LUIS DIEGO PACHECO: All right, so we're going to talk about hypertensive emergencies. Again, this is not going to be the typical preeclampsia talk. It will include the complications you can get for preeclampsia. You can have a [INAUDIBLE] hemorrhage, you can have an intracerebral hemorrhage, you can have an ischemic stroke, and so on.

So these are pretty much the objectives. First, we're going to go through the definition and the pathophysiology of hypertensive emergencies. And then we're going to talk a little bit about each medicine, the medicines that we use most commonly to treat these conditions. And then we're going to go through some little cases, mainly the different kinds of strokes and aortic dissection, and myocardial infarction, which agents to use in each of those conditions.

So the thing with hypertension is that the blood pressure is going to be the product of cardiac output times systemic vascular resistances. So if you are hypertensive, it's going to be one of these two. Sometimes-- and it does not always work-- but sometimes if you see someone that is for example 190 over 90, that's mainly systolic hypertension, right? And theoretically, remember that-- not theoretically, but remember that the systolic blood pressure depends on what? Depends on the stroke volume. It's actually the squeezing of the left ventricle the determines your systolic blood pressure, while diastolic blood pressure depends on the systemic vascular resistances.

That's why-- I don't know if you know, but the earliest sign on vital signs that you get when you're actively bleeding is actually going to be a narrowing of the pulse pressure-- before you become tachycardic, before you do anything else. Because when you're bleeding and your stroke volume starts dropping, what you do is you secrete catecholamines, and you squeeze your blood vessels to increase the systemic vascular resistances and maintain the mean arterial blood pressure. And that squeezing brings your diastolic up.

So what I'm try to tell you is that if you have someone that has mainly systolic hypertension, and you know that the systolic blood pressure depends on stroke volume, maybe those patients will do better with a beta blocker, right? Medicine that will decrease the contractility of the heart.

Versus if you have someone that is, let's say, 140 over 110, that patient is mainly hypertensive on the diastolic compliment. And you know that diastole depends on resistances. So what would be the best medicine for this person? Something that dilates your blood vessels, like nifedipine or nicardipine or hydralazine, or those kind of medicines, right?

One of the important things when we talk about these hypertensive emergencies is that you do remember that there are some organs, mainly the brain, the heart, and the kidneys, that they autoregulate their blood flow. So it doesn't really matter what the blood pressure is in a very wide range, the organ will maintain the perfusion.

So this diagram, this cartoon or whatever here, it actually shows the autoregulation of the brain. So in the brain-- this is someone that is not chronic hypertensive, someone who is healthy. Most of the preeclamptics, for example, can have these. But in preeclampsia you might lose some of the autoregulation. But what happens in the brain is that from MAPs between 50, all the way to 150, it doesn't matter what happens with your blood pressure. The cerebral blood flow is going to be constant.

So what happens is, let's say you become hypotensive, because you went running outside and you're dehydrated, and you are hypovolemic, and your blood pressure drops a little. So what happens is the vessels in the brain, they will dilate so that even with that low systemic blood pressure, you will have less resistance to flow, and you will maintain perfusion to your brain.

On the other hand, if you become hypertensive, like really hypertensive here, then it's the opposite. The blood vessels that go into the brain, they will squeeze so that there will be more resistance to flow, so you will not translate all that systemic hypertension into your brain. Does that make sense? So that's going to be pretty much the autoregulation.

Now, if you become profoundly hypotensive, then you're going to see that the curve is not flat anymore. That means that the cerebral blood flow will be dependent on the mean arterial blood pressure. So you will start getting periods of ischemia, and you might eventually get a stroke, and ischemic stroke. So that's the danger of dropping the blood pressure too much in the brain. Obviously, all the other organs can suffer as well.

On the other hand, if you actually become very hypertensive and you pass the limit of autoregulation, then now, here you can see how the blood pressure and the cerebral blood flow are going to be directly proportional.

So imagine someone with preeclampsia, and they actually have severe hypertension. And then they seize, and then they die. When they go and they have their autopsies done, usually in the brain you find areas of both ischemic strokes, small ischemic strokes, and you find areas of small hemorrhages.

Remember that the autoregulation is not going to be the same. And we're going to talk-- you know what PRES is, right? You heard about PRES with preeclampsia going to talk a little about that. And that's a good example on how the autoregulation is different in different parts of the brain.

So no one really knows why eclampsia happens or why magnesium-- no one knows anything about that. But one way of understanding why is it that you get those two-- both ischemic and hemorrhagic injuries in the brain of women with eclampsia-- is that some areas, they might actually autoregulate very well.

So the patient becomes hypertensive from the preeclampsia, and then you get this severe vasoconstriction around different areas of the brain in order to prevent that hypertension to go into your brain. Because hypertension in the brain is bad, right? If you increase the pressure inside the vessels, those vessels can either pop and give you a hemorrhagic stroke, or they can actually not pop, but if you increase the pressure inside the vessel, you start leaking plasma, right?

That's like when you have a hose that is semipermeable, and you just close it and you open it here, you're going to see how the water starts coming out. Same thing happens in those vessels, and then what do you get? Cerebral edema. That's called vasogenic edema.

So some areas might constrict so much to avoid that that they cause distal ischemia. That's like people with migraines, when you get the vasoconstriction before you start having the pounding pain. Some people get ischemic stroke from that vasoconstriction. So that will explain the areas of ischemia-- excessive vasoconstriction.

