

Pediatric Heart Failure - Pediatric Grand Rounds-3-28-2025-Meeting Recording

March 28, 2025, 12:29PM

1h 1m 33s

● **Kamat, Deepak M** started transcription



Kamat, Deepak M 0:21

May I want to start?



Bansal, Neha 0:23

Yeah, I'm OK.



Kamat, Deepak M 0:24

You ready?

OK.

Good morning and welcome to pediatric grand rounds.

As always, the CME code is in the chat box, and we'll keep repeating it every few minutes. We tend to 15 minutes.

Also reminder, please complete the variations at the end of the grand rounds, which daily Asse.

Helps me write.

Provide feedback to the grand round speakers after their grand rounds.

So it's my great pleasure to introduce this morning's.

Then around speaker doctor Neha Banson, who was my resident and a fellow some time ago, she's attending physician in the Department of Pediatrics at Mount Sinai, Travis Children's Hospital and associate professor of Pediatrics at ICAN School of Medicine at Mount Sinai.

Her clinical focus is pediatric patients who have heart failure due to cardiomyopathy or congenital heart disease as well as pediatric transplant patients.

She also has a keen interest in cardiac care of survivors of childhood cancer. Doctor Bansal received her MBBS degree from a Premier institute in India, Mumbai, known as GS Medical College km Hospital and then from 2012 to 2018 she was the children's Hosp.



Lamour, Jacqueline 1:25

It's.



Kamat, Deepak M 1:39

In Michigan, where she did her pediatric residency as well as fellowship in pediatric cardiology in 2017, Doctor Bansal received her.

Children's Cardiomyopathy Foundation and Kyle John.

The Miski Foundation Research Scholar award doctor Bansal's research focuses on pediatric cardi, MR50, cardiac transplants and pediatric cardio oncology. The work has been published in numerous Pedi journals, books, manuscripts and articles.

She's also host a podcast called in in the Flow Conversations with pioneers in pediatric heart failure and transplantation.

Available for free on all platforms enlisted.

Throughout the world.

Doctor Bansal, thank you very much for accepting our invitation and we're looking forward to your presentation.

The floor is yours.



Bansal, Neha 2:29

Thank you so much, Doctor Kamat.

Thank you so much for the kind invite and drive. Kamat has been a lifelong mentor for me and since my residency days, so I really appreciate this opportunity to be able to present in front of him.

So as he mentioned, my passion is pediatric heart failure.

I do delve into pediatric heart transplant that sort of comes with the territory, but I think pediatric heart failure, it's a very exciting time and pediatric heart failure and so I.

I wanted to just.

Present sort of an overview of what I do on a day-to-day basis.

I have no disclosures and so just starting from the basics.

So cardiac muscle, which is also known as the myocardium, is a specialized type of muscle tissue that forms the walls of the heart.

It is responsible for the rhythmic contractions that pump blood throughout the body.

Cardiac muscle cells that are called the cardiomyocytes are cylindrical and exhibit

striations that are very similar to skeletal muscle due to the arrangements.

The contractile proteins that you must have heard actin and myosin.

So the functional unit of this cardiomyocyte contraction is the sarcoma that consists of the thick filament called the myosin, and the thin filament called the actin, and the interaction between them forms the basis of the sliding filament theory and you can see here in the cartoon.

That the actin myosin kind of form cross bridges and that's how the contraction occurs. And they're very calcium dependent and so.

So the cardiac muscle, you know, the contractile system depends on these cross bridges keep forming as long as the calcium is attached to troponin and it is, it forms the basis of the heart muscle and so.

I don't know if I can't see the chat, but you know cardiac output as we think of cardiac Physiology is the amount of blood that is ejected by the heart per unit time. Most commonly in liters per minute and the simplest and most practical equation describing the card.

Output is.

This was further residents, you know, as we think of cardiac output, we think of stroke volume times the heart rate.

So it's an easy way to understand.

You know, when we think of heart failure.

We see a decrease in stroke volume and as a compensation we see an increase in our heart rate to maintain that cardiac output. And then when both of them are not able to keep up, we see a decrease in our cardiac output.

Going back to our, you know, Physiology days, you all must have heard of the Frank Starling curve or the Frank Starling's law.

So on the Y axis is the cardiac output or the stroke volume, whichever one you want to interchange that with. And then on the X axis is the preload.

Or or the OR the end diastolic volume.

In heart failure. So. So if you look at the curves here.

The curve A represents the baseline curve and you can see that the contractility kind of has a very nice.

Over here you can see it has a very nice curve.

So generally we like to keep it over here.

This is sort of the optimal place for cardiac output, so as you increase the preload, your cardiac output increases beyond a certain point in time.

Obviously if your volume keeps increasing, the heart won't be able to keep up with that cardiac output, so.

So this is sort of like the optimal place where we like to keep our pre load and that helps with our cardiac output and in heart failure we see that this relationship is altered.

This Frank Starling, Kerr's kind of shifts downwards and to the right.

So over here you see the the curve C. This shift indicates that for any given volume or any given, preload the stroke volume is reduced as compared to the healthy heart. And then of course as it keeps getting worse, the curve D over here shows a failing vent.

And you can kind of think about it as we give it more inotropy, we shift the curves back as much as we can towards normal and you know curve B sort of represents somebody who's in an exercise Physiology.

You know you have increased inotropy in those patients, so heart failure and very, very simple terms is deterioration of global cardiovascular performance and it incorporates systolic function, diastolic function, systemic and pulmonary vascular. It's the inability of the heart to keep up with the body's demands.

When we think of how we classify heart failure, you know and and we we think of like what?

What is going on in the body and during heart failure when you have the reduced efficiency of the myocardium and the myocardium results in being very overloaded, and this this increased workload on the myocardium will produce some changes in the heart causes reduced contractility due to the OR.

