[MUSIC PLAYING]

JAMIE JOHNSTON: My goals for today are really to talk about a topic, which I think is really poorly understood. And that's really to recognize the various types of sodium and water disorders. Not just sodium. And to discuss the therapy of sodium and water disorders.

Remember when we talk about hyponatremia or hypernatremia, we're talking about a ratio. We're talking about the ratio of sodium to the amount of water. And that sodium is a surrogate for intracellular potassium. Water distributes throughout all the spaces. So we're going to do a little bit of basic physiology. Very briefly on potassium-related emergencies and describing plan of treatment for potassium problems.

So the one thing is that hyponatremia and hypernatremia are water problems. And if you have edema, or hypovolemia, these are sodium problems. So let's go back to medical school here a little bit. How many APPs in the room? Good. Thank you, colleagues, for coming. You're the future of medicine, by the way, according to UMPC. It's true. Also based on statistics, Pennsylvania is the 48th worst state in paying PAs and nurse practitioners. Just thought I'd throw that out there. Sorry, Neil, I know I'm not supposed to make editorial comments.

If you go by total body water in a 70 kilo man, 2/3 of that is in the cells. Now what's the major intracellular cation? It's potassium. OK? But water freely distributes into this space. The other is extracellular volume, that's one third. And the major cation in that space is sodium. But water, again, freely distributes. If you look at extracellular field volume, 3/4 is in the interstitial space, and 1/4 is in the intravascular space. And basically what we're measuring when we measure sodium concentrations is what's in the intravascular space. And for men it's 0.6, and for women it's 0.5.

One of the things to remember is that in our population, in elderly population, most of the water is not as much as it is in the younger population because muscle mass decreases, and it's replaced by fat. And water doesn't go into fat. So again, this little thing. 2/3 body weight, extracellular space, 1/3 total body water is in the extracellular space. And one of the things to keep in mind is that in that intravascular volume, you have this little red thing, 15%. That is the amount of volume that is regulated. That is where sodium is regulated, that little tiny space. Water is regulated throughout the entire body. And if you look at that, those are the baroreceptors. And the baroreceptors, quite frankly, are in the spaces between the aortic valve and the afferent arteriole of the kidney. So that's the only space. It's really very small. It's only about 15% of 3.5 liters. So again, when you're talking about sodium regulation, that's where that occurs.

Now tonicity is constant when water and sodium change in the same proportion. So the slide right now recognizes sodium and water under normal circumstances. If I go and I change these parameters by increasing the sodium, total body sodium is increased. But if I increase the water, then your sodium concentration is still normal, but the patient's edematous. This is a sodium problem. It's not a water problem, this patient still has normal sodium concentrations.

If I go the opposite direction, and I decrease sodium, and I decrease water proportionately, this patient is now volume-depleted, but they are still eunatremic. They still have normal sodium concentrations. So we really can't tell what the total amount, or what the ratio of salt to water is just by looking at the patient. That's a laboratory measurement. So again, if you get tonicity changes, hypertonicity or hypotonicity, it's because the two parameters have not changed in parallel.

So in this particular instance, we increase sodium, but then we go and we increase water a lot more, that patient's total body salt and water overloaded with water in excess. And basically what you have is hypotonicity in this particular case. Or we could go the other direction. And this is somebody where the sodium concentration decreases. They've got diarrhea, they're vomiting. The only thing that they can keep down is fluids, and so they've lost both. But there is sodium loss is greater than their water loss, and so they're hypernatremic, but volume depleted. A case that we see pretty constantly.

Same thing holds whenever you look at hypernatremia. If you increase sodium, and you don't increase water enough, basically what you have is a hypertonic solution. This would be the case of a cardiac arrest. You're going in you're giving somebody a lot of sodium bicarb, a highly concentrated solution-- a lot of solute-- greater than the amount of water that you're giving, and basically have hypernatremia. Or you have somebody where the water losses and sodium losses are there, water losses are there, but what you've got is somebody that is exercising. They're not eating, and they've lost water disproportionately. Exercising in a hot environment, something like that. And those folks have hypertonicity.

