Chapter 2
Consequences of Ignorance and Arrogance for Mismanagement of Sports-Related Concussions: Short- and Long-Term Complications

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Abstract The major objective of this chapter is to provide my insights on short, and most importantly long-term complications from mismanagement of sports-related concussions. There is an expanding data base accumulating in neuroscience and clinical practice indicating the danger of long-term brain dysfunctions grounded on metabolic and structural deficits in the concussed brain. Although the exact mechanisms of these brain disorders still remained to be elucidated, the medical professionals in charge of concussed individuals have to be knowledgeable about epidemiology, pathophysiology, and current developments regarding evaluation, diagnostic imaging, management principles, complications, and prevention strategies. Clearly, both short- and long-term consequences of a single episode or multiple sub-concussive blows should NOT be overlooked while assessing injured athletes. There are several myths about concussion that I will elucidate in my final statements of this chapter, emphasizing still existing controversies and discrepancies between basic brain science and clinical management of sports-related concussions.

Keywords Concussion • Myths about concussion • Second impact syndrome

Short-Term Risks of Concussion Mismanagement

It is fun to think about how history may have changed if we knew centuries ago what we now know. A number of drivers were praising, as I would too, Dale Earnhardt Jr. for bringing forward his concussion symptoms after the Talladega crash. He did this because he was aware himself, that he'd had a concussion a few weeks before in

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Kansas, and had concerned about his health. Drivers that were commenting were saying that just wouldn't have happened 10 years ago and that's probably true. NASCAR drivers driving cars are similar to fighter pilots on wheels. Their reaction times and their vision needs to be 100 %. I am very glad to see that he brought concussion issues to doctors even though it cost his team a great deal of money.

So, What Are the Short-Term Risks of Mismanaging a Concussion?

The most common result of not imposing physical and cognitive rest after a is taking concussion symptoms, concussion is greatly exacerbating concussion symptoms and causing something that would have recovered in a matter of days into something that may now go on for months and become post-concussive syndrome. Also, a much less common risk is *second impact* syndrome (SIS), which Bob Harbaugh and Dick Saunders first described in a JAMA article in 1984. It is interesting to me to see how my own practice has greatly changed in recent years, because of the awareness of concussions and post-concussion syndrome. I am actually inundated with post-concussive syndrome patients, most of whom have months of symptoms before ever see them.

Approximately 2 years ago, one of my colleagues and I, along with one of his graduate students wrote a paper looking at a retrospective analysis of 215 consecutive post-concussion patients. Those that had post-concussive syndrome had a disproportionate amount of a history of multiple prior concussions, as opposed to this being their first injury. Many of them took a double hit, which means your first hit may be to the head, and then you fall to the turf and slam your head a second time.

Double hits do seem to be associated with symptoms that last longer than a single hit, and may actually involve rotational forces, which are in one direction, and then rebound in the other opposite direction. Some suggest we should be thinking of those rotational forces as being summated or added. I will leave that up to further research that the biomechanists are doing, but it is a very interesting theory and it does correlate with what we see. The most common occurrence with post-concussive syndrome is athletes who are playing while still symptomatic. We wrote a paper a few years ago not about post-concussive syndrome, but catastrophic injuries. It was found that 38 % of individuals were playing while still symptomatic from a previous head injury that was sustained that season. We are finding the same thing with post-concussive syndrome.

Two young children cracking heads is unacceptable, and what we are realizing today, and what we are measuring today are primarily linear forces. For several years we have associated concussion risk being much higher with linear forces 80–100 g. We have also seen from the work of Dr. Guskiewicz and others, that just because you have 100 g impact, it is not necessarily associated with a concussion. Conversely, concussions can occur with *g* forces that are well under 100 g.

We now realize that it is not just those higher hits that are important. Today we realize concussion occurence is multifactorial and there is no known threshold.

