Persistent Motor System Abnormalities in Formerly Concussed Athletes

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Context: The known detrimental effects of sport concussions on motor system function include balance problems, slowed motor execution, and abnormal motor cortex excitability.

Objective: To assess whether these concussion-related alterations of motor system function are still evident in collegiate football players who sustained concussions but returned to competition more than 9 months before testing.

Design: Case-control study.

Setting: University laboratory.

Patients or Other Participants: A group of 21 active, university-level football players who had experienced concussions was compared with 15 university football players who had not sustained concussions.

Intervention(s): A force platform was used to assess centerof-pressure (COP) displacement and COP oscillation regularity (approximate entropy) as measures of postural stability in the upright position. A rapid alternating-movement task was also used to assess motor execution speed. Transcranial magnetic stimulation over the motor cortex was used to measure longinterval intracortical inhibition and the cortical silent period, presumably reflecting γ -aminobutyric acid subtype B receptor-mediated intracortical inhibition.

Main Outcome Measure(s): COP displacement and oscillation regularity, motor execution speed, long-interval intracortical inhibition, cortical silent period.

Results: Relative to controls, previously concussed athletes showed persistently lower COP oscillation randomness, normal performance on a rapid alternating-movement task, and more M1 intracortical inhibition that was related to the number of previous concussions.

Conclusions: Sport concussions were associated with pervasive changes in postural control and more M1 intracortical inhibition, providing neurophysiologic and behavioral evidence of lasting, subclinical changes in motor system integrity in concussed athletes.

Key Words: traumatic brain injuries, transcranial magnetic stimulation, clinical neurophysiology, motor control, primary motor cortex

Key Points

- Collegiate football players who had sustained a concussion more than 9 months earlier displayed persistent alterations
 in postural control and more primary motor cortex intracortical inhibition.
- Neurophysiologic and behavioral evidence is presented for lasting, subclinical changes in motor system integrity in athletes with a history of concussion.

The incidence of sport concussions has substantially increased over the last 15 years, and these injuries are now considered a major public health concern, with an estimated 50000 to 300000 new cases occurring every year in the United States.¹ Although recovery from cognitive impairments after sport concussion has drawn most of the attention from the scientific community in the last few decades, the investigation of motor system abnormalities has recently come to the forefront of the sport concussion literature. Indeed, posturalstability assessment in various stances and on various surfaces was integrated into clinical practice to assist clinicians in determining when concussed athletes who experienced balance problems could safely return to play.² Postural stability typically returns to baseline levels within a few days after concussion on conventional measures of center-of-pressure (COP) displacement,³⁻⁵ but approximate entropy (ApEn) calculation as a nonlinear dynamic measure of postural control has greater sensitivity to subtle physiologic alterations associated with sport concussion.⁴ More specifically, this measure was introduced to detect changes in COP oscillation randomness when participants attempt to stand as steadily as possible on a force platform. In contrast to conventional measures of COP displacement, which can reflect only overall magnitude of COP displacement over a defined time window, the ApEn calculation considers the sequential order of successive data points during a trial.⁶ Therefore, this temporal analysis probably provides a measure of the participant's ability to produce context-adapted, rapid online postural adjustments. Compared with that of nonconcussed athletes, COP oscillation randomness was reduced from postconcussion day 1 to day 4, especially when participants stood still on a fixed surface, in both eyes-open and eyesclosed conditions.⁵ Authors⁵ have advocated nonlinear ApEn measures of postural stability as a valuable measurement alternative to reduce uncertainty in return-to-play decisions,⁵ and assessing long-term recovery of COP oscillation abnormalities beyond the acute postconcussion phase could provide further support for clinical utility. This approach may be especially relevant considering that balance control in a dual-task condition involving gait and a simple mental task was still affected in concussed athletes relative to a control group 28 days after injury.⁷

