11-2005

A Nonlinear Dynamic Approach for Evaluating Postural Control

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A nonlinear dynamic approach for evaluating postural control: new directions for the management of sport-related cerebral concussion

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Acknowledgement

Dr. Cavanaugh's dissertation work focusing on the application of ApEn to the analysis of COP time series in athletes with concussion was conducted in the Sports Medicine Research Laboratory at the University of North Carolina, Chapel Hill, NC (USA) under the direction of Dr. Guskiewicz. The work was funded in part by grants from the Injury Prevention Research Center at the University of North Carolina at Chapel Hill, the National Center for Injury Prevention and Control (USA), the National Operating Committee on Standards in Athletic Equipment (USA), and the National Athletic Trainers' Association Research and Education Fund (USA).

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Figure 1. The six conditions of the Sensory Organization Test (Reprinted with permission from NeuroCom International, Inc.)

Figure 2. COP AP time series collected from an athlete standing still with eyes closed before and after cerebral concussion. Paradoxically, the range of COP displacement decreases from approximately 5 cm before injury (top panel) to less than 4 cm after injury (bottom panel), suggesting an *improvement* in postural stability. At the same time, however, COP oscillations change from being relatively more irregular before injury (ApEn value = 0.8694) to being more periodic after injury (ApEn value = 0.6619). The increase in regularity (decrease in ApEn value) was interpreted as an indication that the postural control system was more constrained after injury.

Abstract

Recent research suggests that traditional biomechanical models of postural stability do not fully characterize the nonlinear properties of postural control. In Sports Medicine, this limitation is manifest in the postural steadiness assessment approach, which may not be sufficient for detecting the presence of subtle physiologic change after injury. The limitation is especially relevant given that return-to-play decisions are being made based on assessment results. This update first reviews the theoretical foundation and limitations of the traditional postural stability paradigm. It then offers, using the clinical example of athletes recovering from cerebral concussion, an alternative theoretical proposition for measuring changes in postural control by applying a nonlinear dynamic measure known as Approximate Entropy. Approximate Entropy shows promise as a valuable means of detecting previously unrecognized, subtle physiologic changes after concussion. It is recommended as an important supplemental assessment tool for determining an athlete's readiness to resume competitive activity.

1. Introduction

Sports injuries are not commonly associated with "balance problems." Reports of disequilibrium or falls after injury are rare, because athletes generally are in excellent health or because most injuries are isolated to relatively few musculoskeletal structures. Sport activities, however, often demand exquisite body control, such that even subtle impairments may interfere with optimal performance without producing obvious unsteadiness. Perhaps more so than most individuals, athletes utilize a wide array of complex strategies involving arms, legs, torso, neck, and head for control of body position to gain advantage over their competitors. Injuries producing weakness, sensory impairment, diminished joint range of motion, or alterations in neural processing potentially can affect the ability to control the orientation of the body in space.[1, 2]

The medical assessment of postural control after injury often includes the determination of postural stability in quiet standing. In the tradition of Moritz Romberg,[3] postural stability assessment typically requires an athlete to stand as still as possible under any one of a number of base of support (double limb stance, tandem stance, or single limb stance) or sensory (eyes open vs. eyes closed) conditions.[4-6] More stable athletes are assumed to be able to stand with less postural sway about a central equilibrium point. In this sense, postural control is operationally defined using an amplitude metric (e.g., magnitude of sway) that denotes precision; i.e., optimal control is evidenced by less movement, or error, about a target position. Athletes who demonstrate normal postural stability (relative to their age-matched peers) are generally assumed to have a healthy postural control system.

This assumption may be fundamentally flawed. During the last decade, a variety of studies have revealed that the variability of center of pressure (COP) location during quiet

standing is not the result of random error.[7-12] Instead, COP oscillations, despite appearing erratic and irregular, contain a "hidden" structure, or orderliness, that emerges in time, presumably as a result of interactions among underlying postural control system components. Recent evidence supports the idea that with advanced age and disease, the complexity of temporal structure breaks down, resulting in more regular COP oscillations.[13, 14] ***Importantly, changes in the regularity of COP oscillations can occur in the absence of changes in postural stability***.[15] Optimal postural control (in quiet standing), therefore, appears to be characterized by COP oscillations that not only are small in amplitude but also are relatively unconstrained and irregular.

For injured athletes, the return of optimal postural control is an important rehabilitation goal. Current measures of postural stability, however, may be inadequate for helping athletes, coaches, and medical professionals judge when complete recovery of postural control has occurred. The purposes of this update are to (1) to review the theoretical basis and limitations of current approaches to postural control assessment; (2) to offer an alternative measurement approach based on a nonlinear dynamics theoretical framework of motor control; and (3) using sport-related cerebral concussion as an example, to review recent evidence that highlights the limitations of current approaches and supports the application of a nonlinear dynamic theoretical framework for revealing unique changes in postural control after injury.

