JOHANNA L. MORTON: So starting off, I just want to give you an overview of what we're talking about. So we're going to start with a couple of questions, talk about the epidemiology and why I decided to say stakes are high regarding stroking in young adults. We're going to just briefly review the 10 causes of stroke in young adults. There are more than 10, but we're just going to stick to that otherwise I could talk for a good 12 to 24 hours on the topic. There are some controversies in management that we're going to go over and then maybe future directions in this field.

Fortunately, I have no disclosures at this time, but maybe that will change by this time next year.

So there's the dancing brain. How cool is that? Obviously, a neurologist, very good.

So this is the fun part, hence the dancing brain. So what rank does stroke hold in leading causes of death in the state of Texas? What do you guys think?

SPEAKER: Second, third.

JOHANNA L. MORTON: Second? OK. So it's actually third. So what do you think it is in the United States? Fourth. Good. So it's third in Texas. So obviously we'd prefer to be fifth, sixth, seventh, not worse overall than the United States. So yay Texas, we have some work to do.

What is the prevalence of hypertension in young Texans? And I picked 30 to 44 because that was an easily pulled data element. What do you guys think? Very good. So 35, I know. I know. Very good.

What's the prevalence of hyperlipidemia in Texans 30 to 44? 50, yeah, I got you. Good. All right, good.

What's the prevalence of stroke among Texans ages 18 to 29 and 30 to 44? So, two data elements-- two parter. Very good. I'm hearing every letter, so I'm just going to tell you. So in 18 to 29 it's 0.5. And then as we get a little bit older it's 1.0. And that's actually pretty consistent with the rest of the country. So that's good news for us. Yay, Texas. That's the stroke team. So that's what we look here at St. David's. So anybody who's not from St. David's, that's what you'll look forward to, having a stroke here in Texas.

So what's the prevalence of Texans ages 18 to 29 and 30 to 34 who could not see a doctor due to cost? So I've heard every letter. Very good. In the younger age, almost a quarter of Texans, by self-report, did not have enough money to see a doctor. So yay Texas, so we have some work to do.

So this is a really interesting slide according to me. These are the metrics in young adults with stroke here at St. David's. These are our patients. So we pulled data from 2011 to 2012 and 2013. So we had a considerable amount of patients noted-- 2013 is obviously not over. And you can see the number of patients dramatically increasing.

And I apologize. I can't use the laser pointer on the screen. So I'm going to be jumping around like a lunatic or using this clicker here.

But you can see the total number of patients dramatically increasing. And there's a number of factors. It's not like the incidence of stroke in young adults is skyrocketing. Obviously, the number of all patients coming to our facility is increasing.

But the number of TPA administration has been pretty constant. The number of patients going to rehab has also increased. And thank goodness, as the number of patients increased, the percentage who have expired has been the same. So this is very interesting data from our stroke program here.

So when we look at the rehab patients, the black line is actually the functional independence measure. And Dr. Lee could probably tell you more about that. But that's actually reflective of how they do in rehab. And that's actually pretty good because I think 100 is almost the maximum score.

SPEAKER: [INAUDIBLE]

JOHANNA L. MORTON: Right. And actually they do, a fair percentage of them. So most years I was able to pull data, most of the patients did go home. They were community discharges, so that's good news for us.

Taking a step back from Austin and the community, about 700,000 to 800,000 adults have strokes every year. And 5% of them are actually less than 45 years of age. And about 500,000 to 800,000 young adults have had a stroke by self-report.

So in Texas the young adults, those patients less than 30 years of age, the incidence is about 0.5. So that's about national average and that's reassuring.

So if you look around 30 to 49 years of age, the prevalence is actually really crazy because there's something about 2009 that dropped the prevalence. I don't know if that's an outlier or what have you. But the prevalence usually hovers right about 1.0. So in Texas the prevalence is pretty much the national average.

And I want to jump on the screen and just point to you that if you look at-- this is the American Heart Association's Annual Heart Disease and Stroke Statistics, you can see that-- can you guys still hear me? So this is less than 30 and this is 40 to 50. This is the national prevalence. The percentage of the population is about 0.5 and 1.0.

