Chapter 8
Acute and Lingering Impairments in Post-concussion Postural Control

Thomas A. Buckley

Abstract The fourth International Consensus Statement on Concussion in Sport reports that 80–90 % of concussions recover in 7–10 days. Impairments in postural control are a cardinal symptom following a sports-related concussion; however, many studies suggest that these impairments resolve within 3–5 days post-injury. Multiple recent studies, utilizing diverse and sophisticated research paradigms, are suggesting that this may be premature and that prolonged recovery could be normal. Therefore, the overarching purpose of the studies reported herein is to investigate impairments in postural control following a concussion and to identify recovery. We investigated the efficacy of "non-novel" tasks including gait initiation, gait variability, gait termination, and static stance and track the individual's performance across time to identify residual impairments compared to performance on the standard clinical assessment battery. In the acute aftermath of a concussion, the subjects demonstrated substantial impairments in postural control across all tasks which are consistent with a multiple previous investigations. However, the novel findings were the identification of persistent and lingering impairments in postural control which were present despite apparent full recovery on all clinical measures. Specifically, the impairments were more apparent when evaluating central control mechanisms (e.g., movement strategies and anticipatory postural adjustments) as standard kinematic variables returned to premorbid values in a timelier manner. These results suggest that individuals may be returning to sports participation prior to complete concussion recovery and could be a mechanism for the high recurrent concussion rate as well as recent speculation associating concussions and other sports-related injuries.

Keywords Postural control • Concussion • Gait • Recovery

T.A. Buckley, Ed.D. (✉)
Department of Health and Kinesiology, Georgia Southern University,
62 Georgia Avenue, Box 8076, Statesboro, GA 30460, USA
e-mail: Tbuckley@Georgiasouthern.edu

S.M. Slobounov and W.J. Sebastianelli (eds.), *Concussions in Athletics: From Brain to Behavior*, DOI 10.1007/978-1-4939-0295-8_8,

Introduction

As discussed throughout this text, sports-related concussion has reached epidemic levels with estimates of up to 3.8 million concussions occurring annually in the United States [1]. However, some estimate that this may only reflect the tip of the iceberg as over half to three-quarters of all concussions may go unreported [2–4]. In order to appropriately manage sports-related concussions accurate, sensitive, and specific diagnostic tools are required. Ideally, athletes would be forthcoming about symptoms following a potential injury, but many athletes are clearly unaware of common concussion symptoms [2, 3, 5]. Further, numerous high-profile cases exist of athletes opening lying about concussion symptoms (e.g., New York Jets quarterback Greg McElroy), admitting they would lie about symptoms (e.g., Brian Urlacher), not report symptoms (e.g., Troy Polamalu), downplaying the seriousness of the injury (e.g., Maurice Jones Drew), indicating a necessity to play through a concussion (e.g., Calvin Johnson), or intentionally trying to sandbag the baseline testing protocol to hasten return to participation (e.g., Peyton Manning) [6]. While standard imaging technology (e.g., MRI, CT) is effective in identifying structural pathology, these same procedures are not sensitive to the largely physiological pathology of concussion [7, 8]. Recent imaging advances including functional MRI (fMRI), diffusion tensor imaging, MR spectroscopy, and others hold promise for future utilization; however, they remain as research tools and are not recommended for routine clinical care [7–11]. Similarly, there have been multiple attempts at identifying a blood biomarker (e.g., 100-B, UCH-L1) of concussion which, although promising, is likely not ready to move beyond research utilization [12–15]. Neuropsychological testing, while a valuable contribution to concussion management, has limitations including low to moderate test–retest reliability, low sensitivity, a small practice/learning effect, potential "sandbagging" of the test, and test administration differences [16–25].

Accurate and timely recognition of a sports-related concussion is critical in preventing associated sequelae. Specifically, the failure to acutely identify the presence of a concussion potentially exposes the individual to the rare, but often fatal, second impact syndrome (SIS) [26–28]. While the specific neurophysiology of SIS remains elusive, it is generally believed to result from altered cerebral autoregulation following a head injury whereby the brain is unable to regulate cerebral and intracranial pressure [26]. This loss of autoregulation results in rapid cerebral vascular congestion, increased intracranial pressure, brain herniation, and often death within minutes [26, 27]. SIS occurs when an athlete who has suffered an initial concussion suffers a second concussion before the symptoms associated with the first concussion have fully cleared [29]. A recent review of catastrophic head injuries highlighted the need to restrict participation until symptom-free as almost 60 % of football players suffering catastrophic head injuries had a previous head injury and almost 40 % admitted to playing despite residual symptoms of the prior head injury [28].

Fortunately, SIS is an extraordinarily rare condition; however, appropriate concussion management is vital to reduce the risk of repeat concussion. Once the individuals suffer a single concussion, they are at a three- to sixfold increased risk of

suffering a second same-season concussion and over 90 % of the repeat injuries occur within the first 10 days post-injury, potentially suggestive of a window of increased vulnerability [30–33]. Further, this repeat concussion is likely to present worse and has a prolonged recovery time [34, 35]. Finally, recent evidence identified over the last decade has suggested an association between concussions and later-life neuropathologies including mild cognitive impairment [36], clinically diagnosed depression [37], potentially earlier onset of Alzheimer disease [36], chronic traumatic encephalopathy [38, 39], and amyotrophic lateral sclerosis [40]. Thus, it is clearly imperative for health care providers to accurately identify concussions acutely as well as properly manage the condition post-injury. Therefore, this chapter will explore the utilization of postural control as a biomarker of both concussion diagnosis and recovery.

Postural Control and Concussion

The phrases postural control, postural stability or instability, balance, and equilibrium are unfortunately frequently used interchangeably in both the lay vernacular and, occasionally, the professional literature [41, 42]. Postural control involves regulating the body's position in space for the dual purposes of stability and orientation whereas postural stability is the ability to control the center of mass (COM) in relationship to the base of support [43]. The COM refers to the weighted average, in 3D space, of each of the body segments and is generally considered to be the key variable in the postural control system [42–44]. The control of the COM during either static or dynamic tasks is generally categorized into three neurological components: (1) motor processes, (2) sensory processes, and (3) supraspinal or cognitive processes [43]. The motor processes include the organization of muscles throughout the body into neuromuscular synergies [43]. The sensory processes comprise three systems: (1) visual system, (2) vestibular system, and (3) somatosensory system [42]. The visual system is primarily involved in planning locomotion and avoiding obstacles; the vestibular system, sometimes referred to as the body's gyro, senses linear and angular acceleration [42, 45]. Finally, the somatosensory system has multiple responsibilities including sensing the position and velocity of bodily segments, their contact with external objects, and the orientation of the body relative to gravity [42, 45]. The role of the cognitive processes in postural control is an emerging area of research with focus on "attentional resources" [43]. There are two primary theories underlying cognitive control of posture: (1) "Capacity theory" which is based on the sharing of a limited set of neurological resources and (2) "Bottleneck theory" which suggests there is a competition between tasks for limited neurological resources and a prioritization occurs [43]. Overall, postural control is the resultant of complex interactions between multiple bodily systems which have to work cooperatively to control the orientation and stability of the body [43].

Nearly all neuromuscular disorders result in some degeneration in the postural control systems and concussions are not an exception [42]. Indeed, the adverse

effects of a concussion on postural control have been well elucidated in the literature [46]. Briefly, a deficit in the interaction between the visual, vestibular, and somatosensory systems is generally considered to be the underlying post-concussion neuropathology [47, 48]. Specifically, post-concussion it is believed that the individual is unable to appropriately integrate sensory input, ignore altered environmental conditions, and apply the appropriate motor control strategies to maintain precise postural control [47–49]. Recently, an increased focus on vestibular considerations for post-concussion balance impairments had evolved and led to recommend for vestibular therapy in cases of delayed or prolonged recovery [50, 51]. Finally, others have speculated that either diffuse axonal injury or the post-concussion neurometabolic cascade plays either a primary or secondary role in post-concussion impairments in postural control [30, 52]. Current clinical assessment batteries of postural control, utilizing either the balance error scoring system (BESS) or sensory organization test (SOT), have suggested that postural control recovers within 1–5 days post-injury, frequently prior to symptom resolution or achieving baseline values on computerized neuropsychological tests [47, 53].

Post-concussion Postural Control Assessment Battery

The original assessment of postural control following a concussion incorporated the Romberg test [54, 55]. The Romberg test, originally developed in 1853, was designed to subjectively assess somatosensory impairment in individuals with neurological conditions [56, 57]. However, the Romberg test was criticized for failing to objectively identifying subtle post-concussion balance deficits [49]. More recently, force plate measures have been developed to assess postural control and are valid and reliable and numerous metrics have been investigated [41, 58–63]. One commonly used research system, occasionally referred to as the "gold-standard," is the SOT which is thoroughly reviewed in separate chapter in this textbook. Generally, the SOT is both valid and reliable with impairments in postural control noted for 3–5 days post-injury and suggested that the vestibular system of the sensory processes is most commonly impaired [41, 46, 64–68]. However, force plate (>$10,000) and sophisticated balance systems (SOT: >$75,000) are expensive, likely cost-prohibitive for the overwhelming majority of sports medicine clinical sites, and may require extensive additional training or the addition of a biomechanist to the sports medicine staff. Indeed, even amongst NCAA Division I athletic trainers, less than 1 % reported utilizing the SOT [69]. Thus, a cost-effective and practical postural control assessment paradigm was required to appropriately assess post-concussion impairments.