The force of contraction due to overloading of the ventricle like we just saw in the Frank Starling curve.

A reduced stroke volume as a result of systolic failure, or diastolic failure, or both.

And again, like we saw in the Frank Starling curve, we saw that you know, as as your volume keeps increasing and heart failure, your stroke volume doesn't really increase. You have increased heart rate.

So remember that equation.

Cardiac output stroke, stroke. Volume times a heart rate.

So to compensate, when your stroke volume goes down, your body increases your heart rate.

And it is stimulated by increased sympathetic activity in order to maintain cardiac output.

Over a period of time you see some hypertrophy of the myocardium caused by the terminally differentiated heart muscle fibers increasing in size. In an attempt to improve contractility.

So you see some some trabeculations you see the overall mass of the heart being being larger as as the muscle kind of grows and 10s out?

Enlargement of the ventricles, which contributes.

To the spherical shape of this of this failing heart.

And all.

All of this is what you see over a period of time in in, in classic reduced ejection fraction, heart failure.

So again, decreased stroke volume decreased, you know.

That the body compensates by increasing the afterload, it tries to increase the preload and that's why you have increase in your end diastolic volume of the heart and then the body also tries to increase the heart.

Rate and all of this is an attempt to.

Increase the cardiac output of the body so the way we we try to quote UN quote treat heart failure is by reversing some of these things.

So we increase contractility of the heart.

By decreasing we help the heart by decreasing the afterload if the heart is super overloaded, we decrease the preload by giving some Lasix and then we also decrease the heart rate to improve the fillings of the heart.

Part is beating really, really fast at the rate of like 140 beats per minute. It doesn't have time to relax.

It doesn't have time to fill and so decreasing that rate kind of helps the heart fill actually a little bit.

As counter intuitive as it may sound, by decreasing the heart, radio may decrease the cardiac output, but the patients don't really feel very good when their when their hearts are beating at the rate of 150 beats per minute. And so in terms of classification or how we.

Think of categorizing heart failure.

It depends on which side of the.

Part is involved left versus right, whether contraction is affected or relaxation is affected.

The systolic function versus diastolic function.

What is a functional impairment of the patient?

So it's it's fine.

One thing, if you see echocardiographically that the the patient has dysfunction, but what is happening to the patient?

So there is functional classification called the New York Heart Association classification. And for Pediatrics we have the Ross classification.

Whether the problem is primarily increased venous back pressure, so behind the heart or adequate arterial perfusion or or front of the heart.

So backward failure.

So everything is backing up.

So you have hepatic congestion, pulmonary edema or is this forward failure, so you don't have perfusion of the of the kidneys or even the extremities. So you're cool and clammy. Whether the abnormality is due to low cardiac output with high systemic vascular resistance or high cardiac output with.

Low, low vascular resistance.

So you must have heard of low output, heart failure and high output heart failure.

Some of the examples of high output heart failure are like being of Galilean patients who who are in heart failure.

But that is secondary to too much, you know, output from the heart and low output failures or classic, you know, patients who don't have enough contractility of the heart and cannot produce enough cardiac output.

One of the common ways that adult people think of heart failure is by dividing it by ejection fraction. So you can have heart failure with preserved ejection fraction or heart failure with reduced ejection fraction.

So so.

If.

If you have heart failure with reduced ejection fraction.

That is the the contractility of the heart is affected. The ejection fraction is affected.

So that is that means that the amount of blood that is getting out of the heart is is affected heart failure with preserved ejection fraction is basically diastolic heart failure.

So the heart is too stiff, the muscles are too stiff and the heart is unable to relax.

And that impairs the filling of the heart.

And that's once the filling is impaired.

That means that not enough cardiac output goes out.

So think of hypertrophic cardiomyopathy patients.

In those patients you have impaired relaxation and that's why you can't.

Have enough cardiac output in those patients.

So Doctor Omdani, who is also one of our my senior Co fellows at at Trans Hospital of Michigan, where I worked with Doctor Kamat.

They studied national databases like kids database and NAIS database, and they looked at the they wanted to see the burden of pediatric heart failure.

And they determined that.

You know, compared the burden of pediatric heart failure in the ERS as well as you know, hospitalization and compared from 2012 to 2016. And they saw that the burden of pediatric heart failure is on the rise.

And you know, majority of the pediatric heart failure related Ed visits and hospitalizations were due to congenital heart disease, conduction disorders, arrhythmias or cardiomyopathy.

And the even though the pediatric heart failure burden was lower when compared to adult heart failure burden on the ER and the hospitals, the deaths in the Ed and in hospital were significantly more likely in children presenting with heart failure than adults and.

We have to take all of this with a grain of salt because all of this is from administrative data that has been coded by administrators and so sometimes using those large databases for making these analysis can be a little bit tricky.

But but there is decent amount of burden on the hospital system for pediatric heart failure and that's why it's very important that we pay attention and you know.

This paper just highlights how much.

Pediatric heart failure is bubbling beneath the surface and we need to develop an innovative solutions for these populations.

When we think of symptoms of heart, failure vary vary greatly based on age.

So in infants, you may see patients who are not feeding very well.

Who are you know, failing to thrive, who have rapid breathing, excessive sweating, a lot of irritability.

You know children.

Older kids, slightly older kids. You can have some fatigue.

Difficulty breathing some.

Poor growth can have some.

You can see some swelling in the legs or abdomen with fluid overload.

And teens often present with exercise intolerance, abdominal pain, nausea, vomiting,

actually abdominal symptoms. Most of the teenagers that I've seen with who come in acute decompensated heart failure are often presenting with abdominal symptoms due to poor gut perfusion, and they can also present with palpitations, chest pain and.

Shortness of breath.