So again, I just wanted showed you. This is the national. And this is the same data. This is National Health and Nutrition Exam Survey, so this a huge administrative database for the entire United States. And then when you look at the incidence, which is different from the prevalence, the rates for ischemic stroke in young adults is about 10 per 100,000 person years. And there's really no difference when you look at females and males and you compare the two.

So what's concerning? So I can give you data until the cows come home. But what's more concerning is that a very large administrative database called the US Inpatient Sample showed that there's actually an increase, so anywhere from 25 to 50% increase in this age range, over the last about 10 years. So that's worrisome.

And when you look at the cost of admission for young adult stroke, it's actually very expensive. And those who have direct patient care can attest to the fact that we order a lot more studies on these patients because a lot of times we don't know what the cause is. And a lot of times it's not routine vascular risk factors. There are some quotes that say it's about $30,000 as opposed to maybe about $9,000 to $10,000 for regular older adult strokes.

There's also an increased trend for the traditional stroke risk factors. And even more concerning is that maybe only half will actually return to regular work. And about a quarter of them actually need a different kind of job, so they don't even have their normal career to return to.

So like I said before, Texas stroke ranks number three. So that's better than number four, obviously. So I just wanted to show you that. That I'm not making these numbers up. And just so you know, Idaho ranks number five. So for all you competitive characters out there, that's something to work towards.

So what are the diagnostic challenges to young adults? Those who would have direct patient care? So we know that they think that they slept on their arm funny. Or they think that it's just a headache. Or they think that it'll go away. So that's why a lot of times there's a delay to diagnosis that makes these patients very hard to take care of.

A lot of them have an atypical presentation. So they come in with neuropsychiatric syndrome. A lot of times they have a movement disorder. We see this very commonly, or a cranial neuropathy, or just one symptom, and again, headache.

And then, of course, the stroke mimic. So we see this very commonly. Is it a seizure or is it a TIA? Is it vertigo or is it stroke? So migraine, of course, is a great stroke mimic. And a lot of times cancer, MS, encephalopathy conversion disorder, we see that very commonly. And this tends to be the patient population in which this presents.

The stakes are high. So this is a young adult patient multitasking, very productive, and I see this all the time. I can't be in the hospital. I work. I work two jobs. I work three jobs. Yeah, they're locked up in their beds. So we see this. It's very difficult.

So what are some causes of ischemic stroke in young adults? Traffic? That's good. I'm just making sure you guys are awake.

So we're going to go through 10 really quickly. And a lot of them have overlap. And it's obviously not an exclusive list. There are many, many, many, many things that cause stroke in young adults. So we're just going to go to through 10. Stop me if you have any questions.

The most common is arterial dissection. So this is about two to three per 100,000 and probably 2% of all strokes. But when you look at young adults, it's about 10 to 15% of all ischemic strokes. So if I had a pointer I would show you that this is the normal pathology. You see this flame shaped lesion on the [INAUDIBLE]. It's formed by this arterial wall layer. And this false lumen develops as blood seeps into this space.

And the incidence is really less clear for veterbral artery dissection, maybe one to one and a half per 100,000. And the V3 arterial segment, or the vertebral artery, is connected to this mobile cervical vertebrae. And this is very mobile. And it tends to become vulnerable to injury. And there is an excellent review article of vertebral artery dissection, or occlusion, due to cervical manipulation through chiropractic therapy. So in patients less than 45 years of age, there's 1.3 per 100,000 within one week of manipulative therapy. So obviously, that's a little bit worrisome.

And so other risk factors for dissections include trauma, drugs, and collagen vascular disorders. I'll talk about again because all of these causes overlap.

So stroke mechanism, so what causes stroke in arterial dissection? So is it thrombal embolism? Or is it hemodynamic compromise? So is it the artery gets too narrow and the brain does not get enough blood flow? Or is it actually just clot formation?

So actually you can have a dissecting aneurysm formation. And that can lead to thrombus formation. And also, another thing to think about, is that intercranial dissections can cause an elevated risk of subarachnoid hemorrhage. So this is something to remember in these patients.

So what do you do for a patient with dissection? A CTA, MRI, MRA with fat saturated protocols, which is what this is. And I would show you if I had a pointer, which I don't. Intercranial dissections usually require cerebral angiography to see that flame shaped lesion that I showed you earlier.

So is TPA safe for these patients? A nod would be helpful. Yes, no, anybody?