Current consensus of appropriate concussion management, both during the sideline or acute concussion assessment or when tracking recovery, calls for a multifaceted assessment battery as no single test is highly sensitive [7, 20, 70]. The fourth International Consensus Statement on Concussion in Sport (4th CIS) recommends a two-component balance assessment: (1) a modified BESS (mBESS) and/or

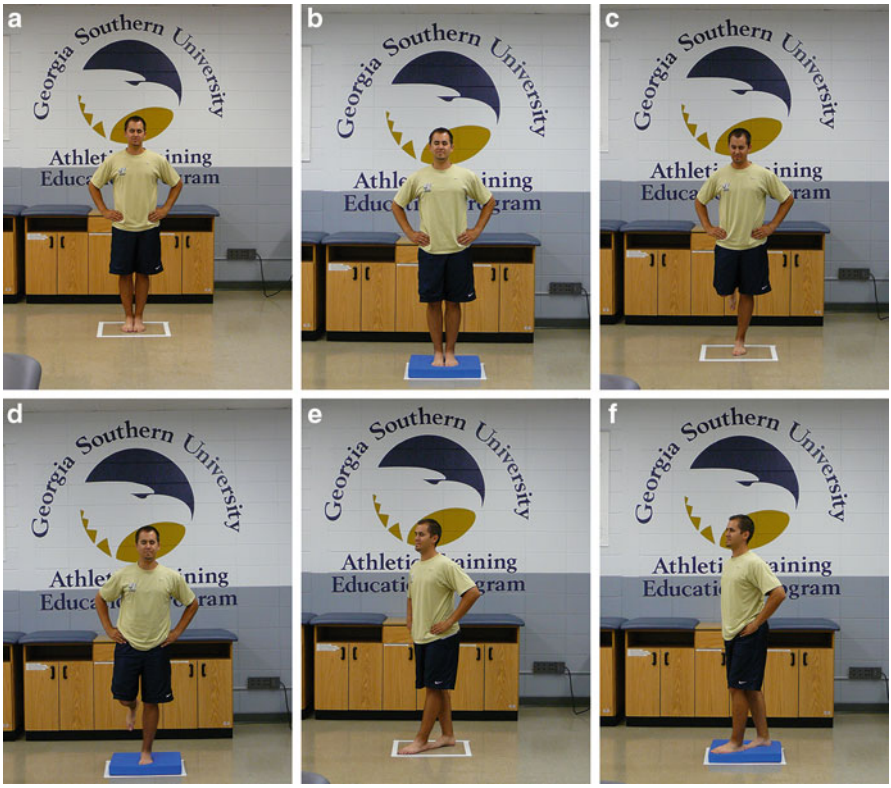

Fig. 8.1 The six stances of the balance error scoring system (BESS) test. Conditions (**a**)–(**c**) are on a firm surface while conditions (**d**)–(**f**) are on a foam surface. Conditions (**a**) and (**d**) have both feet on the surface and in contact, conditions (**b**) and (**e**) are single leg, and conditions (**c**) and (**f**) are tandem stance. Each stance is performed for 20 s and the total numbers of errors per stance (maximum of 10 per stance) are summed for a total score

(2) tandem gait [7]. The mBESS consists of three stances (double, single, tandem) on a single surface which is solid [7]. The mBESS has received limited attention in the literature; however, normative data suggests the scores increase (worsen) with aging and obesity [71]. The tandem gait assessment consists of a heel-to-gait for 3 m along a 38 mm wide piece of tape, a 180° turn, and returning along the same walkway [7]. The test is repeated four times and the best trial time is recorded as the individuals score [7]. While this test has not been evaluated post-concussion, some evidence suggests a dynamic gait assessment may be more reliable and less influenced by fatigue than a static test such as BESS [72].

While the mBESS and tandem gait are the current recommendations of the 4th CIS, the more commonly used postural control assessment post-concussion remains the original BESS test [69, 73–75]. The original BESS consists of three stances (double, single, and tandem) on two surfaces (firm and foam) with errors being counted for deviations from the test position [56, 65]; see also Fig. 8.1.

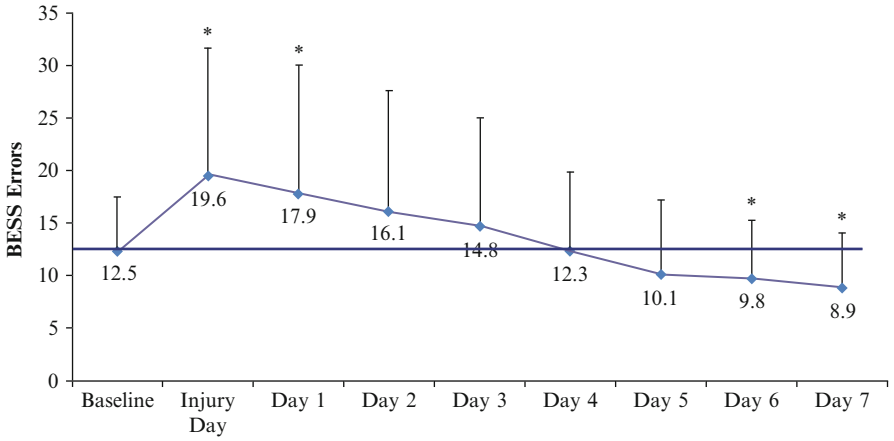

Fig. 8.2 BESS scores across time. There was a significant increase (worsening) in BESS score at immediate post-injury ($p=0.001$) and day 1 post-injury ($p=0.010$). There was a significant decrease (improvement) in BESS score at day 6 ($p=0.045$) and day 7 ($p=0.012$) despite over 20 % of participants still endorsing symptoms for at least 6 days

The BESS appears sensitive to acute concussion with an increase of 6–8 errors post-injury being commonly reported [53, 76]. The specificity of the BESS remains >0.91 through the first week post-injury; however, the sensitivity is low immediately post-injury, 0.34, and continues to decrease to 0.16 over the first 3 days post-injury [76, 77]. Unfortunately, the minimal detectable change values for the BESS test range from 7.3 (intrarater) to 9.4 (interrater) [78]. An additional considerable limitation to the BESS is a noted practice effect, potentially due to the test's utilization of foam to perturb the somatosensory system [79]. Repeat administration, as quickly as the second administration of the test, has repeatedly demonstrated a significant reduction in the number of errors committed [80–82]. Further, this improvement has been noted to persist for the duration of a fall athletic season, 90 days [83]. Our post-concussion assessment protocol involves daily BESS testing, as is common amongst athletic trainers [69], and, similar to previous studies [53], shows an increase (worsening) of BESS score in the immediate 24 h post-injury; however, with repeat administration there was a significant decrease (improvement) of BESS score, as compared to baseline, within a week post-injury, often prior to symptom resolution (Fig. 8.2).

This would inappropriately suggest that balance actually improves post-concussion. These limitations have resulted in the suggestion to conduct multiple baseline testing sessions [80, 84]; however, this is not being incorporated by most athletic trainers likely due to time constraints [69]. Additional limitations of the BESS include fatigue, dehydration, functional ankle instability, neuromuscular training, and testing environment [85–92]. Finally, the influences of previous common sports injuries (e.g., ankle or knee sprains) which occur after the baseline testing but prior to a post-concussion assessment have not currently been elucidated.

Overall, the current utilization of the BESS test, despite being the most commonly used postural control assessment tool, is fundamentally flawed as there is scant evidence that multiple baseline tests are occurring nor that the post-injury limitations are being considered [69].

These assessment batteries are typically performed in a single-task manner (i.e., only a motor task without concurrent cognitive tasks); however, an emerging line of research suggests that dual-task testing may be advantageous in the post-concussion population [93–96]. This is the next logical step in concussion assessment as Winter has suggested that the central nervous system is capable of adapting for a loss of function following a pathology until the patient is deprived of the compensating system [42]. This is consistent with recent findings related to compensatory strategies seen in diverse testing paradigms post-concussion [97–105]. Currently, most post-concussion dual-task testing protocols utilize sophisticated computerized equipment to perform the assessment with balance assessments performed with the SOT and either an auditory or visual switch task as the cognitive challenge [94, 106]. Unfortunately, as previously discussed, these tests are likely impractical for most clinicians as they lack both equipment and training to perform the assessments [69]. A second line of dual-task motor and cognitive challenges involves gait and working memory tasks which will be discussed in the next section.

Postural Control During Motor Tasks Post-concussion

Early evidence of gait impairments following concussion were reported by McCrory based on video analysis of concussions sustained in the mid-1990s by competitors in Australian rules football [107]. Gait impairments, operationally defined as ataxic, stumbling, or unsteady gait, were noted post-injury in 41 % of concussed athletes with the majority manifesting symptoms immediately post-injury; however, a small percentage, 14 %, had a minimal delay of 10–20 s prior to the onset of gait unsteadiness [107]. While not specifically studied, it was speculated that gait unsteadiness involved a brainstem pathology and was multifactorial including postural tone, cerebellar, and labyrinthine function [107]. This study, while limited to gross video observations without true biomechanical assessment, provided foundational evidence of post-concussion gait impairments.

Compared to other commonly investigated neurological pathologies (e.g., Parkinson disease, elderly fallers, stroke, amputee), investigations of gait to identify impairments in postural control post-concussion have been fairly limited. Indeed, there are more review articles (e.g., systematic reviews, meta-analyses) on gait in the elderly than original research articles related to concussion and gait. The majority of gait studies were performed at one laboratory and were largely delimited to grade II concussions, as defined by the American Academy of Neurology (no LOC and symptoms persisting longer than 15 min) [108], had fairly homogeneous and small ($n = 10–17$/group) participant populations for most studies, lacked within-subject pre-injury data, and have involved a variety of gait tasks including single-task gait,

dual-task gait with working memory challenges, and obstacle avoidance tasks [98–104, 109–111]. Finally, not all raw data is provided for all dependent variables of interest limiting the ability to perform a meta-analysis of the findings.

Utilizing traditional clinical measures of balance (e.g., BESS), large cohort investigators have suggested that postural control returns to its baseline value within 3–5 days post-injury [53]. The post-concussion gait studies which have been currently published are limited by lack of within-subjects baseline data; however, they are generally tightly matched to otherwise healthy control subjects. Within this context, gait velocity generally appears to return to a normal value by day 5 or 6 post-injury despite still experiencing concussion-related symptoms [99–101, 111], although in one study it had not recovered by day 28 [110]. This finding and other similar findings need to be taken in context as an apparent practice effect was potentially a confounding variable as the gait velocity steadily increased with each testing session in the healthy control group. However, by day 28 the concussion subjects had still not reached the initial and lowest gait velocity of the control subjects [110]. Similar findings were noted in the stepping characteristics (stride time, width, and length) [111] and sagittal plane COM measurements (anterior displacement and velocity of the COM and the anterior center of pressure [COP]–COM separation) [99, 111]. Frontal plane kinematics may be a more challenging task post-concussion as there is a limited base of support during the single-support phase of gait [112]. During single-task gait, post-concussion participants demonstrated limited increases in the medial to lateral COM range of motion and velocity [98, 102, 104, 111]. These impairments appear to persist for up to 28 days post-injury despite apparent recovery on the traditional clinical assessment battery [104, 111]. Interestingly, in many of these studies there was an apparent recovery on many dependent variables by day 5 post-injury, but significant differences reemerged 2–4 weeks later. These findings suggest either potential differences on day 5 are not statistically significant due to small groups and the possibility of the study being underpowered or some residual consequence of delayed impairment following a concussion. Overall, these gait studies suggest that a conservative gait strategy has been adopted post-concussion, although the rationale for these strategies remains unknown.

Adopting from methodologies utilized with elderly and diseased state patients, the addition of a cognitive challenge to the motor task of gait is beginning to be explored post-concussion. Both impairments in postural control and cognitive processing are known acute consequences of a concussion and thus not surprisingly both are abnormal when tested within 24 h of the injury. Post-concussion gait studies utilizing a dual-task paradigm have largely focused on utilizing working memory challenges (e.g., reciting the months of the year backwards) from the mini mental examination to assess cognitive performance while performing either level over ground gait or obstacle avoidance [113]. A recent systematic review and meta-analysis by Lee et al. [96] suggested that gait velocity and frontal plane range of motion are sensitive markers of dual-task interference in post-concussion individuals (Fig. 8.3).

