Collision Course: Concussions are ticking time bomb for former players

Four players who took part in the concussion project said they would choose to end their lives prematurely if they started to experience signs of dementia.

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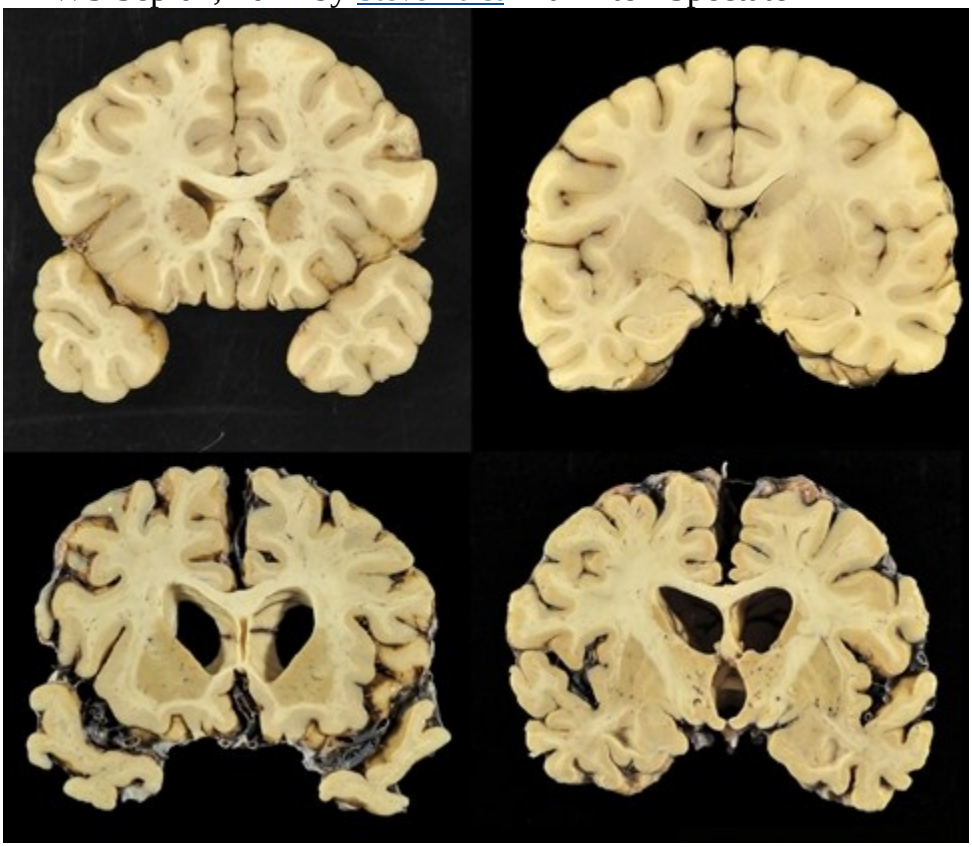


This combination of photos provided by Boston University shows sections from a normal brain, top, and from the brain of former University of Texas football player Greg Ploetz, bottom, in stage IV of chronic traumatic encephalopathy. According to a report released July 25, 2017, by the Journal of the American Medical Association, research on the brains of 202 former football players has confirmed what many feared in life _ evidence of chronic traumatic encephalopathy, or CTE, a devastating disease in nearly all the samples, from athletes in the NFL, college and even high school. - Boston University, The Associated Press

Steve Buist describes what happens to the brain during a concussion.

Fourth and final part of Collision Course, a Spectator investigation on concussion and football. See more series content.

No treatment, no cure, and it can only be diagnosed after death. It's no wonder CTE — chronic traumatic encephalopathy — is the ticking time bomb that strikes fear in many former football players.

CTE is a progressive degenerative disease of the brain with symptoms similar to dementia and it's associated with repeated hits to the head. That much is known.

What's not known is how many concussions or blows to the head are needed to switch on CTE or which players are most susceptible to developing it.

When it comes to brain damage, mounting evidence suggests football is a slow-motion form of Russian roulette for its players — pull the trigger and then wait decades to find out if there was a bullet in the chamber.

"I think people are absolutely right to be fearful if they have significant brain trauma exposure," said Chris Nowinski, co-director of Boston University's CTE Center. "It's a disease that is killing people that we don't really understand how to diagnose, how to prevent and we have no treatments," he added.

