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TITLE: Optimal Movement Variability: A New Theoretical Perspective for Neurologic Physical Therapy

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ABSTRACT

Variability is a natural and important feature of human movement. Using existing theoretical frameworks as a foundation, we propose an alternative perspective to explain movement variability as it relates to motor learning and health; to wit, we propose that mature motor skills and healthy states are associated with an optimal amount of movement variability. This variability also has form and is characterized by a chaotic structure. Less than optimal movement variability characterizes biological systems that are overly rigid and unchanging, whereas greater than optimal variability characterizes systems that are noisy and unstable. Both situations characterize systems that are less adaptable to perturbations, such as those associated with abnormal motor development or unhealthy states. From our perspective, the goal of neurologic physical therapy should be to foster the development of this optimal amount of movement variability by incorporating a rich repertoire of movement strategies. The development of such a repertoire can be enhanced by incorporating a multitude of experiences within the therapeutic milieu. Promoting complex variation in human movement allows either motor development or the recovery of function after injury not to be hard coded, but determined instead by the active engagement of the individual within their environment. Measurement tools that characterize both the amount and complexity of movement variability provide useful means of testing these propositions. To illustrate, we present two clinical case studies, one pediatric and one adult, where we applied our theoretical framework to measuring change in postural control.

INTRODUCTION

Human movement variability can be described as the normal variations that occur in motor performance across multiple repetitions of a task. Variability is inherent within all biological systems, reflects variation in both space and time, and is easily observed. When we throw darts, for example, we are unable to hit the "bull's eye" on every attempt. When we walk, our footprints (e.g., as observed in sand or snow) never repeat themselves exactly. When we stand quietly, we continuously sway around a central equilibrium point without ever remaining exactly still. For some theorists, movement variability can be attributed to random error (i.e., noise).[1] Others suggest that movement variability is not entirely random, and accordingly, may contain important information.[1,2] Using these traditional perspectives as a foundation, the purpose of this paper is to propose a new theoretical model, illustrated with case examples, which we believe broadens the understanding of movement variability and has implications for pediatric and adult neurologic physical therapy.

Traditional Perspectives on Movement Variability

The motor control literature to date contains a variety of perspectives on movement variability. Some consider variation in a given movement pattern to be the result of errors in the ability to predict the necessary parameters for employing the underlying motor program.[3,4] With task-specific practice, prediction errors are gradually eliminated, thereby optimizing the accuracy and efficiency of the movement pattern. Others propose that biological systems self-organize according to environmental, biomechanical, and morphological constraints to find the most stable solution for producing a given movement.[5,6] Increased variability in a movement pattern generally indicates less cooperative behavior between the components of the underlying control system. Decreased variability generally indicates highly stable and cooperative behavior.

Such traditional perspectives are complementary, in that they both recognize that decreased variability results from the efficient execution of a given movement pattern. They also recognize that changing behavioral states may be characterized by increased variability, until a more stable (less variable) movement pattern can be adopted. This proposition implies that a persistent lack of movement

variability in the presence of changing task demands or environmental conditions may indicate rigid, inflexible motor behaviors with limited adaptability.

In our opinion, traditional perspectives do not sufficiently account for the observation that some behaviors, which appear to be stable, paradoxically are performed in variable ways. This is especially evident when we observe elite sports players or musicians performing complex activities (i.e. Michael Jordan taking a jump shot or Yo-Yo Ma playing the cello). Not only is their performance more consistent than that of less capable individuals, but they also seem to have developed an infinite number of ways of performing. Thus, they display a very stable behavioral state underlined by a "rich" behavioral repertoire. If we consider fundamental motor skills (i.e. gait, posture) as complex activities when applied in "real life" contexts, we can actually say that every single one of us is a Michael Jordan in our abilities to walk through crowds or on diverse and challenging terrains. Therefore, it seems that in this sense, variability does not decrease, but rather increases, when we further develop and refine a stable behavioral state.

The idea that variability decreases with skill acquisition in one context (motor learning paradigm) and increases with skill acquisition in another context (the development of a behavioral repertoire) is readily explained by the way in which variability is measured. Typical motor learning curves are constructed using traditional variability measures of skill performance (e.g., standard deviation). Such linear statistical tools quantify the magnitude of variation in a set of values independently of their order in the distribution. The amount of variability continuously decreases and eventually plateaus as motor learning occurs. In contrast, variation in how a motor behavior emerges in time is best captured by tools derived from nonlinear dynamics, for which the temporal organization in distribution of values is the facet of interest. Temporal organization (or "structure") is quantified by the degree to which values emerge in an orderly (i.e., predictable) manner, often across a range of time scales.

The following arguments highlight the distinction between linear and nonlinear measures of variability and provide a strong rationale for applying nonlinear tools to human movement:

1) Typically, in human movement research, kinematic (and/or kinetic) data from several trials are averaged to generate a "mean" picture of the subject's performance or movement pattern. In this

averaging procedure, which is frequently accompanied by normalization, the temporal organization of the pattern is lost.

2) From a statistical standpoint, the valid usage of traditional linear tools to study variability assumes that variations between repetitions of a task are random and independent (of past and future repetitions). However, recent studies[7-11] have shown that such variations are distinguishable from noise and have a deterministic origin (i.e., are produced from lawful interactions among underlying control system components). Thus, they are neither random nor independent.

3) Traditional linear tools provide different answers when compared with nonlinear tools regarding stability of a movement pattern.[8,12]

4) The plurality of human movement patterns and the multitude of motor control feedback loops make movement similar in many respects to other physiological life rhythms (e.g. heart beat), for which variability has been described as exhibiting deterministic dynamics.[13] The underlying fractal-like morphology of many structures of human physiology (lungs, neurons, etc.) increases the likelihood that human movement patterns are controlled by such dynamics.[14]

The fourth argument above requires further elaboration and emphasis. Mathematical techniques from chaos theory (i.e., nonlinear tools) have demonstrated that temporal variations in biological signals, even though they appear no different from random noise, actually exhibit deterministic patterns. These patterns have been defined as "Chaotic" (Figure 1) and can have important implications for clinical medicine. For example, heart rhythms in which the variation in the time interval between subsequent QRS waves is either periodic or random have been associated with heart attacks.[13] Conversely, chaotic heart rhythms are related to healthy states. Similar results have been found in other biological signals such as white blood cells counts, blood pressure control, movement tremor, and gait.[1,7,9,15,16] One interpretation of these phenomena is that chaotic temporal variations in the steady state output of a healthy biological system represents the underlying physiologic capability to make flexible adaptations to everyday stresses placed on the human body.[16,17,18] Accordingly, a reduction or deterioration of the chaotic nature of these temporal variations of a biological signal represents a decline in the "healthy flexibility" within the

underlying control system that is associated with behavioral rigidity and inability to adapt to stresses.[16,17,18]

INSERT FIGURE 1 HERE

An Alternative Theoretical Model of Human Movement Variability

Building on the ideas presented above, we propose an alternative theoretical framework to explain movement variability as it relates to health and motor learning. Proposition #1: We propose that there is an optimal amount of variability in a biological system that is directly associated with health. This variability also has form and is characterized by a highly complex, chaotic structure (Figure 2). Decrease or loss of this optimal amount of variability will make the biological system more rigid. Increase beyond optimal variability will make the system more noisy and unstable. Both situations render the system less adaptable to perturbations and are directly associated with lack of health. Thus, stable yet adaptable systems maintain a rich repertoire of movement strategies containing optimal variability. In Figure 2, we illustrate this point by incorporating on the y-axis the concept of complexity. We believe greater complexity is associated with a rich behavioral state, for which system output is characterized by a chaotic structure. Lesser amounts of complexity are associated with both periodic and random states where the system is either too rigid or too unstable. On the x-axis we have implemented the notion of predictability. Low amounts of predictability are associated with a random and noisy system, while high amounts are associated with a periodic highly repeatable and rigid behavior. In between is a chaotic based behavior where the system in neither too noisy nor too rigid. A similar approach has been used by Tononi et al[19] to relate complexity with integration of information in the brain and cognitive coherence. In our proposition, this model is used to explain movement variability as it relates to health and motor learning.

