. And this is typically treated by aortic root replacement or valve resuspension and aortic root repair.

Coronary malperfusion-- and this again most commonly involves the right coronary artery where it's sheared off or torn in the process-- can be again replaced with an aortic root with a concomitant CABG depending on the health of the coronary artery. Cerebral malperfusion, this again is demonstrated by patients who present with a stroke or a TIA, which typically in our hands require total arch replacement along with a concomitant carotid replacement as needed. An aortic rupture is relatively standard or easier to take care of, because that involves the ascending aorta. And the primary way to replace that, again, is to replace the ascending aorta.

This is again an illustration of where we most commonly see a primary tear in acute aortic dissection in a type A, which really is in the past the sinotubular junction. And again, this is where the primary intimal tear is depicted. And you can see, even though the entire aorta is dissected here, it's just that the primary tear happens to be in this particular site.

And so the goal is not to take out the entirety of the aorta, because that doesn't necessarily add to anything. But the goal is to take out the primary tear. That really is the primary objective. And this is why a lot of arguments exist for doing a limited resection of the aorta and limited repair of the aorta, because the goal is to come out with an alive patient, especially when mortality is anywhere from 20% to 30%.

Now, the secondary objectives are to address aortic root and arch pathology. As you can imagine, the aortic root is not meant to be this way. It's not meant to be dissected this way. And so this is likely to have consequences down the line, which it does. And same thing with the aortic arch and the brachiocephalic vessels. So while you take out the primary tear and address the problem and re-expand the true lumen, you're still leaving dissected aorta behind. And so the goal would be to, again, try to do as extensive of a reconstruction really without increasing your morbidity and mortality significantly while allowing for favorable aortic remodeling.

So for the majority of times, we do resect the proximal aortic arch. And so for us to do that, first do the operation, first of all, we place the patients on cardiopulmonary bypass. But for us to resect the aortic arch even proximal or the entirety of the arch, we do have to stop circulation to the brain and the rest of the body. We take the patient off cardiopulmonary bypass for a limited period of time to expeditiously perform reconstruction on this arch.

And so it's important to do this under hypothermia. And the reason for that is really it allows for margin of error. As you can imagine, as you cool the body down, the brain has less metabolic activity, and which allows us to operate on it without necessarily rushing through the process but still be expeditious along the way. The longer you are under circulatory arrest, the longer you are to have neurologic sequelae. So having said that, cerebral perfusion, for an extended period of time, anywhere from 40 minutes or so, has been shown to be very, very safe with modern technology.

And so this is an example of how we would do this. This is an example of a woven piece of Dacron fabric or polyester graft, which shows that connecting, again, the sinotubular junction to the proximal aortic arch. And while we're doing this repair in the proximal aortic arch, these are two cannulas or tubes that are going up the innominate and the left carotid artery, thereby perfusing the brain bilaterally while we're doing the operation.

At this point, the spine, the gut, the kidneys, et cetera do not get any perfusion. Just something to consider. This is what that looks like intraoperatively in an open aorta where we're putting these cannulas up the innominate artery and the left carotid artery.

This is the other concept like giving the brain an enema, so to speak, called retrograde cerebral perfusion. The way this is done is, again, this is an aorta and a heart cannulated bruised aorta. This is the right atrial appendage, which has a large venous cannula. This right here north is the SVC. Again, head's at the top.

And so the SVC is what would deliver once we take the aortic cannula out and the aorta out and wean off cardiopulmonary bypass would then deliver blood to the brain backwards. And what this leads to is really flushing out emboli and debris from an open aortic arch, which, again, the goal would be to minimize risk of stroke and debris.

This is what this looks like. And the advantage of this, while it's not really nutritive for the brain, it provides a cooling [INAUDIBLE] because the temperature that's going into this SVC cannula is really in the double digits, low double digits, and would allow for all this debris, and [INAUDIBLE], and emboli really to come out of the aortic arch here coming out from the brachiocephalic or innominate artery and the left carotid artery here. And again, the goal is really not to provide adjunctive nutrition but to cool the brain while you're doing this particular reconstruction and to flush out emboli and debris.

