So I don’t think I have to tell you this since you guys basically practice medicine that heart failure is an enormously prevalent condition right now. The estimated prevalence on the most recent reliable data are about 6 million Americans with this diagnosis with an annual incident that’s approaching a million a year. The hospitalizations are really quite high and accordingly the cost of this is pretty extraordinary. It takes up a big chunk of health care expenditures with probably the best estimates being somewhere in excess of $40 billion dollars a year. Now most of that comes from hospitalizations and in turn most of that comes from re-hospitalizations and you guys are probably already starting to get some pressure either subtle or not so subtle about trying to attack this problem of re-hospitalizations for chronic conditions. Heart failure is probably the quintessential chronic disease for trying to make a dent in that. Somewhat surprisingly I think the mortality of this disease despite all the advances that we’ve made, and I’m going to highlight some that you’re very familiar with and then hopefully some that are so new that you might just be first hearing about them, the mortality remains really distressingly high. You can see that if a patient is found to have left ventricular systolic dysfunction with essentially no symptoms, so in other words NYHA class 1 heart failure, they still face about a 20%, 4 year mortality. And then once you get to people who are really severely burdened with symptoms, despite everything that we do with mechanical circulatory support, with transplant, with aggressive medical therapy, we’re still looking at a 50% one year survival. So this disease despite everything we’ve been able to do still takes a big toll.

The future does not look any better. I suppose you could look at this in one of two ways, you could look at the data I show on this slide and say boy that looks pretty bleak, we’re expecting over the
next couple of decades a 25% further increase in the number of people affected with this disease and a cost increase that’s about 3 fold what we see right now. On the other hand, most of the reason that we are seeing this increase, not all of it but most of it, is that the population continues to age, I’m going to show you some compelling data that suggests that this is very much a disease of the elderly and also because we are progressively more successful in treating or at least ameliorating coronary artery disease, valvular heart disease, etc. and so if people aren’t dying of myocardial infarctions, unfortunately it’s somewhat inevitable that a chunk of them will be dying of congestive heart failure, at least developing that disease. Nevertheless, the projections are that we’re going to be seeing an increased number of this.

This gets at some of that epidemiology that I referred to so one way to think about this is that in the U.S. this disease doubles per decade that you live after the age of 50. So if you’re in your 50’s you have about a 1% chance of having congestive heart failure and then each decade after that it doubles so by the time you’re in your 80’s it’s about 10% or so. There’s a slight male predominance throughout. I’ll show you a graph that demonstrates that and in our country despite most of what you’re reading in the literature being sort of largely swayed by non-ischemic or primary cardiomyopathies, the vast majority of heart failure we see relates to hypertension, coronary disease or the combination of those 2 things. And we phrase it that way because its’ very hard to tease out hypertension from CAD, they really go hand in hand in the vast majority of patients.
This is a graphic demonstration of the age impact on the diagnosis as well as the male/female differences. You can see that there’s a male predominance throughout, but while that is most magnified in the fifties and early sixties, by the time patients are elderly that pretty much evens out.

I want to talk a little bit about lifetime risk and how it pertains to heart failure because I think there’s a very important lesson in this. So these data come from Don Lloyd-Jones who’s the chair of preventive medicine at Northwestern and a dear friend of mine. He’s been working on this sort of analysis for a lot of years now and these 2 graphs show the lifetime risk for coronary heart disease broken down by age and sex, so if you look on the left hand graph, let me see if I can point this out to you. If you’re a male in our country and you’re 40 and you have not had any coronary heart disease, over the rest of your lifetime you have about a 49% chance of developing CAD. That doesn’t sound too crazy, we know CAD is a very common condition. If you’ve made it to 80 and you’ve never had CAD you have a much lower chance of developing it in your lifetime, only 24%. And I’m not too surprised by that I think if you’d asked me that question I probably would’ve said if you haven’t had CAD by the time you’re 80, you’re probably less likely to get it before you die, I don’t think anybody is surprised by that.

