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Review

Potential Mechanisms of Acute Standing Balance Deficits After Concussions and Subconcussive Head Impacts: A Review

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Abstract—Standing balance deficits are prevalent after concussions and have also been reported after subconcussive head impacts. However, the mechanisms underlying such deficits are not fully understood. The objective of this review is to consolidate evidence linking head impact biomechanics to standing balance deficits. Mechanical energy transferred to the head during impacts may deform neural and sensory components involved in the control of standing balance. From our review of acute balance-related changes, concussions frequently resulted in increased magnitude but reduced complexity of postural sway, while subconcussive studies showed inconsistent outcomes. Although vestibular and visual symptoms are common, potential injury to these sensors and their neural pathways are often neglected in biomechanics analyses. While current evidence implies a link between tissue deformations in deep brain regions including the brainstem and common post-concussion balance-related deficits, this link has not been adequately investigated. Key limitations in current studies include inadequate balance sampling duration, varying test time points, and lack of head impact biomechanics measurements. Future investigations should also employ targeted quantitative methods to probe the sensorimotor and neural components underlying balance control. A deeper understanding of the specific injury mechanisms will inform diagnosis and management of balance deficits after concussions and subconcussive head impact exposure.

Keywords—Concussion, Subconcussive head impacts, Standing balance, Sensorimotor deficits, Brain injury biomechanics, Injury mechanisms.

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INTRODUCTION

In the United States, approximately 1.7 to 3 million sports or recreation-related concussions occur each year with higher incidence in collision sports such as American football and ice hockey.[127] Concussions, most formally referred to as mild traumatic brain injuries (mTBI), usually result in transient neurological symptoms without apparent structural brain changes in standard clinical neuroimaging.[148,188] More recently, subconcussive head impacts, or milder head impacts that do not lead to clinical concussions,[136] have also raised concerns as athletes can sustain up to thousands of subconcussive head impacts per sports season.[46,231]

Individuals with concussions commonly suffer from deficits in sensorimotor function. Around 30% of sports concussion patients exhibit acute balance problems with some sustaining persistent balance deficits beyond 2 weeks.[21,238] Vestibular and vision symptoms may be even more common, with 67–77% of concussion patients reporting dizziness[140] and 69% exhibiting visual disorders[144] that could contribute to balance problems. Thus, balance assessments are commonly integrated into concussion diagnosis protocols. For example, standing balance testing is a major component of the standardized Sports Concussion Assessment Tool (SCAT),[50,63,245] and balance function is a main determinant of return-to-play/work/school decisions after concussions.[110] Although the focus on clinical concussions is warranted, balance deficits have been detected in collegiate football players without clinical concussions,[157,158,165] which motivates

a necessary perspective shift to investigate subconcussive effects.

Concussions are thought to affect brain function through mechanical deformations of brain tissue often resulting from direct impacts to the head.[103,152,175,180] Considerable concussion research has focused on identifying biomechanical parameters to quantify the risk of sustaining clinical concussions; however, few studies have identified mechanistic links between the biomechanical inputs and the highly variable types and severities of trauma outcomes such as balance deficits. Several review articles have questioned the diagnostic sensitivity of sideline balance tests for concussion,[9,21,167] prompting the question of why balance deficits may be clearly observed in some patients, but not others. As such, some have advocated for more comprehensive post-concussion evaluations looking at different aspects of balance function, namely, postural stability,[92] postural complexity,[21] and sensory deficits associated with postural control,[163,172,226] which may elucidate the origins and mechanisms of sensorimotor or neural trauma. Overall, there exists a gap in bridging biomechanical mechanisms of concussion with resulting post-concussion standing balance deficits.

Given the known variability in the biomechanics of concussive and subconcussive head impacts,[18,136,150,171,173,232,244] a key research question is, can we link head impact biomechanics to the presence/absence of balance deficits, or even to the characteristics of these deficits based on the underlying mechanisms of trauma? To begin to answer these questions, our objective in this review is to consolidate the existing understanding of concussion/head impact biomechanics and the human balance control system, and apply this understanding to analyze acute balance changes observed after concussions and subconcussive head impact exposure. One of the fundamental theoretical frameworks we can use to help organize our thoughts is by viewing standing balance as an intricate multi-sensory feedback control circuit, the subcomponents of which may get mechanically disrupted by concussive and subconcussive head impacts producing observable balance deficits (Fig. 1).

In this review, we begin with a discussion on current theories of concussion biomechanics and an overview of the human standing balance system. The working knowledge on the human balance system presented here allows us to examine commonly employed balance testing paradigms, which vary in diagnostic sensitivity and specificity for identifying balance-related sensorimotor deficits. In a subsequent section, we describe a comprehensive review of studies that examined standing balance function in the acute phase after concussive or subconcussive head impacts. Lastly, we synthesize the observations and gaps within our reviewed literature, focusing on mechanistic insights and future recommendations to address standing balance deficits from concussions and head impact exposure.

BIOMECHANICS OF CONCUSSIONS AND SUBCONCUSSIVE HEAD IMPACTS

The biomechanics of head impacts are typically quantified through head/skull kinematics, assuming the skull experiences little deformation and undergoes rigid-body motion. The head/skull accelerations tend to exhibit an impulse response, with short impulse duration on the order of tens of milliseconds.[18,100,244] Peak linear and rotational acceleration values are usually reported as gross representations of head impact severity. Impacts leading to clinical concussions generally produce higher magnitude head kinematics than subconcussive head impacts. According to head impact exposure data from football, soccer, lacrosse, rugby and hockey studies, concussive peak linear and rotational head accelerations were generally between or higher than 50–100 g and 5000–10,000 rad/s^2, respectively,[18,95,150,224,228,232] whereas subconcussive head impacts were typically between or lower than 10–30 g and 1000–3000 rad/s^2, respectively.[136,171,173,244] Despite these general observations, it is important to note that there is no clear skull kinematic threshold for predicting concussion risk.[93] Prior studies have reported concussion cases with lower head kinematics (e.g., 15–30 g and $<$ 1000–3000 rad/s^2)[15,25,39,95] and high kinematics impacts (e.g., $>$ 90 g and $>$ 5000 rad/s^2) that did not result in clinical concussion diagnoses.[146,157,204]

Instead, concussions are likely influenced by factors such as the impact response of the skull, complex material properties, tolerance of brain structures, as well as the vulnerability of each neural and sensory structure within the head. Given the difficulty in identifying simple head kinematic thresholds for injury, some researchers have investigated more direct predictors of injury risk through brain deformation-based biomechanical parameters. Generally, head linear accelerations have been associated with focal brain deformation and pressure gradients across brain tissue,[96,223] whereas head rotational velocities and accelerations have been mainly associated with diffuse brain tissue strains that may disrupt neural connectivity.[79,103,141,175] Agreeing with the observation that injury severities and symptoms are highly variable across concussions and individuals, the directionality,[68,80] dynamics (e.g. impact duration),[124,177] and magnitudes of impacts can influence brain deformation patterns. For example, in primates, coronal head rotation induced more severe diffuse axonal injury compared with

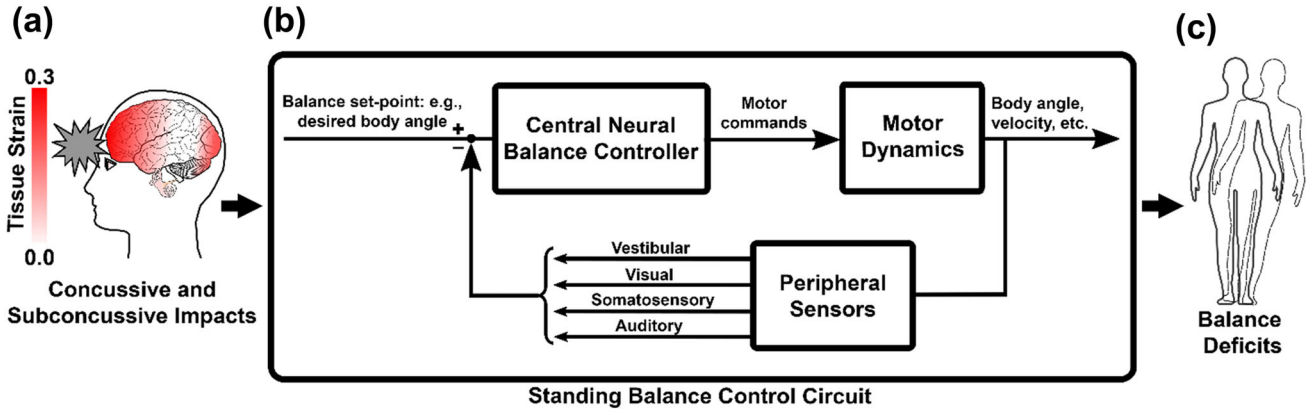

FIGURE 1. Overview of potential mechanisms of head impact-induced standing balance deficits. (a) Concussive and subconcussive head impacts may mechanically deform soft tissue on the head, including sensory and neural components that could be critical for standing balance function. (b) Soft tissue deformation may disrupt one or more components of the close-loop feedback control circuit of human standing balance. This circuit involves central integration of the sensory cues from peripheral sensors (vestibular, visual, somatosensory, and auditory) to calculate the necessary motor commands for the purpose of maintaining the body's balance set-point. (c) Mechanical disruption of this balance control loop could result in global standing balance deficits (e.g., greater postural sway).

head rotations in other planes at similar head kinematics magnitudes.[80] A centripetal theory of concussion also hypothesized that diffuse strains propagate from the surface of the brain towards deeper centers, and the severity of injury correlates with increasing centripetal progression of tissue strains.[176]

Considering the complexity of brain geometry and material properties, investigations of injury mechanisms based on brain loading parameters are supported by finite element model (FEM) analysis. To date, a number of human head FEMs incorporating biofidelic brain geometry and material properties have been developed and used for concussion analyses.[104,113,122,155,220] These FEMs simulate the mechanical behaviour of the brain as a soft deformable body and quantify resultant brain tissue deformations given external head kinematics inputs. Most prior modeling studies have extracted peak brain strain metrics[12,43,180,247] and regional strain metrics in the cerebral cortex,[113,121,180] corpus callosum,[43,121,145,180] basal ganglia,[12,247] thalamus,[12,180,247] brainstem,[180,229,247,248] and cerebellum[43,180] as potential correlates of concussion risk. However, there are limited concussion impact data available for FEM analyses, and so far no clear injury threshold for concussion has been defined using FEM-derived brain deformation metrics.

