The talk today is going to really be focused on recognizing common errors and treating electrolyte disorders, how to treat them and knowing when to ask for help. There’s a lot of misconceptions about salt and water balance and potassium and water balance and where potassium is and you have to consider all those things together; salt and water and potassium and where it is whenever you’re treating these problems. And the format that I like to use because I was just talking with my friend Jay is I like to tell stories because I think we remember stories and because I hated the first 2 years of medical school sitting in a lecture room and I really liked the last remainder of my career when I did nothing except take care of patients so it’s going to be case discussions. And I limited this year to sodium and potassium as the talk.

So just as an overview, yes this is an actual sign in elderly people walking to the cemetery. Yeah I really could not resist. But 81 million by the year 2050, in 2050 I will be 97 years old and God willing and the river don’t rise and the most common problems that we’re going to see are going to be hypernatremia and hyponatremia. The one thing is that there’s a lot of decreases in kidney function in the elderly and the one thing to remember is that a decreased GFR is the norm. Even though my 84 year old, God bless her, had a creatinine yesterday of 0.4, she still only has a GFR of about 60 because just through the aging process she’s lost about 1 ml per year after the age of 40.

The other thing is that the opportunities of going all night and concentrating your urine, you don’t do that when you’re older so you get up in the middle of the night to pee because you can’t concentrate your urine. You can’t dilute it either so you can’t take water loads. Your ability to get rid of acids and potassium goes down and the big thing is if you go and you’re 18 and you drink 2 liters of water
you’re going to get rid of that in about 2 hours, that would be typical. If you’re 80 years old it’s going to take you probably 6-8 hours to get rid of a water load and so the risk of hyponatremia and electrolyte disorders is a lot more common because of the loss of the ability to rapidly adjust.

So the first case, and again, everyone that was here before has a leg up because this is a case I presented before so see if you remember what we talked about. Average retention a week after a lecture is 7% and half of that percentage is what the presenter was wearing, so I have a black suit and a red shirt and a red tie. So an 80 year old comes into the emergency room, she complains of weakness and muscle fatigue. Past medical history is pertinent for hypertension and osteoarthritis. She has recent generalized joint pain and like I’m doing right now which is medicating for my radicular pain, she’s using an over the counter non-steroidal and has been doing so for 10 days prior to admission. She’s sick whenever she comes in and was very, very weak. The medications that she was on, Amiloride, HCTZ, Lisinopril and a non-steroidal anti-inflammatory, her hemoglobin and hematocrit 10.2 and 30.9, BUN is 92, creatinine is 2.5. Notice her baselines, 68 and 1.3. Her K was 8.5. Okay, one of the reasons why she was weak. She was acidemic with 7.24 pH, this was a calculated bicarb of 14.6, glucose was 110 and this was the cardiogram that we got in the emergency room. And unfortunately what she did in the emergency room as we were standing there was to do this, she went from these nice talk peak waves and broadening of the QRS complex and started to go into a sine wave pattern. Now I’m a kidney doctor but I know enough about cardiograms that this actually got my attention. So this is the thought process that went through my head. Calcium gluconate 10%, slow push over 2-5 minutes, IV insulin and IV glucose, beta adrenergic, inhaled albuterol, IV bicarbonate, sodium polystyrene sulfate and a renal consult for immediate dialysis.
Well I was already there so F is no longer a choice. So the question you should ask yourself is what did I do?

And of course you can see this because of the slide format but what I did was I gave her calcium gluconate, a slow IV push and if you’ve never done this, this is one of the most wonderful things in the world. You feel actually kind of like God like whenever you do this because the EKG goes and it goes out from a sine wave and it suddenly compresses down back into a normal EKG and it is very, very quick. Of course you have to stand there and watch because it’s a temporary effect, it may only last an hour so you’ve got to do other things and that’s why the next part of this is to start other medical maneuvers.