Nowinski is also the co-founder of a brain bank based at Boston University that looks for signs of CTE in the brains of deceased athletes and members of the U.S. military that have been donated for study. It's important to note the brain bank's collection is not a random sample. It's possible the results are skewed because those who suspected they had problems may have been more inclined to participate. Nonetheless, the tally from the brains of former football players is truly frightening.

Of the 202 brains of former football players examined post-mortem by the university, 177 showed signs of CTE. The youngest case was a 17-year-old. When the results were restricted to just former NFL players, the number is astonishing — 110 of the 111 brains of former NFLers showed signs of CTE.

Seven of the eight brains of former CFL players also showed signs of CTE.

Recently, 52-year-old former Ticat receiver Ken Evraire announced he will donate his brain to the Boston University bank.

What's equally troubling is that in 20 per cent of the cases where CTE was found, there had been no documented concussion ever reported.

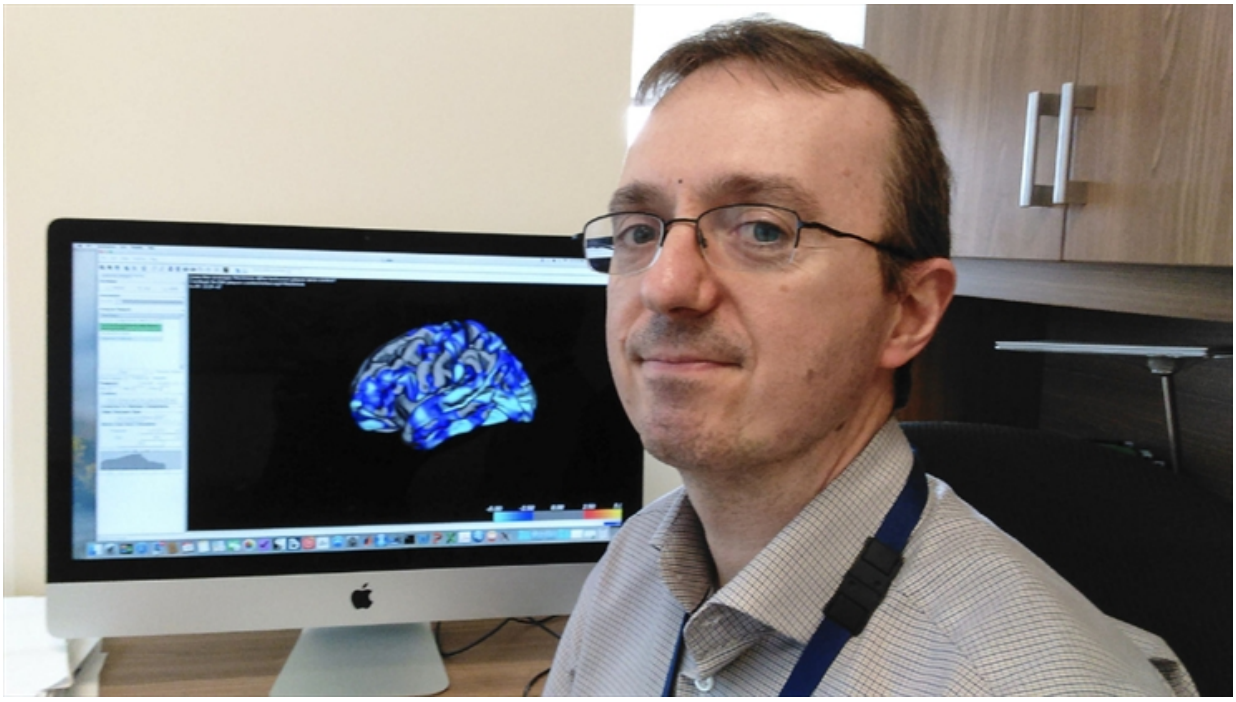
Other findings are just as startling.

Based on numbers compiled by actuaries on behalf of the NFL, former players between ages 50 and 59 had rates of Alzheimer's disease and dementia up to 23 times higher than the general population of similar ages. For players between 60 and 64, the rate was up to 35 times higher.

Which brings us back to the Spectator's landmark CFL concussion project, done in collaboration with researchers from McMaster University and St. Joseph's Healthcare.

The study of 22 retired CFL players showed disturbing differences in brain anatomy, wiring and electrical activity between the players and control subjects — men in a similar age range with no history of concussions or repeated hits to the head.

While the project wasn't designed to provide medical diagnoses or predict future health, it's impossible not to wonder what lies ahead for the players who participated.