INSERT FIGURE 2 HERE

Proposition #2: We propose that human motor development and motor learning processes obey our model of optimal variability as individuals develop healthy and highly adaptable motor systems. In this context, abnormal development may be characterized by a narrow range of behaviors, some of which

may be rigid, inflexible, and highly predictable (i.e., stereotypical), or alternatively, random, unfocused and unpredictable. Motor disabilities many times are described as such.

Proposition #3: We propose that the goal of neurologic physical therapy should be to foster the development of this optimal amount of movement variability by incorporating a rich repertoire of movement strategies. Incorporating a multitude of experiences within the therapeutic milieu can enhance the development of such a repertoire. Promoting complex variation in human movement allows motor development or the recovery of function after injury not to be hard coded, but determined instead by the active engagement of the individual within their environment.

To test the above propositions, appropriate methods for measuring both the amount and complexity of variability should be utilized. To illustrate, we present two clinical case studies, one pediatric and one adult, in which we applied our theoretical framework to measuring change in postural control. The first case describes how our approach was applied to physical therapy intervention for an infant with cerebral palsy that had problems with sitting postural control. The second case describes how nonlinear methods were applied to detect subtle changes in postural control in an athlete with sport-related cerebral concussion. For a more detailed description of the methods used, the interested reader is directed to a review by Stergiou et al.[15]

CASE STUDIES

CASE # 1: Physical therapy intervention for an infant with cerebral palsy

Description: Infants with cerebral palsy have problems with postural control and movement. In spite of considerable investigation, there are many problems in diagnosing movement disorders in infancy and in determining whether treatment is efficacious. Standard neurologic tests that focus on passive examination of a child using reflex testing, imaging, and observations are inadequate for assessing the development of postural control. This failure of testing methods results in missed opportunities for referral for early developmental intervention. Although sitting skills are a part of every motor assessment for infants, none of these tests are sensitive enough to detect small changes in motor control. A measure for increasing

increments of postural control and the variability of this control would indicate responsiveness to specific treatment techniques and improving motor control.

Improved evaluation of early postural control, such as control of sitting, would provide important information about emerging postural abilities and the evolution and adaptive nature of postural control in childhood. In addition, sensitive methods for evaluating the effects of treatment for these infants are needed so that the most effective treatment methods can be disseminated to therapists and provided to infants in early intervention programs. There is insufficient research supporting the efficacy of physical therapy for remediating movement disorders in children.[20,21] In fact, studies measuring clinical outcomes of the most commonly used therapeutic approach, neurodevelopmental treatment, have been inconclusive regarding efficacy, with resulting recommendations to investigate other therapy approaches that may prove more beneficial.[22]

METHOD

Subject: JK was referred for early intervention at the age of 8 months of age because of delays in motor development and increased muscle tone in his right upper and lower extremities, indicating a diagnosis of right spastic hemiplegic cerebral palsy. He was born at term and was otherwise healthy. Developmental tests indicated a significant delay; he was not rolling or sitting independently, and he displayed a severe neglect of his right arm. Recommendations were that he should receive therapeutic services through a transdisciplinary home-based program. However, the family moved out of the school district in which he was evaluated and did not immediately seek services in their new district. Physical therapy intervention finally began at 1 year of age and was instituted using a medical model of therapy provided in a clinic situation, twice weekly for 45 minute sessions. Participation in the research program to study the development of sitting postural control in infants with cerebral palsy was instituted, and parental consent was obtained in accordance with the guidelines of the Institutional Review Board of the University of Nebraska Medical Center. JK had additional problems in the following areas: esotropia of the right eye, aversion to touch on his right hand, and decreased variety of vocalizations. At the time he began physical therapy, he was able to maintain the prop sitting position when placed but would fall backwards or to one

side if left unattended. He disliked the prone position and was able to roll out of prone toward the right side. He preferred to stay in physical contact with his mother and did not play independently on the floor.

Procedure: Evaluation for the research protocol included testing using the Peabody Developmental Motor Scales[23] to verify significant motor delay. He scored at the 3rd percentile in the gross motor component of the Scales for his age, which was considered a significant delay and qualified him to participate. Center-Of-Pressure (COP) measures in sitting were calculated from data sampled at 200Hz from an AMTI force platform. A frequency analysis of both the medial-lateral and anterior-posterior components of all the COP time series from preliminary data indicated that the range of signal frequencies that contained 99.99% of the overall signal power was between 1 and 29 Hz. Therefore, the sampling frequency was set at 200 Hz to allow 10 seconds of quiet sitting behavior for a total of 2000 points for each COP coordinate. This number is considered adequate for nonlinear analysis.[15] The data were analyzed unfiltered for a more accurate representation of the variability within the system.

For each data collection session, JK sat quietly on the force platform while being guarded by the therapist and his mother. Three acceptable trials were selected from the video record using the following criteria: 1) infant was not moving his arms (not reaching, holding an object, or flapping their arms); 2) infant was not vocalizing or crying; 3) infant was not in the process of falling; 4) trunk was not more than 45 degrees to either side. Data were collected at the beginning of treatment, after 8 treatment sessions, and at the end of 16 treatment sessions.

Both linear and nonlinear measures of the COP were used in evaluating postural control. Linear measures included: circle area (mm^2), which is a measure of the circular area that the length of path of the COP covers; range medial-lateral (mm) and range anterior-posterior (mm), which are measures of excursion of the COP in the respective directions. Nonlinear measures included: Approximate Entropy (ApEn), a regularity statistic that quantifies the amount of randomness in a time series; and Lyapunov Exponent (LyE), which is a measure of stability, in both the anterior-posterior and medial-lateral directions for each measure.[8,15] The linear measures generally decrease as postural control increases,[24] a finding from research on standing children and adults. This decrease of linear measures is attributed to a

decrease in variability, which, in the past, has been considered to be a marker of skill development. However, Harbourne and Stergiou,[8] in an examination of the development of sitting comparing linear measures to nonlinear measures, found that linear measures did not change significantly during the development of sitting from prop sitting to independent sitting in typically developing infants. Nonlinear measures, however, did change significantly over time. LyE showed decreasing values, which indicates increasing stability over time from prop sitting to independent sitting, and ApEn first decreased significantly from prop sitting to beginning independent sitting, then increased again when the infants were completely independent in sitting.