Specifically, a pooled mean decrease in gait velocity of 0.13 m/s was noted across the meta-analysis which is similar to a noted decrease of 0.17 m/s across a

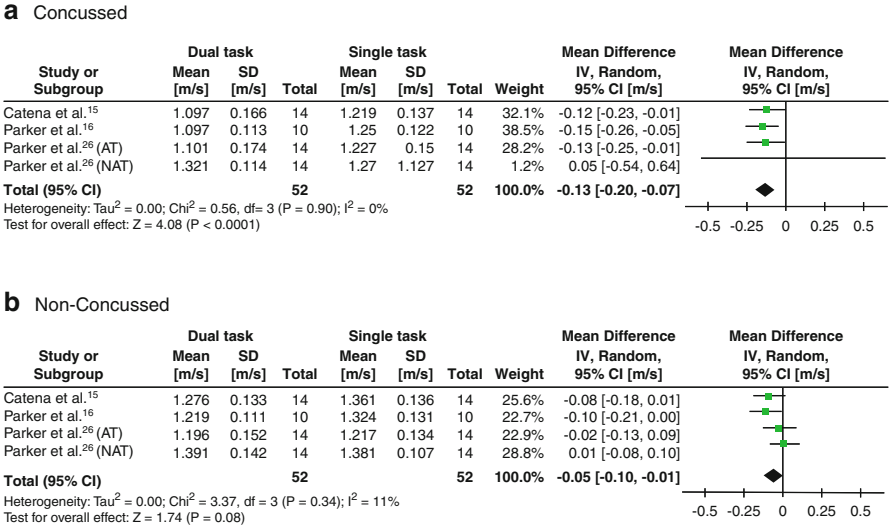

Fig. 8.3 Meta-analysis of dual task. Forest plots for concussed (**a**) and non-concussed (**b**) groups for gait velocity at day 2. (◇): pooled mean estimate of the differences between ST and DT conditions; (□): difference between ST and DT conditions for individual studies; *AT* athletes, *NAT* non-athletes; *horizontal bars* represent 95 % confidence intervals. Lee H, Sullivan SJ, Schneiders AG. The use of the dual-task paradigm in detecting gait performance deficits following a sports-related concussion: A systematic review and meta-analysis. J Sci Med Sport. 2013:16(1): 2–7 (Permission received from publisher)

diverse population of neurologically impaired participants [96, 114]. In the acute recovery phase following a concussion, similar to single-task gait, multiple impairments are noted during dual-task gait. Specifically, when compared to tightly matched control subjects, reductions in stride length, anterior velocity of the COM, and COM displacement in the frontal plane have been reported [98–103, 110]. Consistent with many dual-task paradigms, most kinematic characteristics of gait were reduced with the addition of a cognitive task in both the recently concussed and healthy control groups [98–103, 110]. The recovery patterns of dual-task gait were similar to, but expand upon, the single-task gait with apparent lingering deficits still present up to 28 days post-injury [99–101, 110]. Once again, the frontal plane kinematics appeared most sensitive to the identification of delayed recovery following a concussion [112]. These findings support the necessity of a multifaceted concussion assessment as most cognitive and postural control assessments are recovered far before 28 days post-injury [53]. Further, when compared to a commonly utilized computerized neuropsychological test, there was little relationship with the dual-task gait performance [99]. The authors speculated that dynamic motor tasks, such as dual-task gait, are potentially more complex and challenging than traditional computerized neuropsychological tests and may better approximate the demands experienced during sports participation [99].

The results of these combined studies suggest that impairments in postural control persist for up to 1 month post-injury despite resolution on the traditional clinical assessment battery. Further, an interesting, but unexplained, finding was the apparent recovery within a week post-injury, but residual impairment in performance which persisted up to a month. Future research needs to elucidate the reasons for this altered performance.

Experimental Post-concussion Postural Control

The remaining postural control data presented in this chapter is derived from the Georgia Southern University Concussion Management research protocol. This data represents 84 participants (Ht: 1.74 + 0.13 m; weight: 79.7 + 23.5 kg; age: 19.6 + 1.4 years; 50.7 % with a previous history of concussion [0.8 + 1.1 overall]) who suffered a sports-related concussion. The concussion initial presentations (9.5 % LOC; 34.5 % posttraumatic amnesia) and recovery timelines (symptom-free: 4.8 + 3.1 days; BESS recovery: 2.9 + 2.8 days; standard assessment of concussion recovery [SAC]: 2.2 + 1.8 days) are consistent with previous large epidemiological studies [32, 33, 53]. All participants completed a graduated and progressive return to participation exercise protocol, generally consistent with the third International Consensus Statement on Concussion in Sport (3rd CIS) [7], and the average time to unrestricted return to participation was 12.6 + 5.1 days.

The post-injury assessment protocol has been modified over the years as new information and recommendations have been incorporated. Specifically, the protocol was established in the late 2008 and did not incorporate computerized neuropsychological testing until 2010. The postural control testing occurred in the biomechanics laboratory which contains four force plates (AMTI, Watertown, MA) and an instrumented walkway (GAITRite; CIR Systems, Sparta, NJ); see also Fig. 8.4. Following a concussion, injured student-athletes performed the BESS,

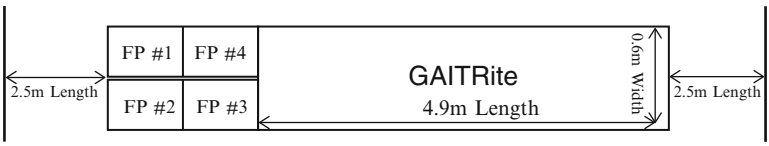

Fig. 8.4 Biomechanics laboratory set-up. The gait initiation trials began with the participant standing on force plates (FP) #1 and #2, having the first football impact on either force plates #3 or #4, and continuing down the instrumented walkway to a target end line 2.5 m beyond the walkway. Gait termination trials transversed the instrumented walkway and terminated with the penultimate step impacting force plates #3 or #4 and the termination step occurring on force plates #1 and #2. Stepping kinematics were recorded from the instrumented walkway during both initiation and termination trials. Buckley TA, Munkasy BA, Tapia-Lovler TG, Wikstrom EA. Altered Gait Termination Strategies Following a Concussion. Gait and Posture. (epub March 11, 2013) (Permission received from publisher)

SAC, and a graded symptom checklist (22-items, 0–6 Likert scale) daily until they achieved their baseline values on each specific test. Participants were tested daily with over 90 % compliance.

Acute Concussion Response

The control of posture and locomotion are interdependent at several levels of the central nervous system [115]. Therefore, impaired posture and gait components may contribute to deficits in locomotion due to adaptive changes in neural control [115]. Many post-concussion balance assessments (e.g., BESS, SOT) are novel challenges and, as described, are subject to a substantial practice effect with repeat administration [79–83]. Thus, we have opted to utilize what we refer to as "non-novel" tasks—these are tasks which are performed as regular activities of daily living and therefore not subject to a practice or learning effect. One task commonly utilized to investigate the interactions between posture and locomotion components is gait initiation (GI). Indeed, GI, the phase between motionless standing and steady state locomotion requiring the generation of propulsive forces, has been shown to be a sensitive indicator of balance dysfunction [116]. GI challenges the postural control systems as it is a volitional transition from a large stable base of support to a smaller continuously unstable posture during gait [117]. From a motor control perspective, GI requires the central nervous system to regulate the spatial and temporal relationship between the position and motion of the COM [118]. Therefore, GI has been used to quantify impairments in postural instability amongst elderly, Parkinson disease, stroke, and amputee patient groups [119–125].

During static stance, the COP and COM are tightly coupled and located just anterior to the malleolus [124]. To initiate gait, they must decouple to generate forward momentum while maintaining upright balance [124, 126]. Initially, the COP moves posteriorly and laterally towards the initial swing limb (Fig. 8.5). This anticipatory postural adjustment (APA), controlled by the supplementary motor area and/or premotor area, involves bilateral tibialis anterior activation and soleus inhibition [127–129]. The initial posterior COP movement generates the forward momentum needed to separate the COP and COM while the lateral COP displacement, controlled by the gluteus medias, propels the COM towards the initial stance limb [130]. This momentum generation is necessary to achieve successful forward locomotion while maintaining upright balance. Thus, the initial posterior and lateral COP displacements are sensitive indicators of balance dysfunction [119, 120, 125, 131].

Following a sports-related concussion, impairments in GI have been noted. A typical healthy adult will displace his or her COP approximately 5–7 cm both posteriorly and laterally during the APA phase of GI. One day post-concussion, the otherwise healthy adults' APA posterior displacement was 2.59 + 1.62 cm; a 131 % decrease compared to a normal healthy adult. Similarly, the lateral displacement of the COP post-concussion is reduced to 3.43 + 1.92 cm; a decrease of ~75 % from a healthy adult. As the posterior and lateral displacement during the APA is believed

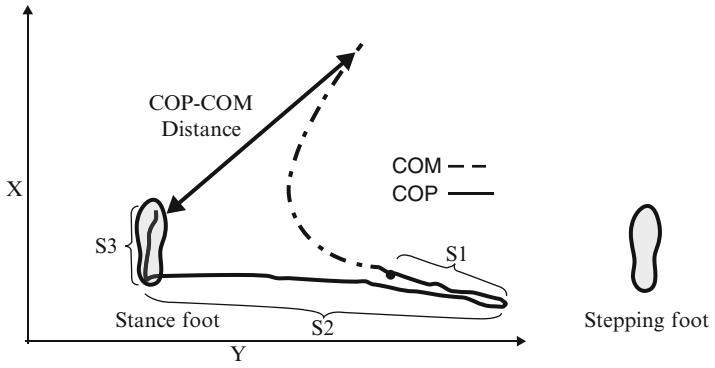

Fig. 8.5 The center of pressure (COP) and center of mass (COM) displacement during GI. When standing quietly, the COP is roughly equally distributed between the 2 ft. Upon movement initiation, the COP is displaced posterior and lateral towards the initial swing limb (S1). The S1 phase is the anticipatory postural adjustment (APA) phase of GI. As the initial swing limb leaves the ground, the COP is then displaced laterally towards the initial stance limb (S2). Finally, as the initial stance limb leaves the ground, the COP moves anteriorly under the foot (S3). Hass CJ, Waddell DE, Fleming RP, Juncos JL, Gregor RJ. Gait initiation and dynamic balance control in Parkinson's disease. Archives of Physical Medicine and Rehabilitation. 2005 Nov;86(11):2172–6 (Permission received from publisher)

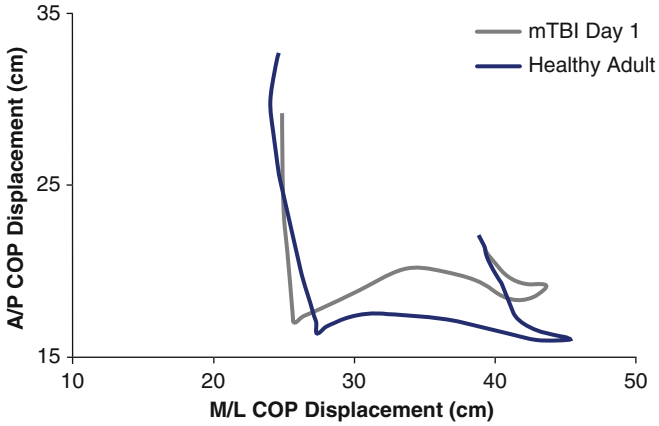

Fig. 8.6 COP exemplar trace for day 1 post-injury and a healthy control subject. The APA phase represents the posterior and lateral shift, towards the initial swing limb, which occurs prior to movement. The normal healthy adult typically has 5–7 cm of displacement in both the posterior and lateral directions; however, the post-concussion reduced both the posterior (2.59 + 1.62 cm) and lateral (3.43 + 1.92 cm) displacements by 131 % and 75 %, respectively

to generate the momentum needed to accelerate the COM forward, it is not surprising that the initial step length (0.60 m) and velocity (0.58 m/s) are substantially reduced compared to population norms [125]. An exemplar COP displacement trace is provided in Fig. 8.6 and particular attention should be paid to the APA phase noting that the COP at movement initiation is nearly identical between traces.