So the whole thing I want you to remember out of this is hyponatremia and hypernatremia are primarily mismatches between salt and water. And that the major thing you have to go about correct these is water, in order to correct the problem. Not sodium. So let's go on.

So the definition, we really consider to be less than 135 milliequivalents per liter. It's the most common electrolyte abnormality that we see, are hyponatremia and hypernatremia. And because of that, I'm going to spend a great bulk of this talk on talking about those particular entities. Hyponatremia, this is a little bit different than what you may have learned in medical school or in nurse practitioner slash PA school.

We really break it down, now, to think of hypertonic, isotonic, and hypotonic. A hypertonic situation is generally when you have another solute present. And the one that's most commonly present is glucose. An isotonic solution is when you've got a substance that is occupying the vascular space that basically decreases the amount of water that's present. And hypotonic is true hyponatremia.

So an elevated glucose. We have all seen this. Someone that comes in-- one of my patients came in from the dialysis unit recently. Confused and so forth. The very first thing I did was check oxygenation and then glucose. She was off the scale, greater than 400, and by the time she got to the hospital, 811. OK? And of course she was hyponatremic, because the glucose acts as a solute and pulls water into the vascular space, and that dilutes out the serum sodium.

So all of you know this rule that you have to add 1.6 milliequivalents to the serum sodium concentration for every 100 milligram per deciliter increase of glucose over 100. Plain and simple, you always have to do this. This is not a salt and water problem. This is a glucose problem. Isotopic hyponatremia, we don't see this as much, but it's something to keep in mind whenever you have somebody that comes in. And they have hyponatremia. And it turns out, you look at their triglycerides, and their triglycerides are very high. Or you look at their protein albumin ratio, and they're gamma globulins are very high. And it turns out that these folks have a proportion of their water that is now taken up by these proteins.

And so when you do a normal lab with the SMA, what you're finding out is that you are measuring the amount of sodium in this aliquot and then dividing it by this aliquot. And so that is not a true measure. And the way around this, is if you have a gammaglobulinemia, or if you have hypertriglyceridemia, is you have to use an ion-specific electrode. And that's in the blood gas lab. So that's one of the things that you should always consider when you're evaluating these problems, is this a true hyponatremia?

And then finally we get into the pathophysiology of hypotonic hyponatremia. And there's a bunch of different flavors for that. The first one is number 5, which is where you have pure salt loss. And I'm sorry folks, that doesn't happen. It really only happens in the first episode of Star Trek, the original series, when there was a salt vampire that sucked salt out of people's bodies. OK. That's science-fiction, that's not the way I practice, I have not seen a salt vampire in this country ever.

One of the cases we get is this. Increased water. We also have free water access. That's a polydipsic. Someone who's drinking a lot of water. This particular case is the case of nausea and vomiting, diarrhea, where you're losing solute and just taking in fluids. Another free water access. Sodium and water excess in both, where water is disproportionately high, or a lot of water excess and some sodium loss.

So let's divide that down. So hyponatremia, we've already talked about the two categories that are on your left. Hypertonic, which is hypergylcemia. Isotonic, which is a pseudo-hyponatremia, and then we have the hypotonics. And this is where you do what I call the poor man's renal biopsy. Or when I'm teaching on the wards, I ask the medical students, what is the kidney thinking? And you can tell what the kidney's thinking by getting urine lights and urine osoms. That's really straightforward.

If the urine osoms are less than 100, and the patient is hyponatremic, then the kidney's doing what it's supposed to do. It's dumping the free water load. And those are your primary polydipsics. Most of the cases I see with that are in the psych wards. They are very entertaining stories. We've had to take toilets out of the room because patients were drinking out of the toilets. We had one patient who actually self placed a garden hose down his throat and was taking in fluid that way. The kidney can handle-- I love this statistic-- 18 liters of water under normal circumstances. A young kidney. Not an old kidney. An old kidney can handle about half that. But that person was hyponatremic because they were basically gavaging themselves with tap water.