In parallel, transcranial magnetic stimulation (TMS) has shed light on persistent motor cortex excitability alterations after sport concussion. Previously demonstrated in patients tested within 2 weeks of sustaining mild to moderate head injuries,⁸ increased intracortical inhibition of the primary motor cortex (M1), as reflected by the duration of the TMS-induced cortical silent period (CSP), was found in previously concussed athletes who had been asymptomatic, on average, for 2 years before testing.9 In addition, this prospective study showed that athletes with multiple concussions who sustained another concussion displayed further increases in M1 intracortical inhibition.9 Another group¹⁰ demonstrated the chronicity of this pervasive CSP duration lengthening in otherwise healthy, former university-level contact-sport athletes who had sustained their last sport concussions more than 3 decades earlier. Although the biological substrates of CSP duration modulation are uncertain, research has pointed to changes in intracortical inhibitory systems of the motor cortex mediated by γ -aminobutyric acid subtype B (GABA_B) receptor activity.¹¹⁻¹⁵ The main inhibitory neurotransmitter in the human central nervous system (CNS), GABA is involved in numerous CNS functions.¹⁶ Slice preparation studies indicate that GABA receptors (particularly GABA_B receptors) play an important role in regulating neuronal excitability and long-term potentiation.^{17,18} Perhaps most pertinent in the context of this study, the administration of GABA_B receptor-agonist baclofen was recently found to suppress long-term potentiation-like plasticity in human M1.19 In animal studies, this increase in GABA neurotransmission prevented long-term potential-dependent motor learning.^{20,21} Although no direct evidence exists for the involvement of GABA receptors in postconcussive brain alterations, abnormal GABA transmission has been reported in rat models of brain injury.^{22,23}

In addition to CSP lengthening,^{9,10} previously concussed former athletes also displayed lower motor execution velocity than nonconcussed former athletes on a rapid alternating movements (RAM) task. This task was selected because RAM velocity is known to decline with age²⁴ and to be altered in patients with moderate to severe head injuries who performed normally on neuropsychological tests²⁵ at least 1 year after injury. Most notably, RAM velocity was strongly correlated with M1 intracortical inhibition anomalies among previously concussed former athletes.¹⁰

Therefore, our purpose was to report on the persistent effects of concussions on motor system functions—namely COP oscillation regularity,⁵ motor execution on an RAM task,¹⁰ and M1 intracortical inhibition^{9,10}—that have not been assessed in young, previously concussed athletes who have long since received medical clearance to return to competition. In addition, we sought to further validate the persistent and cumulative dysfunctions of M1 intracortical inhibition with the introduction of the more widely accepted and less empirically debated long-interval intracortical inhibition (LICI) TMS paradigm.¹²

METHODS

Participants

All 36 participants were active male players (age = 22.3 ± 3.45 years; range, 19 to 26 years) from 2 Canadian university football teams, recruited with the help of the team physician, who provided information about the number of previous sport concussions. Participants were included if they met all of the following criteria: no history of alcohol or substance abuse; no medical condition necessitating daily medication; and no previous history of psychiatric illness, learning disability, neurologic history, or traumatic brain injury unrelated to contact sports. The study was approved by the local ethics committee, and all participants provided written informed consent before testing. Volunteers received financial compensation of CaD \$50 for their participation.

The study consisted of 2 groups. The concussed group was composed of 21 university-level football players who had sustained their last sport concussion more than 9 months before testing.

Lingering concussion-related effects on TMS measures in university football players have been demonstrated in this time frame since injury.9 The number of concussions per athlete ranged from 1 to 5 (mean = 2.65 ± 1.45), and the time since the last concussion ranged from 9 to 34 months (mean = 19.03 ± 13.77 months). Information on concussions sustained while the athletes were at the university was obtained from the medical records, whereas information on concussions sustained before the athletes entered college was mostly self-reported. At the time of testing, concussed athletes were asymptomatic, reporting very few (if any) symptoms on the Postconcussion Symptom Scale $(mean = 2.15 \pm 2.08 \text{ symptoms})$ ²⁶ The control group consisted of 15 university football players who reported no history of sport concussion or neurologic insult. Unequal group sample sizes reflected limited access to high-demand football players within regional university settings. The 2 groups were equivalent in terms of age ($F_{1,34}$ =0.58, P>.05), postconcussion symptoms $(F_{1.34}=0.02, P>.05)$, and level of education $(F_{1.34}=0.21, P>.05)$. All 36 participants completed both experimental sessions.

Procedures

The experiment consisted of two 1-hour testing sessions that took place 1 to 5 weeks apart during the football off-season. During the first session, a concussion history questionnaire, a general health questionnaire, the Postconcussion Symptom Scale, and the TMS protocol were administered. (For more information on the questionnaires, see our previously published article.²⁷) The second session consisted of the RAM task and postural-control assessment.