Shh.

So thinking about functional classification, we know that NYHA classification very well.

You know, stage one, class one being, you have no limitation to physical activity, 2 being you have some limitation.

Class 3 being that you have limitations for activities of daily living.

But you you don't have any symptoms at rest and Class 4 being that you have symptoms at rest and you are unable to do anything without discomfort and so.

That is Class 4 and that is where you know advanced cardiac therapies like ventricular assist devices or listing for heart transplant. Those are the conversations to be had.

But obviously we can't ask an infant what you know what? What kind of discomfort they have with activity.

So Doctor Robert Ross, who is my program director at Children's Hospital of Michigan for Cardiology Fellowship, came up with the Ross Pediatric Heart failure classification. And so it was modified from the NY two classification for infants.

So class one being patients who are asymptomatic, +2 being patients who have some to get Mott diaphoresis with feeding and then for older kids, some Diaphoresis on exertion, Class 3 being marked to keep Mott diaphrangers with feeding.

Poor feeding time.

So patients saying that, oh, my kid is taking, you know, almost.

You know an hour to feed and still that you see that the patient is not growing well on your growth chart.

And Class 4 being, you know, to Get Me Out of tractions, grunting, diaphrangers, all of this at rest. So patients who present with bad myocarditis or acute decompensated heart failure and they're breathing really hard. Those are class 4 patients.

And then ACC and AHA came up with stages of heart failure.

So. So think of them as very different from class of heart failure.

So class is the functional class.

Staging system is basically identifying patients.

Who are like where they are in their heart failure journey.

So stage A being patients who are at risk for heart failure.

So these are patients who, you know, have some risk factors that that is built in into the.

You know, built into the risk factors history of the patient.

So for example, these are patients who have history of hypertension.

Maybe they were exposed to cardio toxic therapies.

Because of chemotherapy or prior history of cancer.

They have a family history of cardiomyopathy and were found to be genotype positive for like a pathogenic variant.

So these are all patients who are at risk for heart failure.

Stage B would be patients who who don't have heart failure but have maybe some signs.

So one of the most classic ones being, you know, if you have asymptomatic cardiac dysfunction.

And patients who have history of chemotherapy, you know for high dose chemotherapy with anthracyclines or if patients have you know increased biomarkers like BNP or cardiac troponin, you know those are patients who are pre heart failure.

They don't have any symptoms, but they have some signs of heart failure.

Stage C being symptomatic. Heart failure patients. So any patients who have any sort of symptoms and signs of heart failure currently and stage D.

He is actually patients who have marked her failure symptoms that interfere with daily life, with recurrent hospitalizations. Despite attempts to optimize medical therapy in these patients.

So these are advanced heart failure patients are the ones that we see in our pediatric cardiomyopathy clinic or heart failure clinic that we are considering for ventricular assist device therapy or talking to the families about listing them for a heart transplantation.

So thinking about etiology of pediatric heart failure, the two big buckets I think of are the congenital heart disease patients.

So Hyperplastic left heart, or teetrology of Fallopians that are, you know, repaired, but have a bad RV ventricle at large VSD patients who are sort of in that high output state or high output heart failure.

So those are the patients who have, you know.

In infants and young children, but also as you think of older Fontan patients.

Have Fontan failure for different reasons that are not pump failure but.

Patients who have other issues with the right sided elevated Fontan pressures. So. Cardiomyopathies is the other big buckets. You can have dilated cardiomyopathy, hypertrophic cardiomyopathy, a restrictive cardiomyopathy. And these are patients. That is the next big bucket that I think of. Well, when I think of patients with heart pediatric heart failure, myocarditis is you know.

A.

A situation where you have inflammation of the heart secondary to viral infections that can inflamed muscle. Obviously the myocarditis patients can have normal function for a while, but then it can develop into a cardiomyopathy like a dilated cardiomyopathy. Or it can recover.

But sometimes my carditis patients present in such bad inflammation that they have severe heart failure in the kid phase and then over a period of time they show some recovery.

Arrhythmias is another reason why you can have reduced cardiac outputs of patients who have ectopic atrial tachycardia long periods of time or SVT over a long period of time that goes unrecognized.

Those are the patients who have heart failure over a period of time. If the arrhythmia is not controlled and the important part about this to remember is that it's very important to recognize.

This because you can have a patient who have.

Who has an arrhythmia and cardiomyopathy secondary to arrhythmia?

So tachycardia induced cardiomyopathy and this is actually a fixable way reason for cardiomyopathy.

So if you fix the arrhythmia, the hope is that over a period of time with some assistance, the heart function will recover.

So these patients can escape, you know, heart transplantation.

Some chronic conditions like metabolic disorders or neuromuscular disease like Duchenne muscular dystrophy. They can also present with some cardiomyopathy.

Kawasaki disease patients, if they have bad aneurysms or if they have some thrombosis and then obviously cancer therapies like I mentioned secondary to anthracycline or chest irradiation. Those are also patients who can develop heart failure over a period of time looking at the ISHOT registry database we found.

That you know that like the two big buckets, like I was talking about.

Majority of the patients who were listed for pediatric heart transplantation formed. Were from dilated cardiomyopathy but almost 40% had congenital heart disease

who were listed for heart transplant, so, so congenital heart disease forms an equal almost equal bucket for a pediatric heart failure you know.

Patients.

And then when you look at the distribution of these over different continents, you see that in North America, congenital heart disease, which is the pink.

Bar graph show is a pretty big bucket for patients listed for heart transplant, which is not true in outside of Europe and North America.

So in Asia or other places where majority of the transplants happen for patients with cardiomyopathies and so, so this this may look very different, pediatric card failure may look very different in different.

Continents.

Then when you look at infants, you know less than one year of age.

