ICSC04 – Emergency Procedures / Rhabdoyolysis
Section 4
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Video Lesson: 02:11

Today, we are going to talk about Clinical Review of Exertional Rhabdoyolysis for the Chiropractic Sports Physician. A little bit about me, I am an assistant professor at Graceland University, in Lamoni, Iowa USA. We are just south of Des Moines, North of Kansas City. It is a small Liberal Arts institution. It is great because we get future healthcare practitioners from all fields. A lot of our students want to go chiropractic, and maybe become physician assistants and PT Athletic trainers. A lot of the content really revolves around the sports population already.

I received my Doctor of chiropractic from Logan University, where I also received my Masters of Science in Nutrition and Human Performance. I then went on and received a Diplomate from the American Clinical Board of Nutrition. I do hold additional sports medicine certifications but have my CCSP, Certified Chiropractic Sports Physician, the International Certificate in Sports Chiropractic, and on top of that, a nationally registered EMT and Paramedic in the state of Illinois.

Exertional Rhabdoyolysis (ER) is how I will refer to it throughout this presentation.

If you are not familiar, you may have heard of Rhabdoyolysis. We often think of kidney damage and when we think about it, when I go into an ER, if I have an athlete or mother and father take an athlete into the emergency department, the diagnostic testing is the same but we know that based on the level of activity that an athlete is doing versus their parents, those are very difference. One of the biggest reasons why we want to make sure that we revaluate and differentiate ER versus just regular Rhabdo is there should be a different set of criteria.

What we are going to show today is the clinical presentation. What should you be looking for sideline and what should you be looking for in an office setting? We will describe the signs and symptoms and compare them to general Rhabdo so that we try and get an understanding of what is that differentiation.

What should we be looking for? It is also important that if we were working at The World Games, the Olympic Games, or at a local high school, we need to know what athletic events may be more prone to developing ER. Know you sports?

I work with the wrestling team here at the university which is one of those top programs and if we have enough time at the end of this presentation, we will go over some of those case studies. We will identify key laboratory findings and look at what a normal diagnostic workup looks like. We will look at what that lab work looks like, what we order in the lab depending on where you are and where your location is which will depend on what sort of testing you could order. All the testing that I am referring to is based on my state license and things that work here in the state of Iowa. However how do we interpret those biomarkers and then how do we apply that knowledge to the actual treatment and management of ER?

It is great that we can recognize it but once we recognize it, then what do we do? We must have a plan and the first thing we need is to get some basic background information to help us understand exactly what we are dealing with. When we hear the word Rhabdoyolysis, it is an umbrella term. The umbrella term is simply used to describe the destruction of muscle cells. As the skeletal muscle cells get damaged through workouts, through activities, through new activity things we are not familiar with, we damage the cells. As we damage the cells, that cell begins to leak all the cellular contents in

the surrounding areas into the bloodstream. Rhabdo in and of itself is a common potentially fatal condition. That is why it is important that we understand this and that we have a full understanding of what it is.

Athletic trainers, coaches, primary care physicians, and sport medicine physicians, all of these physicians and providers will probably encounter Rhabdo at some point in their career. Wrestling, is the sport that I cover predominantly here at the university. We have American football, swimming, bodybuilding, and running. If you are working a marathon race, that destruction, that constant impact, that constant stress on the body can result in the breakdown of muscle cells and muscle tissues resulting in Exertional Rhabdo.

When we look at striated muscles, the myofibrils and sarcolemma are damaged. When they are damaged, the cellular components will leak out. Those cellular components can be a lot of things. Creatine Kinase (CK/CPK) is typically the hallmark and the gold standard for what we test in the lab. We are also going to have Lactate Dehydrogenase, Aspartate Transaminase (AST/ALT), Electrolytes, Calcium, Potassium, and Myoglobin. Myoglobin is really the issue when it comes to renal tubules. If the cell explodes and breaks apart and all these contents go into the surrounding area, Myoglobin is toxic to the renal tubules. That is why you may have heard that Rhabdo is often associated with kidney damage and this is exactly why.

If we look at Exertional Rhabdoyolysis, sometimes it is called in the research as Exertional or Exercise-induced Rhabdoyolysis. It is often diagnosed simply as Rhabdo in the Emergency Department. In the emergency setting, if I am the Emergency Physician, I am going to air on the side of caution. I will go ahead and diagnose it based on the current clinical standards, which will be Rhabdo. I am not going to make that differentiation.

Working in sports though, we need to be able to make that differentiation. We need to be able to understand that it may not be a Physiological Pathological issue, it could just be physiological. They are not interchangeable. That was the position that I took in our recent paper, and I will show you that at the end of this presentation. Work with an Emergency Physician who again agreed that they are not interchangeable yet our hands are kind of tied in Sports Medicine and Emergency Medicine because we just simply don't have enough data as of yet.

There is a debate that revolves around the diagnosis of ER because there is little consensus in the fields. If you ask somebody in the field of Emergency Medicine or Sports Medicine, they are going to tell you that the laboratory findings suggest this. If the laboratory finding suggests Rhabdo, we are going to call it Rhabdo. When in reality, an elevated CK level in a wrestler or marathon runner could just be a physiological response to a recent event, game, or run. ER is thought of as an abnormal event but in literature, if you take a deep dive into the literature, the condition is often just a normal physiological response to tissue repair and tissue adaptation by the athlete.

Think about it, if an athlete comes to you tomorrow and says, "You know what? I have sore muscles but they deserve a new workout today." Are you, the coach or athlete automatically going to assume that it is Exertional Rhabdo? Probably not. There are some other findings that we need to consider. Typically with ER we want to consider physiological. It is often benign. It is often treated pathologically but it is often benign. ER, according to some research, could be the more advanced stage of DOMS, Delayed Onset Muscle Soreness. If you have an athlete who is pre-season or trying to work out or trying to get ready for the season, I do not know how many athletes come in with Delayed Onset Muscle Soreness, it is a very common thing.

Some of the literature is suggesting that DOMS is a milder form of Rhabdo. Again, if you have the ability to test and have research in your facility and practice, if you run a CK level and it does come back elevated, then what? The athlete could have no other symptoms and suddenly, we have an asymptomatic individual with hyper CK in the blood. Well, if that is the case, how do we treat it? Do we treat it as Rhabdo? Do we treat it as DOMS? This is a theory that is gaining some traction, but I think we just need more research on it.

Typically when we look at it, we need 3 clinical morphologies. We need 3 clinical aspects to this. One is severe muscle pain. Again, I am coming to you and saying, "I am sore after a workout", it does not necessarily mean that I am suffering from Exertional Rhabdo. Muscle swelling is something else we want to look for. When an athlete comes to you for muscle swelling, again, this is where we might start thinking of Compartment Syndrome. We need to be understanding that we need to have a keen eye to this. Finally, Myoglobinuria, does the urine look dark? We will look at what urine color we should have versus what is typically presented. Typically, it is this triad, muscle pain, severe muscle pain, muscle swelling, and Myoglobinuria.

As you sit there, just try and think besides a heavy workout, what do you think are some of the things that can cause Exertional Rhabdo or Regular Rhabdo when thinking about the general population? We have a lot. Research has indicated a lot of potential causes, some more than others, and some we agree. Typically, these are kind of the breakdown that we have. Trauma, any sort of trauma to the muscle. That could be Crush Syndrome, some sort of abuse, some sort of electrical shock. Again, if I am electrocuted, that electricity is going to the muscle cells, damaging the muscle cells, and we have a release of the contents into the blood. This is not just an athlete experience. Vascular Obstructions, start thinking about your ischemia. If I have a clot somewhere, some type of infection, an Emboli, maybe I have seizures or if I am cramping a lot, maybe I could damage some of the muscle cells that way, releasing that content into the environment.

This is where we start to talk a lot about athletes when we look at the Environmental Related Emergencies. If you have ever been into a wrestling room or in a wrestling practice room, it is nice and warm. If you ever worked in Football, maybe Football is your sport that you work on a lot, you come back in August, at least here in the Midwest, on hot and humid days, having 2 a day practices. The athletes sweating a lot, they are getting dehydrated. They are not replenishing those fluids before their afternoon session. Football is another one, that is the Hyperthermia. We can also get it in cold weather as well. Then, we have excessive physical activity or muscle strain. Again, if we start a new workout program, maybe we are trying something new. We found the latest and greatest craze online and we want to try that out, if we are unaccustomed to the exercise, we are at a greater risk.

If you have an athlete working with a strength and conditioning coach, if you know the strength and conditioning coach, work hand in hand with them and make sure that you are educating the whole team, everyone involved. Drugs could also be a factor, toxins could be a factor, and then we could have endocrine disorders. As potential causes go, one of the biggest ones especially in the athletic populations, anything involving a heavy eccentric component. Again, if you go back to your introduction to Movement Science Course, if you go back to Weightlifting 101, anything with a big eccentric component can damage the muscle tissues. It is going to damage the muscle fibers, and the z bands. On top of that, if you add in heat and humidity, those have both been linked to an increased risk of developing ER.

Other potential causes that we want to focus on which we will probably see quite often, are some of the ones that I have tried to highlight on this slide, deconditioning. In the college population, athletes go home over the summer, do not do the workout plans like coach says, come back, all of a sudden,

jump in as if they had just left from the end of the season. That is going to cause some muscle damage. They are so unaccustomed to that environment. The deconditioning is from, an office job all summer, and now, the athlete is coming back for two a days in football and has not really spent a lot of time outside. Extreme CrossFit training, not trying to pick on CrossFit but CrossFit is mentioned in several literature articles as being a culprit. Again, a lot of that will stem from unaccustomed activity. We get a lot of novice individuals who come in for CrossFit and start a plan, start a new workout regimen. On top of that, you also have the eccentric component built in.

Dehydration is one of the biggest things that we can focus on and have a direct impact with our athletes, any sport. I am going to keep picking on football and wrestling because it is usually a hot environment. Usually, a human environment, we are usually sweating a lot and we tend to become dehydrated faster. If you look at the wrestling population, it is a weight-class sport and in a weight-class sport, we worry about nutrient deficiencies because we are probably not eating appropriately. We are also probably focusing on weight cutting methods that are not ideal. That is something that we need to be always on the watch for. Electrolyte imbalances are another big key to understanding, that goes hand in hand with dehydration. That also goes hand in hand with cardiac arrhythmias. We will go over some cardiac arrhythmias today. I am sure everyone is familiar with sudden cardiac death. That is something that is pretty high in this population. At least, it is more common in this population than some of the other populations that we would have. That is typically because we have a lot of undiagnosed heart myopathies. Maybe we have electrolyte imbalances going on. We can throw the heart into a funny little rhythm.