The treatment received by JK was based on the approach of the Tscharnuter Akademie for Movement Organization (TAMO).[25,26] This treatment approach provides a rich perceptual environment in which the infant discovers, through active exploration, strategies that are valuable for functional success.[27] Thus, it is consistent with our Proposition #3. The following are principles of treatment utilized in a play environment with the infant: (1) all movements are child initiated; the attention of child is necessary to the task so that the task has value to the child; (2) the environment is set up to require very small changes in skill; (3) the goal is to help the child gather information about possibilities for movement and adapt to very slight changes in force distribution; (4) errors are expected and allowed for learning to take place; (5) an increase in variability of movement is desired and encouraged.

RESULTS

After 16 treatment sessions within a two-month time period (average twice weekly, 45 minutes each time), JK was able to sit independently, reach forward for a toy and right himself, get into the sitting position by rotating from supine and pushing up on his left arm, and was just beginning to move forward over his legs to attain a quadruped position on hands and knees with the right hand remaining fisted. He had also begun pulling to standing at furniture and was using two hands for some reaching and lifting tasks during play.

Figure 3 displays the changes in the magnitude of COP displacements from the initial evaluation session to evaluation after 8 treatment sessions, and after 16 treatments. The values of a typically

developing infant are incorporated into the graph for comparison, as well as the values of another infant receiving a home program as an alternative intervention. The infant receiving the home program alternative treatment is further explained in the discussion section. In general, the linear measures of JK decreased from initial assessment to the assessment made after 8 treatments, which would traditionally be interpreted as a decrease in variability. However, after 16 treatment sessions, the linear measures were approximately equivalent to the measures of a typically developing infant with independent sitting skills at the age of 7-8 months.

INSERT FIGURE 3 HERE

Figure 4 displays changes in nonlinear measures from initial evaluation to the evaluation after 8 treatments and after 16 treatments. Although the linear measures seem to reveal a decrease in variability after 8 treatments, all nonlinear measures showed that complexity increased after 8 treatments. After 16 treatments, the nonlinear measures were essentially equivalent to the typically developing infant's independent sitting measures at 7-8 months of age. In contrast to a possible decrease in variability depicted by linear measures, the nonlinear measures indicated an increase over time in the rich behavioral potential of the system. In fact, JK was beginning many new behaviors such as pulling to stand, getting in and out of the sitting position, and making attempts to crawl on hands and knees.

INSERT FIGURE 4 HERE

Overall, both clinical observations and clinical testing indicated that JK had made progress in his sitting postural control over the period of 16 treatments. The remaining question is whether JK's progress was related to the treatment, or whether his skills in postural control would have developed without treatment. Another question is whether the linear or nonlinear measures were helpful in assessing progress in his sitting postural control. We will attempt to answer these questions in the discussion section.

DISCUSSION

This case demonstrates that nonlinear tools can be applied to measure the development of sitting postural control and can detect changes that are concurrent with functional skill changes. Although

changes in the linear measures indicate fluctuations in variability, changes in the nonlinear measures indicate increasing complexity of the postural control system towards normative values. An increase in complexity would be consistent with increasing the variety of postural and movement strategies available to the child and an emergence of novel skills displayed in sitting, such as the emerging abilities to get in and out of the sitting position, and reach outside of the base of support.

In an effort to determine the value of using nonlinear measures for determining progress and deciding which treatments are successful, it is necessary to compare JK to another similar child who is not receiving equal treatment. Although no two children can be considered exact equivalents, we can compare JK to another child of similar age, diagnosis and functional skill level.

LM was also 12 months of age when he started treatment. He also had a diagnosis of spastic hemiplegic cerebral palsy, and his affected side was the right side. At the time of evaluation he was able to prop sit, but tended to lose his balance to the back or to one side. All of the above are the same features as displayed by JK. However, instead of participating in twice weekly PT treatment sessions, LM received a home program that was checked weekly by a PT. Suggestions from published[28] home exercise programs were provided to the family along with demonstration. These suggested activities included various positioning techniques progressing toward sitting independence, and were of a more static nature than the twice weekly treatment. After 4 and 8 weeks of the home program, data was collected. LM showed changes in linear measures that followed the pattern of increasing variability at 4 weeks and decreasing variability after 8 weeks (Figure 3). This was the same pattern as seen in the typically developing infant; however, LM's values in the linear variables were less than JK's by the end of treatment at 8 weeks. The nonlinear variables showed that LM had the opposite pattern from JK. For LM, complexity decreased, then increased on all nonlinear variables, and after 8 weeks he was further from the values of the typical infant than JK. LM did begin to sit independently, and was able to reach further without loss of balance, but he was not showing the richness of new behaviors, such as getting in and out of sit or pulling to stand, as displayed in JK's behavioral repertoire.

If we use the nonlinear measures to help decide on the success of JK's episode of treatment, we can interpret the data to indicate that JK made greater gains because he showed an overall increase in complexity of postural control, which should lead to the discovery and emergence of new movement behaviors. One month after the post-treatment evaluation (one month after intervention ended) the two infants were displaying different movement skills. JK was crawling on hands and knees, cruising, transitioning in and out of sitting by rotating into quadruped to either side, and had taken a few independent steps. LM was attempting to get into the crawling position, but was unable to crawl. He could stand at a support surface, but was not able to cruise. The indication that less complexity was developing in the postural control of LM at the end of his episode of treatment could lead to the prediction that he did not have the richness and complexity of postural control that would result in the emergence of new movements.

The above comparison of the use of nonlinear tools for evaluation and assessment of treatment efficacy demonstrates the possibilities for using nonlinear methodology in the management of neurologic disorders in infancy. Propositions #1 and #2 of our model are supported by this example, in that increasing complexity reflects an increasing richness of behavioral options related to postural control in developing sitting. Proposition #3 is also supported, because the nonlinear measures provide a methodology for assessing complexity that is not possible with traditional variability measures. In fact, a comparison of the linear and nonlinear measures of JK's COP displacements in the anterior-posterior and medial-lateral planes indicates that changes in variability values on the linear measures are the opposite of the pattern seen in the nonlinear measures. In other words, a decrease in variability, such as noted in JK's measure of range medial-lateral from initial assessment to assessment after 1 month, is measured as an increase in complexity on the ApEn medial-lateral graph during the same time period. Complexity, as measured by ApEn and LyE, appears to reveal the potential for the emergence of adaptive movement skills. This pilot work has been expanded to a larger developmental study using nonlinear tools to examine infants with cerebral palsy and the effects of direct treatment using the TAMO approach versus a weekly home program.

CASE # 2: Detecting subtle changes in postural control in an athlete with cerebral concussion

Description

Sport-related cerebral concussion is a growing public health concern.[29] Athletes who return to competitive activity too early after injury are potentially more vulnerable to injury recurrence, the consequences of which can be catastrophic.[30] Complete recovery of postural control after cerebral concussion is an important determinant of an athlete's readiness to return to competitive activity. On average, athletes who initially present with postural instability after concussion return to their baseline level of performance within 3-5 days of injury.[29] Variable recovery rates, combined with the challenge of objectively measuring subtle physiologic impairments, often make this task difficult and highlight the critical need for developing more sensitive clinical tools for assessment of complete recovery of postural control.