And this is by era. Over a period of time, you can see here that the the blue Big Blue box is is congenital heart disease patients.

So so our congenital heart disease patients kind of formed the majority of the heart transplant patients back in the 90s when heart transplant was starting because we we thought we were not very good at transplant and so.

We, you know, we're listing patients who.

We didn't think we could palliate anymore, and this is, of course, infants less than one year only. But over the period of time, as you can see again, even though we see a lot of cardiomyopathy, majority of the infants who get listed for a heart transplant.

Are are congenital heart disease with almost 57% of those patients?

So the ACC divides the heart transplant, like I said, by 8 stages and so on the left.

Here you see stage 8 heart failure.

These are patients who are at risk and so these are sort of management strategies from the 2022 guidelines for heart failure and of course these are geared towards adults.

So I wanted to bring your attention to one or two things.

One is patients who have exposure to Cardi toxic agents. You know the 4th box there.

So this is where pediatric cardio oncology as a field is developing and it is one of the passion projects of mine. And so you can see that these patients who are exposed to high doses of anthracyclines or radiation doses, these are the patients who.

Period of time develop fibrosis and scarring of their heart muscle and have you know even though they present with some sort of dilated cardiomyopathy it has some

restrictive Physiology in it.

And then the the 5th box there is first degree relatives of patients with genetic or inherited cardiomyopathy.

So we often find patients who are genotype positive but phenotype negative for cardiomyopathies as we do cascade screening for family members.

So if you have one patient who presents with cardiomyopathy.

We often cascade test all the first degree relatives, parents, siblings and so we often find these patients who do have the mutation but don't really have the phenotype yet. And these are the patients who are at risk for heart failure in the future and then other metabolic dis.

Or diseases like muscular dystrophy that form another, you know, patients who have inherited risk for cardiomyopathy.

So these are the patients that I put in the bucket of stage A.

Heart failure.

And then pre heart failure, stage B are patients who you know have sort of asymptomatic LV dysfunction.

So again, cardio, oncology patients, I often see patients in my clinic where patients have received cancer therapies in the past and now they have asymptomatic LV dysfunction.

They're not symptomatic, but their es are in the 40s or even sometimes lower than that.

And so those are the patients that are kind of put in the stage B.

Bucket because they don't have any symptoms from this from this LV dysfunction.

And but then they are definitely at risk for getting worse over a period of time. So as we think of management of heart failure, you think of acute decompensated heart failure. These are patients who are in your ICU and then you think of patients who are chronically out.

Managed and so acute decompensated heart failure.

You know, we we treat them with inotropic support.

We we diurease them.

Intubation helps the left heart and you know so.

We often end up integrating these patients to help offload the LV, and then we have mechanical circulatory support. So in an acute setting in different places it could look different. So ECMO is definitely.

One of the number one ways to support these patients when they come in acutely

decompensated, but now more and more in pediatric patients, we are using ventricular assist devices.

And going elect semi electively and semi urgently in in that as we see a patient. Kind of slide on inotropes.

We go electively for a ventricular assist device.

Implantation and so chronic management of these patients. You know there these are the patients who are outpatient management.

So some of the medications that we use, obviously diuresis is important, but it is not a pillar of what we call guideline directed medical therapy or gdmr.

That's the word that the adults use for these 4 pillars.

Of heart failure therapy.

So the first one being ACE inhibitors or ARBs angiotensin receptor blockers or even Arnie nowadays.

Entresto, as you may know that medication, so beta blockers are definitely another chronic management therapy that we use in patients to reduce like I mentioned before, reducing their heart rate a little bit to help them feel a little bit better.

Mineralocorticoid receptor antagonist.

Or aldactone, you know aldosterone antagonist those are.

Those are very important for reverse remodeling in these patients and we often put that on and there is more and more data coming out for SGLT 2 inhibitors. So this these are these medications were actually being studied in diabetic patients and they found that that significantly reduced heart.

Failure and readmissions for for adult patients.

And so.

They've had a lot of now clinical trials.

That have documented that SGLT 2 inhibitors as chronic therapy helps.

These patients, in many multiple ways.

It's it's kind of still a little bit of mystery as to how they exactly work, but predominantly by glycosuria and and helping them be not as fluid overloaded and it keeps them out of the hospital and there has been very limited pediatric experience at this point in time.

But more and more interest in studying this medication in in Pediatrics.

And then obviously the gold standard for treatment for heart failure, pediatric heart failure is heart transplantation.

So this schematic here kind of shows you, you know, remember the stages stage C

and stage D of pediatric heart failure.

And so as you look at the left, so some of the medications that I already mentioned in terms of treatment, you know diuretics ACE inhibitors to reduce the afterload beta blockers to help with the heart rate aldosterone antagonist to help with the remodeling.

Digoxin is one of the medications that has some inotropic effect.

And so those are the medications that we start when patients start having some symptoms.

So they're in stage C, heart failure. The way we monitor these patients over a period of time is obviously the number one key factor is growth and development.

And so if the patient is not growing, if the patient is not gaining weight on on optimized medical therapy despite getting adequate calories now.

So it's very important that we give these patients enough calories.

So for babies, we recommend 120 kilocalories per kilo per day. So if the patient is not showing significant growth and development, then that is clearly.

You know, a patient who is indeed who is not compensated enough, and those are the patients that we talk about transplant evaluation.

We also monitor them with echocardiography, or if they're older with MRI of their echo windows are not very good.

I want to emphasize something about this, so people often think about, OK.

We started this medication.

Let's get an E2 weeks later.

Let's get an echo the next day.

Day-to-day changes in EF which is very dependent on patients. Fluid status is not something that we that we look for what we look for is meaningful change over a period of time.

So if you are an inotropes, maybe every few days or once a week, we look at the the echocardiogram.

