[MUSIC PLAYING]

RAVI RAMANI: Readmissions has been something on the radar for a number of years. I'm sorry to say, we're not much closer to solving it. But I have some ideas that I'd like to share with you about the problem and how to approach it at least. So without further ado, I just want to describe what we see at UPMC first.

So this is our landscape for admissions for heart failure. So these are all patients who have been seen by a UPMC cardiologists over 2014. And this is about 27,000 patients that have been seen. And often, you can see, most of them don't actually get admitted. But a significant proportion do. And here, the number is about 1,600 patients who get admitted for more than five times in that past year.

There's actually one patient who's been admitted more than 90 times-- 9 0 times. I think a lot of people in this room know who that person is. But it just gives you the scope of the problem. Of those about 6,000 patients account for about 40% of all the admissions in heart failure. So it's a focused problem. It's not all patients with heart failure. But those who do get it, are sick and need our attention.

So what are some of the scenarios that we've seen in patients getting admitted. Well, there are a bunch of different things here. So the classic one is a patient discharge after heart failure admission. The Lasix was decreased and ace held, because of creatinine increase of 1.1 to 1.5. Presented again with heart failure.

Second one is, the patient goes home. Remote monitoring says rate was gaining. There was nobody to get hold of. Patient gets admitted. Third is a non-compliant patient with multiple psychosocial issues. Refused for the treatment-- gets admitted. Next, is hospital-acquired conditions. And then, finally, over diuresis. So all of these just gives you a flavor of some of the problems that patients who are getting admitted with heart failure face.

And therein lies the challenge. It's a messy problem. It's not a clean thing. It's a messy problem. It's got all kinds of issues in it. So what are some of these issues? There are patient issues. There are physician issues. There are factors beyond anybody's control. And there are system issues that are in place to give a patient back into admission.

So let's talk about some of them going forward. There are some disease factors. OK, so one of them is the stage of disease, a stage of heart failure. So this is a very classic diagram of how heart failure progresses. As you can see, in the top graph, there's an initial drop in quality of life. That's usually the presenting admission. You fix that. The patient goes home.

There's a variable period of stability. And after that, heart failure, like many other chronic diseases, being a remitting disease-- exacerbations and intermissions-- essentially, decompensations all the way towards the end of life. And if you look at the bottom graph below that, there are readmission rates. And you can see, there's an initial cluster right around when they present and before treatments are optimized. Then, again, there's a variable period of stability.

And then, right before they die or towards the end of stage, that's when they really have an increase in admissions. We all know this. More than 80% of the cost of health care will be incurred in the last year of life. So that's really a critical point. Where are they in their disease stage?

In addition to that, we've all had patients-- and this has particularly true of diastolic heart failure patients who are very volume-sensitive here or there. So they obviously need much more observations. On top of that, all the other diseases that tend to travel with heart failure-- renal function, renal failure, anemia, ischemia, COPD, hypertension-- all of these are playing contributing roles to the disease state and the risk of readmissions.

There are several tools available to risk score your patients. This is from the Yale University. There's an app available for this. You can just go online and score your patients. They're mostly focused on heart failure or related medical conditions. So they're very disease-focused. Most of them are not really focused on the other patient-driven factors that are equally important, in my opinion, such as social issues, how frail they are, where they're treated and all that, which we've all come to.

All of these are clear but really unquantified benefits-- really don't link themselves to scoring. But this, I find, is a very useful score to see where your patient's at risk for readmission. There's the Yale score. There's a couple of others available on the internet.

There's several patient factors, obviously. We all know this-- medication and diet compliance-- that's a no brainer-- how rich or poor they are, how much insurance they have, how engaged they are in their own care. So this really has consequences. So the graphs on the other side show, really, that this is what we're dealing with. And what this shows here is that, in general, the poorer the patients the hospital is treating, the higher the risk of readmission.

Which is a problem, because some of our most needy, most difficult patients, are really facing a problem of readmissions more than some of our more healthier and more up-to-date patients. BAC factors-- this is Beyond Anybody's Control factors. There's a significant seasonal variation. There's a regional variation. There's obviously race, gender, and age issues.