Postural-Control Paradigm. Participants were instructed to stand as steady as possible in an upright position on a force platform (model OR6-5; Advance Mechanical Technology, Inc, Watertown, MA) with their eyes open and feet side by side, parallel, at pelvis width. Two trials separated by a 60-second resting period were recorded, and each trial lasted 30 seconds. Analyses were computed for the first trial except in the cases of 2 participants who had flawed first-trial recordings because they did not accurately follow the task instructions. We used second-trial recordings in these participants because negligible practice effects were demonstrated in between-trials analyses for participants with 2 valid trials ($F_{1,33}$ =0.025, P>.05). Fur-

thermore, within-subject COP displacement during quiet standing with feet side by side is fairly stable across trials.²⁸ *Postural stability* referred to the root mean square amplitude of COP displacement in both the mediolateral (ML) and anteroposterior (AP) directions. The ApEn values were computed using test trials for the ML and AP components of the COP coordinates.⁵

The RAM Task. Each participant was seated on a straightback chair and told to keep his elbows close to his trunk and flexed at an angle of 90°. He was instructed to rotate 2 handheld spheres as quickly as possible, with maximal movement amplitude (complete pronation-supination at the wrist). To track the participant's hand position and orientation in 3-dimensional space, we placed 4 infrared light-emitting diodes at strategic positions on the spheres. The coordinates of the diodes were recorded at a frequency of 200 Hz using a 3-dimensional motion-analysis system (Optotrak Certus, Northern Digital Inc, Waterloo, Ontario, Canada), and hand orientation was later analyzed using customized analysis software (MATLAB, The MathWorks, Inc, Natick, MA). For each of the 3 conditions (both hands, left hand only, right hand only), 2 periods of 15 seconds each were recorded, separated by a 2-minute pause. Further analyses were conducted on the first-trial data except in 3 cases in which a number of missing diodes prevented appropriate analyses.

Velocity and performance were the main performance measures computed using the algorithms developed by Okada and Okada²⁹ and adapted by others.^{30–32} *Velocity* is a composite measure of range/duration (ie, average angular displacement for a pronation-supination cycle/time per cycle). *Sharpness* reflects the delays associated with changes of direction; more delays reflect less sharp pronation-supination turns. (See references 29– 32 for detailed descriptions of these performance measures). Finally, *bimanual coordination* refers to movement synchrony between hands (ie, smaller values reflect better synchrony).

We derived bimanual coordination scores in the following way. Angular variations of the 2 hands were normalized as a function of each hand's maximal rotation amplitude. We subtracted angular variations computed for the dominant hand from those of the nondominant hand, such that a resulting horizontal line would indicate perfectly synchronized hands. We then computed deviations (in absolute degrees) from the perfect horizontal plane for each sampling point (200 Hz) and averaged them to obtain a bimanual coordination score for each trial.

The TMS Recordings and Data Analysis. The TMS was performed using a figure-8 coil positioned optimally to elicit motor evoked potentials in the right first dorsal interosseous muscle. The CSP duration was calculated at 3 TMS intensities. Five single-pulse stimulations for each of 3 TMS intensities (110%, 120%, and 130% of the resting motor threshold [rMT] intensity) were applied to the left M1 while participants maintained a voluntary isometric muscle contraction of the right first dorsal interosseous muscle at approximately 10% of maximum strength. The CSP duration was calculated with the graphical method described by Garvey et al.³³ An interstimulus interval of 100 milliseconds was used to assess LICI.¹² The intensity of the conditioning stimulus was set at 120% of the rMT, and the test stimulus intensity was adjusted to induce motor evoked potentials of approximately 1 mV peak-to-peak amplitude. Fifteen motor evoked potentials each were collected for the test stimulus alone and for the conditioning stimulus test condition. The LICI was presented as the following ratio: test stimulus/ conditioning stimulus.

Statistical Analyses

All values are expressed as mean±SD. Demographic information, TMS data, postural-stability scores, and ApEn values were subjected to standard descriptive statistics and analyses of variance. Simple contrast analyses were computed to assess between-groups differences for CSP across TMS intensities. Twotailed Pearson correlations were calculated between the LICI and CSP values of previously concussed athletes and between the number of previous concussions and the LICI, CSP, and postural-stability values. Tukey corrections for multiple comparisons were subsequently applied. Power statistics were also computed for between-groups differences across experimental measures.