Dr. Luciano Minuzzi: Professor in McMaster Department of Psychiatry and Behavioural Neurosciences and a member of McMaster Mood Disorders Program based at St. Joseph Healthcare West 5th Campus. He is also a psychiatrist and an expert in the analysis of functional MRI imaging. (Steve Buist, The Hamilton Spectator)

Dr. Luciano Minuzzi is a McMaster researcher based at St. Joe's West 5th campus. He's an expert in brain imaging as well as a clinical psychiatrist. Minuzzi analyzed the thickness of the brain's cortex, where billions of nerve cells reside and compared the players' results to control subjects.

As reported in Part 1 of the series, Minuzzi was shocked at the widespread thinning of the cortex seen on average in the players — so shocked, in fact, that he performed his analyses five separate times because he couldn't believe the results. The question put to him was sobering. If the brains of the CFL participants could be examined after death, how many of them would you expect to find with CTE?

His response was chilling.

"I think it's the majority of them," said Minuzzi.

If there's a saving grace, he added, it's the brain's incredible ability to adapt.

"Our brain is very plastic and very resilient, especially when it's something happening for a long time," said Minuzzi. "Our brain will do its best to adjust. It is possible that if they don't have any symptoms now, the brain is trying to compensate. But we don't know for how long that can happen."

It's not surprising some of the retired players are fearful of the future. Some confess that every time a name's forgotten or a phone number disappears into the ether, they have a flicker of doubt.

Bob MacDonald was an offensive lineman at McMaster University, then with Calgary and Hamilton in the CFL during the early 1990s. His last CFL game happened to be back at Calgary's McMahon Stadium against his old club.

Bob MacDonald

A former teammate head-butted him on a play and MacDonald ended up flat on his back right by the Stampeder logo at midfield, staring up at the sky. Burners — painful stinging sensations caused when nerves in the neck get squished — were shooting down both arms. By this point, he had started teaching and as he lay there in pain, "I'm thinking 'What am I doing? I'm done. My future is teaching,'" MacDonald recalled.

Now 49, he's a teacher at Saltfleet Secondary School and he admits that from time to time, he thinks about what the future might hold for him after years of smashing his head for a living.

"When I'm in the classroom and I can't think of a word," he said. "I have this great stream of thought going and bang, I stop dead — what was the word I was trying to think of?

"I wonder, is that because I have things racing through my mind or, Oh my God, is that a sign of something starting?" he said. "For eight or 10 years, I've wondered that."

Without any prompting, four players who took part in the concussion project said they would choose to end their lives prematurely if they started to experience signs of dementia.

One of them is Don Bowman, now 65, who played in the mid to late 1970s with Winnipeg and Hamilton. He said he doesn't really think about CTE because he already figures he's facing some daunting odds. Both of his parents had degenerative neurological diseases, so he's opted for a fatalistic outlook on the future.

"I'm not turning the clock back and fixing the damage that may have occurred when I played football," said Bowman. "There's a master plan that we don't control and what's going to happen is going to happen."

Bowman's father was diagnosed with Alzheimer's at age 81 and the last three years of his life were "ruthless," Bowman said.

"There was virtually nothing left of him," he said. "That's not ever going to happen to me. So I just leave it at that. I don't think that just living longer is living better."

Each year, just a few days before the Grey Cup, it's customary for the CFL commissioner to deliver a state-of-the-league address and take questions from the media.

So last Nov. 25 in a ballroom of the Toronto Delta Hotel, then-commissioner Jeffrey Orridge stepped on to a small stage and promptly raised eyebrows across the country.


Jeffrey Orridge

Orridge was asked point blank if he was prepared to admit there was a link between football and degenerative brain diseases, such as CTE, Alzheimer's and Parkinson's.

No, he stated simply, "the league's position is that there is no conclusive evidence at this point."

Orridge, who parted ways with the CFL in June after two years, was pressed a second time and again, he declined to draw a link.

"Last I heard," he said, "it's still a subject of debate in the medical and scientific community."

Orridge's statements did not sit well with some of the retired players who took part in the Spectator's concussion project.

"The naivete of that statement is really kind of unbelievable," said Bowman. "It doesn't make any sense. For the person leading the league to make a comment like that, I thought it was really inappropriate."

Former Ticat Mike Morreale called the comments "very upsetting."


Mike Morreale

"Suggesting there is no link is not an educated defence," said Morreale, who's also a former president of the CFL Players' Association. "I don't know how you can come to that conclusion when really there's so much evidence that points there could be. I just want people to be honest and truthful and say 'You know what, this is a sport that you can be seriously injured in and it could lead to brain-related trauma.'