This postural conservative strategy, unlikely to be associated with a fear of falling as is commonly suggested in neurologically impaired older adults, is consistent with gait-based studies comparing post-concussion postural control to healthy adults.

While comparison to healthy individuals is valid, comparing the individuals to their own premorbid performance is ideal. While the observed differences may appear small (i.e., only a few centimeters), the effect size of these differences needs to be considered. Effect size is a measure of the magnitude of the difference between groups and a value of 0.2 is considered a small effect, 0.5 a medium effect size, and 0.8 a large effect size. There is some debate on using effect size on within-subjects measures, but this is largely focused on varying treatment effects which are not present within this data set [132]. Following a sports-related concussion, individuals reduce their APA posterior displacement from a premorbid value of 5.46–2.34 cm, statistically significant ($p<0.001$) with a large effect size (d) of 1.99. Similarly, the lateral displacement during the APA phase is reduced from 5.55 to 3.25 cm, statistically significant ($p<0.001$) with a moderate effect size of 0.51. The reductions in APA COP displacement are likely associated with the reduction in initial step length (PRE: 0.68 + 0.11 m and day 1: 0.60 + 0.09, $p=0.001$, $d=0.37$) and step velocity (PRE: 0.67 + 0.17 m/s and day 1: 0.58 + 0.15 m/s, $p=0.021$, $d=0.27$). These results suggest that the largest impairments are noted in the APA component of GI as opposed to the resulting stepping characteristics. This likely occurs as the APA component is a supraspinal or central control process whereas the stepping characteristics are likely controlled at multiple levels including supraspinal (motor cortex), spinal (central pattern generators), and peripheral (local neuromuscular adaptations) [127–129, 133–135].

The post-concussion response to gait has been well established through a series of studies conducted in Li-Shan Chou's lab at the University of Oregon and has been discussed previously [98–105, 109–111]. Our data is similar with noted deficits on the day following the concussion. Specifically, substantial decrements in performance were noted in gait velocity (PRE: 1.49 + 0.13 m/s and day 1: 1.17 + 0.13 m/s, $p<0.001$, $d=0.77$), mean step length (PRE: 0.76 + 0.04 m and day 1: 0.64 + 0.05 m, $p=0.002$, $d=0.76$), percentage of the gait cycle in double support (PRE: 22.48 + 2.38 % and day 1: 24.59 + 2.23, $p=0.035$, $d=0.42$), and percentage of the gait cycle in the swing phase (PRE: 38.75 + 1.12 % and day 1: 37.7 + 1.21 %, $p=0.05$, $d=0.41$). In a small subgroup, increases in gait variability, expressed as a coefficient of variation, have been identified post-concussion. Gait variability is an indicator of the rhythmicity and gait stability and is known to be impaired in elderly individuals or those with neurological impairments [136–139]. A variability of greater than 7 % has been associated with impairments in postural control in older individuals with neurological impairments; however, in healthy adults normal variability is below 3 % [140–144]. While the post-concussion individuals in this study did not exceed the 7 % threshold, there were increases from baseline (<3 %) to day 1 post-injury (>3 %). Consistent with the Oregon findings, these results suggest a conservative gait strategy is adopted following a concussion. However, the neurophysiological explanation has not been fully elucidated.

These previous findings suggest that, acutely post-concussion, impairments in postural control are identified with single (motor)-task challenges; however, emerging evidence suggests that reallocation of attentional resources and/or neural plasticity may allow the individual to overcome simple single-task challenges [62, 97, 145]. Dual-task challenges examine the effect of executing a secondary cognitive task (e.g., mental processing) on the concurrent performance of a primary motor task (e.g., walking) [145]. Even routine motor activities, such as sitting, standing quietly, or walking, require cognitive processing [146]. Previous investigations noted impaired postural control during both quiet stance and gait in healthy young adults under dual-task conditions [147, 148]. Simultaneous performance of a motor task and a cognitive task may interfere with the performance of one or both, probably due to competing demands for inherently limited attentional resources [149–151]. Utilizing working memory challenges (e.g., serial 7's), post-concussion participants had further reductions in the displacement of the COP during the APA phase of GI and took shorter and slower steps than during single-task GI. Consistent with the findings from the Oregon studies, those differences were also present during gait with significant reductions in gait velocity, stride length, single-support phase, and swing phase when compared to healthy young adults. Interestingly, there were no differences noted within subjects when comparing single- and dual-task gait; potentially due to an inverted "ceiling" effect whereby individual's performance during single task was already dramatically impaired. Indeed, both single- (1.17 m/s) and dual-task (1.15 m/s) gait velocities fell below the 1.2 m/s threshold often cited for a healthy gait in elderly and neurologically impaired individuals [152, 153].

Gait termination (GT) is not a mirror image of GI [154]. Rather, GT is a process by which the central nervous system anticipates, controls, and arrests the forward momentum of the COM without exceeding the borders of the base of support [155, 156]. Further, GT has a known and invariant set of parameters that constrains the multiple degrees of freedom within the lower extremity [157–159]. However, GT poses a unique challenge to the postural control systems because the COM is often located outside the base of support at the onset of GT [126]. As a result, GT is an excellent model for investigating alterations in motor programming and neurologic dysfunction. The central neurophysiologic control of GT is more elusive than GI; however, an fMRI study has suggested that the prefrontal area, specifically the inferior frontal gyrus and the pre-supplementary motor area, likely controls GT [154]. Indeed, GT has quantified impairments in postural control amongst the aging, people with Parkinson disease, amputee groups, chronic ankle instability, and those with general balance disorders [158–168].

The termination of gait requires the coordinated activity of both legs. Indeed, force production is modulated bilaterally such that the lead limb (limb behind the COM) reduces foot push-off propulsive forces as the swing limb (limb in front of the COM) concurrently increases vertical and anteroposterior braking forces [157, 158, 169]. Reduced propulsive forces are caused by soleus inhibition and increased activation of the tibialis anterior while concurrent increases in braking forces are due to an increased soleus activity and inhibition of the tibialis anterior [166, 170].

Table 8.1 Means, standard deviations, 95 % confidence intervals, and effect sizes for gait termination performance

	Control ($n=26$)	Day 1 post-concussion ($n=15$)	Control to day-1 post hoc p-values (effect size)	Day 10 post-concussion ($n=12$)	Control to day-10 post hoc p-values (effect size)
Gait velocity (m/s)	1.32 ± 0.14 (1.26–1.38)	1.16 ± 0.14[a,b] (1.08–1.23)	0.01 (1.14)	1.33 ± 0.19 (1.21–1.46)	0.97 (0.06)
Propulsive (%)	−0.25 ± 0.53 (−0.41 to −0.10)	0.44 ± 0.17[a] (0.24–0.64)	<0.01 (0.85)	0.46 ± 0.13[a] (0.23–0.69)	<0.01 (0.85)
Braking (%)	−0.30 ± 0.20 (−0.39 to −0.21)	−0.05 ± 0.27[a,b] (−0.17 to 0.07)	<0.01 (0.54)	0.16 ± 0.23[a] (0.03–0.29)	<0.01 (0.95)

From Buckley TA, Munkasy BA, Tapia-Lovler TG, Wikstrom EA. Altered Gait Termination Strategies Following a Concussion. Gait and Posture. (epub March 11, 2013); used with permission

[a]Significant difference from the control group

[b]Significant difference from the day-10 time point

Failure to reduce lead limb propulsive forces results in an increased reliance on a single-limb stopping strategy and subsequently longer termination times and a greater number of steps required to control the COM [160]. Therefore, increases in propulsive and braking forces have been identified as sensitive indicators of balance dysfunction and alterations in the cortical control of GT [158, 160, 165]; see also Table 8.1.

In the aftermath of a concussion, GT performance is clearly impaired [171]. Initially, the reduced gait velocity, as previously described, may mask the task performance and, therefore, normalization to gait velocity is required. Further, to more clearly understand the postural strategies utilized, GT variables are compared to performance during standard gait trials. One would naturally expect the penultimate step during GT to have a reduced propulsive force and the terminal step to have an increased braking force relative to normal gait. Conversely, post-concussion the individuals actually increased their propulsive force during the penultimate step and reduced their braking force during the termination step [171]. This highly inefficient pattern of performing GT is suggestive of a central deficit and the selection of an inappropriate motor program to perform the GT task.

While measures of dynamic postural control are insightful in understanding impairments in postural control following a concussion, static stance assessments can also provide additional clarity. Several attempts have been made to quantify static post-concussion postural control utilizing approximate entropy measures but have generally failed to identify differences or noted decreased randomness [63, 172, 173]. The authors speculated that the reduced randomness was secondary to either distorted interactions in the brain or increased co-contraction of the lower extremity musculature; however, these conclusions were drawn from relatively

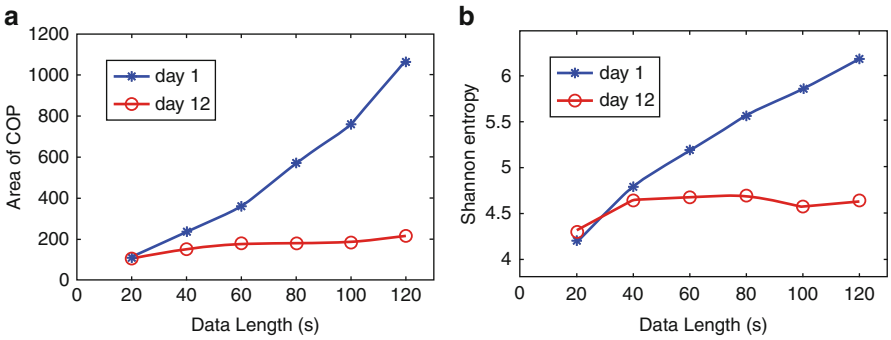

Fig. 8.7 COP area measures and Shannon entropy measures across time. There were no differences between day 1 and day 12 post-injury when the data was sampled for less than 60 s. Gao J, Hu J, Buckley TA, White K, Hass CJ. Shannon and Renyi entropies to classify effects of Mild Traumatic Brain Injury on postural sway. PLoS One. 2011;6(9):e24446 (PLoS One does not require a permission release to republish materials and the article citation is provided above)

short (20–30 s) data sets [173]. When a longer data set (120 s) was investigated, a greater than linear increase in COP area was noted across time suggesting that longer time frames are required for analysis [59]. Specifically, visual analysis clearly shows that at 20–40 s, there would be no differences between groups, but at 120 s clear differences are apparent [59]. Additionally, applying more sophisticated entropy measures, such as Shannon and Renyi measures, successfully identified impairments in postural control [59]. Unfortunately, this study lacks premorbid data, but some early analysis suggests that these trends continue when compared to baseline tests (Fig. 8.7).

