

Motor Function in Former Professional Football Players with History of Multiple Concussions

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Abstract

The objective of this study was to assess the incidence of motor impairment in former professional Canadian Football League (ex-CFL) players with multiple concussions. We investigated motor symptoms and signs in 45 ex-CFL players with multiple concussions and 25 age- and education-matched healthy controls with no history of concussion. Neurological assessment included items from the SCAT3 (Sport Concussion Assessment Tool 3) and the Unified Parkinson's Disease Rating Scale part III (UPDRS-Part III). A performance-based measurement of manual motor function was undertaken using the Grooved Pegboard test. Cognition was measured with patient-reported outcomes for memory, executive and behavioral symptoms as well as performance-based measures of memory and executive function. Symptoms of anxiety and depression were measured using the Personality Assessment Inventory. There was no significant difference between the ex-CFL players and controls on the UPDRS-Part III scores, and neither group reported clinically significant motor complaints. Ex-CFL players did not perform differently from control subjects on the Grooved Pegboard test. In contrast, with regard to cognitive and mood testing, players were more symptomatic: The ex-CFL players reported significantly more memory (77.8% vs. 16%, respectively, $p < 0.001$), executive (53.3% vs. 8%, respectively, $p < 0.001$), and behavioral symptoms (66.7% vs. 20%, respectively, $p < 0.001$). No significant differences were found when comparing ex-CFL players and controls in performance on memory and executive tests. In summary, in a group of retired CFL players who self-reported declines in memory, executive and behavioral symptoms, no motor symptoms were reported and no motor signs were detected.

Keywords: chronic traumatic encephalopathy; concussion; professional athletes

Introduction

SPORTS-RELATED CONCUSSION or mild traumatic brain injury is extremely common with estimates of 1.6 to 3.8 million occurring yearly in the United States.¹ These injuries are particularly common in contact sports, including boxing, hockey, and football, but can also be seen in the military. While most people recover completely from a concussion, approximately 10–15% of individuals do not and have post-concussion syndrome (PCS).²

The more pressing concern today is the relationship between concussion, persistent symptoms, and later neurodegenerative disease. The possible long-term sequelae of repetitive concussions was first described by Martland in boxers.³ He called the condition “punch drunk syndrome” and described one case with onset of

tremor, gait ataxia, and pyramidal signs without cognitive issues 20 years after the boxer's last match.³ Millspaugh later renamed the syndrome “dementia pugilistica” (DP) and described it as a neurodegenerative disease afflicting boxers that included motor, cognitive, and behavioral impairments.⁴ In 1966, Miller coined the phrase “chronic traumatic encephalopathy of boxers” to describe this constellation of symptoms observed in boxers.⁵ These classic descriptions are notable for their concentration on motor features as an early component of this post-traumatic syndrome.

In recent years, delayed neurodegeneration has been recognized in a number of retired athletes who have had multiple concussions in several other contact sports.^{6,7} In some of them, post-mortem evaluation of the brain revealed a pathology now known as chronic traumatic encephalopathy (CTE), which is a tauopathy, consisting of

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abnormal tau inclusions, similar to those seen in Alzheimer disease but distributed in a different pattern.⁶⁻⁸ According to McCrory and colleagues,⁹ the clinical correlates of what they term “modern CTE” have been found to overlap with many neurodegenerative diseases and include cognitive and behavioral signs and symptoms and, to a lesser degree than “classic CTE,” with signs and symptoms of pyramidal and extrapyramidal motor dysfunction.⁹

Early cases of classic CTE reported in boxers showed significant motor manifestations. McCrory and coworkers⁹ described dysarthria, gait disturbance, tremor, and pyramidal signs in classic CTE based on the studies of Roberts¹⁰ and Corsellis and associates.¹¹ Even in the early stages, mild parkinsonism including tremor, bradyphrenia, and lack of facial expression were observed and as the disease progressed, there was overt parkinsonism with rigidity, bradykinesia, and other extrapyramidal signs.¹² Roberts¹⁰ described signs and symptoms in 224 retired professional boxers and found behavioral symptoms, memory impairment, as well as cerebellar, pyramidal, and extrapyramidal features in 17% of these cases. Jordan¹² similarly found a constellation of symptoms and signs in boxers including dysarthria and balance issues as well as more severe signs such as ataxia, spasticity, impaired coordination, and parkinsonism. In the series of CTE cases reported by McKee and colleagues,¹³ 71% of former professional boxers with CTE had motor symptoms compared with only 13% of former professional football players.