Nutrition and diet plays a huge role, these 3 are all connected. If we can dial in an athlete's nutrition, obviously, that is where my diplomate is. That is an area that I have an interest in. If you do not feel comfortable working with an athlete and their nutrition, by all means, there are sports RDs. There are diplomates like me more than happy to consult at any time on an athlete. Other drugs, oftentimes, the general population specially, if we are just talking about regular rhabdo, regular muscle damage, you may have read some studies on statin use. Statins have really gained a lot of following, I would say, within the last 2 decades. In statins, one of the best-known side effects is muscle damage. Nonsteroidal anti-inflammatories, your ibuprofens, and things like that. Again, College kids, at least in my experience, this population loves to take some NSAIDS before a match, before a game, or during a game.

We have recreational drug use just in the United States. Some states now, it is okay to smoke marijuana. Some states still have it illegal. It just adds another layer of things that we need to be able to address. Dietary supplements have been linked immobility, again, if I am just not moving, I could have, you might have heard the old saying, "Use it or lose it." Immobility, if I am not working out, or not moving, I could have tissue breakdown. Then high doses of caffeine.

That takes us to our risk factors. We have covered a lot of causes. At this point, you are probably thinking about what does not cause exertional rhabdo. There are certain risk factors that we can focus on to help pinpoint what population might be at greatest risk. Risk factors and epidemiology, numerous studies have linked rhabdo and ER to various sports and professions. Maybe you are working in a population that is heavy law enforcement. It may be law enforcement training facility close by that you work with. Maybe you are at a military base and work with a Veteran. I am thinking about my days at Logan University and St. Louis, we have Scott Air Force Base close by. We have the South County Veterans. You might have a firefighter training facility close by, these are all linked in terms of profession. These have all been linked because we have heavy gear on, some unaccustomed situations or unaccustomed trainings that we are not just familiar with, and that is going to lead to the possibility that we develop some exertional rhabdom.

These are some populations that we really want to stay focused on. In terms of athletics, again, I have said wrestling and American football, but we also have swimming. Even though there is not a lot of pressure on the joints, it is a high-impact, vigorous sport. Then we have our long-distance runners. For long-distance runners, take Marathon runners and not so much the sprinters. We are causing a lot of chronic damage to those muscles to breakdown, we tend to see at least in this population right here, a higher resting CK level than we would in say, URI. Those who do vigorous exercise and high-intensity resistance training, those have been linked.

We have had cases in Physical Education (PE) classes. Again, this is one more reason why this is a topic of importance to me. As an assistant professor of Allied Health, a lot of our students, I told you, go to PT, maybe Chiro school, athletic training, go that route but we have a lot who want to go and teach. We have a lot of PE teachers coming through a lot of our courses. This is a topic that will directly affect them. There have been changes over the years. Back in my day, if you are late to PE class, you will run a couple of laps around the track, do so many push ups or so many air squats, or something like that. We will cover one of those cases in this presentation.

What are some of those risk factors? Males tend to have a higher prevalence of ER than females. That is often associated with just a greater muscle mass. We have a greater muscle mass, we have more muscles that can be damaged, males greater than females. If we take a look at ethnicity, African Americans tend to have a greater chance of developing ER than Caucasians and South Asians. That is often linked to the sickle cell trait. If I have a sickle cell trait, as an African American, and especially as a male, I am just at a higher risk, higher incidence of developing exertional rhabdo.

That takes us directly to epidemiology. While it is relatively uncommon, I am saying that you probably saw this at some point in your career simply because I have seen four cases, or four believed cases within the past three years with some of our wrestlers. If this is a population that you work with, odds are if they go to the hospital and get some labs drawn, they are at least going to get diagnosed with a high CK level or they going to get diagnosed with Rhabdo. ER itself is potentially fatal, which is what we need to understand and take it seriously. In every year, there is between 22 to 29 cases per 100,000 people. If you're thinking that is quite a lot, other studies report about 26,000 cases per year total, which is still a fairly high number especially if we are in a sports population. Something that I have noticed in the last four years is the athletic community is a small community. As a child, I used to think how cool it was watching somebody play major league baseball, or being in NFL, and now, it still is a huge feat to get to that level but I am amazed at how many players, pro-football, Olympians, and high-level athletes I have been able to meet in just a few short years working in practice.

It is difficult to truly understand ER and define what those cases are, simply because we believed, if you remember from one of the first slides about DOMS, we are going to go on the assumption that DOMS is a mild form of Rhabdo. I personally do not know how many cases of DOMS go to the ER for lab work. I do not test a lot in the office, but if I think an athlete needs lab work for some other reason, I am not opposed to it. If you do find elevated CK levels, what is your standard of care? You cannot just automatically attribute it to them being an athlete. You must unfortunately treat it as if it is a by-product of rhabdo. I know that might be a little hard to understand and wrap your head around. Here I am saying that athletes automatically have a high-level at risk compared to the general population. Unfortunately, all our treatment standards are based on the general population conservative standards. We must meet a standard of care with our athletes. It is difficult to truly determine the frequency of ER because again, I am going out with my professional opinion that DOMS, if we are calling that mild rhabdo, as doctors we are not sending a lot of our DOMS patients or athletes to the ER. We have a lot of misdiagnosed, underreported, and underrecognized ER.

Once we do have it, there is a reoccurrence rate. That reoccurrence rate is as small as 0.08% all the way up to 11%. Typically, what we will associate this is genetic and muscular disorders. If an athlete has 2, 3, 4 cases of rhabdo at that point, the ER is going to recommend further testing. Hopefully, you as a sports physician, sideline provider, you will also recommend additional testing because there must be something going on. This simply cannot be a continuous dehydration issue.

We want to take a look at some of the complications of rhabdo which is why it is so important that we recognize rhabdo on the sideline and try to prevent it. In terms of complications, it can range from metabolic acidosis, DIC, to hypocalcaemia, low calcium in the blood, to hyperkalaemia, that is a high potassium in the blood. Both of those can trigger different arrhythmias. If we have an athlete with a sickle cell trait, we would have sickling collapse that has been documented. You can see in this slide, some of the most severe, most common ones you got here the arrhythmias, and compartment syndrome. Compartment syndrome, we have to recognize, otherwise, that could result in the loss of a limb. Then, acute renal failure is the other one. ARF is how it would be referred to in a few slides. Luckily, the ARF is associated with ER. It is often reversible, so it is not long-term.

Then, we could have end organ failure, cardiac arrest, and even death. Looking at those consequences, renal injury, so again, just injury, not necessarily failure, but renal injury has been reported in about 50% of all cases. When we do our lab work, we run a UA or Urinalysis, we are going to pick up on this. We will pick up on myoglobin and how it has been toxic to the renal tubules and be able to see what that damage looks like, and hopefully, especially if you're in the ER setting, you can start the IV fluid replacement fairly quickly. If we do not, we were at risk of causing acute renal failure. Acute renal failure is rare but about 3 to 7% of all the cases of ER will progress eventually to ARF. That is why early recognition is key here. It is not something that we can just say, " Oh, you know what? Let us go back to the to your room or house, and just drink plenty of fluids." This is something that we want to address fairly quickly but usually, there are other contributing factors. If I am already dehydrated, that is putting a strain, or stress on the kidney. If there is heat stress, if there is trauma, or maybe I have, an underlying disease such as sickle cell, those typically on top of the development of ER is what is going to cause the most damage to the kidneys.

It occurs typically one to two days after the initial muscle damage. You have an athlete, it is Monday morning, we are back at school doing this nice heavy workout to start the week out, and then all of a sudden, we are in the training room or in the clinic. Wednesday afternoon, the athlete is complaining about these muscles and how bad they hurt. That is where we can start to see some of these signs. You can ask him what their urine color looks like. They will tell you, "Oh, it looked like coffee." That is an issue right away. Dark-colored urine, that was one of our hallmarks that we will focus on here in a bit. That right there should be a red flag. We should already think, "All right, we are dehydrated, we are probably having some sort of kidney damage," but renal tubular obstruction will occur secondary to the precipitation of the uric acid and myoglobin.

If we take a look at other research, other studies have reported that elevated levels of CPK and myoglobin as a result of upper-extremity exertion did not lead to ARF versus lower-extremity damage. The correlation could be fairly weak. There is not a lot of research that have examined that but it was interesting to see that upper-extremity over exertion, damage elevated CPK levels didn't really have an effect on the kidneys. Our big takeaway is that while renal failure is one of the most common and concerning issues that we will face with rhabdom or exertional rhabdom, specifically, ARF in the exertional rhabdom patient is almost universally a full recovery, that is a great thing. It is almost universally a full recovery because it usually is a modifiable treatable issue underlying the diagnosis. Dehydration, if I get fluids back in and replenish fluids in the ER, it is going to be IV fluids. If we were

able to kind of catch this early, we want oral hydration. Oral hydration is far superior to IV hydration but I am not going to turn down the bag of fluids in the ER if I get diagnosed with this.

The other thing is compartment syndrome. This is not the same image as this case, but this is a fasciotomy. If the muscles start swelling, again, compartment syndrome, the different compartments of muscle. If they start to swell, and they have nowhere to go, it is just like in the brain right if I fall, I do not know how many people I had with brain bleeds while I was working as a paramedic. They would fall, hit their head, and suddenly, the pressure would start to build up. You get a head CT and that is the end of that story. We can talk about the recovery and the treatment for that. Same thing down in the leg, down in the arm. If a particular muscle group gets damaged, and start to swell, it is going to continue to grow, grow, grow. If it continues to grow, it is going to put pressure on now vasculature. It is going to put pressure on any sort of nerves that are running through. It is often associated with that muscle swelling. It is going to exert that pressure on those nerves. Your athlete may come to you and say, "My toes feel funny, I am tingling, my hands feel funny." Again, as a chiropractor, we could think, "All right. Well, let's take a look at the spine." It is important that we have to also look at the full picture. If it is an athlete, I am going to check the leg if he is complaining of foot pain. I want to follow the leg all the way down. Does anything look abnormal? Does one side look more swollen than the other? Same thing in the arms. This is sometimes a trap for chiropractors. We think, "Numbness in the hand, that is the C5 nerve root. It must be a C5 disc."