It should be noted that concussion biomechanics analyses and FEMs typically do not quantify stresses and strains experienced by sensory end organs or cranial nerves, yet these components may also be perturbed by head trauma and could play a role in commonly observed concussion symptoms (e.g., dizziness, visual disruptions). The sensorimotor control of standing balance requires sensing whole-body motion *via* peripheral sensors located in the head as well as synergistic activation of multiple brain regions. Mechanical blows to the head may generate varying degrees of tissue deformation in one or more sensory and neural components contributing to upright stance and potentially lead to balance deficits. In the following section, we briefly review the anatomy, function, and biomechanics of the human standing balance control system.

THE HUMAN STANDING BALANCE SYSTEM

The primary functional goal of standing balance is to keep the unstable body upright. To achieve this objective, the nervous system must coordinate the activity of appendicular and axial muscles to stabilize the body within a base of support defined by the outer contour of both feet. Mechanically, the maintenance of an upright stance is accomplished by actively modulating the body-exerted reaction torque to counterbalance the torque induced by gravity acting on the upright body. Given that the gravitational torque and the reaction torque are not perfectly balanced, the whole-body experiences motion (termed postural oscillations) when upright. From a control perspective, human standing balance can be modeled as a closed-loop feedback control circuit consisting of three major components (Fig. 1b): peripheral sensors, a balance controller consisting of a network of brain structures, and motor dynamics.[73,87,190]

The peripheral sensors mainly include the vestibular, visual, auditory, and somatosensory subsystems. The vestibular end organs encode gravito-inertial and rotational head accelerations *via* the otoliths (utricle

and saccule) and semicircular canals, respectively.[86,116] Vestibular, visual and directional auditory cues are integrated to form self-motion perception.[26,30,97,192,239] In contrast to the head-centered nature of the vestibular, visual, and auditory signals, the somatosensory system relies on mechanoreceptors distributed in muscles, joints, tendons, and skin throughout the body to perceive relative limb position, movement, and contact with external objects (e.g. foot to ground).[114,186,210] Sensory signals encoded by sensory receptors are transduced into neural impulses and transmitted to multiple neural structures including the spinal cord, cerebellum, brainstem, basal ganglia, thalamus, and cortex. Together, these structures are referred to as the central neural balance controller and are responsible for integrating multi-sensory information, form internal representations of body motion, and generate corrective motor commands to remain upright.[109,190,219,236] Interestingly, these structures have been implicated in concussion biomechanics studies, as mentioned in the previous section, and may thus warrant a closer investigation in their functional links to concussion-related balance deficits.

The brainstem houses a critical network of neurons for sensorimotor integration and control with deep interconnections to multiple parts of the brain (e.g., the cerebellum, sensorimotor cortex) and multisensory systems. Critical brainstem structures for the control of standing balance include the vestibular nuclei, gracile and cuneate nuclei, and reticular formation.[135] The convergence of inputs occurring in the brainstem provides it with crucial capacities to organize and modulate reflexes of vestibular origin, anticipatory/reactive postural adjustments, and muscle tone.[135] Recognized as a relay centre, the thalamus contributes to sensorimotor control by relaying signals travelling between different sensory and cortical motor regions.[28,214] The cerebellum receives inputs from somatosensory receptors *via* the spino-cerebellar tracts and exhibits interconnectivity with the brainstem, thalamus, and cerebral cortex. By combining peripheral sensory signals with motor signals, the cerebellum has been proposed to act as a comparator between motor commands and their resulting sensory effects, a process integral to our internal probabilistic representation and control of standing balance.[135] The cerebellum is also thought to contribute to reactive balance control in response to imposed sensory perturbations, motor adaptation/learning, and refinement of motor commands.[135,161,237] The basal ganglia network contains the motor circuitry thought to be important for the selection/inhibition of competing movement patterns as well as reward-oriented motor learning.[89,153,156] Cortical components such as the primary motor cortex, premotor cortex, and supplementary motor cortex work together to modulate voluntary muscular movements (e.g., planning, initiation, control, execution).[5,202] Cortical motor commands are integrated with subcortical centers to activate interneurons and lower motor neurons within the spinal cord, leading to coordinated muscle activation for the production of restorative torques required to maintain an upright posture.[233,234]

The kinematics and dynamics of standing balance can be modeled using an inverted pendulum.[76] For small body angles, anterior/posterior (A/P) balance dynamics can be simplified into an inverted pendulum pivoting around the ankle joint (Fig. 2a). The corresponding dynamical equation $T/\theta = -mgL$ computes the ankle torque (T) required to keep the body balanced at a given ankle angle (θ), where m, g, L refer to the body mass, gravity constant, and the distance from the body's center of mass (CoM) to the ankle joint, respectively.[213] The medial/lateral (M/L) dynamics differ from A/P, requiring a multi-segment inter-connected model (Fig. 2a), where the lower body is modelled as a closed-chain four-bar linkage and the trunk as an additional inverted pendulum connected at the midpoint of the pelvis.[13,87] Consequently, the dynamics of M/L standing balance varies as a function of stance width.[13,87] Due to the intrinsically different biomechanics underlying the control of A/P and M/L balance, their characterization is often decoupled when being assessed.

Researchers often describe postural oscillations through the body's CoM and center of pressure (CoP) displacement parameters. In the context of standing balance, CoM refers to an equivalent virtual point at which the total body mass is concentrated, while CoP represents an equivalent virtual point where the resultant ground reaction force is applied. In a laboratory or in the field, the body CoP is typically estimated using a force plate, while the CoM can be estimated with motion capture, inertial measurement units (IMUs), or derived from force plate data.[29,53,126] Fig. 2b illustrates a bird's-eye view of typical 2-dimensional CoP displacements measured during standing balance, while Fig. 2c further breaks down the CoP displacements into its A/P and M/L components. As shown, random CoP displacements occur while standing upright (typical magnitude $\leq \sim 1$ cm),[233,235] and are predicted to increase in persons suffering from balance deficits after concussions or head impacts (Fig. 2b).

The biomechanics of standing balance reveals that, due to the whole-body inertia, postural oscillations exhibit dominant components at low frequencies. Indeed, over 90% of the power in CoP displacements is contained below 0.5 Hz.[31,61,227] The power of CoM displacements is concentrated in even lower frequen-