Postural Control and Recovery

As discussed, the traditional post-concussion clinical assessment battery suggests that postural control recovers within 3–5 days post-injury [46, 53]. However, this apparent recovery often occurs prior to symptom resolution or achieving baseline values on computerized neuropsychological test batteries [53]. This suggests that while the current clinical battery may be sufficient to identify the presence of a concussion [70], it may lack the sensitivity to identify residual impairments which persist over time. Each of the non-novel tasks which show impairments in the immediate aftermath of a concussion has also demonstrated residual impairments which persist long after recovery based on standard clinical balance tests as well as self-reported symptoms, cognitive testing, and computerized neuropsychological testing.

A healthy young adult has approximately a 5–7 cm displacement of the COP in both the posterior and lateral directions during the APA phase of GI. However, on the day the individuals pass their BESS test (2.9 + 2.8 days post-injury), which clinically suggests their balance has returned to premorbid levels, substantial significant

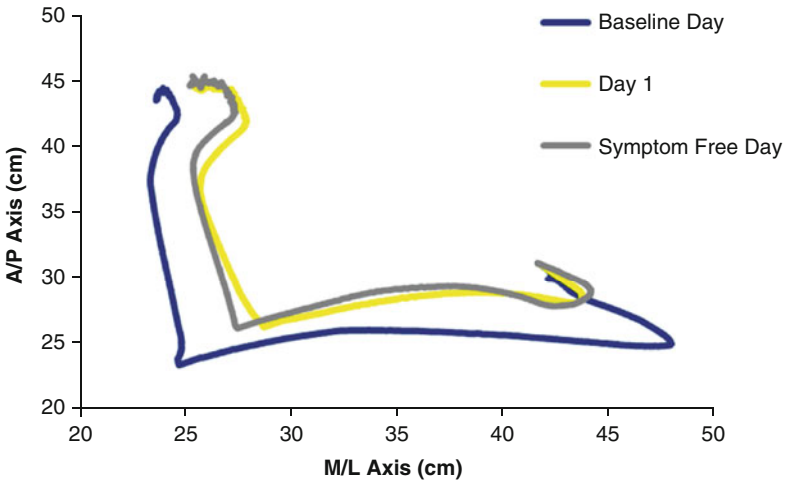

Fig. 8.8 Exemplar changes in COP displacement. The normal healthy adult typically has 5–7 cm of displacement in both the posterior and lateral directions. The posterior APA phase is reduced following a concussion (2.59 + 1.62 cm) and remains reduced at the time (4.8 + 3.1 days) the individual self-reports symptom-free (3.58 + 1.88 cm). Similarly, the lateral displacement is reduced immediately following a concussion (3.43 + 1.92 cm) and remains reduced at the time the individual self-reports symptom-free (4.51 + 2.31 cm)

deficits are noted in both the posterior (PRE: 5.46 + 1.82 cm and BESS: 2.54 + 1.28 cm, $p < 0.001$, $d = 0.68$) and lateral (PRE: 5.55 + 2.13 cm and BESS: 2.93 + 1.63 cm, $p < 0.001$, $d = 0.57$) directions. Similarly, on the day the individual self-reports being symptom-free (4.8 + 3.1 days) there were still impairments in the posterior APA COP displacement (3.58 + 1.88 cm, $p = 0.008$, $d = 0.45$); see Fig. 8.8. Even by 12 days post-injury, the most common time frame for return to participation, the posterior COP displacement was still reduced (4.23 cm) compared to baseline values, although this is within one standard deviation of the baseline. In each case, the standard kinematic stepping characteristics had achieved baseline values thus suggesting the APA phase is a better discriminator of impaired postural control. At no point during the testing protocol, on average, did the posterior COP displacement during the APA phase achieve a value equal to or greater than the baseline value.

The Oregon gait studies interestingly found that most gait kinematic variables returned to baseline values by day 5/6 post-injury and then demonstrated limited impairments at the 2- and 4-week follow-up testing. Conversely, standard gait kinematic stepping characteristics largely returned to baseline values for our subjects prior to return to participation status; however, gait variability assessment remained impaired throughout the recovery process. Specifically, at both self-selected normal pace and at face-paced gait, the variability increased post-injury and either remained flat-lined across time or continued to increase throughout the recovery process. Considering the normal healthy variability is typically below 3 %, the normal self-selected paced step length and step time variability as well as the fast-paced step length, step time, and step width variability all exceeded 3 % at

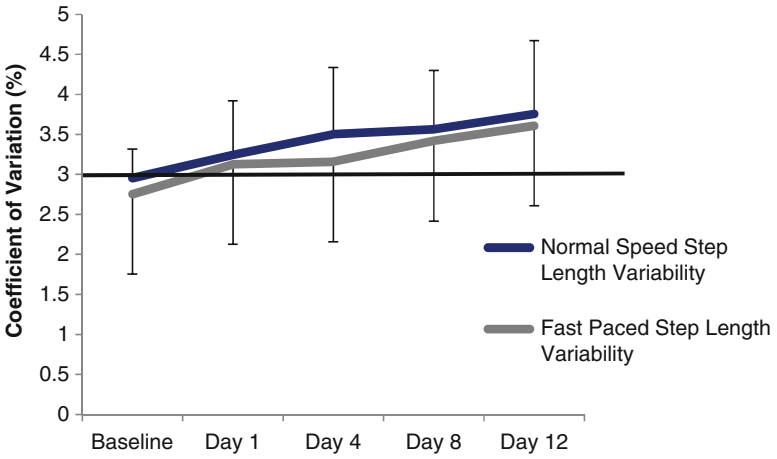

Fig. 8.9 Step length variability. There was a significant increase in step length variability at day 12 post-concussion compared to baseline. Normal healthy adults typically have step length variability at or below 3 %

return to participation (see Fig. 8.9). Similar to previous findings, these results suggest that despite apparent recovery on all clinical assessments, central neurological impairments may still persist.

Consistent with the lingering and persistent deficits noted during gait initiation and gait variability, gait termination remained impaired despite recovery on clinical assessment batteries [171]. By 10 days post-injury, all participants, in this subset, were fully recovered based on BESS, SAC, self-report symptoms, and ImPACT testing; however, clear impairments persisted during GT. Specifically, while gait velocity returned to normal values (actually exceeding the velocity of the control subjects), the post-concussion individuals continued to present with an altered motor strategy for terminating gait. Relative to normal gait trials, the post-concussion individuals continued to have abnormally increased propulsive forces during the penultimate step and decreased braking forces on the termination step—in both cases the motor strategy was actually less efficient than their performance on day 1 post-concussion [171]. While current studies have not elucidated the reasons for this altered movement strategy, it is likely a postural conservative strategy potentially secondary to lingering post-concussion impairments in the prefrontal area [154].

Finally, and consistent with the other reported measures, the impairments in postural control are also noted during static stance trials using sophisticated entropy measures [59]. Once again, despite all participants having achieved baseline values on all clinical measures, impairments in static postural control were still present at least 10 days post-injury; see Fig. 8.10. While this study focused more on the scientific applications of varying entropy metrics, as opposed to clinical application, the underlying mechanisms for the impairments in postural control were not investigated. However, *the results do support the emerging evidence that based on more sophisticated assessment protocols*, *most concussions do not recover within 7–10 days post-injury*.

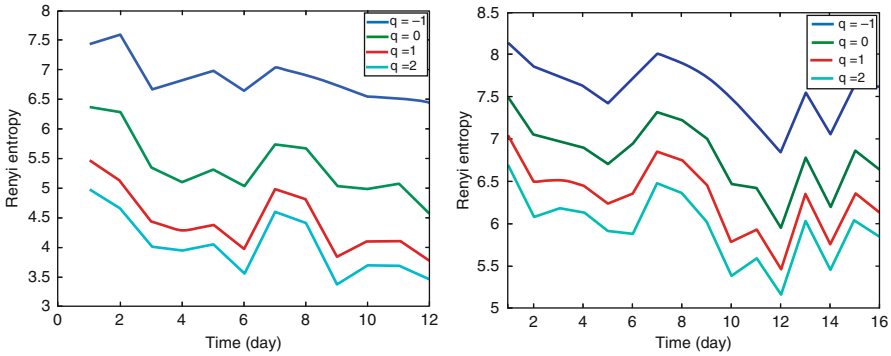

Fig. 8.10 Temporal variations of Renyi entropies for two subjects. Gao J, Hu J, Buckley TA, White K, Hass CJ. Shannon and Renyi entropies to classify effects of Mild Traumatic Brain Injury on postural sway. PLoS One. 2011;6(9):e24446 (PLoS One does not require a permission release to republish materials and the article citation is provided above)

Conclusion

The 4th CIS reports that 80–90 % of concussions in adults resolve within 7–10 days of the injury; however, this is presumably based on earlier epidemiological studies which operationally defined recovery based on self-reported symptoms, SAC, and the BESS [7, 53]. However, a growing body of evidence, utilizing diverse research paradigms including the postural control studies presented herein, has suggested that recovery is prolonged well beyond a couple of weeks [99–101, 104, 111, 174–177]. While the evidence may not fully support the recommendation yet, McKee has proposed a 4- to 6-week recovery period post-concussion to facilitate optimal healing [38]. While it is medically appropriate and ethical to treat concussions conservatively given the emerging association between repeated head injuries and later-life neuropathologies, this is complicated by an already unacceptably low (~50 %) concussion reporting rate [2–4]. Whereas fractures, dislocations, and sprains/strains are often obvious and easy to identify, concussions frequently rely on the individual to self-report the symptoms as severe confusion or disorientation and loss of consciousness are infrequent [32]. Anecdotal evidence suggests that a longer period of time before the student-athlete received medical clearance to return to participation would likely lower the current reporting rate and result in many student-athletes continuing to participate despite having a concussion. Conversely, allowing the injured student-athlete to return prematurely may expose the individual to increased risk of re-injury with potential later-life complications. This paradox will likely continue to challenge sports medicine clinicians for the foreseeable future; however, it is becoming clear that lingering post-concussion impairments persist longer than just 7–10 days.