"But that's a choice I was willing to take," he added.

The CFL did not respond directly to questions from the Spectator about CTE, links between football and neurodegenerative diseases, or Orridge's comments.

In a written statement, the league said "the health and safety of our players is a top priority for our league, particularly when it comes to the assessment, prevention and management of concussions."

The league included a list of safety initiatives that have been recently adopted.

"We are focused on continuing to develop a culture of health and safety across our entire league including players, coaches and management," according to the CFL's statement.

Orridge's denial of a link between football and degenerative brain diseases seems all the more strange, since the CFL has been working with the Canadian Sports Concussion Project, led by Dr. Charles Tator of the University of Toronto.

In May 2013, Tator's research group published a scientific article based on the post-mortem examination of six brains from deceased former CFL football

players. Three of the six brains showed evidence of CTE. The other three brains showed evidence of Parkinson's disease, Alzheimer's disease and Amyotrophic Lateral Sclerosis. In other words, all six brains showed evidence of neurodegenerative disease.

Robyn Wishart, a Vancouver lawyer who has helped more than 200 players file a class-action lawsuit against the CFL and its teams over the long-term damage from concussions, said she "can't even fathom" the league's denial of a link between football and brain trauma.

"It makes my job easier, ironically," Wishart said, "because when they say it doesn't exist, the players stand up and say 'How come I am the way I am then?'"

What was especially puzzling about the CFL commissioner's statement was just how different it was from the dramatic about-face adopted south of the border last year by the NFL. Appearing before the U.S. Congress in March 2016, a top NFL official acknowledged for the first time there is a definite link between football-related head trauma and CTE. Then on Jan. 7, a court-approved settlement became official in a class-action lawsuit launched by former players against the NFL that could ultimately cost the league an estimated US$1 billion. The agreement settles claims made by players and their families that repeated head impacts from football left them with brain injuries.

While the wording of the settlement specifically allows the NFL to deny liability, the scope of the agreement is astonishing nonetheless. About 20,000 former players could be eligible for payments and the agreement will be in place for the next 65 years. There's no cap on the amount to be paid out for qualifying claims, so the total could rise beyond $1 billion.

The league estimates as many as 6,000 former players could ultimately develop Alzheimer's disease or dementia.

As part of the settlement, the league also agreed that players aren't required to prove that their injuries were caused by playing in the NFL to get their compensation. There's even a large grid included in the settlement that spells out the specific payments based on the type of condition and the age of the player at time of diagnosis. The maximum payment, for example, would be $5 million to a player who played at least five seasons and received a diagnosis of ALS before age 45.

The NFL also agreed to pay US$75 million into a fund to provide baseline medical assessments of retired players to determine their level of compensation and US$10 million into an education fund to promote safety and injury prevention to football players at all ages.

"It's interesting that the CFL is refusing to believe statistics whereas the NFL is having to grudgingly admit to statistics," said Bob Macoritti, who played six seasons, mostly with Saskatchewan, in the mid- to late 1970s.

By no means is it a tidal wave, or even statistically significant, but a growing number of players in their prime are choosing to walk away from pro football because of concerns about brain trauma.

Chris Borland of the San Franciso 49ers celebrates after intercepting a pass during game in 2014 in his first season. It was his only season as he left the sport in 2014 concerned for his health. (Bill Kostroun, Associated Press file photo)

Chris Borland was, by all accounts, a promising young linebacker for the San Francisco 49ers who was twice named the NFL's Defensive Rookie of the Week in 2014 during his first season. It turned out to be his only season. Worried about a concussion he believes he sustained during his first training camp, he decided to walk away from the sport at age 24 after doing his own research into concussions.

In January, 23-year-old Buffalo Bills' linebacker A.J. Tarpley also quit after one season. He said he suffered his third and fourth concussions during his season with the Bills, then decided "I am walking away from the game I love to preserve my future health."

The previous March, Husain Abdullah retired at age 30 after seven seasons and five concussions, saying he wanted to have "a sound mind" in his remaining years.

And in July, 26-year-old offensive lineman John Urschel, originally from Winnipeg, retired on the first day of NFL training camp after three seasons with the Baltimore Ravens.

Others weren't able to step away from the game in time.