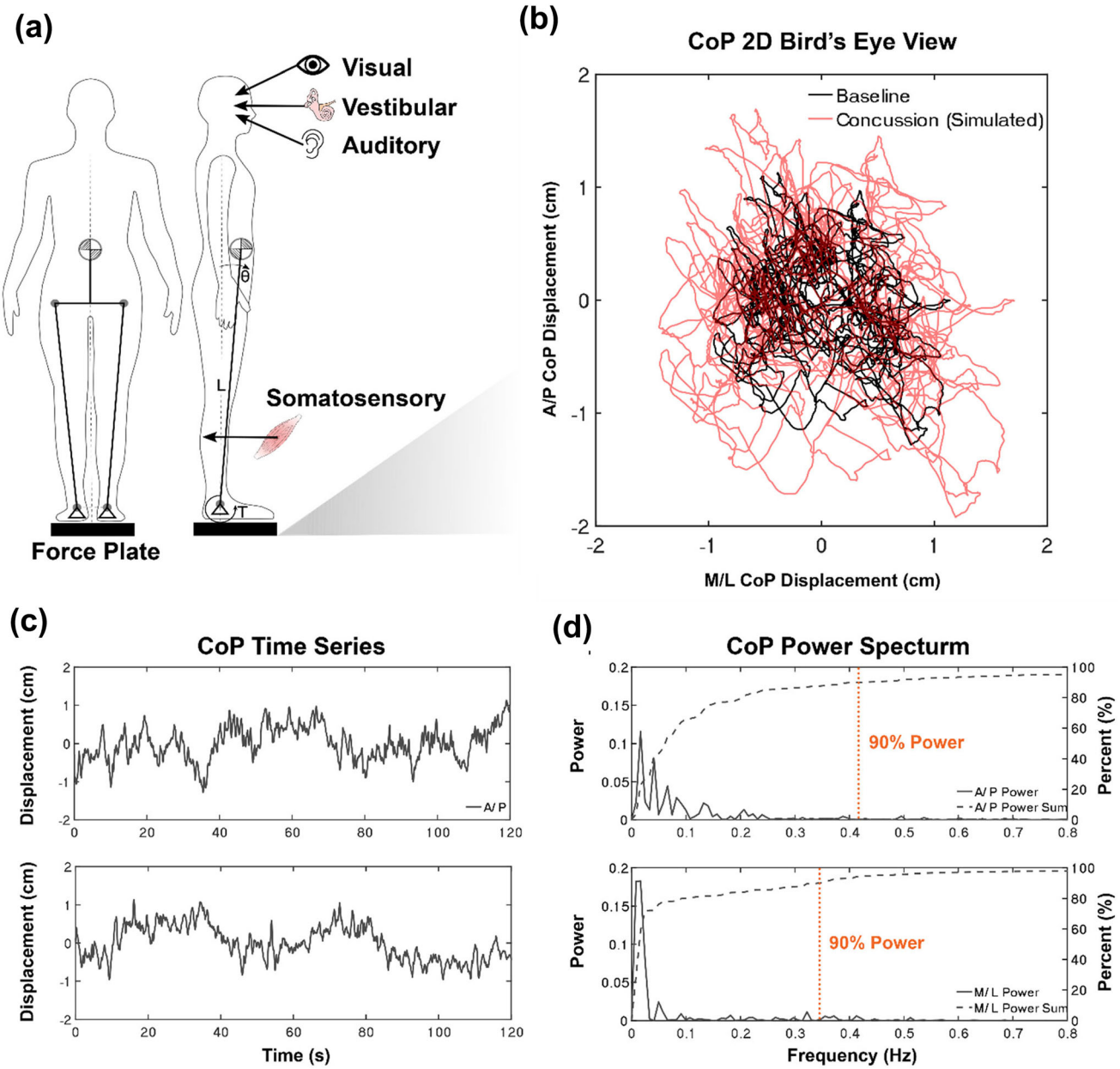

FIGURE 2. Standing balance measurements and frequency characteristics. (a). Biomechanically, anterior-posterior (A/P) standing balance is typically modeled as an inverted pendulum where the ankle torque actuates the body angle, while medial-lateral (M/L) standing balance usually requires a more sophisticated four-bar linkage model. Standing balance behaviors can be quantified by measuring the ankle torque exerted on a force plate to derive the Center of Pressure (CoP). (b). Here we illustrate the bird's eye view of typical 2-D CoP data measured over 120 s. Patients with concussion may exhibit greater postural oscillations (simulated data). The 2-D CoP can be further broken down into A/P and M/L components (c). CoP data have most of the power concentrated in the lower frequency ranges, where 90% of the power is contained 0.5 Hz (d).

cies, with mean frequency at 0.1–0.2 Hz compared to 0.4 Hz for CoP displacements.[178] Consequently, standing balance CoP/CoM need to be recorded over a sufficient duration to characterize the low-frequency characteristics of postural oscillations.[31,55,227] For example, 50 s of data are required to quantify postural oscillations occurring at 0.02 Hz. A prior study found that increasing the sampling duration from 15 to 120 s shifted the relative power of the CoP signals to lower frequencies (mean power frequency shifting from ~0.36 to ~0.15 Hz).[31] Hence, clinical evaluations that rely on brief assessments of standing balance (< 30 s) may lack low-frequency resolution to fully characterize the upright balance behavior. Although most of the postural oscillations estimated from CoP occur at frequencies below 0.5 Hz, the power in the signals may

extend to ~3–5 Hz. The higher frequency CoP oscillations may arise from the net whole-body acceleration, muscular contractions generating small-amplitude postural adjustments or tremor, as well as somatosensory feedback mechanisms.[62,83,132]

STANDING BALANCE-RELATED SENSORIMOTOR ASSESSMENTS FOR CONCUSSION AND SUBCONCUSSIVE HEAD IMPACTS

In this section, we introduce test paradigms designed to assess standing balance or related sensorimotor functions after concussions, starting with common sideline and clinical tests. For sports concussions, the Balance Error Scoring System (BESS) has emerged as a primary balance evaluation,[9,200] where the participant quietly stands with eyes closed during six different conditions varying in standing surface (foam/firm) and stance (single/double/tandem). A shortened modified BESS (mBESS) that only assesses standing balance on the firm surface condition[106] is integrated into the SCAT.[63,245] In each BESS/mBESS test, the participant is asked to stay as still as possible for 20 s while an evaluator counts balance errors. Such balance errors include excessive sway, arm/leg movements, or the participant opening their eyes, which are taken as hallmarks of losing postural stability during quiet standing. Traditional BESS/mBESS testing enables low-cost, rapid sideline balance assessments. Some studies have applied instrumented BESS testing to obtain more objective counts of balance errors.[20,119] However, one recent study has shown poor agreement between human and sensor-based BESS error ratings.[105]

Aside from balance error counts, instrumented standing balance tests utilizing force plate or IMU sensors can also provide further quantification of postural stability metrics, including CoP/CoM displacements and their derivatives such as velocity and acceleration. Time and frequency domain metrics derived from CoP and CoM such as sway path, sway area, range, and standard deviation of the oscillations, as well as mean or median frequency, are typically used to quantify standing balance.[112] These metrics have been frequently applied to assess postural stability in concussion patients. In addition, postural complexity metrics estimate the nonlinear time-varying postural movement pattern. Approximate entropy (ApEn),[184] sample entropy (SampEn),[198] Shannon/Renyi entropy,[19] and multivariate multiscale entropy (MMSE)[1,189] of CoP displacements have been applied, where lower entropy indicates less complex and more regular systems.[42,199] At its core, entropy calculation involves comparing the similarity between data segments throughout the time series as an assessment of regularity (Fig. 3). The entropy calculation process involves multiple post-processing parameters such as the time scale, sequence length, and tolerance (Figs. 3a and 3b). Selections of these parameters influence the resulting entropy value (Fig. 3c).[160] Single-scale entropy only examines the complexity of CoP signals at one fixed frequency, whereas multi-scale entropy provides insights across a range of frequencies.

A common instrumented clinical assessment of standing balance is the Sensory Organization Test (SOT). This test assesses six sensory conditions that vary the visual input (eyes open/eyes closed/sway-referenced) and standing surface (fixed/sway-referenced) while participants stand upright for 20-s trials.[75,169,215] The various sensory conditions assess the individual's ability to use reduced or altered sensory information (visual, vestibular, and somatosensory). The test score for each condition is quantified by comparing the A/P maximum sway angle against a 12.5° sway limit.[36] Visual, vestibular, and somatosensory scores are quantified as the ratio of the score in the altered sensory condition relative to the unaltered sensory condition, and the overall composite score represents the weighted average of all conditions.[16,36,75] The Clinical Test of Sensory Interaction and Balance (CTSIB) and the modified CTSIB (CTSIB-M) are simplified forms of the SOT by replacing the sway-referenced vision and support surface with a visual conflict dome and foam surface[40,211] or fully removing the visual conflict dome condition,[242] respectively.

Beyond standing balance testing, some clinical sensory tests can directly probe sensory dysfunction relevant to standing balance. For instance, the vestibulo-ocular reflex (VOR) can be assessed by computing the gain of the eye compensatory response to head movements, evaluating the vestibular contributions to extraocular muscles for stabilizing the visual image on the retina.[38,203,218,249] The VOR can be assessed for horizontal and vertical linear motion as well as all planes of rotational motion, but clinical VOR testing typically involves imposed head rotation stimuli (e.g., head thrust test[149,240]), active head rotation stimuli (e.g., horizontal/vertical VOR test[4,164]), or both (dynamic visual acuity test[99,128,249]). When direct ocular measurements are not possible, the vestibulo-ocular function can be assessed using the vestibular/ocular motor screening (VOMS) test.[4,164,246] VOMS involves participants performing (1) saccade, (2) convergence, (3) smooth pursuit, (4) horizontal/vertical VOR, and (5) visuomotor sensitivity (VMS) and subjectively rating provoked symptoms such as headache, dizziness, nausea, and fogginess on a scale that ranges from 1 to 10.[164] Other stand-alone visual/oculomotor tests for

