WOLFF-PARKINSON WHITE SYNDROME: SHOULD I BE WORRIED?
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I’d like to talk to day about WPW syndrome, Wolff Parkinson White and really talk about the pathophysiology and what the sudden death is, sudden death risk associated with WPW is as well as how do we re-stratify these patients in clinic. When we do decide to perform ablation, how do we do that and how can we reduce radiation exposure to children not just during ablation, but strategies that can be crossed over disciplines to help reduce radiation exposure in children as a whole.

So why do we worry about this, why do I think about it? It’s because of sad stories like this and those of you may remember reading a story from the Post Gazette earlier this year in January about a Duquesne University sophomore who died in his dorm room at rest and I don’t know anything personally about the case medically but from what I read he was known to have WPW syndrome, he had undergone an ablation at an outside institution that was deemed to have not worked. But according to the paper his family was told that he had a low risk pathway, yet here’s an individual who died. So how is that possible and how do we reconcile those facts.

So to understand that we need to go back in time and the story starts in the early 1900’s with doctors Wolff, Parkinson and White pictured here and they described in 1930 a syndrome of bundle branch block in short PR interval, in healthy young individuals who had paroxysmal tachycardia episodes. And in their paper they described well the hallmarks that we still use today which include short PR interval, delta wave, and wide QRS. Now note they weren’t the very first people in the literature to describe this but they just did it best. They described a case series and really highlighted the
pathophysiology of excess rate pathway conduction that we still know to be true today and hence this disease carries their name.

So what is WPW and how do we think about it? So here’s a diagram of ventricular myocardium and normal HIS bundle Purkinje depolarization. So if you have normal HIS Purkinje activation of the ventricle you get a normal looking QRS and that’s what we want to see. If you have an accessory pathway present and you depolarize the ventricle only through the pathway it’ll look wide like a PVC one, so you get an ECG that looks like this. So if you add those 2 ECGs together you get the ECG of WPW where the initial depolarization or the delta wave is the result of depolarization through a pathway into the myocardium and then the terminal portion of this QRS is due to rapid AV node depolarization as the HIS Purkinje system catches up. So that’s why you see the ECG we see in WPW.

So here’s an example that’s fairly typical and fairly obvious of an individual with a short PR, a wide QRS and a clear cut delta wave easily seen in multiple leads. This is an ECG that I would expect most individuals to identify as WPW. But here’s another individual with a more subtle ECG, but if you look carefully you see again short PR noted in multiple leads, delta wave are slurred up to the QRS and a widened QRS. So this too is WPW in someone. And just because this ECG is more obvious than this ECG that doesn’t tell us anything about the patient’s individual risk characteristics and that’s part of the challenge for us in managing these patients.
So what are the risks? What do we talk about with patients? Well first is a risk of tachyarrhythmias, primarily supraventricular tachycardia, okay. And typically this tachycardia occurs antegrade or forward over the AV node and goes backwards over the pathway to give you a narrow complex tachycardia. But it can occur in the opposite direction where it goes forward over the pathway and then backwards over the AV node and that’ll give you a wide complex tachycardia and I’ll show you a picture of that in a minute. In addition, patients can have atrial fibrillation. It’s rare in kids but certainly more common in kids who have WPW.

And lastly what we all worry about is the risk of sudden death. In that sudden death, the mechanism is due to rapid antegrade conduction of atrial fibrillation over the accessory pathway. So this is some slides of the typical SVT that we see with WPW. So here on the left antegrade conduction or forward conduction down the AV node, retrograde or backwards conduction up the pathway creates a circuit like this and that gives you a narrow complex tachycardia because the antegrade conduction is over the AV node.

If you have the opposite circuit antegrade over the pathway directly stimulates the ventricular myocardium, then it goes backwards up the AV node, that’s the opposite of normal we call that antidromic and that gives you a wide complex tachycardia, and both of these can be found in patients with WPW. But again this is what we really worry about the most when I see patients with WPW is the idea that if you have atrial fibrillation or a rapid, chaotic atrial activity, atria going 400-500 times a minute the AV node automatically filters that. Accessory pathways some of them filter, some of
them don’t. So if you have atrial fibrillation and you have a pathway that allows for rapid transmission this will result in rapid transmission to the ventricle and what we call pre-excited atrial fibrillation and can precipitate ventricular fibrillation. And this is an ECG of an individual with pre-excited atrial fibrillation. The hallmarks are you have a tachycardia that’s very irregular and wide complex.