And there might be some other areas where what is in this graph happens. You just suddenly become so hypertensive, boom, you lose the autoregulation. And now that vessel that was squeezed to prevent that transmission of pressure now suddenly opens up, because you lost the capacity to autoregulate. And when it opens up, lots of blood comes, and then you can pop and get a little hemorrhage. OK? So that's why you don't want to be here, or you don't want to be there.

Now as you know, people with chronic hypertension. When you have someone with chronic hypertension, everyone is like, don't drop the pressure too much. And that's actually a very smart move, because people with chronic hypertension, they have displacement of this curve to the right.

So if you look at this person here-- I just made this up. I don't know, if these are the MAPs, it's probably going to be around here. But just for understanding, this person needs higher MAPs to maintain perfusion, right? And they will tolerate being more hypertensive and still be able to autoregulate.

So that patient-- and this is not necessarily pregnancy, but let's say, I guess, is gynecology or something. Someone comes into a clinic to have something else done, and they're perfect. They're totally asymptomatic, and their blood pressure is 190 over 100. And everyone freaks out and sends the patient to the ER. That patient, that's an urgency, that's not an emergency.

No symptoms whatsoever. Who knows how long that patient has been 190? He's probably around here, without a problem. I'm not saying you don't do anything. Start him on PO medications and titrate him, and over the next few days start dropping the pressure slowly.

But the worst you can do for that patient is just go put an arterial line on them, start on a drip of nicardipine, and then suddenly, you give them an ischemic stroke. Because they need higher pressures, and it takes weeks for this to go back to this.

The other two organs that regulate are going to be the heart and the kidney, as well. We're not going to go through every little thing on each, but it's the same principle. You don't want to drop it too much, or you don't want to have it really, really high.

So what is the definition of a hypertensive emergency? So if you take 20 different textbooks and you open them, they're all going to have different numbers-- more than 200 over 130, more than 220. The thing is, this is not that important. Because just like we said about that lady who has been hypertensive for 20 years and now she goes to clinic and she's 190 over 90. According to this, she's probably going to have a hypertensive emergency.

The main difference between an urgency-- a hypertensive urgency and a hypertensive emergency is that in the hypertensive urgency, you have no end organ damage. You have very high blood pressures, but you have no evidence of end organ damage.

When you have a hypertensive emergency, you have not only the high blood pressure, but you have evidence of end organ damage. And those, the latter ones, they need to be treated right away. And those are the ones that are going to need a main line, and they're going to need parenteral antihypertensives, and so on. Right? And you guys deal, we all deal with those every day, right? Because preeclampsia/eclampsia is considered a hypertensive emergency.

These are just examples of hypertensive emergencies. So someone that has severe hypertension and has neurologic symptoms. They just have a headache, nausea, vomiting, visual symptoms. Or they're focalized-- they cannot move an arm, they cannot move a leg, et cetera. Meaning they could have either an ischemic or a hemorrhagic stroke.

You already know why they happen. The ischemic stroke would be because you autoregulate so much and you end up having ischemia. The hemorrhagic stroke is going to be you lose the capacity to autoregulate, boom, and the vessel just pops, and then you get the bleed. That's how you can get both either an ischemic or a hemorrhagic stroke from high blood pressure. And that's what happens in preeclampsia, as well.

So that could be one. Or the patient that comes in with the hypertension and, not here but here, starts having chest pain. So that is considered an emergency, because it could be a myocardial infarction, or it could actually be an aortic dissection, right? So that actually is going to be important if they have shortness of breath. If they have shortness of breath, it might be from pulmonary edema, just the heart cannot empty that well because you're so hypertensive and you accumulate fluid retrogradely, and develop pulmonary edema.

Severe eclampsia and preeclampsia is considered actually an emergency. We're going to talk about why this happens in a little bit, and why this happens in a little bit.

Or acute post-op hypertension. You don't want someone that you have just operated on to have a bunch of pedicles, just like get in there with a little-- well, we use chromic because we're poor. I don't know, you guys probably use [INAUDIBLE] or something. With a little chromic there, it might just pop and bleed, right? So when you have severe post-op hypertension, that's considered an emergency and you need to treat it there in the PACU.

Now in terms of these two things, why is it that these two things can happen? I don't know if you remember, but the old textbooks used to call hypertensive emergencies something called malignant hypertension. I don't know if you remember. And malignant hypertension, what it is is hypertension in the setting of hemolysis. And hopefully in the next slide we'll understand why this happens and why this happens.

This is just a simplified review of a hypertensive emergency. So we just said the blood pressure is cardiac output times systemic vascular resistances, right? The vast majority of hypertensive emergencies, the vast majority are going to be secondary to a sudden increase in the resistances, not in the cardiac output. So most of them are going to be because this happens. So you suddenly squeeze your vessels more than you should [INAUDIBLE] here with increasing catecholamine secretion.

So a few things happen. So you increase the systemic vascular resistances. You squeeze the blood vessels. When you squeeze the blood vessels, the pressure inside the vessel is going to increase, right? And then that's going to cause mechanical stress on the endothelium. It's going to injure your endothelium. So you're going to get endothelial injury. Remember the endothelium, if you injure the endothelium, your vessels are going to be leaky.

Once you've injured the endothelium, what's going to happen is-- remember how you have, behind the endothelium, if you injure it, you have some endothelial collagen. And that collagen has a lot of tissue factor. And that is going to be a stimuli for platelets to adhere. And when the platelets adhere, the clotting factors are going to adhere to the platelets, and you're going to start creating little clots all around your body.