So there's a theoretical risk of an intramural hematoma formation within that lesion. This is an excellent meta analysis of 180 patients. The mean age was about 46. About 70% of these actually got TPA. And about a third of them got intraarterial TPA.

So they followed them only for about three months, but there was a pooled symptomatic intracranial hemorrhage rate of only 3%. So they did fairly well. About 40% of them had an excellent outcome with one mention of this intramural hemorrhage expansion. And that patient did just fine. So my verdict on this one yes, it is safe.

So after you give TPA to your patients then what do you do? So who would put them on blood thinners? Hands, anybody? Hands. Thank you. Blood thinners, votes? No. Aspirin? Nobody's looking. You're all looking here. It doesn't matter.

All right, fine, I'll just talk. Cochran review is a meta analysis of all the antithrombotics in 2010, so not too long ago. Unfortunately, there are no randomized controlled trials in this topic. There are only 36 observational studies.

So the primary outcome for this review was death from all causes and death or disability. But the secondary outcomes were stroke. And so what did they find? There's no difference.

And I think I say this on the floor all the time, how do we treat them? It doesn't matter. You can anticoagulate. You can use asprin and Plavix. You can use Plavix. You can use aspirin. It doesn't matter.

There was a trend in favor of anti coagulation for death or disability. And of course, the hemorrhages really only occurred on anticoagulations. But that's where that data comes from. So when I say, it doesn't matter, that's what I'm talking about.

Moving on, so the guidelines for secondary stroke prevention really say exactly the same thing. So if you have a stroke and have a dissection, they recommend three to six months on either antiplatelet or anticoagulation. So it's really essentially choose your own adventure for these patients because there's no data. And essentially, if they fail, then you can consider stenting. And if they fail that, you can consider surgery. Or if there's a reason that they cannot be on anticoagulation.

So the vessel will heal with time. We recommend repeat neuroimaging to evaluate that healed vessel prior to changing your antithrombotic. I recommend that they don't weighlift. They don't go on roller coasters. I'm not a fan of chiropractic manipulation only because of that review article and because I've seen a lot of young adults with stroke. I don't use this as a reason not to have aggressive physical therapy and occupational therapy in their short recovery.

Another cause, number two, would be hypercoagulable state. And we see this very commonly in young adults often in concert with a right to left shunt. So that's why on the bottom I also note that the most inherited thrombophilias cause either pediatric stroke or a venous thromboembolism.

So we're going to fly through these. I apologize if there's any burning questions. We can save them to the end.

So the activated protein C resistance is the most common genetic risk factor. It's usually due to a point mutation on the factor V gene. The risk of embolism is increased five to 10 times for heterozygotes or 50 to 100 times from homozygotes. And this is really further increased with oral contraceptives, hormone use, smoking, pregnancy, very tenuously associated with arterial stroke, but we do see this a lot of times in venous sinus thrombosis. So that's good to know and that's why we test for that.

The antithrombin three deficiencies, the non vitamin K dependent plasma glycoprotein that inhibits coagulation or normal coagulation, lysing thrombin, and factor 10A. So it's autosomal dominance. And it's actually common. It's seen in 4% of all patients with thromboembolic events.

And so it also can be associated with many different metabolic conditions. And again, I'm not going to go through all of them. But you're more than welcome to have these slides. It's mainly a venous condition. And the arterial stroke only really develops when there's that right to left shunt. So in a PFO, in a pulmonary AVM, that's when we see this condition associated with stroke.

So the protein CNF deficiency, they're are also very rare, but we do see this, again, with venous thromboembolism or right to left shunting. And I think the protein C deficiency, I mention this because the skin necrosis issue becomes an issue when we start warfarin in these patients. So I always just put this in slides because it's important.

So the post-thrombin mutation is important because it does increase the risk of venous thromboembolism. But arterial association is seen in females quite often. And the prevalence is about 1% to 6%. So that's often seen in the hypercoagulable panel that we do order in the young adults.

So this MTHFR mutation causes hyperhomocysteinemia. So that's genetic and it's acquired. This does cause venous and arterial thromboembolism. And this can cause arterial stroke at a risk of 1.4 to 30%. So it's fairly common. And it's arterial stroke association. Unfortunately, when we try and reduce the risk of homocysteine, it doesn't seem to really correlate with stroke risk reduction.