Look at the same type of analysis though for congestive heart failure. It does not matter whether you are 40 and haven’t had CHF, or you are 80 and you haven’t had CHF, you still have exactly the same risk of developing it in your lifetime and that is because it is so much a disease of the elderly that the prevalence really picks up as you get into the older age groups. I guess the message for you guys on
CONGESTIVE HEART FAILURE – NEW APPROACHES TO AN OLD PROBLEM,
MICHAEL MATHIER, MD

the front line is just because you have a patient that looks hale and hearty in their early 80’s in your
office doesn’t meant that they still don’t have a significant risk of developing congestive heart failure
and you ought to be thinking hard about the risk factors that we’ve identified for the development of
the disease and trying to target aggressive risk factor modification.

Here are those risk factors. I don’t think there’s anything on this slide that will be surprising to you.
We already mentioned CAD and hypertension, we know valvular heart disease plays a role in this,
alcohol and other toxins can, diabetes both working through other intermediary disease states, you
know such as coronary artery disease, but also probably a primary cardiomyopathy of diabetes and
then some kind of lifestyle type of issues including age, obesity, smoking, family history on the
genetic side. Especially in the elderly patients there’s a couple of things that have come out in some
very interesting publications over the last decade or decade and a half. One is the really profound
impact of CKD and I’m talking really mild CKD. So whether you measure it by just a simple serum
creatinine or by creatinine clearance, even very mild degrees of CKD appear to be associated with 2
and 3 fold increases in the risk of developing an incident episode of congestive heart failure.
Another interesting one, sorry my graphics got screwed up here, is an increase in pulse pressure. So
this idea that if you’re measuring a blood pressure especially in an older person and you find that
their pressure is something like 140/60 that kind of increased pulse pressure really implies arterial
stiffness, and arterial stiffness in a consequence is an increased load on the left ventricle and is
associated with the development of heart failure. Then lastly, subclinical thyroid abnormalities I
think we’ve all gotten pretty good on the frontlines for looking for those, but they have been clearly associated with it in the elderly.

A number of these risk factors really point to a certain kind of heart failure and I think you guys know, we really are actively trying to separate out the way we think about what we now call HFREF which stands for Heart Failure with Reduced Ejection Fraction or low EF, versus HFPEF; heart failure with preserved ejection fraction sometimes called diastolic heart failure. The phrase of choice is HFPEF even though it sounds silly because we don’t really know in these patients whether diastole is the true problem. A lot of people feel that for many of these patients diastole actually isn’t too disordered, but instead there is a problem with the coupling of the ventricle to the arterial system and so what we choose to call it is that we have clinical heart failure but at the same time the ejection fraction is preserved. The reason that we really need to make the distinction is that our evidence base differs according to which category the patient fits in and therefore the treatment is likely to as well.

These data from the ADHERE registry I think really point to the magnitude of this particular issue. So ADHERE has been around a long time. It was an industry sponsored but very robust registry that included a lot of hospitals on both the academic and the community side so probably the best real world data we could get in terms of congestive heart failure. And you can see some aspects of it that really tell us what heart failure is and that is mean age in the mid-seventies or so, so we’re talking about elderly. In this case the women were slightly more prevalent that’s because this really skewed
towards older patients, 60% with CAD, 30% with CKD and about 50% actually a little more than 50% of these patients had preserved ejection fraction. This is a registry that really targeted patients being admitted with acute decompensations, primarily fluid overload. So this entity of heart failure with preserved EF is actually the principle dilemma we face in congestive heart failure right now. All the data that we have, all the big studies that have shown benefit of the various drugs that I’ll touch on, that’s all been done in reduced ejection fraction. To date we don’t have a single drug, not one single drug that has been shown to be effective with patients with heart failure with preserved ejection fraction. It’s sort of a big gap in our therapeutic armamentarium.

