The Economist

Concussion

Bang to rights

Science is taking big steps toward understanding the impact of concussion

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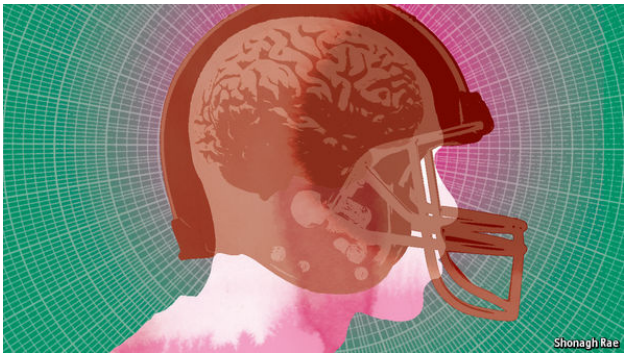

FRED McNEILL, an American-football player, died in November at the age of 63. Between 1974 and 1985 he appeared for the Minnesota Vikings. After leaving them he became a lawyer but in later years suffered from dementia and was told that he had signs of chronic traumatic encephalopathy (CTE), a degenerative brain disease. His recent death has become a milestone in the understanding of brain disorders, for post-mortem examination has confirmed this diagnosis—retrospectively making him the first person to be so diagnosed while alive.

CTE is the physical manifestation in the brain of punch-drunk syndrome—or dementia pugilistica, to give its Latinised, medical name. As that name suggests, this form of dementia particularly affects boxers, who are hit on the head as a matter of course. But doctors now understand that it is also a problem in people like Mr McNeill, who get hit on the head accidentally in contact sports. Mr McNeill, along with several other retired players, volunteered to let researchers at the University of California, Los Angeles (UCLA) use a scanning technique called positron-emission tomography (PET) to look at their brains.

In his case, and in those of others who had suffered concussion (a form of brain injury that leads to headaches, dizziness and nausea, and causes blackouts in 10% of those who experience it), this revealed a pattern of abnormal protein deposits. Mr McNeill's death enabled his scans to be compared with the results of an autopsy. That confirmed the PET-based diagnosis. Specifically, PET was recording deposits in his brain of a protein called tau, which is tied to CTE and other neurological disorders, including Alzheimer's.

Knocked about

Until quite recently, according to Julian Bailes, a director of the NorthShore Neurological Institute in Evanston, Illinois, it was thought that concussion from accidents in contact sports such as American football was a temporary malfunction rather than a permanent injury. This view began to change in 2005, when Bennet Omalu, a pathologist at the county coroner's office in Pittsburgh, wanted to find out why another footballer, Mike Webster, had suffered mental ill-health before dying unexpectedly at the age of 50. Webster had become confused, angry and violent, and even bought a Taser gun to treat his own back pain. Dr Omalu discovered tau-protein deposits in his brain, and proposed that the footballer had died of CTE, something which had previously been recognised as a cause of death only in boxers. This revelation, and his fight with the National Football League, which regulates the game, was the subject of a recent film called "Concussion". Since then, he and Dr Bailes have been collecting the brains of former athletes, and also of old soldiers who have been exposed to explosions, to try to understand who gets CTE, and why.

The road to CTE, their research suggests, does indeed start with concussion. One incidence in five of this then leads to post-concussion syndrome, a period of cognitive impairment that may last months, in which patients have headaches, unsteadiness and other problems. And it seems increasingly likely that, in some cases, repeated concussions may lead to CTE.

In recent years the underlying biology has started to become apparent. Mostly, this relates to the release of certain chemicals when axons, the filamentous connections between nerve cells, are damaged. Concussion is different from blunt-force trauma, such as that which results from getting hit on the head by a rapidly delivered cricket ball. Then, the injury is caused by directly transmitted shock from the impact. Concussion, by contrast, is caused by the internal movement and distortion of the brain as it bounces around inside the cranium after an impact. This bouncing, research has shown, stretches and deforms bundles of axons that connect different regions of the brain. The deformation shears some axons directly, releasing their protein contents, including tau, which with time can form abnormal tangles similar to those found in Alzheimer's disease. It also causes abnormal inflows of sodium and calcium ions in unsevered but damaged axons. These, in turn, trigger a process which releases protein-breaking enzymes that destroy the axon, further disrupting the brain's internal communications.