Like I said, I was going to fly through these because this gets cumbersome. But you can stop me if you want.

So antiphospholipid antibody syndrome is a really difficult syndrome to treat. It's genetic or acquired. It's defined by this recurrent arterial or venous thromboembolism, unexplained fetal loss, or thrombocytopenia. And there's these antibodies that attack, essentially, protein antigens that bind phospholipid anticardiolipins.

Not to get too boring, but the criteria for diagnosis is that one clinical criteria, so that's a vascular thrombosis, which is stroke or pregnancy morbidity, and one lab criteria. So all these lab criteria are essentially part of our hypercoagulable panel here, in St. David's.

So how do we treat this? So if a patient comes in with a stroke what do we do? So there are a lot of different treatments depending on whether or not it's catastrophic or not, or if it's recurrent thromboses, or if this a first time event.

So there was a study where we took patients-- we, I wasn't involved. There was a study where there were patients who-- there were 100 asymptomatic patients who had this positive antiphospholipid antibody syndrome, who had aspirin and some that didn't. And actually there was no prospective reduced risk of any kind of thrombotic event.

But there is actually this group of people who meet to talk about this kind of thing. And they recommended that moving forward that the cardiovascular risk factors are actually really aggressively controlled in patients who are high risk. And that they should receive thromboprophylaxis in high risk situations, like surgery and other things like that.

So this is a-- sorry for the painful, complicated slide. When we order the hypercoagulable panel all the time in stroke patients, this is actually how the pathologists think it through. So we order a bunch of things. We try and figure out is it applicable or not in the patient. We're not supposed to order this test when the patient has an acute stroke, when they're on Heparin and when they're on Coumadin.

The reason why I put this slide in there is because the most important thing-- and I don't have a pointer. I have to remember that-- is the bottom part here, this patient specific narrative interpretation. So the reason why this is important because we have to decide whether or not-- there are a couple of things that can be elevated or abnormal in this interpretive panel here and whether or not that applies to our particular patient. So you can have falsely elevated results when you have a patient with acute stroke or DVT or those kind of things. And they need to be repeated about 30 days after.

And so there are a couple of other conditions that can change coagulation and cause stroke. I'm not going to go into them because it gets really painful. And like I said, I could talk for 24 hours on the topic. And then you all would just walk out, so moving forward.

Isn't there a term for that when the Congresspeople do that? They just talk and talk. Yeah, that's what it would be. Moving forward.

So there are guidelines. So if you take anything away from what I have to say today, this document is excellent. It really has pretty much guidelines for everything that I'm talking about. So basically, if there is some kind of thrombophilia they recommend either aspirin or anticoagulation because the guideline is really not specific. I think that's helpful.

There is no evidence that reducing the homocysteine is helpful. I think that's good to know. If there is an antiphospholipid antibody in a stroke the patient should be anticoagulated.

Number three-- it gets faster after this, so we're not going to be here for six hours. I guarantee it. And it gets more fun too. Number three, so there are things that can cause arteriopathies in patients other than high cholesterol, diabetes, and stroke. So things like inflammation, so that's why we order the sed rate, the CRP, the high sensitive CRP, the ANA panel, all those things in those young patients. Because we're screening for things like Takayasu's arteritis.

So this is really an interesting disease. It affects all of the large vessels that come off of the aorta. And it looks exactly like this. We see this in young women. And it causes all kind of systemic issues, like muscle pain, chest pain. And it also causes clotting. It causes aortic dilation, like the picture down below. And it also is a cause for stroke that's treated under the care of rheumotologists, not neurologists.

Bechet's disease is actually fairly common. I saw it a lot in my previous lifetime due to the patient population. I haven't seen it here yet.

It's also associated with narrowing of the arteries due to inflammations. And it's essentially your body is attacking the arteries and causing inflammation within the vessel wall. So you get skin lesions in the mouth, general ulcers, acne, and then blindness. So patients are blind and then they have weird neurosymptoms, just nonspecific altered mental status and stuff. You do an MRI. And then you see strokes. So that's how that diagnosis is determined.

So primary CNF vasculitis is a very complicated diagnosis. It's very hard to define. It's very hard to treat.