And so when you're dealing with the arch, depending on the tear side is how you decide the amount of reconstruction you want to do. And so this comes with a big balance. So for example, this shows a dissected aortic arch where the intima and adventitia are separated. We typically take a piece of Teflon felt. We sandwich it between these two layers, because if you don't, it's like sewing to peanut butter.

And so once we do this, this allows us to have a robust bolster in doing the surgery. Then we take a piece of woven polyester graft and sew to the proximal arch and then reconnect the dots by sewing to the proximal aorta or the sinotubular junction by the aortic valve.

And more extensive operations. And in Pittsburgh, we do these about 47% to 50% of the time. Whereas, nationally, these are done about 9% to 11% of the time in experienced hands as we replace the entire aortic arch. And the advantages of these are several fold, which we'll get into a little later but primarily where you resect most of the disease of the aortic arch, and you allow for endovascular interventions down the line, which this particular strategy does not.

And so the way we would protect the brain, as we talked about, is really by going down on the temperature, because we know as we cool the body, the metabolic rate will go down. Same thing for the heart. Same thing for the brain. And so the most common way this was done historically and is still done is by what we call deep hypothermic circulatory arrest. When we cool the patient down to about 20 degrees Celsius, we can use retrograde cerebral perfusion, as I mentioned earlier, by giving blood through the SVC, or antegrade cerebral perfusion, where we give blood through bilateral carotid arteries or single carotid arteries.

More recently, a lot of surgeons have now really advertised moderate hypothermia with antegrade cerebral perfusion or you cool the patient down to 20 degrees Celsius. And really, there's not a significant advantage to this. But surgeons do like doing this, because it is a little faster. But again, it doesn't allow for that margin of error that you would otherwise be able to do so in a colder patient population.

Now, we're fortunate, as I mentioned, to have one of the largest neurophysiology departments in the country. And we have 24/7 access to neurophysiology and neuromonitoring. So when we do these operations, we can monitor a patient's brain using EEG and SSEP signals. So we know when the brain is silent and when the EEG is silent.

Most commonly, this is not available at most places in the country. And so we know these are historic data from [INAUDIBLE] that do demonstrate that at about 45 minutes of cooling a patient, 95% of patients have an EEG silent brain. And so when you cool to about 45 minutes, you're typically going to be OK to perform arch reconstruction in the next 20 to 30 minutes. And that's really the concept.

Now, when we're cooling to deep hypothermia, we start seeing a flat EEG. And again, in the operating room, we have leads on the patient's head, arms, and legs that really tell us what the EEG signals look like, what the SSEP signals look like. Then, there's a neurophysiology tech sitting there and communicating with the neurophysiologist and giving us that information live.

Now, that was the aortic arch. What do we do to the aortic root? So most commonly, as I mentioned, it's inconceivable for the dissection to involve the aortic valve cusps. However, as seen from these illustrations, the flap can cause disruption of coarctation of the valve and lead to severe aortic insufficiency.

Now, most patients, when they present with an acute aortic dissection, have normal aortic roots and normal aortic valves. And so again, the goal is to preserve this. And so here we do this by, again, using a piece of Teflon felt and sewing the outer and the inner layer of the aortic root back together and allowing for coarctation of the valve cusps by what we call resuspending them by really hitching these commissioners up and tightening the valve. And that allows us to really eliminate aortic insufficiency in two thirds of our patients. In certain patients, it is unsafe to do so. That is unlikely to lead to long term success.

So this is another cartoon where we take these what we call essentially a neo-media reconstruction where we take a piece of Teflon felt and put this between the layers of the adventitia and intima and sew these to each other. And again, this allows for increased healing, quite frankly, and allows for a robust suture line.

So there are times where we do want to replace the aortic root. There are special categories, and this does happen about a quarter of the time where we see a primary or reentry tear in the root. Patients with known aortic aneurysms, patients with known familiar aortopathies, such as Marfan's, or Danlos, Loeys-Dietz, et cetera, where we know that these patients will continue to have aortic root dilatation despite the aortic root repair. And so these are patients where we typically will replace a root.

And we can do that by putting artificial prostheses in. Or we can do that by preserving their aortic valve and performing what's called a valve sparing root replacement, or a David, operation. The alternative, as I mentioned, depending on the patient's age, is to consider a mechanical or biologic prosthesis. And this way, the entirety of the aorta from the aortic root onwards is replaced and really out of the picture.