How do we know this? Well, these are some of the seasonal variations in heart failure. You all know this. Wintertime, flu season-- that's when they're going to come in. Summertime-- well, they may come in after 4th of July binges or after Thanksgiving. But generally, they tend to stay out of hospital. And notice that the mortality and the hospitalizations, though, tend to follow a similar curve here.

Regional variations-- this is significant also. Where you live determines-- your zip code does determine, to some degree, your outcomes. We know this. Within the UPMC system, the average readmission rate is about 21%-- 30 days. But there's a range from about 15% to 34%, depending on which hospital.

I will not name which ones are good or bad. But one can have a general idea which ones they might be, just based on the demographics of the patients' population that they serve. So there's a significant variation in care and outcomes, depending on where you live.

Physician factors-- the biggest one is inadequate diuresis while in-house. The second biggest one is incorrect post-discharge diuretics, poor hand-off, inappropriate or duplicate medications, inability to handle exacerbation in a timely manner, and lack of consultation. That's not a plug. OK, it is a plug for referring to cardiology. But outcomes have shown that, if a patient with heart failure has been treated by a cardiologist, outcomes are better. But there's a whole bunch of other things in there.

So there's a lot of things in there. And personally, I'm overwhelmed.

[LAUGHTER]

So what do you do? What do we, as physicians, APPs, nurses-- what do we do? What can we actually control? And I think, the one thing, above all, that we can control is the hospital course. And that's something I really want to spend some time on.

So why do heart failure patients get admitted? It's volume overload. You It's signs and symptoms of volume overload-- dyspnea, edema, et cetera, et cetera. There's a bunch of other diseases that may be involved. But it's volume overload that they get admitted with.

How do we treat acute heart failure?

[DING]

[LAUGHTER]

Well how do we actually treat heart failure? It's not so great, OK? At the time of discharge, 50% of patients lose less than 5 pounds. 20% lose no weight at all. And 5% actually gain weight. This is from the ADHERE registry. 26,000 patients in that study nationwide. And this is fairly consistent data.

Now, there are, of course, caveats with getting weights on the floors and all that. But this is the data as it stands. And it really does reflect reality. In fact, it reflects reality at UPMC. We studied 1,100 patients for the same amount of data-- and very, very similar results. On top of that, once we sent patients home, a lot of them waited more than 10 days to see a provider. We know that if the patient is seen early after discharge, the risk of re-admission is lower. The rates of delayed visits are high in minorities.

And finally, we claim 100% compliance with discharge instructions. Does the patients really get it? So these are really factors I really want to control. And above all, is the diuresis part of it. So I'm going to really spend the next few slides just talking about some diuretic management. Because I think that's one factor we really need to control.

So how do we manage heart failure overall? Mr. Spock-- god rest his soul-- said, live long. And maybe, breathe well. So I'm going to talk about that a little bit. In general, we make people live longer through reducing progression of disease. And you do so through neurohormonal interventions and through the prevention of complications. I'm not going to spend a lot of time on this, because the emphasis will be on making people feel better.

And that, we do so through hemodynamic interventions. And we do so, mainly, through diuretics and positive inotropic agents. So how do you make people feel better? We try to fix the pump and we make them pee. So let's delve into that a little bit deeper.

To understand that, let's try and understand what heart failure is. And I like this definition of heart failure. It is-- wherein it's a condition in which the heart either cannot maintain adequate systemic perfusion or can do so only at the expense of increased filling pressures. OK, so there's two components to this. There is systemic perfusion-- that is cardiac output-- and increased filling pressures-- that is, essentially, volume. Pressure's reflected by volume, filling up the heart, your lungs, and causing edema all over the place.

So let's try and dig into that a little bit deeper. So how do we make people breathe better? Again, through hemodynamic interventions. We make people pee through diuretics or dialysis, as the case might be, and through interventions that make the heart perform better-- digoxin, other positive inotropic agents and, in really advanced cases, mechanical interventions, such as transplant and ICDs and all that.