So this is a very rare-- and I'll stress rare-- diagnosis of the small and medium sized vessels. Very nonspecific presentation. It's mimicked radiographically, clinically. The labs CSF or non-specific. The MRI will have some subcortical and cortical strokes. And the angiography can be helpful. Sometimes it's not. And even the biopsy can be helpful, but usually the take home message is that it's a biopsy proven diagnosis.

For the interest of time, I apologize, I'm going to skip that slide.

So lupus also can cause strokes. Sometimes there's a lupus vasculitis, but again, that's actually pretty rare. Usually it's a thrombotic arterial occlusion from the antiphospholipid antibody syndrome. We get premature athero because of uncontrolled blood pressure because the kidneys are involved. Sometimes you can have cardioembolism as well.

VCV virus is very common. And it causes a lot of times middle cerebral artery infarct in young adults. So we'll see multifocal stenosis of the middle cerebral artery, moreso on the angio than the CT angiogram. And the test for this is a varicella virus IgG study. And this is easily treated.

But unfortunately, HIV is very difficult to treat. It does cause stroke in young adults. Sometimes it's a primary vasculopathy. Sometimes it's just coagulopathy because HIV does induce a hypercoagulable state just by the hyperviscosity. Sometimes we have endocarditis or, unfortunately, a ruptured mycotic aneurysm. It's very difficult to treat.

So I think you guys notice that sometimes we do send an RPR on our young stroke patients. Because we're looking for syphilitic arteritis. And we have seen at least two or three over the last year and a half.

So this is a medium and large sized vasculitis. And these symptoms develop five to 10 years after an untreated syphilis. And again, very nonspecific. And these patients, if anybody remembers treating them, they're just totally out of it, unresponsive, the protein's elevated, the white cells are elevated. And the treponemal tests actually are usually negative. So the VDRL and the RPR can be negative. So other alternative test needs to be ordered.

Cardiac etiology anywhere from 5 to 35%. So again, all the TEE's in the young adults that's pretty much why. Because we're looking for the valve. Valvular disease, endocarditis, atrial septal defects.

So interestingly, from a lot of the reports I've been looking at in preparation for this talk, there's actually more of a resurgent and it's been talked about more in the literature about rheumatic valvular heart disease. And so this is group A strep based disease. First you develop the rheumatoid arthritis symptoms. Then the mitral valve becomes involved down the road. And this can actually have a fairly high mortality rate. The lifetime risk of any kind of embolism is very high. If rheumatic mitral valve involvement is detected the involvement of the cerebral embolic events can be 60%. So very worrisome.

So prosthetic heart valves are my worst nightmare. I think we have several on service right now. So the reason why some people choose the mechanical versus bioprosthetic valves is based on durability, thrombogenicity, and the hemodynamic profile. Overall rates for systemic embolism after a surgery for about 0.87 to 3%.

So even the patients totally anticoagulated the valves will actually embolize, the mechanical valves, about 4% per year with the mitral valve and about 2% with the aortic valve. So I think it's important to note that all mechanical valves require lifelong anticoagulation. And after surgery the bioprosthetic valve they actually recommend anticoagulation for three months. And I know that this is a very inconsistent practice. But it's actually class one level evidence. And then [INAUDIBLE] monotherapy after that. And then for an aortic valve, it's class two A evidence, but the same guidelines are recommended.

So infective endocarditis is a very, very common cause of stroke in young adults. And it says rare here, but here in Austin we see this commonly. I don't know if that's because of the patient population. I guess that's what I'm insinuating.

So you can have TIA, stroke, hemorrhage, subarachnoid hemorrhage, or intracerebral hemorrhage, all because you have septic embolization. So the infection gets in the arterial wall, it seeds, the wall becomes unstable, and then you have rupture. So it most commonly involves-- it's a distal MCA artery. But you can really have any distal artery involvement.

And I think it's important note that these patients really should have cerebral angiography prior to open heart surgery to evaluate for a mycotic aneurysm because if you're going to be giving these patients large doses of anticoagulation, you want to make sure that you don't have distal aneurysms that could rupture during open heart surgery.