This is a video, operative video, of a simple aortic dissection. And I'll show you a little bit more complex in a minute. Again, the head's towards the top right now. Feet are towards the bottom. The chest is now open. We're about to open the pericardium. As you remember from the CT scan, that patient had a pericardial effusion. So as soon as you open the pericardium, you can see tamponade like physiology where, again, it's opening up like a [INAUDIBLE].

Here, you see the aorta quite bruised. We typically cannulate that aorta directly. This is done in about 10% to 15% of institutions and surgeons. It's not necessarily a common approach, but we've done this successfully. This was introduced initially in Japan and then popularized in the US. The way we do this is, again, as you can imagine, since there's two lumens, we feel two pops. And now, you don't see blood flow. As soon as you get in the true lumen, you see pulsatile blood flow. And you watch this under TEE guidance before putting a cannu line.

Normally, for routine heart surgery, we would just take a knife [INAUDIBLE] and put this aortic cannula in. But in this particular patient, we watch [INAUDIBLE] TEE. We confirm that it's the true lumen for the entirety of the aorta. That can be visualized. And then we cannulate the aorta. And again, this allows us to be expeditious. There are alternative ways to cannulate like cannulating the groin, which has increased stroke risk, or cannulating the axillary artery, which comes with other kind of complications and delay.

This is the patient's being cooled to moderate hypothermia. Here, you can see the heart is now flaccid, completely drained, slowing down, again, being metabolically inactive. Once you're happy with everything, you cross clamp the heart, stop the circulation to the heart. You cool the heart by giving cardioplegia. And at this point, the brain and the rest of the body is being perfused.

You can see we're taking out thrombus, which is what is formed because of stasis from the false lumen. And again, this will continue to expand if the patients don't get treated right away. And so now we try to take out all the disease tears. Now, you don't necessarily see any tears in the intimal segment right here by the aortic root, but you see a lot of dissected aorta where the adventitia and the intima are totally separated right here.

Now, again, as we mentioned before, two thirds of these patients have competent aortic valves. So we analyze it, make sure that all three cuts are at a good height, make sure they're symmetric. And then once we've decided that we're going to preserve the aortic root, we take a piece of Teflon felt as shown here and put it between the two layers and really sew those two layers to each other.

And that forms our neo-aorta and how we're going to put our new suture line in. And so you try to do this in different segments, of course, while ensuring that you're not covering any of the coronary arteries in the process. And again, this is really the first step before we do this. So now, we've got a put together aortic root, a competent aortic valve, and the valve's stitched up.

ACP stands for antegrade cerebral perfusion. The next thing we do is insert a needle into the innominate artery. We can do this either by poking the innominate artery with a needle like that and connecting that to our pump or we can do that directly after opening the aortic arch and connecting that. And again, this allows for perfusion while we're operating in the arch.

So here, we've cannulated the innominate artery. And so we have unilateral, so right sided brain perfusion. So we can watch this under the carotid by ultrasounding the carotid, making sure there's good flow. We take the cross clamp off. So right now, there's no blood supply going to the gut, kidneys, or anywhere else in the body, only to the right side of the brain.

This particular patient was I believe 80 or 82 years old. And he had a history of carotid stenosis. So we take another cannula and put it in the left carotid artery to ensure we have bilateral protection of the brain. Again, we can confirm that on ultrasound. We then cut out all the disease aorta that we see.

This particular patient only had what we call a DeBakey 2 dissection, where it was confined to the ascending aorta. So that way, this would be a curative operation and not something we'd have to really worry too much about in the future. Once we're done with this, we then take a piece of woven polyester graft and start expeditiously sewing this. And again, the way to do this is you want to make sure that this is watertight. If there are any dissected areas of the aorta, we try to really sew them together by ensuring that they're good and accurate.

Now, at this point while we're finishing up the anastomosis, we try to de-air this appropriately, because we don't want any air trapped in the brain, because the entire, again, aortic arch is open. This is what that looks like in a cartoon segment while we're wrapping up. Once this is done, we recannulate the aorta. This can be done a variety of ways. We go back on cardiopulmonary bypass. And now we have a neo-aorta, which is basically this particular graft. As you can see, that column of blood is now rising as we're slowly starting the pump back on.