For a long time we used to think that these folks, yeah it’s a tough disease, it’s hard to manage your fluid and it is, but it’s a good prognosis and in fact that’s not really so much the case. If you look on the left side of this graph you can see for heart failure with reduced ejection fraction compared to normal controls, age matched etc., there’s obviously a big mortality risk with the presence of low ejection fraction. But if you look at HFPEF, Heart, Failure with Preserved Ejection Fraction, really normal EF, compared again to age matched controls, even though the magnitude or the mortality difference isn’t quite as great, there is still a significantly reduced mortality in these patients. Now most of this is going to come down to the fact that they have, generally speaking, a pretty heavy comorbidity burden. A lot of them have hypertension, diabetes, CKD, etc.. But nevertheless, this idea of thinking of HFPEF as a benign condition that’s just kind of a pain to manage is really misguided. This is a disease that has significant morbidity and mortality.
I want to talk a little bit about how we think about patients because this plays directly into how we treat them. So you guys are all familiar I think with the New York Heart Association Functional Classification, the basic idea that we can make a subjective determination of how sick our patients are with heart failure. So that if somebody has reduced ejection fraction but they’re completely symptom free we call them Class 1. If they have symptoms at rest, we call them Class 4, and we kind of in a very shady way divvy up the 2’s and 3’s according to whether they’re getting symptoms with a lot of exertion or with not much exertion and it’s a really limited system to use because it’s really highly subjective. My three might be somebody else’s two but nevertheless, it’s so engrained in the heart failure literature that we’re kind of stuck with it. Now what the ACC, the American College of Cardiology, the American Heart Association have been doing over the last decade is trying to gradually replace the Functional Classification with a staging system and you see it on the left and you see how it kind of corresponds to what the old NYHA functional classification was. So one difference is that we have a Stage A category and this is to highlight those patients who do not have any manifestation of heart failure but have significant risk for the development of heart failure. So these are folks who have hypertension, diabetes, vascular disease etc., where we’re trying to get the message out that you really have to target these things aggressively in the hopes of reducing the incidents of heart failure. Then you get into Stage B which is structural heart disease but without any symptoms. This corresponds to the old functional Class 1. You get into Stage C which is structural heart disease with either prior or current symptoms that really gets at the idea that you can never go to a lower stage and the importance of that is that we’re trying to highlight the fact that even if, if somebody comes into the hospital and they’re having symptoms and I buff them up and I
get them home, you can’t start to get complacent with that patient. Because even though they feel good now, they’re still at very high risk for decompensating in the future. So it’s another kind of classification schema that’s trying to get us to pay more attention to patients who are at risk for future events. And then Stage D is refractory heart failure that requires special interventions I’ll touch on that towards the end of the talk including mechanical circulatory support and transplant and where that all stands these days.

Just a little further mention of diastolic heart failure or HFPEF, you can tell this slide I made a little while ago before we changed the terminology, again about 50% of heart failure, marked volume sensitivity. What I mean by that is that these folks operate or need to operate in a much narrower volume window than even my patients who have big, dilated hearts with EF’s of 10%. And the reason for that is really summarized in these 2 functional curves. Over here this is basically don’t get nervous it’s physiology, I know we don’t think about that much anymore but this is a Starling curve here and if you’ll remember what the Starling curve suggests is that one of the ways that we increase our cardiac output when we have more demand is by increasing filling of the left ventricle, we have a certain preload reliance to increase in cardiac output. In somebody who’s got hypertensive cardiomyopathy what you see in these patients is if you work backwards instead of thinking about oh yeah I’m going to increase my end diastolic volume to increase my cardiac output, what’s happening in these folks is that as I decrease their filling by diuresing them if I’m not careful and I overdo it they have a very precipitous fall in cardiac output. Okay so that’s one
principle to remember, the idea that these very stiff ventricles have pre-load dependence, you need to give them adequate loading for them to generate a decent cardiac output.