Now the other one, which is the one that we see more of, is a urine osom less than 100, which is a situation where they're holding on to water when they really shouldn't be. They should be dumping the water load. And in one case, you have a hormonal abnormality. It's the syndrome of inappropriate ADH. Which actually is the most common cause, about 40% of hyponatremics in the hospital are SIADH. A tumor associated, medication associated, and so forth. And they will have a urine sodium greater than 20. But they'll have basically a urine that is too concentrated and has too much sodium in it.

Now if someone comes in, and the kidney's saying, I'm dry, I'm holding on to salt, and I'm also holding on to water. They're concentrated. That means they're hypovolemic. They've had a 15% reduction in volume, which is enough to cause the release of volume-mediated antidiuretic hormone. Now to give you an idea about that, that little stimulus is about 15 times higher than the salt regulatory mechanisms that are necessary to cause a change in the salt retention. In other words, a change of 1% in body volume will actually result in the activation of the Renin angiotensin aldosterone system, whereas a change of 15% is necessary to activate ADH to hold onto volume.

And the other situation-- whoops-- with that is the ones we also see heart failure or liver cirrhosis, and that's effective arterial blood volume. Now if somebody asks you what effective arterial blood volume is, it's that space between the aortic valve and the afferent arteriole. That's what they're looking at. So if it's under-profused, the pressure is low, and so forth, the baroreceptors are going to tell you to hold on to stuff. So those are your situations.

Now, the clinical manifestations of hyponatremia are under appreciated. If you drop down to a sodium of 131 to 132, it's going to cause changes that are consistent with being legally drunk. You will have cognitive changes that are associated with that. How many have done the thing on their Wii, the W-I-I, where they've gone and they've looked at their balance just to see how stable they are over their point of gravity? That's dramatically affected in this particular group of people. So what are we concerned about in the elderly population? Falls. So a sodium concentration of 130, 131 in an elderly person is something you're going to have to be concerned about. And those symptoms progressively increase as the sodium concentration goes down. It depends on chronicity and acuity. Mild fatigue, nausea, headaches, a change in cognition, change in balance, all the way down to seizures and coma, which is usually with abrupt changes.

For those of you that are my vintage as clinical practitioners, remember when we used to do TURPs? And we used to irrigate the prostate bed with water? And before we started using glycerated compounds-- I see a lot of head nodding-- and you'd send the patient down to get their TURP, it'd be 140 for their sodium concentration. And they'd come back with a sodium concentration of about 100. That quickly. Those people were in coma and seizing. Simply because the prostate bed is so vascular and absorbs a lot of water. And the amount of irrigant that they use is enormous.

So your general treatment for this, if it's severe, if you have mental status changes, if you have someone that is really bad, they need to be in the ICU. And you need to bolus them with 3% saline. Small amounts, but you really want to try to get them out of trouble quickly. These are usually acute changes and they can be corrected relatively acutely. And you have to check the sodium concentration frequently. Every one to two hours.

I work at a quaternary institution, known as Presbyterian University Hospital. I have to fight sometimes to get my patients into an ICU bed. But this is standard of care based on nephrology practice. These people need to be in ICUs. And if I have to fight with the AOD, I'm going to fight with the AOD. Moderate-- the same thing. If they've got lethargy, confusion, and so forth, they need to be in an ICU. You have to give them a slower infusion of hot salt 3% salient to keep them under control. And if it's mild or asymptomatic, they're 130, 131, you can correct these on the floor, go after what's causing the problem.

