

ICSC01 Head Injuries

Section 2 – CTE

Instructor Rob Reid

Video Lesson: 18:05

I am going to be talking today about Chronic Traumatic Encephalopathy. We are using the term CTE because that is too long. The question we have is what if we were wrong about CTE and concussion? We'll talk about that a bit later and you will see where I am going with that. We need to talk about the definition mechanism of injury, and the histological process as much as we could, we know about, but also, we also need to make sure that we look at a potential problem. I have been around for a long time; 40 years and I am a Sports and Exercise Physician in Australia. I have been Chief Medical Officer for a lot of different sports and I have always used a team approach when I look at these. Whether I am working with an athletic trainer or a sports trainer or another doctor or a chiropractor, or an osteopath, it really doesn't matter to me. I think the athlete's well-being is the most important part of the thing, and I am just part of the team. There is no "I" in team.

Let us recap on concussion itself. Sports-related concussion is traumatic brain injury induced by mechanical forces. Now, those forces may be direct to the brain, or they may be to the face neck or somewhere else in the body that transmitted to the brain. As I see it means if there is a rapid short-term impairment of neurological function that results spontaneously. This is important because it means that it is a functional problem, not a structural problem. It does mean that it doesn't last for a long time or do we don't have consequences? It just means the definition is that, so, they cannot be explained by things like drugs alcohol, medication use, the interesting, they probably maybe in some cases, be able to be described by cervical injuries, but that may be the cranio-cervical junction but that is not very well recognized in the medical profession, but you guys know much more about this than we do.

There are other co-morbidities that are as well, and we need to keep that in mind. The concussions resulting in neuropathological changes, but the signs and symptoms should reflect its functional disturbance, rather than a structural injury, there is a range of clinical signs and symptoms that may or may not involve loss of consciousness, and what we need to remember is that only 10% of concussions are associated with the loss of consciousness. That doesn't mean that they are not all severe, but it just means that the concussive episode that produces a lot of consciousness is not present in all of them. What about the definition? Well, The Concussion in Sports Group, we met in Zurich in 2016, sorry in 2012, but also in Berlin in 2016 and so it is defined as not having any structural abnormalities. It is a historical term that representing low-velocity injuries of corner because our brain shaking, as you can see there. It is a subset of traumatic brain injuries and that's what I am going to talk about. In Europe, they sometimes talk about commotio cerebri as the definition, but we'll talk about concussion.

mTBI which is Minimally Traumatic Brain Injury is not a concussion. It means by definition, it means that there is some structural damage and this is out of the context of concussion itself. It is used interchangeably in some of the U.S. literature and offering that confusing and they should not be used interchangeably. It is a clinical diagnosis made or presumed at the time of the injury. You cannot go back later and say he did not have a concussion, it is very, very difficult. You cannot go back on a Monday or Tuesday after an injury on the weekend and say, well, he didn't have a concussion because it is made at the time of the injury, not when you see them later on, that is whether you are a medical doctor or chiropractor, or anybody else. It can be over diagnosed, but mild concussions are more commonly under-diagnosed, it is symptoms of mild can be delayed, they can be delayed for 24 hours. So, you need to be aware of all of these sorts of things. Tim Stark made an interesting comment, We need a higher level of competency in the outcome clinicians to investigate these things deeper because some of the things that we see in the Medical Profession and not explained by concussion itself but maybe by other problems that Sports Chiropractic can diagnose better and

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so Sports Chiropractor is should be one of the opinions especially when we've got these difficult ones that don't follow the protocol that they are supposed to. So, Sports Chiropractic should be one of the opinions and I agree with them.

Whiplash, these are interesting. Whiplash has been defined as an acceleration-deceleration force transferring energy to the cervical spine. We have got a head sitting on top of that and the problem is that whether it is the cervical spine, or the head, sometimes it is both and some of the problems associated with concussion, some of the symptoms and signs can be problems with the upper cervical spine, and the cranio-cervical junction. There is some very interesting work in this area done by Dr. Scott Rosa in the United States, looking at changes at the cranio-cervical junction, which I find very fascinating. I cannot do it. Well, I can diagnose it, but I think it is fantastic.

The mechanism of injury for concussion. A blow to the brain sets off a flood of all sorts of things that occur, there is an injury to the brain, it is that the whole of the brain that's affected and because of that there are all sorts of problems associated. Changes with an influx of calcium into the neurons and then because of this an extra energy that is required by the neurons to try to get better, but the changes also limit the supply of the brain fuel, which is glucose. So, the higher energy demand restricted flow and oxygen debt, and glucose debt create an energy crisis. This leads to the mental confusion failed memory and may take days to restore the balance. We need a full recovery before athletes return to play, but we need a full recovery which might take longer than we think.