RESULTS

Postural Control

The ApEn values were lower (ie, more regular) in asymptomatic, previously concussed athletes than in control athletes in the AP direction ($F_{1,35}$ =8.90, P<.05, Cohen d=1.03) but not in the ML direction ($F_{1,35}$ =1.48, P>.05, Cohen d=.40) (Figure 1A). In contrast, between-groups analysis of variance (ANOVA) was not significant for RMS amplitude of COP displacement in either the AP ($F_{1,35}$ =1.210, P>.05, Cohen d=.26)



Figure 1. A, Approximate entropy values expressed as the centerof-pressure (COP) oscillation regularity in the mediolateral and anteroposterior directions (range, 0–2). Greater approximate entropy values reflect more COP oscillation randomness. B, Root mean square amplitude of COP displacement for the anteroposterior and mediolateral directions. Smaller COP displacement amplitude reflects better postural stability. $^{a}P < .05$.

or ML ($F_{1,35}$ =1.24, P>.05, Cohen d=.28) direction (Figure 1B).

The RAM Task Results

In a 2 (groups)×2 (hand dominance)×2 (number of hands) 3-way ANOVA for velocity, the 3-way interaction was not significant ($F_{2,34}$ =2.888, P>.05). In sharp contrast to findings in former athletes with a history of concussion at least 30 years earlier,¹⁰ these young, previously concussed athletes performed pronation-supination cycles with greater velocity than the control group ($F_{1,35}$ =6.87, P<.05, Cohen d=0.97). Further analyses revealed that this was true for 2 hand conditions (dominant hand: $F_{1,35}$ =8.02, P<.05; both hands: $F_{1,35}$ =5.87, P<.05), whereas only a trend toward significance was found for the velocity measure computed for the nondominant hand ($F_{1,35}$ =2.89, P<.1). As expected, the main effect of hand condition was significant ($F_{2,35}$ =7.07, P<.05). However, bimanual coordination was equivalent across groups ($F_{1,35}$ =2.28, P<.15, Cohen d=0.57). In the bimanual task condition, computing an overall performance score on the RAM task with equal weight on velocity and bimanual coordination (velocity score×[1/ bimanual coordination score]) revealed that the groups were equivalent ($F_{1.35}$ =1.01, P>.05).

In the 2 (groups)×2 (hand dominance)×2 (number of hands) 3-way ANOVA for sharpness, the group×hand condition interaction was not significant ($F_{2,34}$ =1.78, P>.05, Cohen d=0.51). Groups did not differ according to sharpness ($F_{1,35}$ =3.11, P>.05). Finally, the main effect of hand condition was not significant ($F_{2,35}$ =1.00, P>.05).

The TMS Results

Relative to controls, a 1-factor between-groups ANOVA revealed that previously concussed athletes exhibited lower LICI ratios ($F_{1.35}$ =5.96, P<.03, Cohen d=.82) (Figure 2A). In a 2 (groups)×3 (stimulation intensity) 2-way ANOVA for CSP, the group×intensity interaction was not significant ($F_{2.30}$ =1.17, P>.05). More importantly, the main effect of group revealed that previously concussed athletes exhibited CSP prolongation relative to control athletes ($F_{1.35}$ =15.61, P<.001, Cohen d=1.14) (Figure 2B). As expected, the main effect of



Figure 2. A, Long-interval intracortical inhibition, expressed as the ratio of the conditioning stimulus-test stimulus/test stimulus. Intensity of the conditioning stimulus was set at 120% of the resting motor threshold, and test stimulus intensity was adjusted to induce motor evoked potentials of approximately 1 mV peak-to-peak amplitude. B, Cortical silent period duration when transcranial magnetic stimulation at 3 intensities (110%, 120%, and 130%), expressed as a percentage of the resting motor threshold, was applied to the vertex while participants in each group performed a voluntary isometric muscle contraction of the right hand first dorsal interosseous muscle at approximately 10% of maximum strength. $^{a}P < .05$.

intensity yielded a difference in CSP duration across all groups $(F_{235}=80.11, P<.001)$.