2. Defining and Modeling the Postural Control System

2.1 Terminology and the Task of Postural Control

The task of postural control involves controlling the body's position in space for the dual purposes of stability and orientation.[16] *Orientation* is defined as the ability to maintain an

appropriate relationship between body segments and between the body and the environment. *Stability*, in the broadest sense, refers to the ability of a system to resist perturbations.[17] *Postural stability* defines the ability to maintain a desired postural orientation, either at rest or during movement, in response to perturbations generated from either internal or external sources. For human functional activities performed in standing or sitting, postural stability specifically refers to the ability to resist perturbations such that the whole body center of mass is maintained within the limits of the base of support. *Postural steadiness*, a special case of postural stability, defines the ability to stand as motionless as possible.[18] *Equilibrium*, a term derived from Newtonian mechanics, refers to conditions in which an object is at rest (static equilibrium) or in constant motion (dynamic equilibrium); i.e., not acted upon by unbalanced external forces.[17] *Balance*, often used synonymously with equilibrium, refers to equilibrium about a specified axis, such as the vertical axis in upright standing.[19] *Postural control* and *balance control*, therefore, can be used interchangeably to refer to the act of maintaining or returning the body close to a state of static or dynamic equilibrium.[19]

2.2 Modeling Postural Control

Current models of the postural control system generally have evolved from the seminal work of Nikolai Bernstein (1896-1966), for whom the model of nervous system function considered the whole body as a mechanical system subject to gravitational and inertial forces.[20] For Bernstein, coordinated movement, including postural control, was a problem that involved mastering the many redundant degrees of freedom defined over several levels of biological analysis. The large number of potential degrees of freedom in the control system precluded the possibility that each is controlled individually at every point in time; thus, Bernstein proposed that control of integrated movement was probably distributed throughout many interacting

systems working cooperatively. Consistent with this idea, current models of postural control can be broadly organized into two categories: models that *describe* a system of interacting components and models that seek to *predict* how components interact to achieve postural control. Descriptive models are useful for conceptualizing the multidimensional nature of the postural control system but do not guide the measurement of postural control system changes after injury or in response to rehabilitation.[1, 16]

Predictive models of postural control system output have evolved from both linear and nonlinear dynamics frameworks (Table 1). Linear dynamic modeling is based on a stimulus-response paradigm, in which system output can be predicted from input using linear equations and knowledge of simple interactions among system components. Importantly, the output of the entire system represents the summed output of local interactions.[21] Deviations from expected output values are thought to represent random error. Linear models generally predict that magnitude of variability in a postural control output signal increases in direct proportion to the intensity of the perturbation or the severity of disruptions in the feedback loop.[22]

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The linear dynamics theoretical framework has been used to develop single and multi-link biomechanical models that predict ankle joint torque and/or postural sway angle in response to an external perturbation.[23-25] A simple, early linear model of postural control was based on a sensory-motor feedback loop circuit, in which body position in space was compared to centrally represented equilibrium point using vestibular information.[26] The model was used to explain how postural corrections in response to an external perturbation are carried out through the

musculoskeletal apparatus via a combination of automatic monosynaptic reflexes and postural synergies. More recently, the basic model has been expanded to include multiple feedback loops[27] and concepts of sensory organization,[28] sensory weighting,[29] and sensory noise.[30]

Alternatively, the nonlinear dynamics theoretical framework supports the idea that postural control emerges through the interaction of individual physiologic systems, task demands, and environmental constraints (Table 1.)[16, 31, 32] Nonlinear systems organize themselves according to initial conditions and simple rules that govern the interactions among the most basic, individual components.[21] Because these components form multi-link networks, simple interactions do not predict the behavior of the system as a whole, especially over longer time scales. Depending on initial conditions, small perturbations to the system can have no effect, a proportional effect, or a dramatic effect on system output. In contrast to linear models that analyze the magnitude of output signal variability and focus on individual system components, nonlinear models use the time evolutionary properties of an output signal to draw inferences regarding interactions within the underlying control system.[14] Nonlinear dynamics offer the postural control system the ability to be adaptable and flexible in an unpredictable and ever-changing environment.[22, 33]

2.3 Postural steadiness and the mechanical perspective

The linear dynamics framework and its associated biomechanical models have contributed to the development of commonly employed clinical measures of postural control, for which postural stability is the facet of interest. Postural stability is often evaluated in terms of "postural steadiness," i.e., how closely one can mimic a state of static equilibrium in quiet standing. Using static posturography, postural steadiness is measured as the ability to stand upright as motionless as possible with the vertical projection of the body center of gravity (CG) maintained within the

limits of the base of support defined by the feet. Both non-instrumented (Romberg Test,[3] Balance Error Scoring System[4]) and instrumented force plate paradigms[34] have been developed. The mechanical approach assumes that under normal circumstances, a healthy, developmentally mature individual can precisely regulate the position of the body in space, such that there is only slight variation in whole body CG position about a central equilibrium point. In dynamic posturography, an external perturbation is applied and postural stability is investigated in terms of input / output relationships.

Because measurements of CG variation are technically complicated, cumbersome, costly, and error-prone,[35] steadiness is often measured by center of pressure (COP) variation as a function of time. COP is the point of application of the resultant ground reaction force vector acting on the surface of the force plate.[17] In reality, however, ground reaction forces do not act at a single point, and thus, the COP represents a weighted average of forces applied diffusely over the contact area. In fact, relative to the forefoot and heel, ground reaction forces at the COP for an individual in quiet standing are relatively small.[36] In addition, the COP is a compound signal: it represents not only the position of the whole body CG, transformed by the multi-linked system of the body to the support surface, but the muscle activity used to control equilibrium.[10] The COP corresponds to the vertical projection of the CG only under static conditions in which no horizontal accelerations are applied.[18]

Given that the COP (1) is an “artificial” rather than an anatomical point, (2) does not always correspond to the location of ground reaction force under the foot, (3) is a compound signal, and (4) is not identical to the whole body CG, many investigators have adopted the biomechanical view that unsteadiness in quiet standing is proportional, rather than equal, to COP variability.[18] In this context, the COP reflects the net motor control signal output necessary to correct the

imbalance of the whole body CG.[25, 37] Because COP is a directly measured, highly reliable, and easily quantified parameter, a wide variety of summary statistics reflecting the amplitude of COP variability have been developed.[1, 38-40] Many of these summary statistics have been shown to change reliably under a variety of experimental manipulations, including neurological and musculoskeletal pathology,[41-45] developmental change,[46, 47] changes associated with aging,[38, 48] and changing environmental constraints.[49] In general, greater magnitude of COP variability is regarded as an indication of greater postural instability.