I will try to make the case, although this has not been shown in the laboratory yet by Dr. Hovda that the sub-concussive hits count as well. In terms of how many hits kids are taking over a season, you can see at the college level that while the mean isn't terribly high, the extremes that the individuals are taking can be very high. Certainly at the high school level, over 1,000 hits a season is not uncommon.

Evidence that is emerging that I think should give us all cause for concern [1]. This data that of course needs to be replicated by other investigators, but it is data that shows decreased brain performance from repetitive trauma, even without a recognized concussion. This is presumably from the accumulation of sub-concussive blows over the course of the season. The trauma in this data has mostly been garnered from accelerometers in football helmets.

It is not just metabolic MRS studies that have shown changes, but DTI has shown changes similarly with baseline tests versus end of the season tests with fiber tracks integrity decreasing over the course of the season. Presumably, this is due to sub-concussive blows. Some of the studies have also used computer-based neuropsychological testing, and found deterioration in the test scores over the course of the season when compared with baseline. By using neuropsychological, fiber tract, and metabolic data, there is a suggestion that it may be these sub-concussive blows that are producing deleterious effects on brains. It is an accumulation of this data that has led me to start a book 2 years ago, and certainly the more controversial parts of the book are focusing on children in sports. I suggest taking tackle football away from youngsters *until the age of 14*, no body checking in ice-hockey until the age of 14 (which in the past 2 years has already almost happened with the age moving from 11 to 13), and no heading in soccer until the age 14. It does not make sense to me, that you have a very good batting helmet for baseball players, but then the helmet falls off of the player's head as they round the bases. This subjects them to epidural hematomas due to skull fractures from a thrown ball, when it would be so simple to put a chin strap that holds the helmet on the head.

Also, in sports that do not have helmets right now, but the mechanism of injury is a focal blow, meaning a stick to the head or face, like women's field hockey and lacrosse, I believe there should be helmets. Additionally, I feel very strongly and passionately that we are giving our officials a pass they do not deserve. They should either start calling sports appropriately, or they should be replaced. I think it is terrible that this is a problem that continues to go on. Pop Warner football was founded in 1929, although it was named in 1934 after Glenn Scooby Pop Warner. The organization has thrived, especially since the exposure that it has received on television.

With regard to neck strength, force equals mass times acceleration (g), or force divided by mass equals acceleration. Acceleration is what the brain is experiencing during a hit. If we look at it a different way, change of velocity over time, you have a much less change of velocity with a well-developed neck. Dom Comstock and I, as well as a few others have been working for a number of years on a study, testing neck strength with regard to concussion incidence. The data has correlated that the strongest necks have the lowest number of concussions, and the weakest necks have

the highest number of concussions. Others have found the same theory, that neck strength reduces your risk of concussion. For example, the woodpecker has a large, strong neck, and it moves only in a linear straightforward manner—a woodpecker does not experience concussions, " … never have headache." We are a strong believer in any athlete who is involved with a contact or collision sport should strengthen their neck muscles as well as their other core muscles as much as they can. Youngsters and females don't have testosterone and are not going to bulk up their neck muscles, so they aren't going to look any different, but they can strengthen them.

We do not know what the concussion threshold is today, but we do know that there are curves which suggest that the risk certainly goes up as the linear forces go up. Many of us feel that the rotational forces are more important than the linear, and that is really what should be researched further. This is especially true in sports where the head is swiveled on the neck from a hit like a helmeted football or lacrosse player. The reason that I believe that we do not yet have concussion thresholds despite a lot of good research is that we are not dealing with just biomechanical issues when we think about concussion. It is a complex situation. Yes, linear accelerations need to be known, rotational accelerations would be ideally known as well, duration of impact, location of impact, and tissue strain issues are all very relevant and in need of study.