When you create those little clots all around your body, then when a red cell is going through that vessel, what is it going to do? It's going to pop. So you're going to get hemolysis. And that's why you get the hemolytic anemia, or the hemolysis, which is the old definition of the malignant hypertension. This is why it happens.

And on the other hand, what we were talking about before in terms of [INAUDIBLE], when you actually have all this injury to the endothelium, where you have all these little clots, if you don't do anything about it, and you don't treat this patients-- I mean, I don't think I've ever seen anyone with this, but I'm sure in some parts of the world, it happens-- if you don't do anything about it, these people are going to die. If they don't due from a bleed, they're going to die from end organ damage.

Because all these little clots are going to cause ischemia to different organs, including, for example, the kidney, which is what we were talking about before, here. Acute kidney injury. So you see the creatinine is going up, part of it is going to be because you have all these clots that are compromising the perfusion to the kidney. So you get organ ischemia and hemolysis from this injury.

On the other hand, when you have this increasing systemic vascular resistances, and you increase the hydrostatic pressure inside the vessels, if this is [INAUDIBLE] and this is your pee, and this is your blood, if you increase the pressure here, the filtration gradient will increase. You will filtrate more, right? Because you have very high pressure here. You're going to start peeing a lot, and then that's called natriuresis-- pressure natriuresis. So these patients are going to be hypovolemic. That's why many times it happens.

In some obstetrical textbooks, actually, they tell you-- some people tell you give a little fluid before you give the hydralazine. Because these people have been not well treated for a while, two or three days of being severely hypertensive, they have been peeing sodium and water. And they're hypovolemic, but very squeezed. You give the hydralazine, you open them, and they're empty. Boom, and the blood pressure drops. So you might want to give them a little fluid if they've been hypertensive for a while and not well treated for a while.

So now we're just going to talk about some of the agents, some more used in OB, others less. Do you guys still use-- we use a lot of hydralazine. Do you guys still use a lot of hydralazine? Yeah?

I think everyone uses it. There are some options that theoretically could be better, but it's cheap and people are very familiar with it. A few problems with-- you know, hydralazine is a pure vasodilator. It does nothing to the heart. All it does is just go and open up the blood vessels, drop the systemic vascular resistances.

One of the problems, if you compare it for example with labetalol-- which is in terms of preeclampsia what you use the most-- if you compare it, labetalol will kick in faster. Hydralazine will actually kick in a little slower. It takes 20 minutes to see the main effect of hydralazine, versus 10 minutes in case of labetalol.

One of the other things with hydralazine is, if you don't like what you see, it's going to stick around for a while. It's going to stick around for 10 to 12 hours, so the effect is prolonged. Now, one of the biggest problems-- which actually blows my mind that in OB we use it so often. Because if you go outside of OB-- if you go to neurocritical care-- you won't use hydralazine in the setting of edema. Patients with preeclampsia have edema in the brain, right? Patients with eclampsia, they have PRES. They have edema. Hydralazine has a very big side effect, and it is that it causes dilation of the cerebral vasculature.

And if you're trying to autoregulate, you get this medicine, it opens vessels. So it will worsen the cerebral edema. So for example, in traumatic brain injury or in strokes, people that have high ICPs, we never use hydralazine, or nipride or nitroglycerin. They all have that same problem.

Having said that, in OB we use it, and it's a safe medicine. But just something to-- if you ever had a preeclamptic that for whatever reason gets [INAUDIBLE] and has severe cerebral edema, try to stay away from hydralazine. It might be a theoretical issue, but it's there.

Nifedipine is very bad medicine for hypertensive emergencies. It used to be used sublingual, but it dropped the pressure too much and people got ischemic strokes, so you don't use it anymore sublingual. You can give it by mouth. It takes a while to kick in, and it lasts for a good 8 to 10 hours, as well. Most people actually don't recommend to use nifedipine in the setting of a hypertensive emergency. And I know there's some places, but if you have hydralazine, you have labetalol, you have nicardipine, those are probably better options as compared to nifedipine And nifedipine suffers from the same problem of hydralazine-- it dilates the brain vasculature.

Nitroglycerin-- this is not really a good antihypertensive. Nitroglycerin, remember you have two kinds of vessels. You have the capillaries, but on top of that, you have the arteries and the veins. Nitroglycerin is mainly a venous dilator. That's what it does mainly.

Have you guys ever used it in pulmonary edema? It's a good medicine for pulmonary edema, because what happens is that nitroglycerin mainly works on the veins. And if you dilate the veins, then you sequester blood in your venous system. And if you accommodate more blood there, it's not going back to your chest, so you improve your pulmonary edema.

That's why you give morphine, as well, to people with pulmonary edema. Because morphine is a venodilator as well.

So it is good for people that have hypertension with pulmonary edema. That would be a good medicine to give there on top of another medicine. Or it is good if someone has hypertension with chest pain and you think they have an MI, because it dilates the coronaries.

Having said that, same problem with hydralazine and same problem with nifedipine. Dilates the vessels in the brain. So it's not a good medicine to use when you have cerebral edema.

Nipride? I don't think anyone uses nipride. I mean, there's people that use nipride still, but there's so many better options right now, and safer options. This is a great medicine. This is a super-efficient-- whoever has used it knows what I'm talking about. You start the nipride and the pressure drops within a minute. It's extremely effective.