2.4 Limitations of the biomechanical approach to measuring postural steadiness

Although postural steadiness protocols are relatively simple and safe, and COP variability has demonstrated clinical utility as an indicator of postural control system performance, the mechanical perspective described above has not provided substantial insight into the interactions among system components that produce postural control.[50] A summary of limitations appears in Table 2. One issue is that postural steadiness is a special case of postural stability in which no external perturbation is applied. Instead, stability is inferred from the ability to resist internal perturbations created by skeletal muscle activity, heartbeat, respiration, and cognitive activity.[9, 38, 51] Without external perturbations, and because the internal perturbations are difficult to characterize, input / output, linear dynamics models of postural steadiness are not applicable. A second issue is that traditional linear statistics (e.g., mean, standard deviation) assume that errors in COP location around a central equilibrium point are both random and independent. In this view, because such noise presumably results from measurement error or the degradation in neuromuscular control, averaging the COP variability as a general indication of error is both justified and practical. Averaging, however, suppresses the time-evolving structure of COP

variability and limits the inferences that can be made regarding control system dynamics.[14, 22] In addition, the variations observed in the COP time series have been shown to be neither random nor independent but rather to have deterministic properties.[7-12]

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Inferences based on COP summary statistics also are limited because steadiness has not been theoretically defined in terms of force platform measures.[18] There is no neurophysiological evidence, for example, that supports true static equilibrium in quiet standing as an achievable or even desirable behavioral goal. Alternatively, some investigators have suggested that postural sway necessarily serves as a sensory information gathering mechanism of the control system.[31, 37, 52, 53] There also exist individuals for whom reduced, rather than increased, COP variability may suggest poor postural control. For example, some below knee amputees may appear very steady in quiet standing but cannot effectively respond to an external perturbation.[54]

Another limitation of the mechanical perspective is that there is no external criterion against which measurement validity can be established.[18] In the absence of a "gold standard," investigators have attempted to demonstrate strong relationships between various force plate parameters, under the assumption that measures that represent the same construct should be highly correlated.[18, 55] Although the results of these studies have been mixed, generally the correlations among measures were relatively weak. This finding supports the contention that postural control is a multivariate process[9, 20, 29] and that biomechanical output measures do not adequately capture postural control system dynamics.[14]

A final criticism of the postural steadiness paradigm is that posturography lacks reliable and sensitive sway determinants that can be used for the evaluation of pathological changes.[56] Despite the many reports of excessive postural sway (or COP variability) in the presence of advanced age and disease, static posturography alone apparently is not sensitive enough to reliably detect abnormality in at least a few impaired populations.[57, 58]

3. An Alternative Theoretical Proposition: Measuring Complexity in a Nonlinear Postural Control System

3.1 Evidence for postural steadiness emerging from a nonlinear system

The human body is a complex mosaic of nonlinear dynamical systems,[59] organized within both spatial and temporal domains. Physiological rhythms, i.e., output produced as a result of system interactions over time, are abundant in biological systems and central to life.[60] Well-known examples of physiologic rhythms include heartbeat, respiration, reproduction, and sleep-wake cycles. Less obvious, but of equal physiologic importance, are such oscillations as the release of insulin and luteinizing hormone, peristaltic waves in the intestine and ureters, electrical activity within the cortex and autonomic nervous system, and constriction in peripheral blood vessels and the pupil.[60] In our view, oscillations in a postural steadiness time series constitute a physiological rhythm associated with the postural control system.

Traditionally postural oscillations had been assumed to be a stationary, stochastic process.[61, 62] However, an alternative explanation for the existence of COP fluctuations is that they have a structure that is dependent on the time scale of observation. This notion has been supported by recent research evidence,[7-9, 27, 63] that has confirmed the nonlinear properties of postural steadiness time series. Under fixed task and environmental conditions, nonlinear properties in the

postural system arise due to elastic and damping properties of muscles and the varying time scales (delays and thresholds) of sensory systems.[10, 64] Clinical measures derived from the nonlinear dynamics framework are based on the recognition that the collective interaction among these properties, rather than their individual characteristics, produces the visible, complex behavior of the postural control system .[21]

Complex systems have the potential to generate output signals containing temporal structure imbedded within what appear to be random oscillations. One nonlinear measure, known as Approximate Entropy (ApEn), quantifies the ensemble amount of randomness, or irregularity, contained in a time series.[65] The ApEn algorithm essentially applies a moving window procedure to determine the probability that short sequences of data points are repeated (within a certain error tolerance) throughout the entire time series. Expressing the average probability in logarithmic form (and taking the inverse), ApEn generates a unit-less real number from 0-2. Zero values correspond to less randomly ordered time series that contain greater amounts of temporal structure (i.e., sine wave), whereas values of 2 indicate a completely random and irregular time series (i.e., Gaussian noise).[22]

ApEn represents a class of nonlinear measures that characterize orderliness in the temporal output of a complex system. A separate class of nonlinear tools, known as dimensional measures (i.e., fractal scaling measures like Correlation Dimension[66]), characterize complexity in terms of the degree to which successive points in a time series are related to one another over a range of time scales. Unlike dimensional measures, however, ApEn has the advantage of conceptual simplicity and practical applicability that make it particularly suited for relatively short, noisy biological output signals associated with human movement.

