Thanks John and Fran for the opportunity to come and speak. I’ll try to be, keep on time, I think I’ve been given 45 minutes and I’ve been given the rather large topic of coronary angiography, CT angiography and cardiac MRI to diagnose and manage coronary artery disease we could spend, you know, a whole career talking about it. But I’ll try to create a couple of take home points. I am the clinical director for the cardiovascular institute at UPMC, I have no financial disclosures that would conflict in terms of what I’m presenting today other than to say I am an interventional cardiologist by trade and a rather enthusiastic one and I do work for UPMC.

You know when I give talks in this multidisciplinary types of courses, I always like to consider talks that are right before me because one of the favorite sports I’ve noticed is interventional cardiology bashing if you will. And I noticed that Dr. Johnson took the opportunity to blame cardiologists for all the QC and if you will names that ___ have and of course Dr. Edmundowicz took the opportunity to you know compare us to plumbers if you will which is a pretty, pretty lofty comparison.

My kids will actually tell you that the only difference between plumbers and interventional cardiologists is that if you call a plumber at 3 o’clock in the morning, they’ll tell you to turn off the spigot and they’ll come in the morning. Interventional cardiologists gets up out of bed and comes and takes care of the blockage.
I’d like to do a few things. It’s a big topic so we’re going to start coronary angiography which is a technique which has been around for a long time, near and dear to my heart and then talk about CT coronary angiography and cardiac MR. And really a presentation like this would not be complete today without really talking about some of the issues of radiation exposure and the amount of costs that these tests add to their health care dollars.

Well coronary angiography obviously is a standby and I think that most of us who’ve been in medicine are well aware of the technique, it’s been around for a long time and here’s an example of an angiogram where we inject into the left anterior descending and I always find it useful to review a little bit of the history if you will of how these things came about and I was reminded because I was performing a catheterization with a fellow and the patient asked the fellow who invented cardiac catheterization and she was really unable to come up with the answer. I guess if you really think about it, Dr. Forssman in 1929 could be credited with inventing the catheterization and this is kind of a legendary story in terms of our subspecialty. He was a medical student and he thought that you could place a catheter through the vein to the heart for injection and other purposes and he wanted to prove that on himself. And there are many versions of this story and the most entertaining one is that it was actually a nurse who thought that this was a suicidal move and the claim is that he had to tie the nurse down so that he could perform this. And he did indeed course down into his antecubital vein and insert a catheter. The problem was that he was one floor down from the radiology suite, he obviously wanted to prove that he had done this so there’s stories of him walking up feeling PVCs
because the catheter was ____ on his heart. Now you have to realize that there was no defibrillator at the time so should he have had an arrhythmia that would have been a fatal event.

And this is claimed to be the x-ray where you can see the catheter coming from the left arm all the way into the right atrium. Like many medical institutions his brilliance was immediately recognized by his chief of surgery who immediately banned him from the institution and in fact, he was actually barred from starting, studying cardiovascular medicine and eventually became a urologist. That’s actually a true story. So he was actually a practicing urologist and in 1956 he shared the Nobel Prize for – he was starting the whole field of cardiac catheterization as a practicing urologist.

The next big chapter is Dr. Sones who practiced out of Cleveland Clinic is you will. And this is a depiction of his early catheterization laboratory in Cleveland Clinic and you’ll see the high definition LCD monitor right there and I think all of our surgical colleagues will not the extremely careful sterile technique in practice like in most catheterization laboratories.

Here’s a pediatric cardiologist and at that time there were left ventriculographies and there were tomographies that were being performed regularly. It was actually thought that if you actually inject a contrast selectively into the coronary arteries like we do now it would be a fatal event, that it would immediately cause ventricle fibrillation. So that was thought to be a potential complication. There are many stories about this first angiogram which occurred the day before if you will Halloween. And this is one of them. What is known, I’m not sure about the cigarette being a true
story but what is well documented was that Dr. Sones was a chain smoker and the story is that as they paused for a cigarette the injector actually slipped into the coronary. What is also very clear about multiple accounts, it was an accidental injection, the first coronary angiography. And everyone in the room thought that the patient would BF and they were getting ready to do open heart massage if you will, when the patient was found to be doing quite well actually. It was about a 26 year old gentleman actually from West Virginia who was his first patient. This was a ___ event. After that, coronary angiography came into being and without coronary angiography developments such as coronary bypass grafting obviously, coronary stents would not be possible.