Sudden death, how do we think about sudden death in this disease? Well the problem is it may be their first presenting symptom and that’s one of the challenges is how do we manage these patients. It requires the simultaneous occurrence of 2 relatively rare events. So first you have to have atrial fibrillation which is a rare event in children though more common in kids who have WPW. The literature estimates anywhere from 0-15% of kids with WPW will have atrial fibrillation at some point. In conjunction with that your accessory pathway needs to be one that conducts rapidly in the antegrade direction. And the estimates of this vary widely but anywhere from 10-30% of accessory pathways will conduct rapidly. So if you put those 2 together, atrial fibrillation in an individual with a rapidly conducting accessory pathway you have the propensity or probability for ventricular fibrillation and really it’s probability math we’re talking about here.

So there’s a wide range of estimates of what the actual sudden death risk is. Anywhere from .02% to .5% per year, and I think the average that we found in our natural history study and corroborated another study was it’s really about .1% per year, or 1 in 1,000 per patient year, and that is the number that we quote patients today. The lifetime risk, if you said 1 in 1,000 per year and you
diagnose someone who lives 70 more years in theory it should be 7% but actually the lifetime risk is estimated at about 3-4% risk of sudden death because we believe that a couple of things happen. One is that your accessory pathway risk probably lessens with age because it probably doesn’t conduct better when you’re 60 than it did when you were 16, so that actual percentage per year probably goes down slightly. And secondly as we age we have competing other death risks that impede on that so you’re lifetime risk is probably 3-4%. Of note however, in our patients in children that risk is probably higher than in the average adult population.

So this is a paper from Barry Maron who looks commonly at sudden death in athletes in the United States and this is from his circulation paper in 2009 and WPW makes the list okay. The table numbers were off in the paper but basically it’s 11 patients with WPW who they noted in their study that died suddenly who were athletes. A total of 1,049 patients which makes 1% of the sudden death in their series due to WPW and recognize since WPW is only diagnosed on ECG and not on post mortem this probably is an underestimate of how many people actually died young athletes from WPW.

So what do we know about WPW as a whole? Well it’s fairly common disease in our world, 1 in 3 per thousand. There’s a slight familial predisposition so if you look at first degree relatives some papers have found, not all, that family members may have a slightly increased risk. The majority of patients are likely asymptomatic because if they don’t have a tachyarrhythmia that’s symptomatic it’s just really a potential risk they’re walking around with and the diagnosis often and increasingly
recently is being noted incidentally because someone gets an ECG for some other reason. So a few years ago Nicole Cain one of our former fellows now an electrophysiologist in South Carolina, and I embarked on a study doing the largest natural history study to date, we looked at about 450 patients over a 50 year span here at Children’s Hospital through our database, the median age of diagnosis was 7 years with a slight male predominance. We found structural heart disease in about 10% of patients who have WPW and we found symptoms of diagnosis in about 2/3 of patients. Remember this data is slightly skewed toward an older cohort and in the years past certainly patients would most commonly come to diagnosis because of symptoms. Recently we found however, that more and more patients are being diagnosed incidentally because ECGs are being done for screening purposes for other reasons.

So she found that about 20% of patients had additional symptoms in follow up. Spontaneous resolution is quite rare if we make the diagnosis after 3 months of age and this is an important feature as we talk to families about management strategies because this is very unlikely to just simply go away.

In her study of 450 patients there were a total of 6 sudden deaths which makes a total rate of 2.8 per 1,000. But interestingly we found it clearly differentiated based on presence or absence of structural heart disease. So in normal hearts we also corroborated that 1 in 1,000 number with 2 deaths in normal hearts, but we found 4 deaths in patients with structural heart disease which was an
astoundingly high rate of 27 per 1,000 and that’s a number that had not been reported in the literature previously.