To see what is happening with the patient. But essentially when you put these patients on medications, do you have to wait a long period of time before you start to see the remodeling? And so these patients, you know, may not show you changes in their EF unless you?

Know you have given them optimal medical therapy for a couple of months.

For older patients, we can have exercise testing for these patients. ECG is very, very important, like I said.

Arrhythmias. Tachycardia induced cardiomyopathy could be a very important reason for heart failure and so it's very important to make sure that you've ruled that out as a cause for cardiomyopathy. We monitor their electrolytes because in the acute phase we are giving them a lot of medications in.

And so keeping their electrolyte, it's, you know, cardiac safe sort of to say, you know, potassium optimized as well as magnesium to avoid any tachyarrhythmias.

And then we also follow their BNP.

Levels and you know there has been research studies shown that if over a couple of weeks you're not seeing a marked reduction in your BNP, which is a biomarker for heart failure, you know these are the patients that you don't feel very strongly about, you know being successful.

On oral heart failure therapy and over a period of time, we do, you know, cardiac Cath testing on them to make sure their PVR is not elevated and then you know if they reach stage D which is severe symptom.

We decide that we list them for a heart transplant.

Some of these patients.

Can sit in the hospital for many weeks on Endo pump infusions and you know, we we look at these patients on something called an intermac scale. And if they're sliding on inotropes. So like, for example, if they're still symptomatic despite being on good doses of inotropes or if their.

End organ function is starting to look worse on inotropes.

These are the patients that we semiotactically.

Kind of take to mechanical circulatory support of VAD, or if they're acutely decompensating then we place them on ECMO and then these are the patients who get listed for heart transplant and heart transplantation.

So when we think of a QD compensated heart failure, you think of whether the patient is wet or dry and whether they're cold or warm.

So those are the four ways to think about acute decompensate or heart failure.

So whether they're congested or not congested and whether they have?

Adequate. Prepare for perfusion or they have critical hypoperfusion.

So so when you think of it and divide it into these four categories, you think of patients warm and dry, that is your optimal profile.

That's what you want to focus on to prevent disease progression and decompensation when you think of patients who are warm and wet.

So they have adequate perfusion, but they are very, very fluid, overloaded. The

standard therapy is diuresis. So you treat them with Ivy Lasix.

And you try to get get their fluid back down.

And you try to not let them beat that fluid.

Overloaded. Then you think of patients who are cold and dry.

So these are the patients who are not congested, do not have fluid overload, but you know are have very poor forward profusion.

So these are the patients that I would, you know sort of try to treat them with inotropes. But after a while, if they're still cold on inotropes, that cardiac output. Is not great.

Then these are the patients who are, you know, telling you that they need.

Mechanical circulatory support.

And cold and wet is sort of the worst, right?

So you don't have adequate profusion, and yet you're really backed up.

So you're really congested, so you have, you know.

Liver, that is liver, that has a lot of backed up blood and then you have, you know, pulmonary edema.

So these are the patients that we try to treat with both, but at the same time, you know, these are the patients who who come in very, very sick.

And could be, you know, crashing onto ECMO.

And so when we think about, you know, the schematic, these are this is what happens pathophysiologically.

So let's talk about, you know, let's go 1 by 1.

You know when you have left sided failure or left sided dilation you have increasing your LA pressure and you have lung congestion.

And so you'd have shortness of breath or thoughtnia cough. And then over a period of time you have low cardiac output, low blood pressure.

Which results in decreased organ perfusion can have renal failure, fatigue in teenagers. Sometimes we often see behavior changes and that is something that you know sometimes is under recognized, you know, in patients, teenage patients, you know, these are the patients who start failing classes or who are sudden.

Being very obstructive and oppositional and and that is something that, you know, you have to recognize.

Then we have neurohormal activation as a result of that.

That you have increase in the appi and norappi which increases the heart rate you increase in the angiotensin 2 and Brennan the aldosterone system which increases

the afterload in the body. Your increase in sodium water retention and aldosterone which causes edema in the body and all of these.

Results in adverse LV remodeling, which leads to further LV dilation and failure.

So this is sort of the circle of heart failure that we're stuck in.

When we think of right sided heart failure, we think of you know increased JV.

And liver congestion on liver failure.

So you start to see hepatomegaly.

You start to see, you know, elevated bilirubins you have decreased perfusion to the liver.

You can have ASD, you know transaminitis and so when we think of treating these patients, so we think of you know Lasix for.

Treating your congestion like we discussed to increase some sort of inotropy we think of maybe Digoxin or inotropes actually. But if you think of Chr chronic heart failure, we think of Digoxin.

To think about increased heart rate we, you know, treat them with beta blockers afterload being treated by ACE inhibitors or ARDS. And now Arnie's, you know, treating your aldosterone, you know water retention with aldosterone antagonist like aldactone.

All this results in reverse remodeling of the heart.

Now, all of these medications were studied in adult patients and there have been very limited studies in pediatric and I just wanted.

Find out a few studies that were that were done in Pediatrics and as you can imagine, it's really hard to do trials in Pediatrics. So. So the first one is the CARVEDILOL trial that happened in 2007.

So this is a this is, you know, just studied carvedilol in all heart failure patients, all commoners. And they enrolled about 161 patients in four years and they randomized them to, you know, either placebo, low dose carvedilol, high dose carvedilol.

As we remember, as a beta blocker it's a non specific beta blocker which helps with you know remodeling of the heart. And so you know it's often prescribed as you know first line beta blocker for pediatric patients.

Unfortunately, they found that Carvedilol did not significantly improve cardiac clinical heart failure outcomes in children adolescent who had symptomatic heart failure. But their event rates were very, very low, and so they thought that the trial was maybe under.