In the interest of time I'm going to skip those. So atrial fibrillation, I'm going to mention this because even though this statistic says that it's only found in 0.4% of adults less than 60, when I was a stroke fellow one of my colleagues who was a year older than me actually was diagnosed with atrial fibrillation. So we were about-- never mind. But we were younger.

So it does happen to young people, very young people. So I just want make sure that people don't exclude, don't forget, to put telemetry on these people. Don't forget to order the cardionet on discharge or the ICP monitor, or the crazy things that we do for the older people when we can't figure out why they had a stroke. Because it does happen and I've seen it with my own eyes.

So obviously, A fib is associated with a multitude of things. But it's important to note that once a patient has atrial fibrillation, has a stroke, obviously they're at moderate to high risk for a recurrent stroke. If you believe in the [INAUDIBLE] criteria and you use that and you swear by it, then they're at high risk so they need to be anticoagulated.

Dilated cardiomyopathy is very common. We see this is post-partum. We see this in many drug abusers. There's infectious cardiomyopathy. There's hypertrophic. And there's many, many more. I'm not a cardiologist. THat's why I listed them for you. Takotsubo stress induced cardiomyopathy is very common.

Where do they cardiac emboli come from? They're multi-factorial. Obviously, we get a little bit more stressed out when there's a left ventricular mural thrombus. So just overall, cardiomyopathies are associated with a 4% per year risk of systemic emboli.

That's great, but how do you treat them? Most of the time you can't reverse them. It really depends on what the geology is. So if you just look at just regular heart failures, so reduced injection fraction from heart failure, there was a huge trial last called WARCEF where they looked at strokes, hemorrhage, and death. Because they were trying to figure out, what's the best treatment for these patients who have come in with injection fraction about 20%. And there was really no difference between aspirin and warfarin when you look at everything.

But if you look at just stroke, there was actually significant benefit with warfarin. Well, there's a bunch of different camps. But some of the neurologists pulled that data, even though they're very small numbers, and used that to show that the decrease injection fraction warfarin's probably beneficial in these patients.

PFO, so who anticoagulates patients with PFO? Anybody close PFO? So PFO is the most common congenital heart disease. So that flat function of the one way valve causes this right to left shunt that I've been talking about. And it's present in 20% to 25% of normal people. So probably almost half this section, almost more than that, probably have one. So it's not a big deal.

But when you have a patient with a stroke and a right to left shunt, then it becomes a big deal. And then you throw in a factor V Leiden or something else and then it's a big deal.

So there's really no increase of risk of stroke or death in patients with PFO. And the ANA practice parameter wanted to put down some guidelines-- this back in 2004 and it's been reaffirmed since then. There's really only an increased risk of recurrent stroke in patients with PFO and an atrial septal defect. So they recommended in a cryptogenic stroke with PFO and a atrial septal defect or aneurysm that even with that, even though there's an increased risk of stroke, there's no really benefit to treating with warfarin.

That being said, the stroke guidelines, which are 2010, 2011, they still don't recommend a closure. And they still don't recommend warfarin for this condition. And we'll talk about this briefly a little bit later.

So a cerebral venous sinus thrombosis, very rare. 0.5% to 1% of all strokes. 5 million people annually. Very common in young people. And there was a very, very, very large trial, about 600 of these patients, about 80% of them were less than 50 years of age with a the female predominance. And so when they come in their symptoms are usually due to two things, elevated intracranial pressure and focal deficit. Because they're having venous ischemia, or sometimes hemorrhage, and seizures as well.

So I'm not going to bore you that. That looked better on my screen at home. But if you look at the conditions, which you probably can't see. I apologize, but I'm happy to send you the slides. A lot of these conditions are actually independent risk factors for arterial stroke as well.

So this is actually from the cerebral venous sinus thrombosis guidelines from the American Heart Association. So when people ask, what do I do when I have this, it's actually very straightforward. It's pretty much anticoagulation for about six months.

Drugs, so this slide is for Dr. Moreledge. Not where I was going with that. So obviously a lot of drugs cause a lot of problems, one of which being stroke. And you have pressor effects. You have platelet aggregation. You have foreign particle from the injections, vasospasm, and arrythmias to name a few. And I just summarized some of the effect that we have.

But I did find a case report of a couple of patients from this new K2 thing. And they beat us to-- I was thinking about publishing some of the cases that we have had here in Austin. But apparently these new synthetic drugs are actually much more potent than the natural occurring ones. So that's something that we need to be aware of. And it's very difficult to treat.