From this well orchestrated beginning if you will there are over a million of these procedures that are performed in the United States every year. It is obviously the gold standard in terms of coronary artery imaging. Unfortunately, I think for most of us it’s a very tarnished gold standard and we’ll talk about why that is the case.

Well then Dr. Edmundowicz had showed you this slide about atherosclerotic time table, this is a simplified version and I mention it because it’s very important for us to separate out the preclinical disease, stable disease and then the ruptured plaque which is the end stage disease and the utilization of coronary angiography so the end stage disease is very different and its actually much more useful than so called the preclinical disease and stable disease which we’ll talk about. I’m not going to talk about the preclinical disease because I think that coronary angiography really does not have a role in terms of imaging in this section and Dr. Edmundowicz has covered most of that already.
And this is another picture similar to what I showed you, a stable plaque if you will. There is about a 50 percent narrowing, glistening yellow stuff, hopefully you can see that in terms of lipid laden plaque there. You know your Rib Mac has a honing device right for that plaque if you will. This is a stable plaque and as Dr. Edmundowicz mentioned for reasons that are ill understood currently, it will in some of the cases convert to a rupture plaque. And the whole such syndrome then becomes unstable at that time. But even if this stops here then it may still be asymptomatic. However, if it progresses to an obstructive stage like this then it becomes an acute MI. And this is really the syndrome we’re talking about in terms of imaging and where does coronary angiography fit in in terms of imaging these blood vessels. How does that help us in terms of managing them?

Once again I like to separate out preclinical disease where coronary angiography or catheterization probably doesn’t have much of a role, chronic coronary disease where it’s most controversial and acute coronary syndrome that is once the plaque has ruptured, where I think the role of coronary angiography is much clearer. And with that I mean unstable angina non SD elevation myocardial infarction as well as SD elevation myocardial infarction.

In terms of the acute coronary syndrome, I think the role of coronary angiography is pretty straightforward and clear in the modern treatment of acute coronary syndrome. That is, it is the preferred method of imaging the coronary arteries because not only does it allow relatively rapid imaging of the coronary arteries but it basically provides us the roadmap for the preferred treatment,
which we can talk about the data but for the – which is mostly mechanical in the current age. This is a right coronary artery and the patient is suffering an acute inferior myocardial infarction. This is what you see when you initially perform the catheterization occluded right in the midsection there and this is the result of primary stenting where angioplasty and stenting so that there’s a stent there and reestablish him a normal flow. So in this syndrome I think that there’s not that much controversy that this is a preferred way for us to identify the coronary anatomy and to institute rapid treatment.

However in chronic coronary artery disease the situation is much more complex. One of the questions is when in the chronic coronary artery disease history or course should we start thinking about coronary angiography and why do we do it? What are we really looking for? If you decided that clinically there’s an indication for revascularization either surgical or coronary artery stenting, then the same applies as before. That is if there’s a good road map for revascularization so coronary angiography is still necessary to guide the therapy and in case of coronary stent placement the same equipment is used so that we can act if its coronary stent placement.

However, if you’re not sure whether the patient needs revascularization or you just want to know what’s going on which is oftentimes if you will the question that’s asked for our patients, that is for prognostication purposes then how good is coronary angiography. Here, to me it is much more controversial. It is a pretty good global predictor of prognosis but it’s not a good regional predictor and Dr. Edmundowicz is absolutely correct, we can tell you through a cardio angiography in stable syndrome that the plumbing system is bad. That it’s likely to give you trouble down the line but it is
very difficult for us to tell you which one of the toilets are going to get clogged up first. And that’s where our problem is.

In reality, what our patients and what we really want is a crystal ball to say you’re going to have trouble in the upstairs bedroom or the left coronary artery when we take the initial coronary angiography. But for a variety of reasons which I’d like to spend about 10 minutes discussing, that is a very difficult thing to do and that’s one of the pitfalls of coronary angiography.