So first thing, this is not the first thing we did, we were not going to give her amiloride an ACE inhibitor or a non-steroidal any more and we were really not going to stick a catheter in her and start dialysis because even at UPMC in the Acute Dialysis Unit where we are currently averaging 1,000 dialysis treatments a month, acute dialysis treatments, it still takes a little while to get a catheter in, check the chest x-ray and get somebody on dialysis. About the fastest we’re able to do it is 2 hours. So if you wait for 2 hours this lady is going to be dead, so you have to use your medical maneuvers. The albuterol is a shifter, it pushes the potassium back into the cells. The IV glucose and the IV insulin please give the glucose first, then the insulin, okay. You really want to make sure you don’t make the patient hypoglycemic acutely, that shifts it back in. Then the Kayexalate is a removal agent. One of the things I added this year was cautionary tales about Kayexalate so I really want to make that point. So you have to prioritize, what do you have to fix first and how quickly can you do
it. In this case this was life threatening hyperkalemia, recognize the underlying problem and in this particular case the patient was taking medications with impaired renal function of BUN 68 and creatinine of 1.3 in an 80 year old is about 25% of normal renal function so you have to be monitoring lytes, cautioning the patients about what to take and the next section is the (inaudible) of creatinine and potassium.

Potassium you get rid of primarily by the kidney. Interestingly enough 10% of your potassium goes out through your colon. And the same drugs that affect kidney excretion, affect colon excretion, it’s the same channel. So if you give somebody basically amiloride and block the epithelial sodium channel, they are going to have more difficulty reabsorbing potassium in the colon. So that’s one of the things to keep in mind whenever you affect it. And the colon becomes much more important as renal function decreases. So you’re elderly patient that’s constipated is going to have a little bit more difficulty with potassium homeostasis. 98% is in the cells to give you an idea, blood 4 milli the colon some potassium per liter, a pound of flesh, meat, okay is 150 milli-equivalence per liter. So if your elderly person comes in, falls down the stairs and they have mechanical trauma to their muscles they’re going to release a lot of potassium into their system.

The other thing is if the patient is acidemic as our patient was, hydrogen will go into the cells and potassium will come out and that’s a shift phenomenon that occurs and you have to be aware of that. The components of the evaluation what’s the kidney function, what’s the acid base status, and is the glucose elevated because of the shifts that occur. You can use Cockcroft Gault practically nobody uses Cockcroft Gault anymore, but I use multiple estimates of renal function primarily because none
of the estimates are really good for patients over the age of 65. MDRD is not validated for patients over the age of 65, I want you to remember that. All your estimated GFRs and all your labs are calculated by using the modification of diet and renal disease formula and it is not valid for patients over the age of 65 nor is it valid for someone that’s cachectic from cancer, a liver failure patient, because their muscle mass is not the group that the group that it was validated in so be very cautious about that. I use rough rules of thumb. She’s 80 years old, she’s lost 1% of her function since the age of 40 therefore out of the dock this lady has a function of 60 ml per minute. Then I look at the creatinine and I adjust based on that. Her normal creatinine should be about .6-.7, she’s 1.3, I now divide that estimate of 60 by 2. This lady came into us with a GFR of 30 ml per minute. A quick and dirty and you’ll notice, yeah I know I wrote the slide so I knew what the answers would be, but I didn’t construct the answer that way. But by MDRD she has an estimated GFR of 33 and by Cockcroft Gault she’s 27. So this is a lady that based on the estimates has renal insufficiency but just eyeballing it through a nephrologist’s eyes she’s got a problem as well. And this is why that happens.

As we grow older which I am well aware of, I do not have the muscle mass that I had when I was 20. Only Dr. Resnick has the muscle mass that he did whenever he was 20. And what has happened over the years as I’ve gotten older is that my creatinine production has gone down, but it’s gone down in parallel with my renal function. So the equilibrium point, the creatinine doesn’t change and that’s one of the things to remember. So you can be fooled by the serum creatinine.
This is an old article by Lindeman that shows what I’ve been quoting, that after the age of 40 creatinine clearance falls by .87 and you can see that up to about the age of 40 we have a slight decline and then we really start to take off. And the creatinine production normalizes between men and women by about the age of 65. Then by the age of 80 you’re really only making about 10 mg per kg of creatinine. Arnold Schwarzenegger in his prime was probably making about 28 milligrams of creatinine per kilo. Now I think he’s a little bit less and I keep seeing pictures of him in a bathing suit. The effect of the non-steroidal which as a nephrologist I also think about all the time is it decreases prostaglandin production and if you, the primary effect is to decrease renal profusion, and if you’re dry you really decrease renal profusion so this can precipitate acute kidney injury.