So let's dig into that a little bit deeper. This is a Frank-Starling curve. And this really reflects how the cardiac function behaves when the heart is stretched from increased volume. So as the heart is stretched-- because you're filling it with water-- the actin and myosin fibers align better. And the cardiac output increases. So as you go up on the scale at the bottom, when it says left ventricular diastolic pressure for the normal left ventricle, for the same increase in pressure that is volume, the cardiac output increases.

At the bottom of the red line is a sick heart. And you can see there, for the same increase in the left ventricle end diastolic pressure, which is the volume inside the heart, you get less bang for the buck. So you're straining the heart by stretching it out. But it's not really able to squeeze much more. Let's look at this a little bit more.

So as you increase the left ventricular end diastolic pressure-- that is the left pressure. The pressure back-fills into the left atrium and then into the lungs. The lungs leak out fluid, become stiff and non-compliant, causing shortness of breath. As you increase the pressure, there's more of a tendency in the lungs to leak that fluid, causing pulmonary edema.

And typically-- and I'm going to draw a line in the sand of saying, 18 millimeters of mercury-- above that is going to cause some degree of pulmonary edema. And that number is just a line in the sand. It can be 12 in some people. It can be 20 in some of the people who are chronically compensated.

Now, let's go to the vertical axis here, in which this cardiac index plotted. And here, you can see that, when there's a normal cardiac index, there are no symptoms. But as you drop the cardiac index, perfusion to the brain and other organs decrease. You're going to start to feel tired, fatigued, may have mental status changes, including depression.

So there's cardiac output, which is going to cause decreased perfusion, and there's left ventricular filling pressure that is volume that's going to cause shortness of breath. So fatigue and shortness of breath-- these are two components. But as you decrease your cardiac output or index, you can start to get symptoms. And I'm going to draw another line in the sand saying that below a cardiac index of 2.2 liters per minute per meter squared, you're going to start getting symptoms.

Again, there's a line in the sand. Some people are going to get it higher. Some people are going to get it lower. But by drawing these lines in the sand, that allows us to characterize the patient hemodynamically. And these are called Forrester hemodynamic subsets. And it goes through 1 through 4. You don't need to remember what those are. But these are important, conceptually, in trying to place where your patient is.

So this is what it looks like, 1 through 4 again. And 1 is where the cardiac output is OK and the filling pressure, as the volume status, is OK. And 4, on the other hand, is somewhere where there's a lot of volume and a low cardiac output. And this is what we've all heard of, but I'm going to reiterate here.

The class 1 would be somebody who has a normal cardiac output and a normal filling. Pressure so they're warm-- feel nice and warm. And they're dry. They have no excess of water. Class 2 is somebody with a normal cardiac output, but high filling pressures, high volume. So this is somebody who is warm and wet.

Class 3 has a low cardiac output, but a normal volume status. And they're cold and dry. And cold and wet, where everything is wrong with them. So when I see a patient in clinic or on rounds, I'm mentally classifying them into one of these groups.

So how do you mentally classify them without having all the information? We do so through clinical examination. And how do we do that? Well, the warm and dry patient really will have a blood pressure that's at their baseline. They won't have much of a JVP.

The lungs would be clear. There will be no edema or minimal edema. Kidney function will be OK. And if they happen to be walking around with a Swan-Ganz catheter in them, you'll find that they have a normal cardiac output or normal filling pressures.

The warm and wet is your classic heart failure admission. So this is standard floor admission. Blood pressure will be normal, close to the baseline, or high. They will have JVP, likely. They will have crackles, lots of edema, or ascites. Kidney function will be at the baseline. And cardiac output will be normal. And filling pressures will be high.

The cold and dry patient, on the other hand, will complain mostly of fatigue, maybe depression. Their blood pressure will be on the low side. They may have some JVP. They tend to be more chronic-- minimal or no crackles, no edema, increased creatinine, and a low cardiac output.

