

History of chronic
traumatic
encephalopathy
psychology essay



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Chronic traumatic encephalopathy (CTE) is a syndrome of emotional lability, Parkinsonism, ataxia, and cognitive impairment suffered by athletes who undergo repetitive concussive and subconcussive blows to the head (Cantu 2007). Owing to its initial discovery in boxers, CTE has been variously known as “punch drunk,” “dementia pugilistica,” and “psychopathic deterioration of pugilist.” This paper will discuss the history of research into this fascinating topic, starting with the first descriptions in the medical literature and covering the progress made in understanding the clinical presentation, epidemiology, neuropathology, and genetics of the disease.

CTE was first described by the American pathologist Martland in a 1928 article on the “punch drunk” syndrome in boxers. Martland noted that for years boxing fans and promoters had observed “cuckoo” or “goofy” behavior in fighters. The fighters most often affected were “poor boxers” who would “take considerable head punishment, seeking only to land a knockout blow.” Punch drunk was also common among “second rate fighters used for training purposes, who may be knocked down several times a day.” Martland described the symptoms of punch drunk based on his examination of five boxers. The early symptoms of punch drunk were unsteady gait and slight mental confusion. Some boxers did not progress beyond this stage, while others went on to develop slow movements, hesitancy in speech, and hand tremors. In severe cases, boxers would develop a propulsive gait, Parkinsonian facies, and marked mental deterioration. Martland speculated that the mechanism of brain injury was traumatic cerebral hemorrhages followed by gliosis. This conjecture was based on his observation of multiple

cerebral hemorrhages in people who died from acute traumatic head injury (Martland 1927).

Building on Martland's work, researchers investigated the clinical course and epidemiology of CTE. Critchley observed that CTE can progress in many boxers even after they had retired, a phenomenon that to this day eludes explanation (1957). Corsellis noted that emotional lability and violent behavior tended to precede the Parkinsonian symptoms, ataxia, and cognitive decline mentioned by Martland (1973). Roberts studied the prevalence of CTE in retired professional boxers and found that 17% of subjects exhibited brain damage as determined by neurological exam and EEG abnormalities. Just as Martland observed that boxers who took more hits to the head were more likely to be punch drunk, Roberts showed that career length and number of professional fights were risk factors for CTE. Indeed, 47% of boxers whose careers were longer than 10 years suffered brain damage, compared with 13% of boxers with careers shorter than five years. Likewise, about 50% of boxers who had fought over 150 bouts had brain damage, compared with 19% of those with 50 to 150 bouts and 7% of those with less than 50 bouts (Roberts 1969). This idea of a dose-response relationship between repeated trauma and CTE was supported by the observation that amateur boxers did not suffer neuropsychological deficits due to boxing (Butler 1993). CTE is not unique to boxing, but has occurred in other sports with high rates of head trauma such as wrestling, horseracing, and parachuting as well as a case of battered wife syndrome (Corsellis 1976).

Research into the gross neuropathology of CTE was spearheaded by Corsellis, who studied the brains of 15 deceased boxers, eight of whom were world or national champions (1973). Corsellis identified four common areas of brain damage and their associated clinical symptoms and signs. First, cavum septum pellucidum with fenestrations in the leaflets was a common finding. In addition, the lateral and third ventricles were enlarged and the frontal and temporal lobes were atrophied. These changes were associated with emotional lability and memory impairment. Second, degeneration of the substantia nigra, as evidenced by the loss of pigmented neurons, was associated with Parkinsonian symptoms like tremor, rigidity, and bradykinesia. Third, gliosis and neuronal loss in the cerebellar tonsils was associated with loss of balance and coordination. Fourth, diffuse neuronal loss was associated with an Alzheimer's-like dementia. Eight of the fifteen cases Corsellis studied exhibited all four types of brain damage.

The link between CTE and Alzheimer's was strengthened when in 1967, Constantinidis showed the presence of neurofibrillary tangles in brains affected by traumatic injury (1967). Subsequent research showed that the microscopic pathology of CTE differed from that of Alzheimer's in two important ways. First, CTE exhibited a unique distribution of neurofibrillary tangles in the neocortex. Neurofibrillary tangles in CTE were preferentially distributed in superficial layers of the neocortex - layer II and the upper two thirds of layer III. In contrast, in Alzheimer's they were located primarily in deeper layers - the lower third of layer III and layer V (Hof 1992). Second, whereas beta amyloid plaques are an important feature of Alzheimer's disease, they are not an essential part of CTE. One study showed that

fourteen out of the fifteen brains studied by Corsellis stained positive for beta amyloid deposits (Roberts 1991). However, in a series of 51 CTE cases, McKee found that beta amyloid plaques were present in only 47% of cases (2009).