And again, we want the patient to be totally air free. So this is the patient back on cardiopulmonary bypass. We're inspecting our distal anastomosis. And now, we're going to sew really point one to-- point A to point B together. Whoops. All right. So once this is done, we, again, de-air the heart, wean off cardiopulmonary bypass as expected. This is what a relatively finished product looks like. The heart's not decannulated. It's about to restart here, but this is what we expect.

So this is what we would consider a type A repair 101, where we replace the ascending aorta with what we call a hemi-arch replacement, partial arch replacement. This is an old example of using BioGlue We've learned not to use this as this leads to pseudoaneurysms and a lot of trouble down the line. We resuspend the aortic valve right here with these little sutures that you're seeing. And we really hook up point A to point B. And this is typically the way to do that.

So again, this particular patient presented with coronary malperfusion, underwent a root and valve preservation, extubated same day, and went home a week later. So what happens afterwards? So once you're done with this, we continue to follow these patients, really, for the rest of their lives. And we're the PCPs for these patients. So we monitor them carefully with a combination of echocardiogram, MRI, and CT scan.

And a lot of data from around the world shows that there's a significant risk of re-operation between five to 12 years. And really, the reason for that is that you're leaving some disease aorta behind. And so how do we minimize this re-intervention? Because if you know that you're going to go back and have to redo some of this, that really the goal would be to do extensive aortic reconstruction, or what we call a total arch replacement, in the majority of the patients.

A lot of the time, this is avoided, because it's technically more challenging. It can have higher 30 day mortality. But there are advantages such as potentially complete treatment and, more importantly in our opinion, setting up for future endovascular options as opposed to redo surgery.

This is another example of a patient who presents with an acute type A aortic dissection. This is a large intimal tear. Fortunately for this patient, it happens in the proximal arch. The descending aorta is really free of any disease. And so again, in this particular patient, it's totally reasonable to perform a hemi-arch replacement. The head here is towards the right of the screen. Foot towards the bottom. You can see the true and the false lumen here. We cannulate the aorta, again, as we talked about earlier, going through the false and the true lumen, and by inserting a soft wire that is seen under live TEE guidance here.

Once we confirm this, we heparinize the patient, go on cardiopulmonary bypass. This is the arterial line for cannulation. This is the venous line. This is the cannula and the SVC originally used for drainage, which can then be used for retrograde cerebral perfusion when we stop circulation and any blood supply to the brain and the rest of the body by clamping this particular area and having blood go in this direction. And again, this is what allows us to filter out debris and emboli from the arch.

The next thing we're going to do is, once we start putting the patient on cardiopulmonary bypass and cooling them, is we will then cross clamp the aorta where we stop circulation again to the heart. We've done that here. And we're now cutting out the diseased aorta completely. Again, head's on the top of the screen here. Take some cautery. And again, there's some diseased aorta and segments that we take out to allow for really nothing left behind.

Now, in this particular patient, we go straight to the aortic arch. We take that piece of woven polyester and really start sewing. And in this particular patient, this is a hemi-arch replacement like you saw. Again, it's about a 90 degrees turn. So the head's at the right side of the screen. We recannulate the patient. Go back on cardiopulmonary bypass and, again, connect point A to point B. And in this particular patient, this was a curative operation. This is a technique where we are really taking all the air out. So we don't have to worry about anything.

This is a patient where the valve is competent. So we preserve the valve by, again, hitching up the commissures on all three segments. And again, we sew this particular piece of woven polyester back. And so that's a hemi-arch replacement. So in a total arch replacement, we replace the brachiocephalic arteries, innominate left carotid, and the left subclavian artery.

Sometimes, the left subclavian artery doesn't necessarily need to be replaced. But in our hands, we typically try to do that. We take a separate graft, which is right here, which is a much smaller version, as you can imagine, for the brachiocephalic arteries. This is us sewing the innominate artery together. As you can see, there's still some blood coming out, because we're using retrograde cerebral perfusion through the SVC cannula here.

