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

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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 we 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 had had a concussion a few weeks before in Kansas, and had concerns about his health. Drivers that were commenting were saying that this just would not have happened 10 years ago and that is probably true. NASCAR drivers driving cars are similar to fighter pilots on wheels. Their reaction times and their vision need to be 100%. We are very glad to see that he brought concussion issues to doctors even though it cost his team a great deal of money.

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

The most common result of not imposing physical and cognitive rest after a concussion is greatly exacerbating concussion symptoms, and causing something that would have recovered in a matter of days to weeks into something that may now go on for months to years, and become post-concussive syndrome (PCS). Also, a much less common, but potentially fatal risk is *second impact syndrome* (SIS), which Bob Harbaugh and Dick Saunders first described in a *JAMA* article in 1984 [1]. It is interesting to us to see how our own practice has greatly changed in recent years, because of the awareness of concussions and PCS. We are actually inundated with post-concussive syndrome patients, most of whom have months of symptoms before we ever see them.

Approximately 8 years ago, one of my colleagues and I (R.C.C.), 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 [2]. 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. We 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 [3, 4]. The most common occurrence with PCS 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 [5]. 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 of 80–100 g. We have also seen from the work of Dr. Kevin Guskiewicz and others that just because you have 100 g impact, it is not necessarily associated with a concussion [6]. 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 occurrence is multifactorial and there is no known threshold of force required to produce symptoms. We also recognize that the tools measuring linear and rotational accelerations have significant accuracy challenges that contribute to the problem.

Various laboratory and clinical studies over the last 10 years have demonstrated that the subconcussive hits, those hits that do not result in overt clinical symptoms, count as well. Helmet accelerometer data for youth, high school, and college-level football players have shown the tremendous variability in the number of hits and

repetitive trauma young people are taking over a single season. At the college level [7], while the mean is not terribly high, the extremes that the individuals are taking can be very high. Certainly at the high school level, over 1000 hits a season is not uncommon [8]. While every hit does not result in a symptomatic concussion, the accumulation of hits to the head over a single season, both in football and in soccer, has shown to result in changes in functional connectivity, decreased cognitive performance, white matter, and cerebrospinal fluid markers, demonstrating that seemingly routine and minor hits can have a profound impact [9]. We believe this emerging evidence should give us all cause for concern but of course needs to be replicated by other investigators. We have studies that show structural changes in individuals' brains that have headed more than 1300 times in a given soccer season as well [10].

It is not just metabolic magnetic resonance spectroscopy (MRS) studies that have shown changes, but diffusion tensor imaging (DTI) studies have also shown changes similarly when comparing baseline cognitive assessments prior to and after an athletic season. The integrity of fiber tracts tends to decrease over the course of the season [10, 11]. Presumably, this is due to subconcussive 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 subconcussive blows that are producing deleterious effects on brains. It is an accumulation of these data that led me, the senior author (R.C.C.), to write a book 8 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 has since led to the age moving from 11 to 13), and no heading in soccer until the age of 14 (which has since led to US Soccer banning headers under age 11 and limit heading for ages 11–13).

Further changes suggested were regarding helmets and officiating. It does not make sense that you have a very good batting helmet for baseball players, but then the helmet falls off of the players' heads 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 or ball to the head or face, like women's field hockey and lacrosse, we believe there should be helmets, rather than simply face shields. Additionally, we 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.

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, with change of velocity over time, you have a decreased opportunity for change in velocity with a well-developed neck. Dom Comstock and I (R.C.C.), 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 have 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 [12, 13]. For example, the woodpecker has a large, strong neck, and it moves only in a linear straightforward manner—a woodpecker does not experience concussions. We strongly believe that 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 do not have nearly the testosterone compared to adult males, and are not going to bulk up their neck muscles, so they are not going to look any different, but they can strengthen them.

We do not know the exact combination of linear and rotational forces necessary to reach threshold for a concussion, but we do know that there are curves which suggest that the risk of sustaining a concussion certainly increases 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, ice hockey, or lacrosse player. The reason that we 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, and rotational accelerations would be ideally known as well. But duration of impact, location of impact, and tissue strain issues are all very relevant and in need of study.