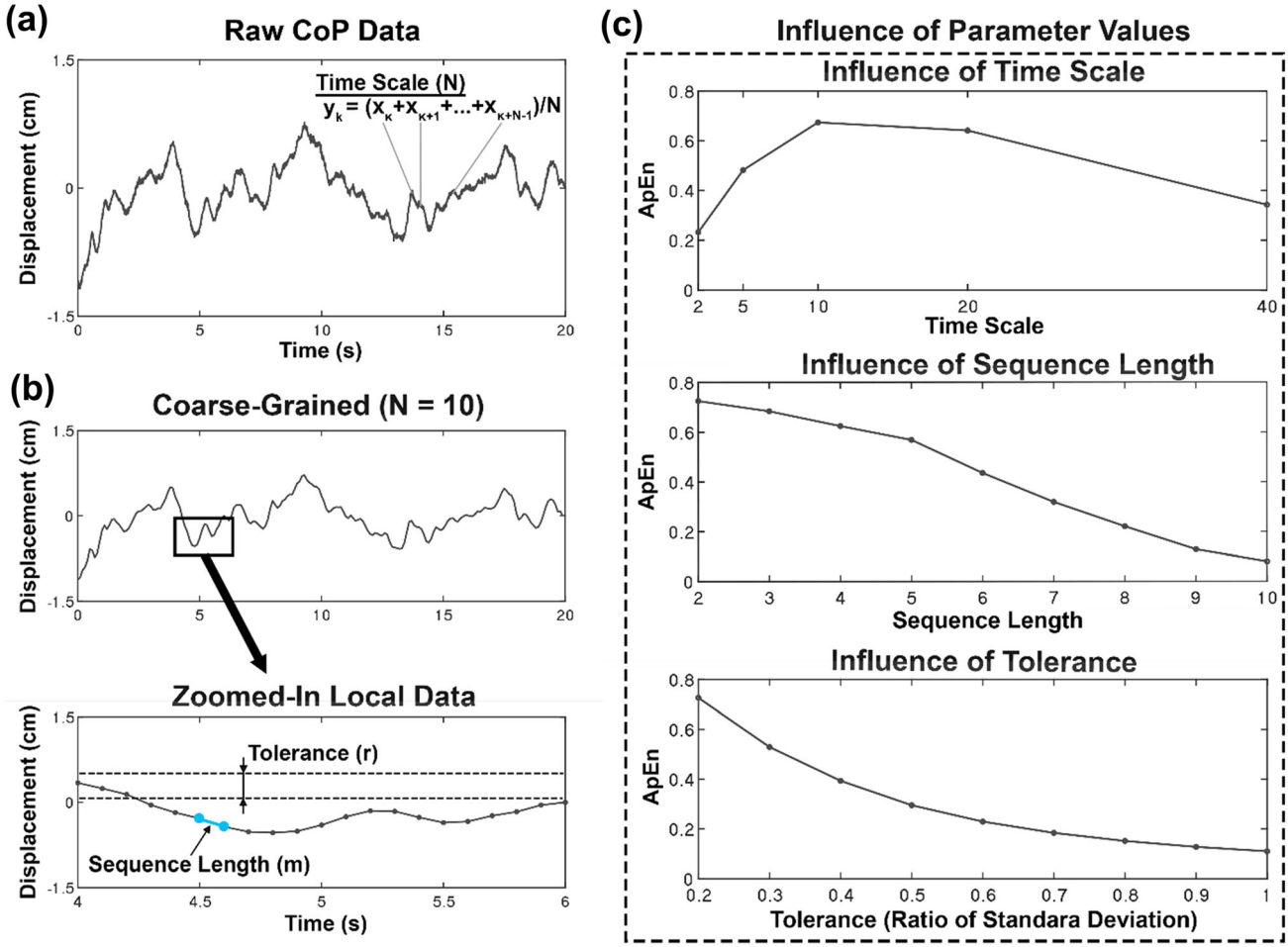

FIGURE 3. CoP Entropy calculation and the influences of hyper-parameters. (a). Raw CoP displacement time series data is typically coarse-grained by the time scale parameter, which is a part of multi-scale analysis to examine varying time scales. Prior single-scale concussion studies usually set the time scale to 10 for analysis. (b). The Approximate Entropy (ApEn), the most used entropy metric for single-scale analysis in prior concussion entropy studies, can then be computed based on the sequence length (*m*) and tolerance (*r*) where m determines how many data points are contained in one data segment for segment-wise comparison and r is the tolerance threshold for assessing the similarity (typically quantified as a distance function, e.g., the maximum distance) between every two data segments. m and r are usually selected as 2 and 20% of the time series' standard deviation in prior concussion studies, respectively. (c). The selection of time scale, sequence length, and tolerance may all influence the final CoP displacement ApEn value to vary between ~ 0.2 and ~ 0.8.

potential concussion screening tests include the King–Devick (K–D) test[77,129] and ocular near point of convergence (NPC).[115,181]

In addition to clinical or sideline assessment of the vestibulo-ocular function, in-laboratory evaluations of eye movements during saccades and smooth pursuit can quantify gaze positional and timing errors post-concussion.[142,168] Also, artificial activation of the vestibular system using currents applied in a binaural bipolar configuration over the mastoid bone (i.e., galvanic vestibular stimulation—GVS or electrical vestibular stimulation—EVS) induces well-defined VOR, perceptual, postural and navigation responses in participants.[37,71,117,154,182,196] The percutaneous current modifies the neural firing pattern of vestibular primary afferents and consequently provides an isolated vestibular error signal with minimal activation of other sensory signals involved in standing balance control.[85,118,125] Researchers have recently used this method to assess standing balance function after subconcussive head impacts.[22,108]

ACUTE STANDING BALANCE-RELATED SENSORIMOTOR CHANGES AFTER CONCUSSIONS AND SUBCONCUSSIVE HEAD IMPACTS

A comprehensive literature search of peer-reviewed English journal articles was performed on MedLine/PubMed, Compendex Engineering Village, and Google Scholar using search terms "(balance OR vestibular

OR visual OR ocular OR sensorimotor) AND (concuss* OR subconcuss* OR mTBI)", with the Google Scholar search limited to the first 100 articles. Only articles observing acute standing balance-related changes within 4 weeks (the majority within 2 weeks) after concussions or subconcussive head impacts were included. We excluded studies with unspecified testing timeframe and studies that only examined persistent sensorimotor deficits (beyond 4 weeks post-concussion/impacts) with potential secondary physiological or biomechanical contributors. In addition, we excluded studies that focused on instrument validation, walking and gait analysis, blast-induced injury, neuroimaging assessments, blood biomarkers, biochemical/cellular analysis, and non-human models. The primary search was conducted within February 2020, along with a secondary search of the bibliographies of the selected papers. In total, our search led to 43 original research articles (concussion: 34; subconcussive head impacts: 9).

Acute Standing Balance-Related Sensorimotor Changes After Concussions

Patients with concussions have consistently shown standing balance deficits in instrumented (e.g. IMU, force plate) tests acutely after injury, followed by recovery within 14–30 days (Table 1). IMU-based BESS/CTSIB tests have revealed acute increases in postural sway velocity, acceleration, and area.[7,82,120,179] Force plate-based standing balance tests also showed higher CoP area/displacement acutely after concussion.[58,166,185,197,212,241] In addition, researchers assessing standing balance with the SOT identified an acute decline in the composite equilibrium score on day 1 in the concussion group compared to the control group, and such group differences usually resolved within the next 1–9 days.[38,45,91,92,94,95,183,193] Furthermore, researchers using SOT sensory analyses have typically demonstrated reductions in vestibular and visual ratios in concussion patients on day 1 of injury, followed by a gradual return to baseline in the next few days.[45,91,92,183] Despite the acute balance impairment-and-recovery pattern commonly observed in the first 2 weeks after injury, abnormal balance performances were still detected beyond the 2-week time frame when participants were asked to perform a cognitive task, suggesting persistent subtle balance deficits that may be exposed with increased cognitive load.[59]

While postural sway generally increases post-concussion, the complexity metrics point toward more regular and predictable postural movements that may persist longer than postural stability changes (Table 1). Cavanaugh *et al.* reported smaller A/P and M/L CoP displacement ApEn within 48 h of concussion compared to baseline.[34,35] These authors further showed that ApEn for M/L CoP displacement declined for a longer duration post-concussion (up to 96 h vs 48 h) and more consistently across all SOT conditions compared to the ApEn estimated for A/P CoP displacement. Other researchers identified decreases in entropy metrics up to 10 days (CoP area Renyi entropy)[78] and 6 weeks (CoP MMSE SampEn)[69] post-concussion. Despite showing lower CoP MMSE SampEn at 3, 21, and 90 days after concussions, Purkayastha *et al.* observed that the A/P CoP range/variability recovered by day 21.[189] From a methodological perspective, these studies have generally digitized CoP at 100 Hz and selected a time scale parameter of 10 for single-scale or 1–20 for multi-scale analysis with a sequence length of 2.[34,35,69,189] Consequently, the frequency range of postural oscillations considered in the CoP entropy estimates was typically $>$ 2.5–5 Hz. These approaches would not capture complexity changes of the dominant low-frequency ($<$ 0.5 Hz) component of standing balance movement.

Sensory dysfunction was frequently identified by symptom provocation from vestibular and visual testing post-concussion (Table 2). Clinical symptoms were identified in 16.8–53.6% of the participants with concussions in various tests of VOMS within 14 days post-injury.[4] Another study found that VOMS symptom score substantially increased from 0.1 on average at baseline to $>$ 2 for the saccade, smooth pursuit, and convergence tests, and to $>$ 3 for the VOR and VMS tests within 21 days post-concussion.[164] Two clinical chart reviews reported around 30% of concussed children displayed vestibular-ocular dysfunction (VOD) within 30 days of injury.[66,67] Other studies found significant increases in NPC distance,[27,64,164] VOR cancellation gains,[38] subjective visual variances,[38] and K–D test completion times with higher error counts[77,129,130] acutely following concussions.

