Speaker: Welcome to the ASHP official podcast, your guide to issues related to medication use, public health, and the profession of pharmacy.

Dave Zimmerman:
Hello, thank you for joining us for Therapeutics Thursdays podcast. This podcast provides an opportunity to listen in as members sit down to discuss what's new and ongoing in the world of therapeutics. My name is Dave Zimmerman and I'm an associate professor of pharmacy at Duquesne university and an emergency medicine pharmacist at University of Pittsburgh, Medical Center, Mercy Hospital. And I will be your host for today's ASHP Therapeutic Thursday podcast. With me, I have the privilege of introducing: Dr. Kate Ciampa PGY2 emergency medicine resident at Massachusetts General Hospital, Louisa Sullivan PGY2 emergency medicine resident at Valleywise Health, and Tiffany Jomoc PGY2 emergency resident at Touro college of pharmacy, Saint Barnabas Hospital. So let's get started talking about today's topic. Recap from the emergency medicine pearls presented at this year's ASHP mid-year clinical meeting. First we have Dr. Jomoc. I know your Pearl was on goal directed resuscitation and cardiac arrest. So what are the some of the critical components of CPR and what role do medications really play?

Tiffany Jomoc:
So CPR is really important in terms of impacting survival. Having quality CPR is really important and some of the things that we can do is minimize interruptions in the compressions with the pulse checks with in between shock pauses and any shifts in compression providers. Another thing we can do is maintain a good rate at a hundred or 120 per minute. In addition, you can also do a really good depth. So usually about what they say two inches or about 50 millimeters. We can also avoid leaning and allow the chest to fully recoil, which allows the refilling of the heart. And with that we can do, insure, firm surface, to allow the recoil to be adequate. In addition, we can also avoid excess ventilation. Usually you want to do less than 12 breaths per minute and the critical components of CPR.

Dave Zimmerman:
And so I have to ask, cause I know we always sing songs during our HCLs training, what is your favorite song to play to keep the correct compression rates?

Tiffany Jomoc:

Right. So usually, it's "Staying Alive" is the most common one, but I think "I will Survive" can be a little bit encouraging too when doing that. And if you're looking for a little bit more upbeat, I tend to like a "Just Dance" by Lady Gaga.

Dave Zimmerman:
All right. It can't go wrong with the Gaga. So one of the tools that you mentioned in your Pearl that we can utilize is coronary profusion pressure or CPP. So why is it important to optimize this and how do you, how do you exactly measure it?

Tiffany Jomoc:
Yeah, so coronary profusion pressure is really important, especially when you're trying to remember that the outcome of doing CPR is a good neurological intact survival. With measuring for coronary profusion pressure, we directly measure the cerebral tissue profusion and hoping that we are measuring and monitoring for that neurological outcome. And of course, I think we were talking about medications before as well, when we, we can do a really good job at administering these medications with epinephrine and causing vasoconstriction. But after we have the fifth, six, seven epinephrine, we can actually lead to a coarse repo perfusion. So making sure that we understand the limitations of the medications and that CPP monitoring for that is actually really important to ensure that we have good neurological outcomes for our patients as well.

Dave Zimmerman:
Okay, awesome. So Dr. Sullivan, I'm going to turn to you now. I know your Pearls titled "Stings in Kids; a Scorpion Antivenom Dosing Strategies for Pediatric Patients". So what are some of the complications following a scorpion envenomation and is there any type of grading system that we can use?

Louisa Sullivan:
Yeah, so the scorpion you should really be worried about is the Bark scorpion, which is normally found in the Southwestern United States and Northern Mexico. And for adults and older kids, the main complications are just going to be pain, numb, numbness and tingling. However, for younger kids we can see more severe complications. So there is a grading system with grades one to four, one is the least severe and four is the most severe. In grade one you'll see some local pain and paresthesias where the stinging occurred. Grade two, that pain may travel approximal to the side of the sting. So if it's in the hand, you can go up the arm, if it's in the foot, it can go up the leg. And then in grade three we start to see either cranial nerve involvement or skeletal neuromuscular

dysfunction. And in grade four we see both, so you can recognize cranial nerve involvement by roving eye movements and tongue specific inflation, and you can see skeletal neuro muscular dysfunction as either flailing extremities or tetanus like back arching.

Dave Zimmerman:
Oh wow. Yeah, it does not sound like a fun time. So I know you mentioned a drug called Anascorp, if I'm saying that right. So how exactly does it work for an envenomation and how do we dose it?