Once we sew this up, we can utilize this as a conduit to then perfuse the brain antegrade. So this is the left innominate arteries done. And so we're now giving blood to the right side of the brain in an antegrade fashion. We now are sewing the left carotid. And once we do this, we have the separate arch graft, so we can now perfuse both sides of the brain successfully. This is what it looks like proximally. This is an example of a good dissection flap where we take out really from the picture.

We're looking down the aortic root now. This is an example of how we would hitch up these commissioners by tightening them up. We do a valve analysis virtually in most patients. We can tell by TEE what's needed. We then size this, because if you put a large size Dacron graft, and you can actually induce AR. Or if you put too small of a graft, then you could potentially not necessarily induce aortic stenosis, but really [INAUDIBLE] competence.

And once you're done with that particular segment, you're done with the top or the brachiocephalic arteries and the distal aorta. And you're finishing at the proximal anastomosis. Then, you re-implant this particular arch graft back onto the neo-aorta. And again, what we call a graft to graft anastomosis really by hooking at point A to point B. Here, the heart's restarted. This is what that looks like. And this is where the rerouted blood supply is going to the innominate and the left carotid artery and left subclavian artery. That's what a completion product looks like.

Now, this is the right neck. From time to time, patients do come in with a stroke and a thrombosed carotid artery. We don't necessarily operate on all these patients, but we operated on patients who we do think can be salvaged. We tunnel a graft from the right neck or the left neck to the chest. This is, again, an example of a thrombosed carotid artery. And we replace this with, again, a piece of woven polyester graft that's appropriately sized. And we tunnel this, and we hook it up to the aorta, essentially doing what we call a carotid bypass. This shows a thrombosed carotid artery, again, because of a combination of stasis.

Frozen elephant trunk really refers to antegrade TEVAR. This is an example of another total arch replacement we're doing. This is I would think the left carotid being sewn by us. And once this is done, once the subclavian is sew, and the left carotid and the innominate artery are sewn, we then cut out this is the distal arch that's being now cut out completely. We look down there. We put a soft wire followed by a pigtail. We typically do this retrograde under IVUS or antegrade under direct vision.

This is an endograft that now we put down or a stiff wire that allows us to do this safely. This helps us with a few things. It allows to cover any secondary entry tears in the descending aorta and allows us for a robust [INAUDIBLE] line, because, again, we use that as [INAUDIBLE] to sew, or to help us really do future TEVARs down the line. And this is us really sewing a surgical graft to that aorta and the surgical endograft.

And so once this is done, again, the finished product would look completely the same. It's like putting Humpty Dumpty back together. And this is what the finished product would look like, with, again, an arch and the aorta reconstructed, and a decent aortic graft. So the Pittsburgh paradigm, as we call it, is, again, about 47% to 50% fall under this category where they undergo a total arch reconstruction.

And again, a lot of this is minutia and detail in our opinion, but these are the typical guidelines that we use internally to allow for more of a complete arch reconstruction. In a lot of patients, a hemi-arch reconstruction is quite plenty. And the way I look at it is, I think it may be a little bit more of an aggressive comparison, but putting a 21 surgical valve in I think in plenty of 80-year-olds, or certain patients, or BSAF one, five, one, six. It may be totally fine. But the goal is to think of the future and really allow for easier future interventions down the line.

And when the carotid is dissected, and the patients present with cerebral malperfusion such as a TIA or a stroke, the goal would be to replace carotids on both sides. Now, this does come with an elevated risk of stroke. And so we do this carefully. Just because we see carotid dissection, we don't necessarily go after it. But a combination of a carotid dissection with stroke or carotid artery thrombosis is when we go after replacing the carotid arteries [INAUDIBLE].

So just to switch gears, so once this patient has their operation done, let's just say they represent back with another episode of chest and back pain. This is what their CT scan looks like, going from the top to the bottom. It looks like they have a rupture in their left chest. It looks like they're type B aortic dissection. And if you look carefully, they have an [INAUDIBLE] right subclavian artery, which comes off distally to the origin of the left subclavian artery right here. It goes retroesophageally to the right side, which makes this treatment a little bit more complex.

So obviously, this is a contained rupture. True lumens compress in certain portions. This was a free on rupture. A patient would not be alive. This is another coronal view of the same patient right here that shows, again, a tear just to the left subclavian artery and the retroesophageal [INAUDIBLE] of the right subclavian artery and the left chest full of blood.