The other curve is the left ventricular end diastolic pressure volume relationship. How filling of the ventricle translates into pressure and that pressure is then filled backwards into the pulmonary circulation. In normal circumstances, this is a very flat curve. I can give you guys if you have a normal heart, a couple liters of saline your ventricle will get increased filling but you won’t see a big increase in pressure and you won’t develop pulmonary edema. But if you have a lot of stiffness in that ventricle, that curve is much steeper and even modest increases in the filling is going to lead to a marked increase in end diastolic pressure and therefore, dyspnea. So these patients if you kind of overlap those 2 curves you have to have them in a really narrow volume window. Too little, their cardiac output falls. Too much and they develop pulmonary edema, shortness of breath. This is why they are so challenging to manage on the out patient side.

Other things that play a role is the super imposition of ischemia which worsens diastolic function acutely. Hypertension which adds some loading that can worsen heart failure, and then atrial fibrillation because typically those hooks go fast, what they want is actually more diastolic filling time, not less. But when they go fast they don’t get enough diastolic filling time. And also they lose their atrial kick which is important when you have a stiff ventricle. So these are all complicating features that we see in diastolic heart failure.
We’re going to turn our attention now to systolic heart failure because even though it probably should share equally with HFPEF this is really where all our data are and really I think the treatment part of this is really best focused on low ejection fraction. So these colors on the pyramids over here on the left represent the stages from A-D. Stage A these are folks remember who we think are at risk for the development of heart failure but don’t have it yet. This is all about kind of lifestyle intervention and risk factor modification. If you have established LV dysfunction but no symptoms we know that ACE Inhibitors and I believe and virtually everybody, beta blockers are important. Once you get up to manifest symptoms and low ejection fraction we’re looking at ACE inhibition beta blockade, typically diuretics, Digoxin which is I think decreasing in its frequency of use mostly because we’ve entered this era of polypharmacy and DIG has a very narrow therapeutic window with a high chance of toxicity. I’m not sure how much it actually contributes in modern heart failure care. Aldosterone receptor antagonists such as spironolactone or eplerenone and then again lifestyle modifications. And then lastly you get into folks who’ve got refractory symptoms we’re doing everything we can and they’re just not making it and then we’re looking at inotropic therapy, mechanical circulatory support and transplant. That’s kind of the paradigm that we’re looking at.

Just a few pearls I think for each of these things, you know diuretics aren’t very exciting to most people, we know they improve symptoms by relieving congestion, remember they have never been shown to improve survival, they’ve never been shown to actually have any heart end points in heart failure predominantly because you can’t really withhold diuretics from a heart failure patient.
There’s some evidence that actually the more diuretic that you use in a patient the worse they do in the long run and understand that gets into sort of a chicken and an egg argument, you know are you using more diuretic because they’re sicker, probably. But it does look like diuretics can have some adverse effects including electrolyte disturbances, activation of the neural hormonal systems that we’re trying to tamp down with our other medications, etc.

Oh so one of the things that I try to do with diuretics just to sort of something to think about is when you have a patient presenting volume overloaded with heart failure regardless of the etiology, you want to be pretty darn aggressive to get them euvolemic and then you want to back off as far as you can and see if you can get them on a minimal dose of diuretics. And most of the time what I’m trying to do with a patient is get them to make their own decision day to day on how much diuretic they need that day and give them some parameters to work with in the hopes that they’re taking just enough to maintain euvolemia without any of this kind of subtle dehydration that I think can cause adverse effects at the renal level and the electrolyte level etc.

Talked about DIG a little bit already, we know that it does improve symptoms and quality of life in old, old studies of heart failure patients with low ejection fraction. In the laboratory and even in some careful human studies you can show that it increases ejection fraction, exercise tolerance typically improves, but there’s no evidence that it improves progression of heart failure or that it decreases mortality, doesn’t have any effect on ventricular remodeling and especially as patients age,
again I think toxicity issues really start to outweigh benefit. And I really only use DIG if I have a patient who is persistently symptomatic despite optimization of their other therapies.