It has a few problems. Same thing with nitroglycerin. Very potent cerebral vasodilator, so don't use it if someone has cerebral edema. Don't use it in myocardial infarctions, because it causes something called the steal phenomena. If this is the coronary that is occluded, it dilates the vessels adjacent to the ischemic area, so blood flow is going to go to the other places and worsen the ischemia in the part of the heart that has actually been hyperperfused.

The other big problem is that most of the textbooks, if you open them, they're going to tell you that the dose of nipride is 0.2 to 10 mics per kilo per minute, and that is incorrect. You should not use more than 2 mics per kilo per minute. 0.2 to 2 mics per kilo per minute. And then you have the issues with the cyanide toxicity, and the thiocyanate toxicity. In pregnancy I don't think anyone uses it, because there are so many other options.

If you happen to use it-- you know how cyanide is the main thing that you're concerned about, right? And cyanide, what it does is it inhibits your mitochondria and you develop lactic acidemia because you block the aerobic metabolism. You cannot be getting blood gases and see, well, if I have metabolic acidosis, then I'll stop it. That's too late. The earliest marker of toxicity from nipride is tachyphylaxis.

So if you have someone that has been doing great 0.3 mics per kilo per minute for one day or two days, and then suddenly now you're at 1.3, and in a few hours you've been going on a higher, higher, higher, higher dose, tachyphylaxis, that's the earliest marker of toxicity.

So then labetalol. Labetalol for sure, everyone here is extremely familiar with it. Remember that labetalol is a combined alpha and beta blocker. When you give labetalol by mouth, it is a three beta to one alpha blocker. So for every three betas, you block one alpha. So even though it's combined, it's more a beta than an alpha blocker. And

When you give it IV, that's even more marked. Seven to one-- seven betas per one alpha. So you're mainly giving a beta blocker with some alpha blocking activity as well.

Compared to hydralazine, it peaks earlier. Usually by 10 minutes you have your effect. And then the effect lasts less. Remember the hydralazine, the effect is going to last for up to 12 hours. this is going to last for three or four hours or so.

Those are theoretical concerns. If you look at the few articles comparing one to the other, there's no difference in outcomes. So you can use either or in the setting of preeclampsia.

The other thing is I don't know why the obstetrical literature says-- and check it if you don't believe me-- if you go there and you start looking at it, most people tell you maximum 220 milligrams per day, or maximum 300 milligrams per day. And I don't know where that comes from.

Because, for example, we use it a lot in neurologic emergencies-- ischemic stroke, hemorrhagic stroke, et cetera. And sometimes we drip them at 8 mgs per minute on things like that. If you see the ACLS guidelines, they tell you you can use from one all the way to eight milligrams per minute infusions. So I've never be able to find where this comes from and what the problem would be if you give higher than that.

Esmolol, it's a great medicine to treat tachyarrhythmia. It is not really a good antihypertensive agent. The beautiful thing about esmolol is that it is a beta blocker that has a half life of anywhere between two to nine minutes. So if you don't like what you see, you turn it off and it's gone in a few minutes.

And it is cleared in the red cells, so you can use it regardless of your liver or your kidney function. So it is very good to treat tachyarrhythmias. For example in obstetrics, if you have someone that has mitral stenosis. People with mitral stenosis, they don't like to be tachycardic. Or you have someone with diastolic dysfunction. Remember, when we talk about diastolic dysfunction in sepsis. If you know someone with that [INAUDIBLE] disfunction, that it's very hard to feel that ventricle and they are going at 140, they have pretty much no cardiac output. You need to slow them to five them time to fill. Same thing if you have a fixed injury in the mitral valve-- if you have mitral stenosis you need a long diastole to be able to fill that ventricle and go through that stenotic mitral valve. Does that make sense?

So slowing them is going to increase filling time. And esmolol is a great medicine for that. So the times we use it in OB-- where did I put the-- there we go-- that's actually where it becomes very useful.

Metoprolol, another beta blocker, this one is cleared by the liver, and then the effect is going to last between four to six hours. So the thing is that, if you are not sure about giving a beta blocker-- let's say you have someone that is postpartum. They bled, but you think you have already resuscitated them-- my advice would be, in the first 12 hours of surgery, don't beta block anyone, unless they are having an MRI or something, because what's the likely thing that that tachycardia is? The likely thing is that it's compensatory.

If you have someone that was perfectly healthy, goes to surgery, now is tachycardic, the likely thing is compensating for hypovolemia. But if you, for whatever reason-- say, this these patient has had three MIs and you are concerned that she is going to have another MI because she's tachying in the 140's-- and you feel that they're not bleeding but they could but likely not, then don't use metoprolol. Just put them on a drip of esmolol.

You start the esmolol, and you see. Blood pressure drops, turn it off. But if you give five minutes on metoprolol and blood pressure drops, it is going to stay down there for a few hours.

And then this is the medicine that we use the most for preeclampsia. We use probably the first time hydralazine-- one hydralazine, one labetalol. But if it doesn't touch them, then this is what we are using pretty much on everyone, which is cardine or nicardipine. And most ICUs, this is what they use for pretty much everyone as well.

It's a very safe medicine. It's not expensive. This is a dihydropyridine calcium channel blocker. So It's like a cousin of nifedipine, very similar.