However, there are also biological issues. There are rarely situations where the biological issues are matched up with the biomechanical situations to truly allow you to look at the whole picture when you talk about concussions. Some of the biological factors include history (how many concussions has somebody had, how severe were they, and what is the proximity of them), anticipation in terms of neck strength, age, gender (girls are more prone), hydration/volume (some science suggests that a dehydrated brain moves more inside the skull and is at greater risk of injury than a well-hydrated brain), as well as underreporting.

Concussions: Structural Versus Functional Brain Disorder

Dr. Ann McKee has found in some individuals that have died shortly after a concussion because of suicide, diffuse axonal swelling as well as axonal damage. In my opinion, concussion in part involves a neural metabolic cascade, a metabolic issue, but in part at least in some concussions, is *also a structural issue*. I believe that as we get better at MRI imaging, like DTI [2], we are going to be able to see those structural changes in concussion that we cannot currently see with routine MRIs. It is common knowledge that concussions are prevalent without loss of consciousness more than 90 % of the time, and that presents a problem. For those of us that have been on the sideline, and I was for a lot of years, it is not a great feeling knowing that we are probably missing multiple concussions for every one of which we are aware.

Subjects' Reports Versus Pure Evaluation

Theoretically by asking after the season, when there is no longer a feeling of possibly letting down their coaches of teammates, the athlete will be more likely to give honest answers. The incidence of concussion reported by individuals from these post-event studies is six to seven times what is known on the sideline. Some years ago we were a part of a Canadian study in which Paul Echlin was the lead author. This study looked at the incidence of concussion in junior "A" ice hockey that was reported from people on the bench, as opposed to physician observers in the stands. The physician observers in the stands had the responsibility of looking at people on the ice. When somebody got up slowly and seemed to have a problem but stayed in, they would go down between periods and examine them to determine if they had a concussion [3].

A seven times greater incidence of concussion was found from the physician observers, as compared with medical personnel on the bench. The national football league knows this is true as well, and that is why it now has athletic trainers up in a booth looking at the same television feeds that you and I get when we watch the game on television. The same feeds are now fed to the medical personnel on the sideline to be used as part of the concussion assessment. There have been multiple examples of individuals who were sent off the field and a body part was examined that was not the head, when it was a concussion that they had sustained. Colt McCoy is the most notable of them.

I am very pleased that Chris Nowinski, co-founder of the Sports Legacy Institute, and I held a meeting, dedicated to documenting the number of impacts occurring as well as potentially identifying what the threshold number should be for cumulative hits. We know that the numbers that youngsters are receiving are appreciable, we know from published data that roughly 60–70 % of those hits in the past have occurred during practice.

Coaching Preventive Strategies

If you change the way that practice occurs, you can dramatically reduce the number of hits individuals are taking to the head. The *winningest* college coach in this country is John Gagliardi from St. John University outside of Minneapolis St. Paul. He has over 800 victories, and during the season over the last 50 years he has never allowed tackling, only games are full contact. During practice the skill drills are all done and people tackling, but do not bring players to the ground—they just wrap people up. The NFL certainly gets that message too, because in the collective bargaining agreement during the 18-week long NFL season, players can only hit 14 times, less than once a week! Things are changing, and what we are doing is taking the trauma out of practice.

We protect little league pitcher's arms with good intentions, without question when we limit the number of pitches they can throw. However, medial collateral ulnar ligaments can be replaced and the arms can come back from high school to pitch in the big leagues. There are many examples of that including some big league players that are on their third Tommy John surgery operation, and still pitching in the big leagues. For a correctable condition, we have pitch counts for youngsters. We think there should be hit counts to the head because obviously the brain cannot be replaced.

We can modify how practices occur, and to their credit, Pop Warner football has reduced drastically the amount of hitting that they allow in practice. I think that we have an issue in soccer with regard to heading. We have studies that show structural changes in individuals' brains that have headed more than 1,300 times in a given season.