Well, how good is coronary angiography in terms of prognosticating coronary artery disease. As you can see this is a very old slide, 1975 but things really haven’t changed that much in terms of catheterization as related to prognostication. What we know basically is that the more coronary artery disease you have, the more atherosclerotic burden you have in the coronary artery tree, the worse the prognosis. The top line is for a single vessel disease, the bottom two lines for a double vessel and triple vessel disease. The graph would essentially be the same today whether you treat with other modalities such as coronary bypass or not. Treatment modalities may shift it but the single, double and triple vessel disease being sorted out by the disease burden will still remain about the same.

So globally we can prognosticate with coronary angiography. Well, how do we do in terms of telling you which one of the vessels is going to cause an infarct when we see it. No very good. I just take the example of the Courage Trial which is probably familiar to most of you, it’s a
randomized trial that looked at coronary artery stenting with medical therapy in stable coronary
disease, but it’s really the latest and the biggest example of many trials that are failed. I know it’s a
little bit difficult to see the graph on the back but fundamentally what it shows is that the coronary
stent arm of the medical therapy and arm overlap in terms of survival and risk of myocardial
infarction. That is trial after trial, coronary stenting in stable coronary disease has failed to prevent
myocardial infarction. And actually if you look at the trials carefully that’s also true of coronary
artery bypass grafting. We can sometimes demonstrate populations that do better in terms of survival
but very rarely in terms of the rate of myocardial infarction with coronary artery bypass grafting. So
that suggests that we have really no good way of predicting which one of those plaques are going to
rupture, which one of the vessels that can cause a myocardial infarction. Why is that? Remember
this is the sentinel event. This changes everything from a stable syndrome to an unstable syndrome.

As Dan alluded to, our ability to if you will predict the vulnerable plaque is currently very limited.
This is really the Holy Grail of interventional cardiology if you think about it. If we could tell you
which one of the plaques we see during a coronary angiogram is likely to rupture and cause a
myocardial infarction we could put a Band-Aid there, that Band-Aid is called a coronary artery stent.
But we could do Band-Aid or some therapy to that lesion to prevent a myocardial infarction but we
currently have a technology to really predict which one of the plaques will rupture. Hence, we can
tell you what the overall situation is but not exactly where it’s going to happen and when it’s going
to happen.
Why is that the case? This is a slide that we teach all first year cardiology fellows. That is, it’s a study from 1995 and what Dr. Falk did was to look at patients who came with myocardial infarctions and these patients had for whatever reason, had an angiogram within 2 years usually before the myocardial infarction and he looked back to see how severe the lesion was in the catheterization before the myocardial infarction was in terms of that infarct related artery. So what’s concluded is this graph on the right. So 60 percent of the time blockage that eventually ruptured and became a myocardial infarction was less than 50 percent. And in fact, only 14 percent of the time was it what we would call a severe lesion, greater than 70 percent. So a large majority of myocardial infarctions actually occur in narrowings that aren’t that severe.

Does that mean that the percent stenosis has really no bearing on that, well that’s not quite true either. Because if you look at this, so if you look at the chance over a 5 year period that a stenosis would go to an occlusion or bring about an infarction, that is a severe lesion from 80 to 95 percent has about a 24 percent chance of progressing to a complete occlusion. Okay? That’s definitely higher than a narrowing that’s less than 50 percent. But the problem is, it’s kind of like an auto accident phenomenon. That is most auto accidents occur not in the high risk intersections, the low risk intersections. Because there are so many more low risk intersections than there are high risk intersections. So yes, if you have an 80 percent narrowing that’s more likely to rupture and cause you a heart attack but the problem is, we probably have thousands of 5 to 50 percent narrowing where we may only have hundred or so 80 to 90 percent narrowings in this population.
So overall it is a good predictor of overall risk but it is not a good predictor of when and what vessels the myocardial infarction would occur. Well, let’s think about that a little bit further. Why does angiography fail in this regard? Well if you think about what we do in angiography, there are many reasons why it doesn’t make that much sense. What do you get when you send a patient for a cardiac catheterization. You get a report from a cardiologist like myself, usually saying, there’s a 70 percent narrowing in the left anterior descending, maybe 50 percent in the left circumflex, but it’s basically describes multiple percentage narrowing. It’s a two dimensional description of what is a 3 dimensional structure that is not even straight, that is full of curves if you will. There’s the issue of reference diameter, there’s observer variability that we talk about because these are the reasons where we progress on, just the anatomic stenosis to more functional assessment in the cath lab.