It works mainly by vasodilating-- so dropping the systemic vascular resistances. And even though it's a calcium channel blocker, it doesn't affect the heart. It doesn't drop the cardiac output. It actually dilates the coronaries as well, and it doesn't have the problem of nifedipine of causing severe cerebral vasodilation.

So it a great medicine. You start it on an infusion at 2.5 milligrams an hour. And then every five minutes, you go up by 2.5's, all the way to 15 milligrams an hour. And if you have not used it in preeclampsia, use it. And you will see how, at least from the way it looks, it looks more efficient than labetalol or hydralazine. Yes, sir?

AUDIENCE: Do you need to do anything special in terms of monitoring [INAUDIBLE]?

LUIS DIEGO PACHECO: No, nothing. The one thing that I would say is, if you're going to use an infusion, at least where I work, we usually get an A-line because if we're going to use an infusion and the cuff is running-- you can have the cuff running every one minute. But remember that, at extreme pressure, the cuff is very reliable. So I would just get an A-line, but that's about it.

The only concerning thing about it, that is theoretical as well, is that since it's a calcium channel blocker, it might increase the chances of postpartum hemorrhage. So just know that it might cause some postpartum hemorrhage. But we use it all the time, and we haven't seen any problems. Yes, sir.

AUDIENCE: [INAUDIBLE] big concern-- I guess my only thing is that from what I've been taught, generally we don't [INAUDIBLE] a calcium channel blocker with magnesium sulfate. Most of our preeclampsia [INAUDIBLE] has sulfates. So what is [INAUDIBLE] to deal with that?

LUIS DIEGO PACHECO: Well, that's a theoretical concern. And it actually came out with nifedipine initially, saying that because magnesium is a calcium channel blocker and then you give another calcium channel blocker, you may end up with respiratory and so on. But that's a theoretical concern. And pretty much all the literature has actually shown that that's not true. And pretty much everyone says you can combine them without a problem.

This fenoldopam is a medicine that we don't use that much and didn't take up, so we're not going to talk about. This is a beautiful medicine. We don't have it. I'm sure if you talk to your intensivists, they may actually have it.

This is like a cousin of nicardipine. It is called clevidipine. It's also a dihydro calcium channel blocker. And it has a half life of one minute.

So you turn on. Don't like it-- turn off. And it is cleared by plasma esterases, so you don't need a kidney or a liver. Nicardipine, you need a liver because it is cleared by the liver.

So this is a beautiful medicine. I don't think there's anything out there in pregnancy but just for you to have it there because people that I know that have used it love it. But it's very expensive. So maybe you can also get them some-- with the cheetahs, you can add that.

Now what we're going to see is just look at some few cases that are actually real cases. And these are OB cases, actually, some from our place, some from other places. So this is someone that just comes in, preeclampsia, maybe has a seizure.

And after they have a seizure, you don't need to image them. That's not indicated unless there's something atypical. So this is someone that had a seizure and then developed cortical blindness. So then, at that moment, you want the image them.

And what you see here is this one is not that impressive. This is anterior. This is posterior. This is a FLAIR MRI. And you see these areas here that are bright? You can see better on the next one.

See here-- anterior, posterior. You see all these areas that are all white? So hyperintense lesions and FLAIR, this is what people call PRES-- Posterior Reversible Encephalopathy Syndrome. And this is how most preeclamptics look or eclamptics. And non-pregnant patients that have hypertensive encephalopathy, this is how they look if you image them.

And mainly what this means is, it's called posterior because it affects mainly the posterior part of the brain. But it can affect the whole brain. It can be anywhere but typically posterior.

Reversible-- because if you image these patients in a month, it's gone. And then encephalopathy because they have symptoms of encephalopathy-- the symptoms of eclampsia and preeclampsia. They have blurred vision. They have nausea, vomiting, a severe headache, visual symptoms and/or seizures.

So that's how it looks. When you do a FLAIR MRI, then you get these hyperintense lesions. So in terms of PRES, this is pretty much what we said.

If you look at this, MRI-- I'm not checking a call. I'm just seeing how much time I have-- in the brain, there is two types of edema, vasogenic edema and cytotoxic edema. Cytotoxic edema is the one you get from ischemia, when you have ischemic stroke. Vasogenic edema, this one is the one you get when you increase the pressure inside a blood vessel and you leak fluid out.

So that's what happens in PRES. That's called vasogenic edema. So it is mainly vasogenic edema, and it happens mainly on the posterior part of the brain because if you look at the circulation of the brain-- really simple. The way I think about it, a neurologist probably would laugh-- but there's an anterior and a posterior system. The carotid system profuses anteriorly, and the vertebrobasilar system profuses posteriorly.

The carotid system is very effective at autoregulating. It is very effective at constricting when you're hypertensive. That's why you get the visual symptoms and the scotoma, because it constricts so much, you get less profusion to the retina. And then you can't see. It's very efficient in doing that.

The posterior part, the vertebrobasilar system, is not that efficient at constricting. So when you're hypertensive, you're going to start seeing higher hydrostatic pressure back here. And if you have higher hydrostatic pressure, you leak out, and you develop that vasogenic edema. And that's what causes PRES.

So PRES, you can see it with preeclampsia. But you can see with lupus and with sickle cell. And you can see it with IVIG. You can also see it with some of immunosuppressants like calcineurin inhibitors.