METHOD

Subject: The individual featured in this case example was an 18 year old male collegiate soccer player, "SP," who sustained a cerebral concussion. Measuring 170 cm in height and weighing 67.6 kg, SP had no history of previous concussion and prior to injury had no reported injuries or medical conditions that interfered with his ability to participate in competition. Concussion was defined as injury to the brain caused by a sudden acceleration or deceleration of the head that resulted in any immediate, but temporary, alteration in brain functions, such as loss of consciousness, blurred vision, dizziness, amnesia, or memory impairment.[31] The presence of concussion was determined during medical evaluations at the time of injury by a certified athletic trainer and a neurologist. As an athlete at risk for head injury, SP had been enrolled in a formal concussion surveillance protocol and had signed a consent form in accordance with the Academic Affairs Institutional Review Board at the University of North Carolina at Chapel Hill.

Procedure: Based on the concussion surveillance protocol, postural control and symptom data were collected from SP at the beginning of the soccer season and once again approximately 1 month later on each of the four successive days after his injury. Postural control was evaluated during the Sensory Organization Test (SOT) using the Smart Balance Master System (NeuroCom International, Inc.,

Clackamas, OR, USA). The system was equipped with a moveable visual surround and support surface that could rotate in the AP plane. Two 9 x 18 inch force plates connected by a pin joint were used to collect COP coordinates at 100 Hz. The SOT consisted of 18 total trials, each lasting 20 seconds, in which SP was instructed to stand with his arms relaxed at his sides, to look straight ahead, and to stand as still as possible without reaching out to touch the visual surround or taking a step. SP wore comfortable attire and was shoeless during testing. Foot placement was standardized based on subject height according to the manufacturer's protocol. The trials were conducted in 3 groups of six each. Each group contains one trial from a different sensory condition (Figure 5). The SOT required approximately 15 minutes to conduct. For the first group of trials, sensory conditions were presented in ascending order (1 to 6). For the second and third groups, sensory conditions were presented randomly.

INSERT FIGURE 5 HERE

A checklist of 17 symptoms commonly associated with concussion was read to SP during each testing session. The list included headache, nausea, vomiting, dizziness, poor balance, sensitivity to noise or light, ringing in the ear, blurred vision, difficulty concentrating or remembering, trouble falling asleep, drowsiness, fatigue, sadness, irritability, and neck pain. At the preseason assessment, SP was instructed to rate the severity of symptoms experienced more than 3 times per week. For the post-injury assessments, SP was instructed to rate his current symptoms. Symptom severity was rated using a 7-point Likert scale, with 0 corresponding to "none" and 6 corresponding to "severe" symptoms. For each testing session, the number of symptoms rated greater than 0 was counted and an aggregate total rating score was calculated.

Data Reduction: Using Matlab software (Mathworks, Natick, MA), we calculated separate ApEn values for the AP and ML components of the COP coordinate time series (N = 2000) from test trials.[15] According to accepted guidelines,[32] average ApEn values for COP time series collected during two trials of the SOT have demonstrated good to moderate between-session response stability for the AP (ICC(2,2) range 0.79 - 0.90) and ML (ICC(2,2) range 0.53 - 0.77) components of COP time series.[33] A surrogation (phase randomization) procedure was conducted as a pre-processing step to identify if the COP data were derived from a deterministic source.[15] ApEn values from the original data and their surrogated

counterparts were compared using the Student t-test ($\alpha = .05$). We found significant differences between all original COP time series and their surrogate counterparts, indicating that the original data were not randomly derived. This confirmation was a necessary component of nonlinear dynamics methodology.[15]

An Equilibrium Score was generated for each trial in each condition based on the algorithm developed for the Smart Balance System.[34] The algorithm uses the peak-to-peak amplitude of COP AP displacement to estimate the amount of postural sway in the sagittal plane. Scores are calculated as the angular difference, expressed as a percentage, between the amount of estimated AP postural sway and the theoretical limit of stability (approximately 12.5º in the AP plane). A lower amount of postural sway requires smaller amplitude of COP displacement to control and produces a higher percentage difference from the theoretical limit. Thus, higher scores indicate greater postural stability. Like the ApEn values, Equilibrium Scores from the first and second trials from each sensory condition were averaged for further analysis. A separate, Composite Equilibrium Score was calculated by independently averaging all trial scores from Conditions 1 and 2, adding these two average scores to the individual trial scores from Conditions 3-6, and then dividing the sum by 14.[34]

RESULTS

At preseason, SP had a Composite Equilibrium Score of 84, consistent with normal postural stability in comparison to data, supplied by the manufacturer, from healthy age-matched subjects. The Composite Equilibrium Score, as well as the ES generated as the simple average from the first two SOT trials (Figure 6), corresponded to relatively low amplitude COP excursion consistent with a healthy state. Preseason ApEn values across the six SOT conditions indicated a moderate amount of complexity for both AP (mean = 0.6532; range = 0.3485 - 0.9156) and ML (mean = 0.8966; range = 0.6872 – 1.11) time series (Figure 6). Also at preseason, SP reported no symptoms listed on the post-concussion symptom checklist (Figure 7).

INSERT FIGURES 6 AND 7 HERE

Cerebral concussion appeared to influence the magnitude and dynamics of SP's COP excursions. For example, Figure 8a illustrates COP AP time series collected during SOT condition 1 (stable platform

and surround, eyes open). At preseason, the maximum range of COP excursion was 1.43 cm, resulting in a single trial ES of 94. In comparison, on the first day after concussion, the maximum range of COP excursion was 3.17 cm, producing an ES of 86. During the same interval, the ApEn value for the COP time series declined from 0.7728 at preseason to 0.5226 after concussion. This loss of complexity appears as a smoother, less erratic, more predictable pattern contained in the post-concussion COP tracing. A similar yet distinct example appears in Figure 8b, in which the maximum range of COP excursion during SOT condition 2 (stable platform, eyes closed) paradoxically decreased from 0.84 cm to 0.7 cm from preseason to after concussion (suggesting an increase in ML stability), while the ApEn values decreased from 1.03 to 0.6787 (suggesting a loss of ML time series randomness). Once again, the relatively lower complexity content of the post-concussion time series appears closer in form to a pure sine wave (ApEn value of 0.0; Figure 1).

INSERT FIGURE 8 HERE

With each subsequent day after concussion, SP displayed changes in postural control (Figure 6) and concussion-related symptoms (Figure 7). As expected,[29] postural stability as defined by average ES, returned to near preseason levels by post-concussion Day 4. During the same recovery period, SP reported approximately 10 symptoms consistent with cerebral concussion, while total symptom severity diminished from a high of 30 on Day 2 to 14 on Day 4. In contrast, post-concussion ApEn values for both COP AP and ML time series remained depressed and became progressively more disparate from preseason values.

DISCUSSION

The case of SP illustrates several important points regarding the application of nonlinear dynamics measurement tools to human postural control data and to the recovery of postural control after cerebral concussion. First, as determined through a surrogation procedure, COP data collected from SP during the SOT using the Smart Balance System contained deterministic structure. This simple yet important fact has been replicated in both healthy and impaired subjects[33, 35-37] and is a necessary requirement to apply nonlinear tools to any human movement time series. Second, ApEn shows promise

as a useful tool for detecting subtle changes in postural control. This proposition also has been supported in larger studies of athletes with concussion[35,36] and highlights the clinical relevance of supplementing traditional measurement tools of postural stability, like ES, with tools, like ApEn, from a nonlinear theoretical framework. Finally, the case of SP reinforces the notion that determining an athlete's readiness to resume competitive activity after cerebral concussion requires caution and a variety of measurement tools applied serially across multiple domains.