So what about this problem in our population, in the elderly? It's about 8% in the community. And it increases with age. The older we get, the more frequently we have this. And so in a Rotterdam study from 2011, about 12% of folks over the age of 75-- I'm sorry folks, this is stuff that as I told Neil, I constantly revise my talks, so I submitted the original slides on 3/25. And I keep working and working and working on my talk, so this stuff has been added. So the take home message in this slide is hyponatremia increases as we get older. For a variety of different reasons. In the population that Neil alluded to earlier, it's about 18% of folks in nursing homes are hyponatremic when they're over the age of 60. And in hospitalized elderly patients over the age of 65, hyponatremia was seen in a third of patients. The reference at the bottom, Clinical Interventions in Aging, 2017, that is a wonderful, wonderful reference. So Clinical Interventions in Aging, 2017, Volume 12, page 1957.

The causes. The elderly have increased levels of antidiuretic hormone. They do not osmo-regulate properly. Their on a lot of medications, thiazides. All of you know this phenomenon of the tea and toast diet. Their appetite decreases. They have an increased incidence of SIADH, but most of them have multi-factorial causes. And what decreases with age? What leads them to this? Well their GFR goes down. During this talk you will lose one nephron. OK? Sorry guys, it's the speaker's fault, you're going to lose an nephron today.

But basically we lose about 1% of renal function for every year over the age of 30. So that by the time you get to be 80, you're at 50% renal function. You can do OK with one kidney, but what it affects is your ability to excrete a free water load. So instead of being able to get rid with 18 liters a day, you can only get rid of nine liters a day. And then your elderly patient reads one of America's newspapers-- not one of America's great newspapers-- one of America's newspapers, the Pittsburgh Post Gazette, and someone says that you should drink eight 12-ounce glasses of water a day, which is total horse hockey pucks. They've proven time and again that drinking a lot of water makes you pee more, but it doesn't help your kidney function.

There's two cases where you drink a lot of water. If you have kidney stones or recurrent urinary tract infections. Tell people to drink to thirst. Their ability to excrete water goes down, their body water changes because of the increased fat content. And because of the increased fat content, what you've got is small changes in water have greater effects on sodium concentrations, because there's a decreased volume of distribution. And then prostaglandins go down as well, can be inhibited by medications, which causes changes in the way that you hold on to water. And then there's increased sensitivity to osmotic stimuli.

I was taught that this was a reset osmostat Older folks just handle water differently. And some of them will go and reset their osmostat down to 126. That's the lowest I've ever seen. Are you going to go after that? Well you can do cognitive testing, balance testing, and so forth. And if they're OK, then you probably should leave it be. And not treat the number, treat the patient. The drugs, thiazides, the last talk alluded a lot to psychiatric drugs. The SSRIs are a direct cause of SIADH. Your serotonin norepinephrine reuptake inhibitors as well, non steroidals, and a variety of diseases that of course we never see in our elderly population. Infections, diabetes, congestive heart failure, liver failure, and renal failure.

So here's a case. I like giving cases. This is a case that I saw during my brief unhappy time in private practice. One year, three months, two days, 15 hours, and 35 seconds. I was asked to see an 83-year-old woman, and I was told when I came on the floor, they said, oh, you should be honored. This is one of our best internists, he never asked for consultations. He's asking for help. He never does this.

So I go into the room-- the console was for hyponatremia-- and there's this really sweet old lady sitting in bed with her feet dangling. She's sitting up, and she's tripodding. Hands on knees, and she's in a little bit of respiratory distress. Her ear lobes are going up and down from her JVD. She has HDR, which makes her hair curl and uncurl. And she's got Anasharca. And I mean, even her eyelashes were edematous.

And the very first thing I noticed that when I walked in the room was that she had 3% saline hanging. 3% saline hanging. So is she volume overloaded? Yeah. Does she have too much salt on board? Yeah. She has way too much salt on board. But she also has way too much water on board. That's the thing to remember. Her sodium was 114, her urine sodium was 5, because she was in congestive heart failure, and her kidneys being stupid, thought I got to hang on to stuff because I'm under-profused. And her urine osom, she had volume-mediated ADH release. So what water disorder does this patient have? Well she has hypotonic hypervolemia. Her total body salt and water are overloaded, but her water overload is much greater than salt.