Powers to recognize differences between, you know, between these events and the

events that they discussed were death hospitalization for more than 24 hours requiring IV medications, you know, worsening heart failure, treatment, treatment failure, you know, withdrawal of consent for other reasons, or worsening NYT or Ross class.

And overall global worsening.

You know, observation by the clinicians.

And so they didn't.

They didn't find a lot of events in these patients and so they thought that that maybe the trial was underpowered because Carvedilol is still being used in pediatric heart failure.

As almost the first line, you know therapy for, you know, pediatric heart failure. One of the things that I wanted to point out was that this had a very heterogeneous population.

So even congenital heart disease patients who had systemic Rv's were also included in this.

So, you know, they also found that maybe the when they did some subgroup analysis that maybe the carvedilol worked better on patients who had systemic LV and not systemic RV.

And so that kind of taught us a little bit about, OK, like maybe we need to be very picky about what patients we pick and study these drugs on because, you know, some of these medications are are not really that effective on systemic Rv's.

The next trial that came was in 2010.

In Allopurin, patients with single ventricles. So this was run by the Pediatric heart network that was established and you know, Doctor Daphne Sue, who was also my mentor.

Ran this trial and they enrolled 185 infants who had single ventricle Physiology into, you know, one us to 1-2 groups.

Enalapril versus placebo?

What they found was that there was no significant differences between the two groups in height Z scores.

So they were growing very well.

They had no differences in their heart failure class, BNP concentrations or ejection fraction.

And death and transplant also did not differ between the two groups and was at about 13%.

And So what they said was that this randomized trial does not support the use of routine use of an allopurinol in this population.

So these are all single ventricle patients who are placed on an allopurinol.

Again, people thought that this study was.

You know.

Underpowered.

So you know, we learned our lessons from some of these prior trials and the latest trial that we had was, you know, in circulation in 2024 Panorama HF trial, which is a prospective trial to assess the angiotensin receptor blocker nebulizing inhibitor.

So Arnie, angiotensin receptor NEBULIZING inhibitor, which is Entresto versus an ACE inhibitor for the medical treatment of pediatric heart failure.

So again, you know this was a big trial and I don't know if you guys have heard about the paradigm trial which was the adult version of the Entresto trial, which was published in 2015.

So you know, these therapies kind of come to Pediatrics a little bit later.

So they enrolled about 375 patients. Again they learned from the prior mistakes and they decided that they would.

You know, only enroll patients who have two ventricular.

Physiology, their ESR less than 45%.

And so they enroll them in, you know, either sacubitril valsartan, which is Entresto versus enalapril, and they studied these patients for endpoints and endpoints. Again being, you know, NYCT class global impression of change and anti pro BNP.

And what they found was that sacubitril valsartan did not show any superiority over an ACE inhibitor, but also did not show any inferiority.

That both the treatment arms showed clinically meaningful improvements and so anecdotally, a lot of the pediatric heart failure providers are starting to use Entresto and their patients over over 8 just because you know the the anti Pro BNP and you know dropped pretty sign.

In these patients and this medication was actually FDA approved.

Based on just the anti pro BNP drops.

These patients.

Does do people I apologize.

I can't see the chat, but do people know what was the first medication that was used to treat heart failure?

I'll give the answer.

It's digitalis so foxglove or digitalis. You know, there's history of people, you know, older ladies having tea parties with a little bit of the talus in their tea, and they all go back home and, you know, feeling better. And so, you know.

Digitalis tea parties is the oldest sort of.

Cute anecdote about heart failure, so thinking about outcomes of heart failure.

You know what?

What are the outcomes? You know it depends on the underlying cause of the.

Patients, you know, unfortunately not a lot of improvement has been made over the years.

Mortality rates for children listed waiting for her transplant still remains pretty high, almost 17%.

We noted that in the CARVEDILOL trial, you know most subjects who were enrolled were Class 2 patients.

So again, remember the functional class of heart failure.

So Class 2 is, you know, patients who.

Were not very, very sick and only two patients with Class 4, but their mortality rate.

Was still 7% and another 11% underwent.

You know, her transplant over eight months of pretty high numbers.

But there was symptomatic improvement in 55% of patients in the 1980s. You know, one year mortality used to be 20%.

So honestly, even though some improvement has been made, there still remains like we have miles to go in, you know, treating, you know, heart failure in children.

And this is a very beautiful depiction of clinical course of heart failure.

From the World Health Organization, you know on the top is quality of life and on the bottom is intensity of care.

So as you can see, over a period of time, you know onset of congenital heart disease, onset of congestive heart failure, your quality of life slowly over a period of time as you start to see decompensation and pump failure, quality of life starts to get worse and worse.

And worse.

And as you start to have pump failure, you know you need to consider mechanical circulatory support, transplantation.

To improve your quality of life and the intensity of the of care for these patients goes up and up and up further.

And so, you know, one of the things that has come about in in the last decade or two

has been ventricular assist device.

So you know, what is the ventricular assist device?

It's an electrical battery operated mechanical heart pump it assist and takes over the pumping function of the heart depending on what side of the heart you're supporting, it can be a right ventricular assist device or an arvad left ventricular assist device.

Or LVAD, or both ventricles, that is by VAD and it allows for delivery of oxygen blood to vital organs of the body can be short term.

Long term, it can be external, you know, pertinently placed or internal that is implantable.

But we still think in pediatric heart failure.

Cardiac transplant is the gold standard for treating heart failure, but we all know that donor hearts is a very scarce resource. We can't as of yet.

Grow hearts in our laboratories.

And so, you know, we need to have.

Some way to keep these patients alive and with improved quality of life while they're waiting for a heart transplant.

So the types of MCS that we think of are short term support devices.

So, you know ECMO, mid term support devices for a few weeks centromeg or pdmac long term support devices, Berlin X core heart or hardware which is discontinued.