And so this would put us in the category of a type B aortic dissection. And so obviously, medical management is really not something that would be appropriate in these particular patients, although appropriate in a lot of patients with type B aortic dissection. Open surgery's largely been abandoned for type B aortic dissection because of high mortality and morbidity with this. And so the standard of care really is TEVAR, which is endovascular treatment.

So what makes a type B aortic dissection complicated? Meaning, when do we do TEVAR for these patients? So malperfusion is really the most common, where patients present with a cold leg or AKI, presumably from true lumen compression, or a left cold arm, for instance, when they present with a contained rupture as is seen or rapid expansion of the aorta.

Virtually almost always, we do rescan these patients at 24 to 48 hours to ensure that there's no rapid expansion. And again, similar to what you saw antegrade, we cover these primary tears. Here's an example of an aneurysm. But the goal is to find a good landing zone proximally and distally and really exclude the primary and secondary tear in the process.

So this is an example of a patient who underwent a carotid subclavian bypass already. This is a pigtail through the left wrist coming to the left subclavian artery, carotid, coming down that gives us a marker of where we're standing. The aortogram shows, again, a contained rupture right here. We already have a stent graft parked along the way to be able to deploy this as soon as we're comfortable.

We typically do this under rapid pacing or by giving adenosine. We've really switched to rapid pacing that allows for more precise deployment. That way, the cardiac output is not significant. So this is a simple deployment, again, of TEVAR, again, covers the primary tear. And a repeat aortogram will show coverage of that tear. And again, no rupture seen, and open left carotid subclavian bypass or transposition there.

We typically have found based on our experience that if we treat these patients down to the celiac artery we do have favorable aortic remodeling. So we do this in an overlapping stent fashion. Here, you can see, again, the true lumen being compressed. We use IVUS throughout the operation to ensure that we're in the entirety of the true lumen. And this is what a completion product looks like.

And this kind of particular flow is not uncommonly seen in patients who have retrograde flow. But again, the goal is to prevent this going all the way back and not necessarily just sitting here. And again, this is differentiated by an [INAUDIBLE]. And it's not uncommon that I get on the phone with [INAUDIBLE] or one of our partners and when we have these discussions as to what's going on what do we think is happening and do we need to do anything?

So in summary, the key concepts for good outcomes, in our opinion, is really time from diagnosis to time on cardiopulmonary bypass for an A or coverage for B. And really, correcting malperfusion is really what determines. And tear based paradigm-- while 70% of these tears are in the ascending aorta, going after the tear is key. And if we see that these tears are in the aortic route. We see these tears are in the aortic arch. We do go after these, and to protect the brain and the heart in the process.

Obviously, this is a multidisciplinary team. From an inpatient to an outpatient, there's multiple more facets of this that I haven't listed here. But obviously, we can't do our job without having a true collaborative multidisciplinary team, whether it's imaging specialists, neurointerventionalists, or ACHD colleagues, our perfusion [INAUDIBLE] staff, or critical care nursing staff, cardiac [INAUDIBLE] cardiology. Obviously, a large number of patients. These are just the inpatient clinical team that takes care of the patient when they show up to the operating room.

So I'm a big proponent of, quote, "knowing what your market value is" or knowing where we stand. And so this is a good example of where we stand as an aortic center of excellence. So IRAD, which is the International Registry of Acute Aortic Dissections-- I think Kim Eagle gave a talk earlier in the year, and he's really credited with creating this. And this was formed the University of Michigan collaboration with MGH over 20 years ago. And it includes 64 centers across the world. And it continues to grow every year by adding centers in each continent.

And so once you treat a patient, you enroll them in the registry. Even if you non-operatively manage the patient, they're still part of the registry. And this allows for a variety of clinical and scholarly opportunities and really was the goal why this was started. And this gives you an example of the number of centers that are around the country. And a lot of this includes centers that you may have trained from, come from, or look at carefully.

And so this gives you an example of where we stand despite us joining about 10 years later based on some of the other leading centers here. And so it's not only that the total number of patients that we see, the total number of patients we treat is also significantly higher than, really, everyone else on that very large list. I think a lot of centers do treat these patients, particularly patients with acute aortic dissections, and don't necessarily follow up.