So did she need more salt? No. What she needed was salt and water restriction. She needed to be diuresed, and she needed something to make her heart work better. This was one of the most challenging consult notes I ever had to write, because I could not write something along the line of renal physiology does not work this way, you-- bup bup bup bup bup. But I was good, and I continued to get consults from this particular individual.

So here's another one. This is a 75-year-old man that you're seeing for hyponatremia, he complains of dry mouth, he's on an SSRI and a thiazide. His serum sodium concentration is 114, his urine sodium is 45, and u-osom is 65. He has no edema on exam. He has normal axillary sweat. Things that people miss, your suppine patients, nobody checks back here in the sacral area for edema. I was never taught this in medical school, I learned this when I was a farmhand as a teenager-- is water runs downhill. You go to the lowest place in the body to look for edema. He had no edema in that particular place. And what does this show you? Well he's hyponatremic, his urine sodium is the normal range. So he's not retaining sodium. His urine osom in his 65, so he's trying very, very hard to basically get rid of water.

So what's going on? What water disorder does this patient have? Well he has hypotonic euvolemia in his total body water overload alone. And this is somebody that really fits into the category of increased thirst, secondary to his psychotropic drugs. And that's what's causing the problem. You also have to think that the thiazide diuretic is contributing to this as well. And so what needs to be done in this particular case, like our last speaker, you have to water restrict them, and you have to stop his medications.

Now is this patient truly euvolemic? Well the answer to that is on physical exam, he's euvolemic. But anyone want to guess about what the minimum amount of fluid that you need on-- or volume you need on exam in order to determine someone's volume overloaded? The salt and water distribute throughout the extracellular space, so you need to be three to five liters overloaded before you can even detect ankle edema or sacral edema. That water gets distributed into the interstitial spaces. The definition of this is clinically euvolemic. You can't detect the edema on exam.

And then finally, we have a 62-year-old man. Hyponatremia, five days of nausea and vomiting, only tolerating fluids. He's read that drinking Coca-Cola, Pepsi Cola, and Gatorade is really good to replace electrolytes. There is a certain farm product that terminology really applies to, something that comes out of the back end of a cow. Anyway, anybody speak amorous lamb sign language? So if you are in a conference like this, and you go like this, that's bullshit. OK. Just so you know that, little thing for you to communicate with your patients.

The only thing that you can buy over-the-counter that's going to help replace electrolytes is Pedialyte. And it comes in multiple flavors to get the kids to drink it. All the fraternity brothers know this, they drink Pedialyte after binging on Saturday. But Gatorade and stuff is totally useless for replacing electrolytes. This patient had orthostasis, he had dry mucous membranes. He had no axillary sweat, which actually on exam for volume depletion, the lack of axillary sweat, I know it's distasteful, but that's the most associated with decreased volume. And he was dry. Sodium concentration was 114. He was holding onto sodium like crazy, his urine sodium was five, and u-osom was 700.

So what disorder does this patient have? He has hypotonic hypovolemia. And it's salt and water loss, with sodium loss greater than water loss. And you'll notice the way I put these. You'll always have to look at the relative losses of both things in the ratio, salt and water. And what needs to be done? This person needs basically, IV fluids. If he's in the unit, lactated ringers, if he's on the ward, isotonic saline.

So treatment of hyponatremia as your primary polydipsia, he has too much water, that's going to be fluid restriction. Hypovolemia, you're going to volume resuscitate the patient. Hypervolemia, you're going to use-- for heart failure and liver cirrhosis-- fluid restriction, loop diuretics, and you're going to try to use vasopressin receptor antagonists. They're relatively controlled in a hospital setting, but sometimes you have to use them. I rarely use them. Most of the time, I can get out of trouble with fluid restriction. Or loop diuretics.