Although most researchers have observed acute standing balance and vestibular/visual deficits post-concussion, some have reported conflicting results using common sideline/clinical tests. Traditional human-based BESS/mBESS error scores have shown inconsistent changes post-concussion: in our review, there were 6 studies reporting human-rated BESS/mBESS scores; 2 of the studies found significant error score changes post-concussion,[147,222] and the other 4 studies did not[29,71,149,240]. However, these 4 studies found significant changes in sway acceleration measured by IMU[29,71] and in select visual or vestibular SOT test conditions.[149,240] It is also worth noting that some studies[149,240] combined acute ($\leq$ 10–14 days) and chronic ($\leq$ 90–120 days) patients in the concussion group, where it be difficult to isolate acute and chronic

TABLE 1. Acute global standing balance deficits after concussions

Article	Patients (sex, avg. age)	Post-injury test time	Protocol (duration, comparison method)	Key observations
Postural stability				
McCrea et al.[147]	94 football players (M, 20.0 years)	0, 3 h, 1, 2, 3, 5, 7, 90 days	BESS (20 s, baseline)	Error score ↑ at 0 h and mostly recovered by day 5
Teel et al.[222]	352 high school/college athletes (NS, NS)	0, 3 h, 1–7, 90 days	BESS (30 s, baseline)	Error score ↑ at 0 h and returned by day 4
Parrington et al.[179]	23 athletes (F/M, 20.1 years)	≤ 24–48 h	Instrumented BESS (30 s, controls)	Error score ↔ and sway (mm/s^2) ↑
King et al.[120]	52 athletes (F/M, 20.4 years)	2.2 days (avg.)	Instrumented mBESS (30 s, controls)	error score ↔, while sway area, ML RMS acc, acc range, power ↑ (double stance)
Baracks et al.[7]	48 athletes (F/M, 20.6 years)	≤ 72 h	Instrumented mBESS (30 s, controls)	RMS sway acceleration and 95% sway area ↑
Gera et al.[82]	38 athletes (NS, 20.6 years)	2–3 days	Instrumented CTSIB-M (20 s, controls)	Postural sway area measured by IMU ↑ in 3/4 conditions
Guskiewicz et al.[95]	13 football players (M, 20.2 years)	≤ 24–48 h	SOT (20 s, baseline)	Composite score ↓ in 8/13 patients; no relationship identified between head kinematics and composite score changes
Guskiewicz et al.[94]	36 athletes (F/M, 19.5 years)	1, 3, 5 days	SOT (20 s, baseline/controls)	Composite score ↓ on day 1 and did not fully return by day 5
Guskiewicz et al.[91]	11 athletes (F/M, 18.6 years)	1, 3, 5, 10 days	SOT (20 s, controls)	Composite score ↓ on day 1 and ↔ by day 10
Peterson et al.[183]	28 athletes (F/M, 20.2 years)	1, 2, 3, 10 days	SOT (20 s, baseline/controls)	Composite score ↓ from day 1–10 and returned by day 10
Register-Mihalik et al.[193]	108 athletes (F/M, 18.8 years)	1.44 days (avg.)	SOT (20 s, baseline/controls)	Composite score ↓ in the headache group
Christy et al.[38]	28 athletes (F/M, 20.7 years)	≤ 72 h or ≤ 2 weeks	SOT (20 s, controls)	Composite/individual score ↓
Resch et al.[195]	40 athletes (F/M, 20.2 years)	≤ 24 h	SOT (20 s, baseline/controls)	Composite score ↔
Cripps et al.[45]	7 athletes (NS, 17.1 years)	24–48 h, 10 day	SOT, CTSIB-M (20 s, baseline/controls)	Composite score ↓ on day 1 than 10 and ↑ with visual distractions than without; CTSIB-M sway ↔
McDevitt et al.[149]	12 athletes (F/M, 20.5 years)	≤ 4–90 days (6 acute: 2–10 days)	SOT, BESS (20 s, controls)	Scores in SOT condition 3/4 ↓ while BESS error score ↔
Wright et al.[240]	12 athletes (F/M, 21.7 years)	≤ 10–120 days (avg. 36 days)	EO/EC, foam/firm surface, with/without rotating visual scene, BESS (20 s, controls)	CoP area and variability ↑ especially for rotating visual scene/foam while CoP velocity and BESS error score ↔
Wright et al.[241]	11 athletes (F/M, 20.4 years)	2–90 days (6 subacute: > 10 days)	EO/EC, foam/firm surface, with/without rotating visual scene, SOT (20 s, controls)	CoP area ↑ especially for rotating visual scene/-foam, while only the condition 3 score in SOT ↓
Rhine et al.[197]	13 children (F/M, 12.8 years)	≤ 6 h	EO/EC double/single limb (60 s/30 s, controls)	CoP displacement ↑ for double-limb EO, and CoP range ↑ for double-limb EO/EC
Dierijck et al.[58]	12 athletes (M, 19.0 years)	72 h, 2 weeks, and 1 month	EO/EC (60 s, controls/72 h)	A/P RMS CoP displacement ↑ at 2 weeks than 72 hrs, ↔ compared to controls, ↔ at 1 month
Powers et al.[185]	9 football players (M, 20.2 years)	1–13 days.	EO/EC (60 s, controls)	CoP RMS displacement and velocity ↑
Munce et al.[166]	1 football player (M, 12 years)	1 h, 2, 6, 12, 20, 27 days	EO/EC with/without cognitive tasks (20 s, baseline)	CoP area ↑ at 1 h, peaked on day 6, and returned by day 27
Dorman et al.[59]	18 children (F/M, 16.6 years)	1st visit ≤ 10 days (4 visits)	EO/EC with/without dual cognitive task (20 s, controls)	CoP area remained different in the 2nd visit but only with the dual cognitive task
Hides et al.[101]	54 rugby players (NS, 24.4 years)	≤ 3–5 days	EC: narrow/single leg/tandem stance on foam/firm surface (20 s, controls)	Sway velocity ↓, muscle cross sectional area and contraction ↑, cervical proprioception score ↔
Slobounov et al.[212]	48 athletes (M, 20.9 years)	3, 10, 30 days	With/without visual variation (30 s, baseline)	CoP area ↔ without visual variation, while ↑ on day 10 and ↓ on day 30 with visual variation

TABLE 1. continued

Article	Patients (sex, avg. age)	Post-injury test time	Protocol (duration, comparison method)	Key observations
Postural complexity				
Gao et al.[78]	10 collegiate athletes (NS, NS)	Daily test (avg. 11.8 days)	Normal (120 s, day 1)	Renyi entropy ↓ nonmonotonically from day 1 to recovery
Fino et al.[69]	6 college athletes (NS, NS)	Weekly test (1–6 weeks)	Normal (120 s, controls)	CoP MMSE SampEn ↓ except week 5 and was more consistent than other entropy metrics
Purkayastha et al.[189]	31 athletes (F/M, 21.0 years)	3, 21, 90 days	EO/EC (60 s, controls)	CoP MMSE SampEn ↓ until day 90, while A/P CoP range and variability ↑ on day 3 and returned by day 21
Cavanaugh et al.[34]	27 athletes (F/M, 19.5 years)	≤ 48 h	SOT (20 s, baseline)	A/P and M/L ApEn ↓ ≤ 48 h
Cavanaugh et al.[35]	29 athletes (F/M, 19.1 years)	48, 48–96 h	SOT (20 s, baseline)	A/P and M/L ApEn ↓ ≤ 48 h; M/L ApEn remained ↓ at 48–96 h in those whose instability recovered

↑ denotes measurements became higher, ↓ lower, ↔ no significant differences; *EO* eyes-open, *EC* eyes closed, *F* female participants, *M* male participants, *NS* not specified.