So this is a graph from our paper and we show that now we have about a quarter of patients being diagnosed incidentally and again I think this is only going to go up as ECG screening for many reasons is being propagated.

This is the age of diagnosis. You see clear cut predominance in infants most of these who presented with SVT and then a distribution later on. This is that same curve broken down by symptomatic versus asymptomatic patients. So here the babies were almost all symptomatic who had SVT but you see in teenagers now more and more coming up who are being diagnosed incidentally for other reasons.

This is the possibility of spontaneous resolution so if we diagnose you before 3 months of age over a 1/3 of those will go away and honestly probably slightly higher than that. But if we diagnose you after 3 months of age it very rarely goes away which is part of the management strategy for these patients.

Structural heart disease was found in 9% of patients and the 2 most common diseases that we talk about in older individuals would be Ebstein’s anomaly, the tricuspid valve which we found in a fifth of patients and congenitally corrected transposition of the great arteries or CCTGA which we found
in 15%. Statistically the most common was actually ventricular septal defect at 25%, except to the best of our knowledge this is not a true association or causal effect but rather an overlap of the fact that WPW is quite frequent in infants and tiny VSDs are also quite frequent in infants as well.

So for this reason we recommend that at time of diagnosis of WPW patients should get an echocardiogram and again their sudden death rate is quite high if you have structural heart disease and WPW which impacts our management strategy for these patients. This is the Kaplan Meier survival curve from the paper which shows that if you have a structural normal heart you have a reasonable longevity, but if you have structural heart disease it drops off quite quickly.

Here are the 17 deaths in the paper, 6 were sudden, and 2 of those were in patients who had normal hearts. One was an infant and one was a teenager and then I mentioned there were 4 structural heart disease patients. One with Ebstein’s anomaly which was mild, one with congenitally corrected transposition, one with hypertrophic cardiomyopathy, and the last with an AV septal defect.

So we’re worried about sudden death so how do we figure out what do we need to think. So with stratification how do we assess what the risk of rapid antegrade conduction through your pathway is? So we start with non invasive testing, resting ECG, Holter monitor, exercise test. And Phil Walker one of our former fellows who’s now an electrophysiologist at the Mayo Clinic and I looked at our data over the last 15 years had non invasive testing and correlated with invasive results. And we talked about the idea that intermittent pre-excitation means that you only have some beats that are
pre-excited, likely means that your pathway doesn’t conduct very fast. So what are we looking for? This is an exercise treadmill and on the right you’ll see ECGs that show pre-excitation. If you look very carefully you see this short PR, delta wave, and a widened QRS here. And in a single beat on the next beat this individual loses pre-excitation, the pre-excitation completely goes away, the delta wave is gone, you have a distinct PR interval and a normal narrow QRS. This is abrupt loss of pre-excitation suggesting intermittent pre-excitation in this individual. So can find intermittent pre-excitation on non invasive testing in anywhere from 10%-20% of patients. We believe based on not only Phil’s data but other data through the literature that seeing intermittent pre-excitation on an ECG or a Holter Monitor is probably less reliable than seeing it on an exercise test to understand true risk and antegrade conduction. But our data and recent data from Boston both raised the question that some patients who had intermittent pre-excitation still conducted quite rapidly on their at the time of their EP study. So the real question is, is it a perfect test, no, but it’s a very good non invasive screen to try and assess risk. So there’s really controversy in my mind over whether intermittent pre-excitation is a guarantee against sudden death, no, but how does it correlate with the risk of sudden death. I think if you have intermittent pre-excitation especially on an exercise test you would likely have low risk antegrade conduction, and your risk is probably less than 1 in 1,000 per year. But quite honestly we’re just lacking the data because the ‘n’ needed to get convincing arguments of correlating this testing with actual sudden death risk is not there. Thankfully patients don’t die that frequently so we can never correlate it with actual sudden death. The best we have is to correlate it with markers of sudden death which is invasive risk assessment.
However, I said we could find intermittent pre-excitation in 10-20% of patients which means that for 80-90% of patients we cannot tell intermittent pre-excitation on non-invasive testing. So if you want to know more about their pathway you have to consider an invasive risk assessment or electrophysiology studies.