How do you treat it? Well before, how you treat it, you know how 20% to 30% of patients with eclampsia, they have normal blood pressures, right? And if you image them, you can see the same thing here. So why is it-- because all this makes sense? If you are really hypertensive and you don't autoregulate and [INAUDIBLE], it makes a lot of sense if you have high blood pressure. But how do you explain it if 25% of people with PRES or eclampsia, they have normal blood pressures?

Well the thing is that, as we said, the autoregulation is not the same everywhere. It's different in different parts of the brain. So these might be people, actually, that they just don't autoregulate at lower blood pressures. And that's just something I read of someone who just made it up, probably, because it's just a way-- but there's nothing wrong with trying to understand things, even if it is not true. But it just helps you understand things, and then you can make the right decisions.

So in terms of treatment, the treatment of PRES is going to be just treat the blood pressure. Lower the blood pressure to whatever you want. Classically, in preeclampsia, between 140 and 160 and 90 to 100, 110-- but just lower the blood pressure. That's pretty much the treatment for it.

And if you suspect it, don't get a CT, because it is very hard to see it with a CT. Get an MRI because the MRI is the one that can tell you if you have cytotoxic edema from ischemia vasogenic edema from PRES. The MRI is the only one that can differentiate those two.

And those are the flavors of the MRI. The radiologist knows this. You can have a FLAIR MRI or a DWI or an ACD. It will actually depend.

But usually with the FLAIR, the one I showed you, you see bright images. If you do a Diffusion Weighted Imaging, it's going to be actually hypointense imaging. And if you do the Apparent Diffusion Coefficients, it's going to be a hyperintense imaging.

Whenever you have any injuries to the brain, you will never go wrong with either nicardipine or labetalol. And this is a very, very good article by an intensivist called Paul Marik about most of the things I'm talking to you. And you can find the reference there. So just use those two things whenever anything's going on in the brain. You will not get in trouble like you might with hydralazine or nipride or those kind of things.

This right here is a patient. This is not her picture, but we just had one of these a month ago. This is a patient who actually had preeclampsia, delivered, and then had the worst headache of her life.

So obviously, when you hear [INAUDIBLE], you think it's [INAUDIBLE] hemorrhage. But [INAUDIBLE] hemorrhage, it's more common with aneurysms. It's not that common.

She was not focalized. When people are not focalized, it is very unlikely you're going to find a hemorrhagic stroke-- like a vessel that just popped-- or an ischemic stroke because of excessive autoregulation. It is very unlikely. You should be focalized. You should be able not to move this part of the body.

This patient had just a severe, severe, severe headache, but she was crying in pain. And sometimes you see some patients that know that they're not-- eh. But there's some that you really know they're hurt. This one was.

So what we did was we thought it could be a thrombosis of the venous sinus. Thrombosis of the venous sinus can actually cause that. Or we actually thought it could be this.

We just called postpartum angiopathy. And this is actually an angiogram. And then what you see is you see how this should be like the normal caliber of the vessels, and see how there is diffuse vasospasm all around. So there is just diffuse vasospasm in the brain parenchyma.

And this can cause severe headaches. It can cause focalizations because you never know if this is the area of the brain-- I don't even know-- that actually makes you talk and now gets hypoperfuse, you might have focalization from this function of this specific area. You can get nausea, vomiting.

So you can get symptoms similar to eclampsia-- nausea, vomiting, severe headache, visual symptoms. You can or cannot be focalized, seizures. So this is called diffuse postpartum angiopathy.

You know how people are scared to give methergine IV-- like methergine, usually when you have someone bleeding, you give it IM? You can get this from methergine IV. And you can get this from cocaine or ecstasy or sumatriptan, the vasoconstrictors for migraine. So all these things can cause that, this diffuse vasospasm.

And that one, what we got on her was an MRA. We got an MRA, MRV looking for a clot in the-- if you suspect PRES, you get a plain MRI, an MRI, FLAIR MRI. Now if you suspect that someone might have a clot in the venous sinus, then you get an MRV, Magnetic Resonance Venography, because you want to go and look at the veins and the sinuses. If you suspect this, you get and MRA, Magnetic Resonance Arteriography.

Now MRA and MRV are commonly done with a contrast medium called gadolinium. And I don't know your radiologist. Our radiologist will never give gadolinium to someone who is pregnant, even though the ACOG says that if you need to, you can use it. But you can do an MRA, MRV without contrast. So all you do is just MRA, MRV, and then you will rule out this or a thrombi in the sinus venous.

Here, in terms of this condition, what you treat it with is you just need to normalize the blood pressure. Nicardipine is a very good medicine here because what you're trying to use is something that will dilate the vessels in the brain. And nicardipine nicardipine is a soft dilator there.

Now you don't want to use something that dilates a lot, because you may end up having, actually, worsening of the cerebral edema. And sometimes they even need like the neuroradiologist to go in through the groin, all the way up there, find the vessels that are occluded and do balloon angioplasty, just open the vessel. So this is a rare condition, but it can happen.

This is actually a preeclamptic patient in a coma and focalized after delivery. And the imaging actually shows intracerebral hemorrhage, right? This is actually an intraparenchymal hemorrhage, with extension into the ventricles.

This is the classical hypertensive bleed. It's called intraparenchymal or ICH, right, intraparenchymal hemorrhage. And it mainly happens in the basal ganglia. So here-- and we're just talking about blood pressure management.