TABLE 2. Acute visual/vestibular sensory deficits after concussions

Article	Patients (sex, avg. age)	Post-injury test time	Key observations
Sensory Organization Test (SOT) sensory analysis			
Guskiewicz et al.[94]	36 athletes (F/M, 19.5 years)	1, 3, 5 days	Vestibular and visual ratio ↓ on day 1 than baseline/controls and did not fully return by day 5
Guskiewicz et al.[91]	11 athletes (F/M, 18. years)	1, 3, 5, 10 days	Vestibular and visual ratio ↓ than controls on day 1 and become ↔ by day 10
Peterson et al.[183]	28 athletes (F/M, 20.2 years)	1, 2, 3, 10 days	Vestibular ratio ↓ on days 1, 2 than controls and gradually returned by day 10
Register-Mihalik et al.[193]	108 athletes (F/M, 18.8 years)	1.44 days (avg.)	Vestibular and visual ratio ↓ than baseline/controls in the headache group
Resch et al.[195]	40 athletes (F/M, 20.2 years)	≤ 24 h	Vestibular ratio ↑ while visual ratio ↔ compared to baseline/controls
McDevitt et al.[149]	12 athletes (F/M, 20. years)	≤ 90 day (6 acute: 2–10 days)	Visual ratio ↓ while vestibular ratio ↔ compared to controls
Cripps et al.[45]	7 athletes (NS, 17.1 years)	1, 10 days	Vestibular ratio ↓ on day 1 than controls; sensory ratios ↑ in visual-distraction conditions
Vestibular/oculomotor motor screening (VOMS)			
Mucha et al.[164]	64 athletes (F/M, 13.9 years)	≤ 21 days	VOMS symptom scores ↑ than controls
Anzalone et al.[4]	167 sports patients (N/A, 15.0 years)	≤ 14 days	36.8–53.6% provocation rate for saccade, smooth pursuit, and VOR; 16.8% for convergence
Elbin et al.[183]	63 athletes (F/M, 15. years)	1–7, 8–14 days	NPC distance and VOMS symptom scores ↑ in days 1–7 and ↓ in days 8–14 than baseline
Other clinical VOR and visual/oculomotor tests			
Ellis et al.[66]	77 sports patients (F/M, 13.7 years)	≤ 30 days	29% of patients met the clinical VOD criteria
Ellis et al.[67]	306 sports patients (F/M, 13.9 years)	≤ 30 days	30% of patients met the clinical VOD criteria
McDevitt et al.[149]	12 athletes (F/M, 20. years)	≤ 90 days (6 acute: 2–10 days)	Horizontal VOR and optokinetic stimulation symptom score ↑, while horizontal eye saccade, NPC distance, K–D, and dynamic visual acuity line difference ↔
Wright et al.[240]	12 athletes (F/M, 21.7 years)	10–120 days (avg. 36 days)	NPC distance, symptom scores of horizontal VOR, optokinetic stimulation, horizontal eye saccade, and dynamic visual acuity line difference ↑ than controls while K–D score ↔
Hides et al.[187]	54 rugby players (NS 24.4 years)	≤ 3–5 days	Asymmetry (%) of the video head impulse test ↔ compared to controls
Christy et al.[38]	28 athletes (F/M, 20.7 years)	≤ 72 h	VOR gains ↑ than baseline

↑ denotes measurements became higher, ↓ lower, ↔ no significant differences; *F* female participants, *M* male participants, *NS* not specified.

effects. Another study did not find significant differences in CTSIB-M postural sway between day 1 and 10 post-concussion or between the concussion and control group.[45] Interestingly, these authors found improved SOT scores in concussion patients when visual perturbation was applied. Similarly, three other studies found improvements in postural stability[34,101] and the SOT vestibular ratio score[195] compared to baseline in concussion patients. The authors of one study argued that the increased stability, instead of indicating an improvement, may result from a decreased willingness to use body sway for sensory stimulation to gather information about the environment.[101] Aside from this hypothesis, significant learning effects have also been identified in the SOT.[243] In general, the evaluation of standing balance over short durations (primarily 20 s)[21,78] could also contribute to inconsistent findings.

Few studies attempted to relate head impact biomechanics information with acute standing balance deficits. Guskiewicz *et al*. used instrumented helmets to measure head linear and rotational accelerations during concussive impacts, and did not find any significant association between impact location/magnitude and post-concussion (< 48 h) SOT composite score change from baseline in 13 concussed college football players.[95] J. Dierijck *et al*. applied skin-mounted head impact sensors and found no clear correlations between head impact data and balance changes (estimated from CoP metrics) in 12 concussed college athletes.[58] The authors of these studies have suggested that the inability to identify potential links between mechanical head inputs and balance deficits may be due to the complex and uncontrolled characteristics of concussive impacts (e.g., varying frequency, magnitude, and location), the time elapsed between the injury and assessment, and other interfering factors such as one's concussion history and repeated subconcussive exposure.

Acute Standing Balance-Related Sensorimotor Changes After Subconcussive Head Impacts

Although subconcussive head impacts are considered milder than concussive head impacts, they have also been associated with subtle changes in standing balance and vestibular/visual function (Table 3)—albeit less consistently. Indeed, only 44% (4/9) of the subconcussive impact studies we reviewed showed at least one statistically significant change in standing balance or related sensorimotor assessments, compared to 97.1% (33/34) of the concussion studies. Soccer heading is commonly used as a controlled head impact model in laboratory settings. A typical header paradigm is to have participants perform 10–20 soccer headers at 1–2 min intervals[24,98,108,173] using a sports ball launcher to reproduce similar head kinematics as soccer games/practices (10–30 g in linear accelerations).[6,170] Launching soccer balls 40 feet away from the participants, Caccese *et al*. reported elevated CoP velocities immediately after headers compared to both baseline and controls; however, there were no significant changes in CoP area or A/P and M/L CoP ApEn.[24] At a relatively short ball launch distance of 22 feet, potentially corresponding to higher ball velocity upon head impact, Haran *et al*. identified significant increases in A/P CoM displacement (at 1 and 24 h) and M/L displacement (at 24 h) in the heading group compared to the control group, with a subsequent return to baseline at 48 h for both A/P and M/L displacements.[98] In the 2 h following soccer headers, participants exhibited lower responses (smaller trunk angle, leg angle, and CoM movement) to isolated vestibular perturbations (GVS) during standing balance.[108] Finally, Nowak *et al*. reported longer NPC distances and increased KD time/errors at 0, 2, and 24 h post-headers.[173]

On the other hand, a number of soccer heading studies that varied the number of headers, ball launching parameters, or testing time frames did not find significant changes in standing balance parameters such as the CoP area/velocity or SOT scores.[17,139,206] It should be noted that the soccer heading studies we reviewed did not measure the head impact kinematics to allow for comparison of impact mechanics or severity across studies. In a field study where instrumented helmets were applied, Mccaffrey *et al*. did not find significant changes in SOT scores following either a low (< 60 g) or high (> 90 g) head impact in college football players at the end of a practice or game.[146] Lastly, Caccese *et al*. perturbed the visual (virtual reality), vestibular (GVS) and Achilles tendon (vibration) of standing participants but did not observe significant changes in weightings between the sensory systems after 10 headers.[22] A notable distinction with previous work from the same group[108] was that here they did not blindfold participants or ask them to stand on a foam surface.

DISCUSSION

Head impacts can lead to mechanical loading of sensory and neural components situated in the head, which may adversely affect multiple structures contributing to the sensorimotor control of standing balance. In this review, we have identified consistent acute standing balance change patterns such as increased postural oscillation/sway and reduced postural com-

TABLE 3. Acute standing balance-related sensorimotor changes after subconcussive head impacts

Article	Participants (sex, avg. years)	Intervention	Test time	Protocol (duration, comparison method)	Key observations
Postural stability and complexity					
Schmitt et al.[206]	31 soccer players (F: 19.4 years, M: 20.7 years)	18 headers in 40 min (30 mph, 82 ft)	0, 24 h	EO/EC (20 s, controls)	CoP area and velocity ↔
Caccesse et al.[24]	160 soccer players (F/M, 12–24 years)	12 headers in 12 min (25 mph, 40 ft)	0 h	EO/EC (120 s, baseline/controls)	CoP sway velocity ↑ while CoP area, A/P and M/L ApEn ↔
Mangus et al.[139]	10 soccer players (F/M, 21.4 years)	20 balls kicked by a teammate (82 ft)	0 h	SOT (20 s, baseline)	Composite score ↔
Mccaffrey et al.[146]	43 football players (M, 20.7 years)	< 60 g or > 90 g	≤ 24 h	SOT (20 s, baseline)	Composite score ↔
Broglio et al.[17]	40 soccer players (F/M, 19. years)	20 headers in 20 mi (55 mph, 80 ft)	0 h	EO/EC, with/without visual dome, foam/firm surface (20 s, baseline)	Total and mean CoP displacement ↔
Haran et al.[98]	16 soccer players (F/M, 21.0 years)	10 headers in 10 min (25 mph, 22 ft)	1, 24, 48 h	Dark, still/rotating visual scene, still/moving surface (30 s, controls)	A/P RMS CoM displacement ↑ at 1 h, while M/L ↑ at 24 h; both returned at 48 h
Caccesse et al.[22]	30 soccer players (F/M, 21.8 years)	10 headers in 10 min (25 mph, 40 ft)	0, 24 h	GVS, visual translating scene, vibration surface (20 s, controls)	Weightings between vestibular, visual, and somatosensory body movement gains ↔
Vestibular and visual/ocular sensory functions					
Hwang et al.[108]	20 soccer players (F/M, 21.0 years)	10 headers in 10 min (25 mph, 40 ft).	0–2, 24 h	GVS (135 s, controls)	Body angles relative to GVS inputs ↓ at 0–2 h and returned at 24 h
Nowak et al.[173]	78 soccer players (F/M, 20.4 years)	10 headers with 1-min interval (25 mph, 40 ft)	0, 2, 24 h	NPC measure, K–D test (duration N/A, controls)	NPC distance and K–D time/errors ↑ at 0, 2, 24 h

↑ denotes measurements became higher, ↓ lower, ↔ no significant differences; *EO* eyes-open, *EC* eyes closed, *F* female participants, *M* male participants.

plexity post-concussion from studies employing instrumented methods. In addition, vestibular and visual deficits are commonly observed in patients with concussions, while the vestibular and visual systems are often neglected in mechanistic studies. Only a few research groups have measured and correlated concussion impact biomechanics information with standing balance outcomes without identifying conclusive relationships. Consequently, the exact pathophysiological relationship between head impact biomechanics, neurotrauma and standing balance deficits remains an open question. Compared to acute concussion, researchers examining the effects of subconcussive head impacts have shown conflicting standing balance outcomes. Through this review, we have highlighted potential limitations associated with standing balance testing such as trial duration, which should be considered for the design of future studies to improve our fundamental and clinical understanding of balance deficits following acute concussions.