The goals of correction and the limits of correction are different. Your goal should be about 6 milliequivilents over 24 hours. That's the goal. Your limit is no more than 10. And the reason for that is because of the osmotic demyelination syndrome, or central pontine myelinolysis. If you correct this too quickly, you're going to wipe out the patient's pons. And if you wipe out the patient's pons, you're going to kill him. And the onset of this usually is two to six days after over-correction. I've seen this happen three months after over-correction. So this is really something where the limits of correction and the goals of correction have to be adhered to.

Patients have neurologic symptoms with this. Dysarthria, dysphasia, quad repeat harasses. About half of my patients that I've seen this in die. And this is becoming historical now, because we really pay very close attention to this. Diagnosis is clinical, the MRI may help. And the prevention is re-lowering the serum sodium concentration. And what you do if that happens, if you go more than six in 24 hours, you liberalize their water. You can give them D5 in water, or you give them DDAVP. You're giving them exogenous diuretic hormone in order to make them hold onto water and try to get them back. But it has to be slow.

Now we're going to switch gears a little bit here and go to hypernatremia, which we see-- when I was in private practice, I saw this once a week-- and I'll bet you you know where I saw it coming from. They were nursing home patients. That's where I saw it. It's always associated with an increase in plasma tonicity. It's usually chronic. It's very, very common in nursing homes and now-- I'm not sure, maybe you folks can help me with this-- there are many states that if someone is admitted with hypernatremia at a hospital, it's considered to be a reportable offense. Because it reflects substandard care. I am unaware of that in Pennsylvania. But it is something that maybe if you moved to California, because this sounds like a California regulation.

This is Clinical Interventions in Aging, 2014. And it's volume number 9, 1987, another really good reference. Common in the community, 3.7%. The geriatric population, if you're admitted with fever about 30%. And about 15% can develop hypernatremia in the hospital. The elderly patient at the end of the hall that the nurses aren't seeing as frequently, and so forth. And they can't reach the water on their bedside table. And in the elderly-- this is one of the things-- it doubles your mortality. It goes from 15% to 33%.

Again, it's because of loss of renal function. Just like we're not able to get rid of a water load, we're not able to maximally concentrate our urine. So for a healthy youngster they can go to a maximum osmolality of about 1,200, and an elderly patient over the age of 65 can only concentrate their urine to 700 to 800. So basically an elderly person has a loss of renal opportunity. They may only be able to dilute down to 150 to 200, and they may only be able to concentrate up to 700, 800. So their water handling is really affected. They don't respond to ADH, and again, they have lower total body water, which affects small changes, cause big changes in serum sodium concentration.

So these are the situations. Pure salt addition with water loss doesn't happen. Pure salt addiction doesn't happen. Case brought up whenever Pittsburgh was a steel town, I used to see hypernatremia in the guys that were working the open hearth. Because US steel would put bowls of salt tablets out and say, you're losing a lot of salt through sweat-- sweat is actually hypotonic, especially in conditioned athletes, it just tastes salty because we can really taste salt, it's something we seek-- and they would come in, and they'd be drinking water, but they would be taking these salt tablets. The amount of salt in a teaspoon full of salt would amaze you. If you would figure it out. It is a lot.

So you can get very hypernatremic very quickly by this particular situation, where you're taking in a lot of water, but you're also taking in a lot of salt, that's number two. The second one, number three, is in sensible losses. And sensible losses are usually respiratory losses. And with respiration, you can lose as much as a liter an hour. Just by exercising in a hot environment. Just through respiratory losses. And then finally, salt and water losses, but with the water losses greater than salt. These are your so-called free water deficits.

Now it only occurs when the patient is unable to drink or they have disrupted thirst. Again, I'm going to repeat that. It only occurs when they have disrupted thirst or when they have no access to water. And so if you're in a nursing home and you're dependent on the help to give you a drink, if you have taken a lot of drugs and you're comatose, and in Pittsburgh parlance, you're found down. That's that situation in the middle, and then there's the unhappy child at the end that's dependent on the parents, and can't turn on the faucet in the tub. Looks like my granddaughter.