So this can easily be your patient preeclamptic that gets a tube to protect the airway, and the imaging shows that. So you know it's an intracerebral hemorrhage. It is a hypertensive bleed.

So how do you treat the blood pressure? What do you do with the blood pressure there? If you look at the current American Stroke Association guidelines-- because this is a hemorrhagic stroke-- they tell you that you should not treat the blood pressure in these patients, unless the pressure is above 180 over 110. That equals an MAP of 130. That's even higher than what you usually use for preeclampsia, right?

You usually use 160 over 110 to start treatment. So that's what they say. Having said that, the hypertension causes expansion of the hematoma. So in the first 24 hours, like 38% of people-- if you look at this hematoma there, this hematoma will actually expand within the first 24 hours if you're hypertensive.

It doesn't-- you don't have to be a genius to understand that, right? If you're hypertensive, you have hypertension you're going to leak more blood. This is going to get bigger.

The reason why the American Society of-- American Stroke Association, I guess, recommends not to treat this is because initially it was thought that all this area was like a penumbra area. It was an area that was ischemic that needed a lot of blood pressure to maintain perfusion, because you have edema. It makes sense, right?

You know what cerebral perfusion pressure is? Cerebral perfusion pressure is the difference-- the pressure gradient between your mean arterial blood pressure or your ICP, intracranial pressure. MAP minus ICP. That's cerebral perfusion pressure.

Do you think this person has cerebral edema? Of course they have cerebral edema. This is a huge insult.

If you have this, and you're limited by the skull, the intracranial pressure is going to be really high. So to perfuse this, you need to have high blood pressure, right? Otherwise, you're going to not be able to pull blood into the cranium, right?

So that was the theory behind let them be hypertensive. There is a-- let's see where I have it here. I don't have the reference here. But there is a recent paper on the-- it was on the article.

There's a recent paper in the *New England Journal of Medicine* two months ago or so that looks at this and actually they say that if you treat the blood pressure-- like us, they randomize patients with these kind of bleeds into 180 over 110, just like the American Stroke Association says-- versus no, I want to treat it. And I'm going to keep the systolic below 140. There's no difference in outcomes, like you're not going to save anyone's life by doing that.

But there is-- some mild neural outcomes might be better, if you treat the blood pressure. So you can either not treat the blood pressure or you can treat it. And understand that you're not really saving anyone's life, but you can.

The 160 over 110 that you use for preeclampsia sounds kind of OK. It's like in between. It's not as high as the current guidelines.

It's not as low as what most studies actually show. And again anything in the brain use Cardene or labetalol. You will never go wrong there.

This is another patient actually that has a-- this is not a preeclamptic patient, but it is a pregnant patient that was focalized in a coma as well. And here what you can see is all this black area. That's a huge ischemic stroke.

That's not hemorrhage. Remember hemorrhage looks bright. This is just ischemia. So this is a huge ischemic stroke.

You see how the ventricle on this side is compressed and everything here? That's mainly because of all the edema. So ischemic stroke is-- what we just talked about was a hemorrhagic stroke. Ischemic stroke is different.

Ischemic stroke-- don't treat the hypertension, unless you're above 220 over 120. Because here it is true-- as opposed to the hemorrhagic stroke-- all these areas are ischemic. And you need to maintain perfusion. So you let them be all the way to 220 over 120. If they actually are higher than this, then you treat it.

This hypertension will eventually resolve in the next few days. And then we're not going to talk about TPA. But remember that TPA, if you need to give TPA to an ischemic, you would never give TPA to that stroke because that's a huge ischemic stroke. You give TPA, it will turn into hemorrhagic right away.

But if you were to use TPA during pregnancy-- remember that you can use it. There's tons of case reports where ischemic stroke for PE, where you use TPA, doesn't cross the placenta. So if it's an emergency, you might use it.

This is also-- and this is a patient of ours who had a fibromuscular dysplasia. And these patients tend to have aneurysms in different parts of their body. And this right here is a [INAUDIBLE] hemorrhage. and it usually comes from ruptured aneurysms. So if you, in terms of [INAUDIBLE] hemorrhage, there's a before and an after. If you have a patient that comes in because a [INAUDIBLE] hemorrhage from an aneurysm-- which are the majority-- if the patient has not been taken care of, the aneurysm has not been clipped or coiled, then you need to aggressively treat the blood pressure, and keep the systolic below 160.

So use labetalol or Cardene. Because you don't want-- remember that aneurysm could still rupture. You don't want to keep bleeders there.

Having said that, the peculiar thing about this disease is that once the neurosurgeon or the neuroradiologist takes care of this, either by coiling the aneurysms or clipping the aneurysm, and they come and they tell you, listen, it's taken care of. The aneurysm is secured. Then you let them be hypertensive and you do not treat the hypertension.

Because the complication that these people have is something called delayed vasospasm. The blood out there irritates the vessels. And it causes constriction and causes severe vasospasm. So you need to have high blood pressures.

Now these things are outside of pregnancy. I don't know exactly what I would do with the blood pressure because with very high pressures you have a risk of an abruption. So I would let them be hypertensive. If the strip looks fine, and everything looks OK, I would probably just let them be hypertensive.

And arbitrarily-- I don't know-- keep the systolic below 180 or something like that. Because if they were not pregnant if this happened postpartum-- and the pressure is 230 over 160, you let them be. You let them be because that's what the body is doing to maintain perfusion to the brain.