Summarizing Standing Balance Deficits After Concussions and Subconcussive Head Impacts

Based on the studies we reviewed, acute standing balance changes post-concussion have been assessed with postural stability (70%), postural complexity (14%), and sensory testing including SOT sensory analysis and other direct visual/vestibular sensory tests (42%). Taken together, one or more statistically significant post-concussion sensorimotor deficits were identified in 97.1% (33/34) of the reviewed concussion studies. In most of these studies, participants exhibited increases in postural oscillations (IMU angular sway and power: 21–97%; CoP/CoM displacement, velocity, area: 25–120%) and decreases in SOT composite equilibrium scores (7–14%) acutely post-concussion compared to baseline or healthy controls.[58,59,91,94,147,166,183,185,189,193,197] While changes in standing balance are often assessed using stability indicators, these metrics have limited specificity to

identify the underlying deficit. Postural complexity metrics, assessed mainly with CoP entropy, decreased by 19–33% post-concussion, corresponding to an increased regularity in CoP oscillations during standing balance.[34,35,69,189] Decreases in CoP entropy were observed for longer periods following concussion than the increases in CoP oscillations (up to 90 vs < 30 days) and some authors have hypothesized the increased regularity is due to decreased brain connectivity or altered muscle contraction patterns after concussions.[33–35,69] However, to verify such hypotheses, future work is needed to explore if/how brain connectivity or muscle contraction patterns affect the entropy of CoP displacements. Finally, some authors have reported that M/L CoP entropy showed more consistent reductions across test conditions and persisted longer than changes in A/P entropy.[34,35] These results imply potentially different postural control changes between the A/P and M/L balance[34,35,98] that may be related to their distinct biomechanical constraints, dynamics, and sensorimotor control strategies.

Approximately 30–50% of adult and pediatric patients show VOD or provoked symptoms with vestibular/visual testing,[4,64,66,67,164] and SOT sensory tests revealed 12–20% and 7–14% declines for vestibular and visual ratios relative to baseline or controls, respectively.[45,94,149,183,193] While direct sensory assessments have been increasingly applied in the past 5–10 years in concussion testing and shown frequent abnormalities, potential trauma to the sensory end organs is rarely considered in concussion mechanism studies. Theoretically, mechanical head impact forces do not just affect the brain, but could also deform/damage the sensing elements of the vestibular and visual end organs and/or their associated cranial nerves. For example, head impacts may dislocate otoconia from the utricle to the semicircular canals and result in Benign Paroxysmal Positional Vertigo (BPPV).[11,123,163] Davies *et al.* have reported a BPPV rate of ~ 15% in patients with varying degrees of traumatic brain injury (from mild, moderate to severe).[49] Other research groups have reported lesions in the optic nerve and retina as a result of transmissions of mechanical energy from head impacts, mostly for severe head injury cases.[47,174,187,208] It appears possible that, for milder concussive or subconcussive head impacts, structures of the vestibular and visual systems may experience inertial loads that result in temporary sensory dysfunction.

Few researchers have included head impact biomechanics information measured through the helmet and skin-mounted IMUs[58,95,166] or reconstructed concussion impacts through medical reports from the hospital's emergency department.[201] In three out of these four studies, the authors have attempted to link head impact exposure or simulated brain tissue strain data to acute standing balance deficits but failed to identify clear associations. All studies, however, had a relatively small sample size ($N = 12$, 13, and 34) for correlation analysis. In addition, head kinematics from helmets and skin-mounted sensors are prone to kinematics measurement errors due to sensor decoupling from the skull,[3,8,111,244] while medical reports may be affected by recall and reconstruction errors. More accurate and comprehensive head impact exposure data along with other information such as concussion history are critically needed to shed light on the possible relationship between mechanical head impact inputs and induced acute standing balance deficits.

Few studies have investigated standing balance changes after subconcussive head impacts and reported inconsistent results, with only 44% (4 of 9) of studies identifying at least one post-impact standing balance-related sensorimotor deficit. As expected based on the relative severity of subconcussive impacts, the authors of these studies reported more subtle balance changes compared with concussion studies. For example, CoP sway velocity increased by 47% or more acutely following concussions,[45] while less than a 3% increase was reported after soccer headers.[24] Considering most balance deficits dissipate gradually post-concussion, subtle balance changes after subconcussive head impacts are also likely to be more transient, and may or may not be detectable depending on the test time point. In addition, exposure levels varied from single football impacts to 10–20 soccer headers between studies. While head impact kinematics were not quantified, they also likely varied between studies due to factors such as soccer ball launcher distance, angle, and speed. These factors could all contribute to inconsistent findings in the reviewed subconcussive head impact studies. More research is required to investigate standing balance deficits after subconcussive head impact exposure. Considering potential subtle transient changes that may depend on impact severity and frequency, subconcussive studies should consider high-sensitivity standing balance measurements, impact biomechanics sensing, and acute test time points. In addition, repeated subconcussive head impact exposure has also been found to be strongly associated with the onset of concussions, suggesting a second mechanism of concussion.[217] To assess this potential injury mechanism, future sports concussion standing balance studies will also benefit from quantifying subconcussive head impact exposure information.

Limitations and Recommendations in Standing Balance Testing After Concussions and Subconcussive Head Impacts

Despite the general agreement regarding acute post-concussion balance changes, there are some factors that may contribute to varied outcomes between studies. First, the initial assessment time point post-concussion was inconsistent and ranged between 0 to ≥ 72 h, as were follow-up assessments. We note that assessment time point selection can contribute to variance in study outcomes. For example, postural sway changes may be more detectable acutely and gradually dissipate by 14–30 days post-concussion. Second, about 41% of studies (14/34) included baseline balance measurements, while the other studies compared participants with concussions to matched healthy controls. One recent study has cast doubt on the benefit of using baseline in BESS due to the higher within-subject than between-subject variance identified over two football seasons.[191] As such, test-retest reliability and individual variance need to be further assessed for any selected tests to determine the need for baseline testing and threshold to indicate a significant change. Third, among the 34 reviewed concussion studies, age and sex considerations were under-explored for interpreting the results of acute post-concussion standing balance assessments. Given the physiological, anatomical, geometrical, and potential tissue mechanics variances across different ages and sexes, these factors may influence the severity of balance impairments induced by similar head impacts as indicated by some prior studies.[10,44] Finally, current studies have used a range of different sample sizes, which could also contribute to variance in study findings. In the 34 concussion studies we reviewed, sample sizes differ: 18 studies had < 30 participants, 11 had 30–90 participants, 5 had > 90 participants (Table 1).

Sideline and clinical diagnosis of concussions often require rapid and large-scale testing. As such, simple and fast tests such as the BESS have been commonly used. However, we identified inconsistent post-concussion findings from human-rated BESS error scores. The subjective human scoring and known significant learning effects[151] could contribute to such inconsistencies. The BESS test may also not be suitable at detecting subtle balance deficits, nor at detecting the origin of observed balance deficits. Even if a subcomponent of the neural control of balance is disrupted, compensatory mechanisms may activate and achieve seemingly normal standing balance performance.[81,233] For example, if the vestibular system is impaired, the visual, auditory, or somatosensory inputs may compensate and provide adequate information to avoid gross balance errors quantified by the BESS. As demonstrated by the more consistent post-concussion changes from instrumented testing, sensor-based measurements may provide higher sensitivity in detecting subtle postural sway changes. Direct sensory perturbations or manipulations may also increase test sensitivity and reveal specific sensory abnormalities that contribute to standing balance deficits. In addition, applying a dual-task paradigm and increasing the cognitive load during a standing balance task may reduce the brain's capacity to cognitively compensate for subtle balance deficits.[32,59]

The frequency characteristics of standing balance should be carefully considered when designing clinical or laboratory test paradigms. Standing balance is composed of a wide range of frequencies with most of the power contained in the low-frequency range.[31,61,227] Based on data from healthy participants, researchers have suggested that the CoP measurement duration should be at least 60 s for quiet standing trials and 300–360 s for altered vision conditions.[31,227] Nonetheless, most concussion studies only used 20–30 s for each trial, limiting the balance assessment to a single oscillation at 0.03 to 0.05 Hz. Only a few of the reviewed studies applied longer trial times such as 60 s[58,185,189,197] and 120–135 s.[24,78,108] Gao *et al.* showed that both CoP area and entropy increased linearly and sometimes even exponentially as the sampling duration increased from 20 to 120 s 1 day post-concussion, which further motivates longer testing time (at least 60–120 s).[78]

The characterization of postural oscillations complexity using entropy-based metrics has revealed a potentially higher sensitivity for balance assessment and longer-persisting changes post-concussion than stability-based CoP metrics (e.g., variance, range, area). Careful study designs and signal processing considerations are required because the results from entropy metrics are sensitive to the selection of post-processing hyper-parameters.[88] To illustrate this, we show how the key hyperparameters (e.g., time scale and sequence length) influence the entropy estimates from a 120-s CoP dataset (Fig. 3c). In existing concussion entropy studies, authors have often chosen parameters that examine entropy over a 2.5–5 Hz frequency band, avoiding over 90% of the CoP signals that are contained below 0.5 Hz. Thus, future CoP entropy analysis may need to reconsider the selection of post-processing parameters to capture and compare low-frequency complexity changes following concussion.