The distinction between recovery curves for ES and ApEn values underscores the differences in their theoretical foundation. ES is based on the traditional concept of generalized motor programs that the amplitude of COP excursion provides a valid indication of postural stability. In this view, COP fluctuations around a central equilibrium point are considered as random error. Presumably, cerebral concussion increases system error and reduces the precision with which body position can be maintained in quiet standing. In contrast, the application of nonlinear methodology (ApEn and surrogation) confirmed that COP variation was not random. Indeed, ApEn, as a measure of predictability, has been thought of as an indicator of postural control system constraint,[38] and by extension, it can be considered as a measure of system complexity. In this framework, constraint refers to any internal or external factor that restricts natural postural sway. More complex patterns of sway fall in between those that are entirely constrained and predictable (e.g., a sine wave) and those that are entirely unconstrained and random (e.g., Gaussian noise) as described in our Proposition #1.

The decline in ApEn values for SP after concussion may have been related to changes in neurophysiological or mechanical constraints on postural control but were likely not related to changes in post-concussion symptoms.[36] Diffuse axonal injury, for example, may have reduced or distorted interactions among neurons in the brain,[39] thereby affecting the regularity of cortical oscillations[40] that were subsequently manifested in a loss of complexity (increased predictability) in patterns of COP oscillation. Alternatively, increased co-contraction of lower extremity musculature, generated by injured athletes in an attempt to gain control over postural sway, may also have reduced the complexity of COP oscillations. Regardless of the explanation, the positive relationship between ApEn Values and

Equilibrium Scores indicated that larger amplitude COP oscillations (diminished postural stability reflected in a lower Equilibrium Score) tended to be more predictable (lower ApEn), whereas lower amplitude COP oscillations (better postural stability reflected in a higher Equilibrium Score) tended to be more complex (higher ApEn). It appears, therefore, that optimal postural control in quiet standing is characterized by COP displacements that are not only low in amplitude (low variability) but also are relatively complex.

SUMMARY

Using traditional perspectives as a foundation, we have proposed a new theoretical model to explain movement variability as it relates to motor learning and health. Our model is based on the idea that mature motor skills and healthy states are associated with an optimal amount of movement variability that reflects the adaptability of the underlying control system. This variability has form and is characterized by a chaotic structure. Less than optimal movement variability characterizes biological systems that are overly rigid and unchanging, whereas greater than optimal variability characterizes systems that are noisy and unstable. Both situations characterize systems that are less adaptable to perturbations, such as those associated with abnormal motor development or unhealthy states. The model supports the proposition that the goal of neurologic physical therapy should be to foster the development of this optimal amount of movement variability by incorporating a rich repertoire of behavioral strategies. Promoting complex variation in human movement allows either motor development or the recovery of function after injury not to be hard coded, but determined instead by the active engagement of the individual within their environment. The model also offers a new set of measurement tools, derived from nonlinear analysis, with which therapists can measure changes in movement variability as their patients improve with intervention or decline in the presence of pathology.

Our approach is not intended to replace the wide array of traditional measurement tools available for physical therapists; nor are we suggesting that traditional approaches to understanding and measuring change in motor control are inferior. On the contrary, we recognize that the field of movement science is moving forward to more rigorously explore movement variability using alternative methodology.

Concurrent with this change, we have provided two clinical case illustrations of how our theoretical framework can be applied in a clinical setting to gain novel and important insights into changes in movement control. Future research and clinical applications ultimately will determine the extent to which our approach and the associated measurement tools can be used to guide intervention and improve patient outcomes.

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FIGURE LEGENDS

Figure 1: At the top panel we graph a time series from a simple periodic function (i.e. a sine wave) and the corresponding phase plane plot which is practically the amplitude (i.e. position) of the time series versus its first derivative. At the middle panel we graph a time series from a chaotic system, the Lorenz attractor, and the corresponding phase plane plot. At the bottom panel we graph a time series from random numbers (with a Gaussian distribution centered on zero and a standard deviation of 1.0) and the corresponding phase plane plot.

Figure 2. An illustration of the theoretical model proposed using the phase plane plots from Figure 1.

Figure 3. Linear measures comparing infant sitting COP. Three infants are represented: a typically developing infant at 5, 6 and 7 months of age; an infant with right hemiparesis who was treated twice weekly for 8 weeks using the TAMO approach; and an infant with right hemiparesis who was managed with a once weekly home program consultation for 8 weeks. The top graph shows the circular area of the path of the COP in mm^2; the middle graph shows the excursion of the path of the COP in the anterior/posterior direction, and the bottom graph shows the excursion of the COP in the medial/lateral direction. Values are the average of 3-6 trials at each monthly data collection period.

Figure 4. Nonlinear measures comparing infant sitting COP. The same three infants from Figure 3 are represented. The top graph depicts Approximate Entropy (ApEn) measures in the anterior/posterior direction; the next graph depicts ApEn in the medial/lateral direction; the third graph indicates measures on the Lyapunov Exponent (LyE) in the anterior/posterior direction, and the bottom graph is the LyE in the medial/lateral direction. Values are the average of 3-6 trials at each monthly data collection.

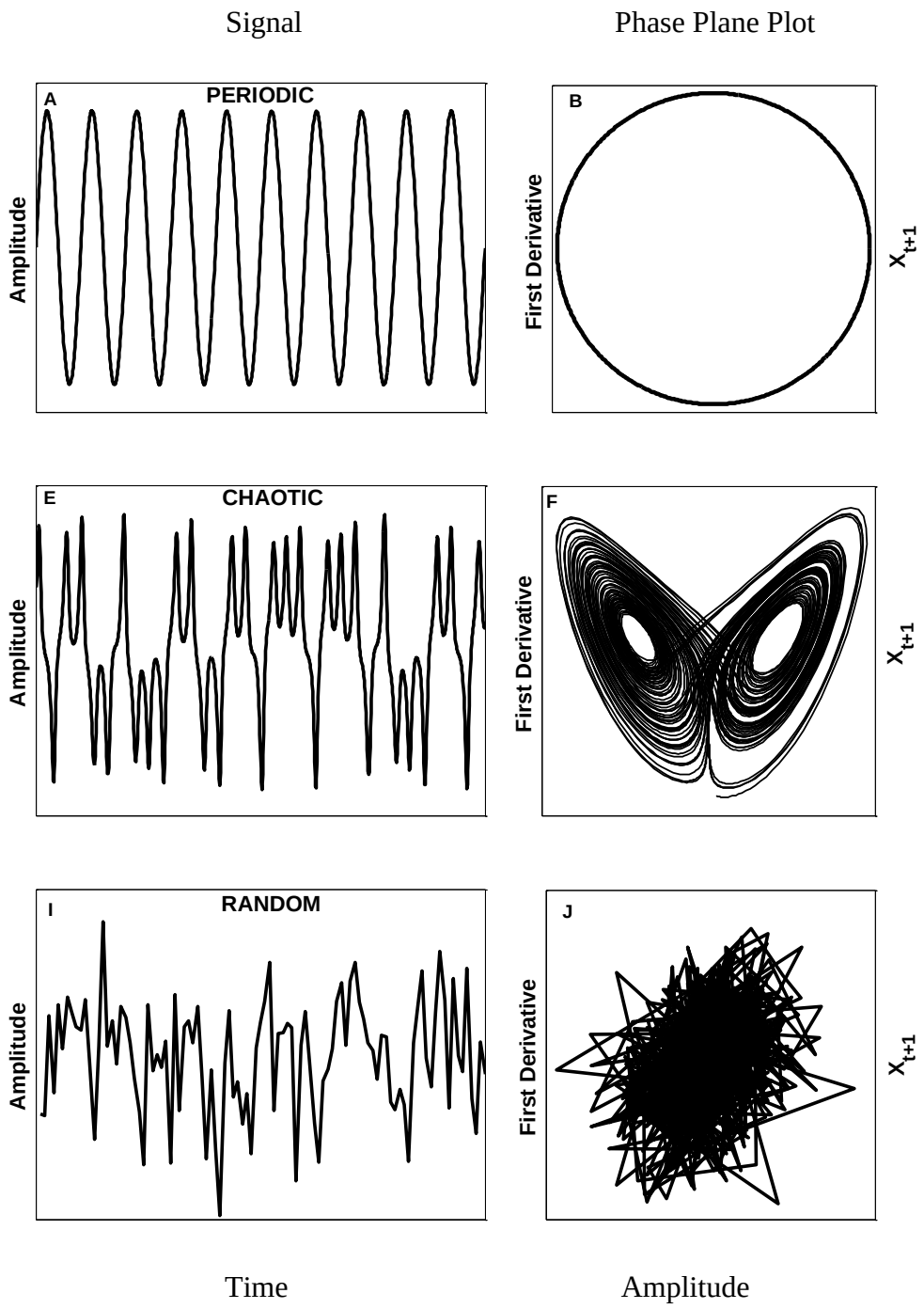
Figure 5. The six testing conditions for the Sensory Organization Test (reprinted with permission from NeuroCom International, Inc.). Vision is absent in conditions 2 and 5. In conditions 3 and 6, the sway-referenced AP angular motion of the surrounding wall reduces optic flow stimulation useful for the perception of self-motion relative to the visual field. In conditions 4-6, sway-referenced angular motion of