However, there are also biological and social factors at play. 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 these risk factors include history (how many concussions has somebody had, how severe were they, and what is the proximity in time of them), 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) [14, 15], as well as underreporting.

Concussions: Structural Versus Functional Brain Disorder

Expert neuropathologist and colleague Dr. Ann McKee has found diffuse axonal swelling and axonal damage in some individuals that have died by suicide shortly after a concussion. Concussion in part involves a neural metabolic cascade, a metabolic issue, but also in part, at least in some concussions, there is a *structural issue*. We believe that as we learn more through neuroimaging studies, like the use of DTI [16], we are going to be able to see those structural changes in concussion that we cannot currently see with routine clinical MRIs or CT scans.

Subjects' Reports Versus Pure Evaluation

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 (R.C.C.) 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. Theoretically by asking after the season, when there is no longer a feeling of possibly letting down their coaches or 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 [17]. A seven times greater incidence of concussion was found from the physician observers, as compared with medical personnel on the bench [17].

The National Football League knows this is true as well; they instituted a policy in 2012 for the use of independent, certified athletic trainers to act as spotters in stadium booths at every game to spot potential injuries that are missed on the field, including concussions. Looking at the same television feeds that the public sees 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. When this was deemed insufficient, the 2017–2018 NFL season introduced the placement of an unaffiliated neurotrauma consultant (UNC) in the league's command center for all games, and later on-site UNCs. 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. Former Cleveland Browns quarterback Colt McCoy is the most notable of them.

I am very pleased that Dr. Chris Nowinski, co-founder of the Concussion Legacy Foundation (formerly the Sports Legacy Institute), and I (R.C.C.) 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, with published data showing that roughly 60–70% of those hits in the past have occurred during practice [18].

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 with people thud tackling, wrapping arms around but not bringing players to the ground. Similarly, Dartmouth College football uses tackling dummies and even a tackling robot in practices so that they are not hitting each other. 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 head 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.

Second Impact Syndrome

SIS [19, 20] 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.

With *SIS,* 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 [19]. 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 quickly increase the amount of blood that is inside the blood vessels, you will 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 [19]. The subdural is not causing much mass effect; however, it is this vascular engorgement of the brain that is

causing detrimental outcomes. This is not vasogenic edema, because there is gray and white matter differentiation. It is a drastic increase in the volume of the brain due to blood in the arteriolar-capillary bed.

Long-Term Risks of Mismanaging Multiple Concussions

Historical Perspective

One of the long-term risks is prolonging your post-concussion syndrome if a concussion is mismanaged, and the other is the issue of chronic traumatic encephalopathy (CTE). CTE is a distinct, progressive, neurodegenerative disease characterized by a pathognomonic lesion of *hyperphosphorylated tau protein* accumulated around blood vessels at the depths of cortical sulci believed to develop due to, at least in part, to repetitive head impacts (RHI) [21, 22].

Do you know who first described CTE? I (R.C.C.) asked this question recently at a conference, and immediately a hand went up and said Bennett Omalu. He did describe the first cases of CTE in National Football League players [23, 24], but not the distinct pathology or the associated clinical syndrome itself. In 1928, forensic pathologist Dr. Harrison Martland described "punch drunk," the clinical syndrome of boxers, which is akin to the clinical features of traumatic encephalopathy syndrome (TES) seen today in CTE [25], but without using those words. In a book that was a tribute to Clovis Vincent that came out in 1949, a number of individuals were solicited to write chapters, 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 the neurological syndrome of boxers was described as "chronic traumatic encephalopathy." Pathologically, the first description was by Corsellis in 1973, who connected neuropathological findings to a retrospective pattern of behaviors seen in a sample of 15 retired boxers. Since Martland first described the disease in 1928, until Dr. Omalu's report in 2005, there were fewer than 50 confirmed cases of CTE in the literature; there are now nearly 400 published cases. However, there are only three confirmed cases of women with CTE—one with a history of domestic violence described in 1990 by Dr. Roberts, one in 1991 by Dr. Hof with a history of autistic head-banging behaviors, and one in 2021 of a 29-year-old with a history of domestic violence.