Numbness along the knee, going across the knee down to the big toe, we must think, "Oh, that is an L4 problem. Let's address that. I am not saying it cannot be. I am just saying as an athlete, as an athletic population, understanding compartment syndrome, and understanding ER now, we need to be focused on that. Nerve injury is often proximal. Multiple nerves can be involved. Here was an example from research. One of the articles that was discussing about how 14 high school American football players all experienced rhabdo during a summer training session. Again, we came back from summer break. We were not accustomed to the environment, we were not accustomed to the activity, to the heat, and suddenly, we started experiencing rhabdo cases. Think about this, 14 cases, high school following push-ups and chair dips. This is compartment syndrome of the tricep muscle.

That was performed continuously, 30 seconds of rest, intervals of 30 seconds until failure. If you've ever tried to do, for me, it's tricep pull down. I like tricep pull-down. I like going to failure. Personally, I obviously try not to develop ER and I do not think I ever have. The consequences though resulted in 14 players being hospitalized. First off, if you are the sideline Doctor, we need to throw up some red flags to the coaching staff, but we had 14 players get hospitalized for exertional rhabdo. We had 3 of those who just started to develop compartment syndrome who all of a sudden needed emergency fasciotomies of the tricep muscle. That would be very similar to this, only on the arm. Basically, the ER Doctor will go in, will make this big incision. Then I have often seen in the hospital setting, it is almost like a yellow yarn. It is a thick thread, they will close it back together. They will approximate it, but they won't close it all the way. The whole idea of cutting this open is to let that pressure out and to let those muscles gradually come back down. This is something that would result in a hospital stay for sure.

As a paramedic, I can't help but start to discuss ECG changes. I do not know how many 12 leads I have run in my life. I think, personally, all athletes should have a 12-lead EKG performed. There is some interesting research out there. Not a lot, but there is some interesting research just to have a baseline. Characteristics of hyperkalaemia, my muscle cells have broken open. I have an efflux of potassium that can cause what is just a hallmark of a peaked T wave. If you remember your PQRST, it is that peaked T wave. P waves could widen and flatten, we could have a prolonged PR interval. These could all lead to bradycardia. Bradyarrhythmia's, if I have bradyarrhythmia, I have a slow heart rate. As that

slow heart rate keeps going slower because I am just continuing to release all these electrolyte abnormalities into the blood, that's when I could have a very slow heart rate that's not compatible with life. It is something that if you have a high trained athlete, do not be afraid of bradyarrhythmia though. A high trained athlete who runs a lot, who has really good cardiac health could very easily have a heart rate in the 40s and be completely fine. Most heart rates are 60 to 100. Over 100, you get tachycardias. Tachycardia, we are typically going to see during activity while we are running, while we are weightlifting, while we are doing whatever activity is. Then it comes back down to resting. The resting heart rate is 60 to 100. For a high trained athlete, we could easily be in the 40s. I have seen a 38 on an athlete before who was in very fit shape. That 38, he was completely fine.

As hyperkalaemia worsens, we start to run into a few things. We could run into Ventricular Fibrillation, PEA, which is Pulseless Electrical Activity, or Asystole. All of these are not good. This is why we learn CPR. This is why we learn how to use an AED. An AED, this is what I tell my first aid and CPR class every semester, the AED is trying to stop the heart. Most people just tell me, "Oh, it's going to shock it into a normal rhythm but how? It just changes it. It just automatically changes it to a normal signs rhythm. Well, that's not really how it works. It is trying to stop the heart. The AED is looking for ventricular fibrillation or ventricular tachycardia. Those 2, it will recognize and say Shock Advised, if it says PEA. PEA could be literally any rhythm but there is no pulse. It is going to show up as a rhythm on the monitor. It is going to show up as a rhythm on the AED. There is no pulse associated. We treat that like Asystole or if you've watched any sort of medical show, it's flatline. If you've ever seen flatline, that's asystole.

When an AED shocks, AED is going to shock vfib, vtach. When it shocks vfib, vtach, it is trying to stop the heart because we want the SA node to kick back into gear. When the SA node kicks into gear, it should in theory create a normal cardiac rhythm. We will talk some more about some EKG's here coming up. Serum potassium levels do not always correlate to ECG changes, but we do have to talk about sudden cardiac arrest is possible, and that is why this is an important topic. Let's look at that hyperkalaemia. Hyperkalaemia, when I say peaked T waves, this is one of the early signs. Again, mild potassium, this is your peak T. See how it almost comes so your PQRST. This is a PT wave with a prolonged PR interval. As it gets worse, as it continues to get worse, we start to just see, it is flattening. We are starting to lose the QRS, 98% of the potassium is going to be found intracellularly. 60% to 70% of the total cellular mass in the human body though is going to consist of cellular muscle. Even an acute necrosis of 100 grams of muscle tissue can be life-threatening, can potentially cause the serum abnormality we take lab work. If you think about it, 100 grams of muscle mass damage is not a lot for an athlete, especially a really built athlete.

I also wanted to show you a 12 lead. Maybe it has been a while since you read EKGs. Maybe you don't feel comfortable reading EKGs. Again, if you don't feel comfortable, do not do this in your practice. Feel free to call, call somebody, refer out, don't be afraid. Here we are, we are talking like some peak Ts right here. If you look at lead 2, lead 2 is kind of the common lead that we just get a good idea of what is going on. You got your peak QRS and then suddenly, a big old T wave. Now that T wave, this is definitely indicative of hyperkalaemia.

Sudden cardiac arrest, I would not be doing this justice if I did not speak of sudden cardiac arrest. This is something that hopefully no one will ever have to deal with within their career. I tell my students in CPR and first aid, you got to learn this stuff simply because you are most likely going to be using CPR on somebody that you love, a friend or family member. For us, I think of the team that I work with, I love all those guys. You become attached to the team that you work with, they become friends, they become family. Knowing CPR is absolutely crucial, which is part of our license to upkeep. That is one of the things that we must have current CPR certifications. The incidence hopefully is something you'll

never see but the incidence is about 1 per 50 to 300,000 patients. It is a wide incidence, but we typically have young athletes who are apparently healthy, who come in. They may have an undiagnosed cardiomegaly, an enlarged heart, or some other cardiomyopathy. For many, no causes ever found. When I say, "no cause", we are talking autopsy. No causes ever found. These individuals pass their preparticipation physicals with flying colors. There is no indication that we should think suddenly in the middle of a soccer game, middle of a football game, you collapse and have a heart attack or sudden cardiac arrest.

What we do want to focus on is one of the theories surrounding some of the cases of sudden cardiac arrest in this population and that is the fact that if I have exercised induced or exertional rhabdo, if I am doing an exercise, I am already more prone to muscle damage. That muscle damage releases all the contents into the cell. I get an electrolyte imbalance which then leads to electrical abnormalities, which then leads to the sudden cardiac arrest. This is feasible, it has been documented in the research being a proposed theory for one of the causes of sudden cardiac arrest. There are multiple causes of an athlete going down. This is also one of the reasons why I think a 12-lead baseline EKG for all athletes would be very beneficial. I think the double-edged sword for some providers is if we find something, we must treat it. I also think that that's why we do it. We try and prevent; we try and find it before it becomes a problem. I have read and heard of stories where athletes get a 12 lead, and they find out they have some sort of heart condition and they never get to play again in their life. I think that is a worry for a lot of athletes but at the same time, you have saved that athlete and I truly feel like that is way more important than you not being able to play forever. We'd rather have you around than not have you around.

Here are those most serious arrhythmias. Like I was telling you, the AED will look for this. This is basically, the ventricles are just kind of quivering back and forth. There is really nothing happening to it. There is no blood flow. Even though there is electricity down here in the ventricle, it is not enough to pump the heart. Same thing with ventricular tachycardia. We have these big waves but again, no pulse. It is going to be pulseless V tach. That is how we are going to treat it. We are going to shock this rhythm. These 2 are what the AED sees. When I get there on scene, I can take the cable from the AED and put it into my monitor. A lot of these have just become interchangeable in the workplace. I could actually start to see what I am looking at. I can see, "Okay, well, it looks like we have a rhythm, but we still have no pulse. I will diagnose it as a pulseless electrical activity." If it is flatline, I will check this and at least 3 leads. Lead 2, 3, and 4, lead A, B1, B2, whatever, I can double-check this. Asystole and 3 leads are deceased. Unless we typically find other reasons to think otherwise.

That takes us to the clinical presentation. What is that clinical presentation of exertional rhabdo looking like? In severe cases of rhabdo, our general symptoms may include fatigue, general malaise, fevers, nausea and vomiting, electrolyte imbalances, tachycardia, so again, fast heart rate, or seizures. This is just a general list, think about the patients that may be coming to you, if you're not in just a sport-exclusive practice, a patient comes to you with fatigue, "I don't feel right. I had some nausea, I am vomiting," rhabdo probably isn't the first thing that we are jumping to, and this is where a good case history, a good medical history is going to come in. What have you been doing recently? What's changed? What's happening? Have you been sick? Trying to get a full picture of what's happening. These general symptoms by them in and of themselves would not suddenly say, "Yep, that is rhabdo." Our clinical presentation, we're looking for muscle pain. Remember, I said severe muscle pain that is intense. It is muscle pain that the athlete has never described before. You ask the athlete, have you ever had this? It has never been this bad. This is like a 10 out of 10. I am sure you have heard it anytime an athlete or a patient goes, "My pain is 10 out of 10." They're just sitting there may be on their cell phone or just kind of smiling like you start to wonder. You can tell an athlete who is in severe intense

pain and that's going to usually be during active and passive ranges of motion. This is where we must actually touch our patients. We want to do a good exam on them. We want to do the active range of motion. We want to do the passive range of motion.

If they are still having this pain with passive range of motion, it is still just as intense, just as severe, our red flags should start waving. We should be focusing on muscle weakness and generalized fatigue. Again, think about it, if I come in with muscle pain and I am already that sore and it hurts that much from the active and passive range of motion, well, of course, I am going to want to focus on the weakness. Generalized weakness, unfamiliar with this exertional level, if it was a new activity, this is where the case history comes in. Then that takes us to the presence of myoglobin in the urine. You can either give them a cup, a urine sample cup, asking to go to the bathroom, fill it up, or just ask them if you don't want to do any sort of UA in the office. If they tell you, it's dark tea or cola-colored in nature, that is a red flag as well. Our clinical pearls, we have 3 primary symptoms. Our 3 primary symptoms, muscle pain, muscle swelling, myoglobinuria. That is slightly different than the diagnostic criteria. The diagnostic criteria are dark cola-colored urine, which typically we will start to see about 12 to 24 hours from initial muscle damage. Myalgia, that severe muscle pain, tenderness, weakness, and edema. If we are getting edema, we really need to be cautious with compartment syndrome. Then the third criteria are elevated serum muscle enzyme. We will do some lab work. This is often done possibly in an emergency setting, so in the ER simply because you can get results a lot faster than you can maybe in your office. That increase CK, it is typically going to start to be there 24 to 48 hours and then it is going to start to gradually decline over the next 7 to 10 days.