Former Chicago Bears player Richard Dent helps carry the casket of his former teammate former Dave Duerson in Chicago on Feb. 25, 2011. The four-time Pro Bowl pick, who played on Super Bowl winners with the Bears and New York Giants, killed himself at his Florida home the week before. (Paul Beaty, Associated Press file photo)

Dave Duerson spent 11 seasons in the NFL as a safety and suffered at least 10 concussions, according to family members. In 2011, at the age of 50, Duerson shot himself in the chest, a desperate attempt to preserve his brain for examination. Duerson's suicide note begged his family to donate his brain to the Boston University brain bank.


Junior Seau

The following year, Junior Seau, a 43-year-old star linebacker who played an incredible 20 seasons in the NFL, also killed himself with a shot to the chest. His brain was also donated to Boston University.

Both of the players' brains showed evidence of CTE.

Not all of the cases have been as high profile as Duerson and Seau, however.

In late April 2015, the Tiger-Cats made a small signing announcement that passed almost unnoticed.

Adrian Robinson Jr. was a 25-year-old defensive end who had been a standout player at Temple University in Philadelphia. He had played 12 games with the Pittsburgh Steelers in 2012, eight games in 2013 split between Denver and San Diego and then he kicked around the practice rosters of a couple other NFL teams.

Three weeks after signing with the Ticats, Robinson was dead. On May 17, 2015, Robinson was found hanging in his Philadelphia apartment. He left behind a baby daughter.

"He went from being one of the nicest guys you'd ever want to talk to, to having a darker edge at times," a lawyer for the Robinson family said.

Robinson's father said he noticed his son becoming uncharacteristically moody and aggressive.

"He was changing," Robinson Sr. told a Pennsylvania media outlet. "He was still my son, but, like, some of the aggressive arguments and fighting, it wasn't normal."

By 2013, Robinson Sr. said, his son began complaining about headaches.

Five months after Robinson's death, officials at Boston University brain bank announced that Robinson's brain exhibited CTE, even though he was just 25 years old.

The family's lawyer said Robinson had suffered several concussions while playing in the NFL.

Three months ago in a Philadelphia court, Robinson's family filed a lawsuit against the NFL and a prominent manufacturer of football helmets. The family alleges the league and the manufacturer are guilty of conspiracy, fraudulent concealment and negligence in the wrongful death of Robinson.

The lawsuit alleges the NFL and the helmet manufacturer engaged in "sham science" and then "published counter-science and marketed ineffective harm-reduction technology in the form of football helmets incapable of protecting the players from sub-concussive and concussive traumas."

The NFL's decision to move to hard-shell, motorcycle-style helmets, the lawsuit alleges, created "a pandemic for those playing the game" because of brain trauma.


Chris Nowinski

"Talk to players and they'll say when the helmets got more comfortable, they became a weapon," said Chris Nowinski, of Boston University's CTE Center.

Indeed, a 2013 scientific article on CTE suggested as much.

"To date, there is no evidence that helmets actually reduce the incidence of concussion," the researchers stated. "In fact, the use of protective gear may actually lead to more aggressive play and may result in an increased incidence of concussion."

John Connolly, a McMaster professor and part of the research team for the Spectator project, said there are grim similarities between football and motorcycle helmets.

"As an E.R. guy told me one time, all it does is keep the mess inside one spot," said Connolly. "Your head still moves around and more important, your brain does. It's the brain moving in the skull that's the problem."

Football, it seems, has reached a crucial crossroads as the scientific evidence about brain trauma starts to pile up.

Can an inherently dangerous sport be made safe to play?

Mike Morreale, the former Ticat receiver, doubts it.

"I don't care what helmet you put on, it's not going to stop your brain rattling around inside your skull and that, to me, is probably the biggest issue," said Morreale, now 46.

As Morreale said, in Part 2 of the series, he was never diagnosed with a concussion but now estimates he may have suffered one in every game he played — a pro career that spanned more than 200 games.
"It's astonishing because no one is compiling all of the players who have said this," said Nowinski. "There are dozens of players on the record with the same statement. That voice doesn't get repeated and the myth gets perpetuated that the number of concussions diagnosed in professional sports is the actual number of concussions that are happening."

Nowinski points to a 2015 study of more than 700 U.S. university football players that found for every one concussion that was diagnosed, there were 26 potential concussions that went unreported or undiagnosed.

"The problem is that the changes at the professional level are only a fraction of the players' careers," said Nowinski. "Youth tackle football has been a tremendous mistake for the health of football players because it just adds way too many years of getting hit in the head while the brain is developing."

There's also a big difference between making football safer and making it safe.

Bob Macoritti, the former Saskatchewan player, says making the game truly safe for players could put it at odds with what fans want.