Boston University School of Medicine Chronic Traumatic Encephalopathy Center

At Boston University School of Medicine, we started with four directors at the Center for the Study of Traumatic Encephalopathy in 2008 (now the BU CTE Center), a part of the BU Alzheimer's Disease Research Center, which was established in 1996 [26]. Original directors included Chris Nowinski, concussion advocate and former professional wrestler, neuropathologist Dr. Ann McKee, famous for

her work in neurodegenerative disease, Dr. Bob Stern, neuropsychologist and clinical research director, and myself (R.C.C.). The Center was established to focus on the study of repetitive head impacts, including the clinical and pathological features associated with contact sport play and military service. Partnerships with the Boston VA Healthcare System, as well as the Concussion Legacy Foundation (formerly Sports Legacy Institute), enhanced collaboration and formation of the VA-BU-CLF Brain Bank, now the largest tissue repository in the world dedicated to the study of CTE. We established an online brain donation registry in 2015 to de-mystify and normalize the process of brain donation; this registry now has over 6,000 people enrolled. We are hoping to register and study brains of asymptomatic individuals that live normal lives, and yet play contact sports. The brain donation registry has greatly accelerated brain donation post-mortem. In the early stages of the Center, we were examining just a handful of cases a year. We have since received over 100 donations in each of the last 3 years. At this time, the VA-BU-CLF Brain Bank houses over 1,000 brains of former athletes and military veterans, the vast majority of whom were symptomatic. Donors range in age from 13 to 99, from youth through professional and Olympic levels of play and 98% are men. Additionally, as a neurodegenerative disease brain bank, it is quite striking that 35% of our donors are under the age of 50. While the majority of donations are from former football players (about 70%), we are increasingly receiving a wider variety of athletes, including several lacrosse, soccer, and rugby players, as well as motocross and BMXers, and amateur wrestlers. Most of these people were symptomatic and were predicted to have CTE by their emotional, behavioral, and cognitive symptoms reported by family members upon donation.

The VA-BU-CLF Brain Bank currently lacks a repository of brains of individuals that were not symptomatic, but they will likely come from that registry. Dr. Stern as P.I., along with Dr. Nowinski, Dr. McKee, myself (R.C.C.), and a crew of graduate students, are doing a longitudinal clinical research study on over a hundred NFL players compared to a group of individuals 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 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. Dr. Stern is also one of the P.I.s on the NIH-funded DIAGNOSE CTE Research Project, a larger, multisite study examining college and NFL players and age-matched controls. 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, as demonstrated by the high rate of CTE in our brain bank, but you cannot be 100% certain until a neuropathological examination is performed.

Dr. McKee's first case was John Grimsley in 2008, a former NFL player who had advanced CTE. The medial temporal lobe was just riddled with this staining identifying *hyperphosphorylated tau protein*. Other notable NFL players examined by Dr. McKee, and that were later publicized, were Dave Duerson, Cookie Gilchrist, Bubba Smith, John Mackey, Ken Stabler, Aaron Hernandez, and three members of the 1972 Miami Dolphins, Bill Stanfill, Earl Morrall, and Bob Kuechenberg. An

important study from 2012 by Lehman et al. examined causes of mortality in former NFL players [27]. They looked at death certificates of a number of NFL players who played over a 10-year period, 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. These brains were not studied, only the death certificates were examined. 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, particularly when Alzheimer's disease can look like CTE.

We do not know from our work in Boston what the incidence of CTE is, and what the prevalence of it is in any population. We know it occurs, and we know that it occurs in a very high percentage of those brains that we examine, but we also know that those brains are in a skewed sample. However, just because a sample is skewed toward symptomatic, high-level exposed individuals does not mean that the work is invalid. In fact, there have been a number of extremely valuable studies over the last few years using VA-BU-CLF Brain Bank data.