Hydration is going to play a huge role here. Hydration status, hydration is a foundational component for any athlete with ER. If we are going to factor in the athlete's sport, their environment, is it hot humid, is it cold environment, and what are their nutritional goals, these can all help us in diagnosing and making a correct diagnosis of ER. This is one thing that I have a hard time getting some of my athletes to understand. I preach hydration levels, but this was the first time this year that I have ever had over-hydration. I partly blame myself for the cramping because again, just like I can go this way and start to cramp and have muscle issues, I can be over-hydrated. Now, overhydration is when you see and again, you might see this in your practice, I see this in class so when athletes walk in with a milk jug full of water. All they are drinking is water, water, water. I am hydrated, I am so perfect. You're missing the electrolytes. You're thinking hyponatremia, you're thinking those electrolytes, you're just really thinned out in the blood. This is where the education piece comes in. Educating that yes, it's important to hydrate, water is great to hydrate but do not overdo it on the water. Make sure that we're throwing in the electrolytes. That is why a lot of the wrestlers like Pedialyte. A lot of football players tend to like other sports drinks. Generally, I don't recommend sports drinks to people just because they are going to work out for 20 minutes. The research doesn't support it.

If it is an activity that you're going to do for over an hour, sports drink may be exactly what you need. When we look at hydration, if an athlete says, "I am crystal clear," I don't want you to be crystal clear. I want you to have some yellow. Now, we start to get dehydration as that yellow starts to get higher and higher and higher. Eventually, we have extreme dehydration where we start to get dark tea color-colored urine. As a refresher, let's talk about some of the dehydration consequences. You can look at the previous slide we had. We had some in terms of overhydration and underhydration but typically in terms of dehydration, at 1% body weight loss, we're going to have thirst. By the time we are thirsty, we are already dehydrated. If I have an increased thirst, maybe I start to have a loss of appetite. Maybe I have discomfort. That right there is going to be a 2%. Now, eventually, we can keep going up to muscle cramps, difficulty concentrating, and this is why I really start to convince athletes that it is important. If they are in the middle of a match, middle of a game, and they cannot concentrate on the

play, they cannot concentrate on what their opponent is doing, and they are 5% dehydrated, that is going to directly impact their performance level.

If we start to get to the stumbling headache, dizziness, and labored breathing, that athlete is out, at that point, we are probably getting some emergency services coming which we have had before. We have had it on the sidelines. Hot humid day, the athlete was not drinking enough. We started some electrolyte therapy on him and then suddenly, he just was not really unresponsive. He just was not really coherent. We activated emergency response because at that point, it is kind of out of our hands. Looking at dehydration, there is another way to look at it if you are an adult beverage drinker. Think of a logger as our good color of urine. Think of a pale ale as we need to hydrate, red ale, we need to hydrate more and if we're a stout, we are coming to see one of us. We need to see a doctor, we need to get some lab testing. It should not look like this. But exercise-related renal impairment usually occurs as a result of dehydration, making sure that we are not dehydrated, making sure that we are keeping our athletes hydrated as best as possible, telling them that fluid losses of 1 to 2 liters per hour, particularly for wrestling rooms, 2 a day football, we are going to have to have an aggressive fluid replacement, especially if I am in a 2 a day. If I am in the morning, one of my colleagues, one of my good friends and colleagues worked football when he was getting his masters, he was a GA. They would make athletes weigh first thing before the morning practice, and he said, it wouldn't be uncommon for them to lose 7, 8, 9 pounds and he would have to stay in the locker room with them or in the athletic training room with them until they rehydrated. They weren't allowed to leave morning practice until they were rehydrated within a pound or 2, there was a percentage of leeway.

Then they could leave, go get lunch, do whatever, and then come back for their afternoon session, weigh again, they had a very good monitoring system. I have been in other places where there is no monitoring system at all. Athletes just go out. Some athletes care about their body enough that they step on the scale, they know what they need to replenish. For the most part, it is not something that is really implemented at a lot of places. We want to make sure we have an aggressive fluid replacement, but we also want to make sure that it is not just pure water because that can lead to hyponatremia. Then finally, most commonly, the ARF, the acute renal failure, it is going to be associated with hyperkalaemia maybe to follow. If we look at biomarkers, so clinical dehydration, if we're doing lab work, if we're able to read lab work from, maybe the athlete brings you back to the lab work from the hospital the notes or you request them. We're looking at sodium, we're looking at BUN, the plasma osmolarity, AST, CK, CK is our big one here, and LDH.

It has been noted that dehydrated athletes with a significance statistically will correlate of sodium and BUN which can then guide you into dehydration and skeletal muscle damage. Dehydration may prevent an athlete from clearing myoglobin. That's our concern. If we get that precipitated, if we get that build-up of myoglobin because I do not have enough fluid to keep the kidneys filtering, keep the kidneys flowing, that is where we get to build up. That is where we get the kidney damaged. You learned a lot so far. Now, we need to start applying some of this. We need to take a look at some differential diagnoses. Without a high index of suspicion, I am willing to bet you are going to miss Rhabdo. I am just willing to go out there. Primarily due to the fact that athletes come in already with muscle pain. They already come in with swelling. They already come in with tenderness. If an athlete comes in with these, I am not just automatically going to assume exertional rhabdo but at least now, after going through this course, you can think, "Okay, you know what? That needs to be on my radar if their urine is fine, if I want to do lab work, that all checks off fine, if you want to rule it out, by all means, rule it out." In my level of differential diagnosis, I always want to go to the most severe first and then work my way down. I want to rule out any sort of life-threatening emergency.

If ER is suspected, I need to have an immediate referral to a higher level of care. As great as it is that I can recognize it, I don't feel comfortable necessarily starting the fluid hydration orally. If I think that somebody is at this level of ER, they are going to an ER. They are going to an emergency department, they are going to get the IVs that I, in the state of Iowa, can't give. They are going to start getting a more definitive line of care. The other causes of that muscle pain and weakness besides ER should also be considered at, as appropriate. If I am working sideline at a football game, the athlete comes in with intense quad pain, it could be a helmet to the quad. It could be a helmet to the leg, or a helmet to the shoulder. There is a lot of reasons to explain muscle pain besides ER but knowing all of your components will help you make a better diagnosis.

If we are looking at muscle pain and weakness, that differential diagnosis may include acute myopathies, periodic paralysis, maybe we have myositis or dermatomyositis, Gan beret, muscle strain, discogenic involvement, again, chiropractors, here we go. It could be a discogenic involvement. In more serious cases, we are ruling out cardiac problems. We are ruling out heat stroke, and we are going to rule out asthma. These are the 3 most common non-traumatic causes of sudden exertional collapse in a healthy individual. Cardiac, respiratory, heat, those 3 things. How do I test, how do I tell? The signs of serious causes of exercise-related collapse may include an altered mental status. Think of your AVPU, your alert verbal pain unresponsive. Think of AVPU, think of systolic blood pressure less than a hundred. When I took my CCSP program, I feel very fortunate because I was taught by 2 fantastic individuals. One was an AT and one was an EMT. My background being an EMT, I could understand where the EMT was coming from. Keeping a blood pressure cuff in your med kit, keeping a stethoscope there because those are all things that we trained on that we learned in practice when we were going through our EMT license. Athletic trainers there and she goes, "You know what, a blood pressure cuff, I do not really use that much. That was not what I was trained on." She had other tools in her toolkit that helped her diagnose.

There is not one right way here but I, personally, being the EMT paramedic that I am, I would recommend having a blood pressure cuff to get at least a systolic blood pressure. I also keep a pulse ox in my bag, just a little finger pulse ox so I can get a heart rate quickly. I get a heart rate, and I get an O2 saturation, and I can tell a lot by an athlete level quickly. Additional keys to that diagnosis. We want to look at dark urine. Dark urine without any other symptoms. That may not indicate ER but rather just a dehydration level and that's fine. That is something that we can see here on the sideline. The athlete comes over on the sideline, comes up after practice, and has dark urine. Now, if we are in the office, maybe we send them for labs. Maybe they are in the ED, maybe they are going to the emergency department for muscle cramping. Automatically, they are going to order lab work. They are going to order a CK and what they are going to find is acute CK levels five times the upper limit. Five times the upper limit is usually about a thousand. If levels go up to a thousand, that is going to be a really conservative, and we will see this, we have a really conservative diagnosis for rhabdo. It is a low threshold. When I say conservative, it is a low threshold has been shown to have a high sensitivity, but it has a low specificity. That is something we need to keep in mind. Criteria, now, we are in the emergency department for sure.

The criteria that may indicate hospitalization, and again, unless you are an emergency room physician, you are probably not making this decision. This is probably not up to you but if it is a high CK level, they may want to stay overnight for observation, and decrease creatinine clearance. Maybe they have elevated serum creatinine. Maybe I have myoglobinuria, I am worried about the kidneys. I will stay overnight. Metabolic disorders or abnormalities, or any sort of signs of compartment syndrome. All of these things are probably a good way to spend the night in the hospital to confirm diagnosis. Once I have gone through my differential, maybe I have ordered some lab test, then I am going to go through

a history first. Let my questions guide what my thinking is. I am going to ask all different questions, three or four different ways to athletes depending on how many times you have done an H and P, a history and a physical, athletes and patients in my experience tend to lie. Not necessarily on purpose but they tend to lie.

If I was in the back of an ambulance and I ask a patient what kind of health conditions they had, they might say "I don't have any. I am fine." Then I get to the ER and the nurse comes in and says, what kind of health conditions do you have and they give the same answer. "I don't have anything. I am fine." Then you might ask, "What medications do you take?" they reply with "I have a list of medications." Some people don't understand that they are taking medications for a reason. They think if I take the medication, I no longer have the problem. Suddenly, you start finding out patients have blood pressure problems, heart, breathing and cholesterol problems. The list is just never-ending.