So what’s the current recommendation for asymptomatic patients which we’re finding more and more of. There was a recent guidelines paper put out by the Heart Rhythm Society endorsed by the Pediatric and Congenitally Peds Society and they said that if patients are old enough about age 8, those patients should get exercise testing. If they clearly lose pre-excitation on their exercise tests they likely have a low risk of sudden cardiac death. Actually in the guidelines statement they say that the risk is low and they would allow them full participation. I think as I know the data and in my practice I say if they lose it on an exercise test I think they’re likely low and I share with families all the data I just shared with you and I say to them I think it’s reasonable to allow your son or daughter to participate in sports for the data we have, but I cannot tell you they’re zero chance. As we go back to that poor young individual who died at the beginning of this talk and we referred to his case, that’s one of the things I think about is that no piece of data at any one point in time is perfect. So just because you lost on you exercise test today doesn’t mean you couldn’t conduct rapidly next week. And what’s even more frightening to me is just because your invasive EP study tomorrow says you’re low risk, doesn’t mean nothing could ever happen to you in the right circumstance, because ultimately we’re just assessing one point in time. If they don’t lose pre-
excitation then I would at least offer families the idea that we should think about invasive risk assessment.

So what’s sports participation, what are our current guidelines for letting people play sports. Because as you know that’s a common and important activity to many young individuals we see. In the United States we’re governed or recommended to be governed by the 36 Bethesda Conference which looks at sports participation in cardiovascular disease and the recommendation was that risk stratification with an EP study is “advisable” in asymptomatic athletes engaged in moderate to high level competitive sports. In Europe they’re a little more strong about this. European Society of Cardiology mandates that all athletes with WPW get a complete risk assessment which includes an EP study.

The other question we get asked a lot is ADHD and really first of all we don’t recommend ECG screening of the population as a whole before prescribing ADHD medication. If you do find WPW in an individual you’re considering ADHD medications and they’re asymptomatic, there really is no contraindication to proceeding with that. And the guideline paper says nicely they recommend “intermittent monitoring and supervision” of a pediatric cardiologist. I’m not sure what that means but it seems to me that it means you can prescribe the medications and then it’s our problem to figure out what to do next.
Alright so we’ve done our non invasive risk assessment we’re not really sure in 80-90% of patients what that means, we can’t tell them what the risk is so we’re going to do an electrophysiology study. What is that and how do we do it? So the catheters we use in the EP lab all have electrode bands on the end of it. These are poles which allow for recording and delivery of electrical stimulus and so this allows us to measure electricity across the heart in different areas. So what we do is put catheters in important areas in the heart, up by your sinus node, down by your AV node in your ventricle and in the coronary sinus which runs in the left AV groove behind the left atrium and left ventricle and that allows us to record electro activity throughout the heart. On a fluoroscopy screen it looks like this, so one catheter up by the sinus node, one catheter down by the ventricle, one by the AV node and one in the coronary sinus. I remember the very first time I ever saw an EP study as a fellow I walked in the room and I saw the screen and I thought to myself wow that’s a lot of catheters to put into somebody’s heart, it looks like an octopus in there. But the point is that we do that because we want to understand electroactivation throughout the heart and what the normal pathway is. And so this is what we see at the screen, on the top here you have surface ECGs they’re stretched out dramatically and that’s why they don’t look like the ECGs you’re used to seeing. And on the bottom of the screen we have intracardiac tracings progressing from the high right atrium to the AV node, to the coronary sinus down to the ventricle and it allows us to see normal progression of electro activity.

So when we do invasive risk assessments for WPW what do we do? Well first and foremost we put patients into atrial fibrillation on purpose, okay that’s one of the primary goals of the study is to
induce atrial fibrillation and measure how fast does that atrial fibrillation conduct to the ventricle, because that’s really the best marker we have to say how risky is their accessory pathway. So the gold standard is called the shortest, pre-excited RR interval during atrial fibrillation called SPERRI. It’s the best predictor of sudden death risk we have but again understand it’s not perfect. So with SPERRI less than 220 milliseconds is the highest risk group we have. Other papers have used 250 or 270 milliseconds and it all depends on whether you want to be more sensitive or more specific. If you use 220 it’ll be more specific. If you use 270 you’d be more sensitive.