Second Impact Syndrome

SIS [4] is simply an individual that has sustained an initial brain injury, who while still symptomatic, sustains another brain injury that may be incredibly mild. What usually happens is that within minutes, there is a loss of autoregulation, which leads to massive blood flow inside the head, and increasing intracranial brain pressure. It is the capillary beds in our brain that have the ability to be dilated and hold extra blood, as do the arterials. In *SIS* this autoregulation, which keeps a constant flow of blood to our brain, is disrupted. In a normal situation, if your blood pressure goes up you find a constriction occurring in the arterial bed to keep a constant amount of blood flowing to the capillaries, and then to the tissue that needs it. On the other hand, the blood pressure returns to normal and the arterials go back to their normal size keeping the same amount of blood flow. When blood pressure goes down, we have dilation in that arterial bed to keep the same amount of blood in your capillaries.

What happens with an *SIS* is that the autoregulation is lost, and with blood pressures that are normal or even above normal because of adrenaline flowing from either pain or exertion, you find dilation in the arterial bed [4]. When that happens with normal or heightened blood pressure, you have a massive accumulation of blood in the capillaries of the brain. The brain inside the skull houses spinal fluid, brain, and blood. If you massively increase the amount of blood that is inside the blood vessels, you will massively increase intracranial pressure and cause brain herniation. Essentially, that is what we are seeing happen. *SIS* is usually bilateral and symmetrical, but it can occur unilaterally and it can occur with a small sliver of subdural hematoma [4]. The subdural is not causing much mass effect; however, it is this vascular engorgement of the brain that is causing the mass effect. This is not vasogenic edema, because there is grey and white matter differentiation. It is a massive increase of the volume of the brain due to blood.

Long-Term Risks of Mismanagement of Concussion

One of the long-term risks is prolonging your post-concussion syndrome, and the other is the issue of chronic traumatic encephalopathy (CTE). At Boston University we started out with four directors at the center for the study of traumatic encephalopathy [5]. Chris Nowinski, concussion advocate, a neuropsychologist Dr. Anne McKee, a famous for her work in CTE, and Bob Stern, who is a co-director. We established a brain donation registry, which now has over 500 people enrolled. We are hoping to register and study brains of asymptomatic individuals that live normal lives, and yet play contact sports. Right now we have over 130 brains, almost all of who have come from symptomatic people or those that have committed suicide. The symptomatic people you could have predicted would have had CTE by the emotional, behavioral, and cognitive symptoms they had.

What we do not have many of, are brains of individuals that were not symptomatic, but they will come from that registry. Bob Stern as P.I. I, along with Chris, Ann, myself and a crew of graduate students, are doing a longitudinal clinical research study on over a hundred NFL players compared to a group with no recognized brain trauma over the course of their lives. Structural issues using a variety of MRI modalities, magnetic spectroscopy, DTI, volume averaging MRI, and also biomarkers are being used to see whether or not we can have a profile that correlates highly enough to make a diagnosis of CTE in living people. The other part of the center is the brain bank with Dr. Ann McKee, which has gotten the majority of the exposure to date. Do you know who first described CTE? I asked this question recently at a conference, and immediately a hand went up and said Bennett Omalu. He did describe the first in National Football League players. Harrison Martlin described dementia pugilistica, he described the clinical syndrome, which is CTE but did not use those words. Well, I will give you the answer, because it was a graduate student that we are working with. He would call it mentoring, but I am not sure who is mentoring whom because he enlightened me to the answer of this. There is a British Medical Journal Article from 1957 by Macdonald Critchley, a renowned British neurologist, and I was aware of that article because a few years ago we did a book called Boxing in Medicine. In another book that was a tribute to Clovis Vincent that came out in 1949, a number of individuals were solicited to write chapters, and it was bound up in a volume and in the book there was one chapter written in English. In that volume the CTE of boxers was written by Macdonald Critchley (1949); therefore, it was the first time that was described. In NFL players Bennet Omalu was the first to recognize it, and he published the first case of it. Currently, CTE can only be diagnosed with certainty after death. You can actually have a very high clinical suspicion if the right clinical profile is there, but you cannot be 100 % certain.