I will do my exam findings as well and take the tenderness into account of the affected muscle group. I will look for possible signs of hypovolemia, take a blood pressure and if it is below a hundred systolic, that is going to be a sign that I want to focus on. Laboratory findings in the hospital. When working in the hospital most likely you will get a CBC, a complete blood count and get what we call a CMP, comprehensive metabolic panel and you will also get a CK panel. For that CK, we are typically looking for things greater than a thousand. That is five times the upper limit. Additionally, we can use a urine dipstick. A urine dipstick can test positive for myoglobin without the presence of microscopic evidence of blood. What we are doing there, this is something that we can use in practice, in our office. Have the athlete go pee in a cup, bring that back. It is just a urine dipstick. We will take a look at what that is. You dip it in, and we can gauge a lot of information based on that. Here is something interesting I found during the research that I just wanted to make sure all providers knew.

We went over, we changed to ICD 10 codes probably 5 years ago, 4 or 5 years ago, I can't really remember. The ICD 10 codes should not be used unless absolute. An Exertional Rhabdomyolysis or a rhabdomyolysis diagnosis should not be used unless absolute, because what the authors proposes of that's going to hinder future research. Right now, we may have a lot of misdiagnosis of rhabdo simply because, right as soon as a patient gets to a thousand, all of a sudden that's criteria. That criteria with some muscle pain and some dark urine, and it is rhabdo.

Early detection is where we come in. We need to be monitoring urine. If we are in a weight class sport specifically, I am more concerned about urine than probably other sports. If you're working with volleyball, you're probably not caring too much about dehydration right here. If you work football, if you work soccer, if you work any sort of outside sport, I recommend and suggest that we focus on early detection of dehydration. What does that look like? The NATA, the National Athletic Training Association really identified 4 factors that may lead to Exertional Rhabdo. Number 1 was performing 2 to 3 minutes of all out vigorous absolute exhaustive, work till failure, maximal, activity, 2 to 3 minutes, that's it. If I am sprinting short distances, right 800 to 1600 meters without adequate rest periods. I am constantly causing that damage, constantly causing that stress, on the quads, on the calves, on the legs, that could be a trigger. Vigorously exercising the first day of pre-season. Like I said, athletes have been off for the summer. Athletes come back, we quickly find out who did and did not do the summer workout programs, and all of a sudden, they act like they haven't missed a beat. They're trying to lift the same 300-pound bench that they did when they left. It may not work out because they are not acclimated. This is a big push, at least within the last couple of years. I remember, from the NATA about acclimating to the weather. You can acclimate to the weather with football.

It is not like we are going to necessarily start 2 days right away. We do a lot better monitoring the field, if we are on turf versus on grass. What's the wet bulb? What's the humidity? Those are all tricks

that we can help our athletes and acclimate them back to activity. Then finally, sprinting at the end of practice, they are sometimes doing work out drills/exercises “gassers or suicides”, sprinting down and back while exhausted and fatigued. The athlete has gone through a 2-hour practice, and now the coach wants me to do work out drills “25 suicides”. We as the doctors need to say, "Is that really going to be the best, for the athlete?" Then we take a look, NATA, again, the National Athletic Training Association. The NATA has given some recommendations on sports programs.

Strengthening and conditioning coaches. There needs to be a transition period for vulnerable times for athletes. All strength and conditioning workouts should be exercise based, scientifically sound, and physiologically represent, representative of that sport. Does it make sense for us to be working out what we are working out? This is something that strength and conditioning coaches, especially us, as healthcare providers, we should really understand. Then conditioning program should begin with work to rest, ratios of 1 to 4 first 4 days of that transition period. We should have separate day workouts and all workouts. Then finally, all strength conditioning workouts should be documented. We should have good records of what did the athlete do. What was the volume? What was the intensity? What was the duration? All of that can be very helpful so that we can help identify, well, 2 days later athletes start coming into the training room and they all have calf problems, or they all have triceps problems.

You go back and you look and redid 13 tricep exercises for every 2 bicep exercises, that's disproportional. Taking a look at diagnostics, we want to take a look at diagnostics. Typically, the 3 things that we are going to get, especially in the ED, it is an electrocardiogram or an EKG, ECG, we are going to get a UA. A urine analysis, we are going to use a dipstick. We can use the same dipstick in the office. We are going to have blood, we are going to get some sort of blood, we are going to get a CBC, we are going to look at renal and liver functions. We want to look at CK levels, at uric acid, and at electrolytes. Those are typically our work diagnostic workup of what we should be doing. Common ECG changes, I won't go through all of these. These are all things that if I have exertional rhabdo, if I do have the electrolyte abnormalities, if I do have any sort of fluctuations, I could have anything from hyper to hypocalcaemia. I could have her hyper or hypokalaemia, hyper or hypomagnesemia and then hyper and hyponatremia. These are all going to be based on electrolytes, sodium, calcium, magnesium, and potassium.

Our most severe Vtach and V-fib is what we shock. PEA, asystole, we don't shock, we just do really good high quality CPR. Here's what a dipstick typically looks like. You have probably seen it or used them in clinic. You may not use them now; it comes in a little container. You pop it off at the top. Just pull this out, follow the instructions by placing the stick in the urine for duration listed on the instructions. Then we are testing different things like specific gravity. We want to look at PH levels, protein, glucose, Ketone levels, bilirubin, blood, nitrates, and leukocytes and if you notice, normal is negative.

I shouldn't have any of these, however If I do have some of these, those would be abnormal. If it came back positive for blood, it is telling me that there's myoglobin in the urine which I can see under a microscope. In terms of myoglobin, this is what I am talking about, tea colored urine. It is one of the most important consequences of significant muscle damage. This can be eyeballed, it is better if I do some testing to it, but if an athlete brings this to me right away, I am going a completely different route on my diagnosis and my plan. I am going to get them, they are going to start to drink fluids, and I am also going to get them most likely to an emergency room. It develops once there's at least a hundred grams of skeletal muscle damage. Small amount, the myoglobin spills over into the urine and the plasma concentrations. If the plasma concentrations reach about 1.5 milligrams per deciliter. That is what causes that reddish brownish tint to it and it may be detected with a urine dip stick or on macroscopic view. Macroscopically, just looking at this, it looks reddish, brown and dark.

Serum levels will typically rise of bio level within 1 to 6 hours, peak about 8 to 12, and then start to come back down to normal within 24 hours. If the serum myoglobin will exceed 0.3 milligrams. Back here, 1.5 milligrams per deciliter spills over. Now we are talking, if we just reach 0.3 milligrams per liter it becomes detectable with a urine dipstick. That's why having a dipstick can be sensitive. You may be on the fence of, well, does that urine look bad? Does it look normal? It looks really dark, but is there anything in it? Use a simple dipstick. The urine dipsticks are inexpensive, they are sensitive tests, ideal for the sideline and office setting but obviously if it finds something abnormal, I could send them to the ER real fast to the emergency department (ED) and have further testing followed up.

I can call the ED and say, ""I am sending an athlete to you who had positive X, Y, Z in the office. I believe X, Y, Z to be occurring." Then the ED will start their diagnostic follow up. You would be amazed at how many facilities will appreciate that initial call. Providers appreciate having some heads up as an athlete comes in. Major drawback is that you cannot distinguish between hemoglobin, myoglobin and hemo rich red blood cells. That is where ER comes in, the ED emergency department, will do that testing and send to the lab so they can figure out what it is. Just another image showing you different colors of urine. Everything from what we call light straw. I would say over hydrated to straw, to yellow, dark yellow, amber and dark.

Same thing here, we just changed them up a little bit amber, red and dark. What I typically find on a lot of the reports that I get back I often see Amber and Dark Amber as being most often listed. We take a look at blood test, we are going to order a CBC, complete blood count, a CMP, comprehensive metabolic panel, and we will do a CK. CK, CPK, we want total serum or plasma here. It is something that I don't really cover too much, but I just want to make sure I say it now, we are not ordering a CKMB. CKMB is looking at cardiac damage, it is a test that I could run if I am concerned that, somebody might have had a heart attack, an MI, I don't want this one.

You need to be very aware of this one because oftentimes when you say, you want a CK, CPK level, the lab for some reason, and it is probably because they are in a hospital and they are probably running way more CKMBs than normal, they will run a CKMB. I have had it done at twice at our local hospital and we are a small hospital. I don't fault them, it was wrong, it was incorrect and I called them up and said, I didn't order a CKMB, I just ordered a CKCMP or a CPK and they just had to re run the test. This will not provide you with the data that you want. This is going to provide you whether or not I had some cardiac muscle damage, which is what I want. I also want you to be aware that depending on what lab and what laboratory technician is working that day, you will either get CK or you will get CPK. They are fairly interchangeable, but their reference ranges are not. They in and of itself are, one is one CK equals CPK, but they have different reference ranges. It just made it difficult when you are trying to go through a case series trying to write that up for a journal and trying to take into account different lab values. Hopefully we will see that here in a few slides. I won't go over the CBC components or the CMP components, but again, this is where we are looking at a lot of that liver and kidney function.

Additional findings, is where we really want to focus. We focus on CK because CK is the gold standard for diagnosis of rhabdo. However, there are 4 other enzymes that could be elevated that we should know in the back of our heads, AST, ALT, lactate, and dehydrogenase. These could be elevated on the lab report, but we are looking for that CK value. If rhabdomyolysis is suspected, the serum levels of the following should be evaluated, and those things are potassium. We have seen why we want to order potassium, hyperkalaemia we want to look at uric acid, we want to look at creatine, lactate dehydrogenase. Then what we may find is that the following could be decreased on lab results, which would just further strengthen your diagnosis of rhabdo, and that's calcium and that's ph.

Let us take a look at creatine kinase, because this is what one of the hallmarks of this condition. Creatine kinase in and of itself, here's a little biochemistry for you, it is part of the ATPCR energy system. Creatine kinase is the enzyme that catalyzes the reaction, transferring the phosphate from the creatine phosphate to the adenosine die phosphate, which helps us make the ATP. I know some of you right now are thinking, "my goodness, I do not care." I can agree, as I dreaded biochemistry in grad school, yet I have opened the medical biochemistry textbook more in practice than I ever did in school. It is amazing that when you get into sports nutrition, how many of these reactions you either learned once and thought, I will never use that again or you want to reference them in a paper, in some research, or a presentation. You want to get this nice image. Don't ever say that you will never open that textbook again, because you may in fact do it. When you are looking at creatine kinase, here is a few things that we want to know. Creatine kinase, it is the enzyme which catalyzes the conversion of creatine to phosphor creatine. There's our PCR, we have the skeletal muscle myocardium and brain. CKMB, CKMM, CKBB those are all rich in enzymes but those are not what we want. When I order a CK level, I just want to know what the CK is, Creatine kinase.