I tell my patient if they’re taking non steroidal that they should be drinking about 8 ounces of water every time that they take it and they should not be restricting their salt intake whenever they’re on that particular medication. The result of the renal failure is they lose the ability to excrete potassium, they can’t retain bicarbonate, they get metabolic acidemia and they shift K out of the cells. So other medical maneuvers the IV insulin and IV glucose we talked about, inhaled albuterol, IV bicarbonate is another shifter. You really want to get the K out and since 1964 when this drug was grandfathered in based on one study we’ve been using poly styrene sulfonate or Kayexalate and it binds K in the gut, it also gives a sodium load and remember for every K that you bind you get 1.6 sodiums so you can throw patients into failure with this. It is generally given with sorbitol in order to get a diarrhea because Kayexalate can cause concretions and get patients into problems. Kayexalate should not be given to anybody that has decreased gut motility and I mean decreased gut motility with the slightest hint or suggestion of decreased gut motility. Because what happens is the sorbitol causes local
hyperosmotic changes, it decreases blood flow to that area, and you get bowel perforations. It is very common in someone that has an ileus to get a bowel perforation with Kayexalate. So if this patient comes in, they’re tympanic they haven’t moved their bowels for 4 days, they said that they have been nauseated and vomiting, this is not a patient you give Kayexalate to. The slide over here, so this is the mucosal surface, the luminol surface of the bowel and these little crystals are Kayexalate crystals in an area of bowel ischemia damage. We write these up this is a big patient safety issue for us. Anybody know what the average lowering of potassium is with 30 grams of Kayexalate? .3, so this lady would’ve gone from 8 down to 7.7 it’s not a good idea and she’s hyperkalemic which means that she has motility disorders as well because of hyperkalemia.

So in this particular case we really do not use this as much. There’s a new drug on the market known by the interesting name of Z which is how we’re referring to it. It seems to be much better and not going to do this. If you have to give Kayexalate give it with water. Do not give it with sorbitol, sorbitol seems to be the bad actor.

The dialysis, dialysis is great, I can take off a lot of potassium very quickly generally I can get people from 8 down to 4 in 2 hours but it takes time to get it started. The other thing you’re going to do is educate the patient a low potassium diet and stop the medications. You would give this patient volume and a diuretic, there’s 2 effects. There’s the direct blockade of the transporter that pulls potassium in or it causes potassium to be excreted and this is done by increasing sodium delivery to the collecting tubule making the lumen more electronegative and increasing K loss. Pictures are always much more valuable. This is where Lasix works in the think limb of the loop of Henle. You
give Lasix you’ve brought this transporter of sodium potassium to chloride that will change the charge here and basically increase sodium delivery to the collecting tubule. That increases transport through this channel and because that makes the charge more negative, more potassium gets pulled out. So this is a nice way of getting out of trouble as well, but this takes several hours to work. The diuretic effect will probably take on average about 45 minutes to an hour before you see it. So that was case 1. Wonderful thing I really loved that memory of giving her the calcium gluconate and watching things back.