We published our first case series of autopsy-confirmed CTE in 2009, where we reviewed 48 cases of boxers, football players, wrestlers, and others with repetitive head impacts [21]. Subsequent papers over the last 10 years have examined the pathology in depth, as well as the various risk factors that we think are involved. Many of these studies examining risk factors, both antemortem in clinical studies, and post-mortem using donated brain tissue, have been led by additional BU colleagues Dr. Jesse Mez, Dr. Michael Alosco, Dr. Jon Cherry, Dr. Lee Goldstein, Dr. Dan Daneshvar, and Dr. Thor Stein. CTE in the context of motor neuron disease [28], epidemiological considerations with concussion [29], subconcussive head trauma [30], military blast exposure [31], clinical presentation [32], research criteria for traumatic encephalopathy syndrome [25], age of first exposure to football and later-life impairments [33–35], corpus callosum and white matter microstructural changes [36, 37], beta-amyloid deposition [38], inflammation [39, 40], MRI, MRS, and PET imaging markers [41–45], cerebrospinal fluid markers [46, 47], Lewy bodies [48], cerebral amyloid angiopathy [49], genetics [46, 50], and duration of play [8, 51, 52] have all been described. Dr. McKee has also led the international effort to characterize the neuropathology of the disease through a series of consensus conferences that have brought together the world's leading experts in tauopathies.

Doctor, Do I Have CTE?

CTE in most people is a progressive neurodegenerative disease believed to be caused by repetitive trauma to the brain which includes subconcussive blows. This is NOT a prolonged post-concussive syndrome, nor is it solely the cumulative effects of concussions. Symptoms characteristically, although not always, begin years to decades after the individual has stopped sustaining brain trauma. 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 neurodegenerative diseases, psychiatric conditions, and post-concussive syndrome before being able to diagnose the disease in living people. The Center receives countless calls and emails every week from families and individuals concerned they have CTE. We always suggest seeing a neurologist, neuropsychologist, or other professional experienced in treating brain trauma to treat the symptoms, which may or may not be associated with CTE. There is no treatment for CTE, but there are ways to manage symptoms and everyday life with neurological and psychiatric conditions [53]. Building a strong network of support from family and friends, eating healthy foods to properly fuel the brain, getting enough sleep, exercising appropriately, and finding meaningful hobbies and activities are just some of the ways in which individuals and families can cope with symptoms. Unfortunately, many cases of CTE have been reported due to those individuals dying by suicide. 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, as suicide in itself is a complex issue. 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 any more of our heroes having their brains examined because of suicide.

In Summary: 10 Myths About Concussion

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

We 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 is not 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 (R.C.C.) 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, but headbands and a plethora of unproven products too. While we encourage research in concussion prevention, we are 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 professional ice hockey player I (R.C.C.) cared 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 demonstrates 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 Are Out

This myth really frustrates us because it is essentially saying in a very naive way that all concussions are created equally, when they are not. Concussions are not created equally, and each one needs to be handled on an individual basis. We 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. It is ultimately a combination of factors, and a larger discussion between a patient, provider, and the family in deciding to retire from contact sports.

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 we are 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 part due to Title IX legislation in the 1970s. In ice hockey, basketball, and soccer, in fact, girls have almost twice as many recognized concussions as boys. We stress "recognized" and 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 due to their weak necks. It could also be both, but only time and research will tell.

Myth Number Ten: Concussions Determine Risk of CTE

This has not shown to be true in the work we have done at the VA-BU-CLF Brain Bank. Our work suggests that individuals that take the greatest amount of brain trauma are most likely to wind up with CTE, not the people that suffered spectacular concussions. We have found the best measure of repetitive head impacts, or concussion and subconcussive blows combined, to be the length of one's playing career. If you play in a sport that takes a higher amount of brain trauma, like boxing or football, you are going to have a greater chance for CTE than if you play a sport like basketball which has less head trauma. If you play football, the linemen 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 at the Brain Bank, we expect to see a similar trend—that boxing seems to have the greatest incidence and football is second. Sports like ice hockey, rugby, lacrosse, and soccer, although they certainly have cases of CTE, now

appear to have a lower risk for CTE, but various risk factors other than contact sport participation are still being studied.

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