ApEn, as a nonlinear measure of complexity, focuses on the time evolving properties of the output signal to draw inferences regarding interactions within the underlying control system. Like other nonlinear tools, ApEn requires the collection of data over a relative long period of time to allow the system's properties to unfold, and thus, is not useful for assessing the effect of a specific stimulus (perturbation) on postural sway that may last few milliseconds.

ApEn also is conceptually distinct from and does not necessarily correlate well with traditional linear tools of time series variability. In the spectral domain, for example, small ApEn values often correspond to a narrow frequency range and large ApEn values correlate with a broad-band spectra and a great frequency range; nonetheless, power spectra analysis and the ApEn measure can produce conflicting results.[67, 68] A similar incongruence can exist between ApEn and amplitude measures of variability (e.g., SD).[67, 69] Thus, it seems that ApEn can complement a frequency analysis and provide useful information about the time domain.

3.2 Change in Complexity and Postural Control

Two hypotheses have been developed to describe how a change in regularity corresponds to a change in underlying system complexity. Both can be derived from the broad idea that the entropic concept of randomness is associated with a diversity of possible control system configurations that could produce a given output signal.[70]

Pincus used mathematical modeling to generate support for the idea that the regularity of system output would reflect the degree of isolation of system components from their surroundings.[71, 72] Systems with a relatively limited number of viable interconnections among components are less complex and generate more predictable output. Fewer components would contribute to the output signal, but each would make a more dominant contribution.

Alternatively, complex, highly interconnected nonlinear systems would have good lines of communication, marked by large numbers of interactions.[73] As a consequence, the output signal would contain influences from a greater number of system components, but with each interaction making a relatively small contribution to the behavior of the composite system.

Newell proposed a similar idea using a degrees of freedom theoretical framework.[14] Accordingly, a more regularly ordered time series is produced by a system with fewer degrees of freedom, and therefore, greater constraint. In contrast, more irregular output is produced by less constrained systems with greater number of degrees of freedom.[14] Completely unconstrained systems with an infinite number of degrees of freedom are produced by an entirely random process. The implication is that either fewer or more poorly organized degrees of freedom reduce the adaptive capability of the individual.[74]

Although support for the hypotheses remains primarily theoretical, together they suggest that a healthy postural control system, because of numerous interconnections among its components, is capable of adapting to a wide variety of task demands and external conditions. Accordingly, when the system is allowed to operate with minimal constraints (e.g., at rest during quiet standing under normal sensory conditions), system output (e.g., COP location) appears to fluctuate in relatively random fashion, presumably reflecting the readiness of the system to rapidly respond to perturbation. In the presence of disease or injury, however, normal interconnections among system components would be compromised, thereby reducing the complexity of the system. As a result, fluctuations in system output at rest would be more constrained, so as to appear more regular and predictable.

3.3 ApEn as a Measure of Postural Control

ApEn has been employed in several investigations of postural control involving non-athletes [11-14, 53, 75] and has shown promise as a clinically applicable tool. The combined results of these studies suggest that healthy, mature postural control in quiet, upright standing is characterized by COP oscillations that are both relatively irregular and small in amplitude. We have recently determined response stability and precision estimates for ApEn of COP AP and ML time series recently for healthy, young adults across six sensory conditions (Figure 1.)[11] Overall, ApEn values for AP time series ranged from 0.50 to 0.84 and demonstrated moderate to good test-retest reliability between trials (ICC (2,1) range 0.52 - 0.75) and days (ICC(2,2) range 0.79 - 0.90). In comparison, COP ML oscillations were relatively more random (ApEn range 0.75 to 0.93), and consequently, were less stable between trials (ICC (2,1) range 0.01 - 0.72) and days (ICC (2,2) range 0.53 - 0.77). As sensory information was withdrawn or degraded, COP oscillations became more regular (lower ApEn) and larger in amplitude (diminished postural stability). Consistent with the aforementioned hypotheses (Section 3.2), we suggested that the removal of accurate sensory feedback not only made it more difficult for individuals to precisely control body position, but also artificially constrained interactions among control system components, so as to produce more regular oscillations in system output.

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4. Recovery of Postural Control After Cerebral Concussion: New Insights Using the Nonlinear Dynamic Theoretical Framework

4.1 Basic concepts of sports-related cerebral concussion

Approximately 300,000 sports related traumatic brain injuries occur annually in the United States.[76] Although the proportion that are repeat injuries is unknown, a study of high school and collegiate football players have reported within season recurrence rates of 14.7%.[77] Importantly, Guskiewicz et al. found that nearly all of the in-season repeat injuries occurred within 10 days of the first injury, suggesting that the brain may be more vulnerable to a second injury during this acute period.[78] There also exists at least some evidence for an increased risk of subsequent traumatic brain injury among persons who have had at least one previously.[79] Additionally, the severity of cerebral concussion appears to increase with recurrent injury.[77] Repeated mild traumatic brain injury (MTBI) occurring over an extended period (i.e., months or years) can result in cumulative neurologic and cognitive deficits,[80] but repeated injuries occurring within a short period (i.e., hours, days, or weeks) can be catastrophic or fatal.[81] In light of these reports, there has been a growing concern regarding the potential for persistent impairments in athletes who sustain multiple cerebral concussions over time. This concern has focused particular attention on the criteria used to determine when and whether an athlete with cerebral concussion is fully recovered and ready to resume competitive activities.