Ann McKee's brain bank is the world's largest now in terms of CTE. Her first case was John Grimsley, which is an advanced case. The medial temporal lobe is just riddled with this staining identifying *hyperphosopholated tau protein*. Dave Duerson's brain was studied, and it was found that he had a moderately advanced case of CTE. Recently there was an article by Lahman and colleagues, who looked at death certificates of a number of NFL players who played over a 10-year period

in the NFL, and they all had to play 5 years or more to be included in the study. When looking at the death certificates of these individuals, they found that the incidence of Alzheimer's disease and amyotrophic lateral sclerosis (omitting Parkinson's disease) was four times higher than what would have been predicted by the national average. We don't know from our work in Boston what the incidence of CTE is, and what the prevalence of it is in any population. It is unknown in the National Football League where we have the greatest number of brains. We know it occurs, and we know that it occurs in a very high percentage of those brains that we examine 45146, but we also know that those brains are an extremely skewed samples.

These brains were not studied, only the death certificates were looked at. These death certificates are filled out by a doctor which is never a happy task—and often a task that subsequently ends up being done as quickly as possible. This leads the information to not necessarily be as thorough as one would hope.

Furthermore, what is being described as Alzheimer's disease can possibly be chronic traumatic encephalopathy. These brains were not studied, so we will never know. The same is true for what is being described as amyotrophic lateral sclerosis, which is probably chronic traumatic encephalymyotrophy, which is a variant of CTE that Ann has described (along with the rest of her colleagues) in NFL players. CTE in most people is a progressive neurodegenerative disease believed to be caused by repetitive trauma to the brain which includes sub-concussive blows. This is NOT a prolonged post-concussive syndrome, nor is it solely the cumulative effects of concussions. Symptoms characteristically, although not always, begin decades after the individual has stopped sustaining brain trauma. The one sport with a fairly high incidence of CTE is boxing. It is fairly common that individuals in their 30s have already started to lose some foot speed, developed slurred speech, etc. We need to know about the risk factors for CTE, and we need to know how we can differentiate them from other situations. Suicide is associated with head trauma, including concussion. In terms of increased incidence, it is also associated with CTE; however, we do not yet fully understand all of the factors involved. It is certain that we do not know the prevalence or incidence of the disease, but we certainly know that we do not want to see anymore of our heroes having their brains examined because of suicide.

Concluding Statement: Myths About Concussion

Myth Number One: You Have To Be Hit in the Head to Have a Concussion

I think that most people now know this is not true. Just from whiplash you can have a concussion, from a blow to your back that snaps your head back or a blow to your chest which snaps it forward, or from a fall on your butt where the forces go up the spine. Of course, when we look at our blast victims, at least in our models, it isn't the pressure wave that is producing the concussion. It is the blast winds that are

associated with it that are causing the head to shake violently and oscillate 10–14 times. This event can give somebody a lifetime of concussions from one blast.

Myth Number Two: You Have To Be Rendered Unconscious to Sustain a Concussion

More than 90 % of athletic concussions do not involve loss of consciousness.

Myth Number Three: Helmets Prevent Concussions

It is possible this could be true, if it were big enough, paired with enough energy attenuating materials maybe, but it is not practical. This would also be putting the neck at risk, so it is not going to happen. We are, however, getting better helmets all the time, and I personally am a strong advocate of going in that direction and not going in the direction of less protection. It is amazing how topics such as this have made their way into the media, because they are things that society in general needs to think about and know.

Myth Number Four: Mouth Guards Prevent Concussions

No, it is not only mouth guards that claim to prevent concussions. I am not against any of it but I am just against claims that cannot be substantiated.

Myth Number Five: You Can Always See a Concussion

You can always see if somebody is unconscious or if they cannot stand up, but you are not going to see most concussions. Most concussions are subtle, and it takes time, especially with mild concussions, to sort out whether somebody has had one or not.