CK is released in other tissue damage, which is why we have different forms and different specific enzymes that we can look at. If we have an MI myocarditis, rhabdo, of course, muscle trauma, severe muscle exertion surgery. If you look at lab work from a surgical patient, any sort of muscle damage, whether it is me working out with a dumbbell or a surgeon coming in with a scalpel, it is still going to cause muscle tissue damage and release of contents into the body. Tetanus, alcoholism or statins. An elevated level of CK is the most sensitive and reliable indicator of muscle injury that will constitute the diagnostic workup and diagnostic hallmark for that matter of ER. Now these are, CK levels are very specific and very affected by race, sex, and physical activity. Those are 3 things that we need to keep in mind.

Usually you will see a spike in CK levels 2 to 12 hours after muscle injury. They usually peak about 24 to 72. We have about a 1-to-3-day window of seeing when they're going to peak, and then they gradually declined. They are going to decline over the next 5-to-10 days. Training for sports and competition, will already have a profound impact on serum CK levels and it has been documented in numerous studies that athletes already have higher CK values than non-athletes. This is the start of my premise that when an athlete goes into the ED for evaluation, they can't be treated the same as somebody who is a couch potato who likes to play video games 23 hours a the day. Their CK levels are already going to be elevated. Treating them, treating those 2 individuals equally in terms of diagnoses is not appropriate in my professional opinion. That's what we wrote in our presentation, in our paper.

Many cases of Exertional Rhabdo are simply subclinical. Some cases are only detected when we start to run CK levels. This is why I go back to the question I asked you early in this presentation, if I am ordering all of these lab tests on an individual and a high CK level comes back, what do I do? How do I treat the patient? Do I automatically pull them because it is a standard of care? Do they have any other symptoms? If you start asking an athlete, do you have any muscle soreness? No, I have muscle soreness. Right now you start checking off the boxes on paper I am checking off boxes for what could be ER, could be Rhabdo, but in most cases it is subclinical.

It may be really important to do for research testing. If I am trying to take a wrestling, football or a swimming population, what is the kind of baseline for this particular group of athletes, and even then, I got different weight classes to think about linemen versus the wide receivers that I have heavyweights versus the 125 pounders. All of this to try and get a reference range is where research really needs to go. CK, CPK it is one of the most used biomarkers to diagnose rhabdo. However, according to research, those levels can range from 5 times. Now 5 times, that's where we get the

diagnostic criteria to call it rhabdo. That's why it is very conservative, but it has been proposed that to 50 times the upper limit should be used.

Think about that, we are going five times to 50 times. Typically, what we see a lot in some of the research, it is a 5 to 10 times range. I can tell you that our local hospital uses the 5 times range. In fact, the local hospital makes it difficult. This is where, if you are working with say LabCorp or Quest Diagnostics, whatever your lab is that you are working with, our facility will only read levels greater than a thousand. I don't even know how high some of the athletes that we have had have gone because the lab sees the cut off as a thousand and that's all they report, it is greater than that. Other labs will come back and give you numbers, 25,460, 15,230. If we take a look at all the research, in the presence of a natural history, just a normal history on a person, serum CK levels greater than 25,000 is likely to be benign in ER patients, think about that. That right there is likely to be benign. Yet we don't even ever get to see levels up to 25,000 because as soon as they are greater than a thousand, they're pulled from play for a week. It is not unusual to see CK levels up to a hundred thousand in some research. When we start to throw in different confounding factors, when ARF, acute renal failure is present in patients with ER, those typical CK levels, we tend to see it at about 15,000. Values have been documented as high as 3 million. That is not a typo, I promise high as 3 million. Those I will say are very rare cases, but I can also tell you that if that patient had been into my local hospital, I would've simply gotten greater than a thousand.

Here is the other problem. There is no standard CPK level for diagnostics for diagnosing rhabdo. That is an issue because our standard reference range is that for most labs are simply low and often lead to overdiagnosis. Because I have no definitive algorithm, because I have no consensus of what level of rhabdo or CK will have detrimental effects, most providers want to air on the side of caution. Clinically, ER is often diagnosed with CK or CPK levels at 5 times upper limits, that's what you just need to know. Five times the upper limit is often a very conservative range. It has been reported like that in multiple sources. There is some suggestion that we should try to raise that to 10 times the upper limit. Typically, patients with 10 times the upper limit of CK, were found to be asymptomatic. No other symptoms just high CK and that's believed to be a better diagnostic value.

As we continue on with the problem, more recently, there has been proposed suggestions of raising that limit to 50,000. No definitive pathological value for CK has been defined either. If we go back a slide, simply because there is no standard in terms of what the diagnostic criteria is, we just use the five times the upper limit as a conservative amount. There is no definitive pathological value for when it becomes dangerous, when it becomes harmful to the body. At this point, we are simply showing the elevation is occurring. The healthcare provider should be aware that some levels even 20 times the upper limit may be seen in performing repetitive strenuous exercise. If I am doing 2 a day work outs, if I am preparing for a show, if I am doing a physique competition, if I am doing body weight lifting, all of that can be just adding to this number.

It can be very scary and let me also say this, if you order this test and LabCorp comes back or Quest diagnostic comes back and says, your athlete CK level is 20,000. You could panic and rightfully, that is a high number, but only CK, CPK values over 5,000 should be concerned for renal failure. That is simply one study that found that other sources say 10,000 is the threshold.

Hopefully by now, I am either confusing you more or I am showing you the importance of what is the standard of care versus where should we be going. We should not be satisfied with a range of, well 5,000 is when we think of renal failure, but you know what? 10,000 is a normal diagnostic threshold, but you know what, 20 times the upper limit for strenuous activity, we just need better reference ranges. We need better research for our patients. At the end of the day, I often tell athletes that they

are their best advocate. I sleep well at night because I know that every athlete that came into see me, I did the best I could for them. I did what I felt was in their best interest.

Let us just take a look at some crazy CK values real fast. I just want to show you some documented cases so that if you ever do see it, you don't automatically just panic. There was an 18-year-old football place kicker, I am not getting hit necessarily all the time, I am just using my legs, 130,000 was his CK value. Another athlete was a 16-year-old male following 2 a day practices at a wrestling camp. I believe it was a five-day camp, he was at 146,000. Then, if you remember, I even told you the physical education class, here is a 12 year old male. He was late to class, his punishment, old school thinking versus new school thinking, maybe you still work with coaches who have an old school mindset of a punishment workout, but we are performing 250 squat jumps, that's it. After performing 250 squat jumps, next couple days, the athlete went in, saw his local trainer, he was having muscle swelling, he was having pain in his legs, didn't have a very good range of motion, was sent to the ER, his value was 244,006. That is a big number. In fact, there is evidence that suggests that CK and CPK values will range between 2 and 10,000 after doing 50 maximal eccentric contractions of the elbow flexors. I was telling you early on that I like to do tricep pull down. If I go tricep pull down to the heaviest I can do 50 of those masks maximal eccentric contractions. If I am trying to work on my arms and then I immediately go get some lab testing, maybe 12 hours after, give it some time to spike up 24 hours after I could have some high CK levels.

Knowing when an athlete is moved from the physiological state to the pathological state is often difficult, and it is often difficult to assess and manage without some sort of clinical diagnostic testing. We want to have expected elevations of CK, CBK may explain why team physicians so us or emergency room physicians often recommend intravenous hydration. At levels of 3000 and over, I am probably going to get fluids. Fluids right there in the ER. I get a couple liters of fluid lactated ringers, normal saline, depending on how my lab recheck goes. If it looks like things are trending down, clear out and mark the urine as I will probably be released. If I am over 10,000 units, I am most likely going to be staying the night in the hospital.

The question is, what is the re-evaluation process and how do I re-evaluate CK levels? This is where we as doctors come back in. The athlete has gone to the ED, been diagnosed with rhabdo, been diagnosed with Exertional Rhabdo and usually what they are told is no activity for a week. Then you going to repeat your lab work. Current laboratory ranges for CK failed to consider gender, ethnicity or any sort of activity level. As a physician, what we do is we can order those lab work and we can use that lab work to make a diagnosis or make a recommendation on whether the athlete can return to play. Usually that return to play is going to be simply based on is their values trending downward?

This leaves physicians to use the same laboratory references for athletes, even though we are not taking into account all of this as they do for a general population, often leading to the incorrect and misguided diagnoses. The athletes will have higher levels and we have said it before, and we will say it again. Athletes have higher levels of CK than non-athletes. Comparing them on a non-athlete scale is pointless, but it is the only thing we got. That is where sports specific ranges start to come in. How does that work? Do we have any of those? There is some research starting to come out showing some of the sport's specific ranges, but it is typically like soccer is one of them, and I believe rugby has one and it is simply based in paper. It is all what I would say, still theoretical.

They have not been adopted by any sort of provider group like the emergency medicine association. They haven't adopted those necessarily. it is just at least research is starting to come out to show yes, there are changes. Yes, we can make a difference. Asymptomatic CK elevation in the absence of symptoms or even minimal non-specific muscle symptoms. Some cramping, some spasm, some

fatigue, an elevated CK is simply an incidental finding. Now, the more severe those symptoms are, we could say, okay, that is a diagnostic finding. More correct diagnosis for elevated CK levels is what we call asymptomatic hyper CK-Emia. I know say that 10 times fast. Asymptomatic hyper CK-Emia, elevated CK levels in the blood asymptomatically. That is exactly how we break that down. Our evidence exists that CK levels will rise after exercise.

They will rise after heavy manual labor. This is why it is not just an athlete issue. If I am working in private practice, in a small town of farmers, but also have a heavy sport population, I will see the everyday farmer who has been outside all day working in the summer heat, throwing hay, which is heavy manual labor. They can be just as susceptible as my football player who's is training twice a day. Increase in exercise or any heavy manual labor, which is increased and cause CK levels to go up to 30 times normal within 24 hours of that activity. Gradually, over the course of a week or so, 7 to 10 days, that value will start to slowly decline.

That is why when I order repeat labs, I am looking to see, the changes. He was 30 times normal on Monday, but by the following Monday it is only seven times. That's a big trend downward and I now know we are going in the right direction. Our concentrations are typically going to peak, usually the 1 to 3 days, 1 to 4 days after exercise. Let us take a look at just a little bit more research as I think evidence based is where we need to be. A lot of my students I ask them, “What does the evidence say”? Prevention is key, that is what the evidence says. Future research should look at sport specific ranges and should explore contributing factors associated with developing ER. It should also evaluate a more accurate CK level for athletes versus non-athletes.