Hypernatremia, again, urine osoms, urine lights are your friend. If the urine osom is greater than 600, then the patient is either hypervolemic because they've been coded and they've gotten a lot of sodium bicarbonate. And they'll concentrate their urine and try to hold onto water. Or they're euvolemic, and they've just had pretty much free water loss. If they're hypovolemic, that's abnormal extra-renal water loss is shown in the middle column. It's either a GI or a skin sores, vomiting, diarrhea, fistula, excessive sweating or burns. Burns can be really bad for fluid losses.

And if the urinary is less than 600, in other words, if they are hypernatremic and they're not holding onto water, something is wrong with the brain, or something is wrong with the kidney. So that's either central DI or it's nephrogenic DI. And the test for that is to get them DDAVP and see if they respond. If they respond, it's central. If they don't respond, then it's a kidney problem.

So this is another case. This is a 69-year-old woman for hypernatremia. Unresponsive, found down at her apartment. She had several empty pill bottles near her. Last seen by friends three days ago. This was an interesting case. The patient was vacationing in the Caribbean, and they life-flighted her to Pittsburgh. Now the last time I checked, there are hospitals in Miami, Fort Lauderdale, Tampa, Atlanta, but they flew her to Pittsburgh.

And basically, on exam, she had no blood pressure. What they had not done was just absolutely amazing. Dry mucous membranes, decreased skin turgor. So dry? Volume depleted? Yes. Yes. Serum sodium was 175, not the highest I've seen. Her urine sodium was really undetectable. The kidneys were trying very, very hard. And her urine osoms were 1,100. So for a 69-year-old to do that was truly amazing.

By the way, the highest serum sodium concentration, I've seen is 215. It was a mental patient that was found down in the woods next to the mental facility where she had escaped, and it was in the middle of summer. And she'd been out there for several days. And she passed. We weren't able to save her. The highest one that I've been able to save was 193. And that was in RVA. And they had not been given fluid for 10 days in the VA.

So these things happen. A lot of time it's iatrogenesis imperfecta. Have you ever heard of that? It's our fault? Yeah. So hypertonic hypovolemia, that's what this patient has. In sensible water losses, and what needs to be done, isotonic saline. You correct the volume abnormality first. This patient needs volume. Otherwise you can't profuse the kidney. You worry about correcting the sodium concentration abnormalities second, and then you calculate the free water deficit and give hypotonic fluid, which in this case could have been a normal saline. 155 milliosoms.

This is the free water calculation. I highly recommend that you get one of the things, the apps, to put on your phone. You can plug it in, and you can plug all these numbers in, you don't have to remember the formula. I hate remembering formulas. It's like Einstein. I don't remember anything that I can look up. I'm not Einstein, but I don't remember anything I can look up. And then you replace normal water losses, which is about 500 mLs, and replace excessive water losses.

You have to do quick iNOs on these folks. If they have a lot of ileostomy losses-- one patient, they had an open abdomen, and they were doing repetitive packing. And they were just soaking the surgical gauze and surgical dressings with fluid. And that was what the fluid losses was. We had to guesstimate that by weighing the dressings. Find out what they were lost. And again, correct by no more than 10 milliequivilents per 24 hours.

Homestretch. That actually is metallic potassium. I always put that in because I think it's really pretty. 99% to 98% of potassium is in the cell. It's only 1% to 2% in the extracellular space. As all of you know it's necessary for nerve conduction, and the biggest complications you're going to see with this are cardiac arrhythmias. Hypokalemia will get ascending paralysis. They'll come in, they'll have leg weakness. And then finally they'll get up to where they're having diaphragmatic weakness and respiratory distress, as well as cardiac symptoms. And then hyperkalemia, which the big thing for that is actually sudden death.

Deficiencies are usually due to poor intake. Do our folks in nursing homes have eating disorders? Absolutely. They have decreased appetite, they might be demented. I always tell my families that whenever your family member stops eating, that's really a bad sign. Because that means the downhill slide is going to occur very, very quickly. And those folks have just decreased intake of potassium compounds.