the force plates reduces somatosensory stimulation useful for the perception of AP self-motion relative to the support surface.

Figure 6. Recovery of postural control after cerebral concussion. Average Approximate Entropy (ApEn) values (left ordinate scale) are represented by dashed lines for both AP and ML time series. Equilibrium Score (ES) values (right ordinate scale) are represented by a solid line. ApEn and ES values are averages based on 2 test trials from each of 6 Sensory Organization Test (SOT) conditions. Note that while ES returns toward Preseason values by Day 4 post-concussion, ApEn values remain depressed.

Figure 7. Number of self-reported concussion-related symptoms and total symptom severity score at preseason and post-concussion.

Figure 8. Center of pressure (COP) displacement time series recorded at preseason and one day after cerebral concussion. Figure 8a. COP anterior-posterior time series recorded during Sensory Organization Test (SOT) condition 1. Preseason values: Equilibrium Score (ES) = 94; Approximate Entropy (ApEn) = 0.7728. Day 1 post-concussion values: ES = 86; ApEn = 0.5226. Figure 8b. COP medial-lateral time series recorded during SOT condition 2. ApEn = 1.03 at preseason and 0.6787 at Day 1 post-concussion. Consistent with reduced ApEn values, post-concussion COP time series appear smoother and less erratic.


Signal
Phase Plane Plot
A
PERIODIC
Amplitude
B
First Derivative
X_{t+1}
E
CHAOTIC
Amplitude
F
First Derivative
X_{t+1}
I
RANDOM
Amplitude
J
First Derivative
X_{t+1}
Time
Amplitude

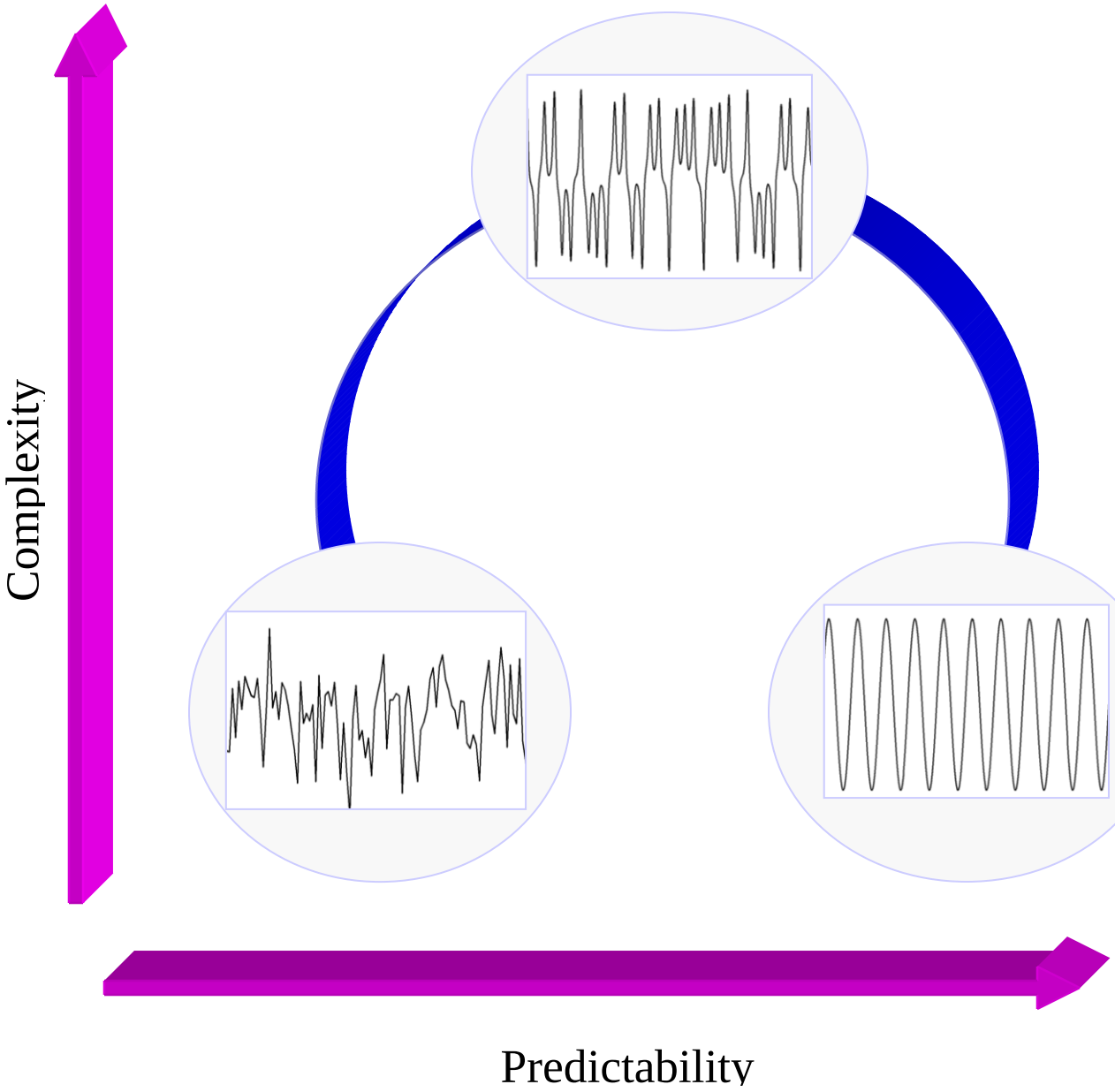
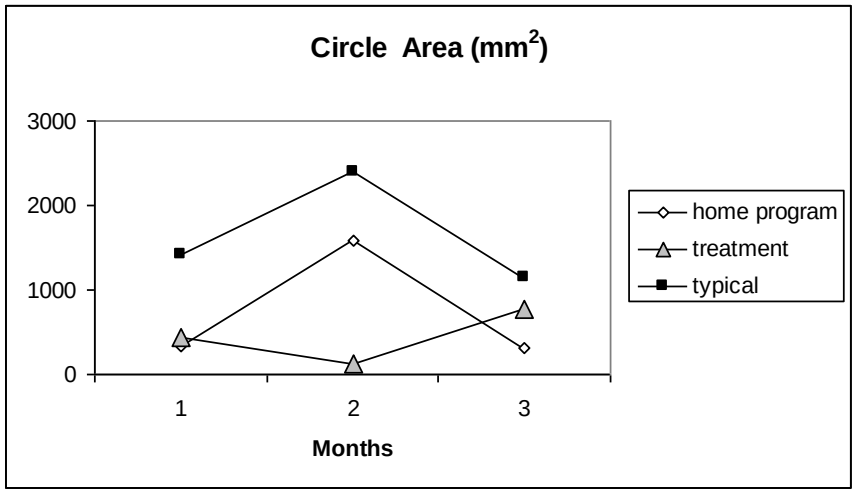
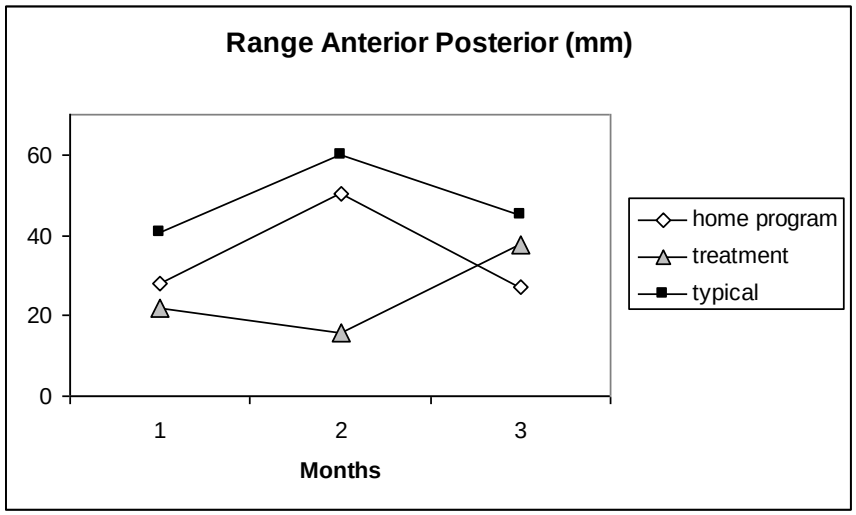