References

1. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375–8.
2. McCrea M, Hammeke T, Olsen G, Leo P, Guskiewicz K. Unreported concussion in high school football players: implications for prevention. Clin J Sport Med. 2004;14(1):13–7.
3. Llewellyn TA, Burdette GT, Joyner AB, Buckley TA. Concussion reporting rates at the conclusion of an intercollegiate athletic career. Clin J Sport Med. 2013;24(1):76–9.
4. Meehan WP, 3rd, Mannix RC, O'Brien MJ, Collins MW. The prevalence of undiagnosed concussions in athletes. Clin J Sport Med. 2013;23(5):339–342.
5. Kaut KP, DePompei R, Kerr J, Congeni J. Reports of head injury and symptom knowledge among college athletes: implications for assessment and educational intervention. Clin J Sport Med. 2003;13(4):213–21.
6. Players still willing to hide head injuries. Associated Press; 2011 [cited 5 June 2013]. Available from http://espn.go.com/nfl/story/_/id/7388074/nfl-players-say-hiding-concussions-option.
7. McCrory P, Meeuwisse WH, Aubry M, Cantu B, Dvorak J, Echemendia RJ, et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. Br J Sports Med. 2013;47(5):250–8.
8. Davis GA, Iverson GL, Guskiewicz KM, Ptito A, Johnston KM. Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. Br J Sports Med. 2009;43(1):36–45.
9. Kutcher JS, McCrory P, Davis G, Ptito A, Meeuwisse WH, Broglio SP. What evidence exists for new strategies or technologies in the diagnosis of sports concussion and assessment of recovery [Review]? Br J Sports Med. 2013;47(5):299–303.
10. Dashnaw ML, Petraglia AL, Bailes JE. An overview of the basic science of concussion and subconcussion: where we are and where we are going. Neurosurg Focus. 2012;33(6):E5.
11. Henry LC, Tremblay S, Leclerc S, Khiat A, Boulanger Y, Ellemberg D, et al. Metabolic changes in concussed American football players during the acute and chronic post-injury phases. BMC Neurol. 2011;11:105.
12. Unden J, Romner B. Can low serum levels of S100B predict normal CT findings after minor head injury in adults?: an evidence-based review and meta-analysis. J Head Trauma Rehabil. 2010;25(4):228–40.
13. Liu MC, Akinyi L, Scharf D, Mo JX, Larner SF, Muller U, et al. Ubiquitin C-terminal hydrolase-L1 as a biomarker for ischemic and traumatic brain injury in rats. Eur J Neurosci. 2010;31(4):722–32.
14. Papa L, Akinyi L, Liu MC, Pineda JA, Tepas III JJ, Oli MW, et al. Ubiquitin C-terminal hydrolase is a novel biomarker in humans for severe traumatic brain injury. Crit Care Med. 2010;38(1):138–44.
15. Jeter CB, Hergenroeder GW, Hylin MJ, Redell JB, Moore AN, Dash PK. Biomarkers for the diagnosis and prognosis of mild traumatic brain injury/concussion. J Neurotrauma. 2013; 30(8):657–70.
16. Barr WB. Neuropsychological testing of high school athletes—preliminary norms and test-retest indices. Arch Clin Neuropsychol. 2003;18(1):91–101.
17. Broglio SP, Ferrara MS, Macciocchi SN, Baumgartner TA, Elliott R. Test-retest reliability of computerized concussion assessment programs. J Athl Train. 2007;42(4):509–14.
18. Randolph C. Baseline neuropsychological testing in managing sport-related concussion: does it modify risk? Curr Sports Med Rep. 2011;10(1):21–6.
19. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using ImPACT. Am J Sports Med. 2010;38(1):47–53.
20. Register-Mihalik JK, Guskiewicz KM, Mihalik JP, Schmidt JD, Kerr ZY, McCrea MA. Reliable change, sensitivity, and specificity of a multidimensional concussion assessment battery: implications for caution in clinical practice. J Head Trauma Rehabil. 2013;28(4): 274–83.

21. Iverson GL, Lovell MR, Collins MW. Interpreting change on ImPACT following sport concussion. Clin Neuropsychol. 2003;17(4):460–7.
22. Erdal K. Neuropsychological testing for sports-related concussion: how athletes can sandbag their baseline testing without detection. Arch Clin Neuropsychol. 2012;27(5):473–9.
23. Glatts C, Schatz P. "Sandbagging" baseline concussion testing on ImPACT is more difficult than it appears. Arch Clin Neuropsychol. 2012;27(6):621–9.
24. Moser RS, Schatz P, Neidzwski K, Ott SD. Group versus individual administration affects baseline neurocognitive test performance. Am J Sports Med. 2011;39(11):2325–30.
25. Resch J, Driscoll A, McCaffrey N, et al. impact test-retest reliability: reliably unreliable? J Athl Train. 2013;48(4):506–11.
26. Bey T, Ostick B. Second impact syndrome. West J Emerg Med. 2009;10(1):6–10.
27. Cantu RC. Second-impact syndrome. Clin Sports Med. 1998;17(1):37–44.
28. Boden BP, Tacchetti RL, Cantu RC, Knowles SB, Mueller FO. Catastrophic head injuries in high school and college football players. Am J Sports Med. 2007;35(7):1075–81.
29. Cantu RC. Recurrent athletic head injury: risks and when to retire. Clin Sports Med. 2003;22(3):593–603.
30. Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train. 2001; 36(3):228–35.
31. Zemper ED. Two-year prospective study of relative risk of a second cerebral concussion. Am J Phys Med Rehabil. 2003;82(9):653–9.
32. Guskiewicz KM, Weaver NL, Padua DA, Garrett Jr WE. Epidemiology of concussion in collegiate and high school football players. Am J Sports Med. 2000;28(5):643–50.
33. Guskiewicz KM, McCrea M, Marshall SW, Cantu RC, Randolph C, Barr W, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2549–55.
34. Collins MW, Lovell MR, Iverson GL, Cantu RC, Maroon JC, Field M. Cumulative effects of concussion in high school athletes. Neurosurgery. 2002;51(5):1175–9.
35. Eisenberg MA, Andrea J, Meehan W, Mannix R. Time interval between concussions and symptom duration. Pediatrics. 2013;132:8–17.
36. Guskiewicz KM, Marshall SW, Bailes J, McCrea M, Cantu RC, Randolph C, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. Neurosurgery. 2005;57(4):719–26; discussion 719–26.
37. Guskiewicz KM, Marshall SW, Bailes J, McCrea M, Harding Jr HP, Matthews A, et al. Recurrent concussion and risk of depression in retired professional football players. Med Sci Sports Exerc. 2007;39(6):903–9.
38. McKee AC, Cantu RC, Nowinski CJ, Hedley-Whyte ET, Gavett BE, Budson AE, et al. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. J Neuropathol Exp Neurol. 2009;68(7):709–35.
39. Schwartz A. Suicide reveals signs of a disease seen in N.F.L. New York Times, 14 Sept 2010.
40. McKee AC, Gavett BE, Stern RA, Nowinski CJ, Cantu RC, Kowall NW, et al. TDP-43 proteinopathy and motor neuron disease in chronic traumatic encephalopathy. J Neuropathol Exp Neurol. 2010;69(9):918–29.
41. Cavanaugh JT, Guskiewicz KM, Stergiou N. A nonlinear dynamic approach for evaluating postural control: new directions for the management of sport-related cerebral concussion. Sports Med. 2005;35(11):935–50.
42. Winter DA. Human balance and posture control during standing and walking. Gait Posture. 1995;3(4):193–214.
43. Shumway-Cook A, Woollacott MH. Motor control: translating research into clinical practice. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
44. Scholz JP, Schoener G, Hsu WL, Jeka JJ, Horak F, Martin V. Motor equivalent control of the center of mass in response to support surface perturbations. Exp Brain Res. 2007;180(1): 163–79.
45. Highstein SM, Holstein GR. The anatomical and physiological framework for vestibular prostheses. Anat Rec. 2012;295(11):2000–9.

46. Guskiewicz KM. Balance assessment in the management of sport-related concussion. Clin Sports Med. 2011;30(1):89–102.
47. Guskiewicz KM, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. J Athl Train. 2001;36(3):263–73.
48. Guskiewicz KM. Postural stability assessment following concussion: one piece of the puzzle. Clin J Sport Med. 2001;11(3):182–9.
49. Ellemberg D, Henry LC, Macciocchi SN, Guskiewicz KM, Broglio SP. Advances in sport concussion assessment: from behavioral to brain imaging measures [Review]. J Neurotrauma. 2009;26(12):2365–82.
50. Chandrasekhar SS. The assessment of balance and dizziness in the TBI patient. Neurorehabilitation. 2013;32(3):445–54.
51. Lei-Rivera L, Sutera J, Galatioto JA, Hujsak BD, Gurley JM. Special tools for the assessment of balance and dizziness in individuals with mild traumatic brain injury. Neurorehabilitation. 2013;32(3):463–72.
52. Mouzon B, Chaytow H, Crynen G, Bachmeier C, Stewart J, Mullan M, et al. Repetitive mild traumatic brain injury in a mouse model produces learning and memory deficits accompanied by histological changes. J Neurotrauma. 2012;29(18):2761–73.
53. McCrea M, Guskiewicz KM, Marshall SW, Barr W, Randolph C, Cantu RC, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2556–63.
54. Jansen EC, Larsen RE, Olesen MB. Quantitative romberg test—measurement and computer calculation of postural stability. Acta Neurol Scand. 1982;66(1):93–9.
55. Thyssen HH, Brynskov J, Jansen EC, Munsterswendsen J. Normal ranges and reproducibility for the quantitative romberg test. Acta Neurol Scand. 1982;66(1):100–4.
56. Riemann BL, Guskiewicz KM, Shields EW. Relationship between clinical and forceplate measures of postural stability. J Sport Rehabil. 1999;8(2):71–82.
57. Khasnis A, Gokula RM. Romberg's test. J Postgrad Med. 2003;49(2):169–72.
58. Riemann BL, Guskiewicz KM. Assessment of mild head injury using measures of balance and cognition: a case study. J Sport Rehabil. 1997;6(3):283–9.
59. Gao J, Hu J, Buckley T, White K, Hass C. Shannon and Renyi entropies to classify effects of mild traumatic brain injury on postural sway. PLoS One. 2011;6(9):e24446.
60. Slobounov S, Cao C, Sebastianelli W, Slobounov E, Newell K. Residual deficits from concussion as revealed by virtual time-to-contact measures of postural stability. Clin Neurophysiol. 2008;119(2):281–9.
61. Slobounov S, Sebastianelli W, Hallett M. Residual brain dysfunction observed one year post-mild traumatic brain injury: combined EEG and balance study. Clin Neurophysiol. 2012;123(9):1755–61.
62. Slobounov S, Tutwiler R, Sebastianelli W, Slobounov E. Alteration of postural responses to visual field motion in mild traumatic brain injury. Neurosurgery. 2006;59(1):134–9.
63. Cavanaugh JT, Guskiewicz KM, Giuliani C, Marshall S, Mercer V, Stergiou N. Detecting altered postural control after cerebral concussion in athletes with normal postural stability. Br J Sports Med. 2005;39(11):805–11.
64. Mrazik M, Ferrara MS, Peterson CL, Elliott RE, Courson RW, Clanton MD, et al. Injury severity and neuropsychological and balance outcomes of four college athletes. Brain Inj. 2000;14(10):921–31.
65. Riemann BL, Guskiewicz KM. Effects of mild head injury on postural stability as measured through clinical balance testing. J Athl Train. 2000;35(1):19–25.
66. Peterson CL, Ferrara MS, Mrazik M, Piland T, Elliott T. Evaluation of neuropsychological stability following cerebral domain scores and postural concussion in sports. Clin J Sport Med. 2003;13(4):230–7.
67. Cavanaugh JT, Guskiewicz KM, Stergiou N, editors. Detecting altered postural control after cerebral concussion in athletes without postural instability. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.