The cold and wet patient, on the other hand, will have low blood pressure and full of water, really short of breath, and having mental status changes and increased creatinine. So once you've seen the patient, you mentally plug them into one of these four quadrants. And why do you do that? Because that tells you how to treat the patient.

So that goes to the next step. So the warm and dry patient-- really, good job. Continue doing what you're doing. The warm and wet patient-- really, you need to make him pee. That's the primary hemodynamic intervention.

The cold and dry patient-- you may need to adjust medical therapy. This may be the patient who is overdiuresed or is over-beta-blocked or something like that. You may need to back off on it. Or you may need to consider advanced therapies if there's nothing else to fix in them.

The cold and wet patient, on the other hand, needs, really, the kitchen sink thrown at them. They may need inetropes. Balloon pumps, VAT, transplant, palliative care and, yes, of course, heart failure referral. So-- yes. But again, when I see the patient, I'm mentally classifying them into one of these four quadrants, because that really determines how to treat the patient next.

So how do we really treat the patients? Diuretics-- very standard. Loop diuretics form the cornerstone of therapy. On top of that, we have the thiazides as synergistic agents for diuresis. The potassium sparing agents are not really used as diuretics. They're more for chronic heart failure management. But the loop diuretics really form the cornerstone of therapy in these patients.

So this is what everybody is scared of when patients come in. One of us goes out and drinks a 6-pack of beer, has hot dogs and nachos and everything. We're not going to go into heart failure. Conversely, we go run a marathon in 100-degrees-- most of us are not going to go into renal failure.

Heart failure patients have a lot less reserve. We all know this. So a little bit of fluid here or there really can tip them one way or the other. And over-diuresis is something-- really-- what people are concerned about. So something like this. I spend a lot of time on. That please laugh.

[LAUGHTER]

But that concern really is the root of inadequate diuresis. And this is something we see as a consistent pattern, this inadequate diuresis. So one reason or the other-- and we'll get into that a second. That really forms a core of two problems. One is the increased length of stay. And the second is the readmission rate.

So what does the ACCHA say? Basically, admit and treat promptly with IV diuretics-- number one. If they're already on loop diuretic therapy, change the same dose to IV or greater. So then, 40-milligrams PO of Lasix a day-- change it to at least 40 milligrams IV of Lasix. And this, you can give as continuous-- intermittent-- doesn't matter. But it should be at least that dose of diuretics as they are getting at home.

If that doesn't work, then go up on the dose or add a second-- that is a thiazide diuretic as a synergistic agent. So-- so far, so good. Most people would agree with this. The problem is the issues that we've seen that happen.

So what we've really seen is two problems that happen with inpatient diuresis. One is that the creatinine goes from 1.1 to 1.4. Lasix gets held. The patient's already feeling a little bit better. So they get started back on their home dose or less. The patient goes home and then gets readmitted with heart failure. That's a very standard admission.

The second is the blood pressure hold parameter is not at UPMC. The standard blood pressure hold parameter is 100 millimeters of mercury. So one blood pressure of 95, when the baseline is about 110, 120-- the nurse holds the diuretic. We, as rounding physicians, don't find out about it till the next morning. That's a shift lost. And if you're concerned about it, you may hold the directs as well.

So what we did was to come up with a consensus statement from the heart failure group. And some of this is reflected here. So we said, first of all, in agreement with the ACCHA, is to start at least the home doses in IV. Then, the second was, if there's insufficient diuresis that is less than a liter a day for most patients, increase the dose by at least 1/3. So if you have 40 mitigants BID of Lasix, go to 40 TID. You can go to 80 BID, if you feel like it But 40 BID is OK.

The third is somewhat controversial. We said, a blood pressure of 90 millimeters of mercury is acceptable in most patients. Exceptions, obviously, If there's significant drop or the patient is symptomatic. And by symptomatic, I don't mean that they get occasionally lightheaded when they get up too quickly or something. That's acceptable. That is OK. And in fact, that's where you want their blood pressure to be.