At this point I would be happy with a broader just a CK reference range athlete, CK reference range non-athlete. If I really wanted to just get greedy, I would say "I want a wrestling, football, soccer and rugby study”. Let us look at a few studies. There are 4 studies which I would like to highlight.

Study #1 Impact of Dehydration on Elite Wrestlers
This study was the impact of dehydration on elite wrestlers. It had 24 elite wrestlers who took in pre-competition weight last practices, they survey those and then there were the biomarkers.

Biomarkers were evaluated we look at the CK levels, we look at the AST, ALT, LDH and protein. We are going to be looking at something in terms of inflammation markers. Now, what researchers found was that serum CK and LDH levels were considerably increased after intense exercise. AST, ALT, CK and LDH are commonly used to evaluate muscle damage. Here is our pretest for our weight loss group and our pretest for a non-weight loss group. We can see higher levels in terms of pre and post.

Study #2 ER with D1 football. D1 NCAA division 1 athletes, research hypothesis that back squat work outs would trigger Exertional Rhabdo. What do we do? Let us do some back squat work outs. What we found was serum CK and creatinine from 96,000 to 331,000. That is a huge, huge spike. Go back to where I was saying how 50 maximal elbow flexors would cause a spike in CK levels. Here, we have back squats. The effective players went to muscle failure. They performed extra squats, you probably seen it if you ever been on the wait room with athletes. Some athletes are going to go extra, some athletes want to try and push their bodies to the extreme. They want to get better. Other athletes come up and ask, "How many of those do I have to do again?" This is the group that did extra work. They did not think that they could complete the workout because they are going so heavy. They were more likely to report symptoms of the affected players.

They did find that protein shakes may have a protective effect and decrease the risk of developing Exertional Rhabdo. One of the theories and this is my theory based on, what I know about nutrition and my background, it is going to be the fact that we have BCAs. We have the Branched-chain amino

acids, the proteins. As the body is looking for energy source, hopefully we are not going to be breaking down muscle tissue. We are going to be using the BCAAs form the protein. Risk of ER, was increased based on the perfect body weight lifting, the number of sets and the time needed to complete the hundred back squats. This is only the third study to report a cluster of ER among D1 athletes.

Study #3 ER + D1 Swimming
Here is a D1, Division 1 swimming, so 7th Collegiate NCAA Division 1 swimmers. They all presented with severe pain and swelling of the tricep. Also severe pain and swelling of pectoralis major and minor. What we had was dark colored urine. We got the hallmarks, we got the pain and swelling, we also have the urine. We take a look, all swimmers denied drug use, alcohol use, supplement use. They said that they are all fully hydrated, so that's great. The physical exam reveals that the tenderness over the palpation of the chest and arms. They had decrease fluxion, we have decrease range as a motion. That's a red flag, we need to think Exertional Rhabdo for sure. Non-pitting edema, non-pitting localized swelling of the bilateral triceps. For these CPK level range from 14,000 all the way up to 157,000.

Depending on what lab you are using, you can see some incredible values for CK, CPK levels.

Study #4 CK Levels Post 2-A-Day
We are looking at CK levels post 2-a-days. Post exercise CK levels were typically 5 to 10 times the upper limit for men and women. The first study to examine CK levels on football players from high intensity exercise. They were in a high heat environment and in a high humidity environment doing 2-a-days. Normal clearance of CK was typically 2 to 3 days, peak levels was about 18 hours as what they found. What that study did not examine was electrolyte imbalances, lactate dehydrogenase, did not examine myoglobin, it was strictly looking at what was the CK, CPK issue. What was that response?

At this point, you are able to diagnose, identify, understand and educate. Now the question is, how do you treat? What do you do? How can you be the best provider that you can be? That is where we come in to talk about sideline management versus treatment.

My first question to you is, do you think this is the same? I am sure some of you would be saying "No" because this is not the same. If I am on the sideline working with an athlete, if I start to suspect this, what can I do to mitigate the risk? I start to ask myself is oral hydration enough? If oral hydration is enough let's start getting hydrated. I need to consider how is the athlete? How is the environment. If it is the hot environment I need to get them out to a cool environment. What are my training variabilities? If it is a wrestler, we keep the wrestling room about 83 degrees. I am going to pull the wrestler out and we are going to get some fluid hydration, sit in the training room to cool off, and see how we are doing versus treatment. If I think they still need further treatment which is often the case, by the time an athlete comes to you, sometimes the hospital is the best place. A lot of athletes like to wait until the last minute, they don't like to tell you that they are not feeling well. All of a sudden, they come to you and sure enough you need to go to the ED. Can they go by personal car, or can they go by ambulance? Are they stable? If they want to go by personal car, the question is, do I feel safe with them going by personal car? Do I feel comfortable with another athlete taking them? Sometimes the athlete will say "Can I have my friend take me?" Well, maybe, but if I think that you are at jeopardy, our hospitals 20 minutes away, if I feel that you can't make that 20 minutes without having some sort of adverse effect, I don't want to put that pressure on to another athlete. I am going to say, no, we need to call an ambulance. If I think it is that severe the ambulance the paramedics when I get there I could start IV fluid resuscitation at that point. I can start the fluids you can at least start to get the fluids and then by the time I get to the hospital I have give you 500 ml. Once you get to the hospital, the athlete is probably going to get at least another liter and a half of fluid at ED. Here is our treatment

overview, mild cases may go undiagnosed and maybe manage an outpatient setting with oral hydration and rest if I catch it early enough.

I see a lot of providers around me, who have a conservative management. They will get some IV fluid replenishment in the ER but then it is just no activity for a week. Conservative management consist of rest and rehydration and that can simply be adequate. If I am in the clinic setting if I am in the hospital-based setting, I am probably going to get some intravenous rehydration, a revaluation of my labs before I go. Typically, what we find is we find lactated ringers, LR or 0.09% normal saline is being our two most common fluids of choice. With the rehydration go about 300 ml per hour. Now, the need for hospitalization is really based on the intuition of the provider and I respect that. There are certain times that my gut says something, and I just have to listen to. I must send you for an x-ray, I have to send you to the hospital. It's just there's certain red flags, certain things we hear in our history and our presentation that make us think this is going to be best for you. It is up to the provider in most cases. This decision is often derived from the physician's intuition and not necessarily based on simply laboratory findings, it is again treating your patients first. You can have some crazy laboratory values and things going on but treat your patient first. With the potential of more serious consequences developing, the health care provider may just want to keep you overnight. Just keep you there for observation make sure nothing happens, repeat labs in the morning. Everything looks fine, you will get discharge. From the sideline perspective, if you work weightlifting, if you work swimming, if you work football, wrestling, or running I need you to know that you are working a sport that has a high index of suspension. Have a high index of suspension if you are involved at any of those.

The World Games 2022 are coming up, and if you are working the body building section, the weightlifting piece those are athletes that we just need to be concern about. Summer in anywhere, but summer in the south it could be hot and humid. Any sort of outdoor sport, any sort of swimming sport, these are all things that would be really beneficial just little keys as I go into that environment. I am worried about XYZ , not so much worried about hypothermia and frostbite in the middle of July in South Florida. It is just not something that I am concerned about. Here in Iowa currently, I am worried about frostbite for a lot of athletes, especially anything outdoors. Patient history will help lead to your possible diagnostics, then eventually to diagnosis. With myoglobinuria can easily be detected in the clinic or sideline setting with the dipstick analysis. That is going to be one of the simplest and easiest ways, inexpensive too so I would suggest just buy some of those. Definitive treatment again ER, got to the hospital you will get dipstick, UA and the ED usually which can be positive with the presence of blood but microscopically is not going to show red blood cells. That's going to tell you that the dipstick was finding the myoglobin. CK values will aid in diagnosis but one of the challenges that even an emergency medicine physician or sport medicine physician is going to have his determining whether that is clinical, in terms of pathology or is that just physiological. Being able to differentiate physiological ER versus pathological ER is going to be big.

Hospital admission maybe required, you may end up getting 4 to 11 liters in the first 24 hours depending on what your kidney function looks like. This is the time that we want to try and prevent acute renal failure. If I am going to get 11 liters of fluid into them, that is going to be a lot of weight to cut. Then it is just your job as provider to make sure they are cutting it right, because the athlete is just going to immediately say "All right, I will just dehydrate myself again" and then we are back into the same position. Once treated the last thing an athlete really need to know is what's my return to play protocol. What's my return to play? A return to play is basically evaluating and assessing risk factors. This is a further complicated by the fact that there's no standard in terms of laboratory markers, like I said I just have to evaluate all right you went in on a Saturday you were four times CK and now you are one-time CK, you are trending downwards after 7 days of no activity you have

improve range of motion. You have full range of motion, you have no muscle pain, I feel comfortable you are starting back with some light activity. Laboratory Testing could rely on those biomarkers as a baseline. An athlete's physician should be closely consulted, and the patient must be closely monitored during a return to play. Luckily for me I have a great relationship with the athletic training staff. In fact, there is an athletic trainer and myself at pretty much all practices.

If I want to release an athlete back, I just have that conversation with the trainer it is possible that if you are in the office setting most days of the week, have that conversation with the trainer who is probably at the practice. Make sure that they know that they have to watch them coming back just because you just because you have been released does not mean you're fully released. Think of your concussion protocols, if I have a concussion, I have a gradual return to play it is not just, now go back to do everything you did right before you went to the hospital. Sports Medicine Physicians and ER, emergency medicine physician should be familiar with the high-risk patients. Those are ones with sickle cell disease familial history of rhabdo, any sort of history of hypothermia, those were recurrent episodes of ER. If this is my third or fourth time exertional rhabdo I need to be concerned about that. Patients deemed high risk, should be referred for further follow-up, an examination for further studies. Then when prop diagnosis and treatment is initiated, honestly this has a great full recovery and full return to play with usually minimal downtime of about a week to 2 weeks at most. This really is as serious as it can be, is something that we can manage quite effectively. Then finally the Champ Guidelines, which is broken down into phase 1, phase 2 and phase 3. I find that these are helpful, 72 hours of no activity and oral hydration, get good sleep, after the 72 hours you can repeat serum CK. I typically repeat after a week. That is what the ER Doctors around here wanted. If it is something in my personal practice, if I catch it, I can follow champ guidelines and still be within the standard of care. I never override an ER physician though. When ER physician sends back a discharge that says repeat labs in 7 days, I am not repeating labs in 4 days to try and get those athlete back any sooner.