And I'll talk about that at heart, mate. Three and then.

You know, if you're adult sized patient you can also get something called total artificial heart.

Which Pediatrics has limited experience with and when we think of LVAD or ventricular device, we categorize them by what are they as a bridge to?

So they could be a bridge to transplant.

So these are patients who are not expected to survive to transplant and you know, wait for a donor heart. So they benefit from an improved functional capacity, quality of life and they enter transplant in better physical and functional shape. We think of them as.

Destination therapy.

So these are patients, you know, who do not have.

Who have end stage heart failure but are not eligible for heart transplant are patients who we know have limited.

Life expectancy.

So patients when Duchenne muscular dystrophy, those are patients that are often offered LVAD as a destination therapy knowing that they would never be eligible for a heart transplant.

Patchaw recoveries to think of like patients with myocarditis.

This or you know, a cute onset of their cardiomyopathy that you don't know whether these patients might recover over a period of time.

And then if you're not sure about what is going to happen with this patient and you don't want to listen for a transplant because you're just getting to know these patients.

It can be placed as a bridge to decision.

You know, thinking about like, how often do we do Vads more and more frequently. Actually, you know about 20 to 30% of patients who are listed for a heart transplant. Are bridged to a transplant with mechanical circulatory support.

So the pink bars are.

Acmo patients who are supported with ECMO, but blue bars are patients who are supported with a ventricular assist device, and these are pediatric heart transplant patients and you can see over a period of time more and more patients are being supported with ventricular assist devices and less and.

Less patients are being supported with ECMO just because we know that the the adverse events with ventricular device are much less than an echo.

And as you can see here, the distribution by age group pretty similar across the board, though infants maybe sometimes are being still more supported with ECMO. But but more and more patients above the age of 1 year are supported with avad and when you split them up by.

The two big buckets that I mentioned about heart failure, congenital heart disease versus cardiomyopathies, you know, obviously like cardiomyopathies, it's much easier to place a ventricular assist device because of the way the devices are.

And I'll go that over that in a minute.

And as you can see, over the eras, these are over five Year 5 to 8 year intervals.

You can see the the blue bar is ECMO that is getting less and less and the sorry the light blue is ECMO and the dark blue is vad and you can see in the current era VAD patients who who are less than one year of age are being.

Supported more and more with VAD and less and less with ECMO.

The majority of the patients are still being transplanted without any device support.

And again, looking at patients less than one year divided by their etiology, again,

most patients who are supported with a VAD are dilated cardiomyopathy patients and less so as congenital heart disease. But as you can see here, you know still a majority of the patients are being trans.

Despite, you know, without mechanical circulatory support but a decent number R you know if you see here less than one year of age between 2005 and 2018.

Dilated cardiomyopathy patients.

Almost 30% of the patients were supported with the ventricular assist device.

That's a big number.

When we look at outcomes or survival after transplantation.

This is what I meant that patients who are supported with only an ECMO, that is the light blue line.

Over here you can see that their survival over post transplant is is remarkably different than the other two lines, and the green line is the patients who are supported with the VAD.

And the red line of the patients were supported with absolutely no mechanical circulatory support.

And so those lines are almost overlapping, showing us that long term survival post transplant in patients, whether you're supportive of the VAD or with no device are almost similar, but the patients who are on.

Some of it has to be taken with a grain of salt because those patients are placed on akmar, probably sicker, to begin with.

Or have congeal heart disease that is very hard to repair.

And so those are the patients who have relatively poorer outcome.

And in most events that we think of with Vads are, you know bleeding obviously because these patients are on anticoagulation strokes because obviously all these patients who are have these foreign surfaces, you can have clots build up on them and you, they can obviously travel to the brain.

Because they're supported often on their left side pump thrombosis.

So you know, these are patients who whose pump stops working because of clots in the pump.

Infection because they're in the hospital.

Most of them have a lot of lines like central lines piclines in addition to the candela sites, and given that these canal are sitting right in the heart, you can have arrhythmias. And obviously This is why we think heart transplantation is a gold standard treatment for heart fail.

In these patients, because you know, VATS of course have adverse events. So talking about what kind of different metric or devices we have for Pediatrics, the 1st and the foremost is.

Berlin X score heart and you can see here this is this is the device over here.

It kind of sits outside the heart and it's a pulsatile device.

It has valves in it that one big flip side is that it has this huge aged computer that is huge.

It's called and sits at the bedside of the patient so these patients can never leave the hospital.

There is a clinical trial being done for what we call an active driver.

So those patients would be a little bit more mobile and the computer is a little less bulky, but still you know these patients you know you can imagine like this pumping thing that is sitting outside the heart.

Be supporting the heart.

You know it it.

These patients don't really leave the hospital and can live here for months, if not sometimes even years.

Waiting for a heart transplant. So in 2021 there was a Berlin Post surveillance study.

The Berlin heart study that was done back in 2007 to 2014 had 320 patients, and this study using what we call the Action Network, which I.

Will touch upon later, you know, enrolled.

Another 72 patients and we found that we are now supporting younger patients, patients who are smaller patients with we're more likely to have congenital heart disease and we are using bivalirudin and we have reduced stroke in these patients by 40%. And what we call pump exch.

By 40% and almost 86% of these patients receive a successful outcome, which could be either a transplant or recovery.

Talking about an implantable device.

Want to just briefly mention Heartmate 3 which is the latest and the greatest adult device?

So it is a fully levitated.

Central rotor magnetically levitated that has no mechanical bearings and so it is implanted into the pericardial space over here.

So you see it on the chest X-ray of the patient.

You know it is the size of half the palm.

It has some intrinsic pulsatility which reduces stasis and minimizes thrombus formation.