Complexity
Predictability

Figure 3


Circle Area (mm2)
3000
2000
1000
0
1
2
3
Months
home program
treatment
typical


Range Anterior Posterior (mm)
60
40
20
0
1
2
3
Months
home program
treatment
typical

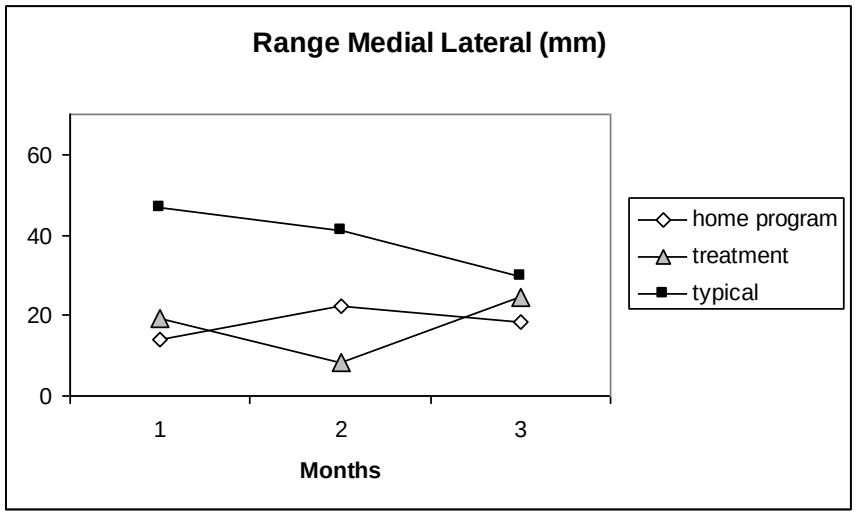

Range Medial Lateral (mm)
60
40
20
0
1
2
3
Months
home program
treatment
typical

Figure 4

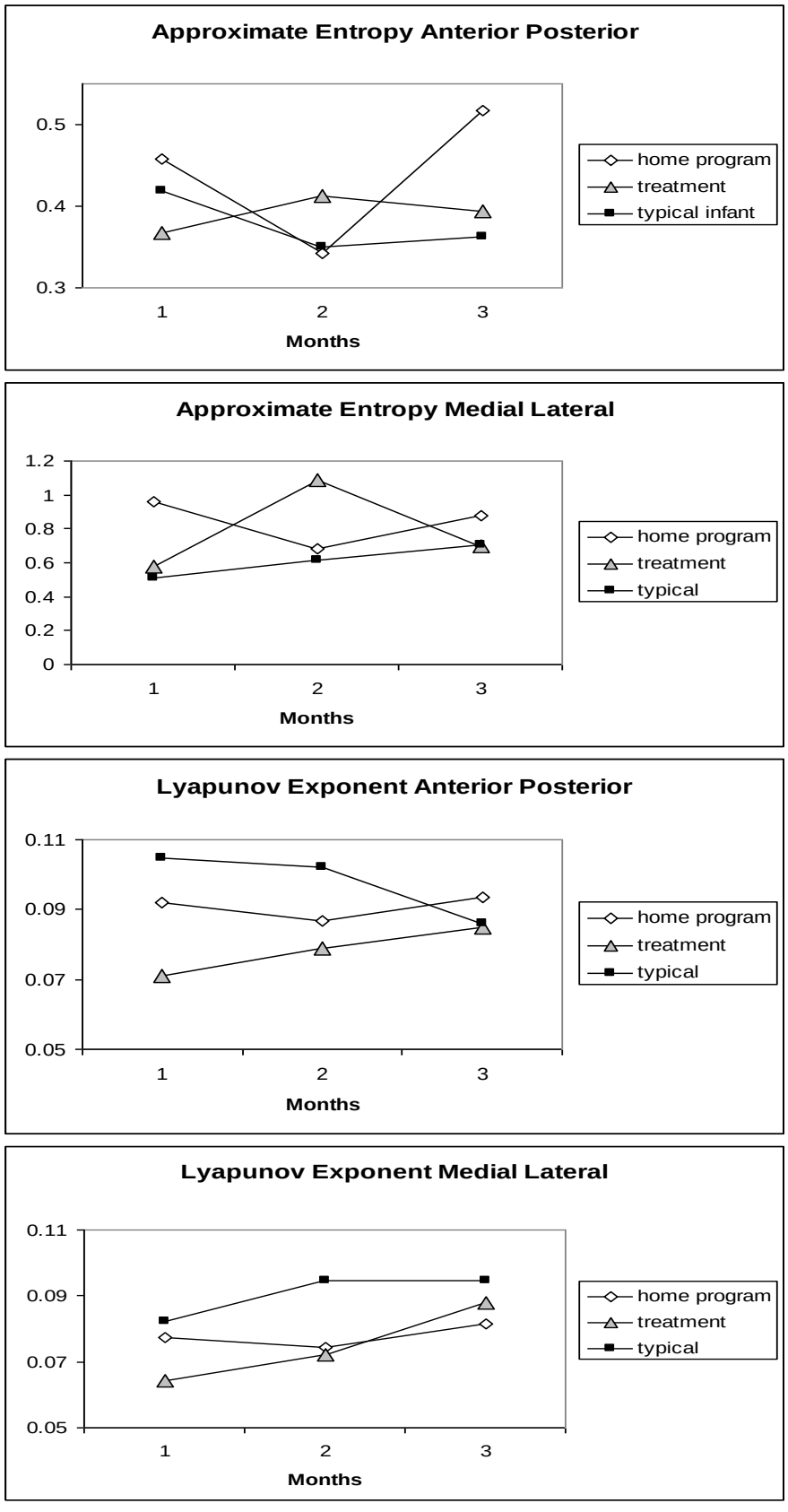

Approximate Entropy Anterior Posterior
0.5
0.4
0.3
1
2
3
Months
home program
treatment
typical infant
Approximate Entropy Medial Lateral
1.2
1
0.8
0.6
0.4
0.2
0
1
2
3
Months
home program
treatment