Furthermore, LICI in concussed athletes correlated with the duration of the CSP elicited when pulses were delivered at intensities of 120% and 130% of the rMT (120%: r=0.479, P<.05; 130%: r=0.501, P<.05), whereas the Pearson correlation computed with CSP at 110% did not reach statistical significance (r=0.214, P>.05).

Two-tailed Pearson correlations between the number of previous concussions and the LICI ratio values were correlated (r=0.47, P<.05). Similarly, CSP duration correlated with the number of previous concussions for both 120% (r=0.52, P<.05) and 130% of rMT conditions (r=0.49, P<.05). The correlation between COP oscillation regularity and the number of previous concussions did not reach significance (r=0.261, P<.15). Finally, velocity scores on the RAM task were not correlated with the number of previous concussions (r=-0.114, P>.05). The Table summarizes data from the 3 main outcome measures.

DISCUSSION

Relative to athletes who had no history of concussion, the current study revealed 3 main findings about previously concussed athletes who had returned to competition 9 months before testing: (1) They exhibited a persistent decrease in COP oscillation randomness only in the AP direction while displaying equivalent RMS amplitude displacements on COP measures, (2) they performed normally on a RAM task, and (3) they demonstrated an increase in intracortical inhibition in M1, the extent of which increased as a function of the number of previous concussions.

Consistent with previous data,⁵ previously concussed athletes who resumed competition more than 9 months before testing still exhibited greater COP oscillation regularity according to the ApEn measure of postural control, despite equivalent postural-stability scores on conventional, linear measures. Although the functional significance of greater COP oscillation regularity with regard to postural stability is still largely unknown, previous authors^{5,34} suggested that it represents an adaptive compensatory mechanism to allow concussed athletes to achieve postural stability. More specifically, we know that ankle muscles dominate the regulation of postural stability in the AP direction²⁸ and that contracting these muscles increases control over postural sway and, consequently, decreases COP oscillation randomness. One possible explanation for increased COP oscillation regularity could therefore be that concussed athletes deliberately increase cocontraction of the lower extremity musculature to compensate for postural-stability losses.⁵ Another possibility is that concussive injuries result in stiffened lower extremity musculature. However, acquired lower musculature stiffness after concussion is at odds with concussed athletes' increased M1 intracortical inhibition, considering that muscle stiffness has been associated with reduced M1 inhibition.^{35,36}

In parallel, the present increase in COP oscillation regularity specific to the AP direction contrasts with a previous report⁵ on concussion-related effects that showed increases in both the AP and ML directions on postconcussion day 1 and decreased ApEn values in the ML time series at day 4. Although underlying concussion-related pathophysiologic substrates that might mediate this increased COP oscillation regularity are unknown, a recent group⁶ applied ApEn calculations to assess the effects of a secondary cognitive task on postural stability in healthy young adults. They showed higher ApEn values in the COP AP time series that were not apparent on conventional linear measures of postural control. This added measurement sensitivity was proposed⁶ to originate from the fact that ApEn takes into account the sequential order of successive data points, in contrast with traditional linear measurements, which can reflect only the overall magnitude of COP displacement. Moreover, the authors⁶ suggested that higher ApEn values specific to the AP direction during the dual-task condition reflected this documented higher ApEn measurement sensitivity. This suggestion is consistent with a previous report²⁸ showing that control of AP displacement by the ankle muscles is the chief mechanism of upright postural control when the feet are side by side and that a force platform records more AP displacement in this condition than ML displacement.

In sharp contrast to formerly concussed athletes who experienced concussions 30 years earlier¹⁰ and demonstrated motor execution slowness on the RAM task, young concussed athletes attained significantly better scores than controls. However, when equal weight was attributed to velocity and bimanual coordination precision, performance was equivalent

Motor System Measure	Dependent Variables		Statistical Analysis	P Value
Postural control	Approximate entropy	Anteroposterior	F=8.90	<.05
		Mediolateral	F=1.48	>.05
	Linear postural-	Root mean square		
	control measure	amplitude	F=1.24	>.05
Rapid alternating movements	Velocity		F=6.87	<.05
(pronation-supination cycles)	Bimanual coordination		F=2.28	>.05
	Sharpness		F=3.11	>.05
	Overall performance index		F=1.01	>.05
Intracortical inhibition of	Cortical silent period		F=15.61	<.05
primary motor cortex	Long-interval intracortical inhibition		F=5.96	<.05
Correlation with number of	Cortical silent period	120% of resting motor		
previous sport concussions		threshold	r=0.52	<.05
	Long-interval intracortical	130% of resting motor		
	inhibition	threshold	r=0.047	<.05
			r=0.49	<.05