68. Register-Mihalik JK, Mihalik JP, Guskiewicz KM. Balance deficits after sports-related concussion in individuals reporting posttraumatic headache. Neurosurgery. 2008;63(1):76–80; discussion 80–2.
69. Kelly KA, Jordan EM, Burdette GT, Buckley TA. NCAA Division I athletic trainers concussion management practice patterns. J Athl Train. 2013 [epub ahead of print].
70. Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. Neurosurgery. 2007;60(6):1050–7.
71. Iverson GL, Koehle MS. Normative data for the modified balance error scoring system in adults. Brain Inj. 2013;27(5):596–9.
72. Schneiders AG, Sullivan SJ, Handcock P, Gray A, McCrory PR. Sports concussion assessment: the effect of exercise on dynamic and static balance. Scand J Med Sci Sports. 2012;22(1):85–90.
73. Ferrara MS, McCrea M, Peterson CL, Guskiewicz KM. A survey of practice patterns in concussion assessment and management. J Athl Train. 2001;36(2):145–9.
74. Notebaert AJ, Guskiewicz KM. Current trends in athletic training practice for concussion assessment and management. J Athl Train. 2005;40(4):320–5.
75. Covassin T, Elbin R, Stiller-Ostrowski JL. Current sport-related concussion teaching and clinical practices of sports medicine professionals. J Athl Train. 2009;44(4):400–4.
76. McCrea M, Barr WB, Guskiewicz K, Randolph C, Marshall SW, Cantu R, et al. Standard regression-based methods for measuring recovery after sport-related concussion. J Int Neuropsychol Soc. 2005;11(1):58–69.
77. Mulligan IJ, Boland MA, McIlhenny CV. The balance error scoring system learned response among young adults. Sports Health. 2013;5(1):22–6.
78. Finnoff JT, Peterson VJ, Hollman JH, Smith J. Intrarater and interrater reliability of the balance error scoring system (BESS). PM R. 2009;1(1):50–4.
79. Pagnacco G, Carrick FR, Pascolo PB, Rossi R, Oggero E. Learning effect of standing on foam during posturographic testing preliminary findings. Biomed Sci Instrum. 2012;48:332–9.
80. Hunt TN, Ferrara MS, Bornstein RA, Baumgartner TA. The reliability of the modified balance error scoring system. Clin J Sport Med. 2009;19(6):471–5.
81. McLeod TCV, Perrin DH, Guskiewicz KM, Shultz SJ, Diamond R, Gansneder BM. Serial administration of clinical concussion assessments and learning effects in healthy young athletes. Clin J Sport Med. 2004;14(5):287–95.
82. Valovich TC, Perrin DH, Gansneder BM. Repeat administration elicits a practice effect with the balance error scoring system but not with the standardized assessment of concussion in high school athletes. J Athl Train. 2003;38(1):51–6.
83. Burk JM, Munkasy BA, Joyner AB, Buckley TA. Balance error scoring system performance changes after a competitive athletic season. Clin J Sport Med. 2013;23(4):312–7.
84. Broglio SP, Zhu W, Sopiarz K, Park Y. Generalizability theory analysis of balance error scoring system reliability in healthy young adults. J Athl Train. 2009;44(5):497–502.
85. Susco TM, McLeod TCV, Gansneder BM, Shultz SJ. Balance recovers within 20 minutes after exertion as measured by the balance error scoring system. J Athl Train. 2004;39(3):241–6.
86. Wilkins JC, McLeod TCV, Perrin DH, Gansneder BM. Performance on the balance error scoring system decreases after fatigue. J Athl Train. 2004;39(2):156–61.
87. Fox ZG, Mihalik JP, Blackburn JT, Battaglini CL, Guskiewicz KM. Return of postural control to baseline after anaerobic and aerobic exercise protocols. J Athl Train. 2008;43(5):456–63.
88. Onate JA, Beck BC, Van Lunen BL. On-field testing environment and balance error scoring system performance during preseason screening of healthy collegiate baseball players. J Athl Train. 2007;42(4):446–51.
89. Weber AF, Mihalik JP, Register-Mihalik JK, Mays S, Prentice WE, Guskiewicz K. Dehydration and performance on clinical concussion measures in collegiate wrestlers. J Athl Train. 2013;48(2):153–60.

90. Docherty CL, McLeod TCV, Shultz SJ. Postural control deficits in participants with functional ankle instability as measured by the balance error scoring system. Clin J Sport Med. 2006;16(3):203–8.
91. McLeod TCV, Armstrong T, Miller M, Sauers JL. Balance improvements in female high school basketball players after a 6-week neuromuscular-training program. J Sport Rehabil. 2009;18(4):465–81.
92. Erkmen N, Taskin H, Kaplan T, Sanioglu A. The effect of fatiguing exercise on balance performance as measured by the balance error scoring system. Isokinet Exerc Sci. 2009;17(2):121–7.
93. Broglio SP, Tomporowski PD, Ferrara MS. Balance performance with a cognitive task: a dual-task testing paradigm. Med Sci Sports Exerc. 2005;37(4):689–95.
94. Resch JE, May B, Tomporowski PD, Ferrara MS. Balance performance with a cognitive task: a continuation of the dual-task testing paradigm. J Athl Train. 2011;46(2):170–5.
95. Teel EF, Register-Mihalik JK, Troy Blackburn J, Guskiewicz KM. Balance and cognitive performance during a dual-task: preliminary implications for use in concussion assessment. J Sci Med Sport. 2013;16(3):190–4.
96. Lee H, Sullivan SJ, Schneiders AG. The use of the dual-task paradigm in detecting gait performance deficits following a sports-related concussion: a systematic review and meta-analysis. J Sci Med Sport. 2013;16(1):2–7.
97. Chen JK, Johnston KM, Frey S, Petrides M, Worsley K, Ptito A. Functional abnormalities in symptomatic concussed athletes: an MRI study. Neuroimage. 2004;22(1):68–82.
98. Parker TM, Osternig LR, Lee HJ, van Donkelaar P, Chou LS. The effect of divided attention on gait stability following concussion. Clin Biomech. 2005;20(4):389–95.
99. Parker TM, Osternig LR, van Donkelaar P, Chou L-S. Recovery of cognitive and dynamic motor function following concussion. Br J Sports Med. 2007;41(12):868–73.
100. Catena RD, van Donkelaar P, Chou L-S. The effects of attention capacity on dynamic balance control following concussion. J Neuroeng Rehabil. 2011;8:8.
101. Catena RD, van Donkelaar P, Chou L-S. Different gait tasks distinguish immediate vs. long-term effects of concussion on balance control. J Neuroeng Rehabil. 2009;6:25.
102. Catena RD, van Donkelaar P, Chou L-S. Altered balance control following concussion is better detected with an attention test during gait. Gait Posture. 2007;25(3):406–11.
103. Catena RD, van Donkelaar P, Chou L-S. Cognitive task effects on gait stability following concussion. Exp Brain Res. 2007;176(1):23–31.
104. Catena RD, van Donkelaar P, Halterman CI, Chou L-S. Spatial orientation of attention and obstacle avoidance following concussion. Exp Brain Res. 2009;194(1):67–77.
105. Howell D, Osternig L, Van Donkelaar P, Mayr U, Chou L-S. Effects of concussion on attention and executive function in adolescents. Med Sci Sports Exerc. 2013;45(6):1030–7.
106. Okumura MS, Cooper SL, Ferrara MS, Tomporowski PD. Global switch cost as an index for concussion assessment: reliability and stability. Med Sci Sports Exerc. 2013;45(6):1038–42.
107. McCrory PR, Berkovic SF. Video analysis of acute motor and convulsive manifestations in sport-related concussion. Neurology. 2000;54(7):1488–91.
108. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology. 1997;48(3):575–80.
109. Parker TM, Osternig LR, Van Donkelaar P, Chou LS. Gait stability following concussion. Med Sci Sports Exerc. 2006;38(6):1032–40.
110. Parker TM, Osternig LR, Van Donkelaar P, Chou LS. Balance control during gait in athletes and non-athletes following concussion. Med Eng Phys. 2008;30(8):959–67.
111. Parker TM, Osternig LR, Van Donkelaar P, Chou LS. Gait stability following concussion. Med Sci Sports Exerc. 2006;38(6):1032–40.
112. Van Donkelaar P, Osternig L, Chou LS. Attentional and biomechanical deficits interact after mild traumatic brain injury. Exerc Sport Sci Rev. 2006;34(2):77–82.
113. Bell R, Hall RCW. Mental status examination. Am Fam Physician. 1977;16(5):145–52.
114. Al-Yahya E, Dawes H, Smith L, Dennis A, Howells K, Cockburn J. Cognitive motor interference while walking: a systematic review and meta-analysis. Neurosci Biobehav Rev. 2011;35(3):715–28.