Eccentric stenosis, well it doesn’t take rocket scientists to quickly think about what we’re trying to do with identifying a stenosis as a 50 percent or 70 percent. If you look at these two narrowings, what does it mean that it’s 50 percent narrow. You would think they were talking about the area but we’re not. Most of the reports are really talking about simply a two-dimensional picture. So if you look at this, if you’re looking at it from this angle, going straight down, it’s really not 50 percent narrow. If you look at it from this angle it looks to be pretty severely narrow.

What you get when you get a report of a catheterization is basically the angle that seems to give us angiographically the most severe narrowing as a two dimensional picture. If you think about it it’s
pretty unlikely that that’s going to be an adequate description of the blood flow through this vessel. But that is the gold standard as limited as it is.

Well how about the question of reference diameter. Here’s the left anterior descending injection, here’s the left main, distal left main with some narrowing and LAD throughout. If you get an intravascular ultrasound or a CT coronary angio on this vessel I can tell you that the whole vessel will be diseased, there’s an atherosclerotic plaque from probably there down to there. So how do we go about describing a vessel like this? Is this a 50 percent narrowing? If you say that the reference diameter is right there, you could say it’s a 50 percent narrowing. If you think that the vessel really should have been this size, maybe an 80 percent narrowing. So these are very difficult questions and what you’re getting really and it’s important to understand is really a semiquantitative and really more of a qualitative interpretation from the angiographer or the cardiologist.

And has it been studied? Well, it’s been studied ad nauseam and even earlier than 1976 this is a circulation paper where four observers looked at the same angiogram and this is kind of a scary concept if you will and you can see how variable some of the readings were, pretty remarkable. If you think about the angiogram right before in that slide, you can kind of understand why you would get such variable readings from an angiogram.

So with that, despite that obviously it gives some prognostic value and it’s not that surprising that we’re not very good at really predicting when an infarction will occur. So what else can we do? So
as a field from a coronary angiography standpoint and as cardiology, we’re moving away a little bit from a simple two dimensional description as narrowing of a functional description as you will. And following Dr. Johnson’s if you will, a trend, this is called a Fame study if you will which is a fractional flow reserve versus angiography in guiding the percutaneous coronary intervention. So the concept is that as this ___ shows what the angiographer does is to put a wire across the narrowing and instead of just measuring the degree of narrowing, we measure the pressure gradient under certain circumstances with is hyperemia. That is how much of a pressure gradient difference is there between the mean arterial pressure before the narrowing and after the narrowing and this ratio is what’s called FFR and its been validated to show that if this number is less than .75 which is not an important number except for those people who do it. But below a certain number it has a much higher risk of that narrowing progressing on if you will. So it doesn’t tell us exactly when it’s going to rupture but it’s a better concept than simply looking at the anatomy and saying there’s the present stenosis and trying to guesstimate about what the chances are that such a thing is going to rupture.

Well how does it work? The Fame Study came out about a year ago, it’s a pretty revolutionary study if you will. From the back the red line is basically if the percutaneous intervention, the coronary stenting was guided by angiographers interpretation of angiogram, I just showed you all the pitfalls of that. On the blue line the coronary stenting was guided by measuring the FFR in all of the narrowing that was thought to be possibly significant. That is if the FFR was greater than .75 nothing was done, if it was less than .75 than coronary artery stent placement was placed. And the remarkable thing is that such a guidance will actually decrease the rate of repeat revascularization,
even seem to decrease the rate of myocardial infarction and some hint of increased survival with such guidance. So these are the things that in the modern cath labs, we’re moving more and more towards if you will, so called functional assessment of the narrowings that look severe to try to make a decision about whether it makes sense to perform mechanical therapy what we call Band-Aid therapy if you will.