And again, as I said, we serve as the PCPs for patients with aortic disease. I think last week, [INAUDIBLE] call me saying, well, I have a patient with a 4 and 1/2 centimeter aorta, really no concern, tall man who weighs quite a bit. And I said, chances are he'll probably never need surgery. But having said that, we do want to make sure that they're carefully followed. So again, it's not uncommon for us to see-- and a majority of these patients don't necessarily need surgery right away.

And again, I think this is something that we take pride in that our outcomes do continue to be significantly better than what peer groups look at. And these are not necessarily all centers across the country. These are just IRAD data, which have-- or I should say international mortality about 20% to 30% where we really dropped significantly. The stroke rate has really helped by using aggressive neuromonitoring like we do and allowing us to know when the brain is ready for us to do circulatory arrest and how to rewarm the patient.

Our typical protocol is one phone call. Depending if you're on a non-Oakland campus, you get one of the three of us on the line. Patients get directly transported. And we believe in a whole concept of subspecialization despite having obviously a large number of surgeons in the service line who are extremely talented and have excellent outcomes with everything else. Aortic surgery, just like a lot of other niche operations, does have a volume outcome relationship that has been demonstrated time and again.

And so some practical tips for, quote, "first responders" to think of, anti-impusle therapy is always a good way to start. Once you cannot get the blood pressure down that way, after, a little reduction is typically the next move. Most of the times, these patients are scanned with a PE protocol, which is, again, expected, because that's much more common than an acute aortic dissection.

But I would encourage everyone, if there's any suspicion at all, to scan the patient's abdomen and pelvis as well, because that gives us a better picture and idea of what is going on and what is dissected. I think things such as extended dissection, presence of pericardial effusion, and a pulse exam, or an abdominal exam, or pain, rather, and how acidotic the patient is give us an idea of how malperfused somebody is. And again, we think about this a lot, because it allows us to see how disease and how in bad shape these patients are.

So typically, I think these cases are missed, again, because of low suspicion. And I do think it is very hard on certain patients, because about 20% to 30% of the patients do not present with chest pain. They'll present with stroke and an MI. And stroke and MIs are significantly more common. And I think going down that pathway is totally reasonable. But I think if there is a suspicion of an acute aortic dissection, I would have a very low threshold of scanning these patients.

And again, same thing with acute kidney injury. I think it is likely that these patients may recover after an acute kidney injury episode, but it's unlikely that they'll recover if a dissection gets missed. And so it doesn't matter if the patient necessarily needs aortic surveillance or typically medical, endovascular, hybrid, or open surgery, a complex aortic surgery, the goal is for the aortic center at UPMC to be a one stop shop, where we're able to take care of these patients and really follow them for the rest of their lives from point A to point B.

And so what do we think the future looks like? So this is an example of a 60-something-year-old patient that presented. She was on dialysis. She had a recent stroke. She was in a nursing home. She has a type A aortic dissection here. And this is a patient we thought was not suitable for open surgery. This shows a catheter, again, in the true lumen.

And we decided to treat this patient endovascularly by covering her primary entry tear. And again, the goal for this was, again-- she was only 60, but, again, recent stroke, [INAUDIBLE], nursing home, somebody who we thought was going to recover, but unlikely to survive open surgery. And these are right now off label therapies. There's a lot of pivotal trials that are going to be down the line. This is what the aortic based industry has concentrated on for the past decade. However, they've been unsuccessful because of the dynamic nature of the ascending aorta, because of the aortic root complex, because of sinus segment, et cetera.

So I think when we do these, it's important, obviously, not necessarily to cover any of these coronary arteries while covering the primary tear. So it's not uncommon for us to make fine adjustments. And this particular patient initially was felt that we would potentially cover the coronary artery. And so the endograft was moved. And then, you can utilize a second endograft by really in a balloon segment by essentially fixing it in place, which is what this shows. Sorry.

The goal, again, is to notice to make sure that once you've ballooned it, which we typically don't do, that you've covered the primary tear. The true lumen, obviously, looks well expanded here. And to have a relatively, again, it is unlikely you're going to get an A-plus result this way. But the goal, again, would be to cover the primary tear in this particular patient.