ACE inhibitors are really the cornerstone of therapies here and I’m going to just kind of combine my comments about ACE inhibitors and ARB’s together in numerable studies done over a couple of decades we’ve been able to show that ACE inhibitors improve both morbidity and mortality in heart failure with low ejection fraction. ARB’s have also been shown to do that. I tend to favor ACE inhibitors because they were the original medicine and they were the cornerstone of all these trials. There’s some very subtle pharmacological arguments to suggest that they might be better. The only reason I ever go with an ARB really is if the patient has a cough that I become convinced is ACE related, that’s the only side effect that I’ll switch over for. Remember that if a patient has trouble with an ACE inhibitor because of hyperkalemia or acute kidney injury an ARB is going do the very same thing to them typically, so I won’t switch for that purpose.

Otherwise quite honestly I think the effectiveness of the two drugs is about equal. I also don’t think there’s much data at all to suggest that one ACE inhibitor is better than another or that one ARB is better than another. It just seems like they have beneficial class effects.

So one of the exciting things that’s happened recently, poor us in the heart failure field, we’ve gone about a decade or more without a positive drug trial, maybe 15 years and you may have heard that this study was just announced at the European heart meetings. This is an agent that goes by the
general name of Neprilysin inhibitor it does not have a trade name as yet, it just has a little letter and number code so I won’t bother you with it. But the idea of this drug is that it inhibits an enzyme that breaks down your natriuretic peptides, you guys know that you elaborate natriuretic peptides to try to maintain cardiovascular homeostasis in heart failure. Natriuretic peptides help you get rid of sodium thus the name, natriuretic peptide. But they’re also vasodilatory and they probably have very beneficial local myocardial effects, but you break them down very rapidly because of Neprilysin and so this agent gets in the way of that breakdown, it increases your endogenous natriuretic peptides, it also increases your endogenous bradykinin which may or may not be a useful vasodilating agent in heart failure. But it also may increase the angiotensin system signaling and so what happens with this drug is that they combine the Neprilysin inhibitor with an angiotensin receptor antagonist to try to offset that part of it. And so they asked a very simple question in this study which was if we compare the old standard Enalapril as cornerstone heart failure therapy to this new drug that we think would both antagonize the renin angiotensin system but also increase endogenous natriuretic peptides and endogenous bradykinin, would we see improvement. And low and behold the improvement was actually quite impressive, very large study, over 8,000 patients and you can see that there was a clear improvement in the cumulative probability of the primary outcome which his a combination of heart failure, hospitalization and mortality. This is especially impressive because remember this is not a placebo controlled trial, this is an active treatment trial so you’re going against the kind of reigning world champion and you come in with a pretty significant improvement over what we were seeing before. This trial hit all the things we look for in a congestive heart failure trial meaning that patients were really well treated, they were on evidence based therapy otherwise,
the follow up was done very carefully, there was a nice, equal distribution of how care was given otherwise, so there’s been a fair amount of excitement over this and we’ll have to see how it plays out on the regulatory side.

Beta blockade now of course is also a cornerstone of heart failure with low ejection fraction treatment. You can see all the different trials on the left here, I won’t bore you with the details. Typically we try to avoid the beta blockers that have not been shown to be beneficial in heart failure, so no Atenolol and no short acting Metoprolol is preferred, and instead we like to see long acting Metoprolol, Carvedilol or Bisoprolol which are the 3 drugs that have been shown to be efficacious. When we’re talking about using ACE inhibitor or ARBs I generally believe that a little dab will do you, in other words it doesn’t appear that there’s much benefit to high dose versus low dose. That’s the opposite with beta blockade. It appears that the higher you can gradually move your patient to in terms of beta blocker dose, the better the outcome will be. So we really try to get patients on low dose ACE and beta blocker to start with and then very gradually go up on the beta blocker to our really maximally tolerated dose.

Spironolactone or Aldosterone antagonists have been shown to be effective in heart failure with low ejection fraction. Many of us were surprised by this, this is the RALES trial which was published now a little over a decade ago. Because we though that if you were on an ACE inhibitor you wouldn’t elaborate much Aldosterone and therefore, you wouldn’t need to antagonize it but of course the body is a stubborn organism and it has other ways to elaborate Aldosterone and
Aldosterone is a bad actor in heart failure. It contributes to vascular and myocardial fibrosis and heart failure progression and you can see that using Spironolactone whether it’s in an advanced heart failure patient like this, or in more recent studies in a more moderate heart failure population really is associated with improvements in both mortality and morbidity.