Just to kind of reiterate the point in a separate study done in 2001, like I said, if .7 FFR or less than .7 FFR is the magic number for multiple studies showing that FRR less than that is associated with poorer prognosis and of greater that that. You can see in this population all these dots once again represent single patients, this is the percent stenosis as read by the angiographer. The percent stenosis is really all over the map. There’s some percent stenosis that was read as 40 percent if you will. Where the FFR was less than .5 so there was a significant pressure grade across such lesions. Other lesions that look like 70 per cent here where the FFR is normal. So once again demonstrating that the percent stenosis as a single entity tends not to be so reliable. And this is once again in chronic stable angina, chronic coronary artery disease, it’s a different ballgame when you’ve already ruptured a plaque and are suffering from an acute coronary syndrome.

What are other technologies we’re using. We’re forever, like I said the Holy Grail is to predict which plaque will rupture. Just a brief word on, it’s optical coherence tomography if you will where with the catheter inside the blood vessel, this is a coronary artery, I believe a right coronary artery we’re able to image in this very new technology that was FDA approved about 3 months ago. And I
just want to show you a picture. This side is essentially normal, this is what a normal blood vessel should look like, this is the lumen of the blood vessel where the blood is flowing. And here’s this thin wall. Here is what all the cartoons that Nan and I have been showing here, here’s the plaque if you will. And you see that’s a heterogenous plaque. And what we’re trying to learn and see is what are the characteristics of the plaque which will tell us that it’s the vulnerable plaque. Has a high likelihood of rupturing because some plaques will stay like this for years and never change. Those we don’t need to do anything to treat other than systemic treatment. This technology is not quite there but it’s probably the most promising in terms of having the ability to predict which are vulnerable plaques if you will.

And here is an example of such a ruptured plaque. You can see here instead of that fluid line you saw on the blood vessel that was just above this segment, here it’s clearly what we call a flab, that is the plaque has ruptured and it is intruding into the lumen and this is what likely will cause thrombosis and acute coronary syndrome.

So how about coronary angiography. It’s really a tainted gold standard, its limited in many ways, it’s actually remarkable it does as good as a job that it does in terms of prognosticating. You can see this from the New England Journal editorial, 1987, people have been predicting the demise of coronary angiography from clinical usefulness standpoint for many, many years yet it’s hung on. The last sentence is interesting. It says only when technology can offer noninvasive imaging methods capable of demonstrating the anatomical distribution of the obliterative process in the
coronary arteries will coronary angiography die. He was talking about CT coronary angiography because that’s exactly what that technology does and that technology came to us in about 2005 and yet we’ll talk about why it hasn’t taken off more than it has. You would think from my description of coronary angiography that CT coronary angiography would have completely replaced it by now.

Everyone’s now familiar with kind of the basics of the CT as you’ll recall a rotating detector and a radiation source if you will. But what’s different is that we now have multi-detectors, that is standard being a 64 detector CT that rotates. The challenge in terms of coronary angiography with a CT was the epicardial coronary arteries were too small, they were moving and so the detectors weren’t fast enough. What’s different now is the gating technology and the multi detector allow such fast acquisition, we can now get good images of the coronary arteries of the CT.

When it began and commercially available in about 2005 there was much hoopla, the cover of Time, this a 3D dimensional depiction and look what the headline says, stop a heart attack before it happens. From this article you think that they already found a way to really identify the vulnerable plaque but that’s really not the way you would stop the heart attack.

What came to us in about 2005, obviously the advantage is that it does give a coronary angiogram and this is different than calcium score where Dan mentioned, just measured the calcium, it doesn’t, calcium score doesn’t tell you that there’s action obstruction. CT coronary angio actually shows us where the blood vessel is in percentage obstruction. The advantage is it’s non invasive and it allows
visualization of the total plaque burden and as you’ll see it allows us some plaque characterization. But we’re not sure what that means right now. And it certainly gives us very, very pretty pictures as you can see from here. It is a 3D reconstruction, a CT coronary angiography with an aortic root here, there’s a right coronary artery coming down around, its actually an anomalous coronary artery, less circumflex which really comes from the right cusp and courses around. So it gives us very beautiful images, allows us to really see how the coronary artery traverses throughout the anatomy.