Concussive injury also damages the blood-brain barrier. This is a system of tightly joined cells surrounding the capillaries that service the brain. Its purpose is to control what enters and leaves the central nervous system. One consequence of damaging it is to release into general circulation a brain protein called S100B. The body mounts an immune response against this protein, and the antibodies it generates can find their way back into the brain and harm healthy brain cells. Researchers propose that repeated damage could set the stage for a continuous autoimmune-type attack on the brain.

The relationship between brain injury and behavioural symptoms is, nevertheless, unclear. The work at UCLA suggests a characteristic pattern of protein deposits in people who have suffered repeated concussions. The death of Mr McNeill suggests that may lead to CTE and dementia pugilistica. But many uncertainties remain, according to Huw Morris, a clinical neuroscientist at University College, London.

One challenge is to connect concussion, post-concussion syndrome and CTE itself. The immediate relevance of this will be to help understand whether a particular injury is likely to have long-term consequences. In this context yet another protein released during concussion, SNTF, is of interest. Work on concussed ice-hockey players suggests its level in the blood after a blow to the head predicts the severity of a concussion. A test for this, or for S100B, might provide a way to determine an injured player's prognosis.

That is a matter of interest both to sportsmen and—women, and to the clubs they play for. Neither coaches nor athletes tend to be good judges of fitness to play after a head injury. It seems likely that many people return to the field long before the brain's physical healing is complete. And players tend to underreport symptoms. A survey of American university athletes in 2014 found that 20% believed they had suffered a concussion, but almost 80% of those decided to continue to play rather than seek medical attention. Most felt their concussion was not serious and were concerned that revealing their injury would affect their standing with the team.

To try to find an objective measure of the damage an episode of concussion has caused, Dr Morris and his colleagues are studying players at Saracens, a rugby club in London. Participants will wear impact sensors to monitor collisions and give blood samples at the end of every game. The study hopes to find chemicals associated with brain injury that could be used to develop a blood test. A similar project is under way at the Translational Genomics Research Institute, in Phoenix, to study American-football players at Arizona State University.

Brain scanning, too, may have something to contribute. Magnetic-resonance imaging (MRI), a mainstream and widely available technique, can reveal reduced blood flow in the brains of athletes who have had concussions, even long after the injury. An MRI–based test might thus be developed to determine when or whether a player is well enough to return to play. There are also commercially available apps called HeadCheck and Braincheck, which are intended to help athletes work out their baseline brain performance and allow the organ's health to be tracked. They are not approved as medical devices, so cannot be used to diagnose concussion. But they may let players, especially amateurs, monitor themselves after blows to the head to search for signs that they might need medical advice.

Suffer the little children

In the end, adult athletes can make up their own minds about what risks they wish to take.

Children, though, cannot. It is therefore children who should attract the greatest attention. Not only are some children obliged to play contact sports at school, but they may also be participating in an environment that encourages a "stiff upper lip" when they are injured. Yet it has been clear since a study published in 2012 by Andrew Mayer at the University of New Mexico in Albuquerque that subtle brain changes in children who have sustained a concussion persist for months after the injury, even when there are no longer any obvious symptoms.

Work published last December by Charles Hillman of the University of Illinois found that children who had sustained a single sports-related concussion still had impaired brain function two years later. Ten-year-olds with a history of concussion performed worse on tests of working memory, attention and impulse control than did uninjured confrères. Among the children with a history of concussion, those who were injured earlier in life had larger deficits. This study was small (it involved 15 injured participants) but if subsequent research confirms it, that will be great cause for concern.

Concussion—once an invisible injury—is rapidly being illuminated. Many sports-lovers have been disturbed to learn that injuries on the field have caused their heroes to undergo profound personality changes. And many parents now worry, with good reason, about their children playing sports. Taking one for the team is all very well. But the price of doing so may be paid over a lifetime.

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