Throughout history, the pathophysiology of cerebral concussion has been a matter of dispute.[82] Recent evidence, however, suggests that cerebral concussion produces functional rather than structural neurophysiologic changes in the cortex and brainstem reticular formation.[83] The latter disturbance in particular is presumed to account for the autonomic, motor, and postural

impairments that occur in many individuals.[6, 83] Subtle vestibular deficits also may be possible in the MTBI population.[84] Animal studies of mild head injury have associated an increased brain demand for glucose, combined with a reduction in cerebral blood flow, with an extended state of vulnerability to further neuronal loss.[85] From a complexity perspective, these concussion-induced pathophysiological changes might reflect alterations in the patterns of interaction among components of the central nervous system. Diffuse axonal injury, resulting from direct trauma to neurons or secondary metabolic sequelae, may reduce or distort interactions among neurons in the brain.[86] As a result, brain regions might become less coupled to one another, thereby increasing the regularity of cortical oscillations.[68] Indeed, the symptoms of minor concussion (being "stunned, dinged, or dazed") are often strikingly similar to those of minor epilepsy, a condition in which patterns of cortical activity become more synchronized.[83] Because patterns of brain electrical activity are known to be reflected in patterns of electrical signals descending to the periphery,[87] it is plausible that changes in patterns of COP oscillation after concussion reflect changes in cortical oscillatory activity.

4.2 Recovery of postural stability after cerebral concussion

The transient nature of most cerebral concussion symptoms and impairments suggests that postural instability, when it exists, resolves relatively quickly. Indeed several longitudinal studies in college athletes have demonstrated a typical pattern of recovery in which postural stability returned to baseline within approximately 3-5 days.[5, 88-90] These studies were similar in their use of a protocol that required subjects to stand quietly for 20 seconds on a force platform under 6 different sensory conditions (Figure 1.) The conditions varied in terms of the amount and quality

of environmental cues available, along a continuum of easy (accurate somatosensory and visual cues) to more difficult (inaccurate somatosensory, and unavailable or inaccurate visual cues).

Guskiewicz, Perrin et al. used two indices of COP displacement about a fixed, central reference point to quantify impairments in postural stability in two cohorts of college and high school athletes with cerebral concussion.[88] One cohort included football players who had undergone baseline testing (n = 10), while the other included athletes from other sports who had not undergone baseline testing (n = 9). All subjects were matched to healthy comparison subjects. Although precise balance scores were not reported for each sensory condition, the degree of balance impairment in all subjects with cerebral concussion appeared to increase with task demand. The authors concluded that athletes with acute cerebral concussion display problems using various combinations of somatosensory, visual, and vestibular information for control of upright posture during the first few days following injury.

In a follow-up study, Guskiewicz, Riemann, et al. used a sway referenced, force plate driven, computerized balance assessment tool (NeuroCom International Inc., Clakamas, OR, USA) to quantify postural steadiness in a sample of 11 college athletes who had sustained a cerebral concussion.[89] The Sensory Organization Test (SOT) protocol was used for data collection (Figure 1.) Subjects with cerebral concussion were tested on the first, third, fifth, and tenth day post injury. Healthy subjects were used for comparison. On each test day, SOT composite equilibrium scores were calculated as the average of a group of 14 individual condition scores that were based on the AP component of the COP trajectory. The equilibrium score represented the margin of stability, represented as a percentage difference between the peak-to-peak COP amplitude and theoretically defined limits of stability. A higher score indicated a greater margin of stability, i.e., smaller range of COP displacement. Additionally,

visual, vestibular, and somatosensory ratio scores were calculated to determine relative differences between the equilibrium scores of various conditions. The results revealed not only that composite equilibrium scores returned on average to near baseline levels by Day 3 post injury, but that athletes initially appeared to have more difficulty using visual and vestibular information for control of body sway. Importantly, the authors emphasized that the clinical presentation of athletes with cerebral concussion was quite variable. In some athletes, acute balance deficits were present in the absence of amnesia and/or other post-concussion symptoms. In other athletes, cerebral concussion did not affect the postural control system but resulted in somatic symptoms or cognitive problems.

Mrazik et al. reported the composite equilibrium score in four college athletes who sustained a cerebral concussion.[90] Three of the subjects each had a different severity level of injury (Grades I-III) but no history of prior cerebral concussion. The fourth subject had a Grade II concussion but had had a previous concussion. Pre-injury (pre-season) equilibrium scores were reported. No healthy subjects were used for comparison. The results revealed that severity of cerebral concussion appeared to correspond to the severity of balance impairment. Despite the varying histories of the subjects, all equilibrium scores returned to baseline levels by the fifth day post injury.

The entire group of concussion studies cited above suggests that force plate measures of postural stability may be more sensitive than traditional clinical measures, e.g., Romberg test, for identifying balance deficits in individuals with cerebral concussion. Furthermore, sway-referenced testing systems, which allow the manipulation of various sensory conditions, appear to be necessary to optimize the sensitivity of postural steadiness testing. It is important to recognize, however, that force plate measures alone have not consistently revealed postural

control system alterations post injury.[89] This inconsistency suggests several possibilities; that (1) not all concussions produce postural control impairments; (2) postural control impairments in some athletes resolve in less than 24 hours (before initial post injury testing); or (3) COP parameters derived from biomechanics are not sensitive enough to detect subtle alterations in postural control after cerebral concussion. Especially because some athletes (collegiate football players) appear to be at increased risk for a within-season repeat concussion within the first 10 days after a first concussion, the sensitivity of clinical measures for detecting physiologic abnormality has important implications for decisions regarding an athlete's safe return to competition.