And here it gives us plaque morphology and this is a different way of looking at it, different way of processing it if you will. Here’s the left anterior descending coming and this is what we call a soft plaque, we have no idea if its really soft but it’s called soft because it looks like that in contrast to something like this where you have calcium so that’s called hard plaque. And here’s another plaque right here that intrudes into the lumen and these are just small, for example there’s a picture there, it’s a calcified lesion also. So certainly CT coronary angiography is very successful. Once again in times of identifying the lumen stenosis, let’s remember the same problem applies in terms of chronic coronary artery disease, in terms of what that means, which one of the narrowings is going to give us the heart attack, those are really the magic questions which still haven’t been answered.

Why hasn’t it taken off more? CT coronary angiography – because it’s non invasive, it should be more readily available. There’s some limitations. Radiation dose is pretty considerable. In fact, it’s higher than most diagnostic angiography, diagnostic cardiac catheterizations. The contrast exposure
is actually higher in volume for renal failure patients or renal challenged patients than for diagnostic catheterization. And even though the radiologists have gotten much better there are some heart rates limitations in that it has to be regular, A-fib doesn’t work. PVCs don’t work and we have to induce relative bradycardia with beta blockers in most cases. And in addition, one of the barriers it has some very inconsistent reimbursement and the reality is that has to really decrease the growth rate whether rightly or wrongly of CT coronary angiography in this country.

What is it good for? It is actually the study of choice for anomalous coronary arteries, if you have a coronary artery that’s does not fill up normally, the usually 3 coronary artery QT, CT coronary angiography probably gives us the best picture. It has excellent negative predictive value, that is if you need a study to rule out coronary artery disease, someone with an equivocal stress test, someone who for whatever reason, medical, social or psychological really needs to rule out coronary artery disease and you get a negative CT coronary angiography, for most of us that’s as good as having a clean chords in a cardiac catheterization.

And here’s a metaanalysis of multiple studies. Blue is the negative predictive value, all excellent, 90 to 100%, and really depends on what the end value is, whether you are saying it’s coronary ischemia or presence of some coronary artery disease, but suffice it to say most of us believe that the negative predictive value of CT coronary angiography is excellent. Positive predictive value is all over the map, the red bar. And partly because that depends on, as you might imagine, what the pretest probability is and what the population is that’s being tested.
Just this week in the Journal of American College of Cardiology, if you will, ACC multi-speciality if you will committee has come up with the most current appropriate use criteria for CT coronary angiography. So if you want to look that up it really is an exhaustive list. What they did was to go through 93 clinical scenarios and they felt that in 35 of these that CT coronary angiography was reasonable to use and appropriate. I’ll summarize it by saying mostly agreed that the best, it was the test of choice for anomalous coronary arteries, which is a really small population that in general it is reasonable to use in stable coronary artery disease to identify the severity of coronary artery disease. It is less useful in acute coronary syndrome for the reasons that we discussed before in terms of roadmapping therapy.

Here is a different picture, once again here is an anomalous coronary artery, and the reason that this is different than the other picture is just the way software has post-processed it and shown the other structures of the heart. Left anterior descending coming down here and the right coronary coming down this side of the heart, and here once again this shows an anomalous coronary artery that traverses anterior to the pulmonary artery and the RV and to the anterior wall. So it’s an LAD that actually comes off the right side of the heart, this is another picture of the same thing. These are actually very difficult to fully understand the course when you are doing coronary angiography, so this is a big advancement; but once again, a pretty small population.
Well how about cardiac MR? This is a picture from Dr. Eric Sherba who is the Director of Cardiac MR, UPMC just reminding us that this is not like your father, I guess as the commercial goes your father’s MR. Current MR, not necessarily for cardiac MR or necessarily, it is wide bore, not totally open per se. And in fact as odd as it seems if you think about the old pictures of MR, one of the advantages of cardiac MR for cardiac studies is they can accommodate very large patients without degradation of the images. That is as long as the patient fits here up to 510 pounds then we can get images that are the same quality as a 90 pound person, which is not true of nuclear imaging, echocardiography or coronary angiography.