But it's a continuous flow device. So in Pediatrics, we studied these patients who got heartmade 3 implanted. As you can see, most of them were 15 years of age. And so the it's implanted in larger patients, though we have implanted in 17K patient, most of them had.

Dilated cardiomyopathy and you know 61% underwent heart transplant and this could also be offered as a destination therapy.

And you can see strokes is only 6% ischemic.

Stroke and 1% hemorrhagic stroke.

Uh, but if you were a small kid who got this large device, you had a higher incidence of infection, renal dysfunction and stroke. Wanted to talk about Action, Learning Network, advanced cardiac therapies, improving outcomes, Network that celebrated A7 year anniversary in 2024 started as a.

Vad Qi.

Research learning Network but has now multi pronged industry research.

It has industry partners now.

And we now have more Vads with pediatric labels.

So heart made three actually has a pediatric.

Label now and it is doing a lot of pediatric. A patient reported outcome research and so not just improving hard outcomes like strokes but also patient reported outcomes like quality of life.

So this is a a learning network that is unique to pediatric heart failure that is dedicated to improving pediatric care. And just an example of them is you know they called this.

ABC.

Model which you know they worked on anticoagulation.

And blood pressure control and communication skills of the ICU team. And they found that the reduced the stroke rate in Berlin heart patients by 50%.

So from 30% they almost brought it down to 12% stroke rate just by improving anti coagulation protocol, standardizing it, standardizing blood pressure control and standardizing communication across ICU teams.

And so I think that there is a lot to be said about learning networks and you know, communicating with each other.

Across.

Across centers, so each VAD hardware used to be.

A small ventricular device that was actually very helpful for our patients because it was implanted and these patients could go home and you can see here in the picture comparative to the Heartmate three, it was a much smaller device, but once the heartbeat three came to the market.

Medtronic decided to stop HVAD being distributed, and that has been a huge challenge for a pediatric patient.

Our pediatric patients are now being forced to implant with Berlin, Berlin hearts and are being forced to stay in the hospital.

We are having a hard time finding Berlin, Canadas or icoses.

We're having a hard time supporting these patients, you know who come in acute decompensated heart failure. We're having to use less efficient devices in our kids.

And there is a huge need for advocacy. And you can see here the change in the type of devices we use you.

Know with the.

You know this?

This dark blue being, you know the H VAD and that has completely disappeared.

In in in the current you know after 2021.

And so we're having to use less efficient devices like the centromed or the PD Mac that have a relatively higher stroke rate.

And so, you know, I know I'm running out of time.

I just briefly wanted to talk about, you know, looking at overall assessment of these patients. We talked about ekg and anti arrhythmic medication.

Nutrition is a huge thing.

We need to make sure these patients are growing. A lot of these patients are placed on anticoagulation because stasis leads to clots and so patients are placed on aspirin if they have non compaction or bivalirudin, or Coumadin if they have thrombus. We have to consider ICD in these.

Patients, if we're sending them home and exercise is a huge area that still needs a lot of research as to what kind of exercise limitations we give to these patients.

Because we also don't want them to be couch potatoes and become a beez.

And lastly, I just wanted to touch upon a multidisciplinary model for pediatric heart failure patients.

It is very important to partner with your colleagues, so while you should have a heart, you know, talk to your heart transplant colleagues. We also need to talk to patients

who take care of single ventricle patients or single ventricle program genetics team to make sure that we are.

Getting a full genetic assessment on these patients or oncology partners to send US patients.

Beyond for heart failure are neuromuscular colleagues, neurology, colleagues to send US patients with muscular dystrophy and you know, we need not just Md's, but also our nurse practitioners, social workers, psychologists, pharmacologists to help us in care of these patients in a very multidisciplinary model. So in summ.

Our failure defined as inability for heart to keep up with the body's metabolic demands.

Remember this equation.

Cardiac output stroke volume 10's heart rate.

There are many ways to classify and stage heart fail.

And medical managements consists of anthro diuretics, arneys beta blockers.

We are doing more and more Vads almost 30% where the pump takes over the heart function and we now have multiple MCS options in pediatric heart failure like Berlin hearts or heart threes.

But there are many adverse events associated with this, so it's only in patients reaching cardiogenic shock and heart transplant is still the gold standard for treatment for heart failure.

And thank you so much for giving me this opportunity.

I really appreciate it and I'll be happy to take any.

Questions.



Kamat, Deepak M 59:55

Thank you, Doctor Bansal, for that wonderful presentation on pediatric heart failure. Doctor abarbanell.

Do we have a question?

I saw your hand up.

I don't see. OK.

Anybody has any questions or comments for Doctor Bansal?

We do have a few minutes because we have faculty meeting right after this, right?

There are two thank yous in the chat box.



Bansal, Neha 1:00:41

No, thank you so much. And you know, I actually ended up speaking with Doctor Aaron.

I apologize.

I'm forgetting yes yesterday because, you know, I wanted to see what he thought about me talking.



Kamat, Deepak M 1:00:51

About a bourbonian.



Bansal, Neha 1:00:58

So he was very kind to give me some pointers.

So some of the last minute slide Tweakings were thanks to him and what he wanted me to address today.



Kamat, Deepak M 1:01:07

OK.

I think I had a question, but he may have to leave.

Thank you, Doctor Bansal again for that wonderful presentation.

Thank you all for attending this morning's grand round.

We have a faculty meeting which is already started.

We talk to you later. Thank you.



Bansal, Neha 1:01:22

Thank you so much.

Thank you so much, Doctor Kamat.



Kamat, Deepak M 1:01:24

Bye bye bye.



Bansal, Neha 1:01:25

All right.



Bansal, Neha 1:01:25

Thank you all, bye.

● **Kamat, Deepak M** stopped transcription