4.3 Detecting altered postural control in athletes without postural instability

In light of the issues emphasized above and continued concerns regarding the sensitivity of postural stability measures, we employed ApEn to examine changes in postural control in a sample of 27 male and female collegiate athletes without postural instability following cerebral concussion.[15] Using a longitudinal design, athletes were tested using the SOT at preseason and within the first 48 hours after injury. A clinically normal Composite Equilibrium Score, no more than 5% below preseason values, verified the absence of postural instability after injury. For comparison, thirty healthy non-athlete subjects were tested on two occasions. ApEn was applied to the AP and ML components of the COP time series collected using the SOT protocol. Whereas for the healthy subjects, ApEn values remained stable across days, ApEn values among the injured athletes generally declined across all sensory conditions. More dramatic increases in COP regularity were observed for the ML time series. The result is important, not only because a change in postural control was detected in the absence of postural instability, but also because it

revealed, in contrast to previous studies using ApEn, that the relationship between COP amplitude and regularity is not linear. ApEn is not a simple surrogate for postural stability measures, but provides unique information regarding the state of the postural control system. An illustrative example is presented in Figure 2.

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The increase in regularity of COP time series was interpreted as an indication that the postural control system was more constrained after injury, as a result of either mechanical stiffness or neurophysiologic impairment.[15] This interpretation is consistent with the aforementioned theoretical propositions,[14, 72, 73] which together suggest that an overly constrained postural control system may be less able to mount a physiologic response to a particular task or environmental demand. For athletes returning to competitive activity after concussion, impairment in the adaptability of the postural control system may limit performance during rapidly changing task and environmental conditions. To substantiate this idea, we believe that new post-concussion assessment protocols could be developed, in which athletes not only are re-evaluated during the performance of individual sport-specific tasks, but also are assessed using multiple, simultaneously presented, and rapidly changing tasks and conditions.

4.4 Comparing the recovery of postural stability with the recovery of altered COP regularity

To determine if changes in COP regularity after concussion parallel the recovery of postural stability, we studied COP data collected using the SOT from 29 athletes at preseason,

within 48 hours after injury, and between 48 and 96 hours after injury.[91] The sample included injured athletes with and without postural instability. The number and severity of concussion-related symptoms was also recorded at each time interval. Consistent with previous reports, impaired postural stability resolved within 3-4 days. Importantly, however, ApEn values for all subjects remained depressed, even among athletes whose initial postural instability had resolved. There was no significant relationship between changes in ApEn values and symptoms after injury, suggesting that the changes in postural control occurred independently from changes in other physiologic systems.

This pattern of results conflicts with a traditional linear dynamics theoretical perspective, in which an input stimulus (i.e., concussion) would produce a predictable and proportionate output response (i.e., series of impairments) according to a fixed set of physiological and biomechanical determinants. In contrast, our results support a nonlinear theoretical framework, according to which the postural control system contains a group of highly interconnected neuromuscular, musculoskeletal, and cognitive subsystems whose interactions are dynamic and self-organizing. Consequently, relationships among various subsystems, and the resulting global system output, are not necessarily predictable or proportionate to a given input. We would not expect, therefore, for concussion to produce predictable postural control impairments, or for the recovery of postural control necessarily to be related to symptom resolution.

As stated previously, recent evidence suggests that cerebral concussion produces functional rather than structural neurophysiologic changes.[83] We contend that the disruption in interconnectedness presumed to occur within the postural control system could account for an increase in system constraint without producing detectable impairments in individual sensory or motor subsystems. This proposition allows for the possibility that traditional performance

measures of postural stability, as well as neurological measures of sensory and motor systems, would not necessarily reveal concussion-related impairments. The proposition also suggests that nonlinear measures, which reflect the amount of global postural control system constraint, should be included as an important component of post-concussion medical assessment.

5. Summary and Recommendations for Postural Control Assessment Approaches in Sports Medicine

Measuring the extent of postural control system changes after cerebral concussion remains an important challenge in Sports Medicine. The traditional biomechanical approach, which is based on a linear model of system behavior and primarily concerned with postural stability, appears to lack diagnostic sensitivity in cases of mild injury. As an alternative, Approximate Entropy, a nonlinear measure of postural control, has revealed subtle system changes after concussion that appear to be more prevalent and last longer than thought previously. Given the proposed link between the complexity of control system interactions and the capacity for adaptation to stress, the failure of an athlete to recover pre-injury levels of COP irregularity may have important implications regarding the safe return to competition.

There currently exists no validated method for using ApEn to classify the integrity of postural control in absolute terms. Thus, unlike biomechanical balance measures, for which static equilibrium is the theoretical, albeit unattainable, goal, ApEn values measured on one occasion are not clinically useful. ApEn is best suited for measuring changes in postural control, especially in circumstances where subtle abnormality increases the likelihood of subsequent injury. In this sense, ApEn appears to be a valuable supplemental tool for determining the state of the postural control system.

The implications of this review for clinical practice and future research are significant. The evidence concerning ApEn as a measure of change supports the widespread use of preseason baseline testing for all athletes at risk for sport-related cerebral concussion, especially when pressures to rapidly return to competitive activity are considerable. Whenever possible, traditional measures of postural stability should be supplemented with ApEn applied to COP time series. This implies that in an optimal clinical setting, quantitative (force plate) measures that capture COP variability are an important and necessary assessment tool, and that software should be developed to simultaneously generate COP amplitude and regularity indices. Finally, the review implies that other types of sport-related injuries that affect postural control during athletic performance might benefit from similar multi-modal assessment and the application of a nonlinear theoretical framework to recovery.