Cardiac MR advantages are pretty obvious. There is no radiation, obesity is less of a limitation and in terms of what tests we actually do for cardiac MR there’s actually less so-called coronary angiography. That is it’s less direct visualization of the coronary arteries and more regular stress testing and cardiac MR is currently the gold standard for infarct detection and sizing in an intact patient. If you look at most clinical trials that want to see what exactly the size of the infarct is, whether there is a truly infarct or not, cardiac MR is the best for accounting. It’s excellent wall motion definition and despite our biases it turns out that the cost at least currently is about equivalent to a CT or a – equivalent to echocardiogram or nuclear imaging. Nuclear imaging actually tends to be more expensive, which is surprising.

Unfortunately this is not going to play, we had some technical difficulty but this is a picture of cardiac MR trying the wall motion which is excellent. What are the limitations of cardiac MR?
Coronary angiography is less mature than CT, so I don’t think it’s really, most of us agree, ready for prime time in terms of actually visualizing the coronary arteries, and the availability is severely limited. It actually requires a pretty special software, so your regular MR machine being used to do backs, neuro MR doesn’t mean that they will be capable of doing the cardiac studies. And it requires a fair amount of physician and technologies expertise which is not widely available, and one of the reasons that MR probably will not take off that much in terms of cardiac imaging. Pacemakers and defibrillators are currently a contraindication, although that’s probably changing. Manufacturers are coming with MR compatible pacemakers and defibrillators and probably within the next 5 to 10 years will be less of an issue.

And just one of the questions that we get asked a lot, coronary stents are not an issue. If you look at the labels they say to wait 6 weeks, we routine perform MRs, cardiac and otherwise, in patients with stents even with 24 hours. There’s never been a reported case of any problems, so that’s never an issue with the current modern day stents.

Well, what about cardiac MR? It’s really the study of choice for structural heart disease, congenital heart disease and certain infiltrated conditions and hypertrophic cardiomyopathy. But its use in terms of coronary artery disease is relatively limited, mostly looking at stress tests, wall motion sizing of the infarcts.
Well we can’t really talk about imaging without really talking about radiation exposure in dollars, because this is very pertinent. If you look at cardiology the usual cardiology bashing is that we are trying to bankrupt the economy, which is somewhat justified, 2009, $2.5 trillion in healthcare spending, cardiac and vascular and neurovascular disease accounts for almost 20% of that. It’s not really clear but some accounting says up to 20 to 25% of that is due to imaging.

How good are we at growing imaging procedures? Very good. The blue line is cardiac catheterization from 1979 to 2006, you can see almost a straight up curve. The more alarming green line is that is the number of percutaneous coronary interventions performed in this country, now over a million. You’ll be glad to know that they did finally flatten out over the last 5 years. So in fact the number of PCIs being performed in the United States has actually decreased slightly, but not much, they are still pretty high.

And I guess Germany now you know that catheterization was invented there is really the only western country where you are likely to be undergoing a cardiac catheterization is higher than the United States. We cath about .4% of the patient population in the United States every year, it’s about 4 times the rate, if you will, of our neighbors in Canada and well over 4 times the rate in United Kingdom.

And how fast is imaging growing? This is a Medicare curve if you will, the top line that’s the imaging services, all the other lines are other physician related services, clearly outstripping
everything else. The number of CT scans in this country is no surprise if you will. This isn’t cardiac CT but this is the number of CT scans in this country.

Well, in addition to the obvious questions about costs it’s raised a significant amount of appropriate questions about radiation exposure. What are we doing with all these imaging procedures? And if you look at Brenner’s estimate from 2010 this is a pretty alarming if you will statistic. In 1980 30% of U.S. population’s radiation exposure was thought to be due to medical imaging, in 2010 it’s thought to be 50%. And cardiac testing seems to account for about 30% of that exposure.