But in general, a very conservative blood pressure hold parameter that's 100 or more is way too conservative. Too many Lasix doses get hold. The next one is a creatinine increase. After much discussion with the Renal Department and ourselves, we agreed on a 50% increase in creatinine that is acceptable in most circumstances.

These are not national guidelines. There is little data to back this up. It is hard to get data on this. But a creatinine increase from 1.1 to 1.4, for example, is not sufficient for people to hold diuretics. You should really continue diuresis through that. And most patients will make it through that with stable creatinines.

And the other thing I didn't put down here is-- I've noticed-- epidemic of Lasix or diuresis holes among the house staff, because the potassium is 3.1. So Lasix is being held for potassium hypokalemia. There's no reason to hold diuretics for hypokalemia. The treatment for that is replacing potassium, not holding the diuresis.

The last thing here is exam. Unfortunately, despite all the work, there is really no good noninvasive way to assess volume without actually doing a good physical exam. And even that is insensitive, even for heart failure specialists. But it is what we have. So what we suggest, really, is not relying just on the lung exam.

The lung exam, especially in chronic heart failure patients, may be completely normal in your heart failure patients, despite having elevated reges. So one needs to use the JVP, any distention in the abdomen, peripheral edema. And yes, you can use the BNP, particularly a declining BNP, as a guide to therapy. The lung exam and chest x-rays are less sensitive. And I urge you not to rely just on that.

So the concerns, again, with over-diuresis are if the patient's creatinine does rise. And one thing we really need to make sure is, where's the patient? So this is a daily assessment, not just the first time where the patient is-- which quadrant do they fall in?

Initially, most of them will present in warm and wet. The creatinine rises and they still have volume-- they're not cold and dry. That is not over-diuresis. They have probably moved from warm and wet to cold and wet. So that is something, really, what one needs to pay attention to. So if they still have volume, creatinine is rising and the blood pressure has fallen, again, they're more likely to be cold and wet.

Of course, you got to think of other things, like the CAT scan with contrast they got in the ER for rule out PE, when they first presented. That can be causing the real function. So of course, rule all that out. But again, the volume status is the key determinant of how you treat the patient.

So if they do more from warm and wet to cold and wet, then you need to up your game. And this really is where inotropes come in. So again, some Frank-Sterling curves here. As you can see, as the filling pressure left ventricular end diastolic pressure increases, the cardiac output increases in the normal green-line. Whereas, with the sick heart, for the same amount of pressure, same amount of volume, you get less bang for the buck.

So what inotropes do here is change the line from here to here. So for the same amount of volume, you're whipping the heart into beating stronger. So again, in the patient you think has moved from warm and wet to cold and wet, that's where inotropes will come in. And obviously, this will be somewhere where you'll be considering advanced cardiology consult as well.

So when do we use inotrope? They are essentially palliation of bridge agents. Because none of them improve mortality. They may, in fact, increased mortality-- you need both signs of severely decreased cardiac output, which can be hypertension, increase [INAUDIBLE], mental status changes, and signs of refractive volume overload, such as lung congestion, JVP, and peripheral edema.

The classes of agents-- the most common one we use is dobutamine. And the other one is milrinone-- essentially interchangeable, despite what the druggists might say, in terms of side effect profile and all that. A lot of side effects are possible. It can cause hypotention through peripheral vasodilatation action. And every known arrhythmia known to mankind can be caused by inotropes.

All of them can increase ischemia, because there's no free-ride. You're whipping the heart into beating stronger. And all of them do increased mortality. So again, these are bridge agents or palliation agents to get them out of the hospital, make them feel better.

So in-patient therapy-- the principles, again, are we want to treat the hemodynamic issue first. So when they come in with heart failure, I wouldn't worry so much about adjusting the beta blocker doses or something else or beginning of the ACE inhibitors as a first line. The first line is really to get them dry. And this is really diurese. And aim for one liter or greater per day.