We can measure something called the effective refractory period or the ERP of the accessory pathway and we can look at how fast does that pathway conduct when you pace the atrium. Logically saying that if it conducts faster and faster we think that pathway has potential higher risk. So this is that same ECG I showed you earlier of pre-excited atrial fibrillation and if we wanted to measure the SPERRI in this patient what we would do is find the shortest RR interval and measure them. And remember that in ECGs big boxes are .2 seconds or 200 milliseconds and so that SPERRI I’ve highlighted is about 100 milliseconds so this individual we’d say is in a high risk category for sudden death based on their invasive risk assessment. So the gold standard studies that have been done now 20 or 30 years ago were done in an adult patient without general anesthesia, recognize that anesthetic both inhaled and IV may affect pathway characteristics. Patients were in their baseline condition. In other words they didn’t have isoproterenol or other sympathetic agonists on board to try and stimulate conduction. Why do we know that now, because we know from the data in the natural group that isoproterenol clearly affects conduction, so this one here if you focus
on SPERRI, less than 250. In the baseline condition only about 5% of their patients conducted very rapidly but when they gave their patients isoproterenol about 40% of patients conducted rapidly. Isoproterenol is a sympathetic agonist and we believe that in some ways it stimulates exercise, so people use it in this day and age somewhat to counteract the fact that patients are under anesthesia. The idea being well if you conduct rapidly on isoproterenol does that mean that perhaps that the surrogate marker for what you could do in real life one day as you ran around and your catecholamine state went up. So at the end of the day we recognize it’s an imperfect marker because thankfully 40% of patients with WPW are not dropping over dead out there. So the challenge of the isoproterenol data today is how do we interpret that. None of us like to see somebody who conducts down to a very rapid heart rate a SPERRI, a rate of less than 200 milliseconds on isoproterenol. It makes us nervous because it makes us feel like wow, this pathway does conduct really fast that seems like that could be bad for you. But the problem is I don’t have a large volume of data to correlate that to, to guarantee to you that that is a surrogate for a sudden death risk.

So how do we decide who needs an ablation then once we’re there, we’ve done an invasive risk assessment? Well first of all the easy one is symptoms. The patient came to the cath lab because they’re having SVTs because they’re having palpitations that we believe are SVT recurrently, ablation is a definitive way to treat them. If they have an invasive risk assessment that suggests that they’re high risk we probably should treat that as well. So if they have a SPERRI less than 250 which is the number we use in our lab or of they have inducible SVT then that’s something we believe we should eliminate for.
Patient/family preference and lifestyle choices, what do I mean by that. Well one of the most common things that patients and families will tell me is well if you’re already there I just want you to get rid of this because I don’t want my son or daughter to have to live with this from now on. And I think that’s a very reasonable choice and I’ll show you why because I think the risk profile for ablation is quite low. And then lifestyle choices and this is another common indication we have. Individuals who went to the military, the military will not accept you if you have WPW, it doesn’t matter if you’ve been asymptomatic, it doesn’t matter if you have the most reassuring treadmill in the world, it doesn’t matter, they just won’t take you. So you have to have an ablation if you want to go into the military. Police academy, fire academy are similar though my experience has been not quite as restrictive as the military and I’m sure commercial pilots are just as restrictive as the military though I haven’t had personal experience with a commercial pilot yet. But those are some reasons.

Associated structural heart disease this goes back to the data in Nicole’s paper where we found a very high incidence of sudden death in those individuals with WPW and structural heart disease. So for that reason if you had significant Ebstein’s anomaly and WPW we believe it’s in your best interest to eliminate that WPW.

Then lastly ventricular dysfunction, there are real case reports of individuals who have dyssynchrony from their pre-excitation so they have dyssynchronous activation of their ventricle and that creates
ventricular dysfunction and in individuals who are susceptible to that, ablation of the accessory pathway will often normalize function.