And then we get to device therapy with low ejection fraction remember virtually any patient who wants the therapy should have a defibrillator it’s been shown pretty clearly that once your ejection fraction is persistently below 35%, so long as you are not Class 4 and going to die from pump dysfunction in the near future, we recommend defibrillators. And also remember that if you meet certain electrocardiographic criteria namely if you have a left bundle branch block in addition to an ejection fraction less than 35% and you have Class 2-3 heart failure symptoms, a CRT device or a bi-ventricular ICD is the preferred device. The CRT itself even in the absence of the defibrillator has been associated now with improved mortality, so pretty typically we would get those patients a combination CRT defibrillator.

So that’s all the stuff that we write on prescription pads and that we spend a lot of money putting in but in fact if you’re going to be successful at managing patients with congestive heart failure, a lot of it comes down to what’s on this slide. Like any chronic disease there has to be a lot of attention paid to things such as what the patient’s goals are, you know what they’re trying to achieve with their treatment, where are they, how much functional ability are they really looking to get back to, what’s the home environment, what hurdles might be present in the home environment that are going to get
in the way of successful therapy, how their compliance with treatment is, how their compliance with lifestyle adjustments are. We spend a lot of time on education of these patients, I have an amazing nursing staff that really does the bulk of our education for our patients, which is an ongoing process. It’s not just a one time thing. And all of these things have been linked to readmission rates. So if you are interested in readmission rates and if you aren’t today you certainly will be in the future because that’s going to be linked to all of our pocketbooks not to mention that patients just do better if they’re not getting into the hospital over and over. These are things that we’re really going to have to make a push for.

Other things that I think are important and often overlooked is depression is incredibly common in congestive heart failure. Some studies suggest maybe 60%. Bruce Roman does amazing research on this topic and has shown not only the prevalence but also how much better patients can do when it’s treated well. Sleep disorders are very common in this. Some of you may know I’m married to a sleep doctor so I hear about this all the time whether I want to or not, so we do very aggressive screening for sleep disorders, and then sexual dysfunction which plays into overall quality of life obviously.

Now I’ve been harping on this kind of readmissions and these metrics we’re going to be held to and I want to just show some stuff that’s going on at UPMC that I think is very exciting. One of the things that we get held to in terms of a standard is trying to have as low a length of stay as we can. It’s really challenging in heart failure as it is in many chronic conditions because there’s probably
something of a reciprocal relationship between length of stay and readmission rates and we’re really going to be held to high standards for both of those things. If I rush a heart failure patient out of the hospital before they’re ready to go it doesn’t take a rocket scientist to know that they have a higher chance of coming back into the hospital within a short window. So we’re trying to find ways that we can shorten length of stay by seeing where we’re inefficient right now, not so much by short change a patient’s treatment, but to understand where we can improve efficiency. And one of the ways that we can do this are through these pathways. These are being developed jointly through Robby Romani on the heart failure side in my group spends a lot of his time on this. He works with _____ and others in the technology development center and they’re trying to come up with these very streamlined, very user friendly and still flexible, because nobody wants to take away physician autonomy, pathways that we are hoping are going to make a positive impact on quality of inpatient care as well as length of stay. And you can see that you basically just move through a patients admission, you start with their initial admission features and then you go through each of the days of their stay. There’s prompts, there’s reminders, there’s ability to link to evidence etc., in an effort to try to improve our overall systemwide approach to congestive heart failure.