You must treat to completeness. And that is as close to euvolemia as possible. We know, from other studies, that 40% of patients are discharged too early. And this is across the board. This is academic centers as well as community hospitals. Increase in creatinine of 50% is OK in most circumstances. Systolic blood pressure-- 90 is acceptable, unless it's a drop or the patient's symptomatic.

And make it a point to address the exacerbating factors. And this could include social work consults, dieticians, and other heart failure education that may be available to your hospitals. And if you think the patient's advanced disease, do consider palliative care. Because a lot of them will fall into that where, really, this end-of-life management is warranted.

And that cycle of repeat admissions-- no one wants to die in the ICU with six tubes sticking out of them. And this is what happens to a lot of these patients. So discharge criteria-- when is a patient ready for discharge? Optimal volume status achieved-- so this, again, is as close to euvolimia as possible, not just that the patient's feeling better.

The patient needs to be stable on what medicines they are going to go home on. So what this really means is that 24-hour stability on PO medicines, PO diuretics, and stable volume-- that is, once you switch them from IV to PO, they're not getting fluid back-- the blood pressure's stable, and the renal function is stable. If all three are met on PO, then they're medically ready for discharge.

Their education is complete. There's standard precheck orders. Please, get heart failure education on your patients. It's a pretty good service in most hospitals. The exacerbating factors are addressed. And again, consider social factors when thinking about this.

And there's a post-discharge plan in place. It is most likely not sufficient just to tell them to call their PCPs upon discharge. That almost never happens. Ideally, the appointment should be made before the patient is discharged. So how does the transition work? There are several key concepts. These are based on consistent statements from the ACC and the Heart Failure Society of America.

Questions to ask are-- are they familiar with their medications? Do they have access to their medications? Do they understand what kind of symptoms are problematic and whom to call? And do they have a follow-up visit scheduled? And are they able to get to that visit? So some simple straightforward things. It will take some time, but your floor managers or yourself can definitely take care of this.

Does this work? Yes, this is from the *New England Journal* about 10 years ago now. And this is intensive care management. And you can see that readmission rates are decreased with intensive care management. And these are education, medication review, and a follow-up visit, including a social service consultation. So it does work.

The problem is that it requires a lot of manpower. It's really labor intensive. And most centers cannot really duplicate these other studies. So we at UPMC have taken a slightly different approach. What we've tried to do is to virtualize this using remote monitoring. So pretty much any patient being discharged from UPMC hospital with a diagnosis of heart failure will be eligible for remote monitoring, regardless of pay, free of cost to them, for a period of 90 days.

So what is remote monitoring? It's, basically, the patient at home. Again, I'm sorry. My letters don't seem to have come up. But getting the biometrics and a symptom checked done-- a lot of cases, with self-education, they can manage it themselves.

If that doesn't work, it gets bumped up to a call center, which is run by our UPMC Jefferson Home Care Agency. So it's RN run. They can take orders. And they can manage a lot of things on their own. And if that doesn't work, it goes to this inappropriately happy person, who's a physician, who can then manage the case accordingly.

So this is something we've been doing for a number of years. We've had some success with it. But this is what the overall data shows. The data is mixed in that, this is from the Cochrane database. It's a meta analysis. So you can see this bottom diamond at the bottom, showing a trend towards benefit.

And I think a lot of the problem is that it's very focused on symptoms. What we have tried to do at UPMC is build, really, care management around remote monitoring. And with that, we have had-- I'm sorry, again-- my numbers-- but a drop in readmissions from about 21% to 14% over this two-year period. So this is really something we're pushing for. And I encourage you to use it for your patients.

So in summary, it's necessary to view the hospitalization as part of the patient's overall situation. The patient doesn't stop existing once they're discharged. So it's really important to remember that. And that has implications and sign-offs and post-acute transitions.

Mentally, plays the patient into low, medium, high risk categories, depending on your overall Gestalt feeling. You can use a risk score if you feel like it. Treat to completeness while the patient is in-house. Discharge the patient when discharge criteria are met. Have a transition plan in place prior to discharge. And use available resources, particularly remote monitoring, post-discharge. And thank you.

[APPLAUSE]