Whereas motor signs and symptoms including dysarthria, gait disturbance, tremor, pyramidal dysfunction, and parkinsonism were a prominent feature of classic CTE, modern CTE as described mainly in former professional football players has been associated with fewer motor symptoms and seemingly earlier cognitive and behavioral symptoms.^{9,13-15} In classic CTE descriptions, mood, behavioral, and cognitive symptoms appeared later.⁹ In view of the differences in descriptions of motor dysfunction in classic (primarily boxing-related) CTE and modern (primarily football-related) CTE, we aimed in our study to specifically investigate the incidence of pyramidal and extrapyramidal motor impairment in former professional football players.

Methods

Forty-five former professional Canadian Football League (ex-CFL) players, each with a history of multiple concussions, and 25 age- and education-matched healthy male controls (HC) with no history of concussion were recruited through the Canadian Concussion Centre (CCC). Subjects (ex-CFL players and HC) were excluded if they had a previous history of diagnosed neurological or developmental disorder, psychiatric illness, stroke, or other illness affecting the brain. The incidence of concussion was ascertained by self-report, determined by the player's ability to recall injuries caused by a blow to the head or body that resulted in concussion symptoms including at least one of the following: headache, nausea, vomiting, dizziness/balance problems, fatigue, trouble sleeping, drowsiness, sensitivity to light or noise, blurred vision, difficulty remembering, or trouble concentrating.¹⁶ Subject demographics are listed in Table 1. All participants completed a standardized history intake and neurological examination. The University Health Network Institutional Research Ethics Board approved the study, and formal consent was obtained from all subjects.

Neurological examination and neuropsychological assessment

The assessment included a history-taking portion and a standardized neurological examination that included the SCAT3 (Sport Concussion Assessment Tool 3).¹⁶ During history-taking, all sub-

TABLE 1. DEMOGRAPHIC DATA FOR RETIRED FOOTBALL PLAYERS AND HEALTHY CONTROLS*

	Ex-CFL (n=45)	Controls (n=25)	p
Age (years)	53.38±10.31	49.96±10.93	0.198
Education (years)	16.13±1.88	16.12±2.26	0.979
CFL career (years)	7.91±3.58	0	-
Number of concussions	5.07±4.21	0	-
Time between testing and last concussion (years)	17.33±14.76	0	-

CFL, Canadian Football League.

*Mean±standard deviation.

jects reported first symptoms (present after last concussion) and current symptoms in the following domains: memory, executive function, language, visuospatial, motor, sensory, behavioral, constitutional, and headache. Each domain consisted of several statements, and subjects responded with no “0” or yes “1” to indicate whether they experienced those symptoms after their last concussion. Current symptoms were rated on a 3-point scale with “0” indicating no problems, “1” indicating problems observed but not interfering with day-to-day life, and “2” indicating problems interfering with day-to-day function.

For the purposes of this study, the 3-point current symptom scale was converted to a dichotomous score wherein “0” indicated absence of a symptom and “1” corresponded to problems present that may or may not interfere with day-to-day function. An overall score was obtained for each domain for current symptoms. A larger sum indicates increased concerns in that domain.

The neurological examination assessed for motor signs including pyramidal, extrapyramidal, and cerebellar function. The Unified Parkinson's Disease Rating Scale part III (UPDRS-Part III) was used to assess extrapyramidal signs. The Grooved Pegboard test, which measures manual motor speed and dexterity, was used to test motor coordination and speed in both the dominant and nondominant hands.