"You might be seeing flag football in 15 to 20 years," said Macoritti, "unless they can create something that is so protective of the head — and I don't see how you can do that."

As Part 3 of the series noted, CFL players aren't eligible for workers' compensation benefits or long-term disability, and they're not allowed to sue their employer in court.
Their league refuses to acknowledge a link between football and degenerative brain diseases. They're at enormously higher risk of developing Alzheimer's disease and dementia than the general population.

And for those retired players tested as part of the Spectator's concussion project, the results strongly suggest evidence of long-lasting brain trauma years, even decades, after their careers ended.

"Up to this point, people who play a professional sport like football or hockey, we just thought of it as a game," said Dr. Mike Noseworthy, director of McMaster's School of Biomedical Engineering and part of the project's research team.

"But this is their job.

"Going forward, we have to think it's not just about breaking bones or blowing out a ligament," Noseworthy said. "There are serious risks to the brain as part of their job.

"And if you sign up to do this job, you have to understand the risk you're getting into."

What is CTE?
In 1928, a scientific article in the Journal of the American Medical Association carried a short, catchy and rather unscientific title: "Punch Drunk."

Written by Harrison Martland, a New Jersey doctor, the article was the first to scientifically link the symptoms of brain damage suffered by boxers to the effects of repeated concussions and blows to the head.

By 1937, the syndrome of slurred speech, motor skill problems and memory loss was given a more dignified title — dementia pugilistica, which is now recognized as CTE.

CTE — chronic traumatic encephalopathy — is a progressive degenerative brain disease associated with concussions and repeated hits to the head that don't

necessarily rise to the level of a concussion. The onset of CTE begins in mid to later life, long after the effects of the original trauma have cleared. Symptoms of CTE include short-term memory loss, emotional instability, difficulty planning and carrying out tasks, depression, suicidal thoughts and some of the motor skill impairments seen in Parkinson's disease.

CTE can develop from hits to the head that cause the typical symptoms of a concussion as well as so-called sub-concussive hits that don't produce symptoms.

The exact mechanism of how concussions and sub-concussive hits lead to CTE is still being studied. It's also not clear how many concussions or sub-concussive hits need to be absorbed for CTE to develop. One recent scientific study suggested just one concussion could lead to findings consistent with CTE.

It's believed that the repetitive blows build up damage in the axons of the brain's nerve cells. Axons are the long wires that transmit electrical signals from one nerve cell to the next. When the brain gets rattled, the axons can get stretched or squished or even sheared. That damage is then thought to start a cascade of chemical events.

At the microscopic level, the internal framework of the axon is supported by microtubules, and one certain protein called tau is responsible for assembling and stabilizing the microtubules. With CTE, the damage to the axons can cause an abnormal flow of chemicals back and forth across the outer membrane. As some of these substances flow in, they add little chemical appendages to the tau proteins that cause crucial changes to their shape. The tau proteins start to form tangles that disrupt the framework of the axons and interfere with the transmission of electrical signals.

Over time, the jumble of misshaped tau proteins begin to build up, which can lead to the development of symptoms depending on the area of the brain that's affected.

What makes CTE such a concern is that suspected cases can only be diagnosed definitively by examining the brain after death. There are no blood test markers, for example, or brain imaging techniques that would conclusively show the presence of CTE in living people. There are also no treatments.

"You can't diagnose CTE with any known accuracy in living people," said Chris Nowinski, co-director of Boston University's CTE Center.

Nowinski's interest in the concussion issue is as much personal as professional. He played football for Harvard University then became the first alumnus of the school to wrestle in the World Wrestling Entertainment. Nowinski suffered a

concussion in 2003 and had to retire because of post-concussion problems that wouldn't subside.

He began researching the medical literature, he said, "trying to understand why I wasn't getting better," which then led to a book he wrote titled "Head Games: The Global Concussion Crisis."

"We are starting to understand some telltale signs of the disease that give clinicians confidence — without absolute confidence — in the diagnosis," he said. "Certainly within 10 years we'll have a much better idea of how to diagnose it."

Preventing CTE is actually the easiest part, Nowinski added.

"Don't hit people in the head," he said. "That part is well understood."

Stages of CTE

The Boston University centre has identified four stages of CTE symptoms which are gathered post-mortem through interviews with family members.

Stage I: Headaches and problems with attention and concentration;

Stage II: Depression and short-term memory problems;

Stage III: Cognitive impairment and problems with planning and organization;

Stage IV: Evidence of full-blown dementia.