CTE is defined as a progressive degenerative, disease of the brain. When people with a history of repetitive brain trauma often athletes, but not always. It is symptomatic concussions but also, we think that asymptomatic is sub-concussive hits, play a part as well. It has been known to affect boxers, it was called the punch-drunk syndrome would dementia pugilistica, but we think it is because of these concussive episodes and Boston University CTE Center is doing a lot of work on this. Obviously, this is a big, big thing in the states. So, the NIH had a CTE Conference in March 2015 and the neuropathology which is pathognomonic of CTE is an accumulation of tau which is a protein in the neurons and the glia at the depth of the cortical sulci. The problem with that is that it can only be diagnosed after death because post-mortem, you're a pathological analysis is required to find this tau protein. We cannot use any of the normal methods likes MRI, CT or anything like that to diagnose CTE during life. The concerns that we have is this, we cannot diagnose it during life, but we don't really want to get to death before we diagnose it. People are not going to donate their brains to science when they are still using them. It is a difficult conundrum that we face, but Boston University CTE Center and a lot of others are doing a lot of work in this area. Tau protein aggregation has been implicated as a result of amyloid protein aggregation, but it is interesting that we actually, we are going to come onto the difference between this and parkinsonism or Parkinson's disease later on.

A cause-and-effect has not been confirmed we would like to, but we think that repeated brain trauma triggers this Progressive degeneration including the build-up of tau but it takes too long for this to occur to make it easy for us to make the connection. It could be decades after the last brain trauma or the end of the act that led to even caught involvement before somebody really shows the problems with CTE. We do not know the number of head injuries or the type or the amount of head injuries that are required to produce CTE and we also have found that not everyone with a history of repeated brain trauma has it makes it difficult.

When we look at this picture of Parkinson's disease, we can see that the substantia nigra is a lot smaller in somebody with Parkinson's. We see something similar in somebody with CTE. So, obviously, it is different to normal, but again, we need to pull the brain apart before we could find it. So, the degeneration in the brain is associated with the common symptoms and these can be all of those that are also post-concussive syndrome or symptoms that we can see. Parkinsonism is not one, but confusion, headache, depression, impulse control problems and things like that are all seen through all of these. Eventually, we get a progressive

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dementia and that is probably something that finally has these patients die, but again, we cannot describe it during life, unfortunately. The post-mortem analysis indicated that the symptoms are associated with the neuropathological changes, but their changes because there is atrophy of certain structures as well as degeneration of myelinated neurons. We have a difficulty in trying to describe this or define this during life, other changes with the degeneration of the neurons, etc. We have enlargement of the lateral at the ventricles and septi fenestrations and things that we again cannot define during life. We must have a brain on a table to be able to have a look at these.

Microscopically. There is accumulation in the neurons of tau protein, as I said before these aggregations and filaments called neurofibrillary tangles, but you don't need to know all of that. But there are other small vessels in the frontal and temporal lobes but also they are prominent in the basal ganglia. When we talk about parkinsonism, we talk about the basal ganglia as well, but this is a bit more diffuse and so, when we look at this and even if we look at Alzheimer's there is a very different look to these. Alzheimer's. Well, again is similar because the tau protein is increased, but they are different, they are amyloid plaques. What we see in CT is a very diffuse amyloid and tau protein aggregation. So, there are similar other brain problems, it is difficult to define whether they are on your genetic factors or whether it is just because of brain injury.

When we assess these, it is difficult there is a lot of different things that we look at difficulty thinking it involves behavior, depression, memory loss, difficulty planning, the executive functions of one of the things that that are important, but again, it makes it difficult for the person who has it because they don't have the executive function to follow it up, emotional stability. The problem is this can occur in other neuropathological problems or other diagnoses, which don't include CTE. We cannot make a definitive diagnosis on these because these are the functions but not CTE's defined as a structural problem. It is almost the opposite of a concussion.

What about prevention? Yes, it is important really important. Can we prevent it? We don't have a treatment for CTE. We cannot make these brains better when we can prevent them and we think that the way to do that is, because it is associated with a recurrent concussion, we need to do something about that. If somebody's had one concussion, we know that they are more likely to have another head injury. We don't know how long they will likely have another head injury, but we do know that there is some association, we don't know whether it is because they have not really fully recovered from the first one, or whether they have set themselves up, if you like for a second heading toward the recurrent, current recommendation is to prevent the mild traumatic brain injuries, but don't play sport that that project can produce these things, but we also need to prevent additional injury after a concussion. If somebody is completely back to normal before a return to sport, they are more likely to have any other injury that includes another concussion. We need to make sure that the concussion's treated properly.

Can we treat it any other ways? There are some interesting things happening at the cervical cranial junction or creating a cervical Junction I should say, some interesting studies done by Dr. Scott Rose in the States which a lot of you may be familiar with. He is talking about the change in some in this Junction, which may make a difference to some of the things that we see in CTE because of a change in CSF flow. The medical symptom system doesn't really understand what the CSF flow is or why we can sort of semi-define it, but there is some interesting studies done by Scott Rosa and by a number of other people too, who may be able to change some of the early symptoms and signs of CTE, but this is still in the early stages and we not sure that we have definitive changes that we can do with that yet. There are a few references there one is the Rosa Clinic by Scott Rosa also the two BJSM references are for the Berlin CIS, The Concussion in Sport Group meeting in 2016, and 2012 and Bruckner and Khan's book, has some good information in it as well.

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