Your kidney is really lousy at holding onto potassium. Because historically, evolutionary status, our potassium intake has always been very high. A lot of fruits and vegetables. So our kidney is really good at getting rid of potassium, but not really good at very holding onto it. And then you can have increased losses as well with GI losses, bulimia, and so forth.

Rules of thumb to get you out of trouble. You should always try to correct potassium up to 3.5. Why is that? Under 3.5, your blood pressure starts to increase. Under 3.5, your chance of muscular weakness and cardiac abnormalities start to increase. As a rule of thumb, if they're less than three, that's a total body potassium deficit of about 100. If they're under two, that's a total body potassium of less than 200.

You can give K about 10 milliequivalents per hour on the floor. Really be careful with this, potassium is extremely caustic. It can cause burns and sclerosis of the veins. If someone's really in a lot of problems, put them in the unit, put in a big central line, and you can go up to about 20 milliequivilents per hour. In addition, you should be using oral supplements.

Hyperkalemia, a lot of the times I see this, is because of shifts. Again, the increased glucose in the blood pulls more water out of the cells and with it, potassium. And then metabolic acidosis causes hydrogen to go into the cells and potassium to come out. And kidney failure is where you're going to see this the most. In an end stage renal disease.

I've had patients where I've dialyzed them, I take them down to a potassium of three. They go back to the ward and the eager house staff goes and gives them a potassium infusion. And then I have to dialyze them again four hours later because now they've re-equillibrated, and they're potassium is now seven. OK. It takes about six hours after dialysis for your potassium to reequillibrate. So you really should not be measuring potassium within the first six hours after dialysis. And if you get a hyperkalemic patient, always recheck the potassium, and always check an EKG.

So my last cautionary tale, and I've been beating this drum now for close to a decade, and I'm going to continue to beat it. This was a 77-year-old that we saw. The consult was for hyperkalemia. Her K was 5.3. This is not a significant hyperkalemia, but we were asked to see this patient. She'd been status post-colectomy for Crohn's disease. She had a post-op ileus. The house staff gave her kayexalate with sorbitol. For a K of 5.3 with no EKG changes.

So we were called in after the fact, and what we found was a patient with abdominal pain and tenderness. She had a rigid abdomen. She had free air in her abdomen, and she had perf'd. And then she went on to an emergent x-lab and had a small bowel perforation. And one of my colleagues, Dr. Ochoa, who's no longer here and I dearly love, sent this to path and this is what we found. These are sodium polystyrene, this is kayexalate that is actually in the area of the perforation. The sorbitol is thought to cause a hyperosmotic stimulus, decrease renal blood flow, and leads to tissue necrosis and bowel perforation.

So my advice to you is never use kayexalate in anybody that has any type of gut motility issues. And you should probably not use sorbitol with it. The benefit is small. So did this patient need to be treated? No. The treatment for emergent hyperkalemic should be based on EKG. These are other emergency treatments, which all of the you know. IV insulin, and IV glucose. Albuterol to shift it back into the cells. Bicarbonate to shift it back into the cells. Calcium to stabilize the voltage across the membrane. And eliminate it from the body with a loop diuretic or K binder. If you asked for dialysis, that's going to take me two to four hours to get it set up, especially if the patient doesn't have a catheter. So remember you should know these ones first.

The new stuff, not so new, Patiromer has now been out. If you have someone's who's chronically hyperkalemic, Patiromer is the stuff. It on average lowers you down by about a milliequivalent per liter. The average lowering for kayexalate is 0.3. OK. Patiromer is much better. And the other new kid, this is ZS-9, which the FDA has just approved. It's another chelater, and it's another good one. They're expensive, you might run into trouble with formularies. And with that sunny picture from the Oregon coast, I'm done. And I think I have a couple minutes to answer questions. Thank you very much.