These are the radiation, estimated radiation doses of the various procedures if you will. If you will, you can use kind of the standard as a chest x-ray. I think there are probably some surprising aspects for most people. Coronary angiography is 7 or 70 times what you would get from a regular chest x-ray, 64-slice CT coronary angio is twice that of a coronary angiography, which is surprising. But what is most surprising is the stress testing that we order all the time. The nuclear stress testing depending on how it’s done is anywhere from 120 to 410 times the radiation dose of a chest x-ray, and I think all of us can identify patients who get stress testing for one reason or another almost yearly or every 6 months, so you can just think about the cumulative radiation dose. The fact is we don’t really know how much of a cancer risk these are, but I think it’s safe to assume that that once you start getting repeated testing with these modalities that you run into a significant cancer risk in that regard.
Well, what can we conclude? Coronary angiography is still the gold standard. It is very, very imperfect even from a person like myself who performs it on a day to day basis. It has significant limitations in terms of the information that we want to get out of it. But it’s likely to stay because it is the platform through which we treat acute coronary syndromes and once the plaque has ruptured. It’s role in terms of stable disease I think will change. CT coronary angio seems to have the lead in terms of increasing utilization for stable coronary disease. However, it’s still not clear what we are going to do to the information, right? As Dr. Edmundowitz has explained, since we can’t predict which plaque is going to rupture how much do you really need to know about how much atherosclerosis there is in a patient? If you already know, then you are going to maximize preventive and medical therapy obviously. Cardiac MR in my opinion will likely remain a niche type of a imaging because of the difficulties of access and the technical expertise and these other modalities are more readily available.

I think it’s important to really realize that the concerns regarding radiation exposure are real in terms of cardiac imaging. It’s become such a large chunk of imaging and how we manage cardiac patients it is a significant issue, we do have the capacity to really bankrupt the healthcare economy, if you will, because of the rapidly expanding number of testing that we are doing. The biggest issue with cardiac imaging, coronary artery disease, is really not necessarily the limitation in technology but our decision making is not as advanced as it should be. When should we use it? What is this most, if you will, the highest yield in terms of the currently available technologists that are available?
I just want to take one minute just to show you a case which really exemplifies many of the conundrums that we get into because of the availability of all these techniques. This is a real case that I have been taking care of for about 10 years and I think it’s actually Dr. Saloma’s patient. AC presented when he was 52 years old, family history of coronary disease, not horrible. He was sedentary, he was certainly concerned about coronary disease and not unreasonably from Dr. Edmundowitz’ presentation he got an EBCT. He was 95th percentile in the calcium score, he looked it up and all those graphs that Dr. Edmundowitz was showing you, and he said whoa, apparently it’s kind of high. I’d like to get a stress test. Not unreasonable. He went 9 minutes and 30 seconds on the protocol, not horrible for a 52 year old, absolutely no chest pain. He is absolutely asymptomatic no matter how many times you ask him, and how many differential angina questions you ask him. ST depression, inferior hypokinesis and after hearing about this, he realized that this actually increased his risk for a cardiac event, so we proceeded to a cardiac catheterization.

Well it wasn’t good, he’s got 3 vessel disease, he’s got a completely occluded right coronary artery, completely occluded left circumflex artery with good targets and the LAD has a severe stenosis, if not a total occlusion. So what do we do now? He’s got very severe 3 vessel disease, completely asymptomatic, 52 year old gentleman. Well, what you can tell him is that in terms of the world literature all of the randomized clinical trials that demonstrates mortality benefit or revascularization in asymptomatic coronary artery disease patients is contained on this page, okay. There are none. There is no randomized control trial that’s going to tell us that revascularization will decrease this gentleman’s mortality. You can also let him know that of more than 300 sudden cardiac deaths in
this country, more than 50% of those never have a warning sign. They probably all look like this
gentleman, but what do we do? What are we supposed to do?

I can tell you what we did. So he underwent coronary bypass grafting in year 2000 and he got
aspirin, statin, beta blocker. Since he never had any warning signs it wasn’t really clear how to even
monitor. So he’s had a stress test in 2005 because we kind of believed the 5 year warranty period of
the bypass surgery, and then yearly since that time. They have been totally negative, he is totally
asymptomatic, totally normal lifestyle. Of course after the initial down time of his coronary artery
bypassing, bypass grafting.

But the real question is thinking about a case like this, what is the reality? Did we really save his
life? Did we find an impending disaster before it occurred? Or what would have happened to him
otherwise? Or did we just expose him to life threatening surgery and probably $100,000 worth of
medical care without really changing anything else other than putting him out of work for about 6
months? Thank you very much for your attention.