The next picture is a hint for case 2. Okay just a little hint alight. So this is another one of my, this was basically expert review case for me. It's about 20 years old now, but this was a 75 year old man was a resident of the nursing home. He had been in a high density line operator and basically was electrocuted and was non com pos mentis, he was demented and in the fetal position and was being kept alive at the nursing home. And basically he was transferred to an Oakland hospital okay no names for replacement of his gastrostomy tube. The guy was demented in the fetal position with multiple muscular contractors. I really could not get a history out of him. Physical exam was notable for diffuse flexion contractors of his arms and his legs and he was unresponsive to verbal stimuli. his gastrostomy replacement was scheduled for Monday. Time passed so we're now Wednesday, Thursday, Friday, Saturday, Sunday, 5 days since his gastrostomy tube fell out and his sodium was 175. This is not a record for me. My record for a serum sodium was a lady that was found out in the woods in West Moreland County, her sodium was 221, okay and we did not save her. She was hyper pyrexic and we never figured out why she was out in the woods but she was 221. K was 5.1, total Co2 18, chloride 142, BUN 35, creatinine 1.5. The OR was canceled and just like this talk,
time passed and it was now Wednesday, we're a week after admission and we're now up to 193 with the sodium, K was 5.1, total Co2 was 16 and they consulted nephrology because they realized that something wasn't quite right. So they did the first one so you're now down to 4 choices, okay. Immediately start an IV of D5 and water, 250 an hour, examine the patient, calculate the free water deficit or start IV normal saline at 75 MLs per hour. So what would Ozzler do, okay? Little arm band WWOD, what would Ozzler do, and what Ozzler would do would be he would examine the patient which is the right thing to do in this case. This guy was so, my one associate would say he was dry as a tick which I've never quite understood that but he was a potato chip and really very crisp potato chip. He had decreased skin turgor which right off the bat told me that he was 5% down, pushing on his thigh was like pushing on play dough, so he had doughy thighs which makes him 10% volume depleted based on physical exam, of course he was not sweating and had no mucosal moisture and his blood pressure was 60 over palp. Testing skin turgor, young people which some of us see occasionally you test the forehead, when they get to be about 65 you go down and you test the sternum and whenever they're 80 as I did yesterday on my patient I went up to her and I said please forgive me but I have to give you a pinch on your thigh and she thought that was very funny and so did her daughter and she told me I was being fresh. But in the elderly population it's the least sun exposed area in people that are not nudists and therefore it's really the best place to test for skin turgor. For those of us in the audience with gray hair you can illustrate skin turgor by pinching the skin on the back of your hand and all of us have decreased skin turgor because of solar damage, okay and being a little bit more mature. So what did I do? Well the first thing in this patient is this patient is hypotensive, you have to fix the volume compromise first. Restore blood flow to the kidney, restore the heart and so basically in this particular case we gave started an IV at
75 MLs an hour, I actually gave a liter of normal saline first. The sodium concentration in this is 155 which is roughly 40 lower than what the patient has. So you can run into trouble with the patient getting volume expansion in their brain and causing cerebral edemas, not a good idea to give it quickly.

New thing this year based on the newest guidelines, the correction rate should be no more than .5 milliequivalents per hour. The total correction rate for 24 hours should be no more than 8 milliequivalents in 24 hours. So remember that, this change has to be done very, very slowly because the brain tissue has adapted by increasing solutes within the brain tissue and unless you give the brain time to pump that out and you give them a lot of water, the brain is going to soak that up like a sponge and you're going to end up with herniation of the brain.

So once the hemodynamics have been proved, I would be happy if this guy got up to 90 I really would. Then I'm going to calculate his free water deficit and then I'm going to do this. So where to start assess their volume status, he's hypovolemic so he's had water and salt losses in this particular case the water losses were greater than the salt losses probably through insensible losses. You can get isovolemic water losses, we had a patient that just was discharged yesterday with DI, he did fine I mean he drank his 6 liters of water a day and was you know if he's in the OR and comes out of the OR and he no longer has access to free water he's going to get hypernatremic. So you have to think for their kidneys whenever they're not conscious. The average water intake for a DI patient secondary to lithium is about 5 liters a day. They drink a lot of water, it's fine, they need to drink a lot of water And in hypervolemia which we usually see in code situations where they get a lot of
sodium bicarbonate and a lot of saline is usually hypertonic saline solutions or primary hyperaldosteronism, these are relatively rare. Your most common are going to be your hypovolemics, that's what you're going to see in our population. The free water deficit there are a variety of formulas there if you had the (inaudible) the free water deficit is there, I'm not going to repeat it. And basically this guy's water deficit was about 9.5 liters, which was pretty appropriate because he had no IV's and was losing about 500ccs a day through insensible losses. So we'd figure he's about that.