Then phase 2 is your light activity no strenuous work, physical activity at their own pace. Then phase 3 is your gradual return to regular sport and physical activity. Quickly, because I just feel like with my background I would be doing an injustice if I didn't talk about at least a little bit of nutrition. Some research has suggested that consumption of protein shakes may possibly protect against the ER. That is something that is beneficial to know and usually what the other studies have found is because of those VCAAs. Those VCAAs can also reduce serum levels of CK. You may have heard before that creatine monohydrate could cause exertional rhabdo. The most recent study that I found creatine monohydrate was extensively studied and it does not play a role into the development of exertional rhabdo. Then proper hydration is absolute key.

Here are some final key points that I want you to take away. I want you to remember some red flags for me. Patients presenting with severe muscle pain, with decreased muscle strength or myoglobin urea, we should all be worried. Common findings increased myoglobin and increased CK levels those are two things that you really want to be focusing on. Diagnosis is often made with the help of laboratory findings good clinical history. As we take some of those takeaways home, right exertional rhabdo is strong in the eccentric component some muscle lengthening. Downhill running, negatives, delayed on-set muscle soreness, which is I am sure something that we have seen several times in a given season for our athletes, has been suspected as being a mild form of a rhabdo. It maybe suspected after athlete presents muscle stiffness following eccentric exercises and is often likely due to the inflammation and sensitization of nociceptors sectors. This is where that pain comes in. Exercise related muscle injury can be considered a continuum from DOM's to rhabdo, keep that in mind. More than half of all patients do not report muscle symptoms. Even though I said that's one of the triad that we want to look for, half of all patients never have the muscle symptoms, they never have the stiffness,

they never have the soreness. Myoglobin urea is found in only half of patients. If an athlete comes back to you and says "Yeah, it is just yellow urine" it does not mean that they do not have it. We cannot exclude the syndromes simply based on that having myoglobin urea. Then less than 10% of all patients present with all three conventional symptoms. At the end of the day, now that I probably really confuse you even more, I want you to know that this all comes down to your ability to question the patient, take a good history, get to know your athletes. This is where you are going to be able to pick up on those things to be the best clinician, the best diagnostician that you can be.

It is imperative to understand that exercise can increase CK, CPK levels in blood. Research is needed to establish specific references. At this point, I just want an athlete versus non-athlete range. Bringing awareness to healthcare providers regarding diagnosis, treatment and management. Mini athletic training and exercise physiology textbooks nowadays simply do not cover ER or just never talk about it altogether or give you a very poor clinical picture that is nowhere near this presentation. Healthcare providers working with athletes, at the end of the day we need to educate. You need to educate the athletes, needs to educate the coaches, need to educate the other health care providers around us, so that we can promote and practice mitigation techniques so, the education dissemination of appropriate information. Maybe we create a flier, something we put up in the training room. I have pull some things from the Olympic committee and put up in the training room for our athletes, just because there is a lot of good resources out there that they do not get to see.

We are going to run through just a couple of Case Studies.

Case #1

24 year old Caucasian male presented to a local clinic with vomiting, muscle cramps and low urine output. We got the cramping, vomiting so dehydration coming in and low urine output. Dehydrated. He received IV fluids 500ml at the clinic and was referred to the emergency department for re-evaluation. Once in the emergency department it was found out that he had lost 17 pounds in 2 days trying to meet his weight for an upcoming wrestling tournament. It was confirmed that he was experiencing muscle cramping, dehydration, and decreased urine output at FED and actually unable to urinate and when he did it was brown. My question is, what is next? The typical workup was this. Got a CBC, CMP, CPK and a UA. This athlete had these findings, his CPK, everything about the cramping, everything about the urine you thought instantly "he is going to have a high CK level." His CK was 460, about 2 and a half times what their upper limit was. If you take a look, dark yellow, specific gravity hydrated and then trace-lysed amount of blood. This patient got 2 liters of normal saline, was placed on no activities for five days in a gradual return to play. Final diagnosis rhabdo. My question is, did that meet the criteria for rhabdo? Rhabdo, dehydration, muscle cramping and low urine output. Well, here he is return to play, he follows the 5 day recommendation. Six days following the ED visit, we did follow up lab work. Patient began experiencing muscle tightness at the practice. Did not tell anyone. Seven days followed the initial ED presentation, two days after a return to play, his pain has increased back. It was newly developed nausea, vomiting, muscle pain, flank pain was evaluated in the clinic. This time more labs were ordered. Again, CBC, CMP, CPK and UA those are all re-ordered.

CK were still 353, BUN does not look to terrible, color was just called darker. Sodium and Potassium were at the normal ranges. At this point, the ER doctor did this, we had 2 liters lactated ringers. He was pulled for a week now and he was told to consider metabolic myopathy. This is a repeat, just one after another. A repeat incident of Rhabdo, the athlete was told to consider metabolic pathways or metabolic myopathy. He was diagnosed with heat exhaustion, hyper uricemia, elevated CPK, acute kidney and dehydration. If you look, nowhere there was the word rhabdo or exertional rhabdo mentioned. Even though were being treated as such.

Case #2 Clinical example number 2, 21 year old African American male. Based on everything that you have learned so far, maybe a sickle cell trait but he presented to the ED by EMS after being choked out at a tournament. The shortness of breath following a wrestling tournament, he passed out, when he regains consciousness, he know that he was having trouble breathing, difficult to swallowing and his voice was different. We start to think maybe we have a crush injury at the larynx. He goes by ambulance, get's a CT. They, of course, do labs. He admits in the ER that he had been cutting weight for several days. He has not been starving himself though, that was his quote but the last time he urinated was before today's match. Diagnostic was CBC a BMP just a basic metabolic panel, CK and then we are looking at the heart now, CKMB. CK, 1,220, so we are definitely over that five times upper limit, we can meet criteria for the CK values now. UA was not obtained, obviously the ER felt more strongly on breathing issue, which I do not necessarily blame them.

If you take a look at the treatment that he got. He got 2 liters of normal saline, dexamethasone, Iopamidol and was discharged. A lot of this was for the upset stomach. Discharge return home with the team. Diagnose with a choking injury, mild Rhabdo based on the CK. Just like our last individual, once he was home, he woke up in the middle of the night, was feeling dizzy, felt bad, and ended up having a syncopal episode in the dorm. Roommate called 911, EMS arrived took him to the local ED, this sure look awfully familiar. This is pretty much the work up of our local hospital, got a CBC, CMP, CK/CPK and a UA. At this time this is where I am telling you and showing you that all they say is greater than a thousand. He was 1,220 at the last hospital, he is greater than a thousand at this one. I do not know if he was still going up, based on what I know and based on the research that I have had, he is continuing to go up. AST was altered, we start looking at amber urine. What are the red blood cells? His treatment was a little bit different. He had 2 liters of normal saline, some Tylenol for the pain, Zofran for the nausea. He was admitted overnight for observation because he did have a syncopal episode and they were worried about the breathing at that point. His diagnosis was recurrent Rhabdomyolysis even though he just been diagnosed 8 hours before it reoccurred, mild hypokalemia, inverted T-waves which they consult towards a cardiologist. Cardiologist came back and said that was a normal juvenile persistent pattern. Then on CT, they did find a cyst of the panela gland which was just a simple incidental finding.

Case #3 - 18 year old male presented to the ED by athletic trainer. Athletic trainer following a history of 2 syncopal episodes. First syncopal episode was earlier in the day and the second syncopal episode was inside the locker room after wrestling practice. Patient was vomiting over a toilet when he passed out for approximately 1 minute. No other neurological deficits were noticed. One's in the ER it was found that he had bilateral flight pain that he rated 3 out of a 10. He had other muscle cramping and aching. Patient said he had a history of colitis, initially stated that he had been working out that day, was drinking some water but later admitted that he had not been drinking or eating for several days as he was trying to loose weight for a wrestling match. Local Hospital ordered the CBC, CMP, CPK and UA to which we find pretty elevated CPK levels were 848, upper end is 170, we are getting pretty high to those five times limit. Urine is amber, we are dehydrated with our specific gravity and that was all the data that we really had. This patient, 2 liters of normal saline, CBK remains elevated but has treaded downwards. This patient spent some time overnight, has trended downward. Patient should not be at any undue risk of discharge, he was then discharged with the diagnosis of elevated CK, not even Rhabdo, elevated CK and dehydration. I would argue that you could have put rhabdo here if you wanted to follow the other clinical presentations such as the cramping, urine being darker and the elevated CK he would technically be able to meet it based on current standards.

Case #4 - 18 year old male presented to the ED by athletic trainer again, after a history of 3 syncopal episodes following strenuous activity. He said that the syncopal episode had occurred in the weight

room earlier that day. He felt very light headed, leaned up against the wall and felt as though he was going to pass out. Similar episodes occurred on Monday, 5 days earlier. When he went to the emergency department, patient denied any pain. He did note some muscle cramps in his hand and legs. Stated that he was trying to cut weight for an upcoming wrestling match but he had not been drinking and he did report a history of migraines. At this point, diagnostic workup looks pretty similar to everybody else because it is the same hospital. Here we go, we found CPK levels greater than a thousand again I cannot tell you what they were because I simply do not know. BUN was elevated, urine was amber, specific gravity was showing dehydration. Here is his diagnosis, elevated CK levels and dehydration. The consistency here from the same hospital, different providers is everything from elevated CK to Rhabdo to persistent Rhabdo. This is all coming down in my professional opinion just showing why we need better diagnostic criteria. I am not saying that any of these athletes were treated wrong, as they were not treated wrong and the standard of care was absolutely applied but for what is there is so inconsistent. He received 2 liters of normal saline and normal kidney functions, CK was starting to trend downwards so, he was released.

All of that is what led us to write this paper. Doctor Thomas is an ER physician, board certified and I worked on this and it was our paper for the re-evaluation of the diagnostic criteria for Exertional Rhabdo and collegiate wrestlers. In research you want to pick a topic, my topic was topics was population of collegiate wrestlers. Though I do believe this really applies to other sports, but this was a case serious. It was a case that I just run through, that is why it went kind of fast because you have the ability to go and find this if you would like. Then, it is a review of the literature, which really covers a lot of what was presented here today. I greatly appreciate your time. I hope you learn a thing or two.

I hope you really enjoyed the presentation. I hope you found it to be of value. This is a topic that I particularly enjoy, not enjoy necessarily dealing with, but it is definitely something that I think a lot of providers will see and just may not fully understand. Having the opportunity to share that with you, share some of this knowledge, and share some of these resources is truly an honor. If you have any questions, please by all means, reach out.

To FICS to the ICSC you are more welcome to contact me, they will have a way if you have any questions, comments, or concerns about this presentation, I am more than happy to speak with you.

[END]