Another thing that’s pretty exciting and now we kind of switch and get into the question of how we’re going to decrease readmission rates, is remote monitoring technologies and some of these have been around for awhile, and some of them are really brand new. But even simple symptoms have, I’m sorry simple systems have been shown to improve our ability to keep folks out of the hospital. So simple scales that give reminders about you know what medications and when to take them,
maybe some of them ask simple questions about symptoms etc., sometimes these are combined by blood pressure measurements. These give direct feedback to patients, they also go to usually a nurse or another care provider that can give direct feedback to patients, or all the way to the physician who can then give the feedback to the patients in an effort obviously to catch perturbations in the patient’s course early so that we can make interventions and keep them out of the hospital.

This is very cool technology here. This is a cardioMEMS device, this is a very simple device that can be dropped into the pulmonary artery it just has a coil of wire in there, there’s no batteries in this. The whole thing is only about 4cm. long by a cm. wide, blood flow pushes it so that it lodges in the pulmonary artery and then at home the patient can lay on this pillow and this device here will send a signal to that device, it is reflected back and the frequency change that comes from the reflection translates directly into a pulmonary artery pressure tracing. You can see the device here, it’s very tiny, you can see it going inside to here and this is the kind of data I get on my desktop looking at a patient’s pulmonary artery pressure over time as I am adjusting their heart failure medications. This is now approved by the FDA to decrease hospitalizations in heart failure and we’re just starting to roll it out at UPMC. It’s very cool stuff. I’ll skip that slide.

So now we get into the end stage stuff. Transplant is still very valuable, people do very well with it, but here’s the problem we are absolutely capped in terms of numbers of organs available in our country and you can see that we’ve been capped now for 20 years. We have not made one dent in trying to increase donor supply. We do approximately 2,200 heart transplants a year in the U.S.
That’s why many people will say that transplant is to heart failure as the lottery is to poverty. Very, very few people unfortunately are going to be able to take advantage of this technology because there just is not adequate availability. So we’ve been looking at replacement strategies. This is our original heart pump, or left ventricular assist device, or mechanical circulatory support. You can use your phrase of choice. You can see how big and bulky it was, it was really a fairly brutal kind of medieval device but through lots of bioengineering advances we’ve gotten smaller devices and now even smaller devices. This is the typical pump that we implant now. You can see how tiny it is, it works on a centripetal system instead of a pusher plate. The funny thing about that is that once a patient has one of these and goes home and comes back to the office they are pulseless, so it’s kind of odd, all you get is a mean pressure by Doppler you don’t actually get a blood pressure. You can see how this sits in the heart here this is its mechanism inside. The outcomes with this device are actually getting to be incredibly good. You can see now 3 year outcomes with 70% survivorship which is really astounding given that you’re talking about a 50% one year mortality in this population. These are how we, the list of how we use this device. Some folks will really crash and burn at the time of an open surgery, an open cardiac surgery and we’ll put one of these in as a temporizing agent to see if their heart recovers. Commonly we’ll use it as a bridge to a bridge or a bridge to a decision. The idea that if a patient might be a transplant candidate but we don’t know yet if they’re going to be we can put this in and then we can figure out if they’re going to be a good transplant candidate. We often use them as bridge to transplant. I might have a guy waiting on the list and he’s starting to go down hill and he’s not going to make it until he gets a heart, I can put one of these in, he can rehab, he can get healthy again and then he can get a heart with less of a time
constraint. Or now increasingly we use them as, what’s kind of ominously called destination therapy. That means that this is your treatment, this is the treatment for your heart failure and it’s what you get for the rest of your life. And we now have survivors who are out 5 years at home playing golf, riding horses, doing stuff like that with a pump like this in. It’s obviously not for everybody but we’ve implanted some folks well into their seventies who’ve done really well and I think it really is where the future of advanced heart failure care is going.

Then I’ll just conclude by saying remember older folks are the ones at the highest risk for this, there’s lots of risk factors so keep an eye out for them and try to be aggressive in manipulating them. The standard therapies work real well but it still a very morbid and mortal condition. The higher order therapies can be considered in virtually any patient and we would encourage you guys to get us involved if you think you have a patient who might benefit and I think where we’re going to go in the future is really going to come from better monitoring, better disease management strategies. Thanks.