Future Sports Medicine investigations should attempt to determine (1) the response stability of ApEn values in athletes with concussion and other injuries; (2) how long ApEn values remain depressed after injury; (3) what factors correlate with the eventual return of ApEn values to pre-injury levels; and (4) what specific neurophysiologic or mechanical mechanisms explain the changes in COP regularity after concussion. Ideally, these investigations would lead to the determination of whether reduced ApEn values after injury are associated with an increased risk for subsequent injury recurrence.

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Table 1. General Characteristics of Linear and Nonlinear Postural Control System Models

	Linear	Nonlinear
Paradigm	• Stimulus-response	• Self-organization
Degree of postural control reflected in the output signal	• Predicted based on input (perturbation) parameters and known linear relationships among system components	• Dependent on initial conditions and nature of multi-linked interactions among system components. Predicted over various time scales.
Signal Variability	• Random error	• Can contain temporal structure that reflects the organization of the underlying control system
Measurement Implications	• Error magnitude is proportional to the intensity of the perturbation or the severity of disruption in the feedback loop.	• Error magnitude is irrelevant. Temporal structure is measured as patterns of variability.

Table 2. Limitations of the Biomechanical Approach to Measuring Postural Steadiness

Limitation	Effect
• No external perturbations applied	• Unable to apply linear dynamics measurement framework based on input / output relationships
• Signal variability summarized using an “average” statistic (e.g., standard deviation)	• Averaging suppresses temporal structure of variability
• No evidence to support true static equilibrium as an achievable or desirable goal	• Poor content validity
• No external criterion (“gold standard”)	• Poor criterion validity
• Lack of reliable and valid sway determinants for evaluating pathological changes	• Poor predictive validity

Figure 1

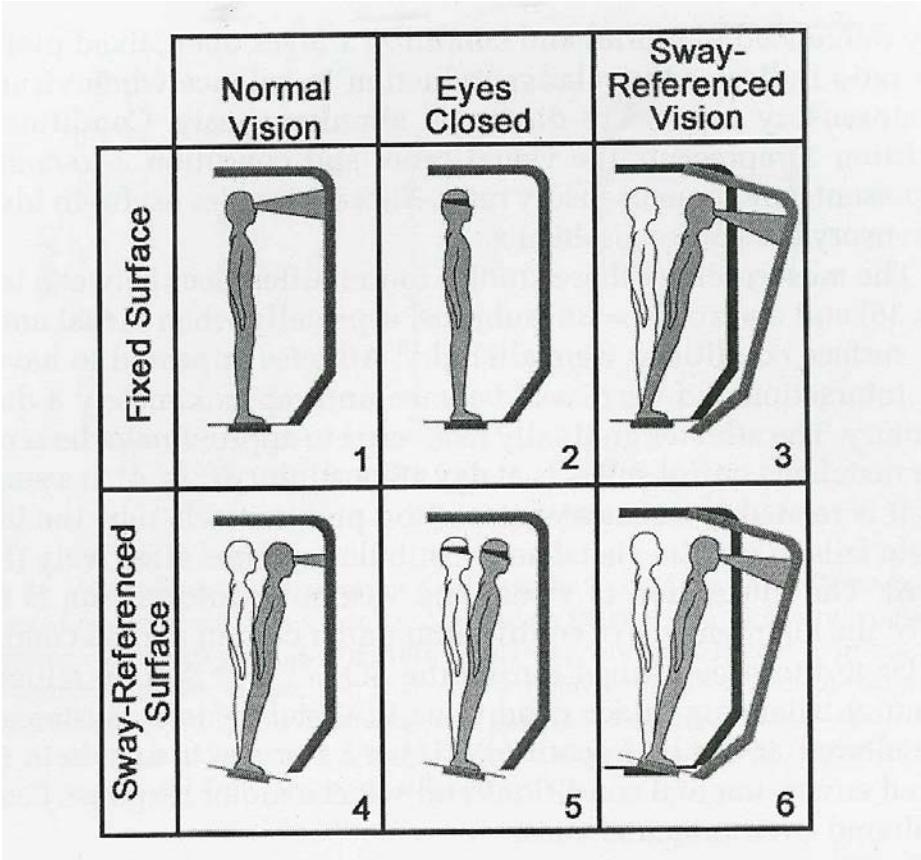
Normal Vision
Eyes Closed
Sway-Referenced Vision
Fixed Surface
Sway-Referenced Surface
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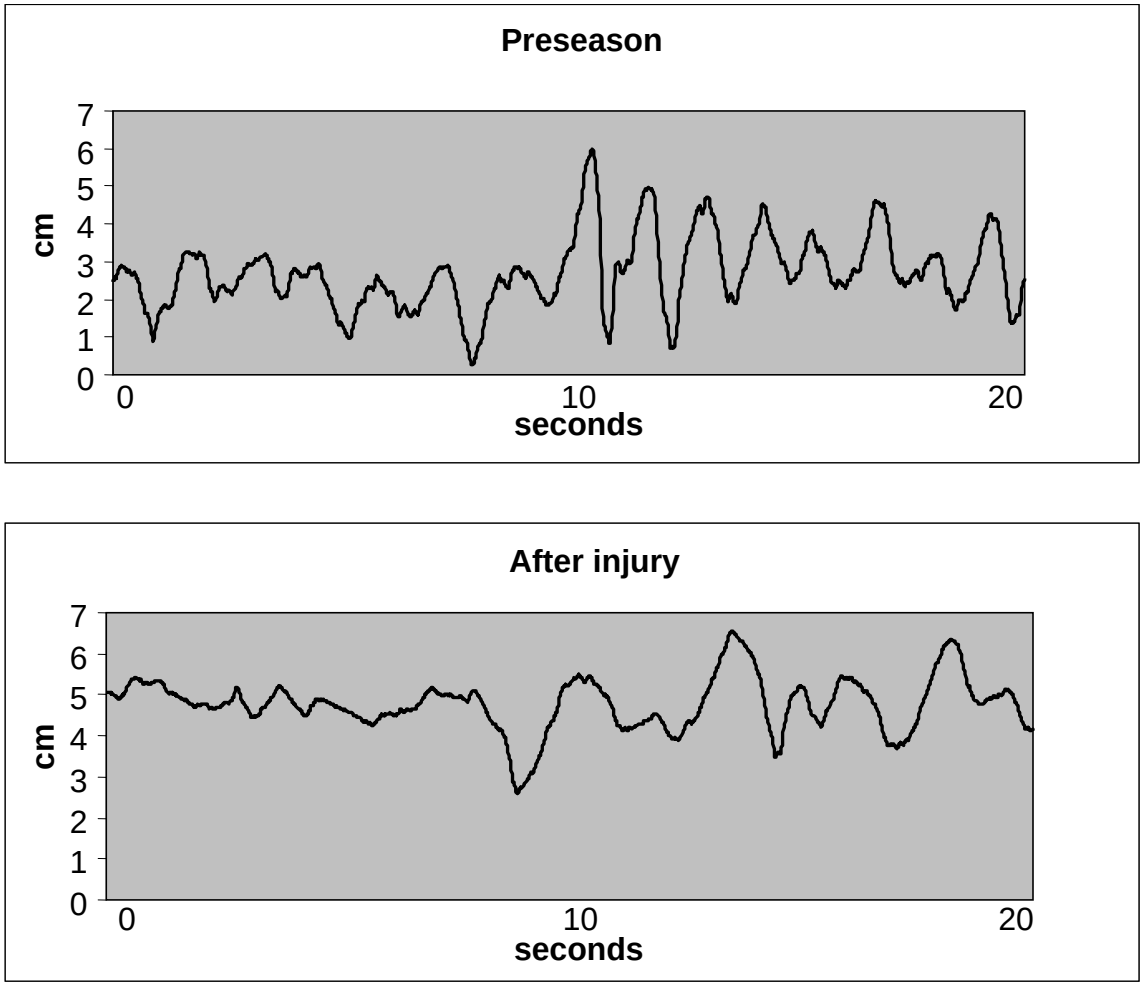
Figure 2.