And then the rate of fluid replacement which was also on your handhelds and your apps, 130 MLs of free water per hour and don't forget the insensible losses of 500 MLs per day. Why not correct quickly? It's what I already talked about is that the brain becomes hyper concentrated, you give a dilute solution, 155 milliequivalent sodium chloride is a dilute solution to this patient, and the brain will soak that up like a sponge. You've got 8% up here that's unused, in Congress it's about 70% but you can expand this space by about that much, sorry. But what can happen is the brain swells and you can get uncal herniation. The brain has the consistency of thick toothpaste and so it'll go right through the uncus. And this is one of the major reasons why this happens to our patients an inability to get free water, and I nearly always see this situation in the elderly or in neonates. The team didn't recognize it so it was a failure of recognition, I guess they assumed that the patient had roots or some sort of thing where they could just absorb water from the environment, they assume that with our dialysis patients as well that if you're a dialysis patients you don't need to drink because you can't get rid of it you still have insensible losses. So you have to supplement, everybody needs a little bit of water and salt and they didn't get labs so this is what I wrote in my report about what the failure was.
Case 3 I've already given you the really awful first slide but I really love this slide because I was a gymnast in high school and this is a hard move and to do it in a position like this this lady is pretty doggone cool. So this is a 75 year old woman negative past medical history. She's got a little bit of hypertension, on repeat 155/100 she's very health conscious, still practicing gymnastics and follows a low salt diet. She exercises regularly. We did confirmatory blood pressures on her and she was hypertensive on multiple occasions and this one is on me guys, okay so I'm not so egotistical that I'm not going to report my mistakes. This was one of mine. I thought oh what a big boy am I, I started on a little hydrochlorothiazide, I talked about all the different things that she had to do, low salt diet, all this that and the other thing. She called me 3 weeks later and was weak and had cramps in her hands and feet and she noticed that she really had not been feeling very well, wasn't eating very much and she was constipated. At that point I thought oh crap I knew what I had done wrong and I took her to the emergency room and what she had was same history on the phone, blood pressure was fixed, pulse was 110, oh what a big boy am I, she also had decreased skin turgor so her volume was down about 5% and her potassium wonderfully enough was 2.1, and I had given her pre renal, acute renal failure, her calcium was up a little bit and on the cardiogram she had a very prominent E wave. Rule of thumb if your K is 2.1 you're probably down about 250-300 milliequivalents of potassium just as a rule of thumb. So I was feeling pretty stupid at this particular time. They had consulted me so again A. is not your choice. I was actually down there sweating bullets, they didn't need to consult me. So start a K sparing diuretic, a slow release oral potassium supplement, a big glass of Florida orange juice or an IV or normal saline and 10 milliequivalents and run it at 100 an hour cardiac monitoring and oral K supplementation.
A trick for recertification exams if they're poorly written the longest answer is usually right, and that was what it was in this case. So she is symptomatic and has EKG changes so you've got to treat her right away. And the best way to do it through a peripheral line is to give her IV fluids with of K in it and again with each one milliequivalent K deficit is about 200 to 400. You have to do this in a monitored setting because of her heart problems. And you can get higher K concentrations but if you've ever had any of your patients get higher than 10 peripherally, they'll tell you how much it burns. I know at least one person in the audience that knows that particular phenomenon this is not good and you ruin that vein forever because it will sclerose.

So what I had done was I started on a K wasting diuretic. I failed to start a K supplement or tell her to enrich her diet in high K foods, and I failed to get repeat labs a week after I started it and I really now after having done this I got burned once I'm not going to get burned again. I always check the labs a week after they are on the medication and I always do this now. This was on me. And this is what I had done. This is, I had gone and I increased sodium delivery, increased the negative charge and pulled K out and didn't replace it and that was the whole thing.

Case 4 another subtle hint, this was the source of the Nile, okay. Unfortunately, I have not been there yet. But this is an 85 year old again, past medical history she just comes in for a routine checkup, sounding familiar? Blood pressure 155/100 health conscious, follows a low salt diet, she's in Silver Sneakers goes to the mall really outdistanced her friends, does not stop at the store, doesn't spend a lot of money which is one of the major side effects of Silver Sneakers and we confirmed that she's
got high blood pressure and started her on a hydrochlorothiazide, have her come back in a month, she's feeling very well, she's taking your advice very seriously, low salt dash diet, drinking eight 12 ounce glasses of water a day because she read in the Pittsburgh Press that water is good for your kidneys. Okay, it's an urban myth. Water is good for your kidneys if you have recurrent urinary tract infections or if you're a stone former, it is not good if you're an 85 year old lady. You should, the big thing I tell my patients that say well doc how much should I drink, I tell them to drink when you're thirsty and drink to quench your thirst. And there's some little provisos that go along with that if they have congestive heart failure and such, but for the most part that rule works pretty well.

So now I fixed her blood pressure again, this one is not on me. By this time I had learned something. Blood pressure is 110/56, I ordered labs, remember it's been a month since the hydrochlorothiazide and tell her to come back in 3 months. The lab calls me with a critical lab value this sodium was 98 and this was from when I was in intern and this patient came in and was on 9 North in Montefiore, this delightful lady sitting on the edge of the bed saying why am I here, I don't know why I'm here. And I didn't know why she was here until I went and got the labs back and then I sort of had to go change my shorts and basically this, I admitted her and what do you do first? Three tablespoons full of table salt only if you know how much sodium chloride is in 3 tablespoons fulls of table salt, an IV or 7% saline which patients will tell you is hot salt. I examine the patient hint, hint, nudge, nudge, calculate the free water clearance and start a IV normal saline at 75 MLs per hour and of course you already know the answer what would Ozzler do, he'd examine the patient because your treatment options really depend on the patient's volume status and your rapidity of
correction depends on the patient's symptoms. This is a chronic problem it should be corrected slowly. If it's an acute problem, try to correct it quickly, but chronic, no.