Myth Number Six: Your Next Concussion Will Be Worse Than Your Last

Wrong. A Bruin's player scared for had his first concussion that consisted of four and a half months of symptoms, causing him to lose a season. With his second concussion, he experienced 2 weeks of symptoms, lost a month of playing, and was back playing the rest of that season. With his third concussion, he had 4 days of symptoms, and was back in 2 weeks. That was an exception, and it is not usually

what we see, but it demonstrate that every concussion is unique. You cannot predict what the next one is going to be, unless somebody is on a trajectory that they are more easily concussed and each concussion is lasting longer.

Myth Number Seven: Three Concussions and You're Out

This myth really frustrates me because it is essentially saying in a very naive way that all concussions are created equally, but when they are not. Concussions are not created equally and each one needs to be handled on an individual basis. I strongly believe that you need to record in verbiage how long the symptoms lasted with each concussion. This way in the future, others working on managing an individual's concussion can have an idea about how severe their previous concussions were. If symptoms lasted months, that is not the same injury as symptoms that lasted hours or only a day.

Myth Number Eight: Signs and Symptoms Occur Immediately

Incorrect. Some individuals have very little in the way of symptoms immediately, and some are not aware that they have had a concussion immediately. How much of that is related to adrenaline and rationalization I'm not sure, but it is a reality that many people worsen hours after the incident. Some may not have symptoms really worsen until later that night or the next morning.

Myth Number Nine: Boys Suffer More Concussions Than Girls

The number of girls are now almost equal to boys in most sports. In ice hockey, basket ball, and soccer, in fact, girls have almost twice as many recognized concussions as boys. I stress "recognized" and I keep repeating it because we do not really know that they have twice as many concussions; but twice as many are recorded. It is possible that this is due to the fact that they are more honest in reporting their symptoms, or that is due to their weak necks. It could also be both, but only time will tell.

Myth Number Ten: Concussions Determine Risk of CTE

This has not shown to be true in the work we have done. Our work would suggest individuals that take the greatest amount of brain trauma are most likely to wind up with CTE, not the people that suffered spectacular concussions. If you play in a sport that takes a higher amount of brain trauma, arguably calling boxing a sport, you are going to have a greater chance for CTE than if you play a sport like football which has less head trauma. If you play football, the lineman are going to take a lot more hits to the head than the wide receivers or the quarterbacks, although the wide receivers and the quarterbacks may take a more spectacular hit. In a sport like ice hockey, you are going to take hits equal to or greater than a football player occasionally, but not as frequently. As we accumulate more and more cases going forward, we expect to see a similar trend—that boxing seems to have the greatest incidence and football is second. Sports like ice hockey, although it certainly has cases of CTE, now appear to have a lower risk for CTE.

References

1. Cantu RC. Role of diffusion tenser imaging MRI in detecting brain injury in asymptomatic contact athletes: World Neurosurgery; December 2013.
2. Bigler ED, Maxwell WL. Neuropathology of mild traumatic brain injury: relationship to neuroimaging findings. Brain Imaging Behav. 2012;6(2):108–36.
3. Cantu RC, Guskiewicz K, Register-Mihalik JK. A retrospective clinical analysis of moderate to severe athletic concussions. Am Acad Phys Med Rehabil. 2010;2:1088–93.
4. Cantu RC, Gean AD. Second-impact syndrome and a small subdural hematoma: an uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. J Neurotrauma. 2010;27:1557–64.
5. Baugh CM, Stamm JM, Riley DO, Gavett BE, Shenton ME, Lin A, Nowinski CJ, Cantu RC, McKee AC, Stern RA. Chronic traumatic encephalopathy: neurodegeneration following repetitive concussive and subconcussive brain trauma. Brain Imaging Behav. 2012;6(2):244–54.