115. Mille ML, Hilliard MJ, Martinez KM, Simuni T, Rogers MW. Acute effects of a lateral postural assist on voluntary step initiation in patients with Parkinson's disease. Mov Disord. 2007;22(1):20–7.
116. Chang HA, Krebs DE. Dynamic balance control in elders: gait initiation assessment as a screening tool. Arch Phys Med Rehabil. 1999;80(5):490–4.
117. Hass CJ, Gregor RJ, Waddell DE, Oliver A, Smith DW, Fleming RP, et al. The influence of Tai Chi training on the center of pressure trajectory during gait initiation in older adults. Arch Phys Med Rehabil. 2004;85(10):1593–8.
118. Mille ML, Johnson ME, Martinez KM, Rogers MW. Age-dependent differences in lateral balance recovery through protective stepping. Clin Biomech. 2005;20(6):607–16.
119. Hass CJ, Waddell DE, Wolf SL, Juncos JL, Gregor RJ. Gait initiation in older adults with postural instability. Clin Biomech. 2008;23(6):743–53.
120. Hass CJ, Waddell DE, Fleming RP, Juncos JL, Gregor RJ. Gait initiation and dynamic balance control in Parkinson's disease. Arch Phys Med Rehabil. 2005;86(11):2172–6.
121. Brunt D, Vanderlinden DW, Behrman AL. The relation between limb loading and control parameters of gait initiation in persons with stroke. Arch Phys Med Rehabil. 1995;76(7): 627–34.
122. Halliday SE, Winter DA, Frank JS, Patla AE, Prince F. The initiation of gait in young, elderly, and Parkinson's disease subjects. Gait Posture. 1998;8(1):8–14.
123. Tokuno CD, Sanderson DJ, Inglis JT, Chua R. Postural and movement adaptations by individuals with a unilateral below-knee amputation during gait initiation. Gait Posture. 2003;18(3):158–69.
124. Polcyn AF, Lipsitz LA, Kerrigan DC, Collins JJ. Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. Arch Phys Med Rehabil. 1998;79(12):1582–9.
125. Vallabhajosula S, Buckley TA, Tillman MD, Hass CJ. Age and Parkinson's disease related kinematic alterations during multi-directional gait initiation. Gait Posture. 2013;37(2): 280–6.
126. Jian Y, Winter DA, Ishac MG, Gilchrist L. Trajectory of the body COG and COP during initiation and termination of gait. Gait Posture. 1993;1(1):9–22.
127. Brunt D, Short M, Trimble M, Liu SM. Control strategies for initiation of human gait are influenced by accuracy constraints. Neurosci Lett. 2000;285(3):228–30.
128. Massion J. Movement, posture and equilibrium—interaction and coordination. Prog Neurobiol. 1992;38(1):35–56.
129. Chang W-H, Tang P-F, Wang Y-H, Lin K-H, Chiu M-J, Chen S-HA. Role of the premotor cortex in leg selection and anticipatory postural adjustments associated with a rapid stepping task in patients with stroke. Gait Posture. 2010;32(4):487–93.
130. Winter DA, Prince F, Frank JS, Powell C, Zabjek KF. Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol. 1996;75(6):2334–43.
131. Hass CJ, Buckley TA, Pitsikoulis C, Barthelemy EJ. Progressive resistance training improves gait initiation in individuals with Parkinson's disease. Gait Posture. 2012;35(4):669–73.
132. Vincent WJ. Statistics in kinesiology. 3rd ed. Champaign, IL: Human Kinetics; 2005.
133. Tate JJ, Milner CE. Real-time kinematic, temporospatial, and kinetic biofeedback during gait retraining in patients: a systematic review. Phys Ther. 2010;90(8):1123–34.
134. Prochazka A, Ellaway P. Sensory systems in the control of movement. Compr Physiol. 2012;2(4):2615–27.
135. Cheron G, Duvinage M, De Saedeleer C, Castermans T, Bengoetxea A, Petieau M, et al. From spinal central pattern generators to cortical network: integrated BCI for walking rehabilitation. Neural Plast. 2012;2012:375148.
136. Roemmich RT, Nocera JR, Vallabhajosula S, Amano S, Naugle KM, Stegemoller EL, et al. Spatiotemporal variability during gait initiation in Parkinson's disease. Gait Posture. 2012;36(3):340–3.
137. Hausdorff JM. Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. Chaos. 2009;19(2):026113.

138. Wittwer JE, Andrews PT, Webster KE, Menz HB. Timing variability during gait initiation is increased in people with Alzheimer's disease compared to controls. Dement Geriatr Cog Disord. 2008;26(3):277–83.
139. Lindemann U, Klenk J, Becker C, Moe-Nilssen R. Assessment of adaptive walking performance. Med Eng Phys. 2013;35(2):217–20.
140. Nakamura T, Meguro K, Sasaki H. Relationship between falls and stride length variability in senile dementia of the Alzheimer type. Gerontology. 1996;42(2):108–13.
141. Hausdorff JM, Zemany L, Peng CK, Goldberger AL. Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children. J Appl Physiol. 1999;86(3): 1040–7.
142. Hausdorff JM, Edelberg HK, Mitchell SL, Goldberg AL, Wei JY. Increased gait unsteadiness in community-dwelling elderly fallers. Arch Phys Med Rehabil. 1997;78(3):278–83.
143. Beauchet O, Annweiler C, Lecordroch Y, Allali G, Dubost V, Herrmann FR, et al. Walking speed-related changes in stride time variability: effects of decreased speed. J Neuroeng Rehabil. 2009;6:32.
144. Frenkel-Toledo S, Giladi N, Peretz C, Herman T, Gruendlinger L, Hausdorff JM. Effect of gait speed on gait rhythmicity in Parkinson's disease: variability of stride time and swing time respond differently. J Neuroeng Rehabil. 2005;2:23.
145. Armieri A, Holmes JD, Spaulding SJ, Jenkins ME, Johnson AM. Dual task performance in a healthy young adult population: results from a symmetric manipulation of task complexity and articulation. Gait Posture. 2009;29(2):346–8.
146. Silsupadol P, Lugade V, Shumway-Cook A, van Donkelaar P, Chou LS, Mayr U, et al. Training-related changes in dual-task walking performance of elderly persons with balance impairment: a double-blind, randomized controlled trial. Gait Posture. 2009;29(4):634–9.
147. Kerr B, Condon SM, McDonald LA. Cognitive spatial processing and the regulation of posture. J Exp Psychol Hum Percept Perform. 1985;11(5):617–22.
148. Ebersbach G, Dimitrijevic MR, Poewe W. Influence of concurrent tasks on gait—a dual-task approach. Percept Mot Skills. 1995;81(1):107–13.
149. Shumway-Cook A, Woollacott M. Attentional demands and postural control: the effect of sensory context. J Gerontol A Biol Sci Med Sci. 2000;55(1):10–6.
150. Plummer-D'Amato P, Altmann LJP, Saracino D, Fox E, Behrman AL, Marsiske M. Interactions between cognitive tasks and gait after stroke: a dual task study. Gait Posture. 2008;27(4):683–8.
151. Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. Gait Posture. 2002;16(1):1–14.
152. Langlois JA, Keyl PM, Guralnik JM, Foley DJ, Marottoli RA, Wallace RB. Characteristics of older pedestrians who have difficulty crossing the street. Am J Public Health. 1997;87(3): 393–7.
153. Hoxie RE, Rubenstein LZ, Hoenig H, Gallagher BR. The older pedestrian. J Am Geriatr Soc. 1994;42(4):444–50.
154. Wang JJ, Wai YY, Weng YH, Ng KK, Huang YZ, Ying LL, et al. Functional MRI in the assessment of cortical activation during gait-related imaginary tasks. J Neural Transm. 2009;116(9):1087–92.
155. Perry SD, Santos LC, Patla AE. Contribution of vision and cutaneous sensation to the control of centre of mass (COM) during gait termination. Brain Res. 2001;913(1):27–34.
156. Sparrow WA, Tirosh O. Gait termination: a review of experimental methods and the effects of ageing and gait pathologies. Gait Posture. 2005;22(4):362–71.
157. Bishop MD, Brunt D, Pathare N, Patel B. The interaction between leading and trailing limbs during stopping in humans. Neurosci Lett. 2002;323(1):1–4.
158. Bishop MD, Brunt D, Kukulka C, Tillman MD, Pathare N. Braking impulse and muscle activation during unplanned gait termination in human subjects with parkinsonism. Neurosci Lett. 2003;348(2):89–92.
159. O'Kane FW, McGibbon CA, Krebs DE. Kinetic analysis of planned gait termination in healthy subjects and patients with balance disorders. Gait Posture. 2003;17(2):170–9.

160. Bishop M, Brunt D, Marjama-Lyons J. Do people with Parkinson's disease change strategy during unplanned gait termination? Neurosci Lett. 2006;397(3):240–4.
161. Menant JC, Steele JR, Menz HB, Munro BJ, Lord SR. Rapid gait termination: effects of age, walking surfaces and footwear characteristics. Gait Posture. 2009;30(1):65–70.
162. Vrieling AH, van Keeken HG, Schoppen T, Otten E, Halbertsma JPK, Hof AL, et al. Gait termination in lower limb amputees. Gait Posture. 2008;27(1):82–90.
163. Vrieling AH, van Keeken HG, Schoppen T, Hof AL, Otten B, Halbertsma JPK, et al. Gait adjustments in obstacle crossing, gait initiation and gait termination after a recent lower limb amputation. Clin Rehabil. 2009;23(7):659–71.
164. Miff SC, Childress DS, Gard SA, Meier MR, Hansen AH. Temporal symmetries during gait initiation and termination in nondisabled ambulators and in people with unilateral transtibial limb loss. J Rehabil Res Dev. 2005;42(2):175–82.
165. Wikstrom EA, Bishop MD, Inamdar AD, Hass CJ. Gait termination control strategies are altered in chronic ankle instability subjects. Med Sci Sports Exerc. 2010;42(1):197–205.
166. Tirosh O, Sparrow WA. Age and walking speed effects on muscle recruitment in gait termination. Gait Posture. 2005;21(3):279–88.
167. Oates AR, Frank JS, Patla AE, VanOoteghem K, Horak FB. Control of dynamic stability during gait termination on a slippery surface in Parkinson's disease. Mov Disord. 2008;23(14): 1977–83.
168. Cameron D, Murphy A, Morris ME, Raghav S, Iansek R. Planned stopping in people with Parkinson. Parkinsonism Relat Disord. 2010;16(3):191–6.
169. Crenna P, Cuong DM, Breniere Y. Motor programmes for the termination of gait in humans: organisation and velocity-dependent adaptation. J Physiol. 2001;537(3):1059–72.
170. Hase K, Stein RB. Analysis of rapid stopping during human walking. J Neurophysiol. 1998;80(1):255–61.
171. Buckley TA, Munkasy BA, Tapia-Lovler TG, Wikstrom EA. Altered gait termination strategies following a concussion. Gait Posture. 2013;38(3):549–51.
172. Sosnoff JJ, Broglio SP, Shin S, Ferrara MS. Previous mild traumatic brain injury and postural-control dynamics. J Athl Train. 2011;46(1):85–91.
173. Cavanaugh JT, Guskiewicz KM, Giuliani C, Marshall S, Mercer VS, Stergiou N. Recovery of postural control after cerebral concussion: new insights using approximate entropy. J Athl Train. 2006;41(3):305–13.
174. Mayers L. Return-to-play criteria after athletic concussion—a need for revision. Arch Neurol. 2008;65(9):1158–61.
175. Dupuis F, Johnston KM, Lavoie M, Lepore F, Lassonde M. Concussions in athletes produce brain dysfunction as revealed by event-related potentials. Neuroreport. 2000;11(18): 4087–92.
176. Gosselin N, Theriault M, Leclerc S, Montplaisir J, Lassonde M. Neurophysiological anomalies in symptomatic and asymptomatic concussed athletes. Neurosurgery. 2006;58(6): 1151–60.
177. De Beaumont L, Brisson B, Lassonde M, Jolicoeur P. Long-term electrophysiological changes in athletes with a history of multiple concussions. Brain Inj. 2007;21(6):631–44.