This is a very busy slide but this is what I do for all my patients with hyponatremia. Are they volume depleted, are they volume overloaded, or are they the clinically euvoletic but with about 2-3 liters of extra water on board that you can't detect. So that's the big thing about this, this is clinical euvoletic. This one with low volume is going to be renal losses and extra renal. Your urinary sodiums will give you the clues if it's an induced renal loss the sodium will be high. If the kidney is okay then the urine sodium will be low and really these things have to be based on the symptoms. If you are clinically euvoletic you're handling salt appropriately so the urine sodium will reflect your intake but the osmol will be inappropriately high for they hyponatremia that's the clue there. And with the nephrotic syndrome you either are sodium and water retentive or you're having difficulty getting rid of the salt and water. So if you're sodium retentive, sodium will be low osmolality will be high and in this particular case sodium will be high and osmolality will be sort of around 250 in that general range because the kidneys aren't working. And the therapy really depends on the exam and the acuity of the problem. So she was awake and alert and really pretty worried and believe me she was the cutest lady I can just picture her in my head right now, thin, petite, I really thought I'd get cookies out of the deal but I didn't. Blood pressure was 116/60, pulse was 80, standing blood pressure fell so she's mildly orthostatic, no edema, decreased thigh turgor, I always ask can I pinch your thigh and they usually pat me on the head and say is that naturally curly hair and I say yes and put up with having my head patted. And believe me it happens all the time. The worst was when I had 3 sisters and they all lined up to pat my head as I walked out of the room. Something about curly
hair and older women. She had hypovolemic hyponatremia. It's naturally curly I don’t put my hair up in spoolies at night. Basically her salt loss has been greater than her water loss and the probably cause is her thiazide diuretic and water loading. And this is very, very common for hyponatremia. Ninety-seven percent of your cases of this are iatrogenic, in other words iatrogenesis in perfecta the doctor did it or are non osmotic ADH release the patient's volume depleted. Slow or fast depends on the symptoms. For acute rapid correction 4-6 milliequivalents at 3% saline and then back off, okay you don't want to do it too quickly. Chronic you change it over a few days and again the correction is no more than 8 milliequivalents per day.

I added 2 very nice references which on your handout you probably can't see unless you have a magnifying glass or if you're a high myope like me, I'm a microscope. That's Stern's treatment of hypernatremia and Current Opinion and Nephrology and Hypertension and Verbalis which is he was actually an endocrinologist here in the American Journal of Medicine in 2013. These are high yield.

Our patient we stopped the diuretic, she was volume depleted so we gave her a liter of saline, close monitoring about every 3 hours, this is one of the typos that I found. This should be 8 milliequivalents per day okay and another typo, well the reason you don't want to correct it is because you can get central pontine myelinolysis it's basically demyelination in the pons because of the rapid correction. Much more common in women in childbearing years but the one that I saw was in an 80 year old and it developed 6 weeks after the correction so you have to be sure you don't correct too quickly. Close monitoring not every 21 to 22 hours, but every 1-2 hours, decreased replacement or decreased fluid tonicity if you correct too rapidly and in some patients we give them
exogenous DDAVP if they correct too rapidly. So in our patient if they went from 109 up to 119 in 12 hours I'm stopping the saline I'm giving a little bit of DDAVP, and I'm cutting back and I'm giving her a little bit of free water in order to get her back down to the appropriate change. Chronic changes you correct slowly.

So the take home points that I want you to have from this are identify what caused the problem, it's very critical to correct the underlying problem, interpret it in the context of the particular patient you're seeing, if it happens quickly correct quickly, if it happens slowly correct slowly, finish line is coming up.

These are 2 excellent articles that reflect the current knowledge about this and for my final statement do not treat asymptomatic urinary tract infections, please do not treat asymptomatic urinary tract infections. Straight cathing is better than Foley's and that's it. Thank you very much.