Louisa Sullivan:
So Anascorp is indicated in grade three and grade four Barks scorpion envenomations and it works by binding the venom and enhancing elimination. It has traditionally been dosed as three vials in 50 mils of normal saline over 10 minutes, followed by an additional vial every 30 to 60 minutes as needed until symptoms resolved. However, at our institution we started looking at dosing at as a single vial every 30 to 60 minutes as needed. And found similar outcomes with some pretty major costs savings.

Dave Zimmerman:
Pretty interesting. So anything that we need to watch out for from a monitoring or administration?

Louisa Sullivan:
Sure. So once you get an Anascorp, you're really just looking for a resolution of symptoms, so that's how you can tell it's working. And then the only major adverse effect you're really looking for is allergic reactions, since it is a horse derived antibody formulation.

Dave Zimmerman:
So now we're going to turn to Dr. Ciampa. Her presentation is titled: "All about that Dose; Naloxone for Clonidine Toxicity". So what are some of our symptoms that you would see in a patient that presents with a Clonidine overdose?

Kate Ciampa:
Yeah, so in as with any overdose, really the majority of symptoms that you'll see are an exaggeration of the clinical effects of the medication. So with Clonidine overdose, you're going to see cardiovascular impact. So hypertension and bradycardia. And you'll also see some CNS impact with the CNS depression. And the CNS depression is actually the most frequently seen adverse effect in a Clonidine overdose. It has a relatively rapid onset after the overdose. So within

30 to 90 minutes, you should expect to be seeing these things and it should resolve over 12 to 36 hours. I think something that's important to understand about why these effects are happening is understanding different receptors that are involved with the, the toxicity that you see. So with the cardiovascular toxicity, you're seeing both central and peripheral alpha receptors and the peripheral alpha receptors are only really being hit when you are overdosed in an overdose situation.

Kate Ciampa:
So initially when someone presents with a Clonidine overdose, you're going to see hypertension, but this is transient as that massive bolus of Clonidine in your peripheral alpha receptors occurs. And then the centrally mediated hypotension is what you will see mainly during the course of the the overdose. And then to talk a little bit more about the CNS depression. So in addition to hitting alpha receptors, Clonidine is also binding to imidazoline receptors and there are three different imidazoline receptors. The first one is involved with, again, with the hypotensive effects, the second imidazoline two receptors. This is mediating endogenous opioid release. And so this endogenous opioid then goes on to stimulate the opioid receptors and cause some of that CNS depression that's characteristic of Clonidine overdose. The imidazoline receptor is involved with glucose homeostasis and is not really something to be concerned about an overdose situation.

Dave Zimmerman:
And when we go to to management, I had no idea. And you mentioned during the talk that you can use Naloxone for quantity and toxicity, so exactly how does this work?

Kate Ciampa:
Yeah, that's a, that's the great question right there I, it hasn't been super well-described. There is a little bit about it in the Goldfrank's toxicology textbook, so it's thought that it's mediated through the imidazoline receptors. So imidazoline to your [inaudible 00:08:57] that it, when Clonidine emulates imidazoline to receptor, it's stimulating the release of endogenous opioids and then those endogenous opioids go on to stimulate the opioid receptor. And Naloxone is doing its normal business of interrupting the binding of opioids to the opioid receptor. So then Naloxone doesn't really interact with Clonidine at all and it's not impacting Clonidine binding to its initial imidazoline receptor. It's blocking the downstream effects that are the downstream effects of Clonidine binding to imidazoline. It's a little bit convoluted and it's super not super well described, but that's what is thought to be the mechanism.

Dave Zimmerman:
Always an area for more research than I guess, and I know that you brought up a debate of high dose versus low dose, so which is the correct dosing strategy that we should use.

Kate Ciampa:
So I think the study I will be discussing in my Pearl looked at using higher doses because they broke people down into what they called responders versus non-responders with Naloxone. And so the responders were individuals who got Naloxone after a Clonidine overdose and their mental status improved. The non-responders got a dose of Naloxone and did not improve. And the the two groups didn't really vary in their different dosing strategies. But the purpose of giving a high dose is to say to effectively say that, okay, we know it's not the fact that we gave a low dose of Naloxone and that's why they're not responding. So you really want to test them by giving them a higher dose. And in the study, the median dose they used was 10 milligrams. So that would be the dose that I would recommend to the providers in my ed if they are using it for Clonidine overdose.

Dave Zimmerman:
Alrighty. Some great stuff there and a bunch of great topics. That's all the time we have today though. So I want to thank doctors Ciampa, Sullivan, and Jomoc, some of our outstanding PGY2 emergency medicine residents for joining us today to discuss their Pearls topics they that they presented at the ASHP midyear meeting. Join us here every Thursday. We will be talking with ASHP member content matter experts on a variety of clinical topics.

Speaker:
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