So in the interest of time, I'm going to skip that. Move on to Moyamoya disease, which is something that we see pretty commonly. Contrary to what people believe, this is not a disorder of only Asian descent. It is a non-inflammatory occlusive vasculopathy. And it's associated with many, many, many conditions.

It involves one or both distal internal carotid arteries and either anterior middle cerebral arteries. And what happens is that you get this abundant angiogenesis because as these arteries slowly occlude you begin to develop this collateral network. So it's very difficult to treat. We start with antiplatelet therapy, maybe antiepileptic therapy, steroids, and sometimes bypass. But overall very, very debilitating and difficult disease to treat.

In the interest of time, I'm going to skip these, but I'm happy to talk about them. And I'm going to talk about our RCVS because this is another condition that we see very, very commonly. I think it's under diagnosed here at our center.

So this is characterized by a very sudden headache. And diffused segmental construction of multiple arteries, usually the large arteries, sometimes the medium arteries. And the criteria for diagnosis is reversibility. Unfortunately, sometimes the patients are discharged because they're clinically stable and we never get that follow up scan.

And if you can see, the associated terms and synonyms are endless. But most typically it's referred to as Call-Fleming syndrome. That was the original term. I think it's from the 1980s.

So it's a spectrum diagnosis. I think that's very important. Migrainous vasospasm fits into this bucket diagnosis. Drug induced vasospasm and there's a lot of things that fit into this diagnosis. But there are no validated criteria right now. And [INAUDIBLE] is the guru on the diagnosis. And most important things are that there's vasoconstriction, there is no aneurysmal subarachnoid. But you can have cortical subarachnoid, normal CSF, headaches, usually acute, usually thunder clap, and reversibility.

And this is just a helpful slide to figure out whether or not you're dealing with our RCVS, primary CNS vasculitis, subarachnoid, or a dissection. And the take home message is that calcium channel blockers, usually verapamil, 180 three times a day, is associated with very good outcomes.

So this is a great slide. So number 10, probably the most important, is premature atherosclerosis. So if you look at the vascular risk factors here in young Texans, just take a look at the prevalence of obesity. We went through hypertension and hyperlipidemia. Those who don't have five fruits or vegetables per day. Those who haven't had their lipids checked. Those who haven't seen a doctor. Those who don't have health insurance, but I'm sure that'll change pretty soon.

So the Health in Case Stroke Registry is actually 1,000 patients. And very similar to what I'm talking about today. Do you see 30 to 34? So large artery and small artery atheros right here, so do you see 30, 39, 44? Do you see the trend? Right about 30 is where everything goes downhill. I mean emerges. It all emerges after 30. We're getting there.

So the prevention of atheros really should target young adults. And I don't think that we're doing a great job.

But I apologize. I want to admit something to you that I forgot to mention. What is the number one diagnosis of stroke in young adults? Number one?

So it's actually cryptogenics. We don't know. We've done every test we can and everything's normal. So that happens about 20% to 30% of the time. And that's OK.

So we're going to go through a couple of controversies in stroke real quick. We're not going to read all the words on the slide. Don't worry. I see your eyes, Dr. Waldron. I got you.

So I think it's important for people to know the data behind these PFO closure trials. So when I say, no, we're not going to close the PFO, there's a reason behind it. I just don't walk around prancing my opinions for no reason. There's a lot of data that's been done. And there still needs to be more studies on the closure of PFO. But I just want to give you a heads up and you guys can Google these later or have my slides probably.

So the first trial-- and these all have been done within the last two years-- was closure. I'll tell you about this one and then we'll just move on. So closure was about 500 patients had PFO closure, about 500 had medical therapy. Medical therapy was either aspirin, warfarin or both. The endpoint was stroke or TIA at two years 30 day mortality or mortality past 30 days. So at the end of the day, after two years, there was really no difference between both groups either for the primary endpoint, for stroke or for TIA. So no difference, closure or medical therapy. So PC trial.

Second one, back to back, same thing, closure or medical therapy, same endpoint, for years, not two. No different. Intention to treat and [INAUDIBLE] protocol cohorts, not significant.