For cognitive testing, the subjects underwent a complete neuropsychological battery that included the Rey Auditory Verbal Learning Test (RAVLT) for assessing memory acquisition and retention, Trails A for speed of processing, and Trails B and the Symbol Digit Modalities Test, (SDMT-Oral) for timed, motor and nonmotor (respectively) measures of executive function. The Personality Assessment Inventory (PAI) subscales for anxiety and depression were used to assess symptoms of anxiety and depression, respectively.

Statistical analysis

Statistical analysis was performed using SPSS version 23 (IBM Corp., Armonk, N.Y.). Chi-square or Fisher exact tests were used to compare ex-CFL and HC groups on self-reported measures and neurological signs. Student *t* tests were performed to compare age, years of education, and performance on neuropsychological tests between ex-CFL and HC groups. We ran an analysis of variance to compare the three groups of ex-CFL players.

Results

Demographic data

Subject age, years of education, self-reported number of concussions, time as a CFL player, and time between study visit and last concussion are summarized in Table 1 for each group. There was no significant difference between the two groups for age or education.

Neurological examination and neuropsychological assessment

Motor outcomes. There were no significant differences in self-reported motor symptoms between the two groups. Similar to HC subjects, ex-CFL players reported few motor complaints. Three ex-CFL players complained of motor weakness that did not interfere with their day-to-day function, and their power was normal on neurological examination. Two ex-CFL players had unsteadiness as a complaint, but they had a normal neurological examination.

On physical examination, one ex-CFL player had mild dysmetria on heel-shin testing and another had mild slowing of right hand finger tapping. Two other ex-CFL players had mild left upper extremity action tremor, and one of these had wide-based gait. No HC subject had pyramidal or cerebellar dysfunction on examination. At the group level, the pyramidal and cerebellar findings did not differ significantly between the ex-CFL and HC groups.

Three ex-CFL players had a UPDRS-Part III score of 4, three had a score of 3, and three had a score of 1. One HC subject had a UPDRS-Part III score of 4. The UPDRS-Part III scores did not differ significantly between the ex-CFL and HC groups (chi square (df 3, n = 70) = 4.01, p = 0.260).

Performance-based measure. On the Grooved Pegboard test assessing motor coordination, ex-CFL players did not perform differently from control subjects using their dominant (45.29 vs. 46.92, p = 0.574, respectively) or nondominant (77.69 vs. 81.24, p = 0.477) hand.

Cognitive outcomes

Participant-reported outcomes. Table 2 shows that as a group, the ex-CFL group self-reported significantly more memory problems (35/45 [78%] vs. 4/25 [16%], p < 0.001), executive problems (24/45 [53%] vs. 2/25 [8%], p < 0.001), and behavioral symptoms (30/45 [67%] vs. 5/25 [20%], p < 0.001) than the HC group. Twenty ex-CFL players complained that their short-term memory symptoms were interfering with day-to-day function. Seven of the ex-CFL players with memory complaints also had complaints of executive deficits, but these did not interfere with day-to-day function.

Performance-based outcomes. Table 3 shows that there was no significant difference between the ex-CFL and the HC groups on SDMT-Oral version (0.53 vs. 0.32, p = 0.369), PAI-anxiety (47.16 vs. 43.72, p = 0.105), PAI depression (49.30 vs. 45.24, p = 0.121), RAVLT learning score (6.59 vs. 6.80, p = 0.626), RAVLT long delayed recall score (-0.25 vs. -0.29, p = 0.885) and short delayed recall score (0.13 vs. -0.13, p = 0.282), and the Trails

TABLE 2. SUBJECTIVE COMPLAINTS FOR MEMORY, EXECUTIVE AND BEHAVIORAL SYMPTOMS IN RETIRED FOOTBALL PLAYERS AND CONTROLS

Assessment	Ex-CFL players n (%)	Controls n (%)	Chi square	p
Memory complaints	35 (77.8)	4 (16.0)	24.859	< 0.001
Executive complaints	24 (53.3)	2 (8.0)	14.147	< 0.001
Behavioral complaints	30 (66.7)	5 (20.0)	14.000	< 0.001

CFL, Canadian Football League.