Table. Statistical Analysis of the 3 Main Motor System Measures Reflecting Group (History of Concussion, No History of Concussion) Main Effects

across the groups. Relative to controls, previously concussed athletes therefore appeared to favor speed over movement accuracy. This qualitatively distinct performance across groups may be mediated by various factors extraneous to concussions, including performance motivation. Athletes in the acute postconcussion phase are highly motivated to downplay the effects of the injury to accelerate return to play.^{37,38} Although we are only speculating, the speed-accuracy tradeoff we found might reflect greater performance motivation among previously concussed participants, especially because the task instructions placed more emphasis on speed than on movement precision. Unlike the older, previously concussed athletes who displayed CSP prolongation that strongly correlated with motor execution slowness,¹⁰ the young athletes displayed prolonged CSP duration without motor execution slowness. This inconsistent pattern across age groups, coupled with unknown concussionrelated pathophysiologic features affecting both CSP and motor execution speed, warrants caution when interpreting findings. The normal aging process has repeatedly been associated with motor execution slowness,³⁹⁻⁴¹ so a history of sport concussions might render the aging, concussed brain particularly vulnerable to further movement slowness, at least partly through lifelong intracortical inhibition abnormalities. Given that many professional athletes retire in their late thirties, longitudinal studies could be helpful in characterizing the presence of motor execution slowness and possibly associated functional impairments in formerly concussed athletes. Similarly, knowing that aging is associated with increased amounts of postural sway,42 which may ultimately lead to falls, longitudinal follow-up could inform us about the long-term repercussions of concussionrelated increases in COP oscillation regularity in comparison with former athletes lacking a history of concussion.

Among active university football players, those presenting with a history of sport concussion showed more LICI and longer CSP duration relative to their nonconcussed counterparts. In accordance with numerous studies43-45 suggesting that CSP and LICI reflect similar M1 intracortical inhibitory mechanisms, LICI correlated with CSP duration. Furthermore, altered M1 intracortical inhibition was strongly associated with the number of previous concussions: athletes who sustained more concussions typically exhibited more M1 intracortical inhibition. In conjunction with the demonstrated direct increase in LICI with intake of the GABA_B agonist baclofen,¹² our results provide compelling evidence that sport concussions induce longlasting alterations of intracortical inhibition at least partially mediated by GABA_B receptor activity.^{11,13–15,46} Although Pearson correlations between M1 intracortical inhibition indices and the number of previous sport concussions are considered strong,⁴⁷ we should remain cautious when interpreting such associations because derived coefficients of determination (r^2) indicate that only 25% of M1 intracortical inhibition variance can be explained by the number of previous sport concussions. Consequently, other intervening factors may contribute to the known long-term and cumulative effects of sport concussions. The absence of correlations between COP oscillation regularity and CSP or LICI in concussed athletes also points to the complexity of the pathophysiology of concussion. This finding is consistent with recommendations in the consensus statement of the Third International Conference on Concussion in Sport⁴⁸ suggesting that multidisciplinary assessments benefit the management of patients with concussion.

Having to rely on concussion history self-reports as opposed

to medical records for sport concussions that occurred years before testing is not optimal. Prospective studies conducted with young athletes followed longitudinally are therefore needed to validate the persistent, cumulative effects of concussions observed in the present study. Another major limitation to the present study is the lack of imaging results. In fact, one possible explanation for our findings could be their potential association with structural damage related to sport concussions; in addition to exhibiting more severe postconcussion alterations, athletes with multiple concussions are more likely to have sustained structural damage. Adding structural imaging in future studies would be instrumental to systematically addressing this issue.

In summary, we showed that sport concussions induced pervasive changes in postural control and more M1 intracortical inhibition, providing neurophysiologic and behavioral evidence of lasting, subclinical changes of motor system integrity in previously concussed athletes. Normal performance of young, previously concussed athletes on a RAM task also suggests that rather than being induced by sport concussions alone, motor execution slowness symptoms evidenced 30 years after concussion¹⁰ seem to be at least partially mediated by the combined adverse effects of aging with a history of sport concussions.

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