The pattern of neurofibrillary tangles observed in CTE overlaps with the areas of neuronal loss identified by Corsellis. McKee observed neurofibrillary tangles and tau-immunoreactive astrocytes in parts of the neocortex, basal ganglia, cerebellum, brainstem and spinal cord. The density of neurofibrillary tangles was particularly high in the hippocampus, entorhinal cortex, and amygdala. This suggests involvement of the papez circuit and may explain the emotional lability observed in CTE. In addition, neurofibrillary tangles have been found in the substantia nigra and cerebellum. (McKee 2009).

Recent research has suggested several mechanisms for brain injury in the setting of repeated trauma. Neurofibrillary tangles in CTE have a characteristic perivascular distribution, grouped around small intracortical vessels (Geddes 1999). This finding suggests that trauma may damage the blood-brain barrier, releasing neurotoxins that promote the formation of neurofibrillary tangles around blood vessels. In a similar vein, another study found that in many areas of CTE-affected brains the microvasculature was less dense and tortuous than normal. In addition, the distribution of this pathological microvasculature was highly correlated with the distribution of neurofibrillary tangles. The proposed explanation was that trauma damaged the microvasculature and led to the growth of neurofibrillary tangles. (Buee 1994). On a related note, neurofibrillary tangles in CTE were found to contain

higher levels of iron and aluminum than those in Alzheimer's disease, possibly due to damage to the blood-brain barrier (Bouras 1997).

Diffuse axonal injury is a second possible mechanism of injury. After a concussion, disruptions in axolemma permeability and in axonal transport can lead to axotomy within 24 hours (Maxwell 1995). Indeed, in one study eighty percent of patients who died from acute head trauma showed immunocytochemical evidence of axonal injury (McKenzie 1996).

A third mechanism of brain injury is the deposition of beta amyloid. Although beta amyloid plaques are present in only half of CTE cases, studies have shown that beta amyloid deposition increases after head trauma (Gentleman 1993). In addition, beta amyloid concentration in the brain is correlated with neurological recovery following head trauma (Brody 2008).

Genetic studies suggest that the apolipoproteinE e4 allele predisposes to worse outcomes after traumatic brain injury. One study finds that patients with the APOE e4 allele have a two-fold higher risk of death, vegetative state, or severe disability compared to those without the allele (Teasdale 1997). The mechanism by which APOE e4 influences recovery from traumatic brain injury is unclear, though a role in neuronal repair has been suggested (Chen 1997).

There are many unanswered questions regarding CTE. First, recent case reports indicate that CTE can occur in professional football players and soccer players (Omalu 2005, Matser 1998). The prevalence and risk factors for developing CTE in populations other than boxers are unknown and require further investigation. On this front, public awareness is increasing <https://assignbuster.com/history-of-chronic-traumatic-encephalopathy-psychology-essay/>

and more than 250 current and former NFL players have pledged to donate their brains to the Center for the Study of Traumatic Encephalopathy (CSTE) at Boston University School of Medicine (CSTE 2010). Second, the observation that CTE can present years after retirement from sports cannot be explained by current theories of CTE pathophysiology. Third, there has been no research into potential treatment options, though Parkinson's and Alzheimer's drugs have been used speculatively. Fourth, current preventative measures consist of "return to play" guidelines that sideline players who suffer concussions until their symptoms resolve. This is based on the finding that the risk of a second concussion is increased in the period following a concussion (Cantu 2003). However, no protocols for measuring degree of neurological impairment and reinjury risk in athletes have been developed.

Chronic traumatic encephalopathy is a progressive neurodegenerative disease marked by emotional lability, Parkinsonism, ataxia, and cognitive decline. Since its first description by Martland, much has been learned about this disease. CTE occurs in professional athletes who suffer repeated head injury in a variety of sports, but has not been found in amateurs.

Pathologically, CTE presents with neurofibrillary tangles in a distribution unique from Alzheimer's and with beta amyloid deposits in about half of cases. Various mechanisms of injury have been proposed, though none have been proven. Genetic studies suggest that APOE e4 may promote CTE. Areas ripe for future research include the prevalence of CTE in sports other than boxing and the pathophysiology, treatment, and prevention of this disease.