TABLE 3. COMPARISON BETWEEN EX-CFL (N = 45) AND HEALTHY CONTROLS (N = 25) ON ASSESSMENTS OF COGNITION AND MOOD

Assessment	Ex-CFL M (SD)	Controls M (SD)	p
Grooved Pegboard dominant hand	45.29 (10.65)	46.92 (13.07)	0.574
Grooved Pegboard Nondominant hand	77.69 (20.85)	81.24 (17.97)	0.477
Trails A T-score	57.11 (9.81)	50.36 (14.49)	0.023*
Trails B T-score	55.73 (11.07)	50.04 (12.18)	0.051
SDMT Oral Z-score	0.53 (0.94)	0.32 (1.0)	0.369
RAVLT learning score	6.59 (1.66)	6.80 (1.78)	0.626
RAVLT Short Delay Z-score	0.13 (0.97)	-0.13 (1.00)	0.282
RAVLT Long Delay Z-score	-0.25 (1.05)	-0.29 (1.06)	0.885
PAI Depression	49.30 (10.60)	45.24 (9.69)	0.121
PAI Anxiety	47.16 (7.81)	43.72 (9.15)	0.105

CFL, Canadian Football League; SDMT, Symbol Digit Modalities Test; RAVLT, Rey Auditory Verbal Learning Test; PAI, Personality Assessment Inventory. *p < 0.05.

B score (55.73 vs. 50.04, p = 0.051). Ex-CFL players performed better than the HC group on the Trails A (57.11 vs. 50.36, p = 0.023).

Mood outcomes

There were no significant differences between the groups on the PAI clinical scales of anxiety (47.16 vs. 43.72, p = 0.105, respectively) or depression (49.30 vs. 45.24, p = 0.121, respectively).

Position played

To investigate whether position played had an effect on our results, we divided up the players into linebacker, tight ends (n = 16), skill positions (n = 9), and lineman (n = 19) and found no significant difference in age, education, years played, total number of concussions, or performance on any of the cognitive or motor performance-based outcomes.

Discussion

We evaluated motor symptoms and signs in ex-CFL players with histories of multiple concussions and a group of age- and education-matched controls without a history of concussion. We also reviewed complaints of memory, executive, and behavioral changes in both groups. In this study, there were no significant differences in motor symptoms and signs between the ex-CFL and HC groups, although pyramidal, cerebellar, or extrapyramidal abnormalities were found in a minority of ex-CFL players. Although we did observe significantly more memory, executive, and behavioral complaints in the ex-CFL players, like others, we did not find a significant difference on neuropsychological assessment for cognition, executive function, and mood between the two groups.^{17,18}

Previous research on CTE has described symptoms from multiple domains, specifically in cognition, behavior, mood, and motor function.¹⁹ Classic CTE, the dementia pugilistica originally described in boxers, was associated with significant and early motor

symptoms, particularly parkinsonism.¹⁹ Dementia pugilistica was observed mainly in boxers with long careers and high numbers of blows to the head.^{13,20} Modern CTE, described primarily in former professional football players, has been associated only rarely with motor deficits including parkinsonism.⁹

The reason for this difference in motor symptoms between dementia pugilistica (classic CTE) and cases of modern CTE is unclear. One possible explanation may be different mechanisms of injury in dementia pugilistica compared with modern CTE. Recent cases of CTE tend to be the result of multiple concussions in athletes sustained in a variety of contact team sports including football and hockey. Angular acceleration and torsional injury to the brainstem and cerebellum may be more common in boxing than other contact sports, and this type of injury may predispose to parkinsonism and cerebellar features.^{19,21} Perhaps the most likely explanation is simply related to the enormous differences in total blows to the head incurred by boxers in comparison with players in contact team sports.