Respect trial, 980 patients, closure or medical therapy. Same thing, stroke, fatal stroke, early death. But if you looked at the protocol cohorts, the event rate for closure was a little bit less. So they got a little bit excited about that.

At the end of the day, all trials really had small numbers of primary events. And there was a really short follow up period. And all three randomized clinical trials had no significant benefit by intent to treat analyses.

So if do close PFO's? Maybe, good. So I think in certain patients, maybe some patients who have a PFO and recurrent stroke, some patients who have venous hypercoagulable states, some patients who have ASA or ASD, there's certain patients who I would. Pelvic DVT? Perhaps. But as a general rule, no.

So migraine and strokes, are they connected? So migraine's common, strokes common, does migraine cause stroke? So without a specific criteria, you could really say that every stroke patient that walks in the ED with a headache is really a migrainous stroke versus just calling it a cryptogenic stroke.

So I think it's really important to have a diagnostic criteria for every migrainous infarction. Because I think it can be a slippery slope. The incidence of migrainous infarction, according to those who are believers, is about 1.44 cases per year per 100,000. And there's a whole slew of proposed pathophysiology.

How do you treat migraine and stroke? Just continue antiplatelet therapy, prevent the migraine from happening in the first place, avoid triptains because there's a theoretical risk of venous spasm worsening any kind of pre-existing atherosclerosis. So maybe, I'm not that excited about that.

So is it safe to treat stroke mimics with TPA? This is a really good meta analysis. So this is about 500 patients and only 50 patients were actually misdiagnosed. Now if you look, it's conversion disorder, complicated migraines, seizures. And these patients tended to be younger. They tended to be milder. No case of symptomatic intracranial hemorrhage in these stroke mimics, all 50 of them.

So then they looked at six more studies. And they had another 1,700 patients. Another 150 stroke mimics that got TPA, still no symptomatic intracranial hemorrhage. So yes, why wouldn't you? Right? All right, good.

All right, so we're homestretch now. So what do you do? So this is my recipe for a stroke in the young adult. So as they go through the normal protocol, St. David's stroke protocol, and everything comes back normal. So then we think about angio. We think about TCD. We think about the flow dynamics. We think about do they need lower extremity duplex because they have a PFO? Do they need a pelvic MRV? Do they need a long term cardiac telemetry monitor?

And I think it's really important to remind people who are looking at these patients that they need to work with other specialists. So they need their hematologist or nephrologist or pathologist if they're going to do a biopsy. Psychiatrists, opto is very important. So it's very important to keep in mind that you're not solo when you're working up these patients.

When you're ordering these tests it's important to keep in mind the diagnostic yield can be low. But it's still important. And Dr. Lee might be able to tell us a little bit more about the prognosis of these patients. If you just want to read the top line, the estimates on the functional independence, that's a modified Rankin of less than or equal to two. It's very high. So they tend to do well. So that's a good thing to keep in mind.

[INAUDIBLE] giving me the evil eye, so I'll speed up a little bit. So we're on the last couple slides there. Moving forward, so obviously, these patients are very difficult to treat. They're unique. They're challenging.

They have a longer expected survival because they're younger. So much more disability associated with that fact. They may be responsible for generating their household income or securing child care or elder care or both. It's a very stressful situation. For them it might require additional resources to be able to manage that on both sides, on the patient side and on the provider side.

This is a great website. And just so you know, this citation here is actually the American Academy and Reality Consensus statement paper on stroke in young adults. So if you want to refer to anything, that's an excellent paper. And it talks about resources. Well, it talks about everything from evaluation, treatment, and moving forward in stroke in young adults.

But obviously, there's a need for heightened awareness because it's not recognized. But we need on the discharge and rehab and recovery side, we need better resources for networking for how to handle issues like relationships, careers, rehab back into the community.

So in conclusion, stroke in the young adult patient presents diagnostic and therapeutic challenges. The heightened awareness and education in this young group is needed to decrease the preventable burden. So we identified in the state of Texas that preventable disease burden. So obviously it's rare, but there's a large potential long term disability. And the economic loss is disproportionate in this patient population.

And then the socioeconomic factors of this age group present unique needs that may influence rehab and outcomes. And obviously, more work is needed to improve awareness, access to care, and resource. I think I've talked enough. I think we're done.