In terms of acute-- this is very, very common in obstetrics, right? You have someone that comes in hypertensive with pulmonary edema. So remember when they have pulmonary edema-- and this is not from infection, which is what we talked about earlier-- this would be cardiogenic pulmonary edema. It's a problem with the heart.

There's too many problems. It can either be systolic or diastolic. Systolic means, like let's say you have a dilated cardiomyopathy, dilated peripartum cardiomyopathy. Part of the left ventricle is dilated, and it has a very low ejection fraction-- let's say 10% percent.

And that patient comes in. He's hypertensive with a 10% ejection fraction and has pulmonary edema. So how do you treat that?

For the pulmonary edema part, you can give nitroglycerin and Lasix, right? But how do you treat the hypertension? Which agent are you going to use?

Here you don't really want to use a beta-blocker, right? Because you have someone with a 5,10% ejection fraction. So what do you want to do is use a pure vasodilator.

So you can use nicardipine. Or you can use nipride. Or you can use clevidipine. Or you can use hydralazine-- a pure vasodilator. This is probably what we will use the most-- like nicardipine with nitro, Lasix.

On the other hand, let's say this patient also has a problem in the heart. They come here with pulmonary edema. You get the echo.

The echo shows 65% ejection fraction. Everyone sees that and says, oof, it's not the heart. And they don't even read the rest of the report.

25% to 50% of patients that present to the emergency room with cardiogenic pulmonary edema-- 25% to 50%-- they have diastolic dysfunction. They don't have a low ejection fraction. This is what we were talking about earlier, right? It's the heart that is concentric hypertrophy, very strong muscle-- squeezes 70%, but cannot fill.

So if it cannot fill, and you're really hypertensive, then it's even harder to empty the small amount of blood that can fill that ventricle, right? So you start accumulating blood backwards, and you develop pulmonary edema. So look for systole and diastolic dysfunction. If you have diastolic dysfunction, the best things to use are going to be something that relaxes the muscle of the heart-- calcium channel blocker or a beta-blocker. And same thing-- nitro and Lasix to take care of the water in the lung, right? Yes, sir?

AUDIENCE: I thought that the medical literature showed that for diastolic dysfunction, people were treated with [INAUDIBLE] and ACE inhibitors.

LUIS DIEGO PACHECO: Yes.

AUDIENCE: [INAUDIBLE].

LUIS DIEGO PACHECO: Yes, it is. And that is when you are not acutely decompensated. When you come in with frank pulmonary edema and you're on a ventilator, or you're really really sick and you're [PANTING], you would never use a beta-blocker. It's contraindicated.

Once they actually get better, then yes, they need to go home with Corvalol, which is [INAUDIBLE] and with an ACE inhibitor. Because what happens is when they have this sick heart that barely contracts, the beta-blocker will slow the heart rate, increase the filling time. So we will increase the chances that the ventricle will fill and squeeze a little bit more. But that will be for maintenance not acutely. So in terms of-- if someone comes in hypertensive with an MI, obviously beta blockers are going to be the best option and nitroglycerin to help delay the coronaries.

Real fast, an aortic dissection-- if you have an aortic dissection, and you're going to treat the blood pressure, here you need to bring the pressure down really fast. You need a systolic below 120 really, really fast. Because this thing is just progressively dissecting through the wall of the aorta. And they are going to die otherwise.

You need to be careful because if you give a vasodilator initially-- let's say you give them hydralazine or you give them nicardipine only that-- and you don't block the heart, when you dilate it's going to be easier for the hard to squeeze. It's going to increase the stroke volume. And you're going to dissect more.

So beta-block the heart first and then give the vasodilator and use medicines that are short acting. Because if you don't like it, you just turn it off. Because these people can actually rupture this. And they're going to become very hypotensive.

So start esmolol, and then give Cardene. But use the esmolol first. Or you can use monotherapy with labetalol if you want. Because remember it's both an alpha-beta, even though it's more beta than alpha.

And we're not going to look at this. The last thing is this, which is going to be-- this is the last slide. If you have someone with a pheochromocytoma-- which can happen. It's not unheard of. And in pregnancy, that might be the first time you see them because with the kicks of the baby and the growth of the uterus it actually stimulates secretion of catecholamines from the super-renal. So you can actually see it.

If you have someone that is diagnosed with a pheochromocytoma, do not start them with a beta-blocker. Because what happens-- if you just give them say metoprolol or you give them any beta-blocker you choose-- if you block the beta receptors, then when you secrete the catecholamines, all that is available are going to be the alpha receptors. So you're going to cost severe hypertension from the alpha constriction. And you might have a stroke and die.

So you need to start with an alpha. Who cares if the patient is going to be tachycardic for a while? Start blocking the alpha receptors first. That's done with phenoxybenzamine, right?

So that's a pure alpha blocker. You start giving that, start like 10 milligrams BID. And over the next few days you start increasing to a max of 40 TID.

And then when you start giving the alpha blocker and the pressures look good you're going to see that one day the patient is going to start becoming tachycardic. When the patients starts becoming more tachycardic than the baseline, what is it? You have already blocked most of your alpha receptors.

And now the catecholamines are going to the beta receptor. And then there, you give them a beta-blocker. But the important thing to remember here is just don't use a beta blocker as monotherapy because you can give them very severe hypertensive crisis, OK?

And I think that's pretty much all I have for this. And just thank you very much again for the invitation. And I had a blast. Thank you.

[APPLAUSE]