Fundamental investigations to understand the underlying mechanisms of standing balance deficits can benefit from more specific and quantitative testing paradigms. For example, VOR tests involve a three-neuron neural circuit and quantitative instrumented

tests may reveal specific impairments in this circuitry. Also, electrical activation of the vestibular system enables specific activation of the vestibular afferents without applying a motion to the head and activating other sensory afferents. This approach, along with other sensory-specific signals (e.g., virtual reality), can help in determining the sensory contributions to the balance deficits observed post-concussion. While such tests often involve specialized equipment, they may be necessary to further probe the specific effects of head impacts on critical neural circuitry and pinpoint the cause of sensorimotor symptoms or balance deficits. Emerging standing balance robotic devices can be used to create mechanical/sensory perturbations and manipulate the constraints/relationship between sensory cues and motor control outputs (e.g. altering the brain representation of the body and environment), thus providing researchers with unique opportunities to explore and probe the standing balance circuitry, its adaptability and the underlying components impaired post-concussion.[72,74,107,133,134,190] In addition, accurate head impact biomechanics information paired with the mechanistic sensorimotor/balance tests are required to start understanding the chain of events from mechanical head impact to functional disruptions.

Locomotor assessments, while not included in traditional sideline concussion tests, have also emerged as a common post-concussion test paradigm to assess sensorimotor function.[70,138] Similar to the balance deficits reviewed here, recent review papers have reported locomotor abnormality acutely after concussion that recover over time,[70] with consistent evidence of increased M/L CoM displacement in concussion patients during walking.[138] Measuring CoM displacements during locomotion may help examine the neural control of balance while walking, providing a balance assessment during a dynamic task that involves changes in body configuration over the gait cycle, requiring temporal coordination of movements.[194] Future research should investigate potential links and/or differences between standing balance and locomotor deficits after concussions and subconcussive head impacts.

Linking Brain Deformations with the Standing Balance Circuitry

Given limitations in existing studies, we have insufficient evidence to establish the mechanisms of head impact-induced standing balance deficits. Further evidence may be drawn from a broader range of mechanical modeling, neuroimaging, and sensorimotor assessment studies to hypothesize potential neural components key to such balance deficits. As mentioned earlier, several brain regions implicated in concussion biomechanics studies are intricately linked with the control of standing balance.

The brainstem has been identified as a region of interest in concussion mechanism studies and linked with severe concussion outcomes such as loss-of-consciousness.[54,175] FEM simulations of concussive head impacts have proposed shear stress at the brainstem as a predictor of concussion.[180,248] Supporting these modelling studies, participants exhibited prolonged inter-peak latency in brainstem auditory-evoked potentials within 48 h after a mild head injury.[207] Also, participants with chronic concussion and loss of consciousness at the time of injury exhibited white matter integrity and abnormalities in the brainstem compared to controls.[54,131] Brainstem structures, and in particular the vestibular nuclei and reticular formation, are thought to be critical for the control of standing balance. Indeed, researchers have found correlations between brainstem lesions in MRI and participants' inabilities to maintain balance using single-leg stance condition.[216] Some researchers have examined the vestibular control of balance using EVS methods and reported conflicting outcomes, with either larger motor responses to a vestibular error signal after a bout of 10 headers[108] or no upregulation of the vestibular control of balance for high heading exposure compared to low heading exposure soccer players.[23]

Anatomically, the brainstem and cerebellum are in close proximity, and the cerebellum is another key structure implicated in concussion mechanisms as well as standing balance function.[161] Guskiewicz *et al.* hypothesized that a coup-countrecoup mechanism caused by crown impacts can indirectly concuss the cerebellum resulting in postural stability deficits.[95] Some concussion FEM studies have reported mean maximum principal strains > 0.2 in this region.[43,180] Using diffusion tensor imaging, Mallott *et al.* identified abnormality in the cerebellar tracts acutely post-concussion.[137] Standing balance deficits have been consistently identified in patients with cerebellar lesions[57,84,162] and, depending on the location of the lesions, different balance deficits have been reported.[57] Specifically, increased postural sway of high velocity/low amplitude and low frequency/high amplitude was identified in patients with anterior lobe atrophy and vestibulo-cerebellar impairments, respectively. The cerebellum also plays an important role in sensorimotor adaptation and learning. For example, in common balance test conditions where sensory inputs are manipulated (e.g. sway-referenced vision or somatosensory information), the cerebellum is thought to re-calibrate motor actions when consistent errors are encountered.[14,102] Cerebellar deficits could indeed lead to an increased difficulty for patients with concussions to adapt to sensory manipulations during

standing balance (e.g., SOT test). Indirectly supporting this possibility, visuomotor adaptation difficulties involving upper limb tasks have been confirmed in concussion patients, revealing larger within-subject variance and greater differences compared to controls, and such adaptation deficits persisted up to 18 months post-concussion.[209,230] Notably, there is a lack of tissue displacement data for the brainstem and cerebellum to validate FEMs, leading to uncertainties in brain deformation modeling studies to further examine these areas of interest.[65]

The basal ganglia are thought to contribute to reward-based sensorimotor reinforcement learning.[60] Lesions in the basal ganglia have been associated with reduced sensorimotor adaptation and learning.[41,48,159] One computed tomography study identified focal impairments in the basal ganglia area in 55.2% of the patients with mild or moderate brain trauma.[90] The thalamus is situated at the intersection between multiple neural regions including the cortex, cerebellum and brainstem. Patients with unilateral thalamic lesions were reported to suffer from sensory loss and inabilities to stand.[143] Several functional magnetic resonance imaging studies have found abnormalities in this area in concussion patients.[221,250] Strains in the thalamus have also been associated with concussions in FEM analysis.[247] Zhang *et al.* hypothesized that the high strains induced by coronal head rotations in the thalamic-midbrain area were due to the impinging effect exerted by the falx cerebri.

Aside from regional considerations, diffuse brain deformations could potentially lead to widespread axonal connectivity disruptions. Researchers using diffusion tensor imaging have frequently found diffuse connectivity disruptions post-concussion.[2,137,205] The potential diffuse decoupling of brain regions has been hypothesized to manifest as decreases in neural complexity that could be identified using postural entropy.[34,35] Some researchers have proposed that concussions and subconcussive head impacts may inhibit neural transmission inside the corticomotor regions, resulting in the dampening of neural muscular control.[51,52,56,225] Increased latencies between primary motor cortex activation and lower leg muscles were identified using transcranial magnetic stimulation in university boxers/Muay Thai athletes following three rounds of sparring bouts.[56] Similar observations have also been reported after repeated concussive exposure.[51,52,225]

Summary

Overall, there remain major gaps in the data available to investigate the mechanisms underlying acute standing balance-related sensorimotor changes after concussions and subconcussive head impacts. The key points from our findings are summarized below:

- Instrumented standing balance testing after concussions has demonstrated prevalent acute increases in postural sway within 1–2 days post-concussion, followed by recovery to baseline within 14–30 days post-injury. Subconcussive studies have shown subtle and inconsistent standing balance changes.
- Postural complexity, specifically CoP entropy decreases, may persist for longer than the increases in postural sway post-concussion; however, the physiological relevance of such changes remains unclear.
- Clinical and laboratory standing balance testing should consider the low-frequency characteristics of standing balance dynamics and increase trial duration from 20 s to at least 60–120 s.
- Investigations of the mechanisms underlying acute standing balance deficits after concussion should consider emerging sensorimotor testing paradigms that can quantitatively target and characterize the specific sensory and motor components underlying these balance deficits.
- Concussion mechanisms studies should consider the potential role of vestibular and visual end organs and their cranial nerves in common concussion deficits.
- Brain regions such as the brainstem, cerebellum, basal ganglia, thalamus, and cortex have been identified as regions of interest in concussion mechanism studies and are known to contribute to standing balance, but their links to standing balance deficits following concussion need further confirmation.
- More accurate head impact biomechanics measurements are needed to link head impact kinematics and mechanical deformations in balance-related sensory/neural components with global balance changes as well as local functional changes in neural structures/pathways contributing to standing balance.

Research into the mechanisms of concussion has mostly focused on brain mechanics and function, despite increasingly broad definitions of the concussion that consider a wide range of neurocognitive, sensorimotor, neuropsychological, and physiological symptoms. Given their prevalence, investigating the mechanisms of standing balance deficits could be one major step towards explaining the variance we see in post-concussion outcomes. Understanding the underlying acute brain injury mechanisms will inform more effective practices in applying standing balance testing

for diagnosis, monitoring, and management of mild142 traumatic brain injury.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

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