So what is ablation and what are we doing exactly? So this is from Fred Morady New England Journal article. You see here the atrial myocardium in cross section we have ventricular myocardium and the AV valve and in this case the mitral valve. So these accessory pathways are essentially though to be anomalous connections which span directly from the atrium to the ventricle are allowing for bypass of electrical conduction. In this paper they show a retrograde approach from the aorta to the left ventricle, to the ventricular annulus here, we use a prorate approach going transeptal to the annulus here which allows for attack of this accessory pathway. So what we do during an ablation we hope is essentially I want you to focus here at this side of the screen on the left, this is WPW I’ve got it sort of drawn out and stretched out but you here slurred up stroke delta wave wide QRS, as we apply RS energy and destroy that tissue you see an abrupt and immediate change of QRS morphology to a normal QRS and that accessory pathway is eliminated.

So what can we tell patients about public outcomes for ablations in young patients. And a lot of this data comes from the Pediatric Ablation Prospective registry which published data in the late 90’s or late 2000’s, so recognize about 10-15 year old data. And the acute success at that time was in 90-97% and it clearly varied with accessory pathway location, so left free wall were clearly the best, but lower success of the right free wall or septal region. Recurrence rates were widely different among different case series and locations, anywhere from 0 to 24%. It’s variable among case series and
among follow up because follow up times and protocols were different. But the risk factor deemed common or the presence of structural heart disease as well as multiple accessory pathways.

What are the risks of ablation? So it’s from the same papers, the overall rate of any complication was 3.2%. Now recognize this is a very tightly controlled registry with prospective data so they’re very meticulous about how they recorded complications and they recorded vascular complications in 1-2%. Coronary artery injury is really unknown but estimated between .5 and .8%. In a large adult series of about 1,300 patients AV block was seen in .1% and again higher in septal substrates and death in a very small percentage. Analyze so there’s radiation exposure early in the registry the mean fluoroscopy time given to children was 40 minutes.

What do we do here at Children’s? Well ablation here typically involves an overnight observation of post ablation. We offer patients general anesthesia, older patients we offer them conscious sedation if they’d prefer but I have yet to meet a teenager who prefers to have conscious sedation when offered general anesthesia for this procedure. We tell them recovery time is usually less than a week it’s all related to IV access from the venous access in the leg and we perform both radiofrequency ablation and cryoablation dependent on accessory pathway locations. And so what I did for this talk was look at our experience in WPW specifically over the last 3 years. I included patients only who were undergoing their very first EP study, so patients who were referred from outside centers with prior failed attempts elsewhere or our own patients with prior failed attempts I did not include for the purpose of this analysis. We had a total of 51 cases over those 3 years and we
had acute success in 100% and we had recurrence in one. So I think we can say our results here are on par or better than what’s been published nationally thus far and we had no cases of permanent AV block and no major complications in this group. So this helps me to tell families that I think our chance of success is very good, our chance of recurrence is quite low, and I think the risk of complications is really minimal compared to what’s published in the literature. So what I tell patients in my clinic is I feel like to be fair to them I quote them a total success rate of about 90% incorporating the possibility of recurrence.

So we’ve done ablations as many people have done for years using fluoroscopy, we use x-ray both biplane x-ray, anterior, posterior and lateral to place catheters with wires to verify venous access, to put the catheters in place, to map the arrhythmia substrate, to gain access to the left atrium with transseptal procedure and to perform the ablation. Well as we know there are negative effects of ionizing radiation, so in my mind is we have brought down the actual risk of ablation with regards to thrombotic complications, stroke, death. What we’re left with as far as a risk profile is really radiation exposure. And the issue is there’s higher risk in children. Children have more rapidly dividing cells, they have greater life expectancy, and so it’s estimated that children’s radiation risks are at least 3-4 times higher than those of adults for the comparable radiation dose. And really in the future it’s clear there’s going to be mandatory tracking of radiation exposure for all patients within hospitals across all modalities of testing. We’re going to need to be able to tell patients how much radiation did you get as the result of this test, how much radiation have we given your child total as a
result of all the tests we’ve done in our institution. You can imagine that in the adult world this is a much bigger issue because they’ve got exposure of radiation in multiple different locations.