Preseason
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A Nonlinear Dynamic Approach for Evaluating Postural Control
Cavanaugh, JT, et al.

Additional Amendments

1. Page 8, Figure 1. Caption should read as follows:

   "Sensory Organization Test – Six Conditions. Used by permission NeuroCom® International, Inc."

   Note: No manuscript citation is required for this figure.

2. At the time we prepared this manuscript, Figure 2 was original. Since then, however, a second manuscript containing a more complex version (4 panels instead of 2) of the figure has been accepted for publication in *BJSM*. That manuscript describes one of our concussion studies in full detail. Because I have already approved the final proof of the *BJSM* article, I feel obligated to request permission from the BJSM editor to adapt that figure for use in *Sports Medicine*. Please rest assured that I will fax or email a copy of the permission to you as soon as it arrives.

3. Amended sentence (underline for editorial use only): "Importantly, however, ApEn values for COP ML time series remained depressed, even among athletes whose initial postural instability had resolved.

4. Please add: "The authors have no conflicts of interest that are directly relevant to the content of this manuscript."

5. Reference #11 should read:

   Cavanaugh JT, Mercer VS, Guskiewicz K. Response stability estimates for the Sensory Organization Test: Equilibrium Scores and Approximate Entropy values in healthy young adults. *Gait & Posture.* 2004;20(Supplement 1):S55

6. Reference #12 should read:

   Cavanaugh JT, Mercer VS, Guskiewicz K. Effect of a secondary cognitive task on the temporal structure of postural control: implications for the dual task paradigm. *Gait & Posture.* 2004;20(Supplement 1):S54

7. Reference #15 should read:

   Cavanaugh JT, Guskiewicz K, Giuliani C, Marshall SW, Mercer VS, Stergiou N. Detecting altered postural control after cerebral concussion in athletes without postural instability. *BJSM.* 2005 (In Press).

8. The citation reflects the extent of the information listed on the header of the referenced article. Only the year of the symposium (1983) is listed.

9. Reference #35 should read:

   Hasan SS, Robin DW, Shiavi RG. Drugs and postural sway. *IEEE Eng in Med Biol Mag.* 1992;11(4):35-41

10. The dates of the symposium: June 13-15, 1989. APTA = American Physical Therapy Association

11. Reference #59 should read:

    Glass L, Mackey MC. *From Clocks to Chaos: The Rhythms of Life*. Princeton, NJ: Princeton University Press; 1988

12. Reference #91 should read:

    Cavanaugh JT, Guskiewicz K, Stergiou N. New insights into the recovery of postural control after cerebral concussion. *JOSPT.* 2005;35(1):A77-78