Modern cases of CTE are associated with earlier cognitive and behavioral impairment compared with classic CTE. Cognitive features observed in CTE, based on retrospective studies, include memory impairment, executive dysfunction, lack of insight, perseveration, impaired concentration, language problems, visuospatial dysfunction, reduced intelligence, and dementia.²² In the current study, we found a high frequency of subjective cognitive complaints of memory problems and executive impairment in ex-CFL players, significantly different from the HC group. It is unknown whether these memory complaints represent early involvement or injury of the hippocampus, entorhinal cortex, and/or medial thalamus or to other, nonlimbic dysfunction—i.e., frontal lobe structures.²³ Previous studies in this group have revealed focal injury to both gray matter and white matter structures.^{24–27}

Behavioral changes are described in both classic and modern CTE. These personality changes include explosivity, loss of control, impulsivity, aggression, rage, physical and verbal violence, inappropriate speech, childish behavior, disinhibition, social inappropriateness, paranoid delusions, psychosis, and social isolation.²² Although present in both classic and modern CTE, behavioral changes feature early on in modern CTE whereas in classic CTE, there has been a greater emphasis on motor deficits in addition to behavioral changes early in the disease. Studies have implicated injury to the orbitofrontal regions in football players as an explanation for behavioral and mood issues in those with CTE.^{28,29} CTE-specific tau pathology has been observed in limbic structures in definite cases of CTE and may also explain behavioral symptoms.²³

The clinical signature of modern CTE remains to be elucidated because it has been impossible to perform prospective studies without *in vivo* detection of CTE. Based on a retrospective chart review, progressive cognitive and behavioral impairments were found to include deterioration in social and cognitive function, altered mood and behavior, changes in interpersonal relationships, criminal and violent behavior, alcohol and drug abuse, hyperreligiosity, headaches, and generalized body pain.⁶

Again based on retrospective chart review, McKee and colleagues⁶ endeavored to classify the clinical features of CTE by dividing the disease into four stages that included: stage 1—headaches, loss of attention and concentration; stage 2—depression, impulsivity, and short-term memory loss; stage 3—decision-making dysfunction and cognitive impairment; and stage 4—dementia, word-finding difficulty, and aggression. This modern staging proposal has not been evaluated prospectively, and the relationship between clinical

features and amount and distribution of tauopathy is currently unknown.⁶

As well, copathology is encountered frequently in CTE, making it impossible in certain cases to ascribe accurately the etiology of a symptom.^{6,30} Hazrati and coworkers³⁰ reported the pathological findings on six retired CFL football players with a history of multiple concussions and progressive neurocognitive decline. They found CTE in only half the cases, and all three CTE cases showed concomitant neurodegenerative pathology including Alzheimer disease, vascular disease, and Lewy body disease. McKee and associates¹³ also found that 52% of CTE cases had concomitant neurodegenerative pathology, with Alzheimer disease and Lewy body disease being the most common.

Gardner and coworkers³¹ reported a 44% increased risk of Parkinson disease in patients with traumatic brain injury compared with those who had trauma to other parts of their bodies. They concluded that traumatic brain injury can be an independent risk factor for other neurodegenerative diseases, although these patients were older, and falls could have been a symptom of their disease onset in some cases. Interestingly, Fournier and colleagues³² reported that head injury does not affect the disease progression or the neurodegenerative process in patients with amyotrophic lateral sclerosis.

Limitations of this study include recall bias for the number of concussions incurred, because players may not recall many minor injuries or some more serious concussions that were associated with amnesia. Further, there was a variable interval between a player's last concussion and assessment such that longer retired players who played when concussions were less readily recognized may have had more difficulty recalling their concussions. Comorbidities such as pain, obstructive sleep apnea, and substance abuse, which can affect cognition and mood, were not included in this analysis, because the focus was on motor symptoms. Last, cerebrovascular disease, which has been overlooked in many of the current case series, can affect cognition and motor function and can be present as a comorbidity in many cases of neurodegenerative disease. This would need to be considered in evaluating the cause of parkinsonism in cases of CTE.³³

Conclusion

In the current study, we found considerable memory, executive, and behavioral symptoms in the group of ex-CFL players compared with the HC group. There was, however, no significant difference in symptoms or signs of motor impairment, although motor signs were found in a small minority of the ex-CFL group. These findings highlight an important difference between modern and classic CTE that warrants further study.

Acknowledgments

The authors would like to thank the participants of this study for their contribution.

Author Disclosure Statement

No competing financial interests exist.

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