So 3 dimensional mapping offers us the ability to localize catheters without using fluoroscopy, so it reduces radiation exposure and there are individuals who published the possibility of using zero fluoro to do ablation. You can mark points in 3 dimensional space and create a geometric map of the heart which allows you to create a complex electrical map and do complex arrhythmia mapping. So this is a 3 dimensional movie clip of essentially how 3-D mapping works. It’s essentially a GPS system for catheter location. You place patches on the patient’s skin which allows you to visualize and manipulate catheters in 3 dimensional space and track them without having to use radiation. So what we’ve started doing is we use this for catheter insertion. So if you watch here we’ll track this catheter up as it comes from the IVC all the way up femoral vein to IVC, we can stop at the point where we’ve reached the heart and we have recorded electrical signals so we know we’ve reached the heart. We can mark that as the IVC, we can then advance that catheter push it up, go into the superior vena cava and then we can mark where that ends because we can where electrical activity stops and it allows us to manipulate and place catheters with no radiation exposure.

Not only can we place catheters we can move them around. So here we've created the geometric shell of the right atrium in the coronary sinus. This catheter is going to come down. The coronary sinus is located posteriorly on the left side of the heart, so you'll see here we'll move this catheter, position it over and get it placed in the coronary sinus. This is something that used to take biplane
fluoroscopy to do, we now do this with zero radiation exposure. And then last we create a model and you can manipulate it in the 3-dimensional space. So you can take here this model of the IVC, SVC, right atrium and coronary sinus with several catheters in place and you can rotate it around, see the tricuspid valve there, see the rest of the right atrial surface, you can visualize where your catheter is and where it moves in 3-dimensional space. In the future we can also incorporate existing CT or MRI images into our map so we can then corroborate those images and figure out where we are working inside a patient's heart.

This is an example of - this is how we tag lesions. So if we are placing ablation lesions down we can know exactly where we are in 3-dimensional space. So basically what we did at the beginning of - about a little over a year ago we decided we were going to drastically reduce how much fluoroscopy we gave patients for ablation procedures. So first of all what we did was shift it to the low frame rate to minimize radiation, we for vascular access we changed the frame rate to 0.5 frames per second, and for maneuvering catheters instead of being at 10 or 15 frames per second, which we were previously, we chose 3 to 6 frames per second for manipulation of catheters and transseptal procedures. And we use the 3-D mapping system called Velocity made by St. Jude Medical for catheter placement, for mapping ablation and lesion location. And so now we've done this since June, 2013. It allows for minimization of radiation exposure to both patients and to staff. We typically have plenty of vascular access with really virtually zero radiation exposure, most times we see 0 mGy recorded, it's 0.0 minutes because it's less than 6 seconds of radiation and it's 0 mGy. And
once you do that you can then remove lead which reduces the risk of lead associated work injury over your lifetime.

So basically this is data looking at the original timeframe of this study, first 36 patients we did the standard procedure, the last 15 we've done since then. The fluoroscopy time is reduced from 23 minutes average to 5 with a markedly significant P value and a reduction of about 80% in fluor time. And a radiation exposure in mGy of 340 down to 61, again a very significant P value, but again a reduction of about 80%. And there is no change in success rates or risk of complications.

So in summary WPW is a common pediatric condition that you will encounter, it carries symptomatic risks for patients, tachyarrhythmias, SVT, atrial fibrillation and sudden death. And unfortunately sudden death may be the first manifestation. There is a risk of 0.1% or 1 in 1,000 per year with a lifetime risk to each individual of 3 to 4%. Noninvasive risk stratifications is helpful but it's not a guarantee against future events, and so as families tell me I want to know for sure and I say that noninvasive risk stratification is not good enough. And invasive risk assessment is more comprehensive but ultimately ablation is really the most definitive way for us to eliminate those patients' risk and I think going forward we are able to perform ablation with less and less risks and really with minimal exposure to ionizing radiation which greatly diminishes the risk profile of choosing it as a strategy for patients with WPW.
As you know patient care is a team effort, I'm fortunate to be part of a great team and I'm sure I've forgotten people and I apologize for that but thank you very much.