typical
Lyapunov Exponent Anterior Posterior
0.11
0.09
0.07
0.05
1
2
3
Months
home program
treatment
typical
Lyapunov Exponent Medial Lateral
0.11
0.09
0.07
0.05
1
2
3
Months
home program
treatment
typical

Figure 5

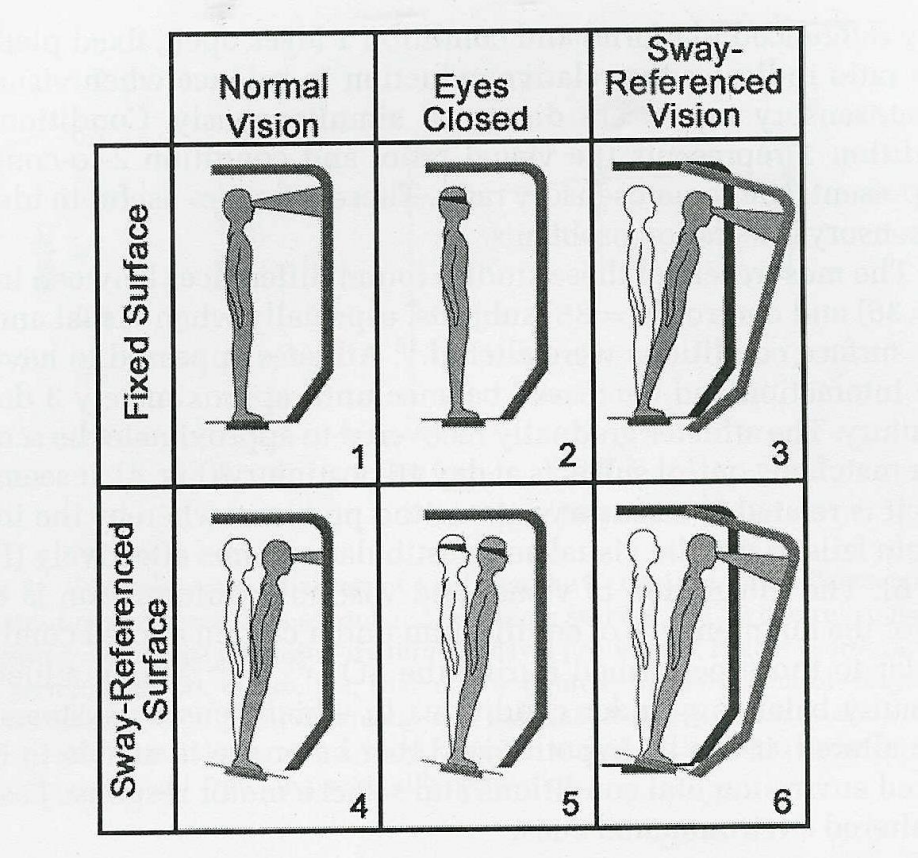

Normal Vision
Eyes Closed
Sway-Referenced Vision
Fixed Surface
Sway-Referenced Surface
1
2
3
4
5
6

Figure 6

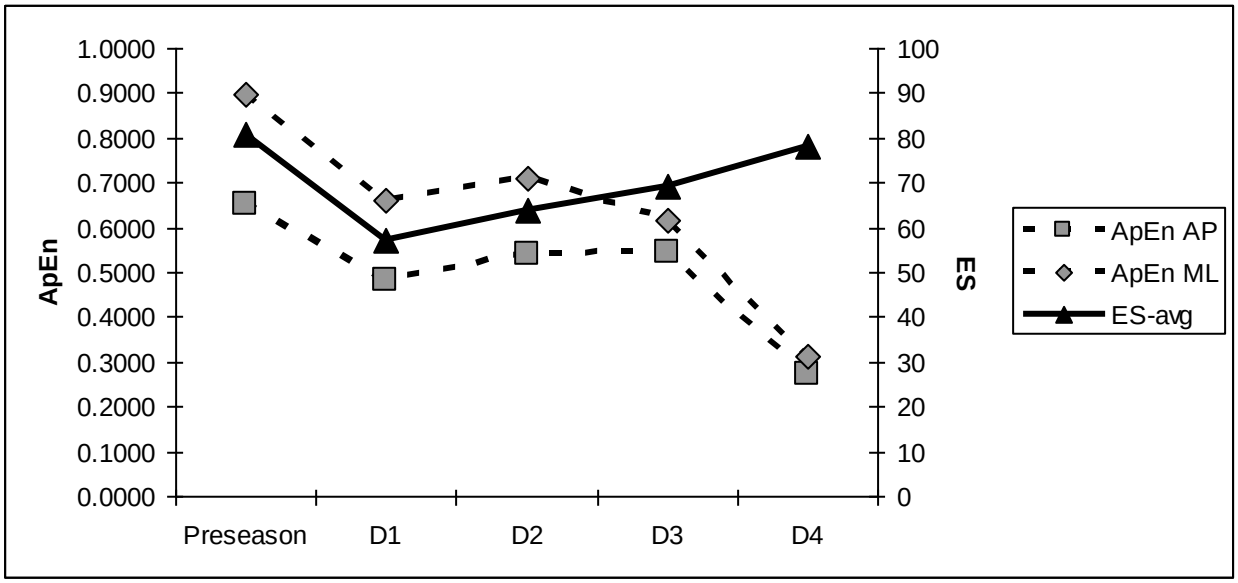

1.0000
0.9000
0.8000
0.7000
0.6000
0.5000
0.4000
0.3000
0.2000
0.1000
0.0000
ApEn
100
90
80
70
60
50
40
30
20
10
0
ES
Preseason
D1
D2
D3
D4
ApEn AP
ApEn ML
ES-avg

Figure 7

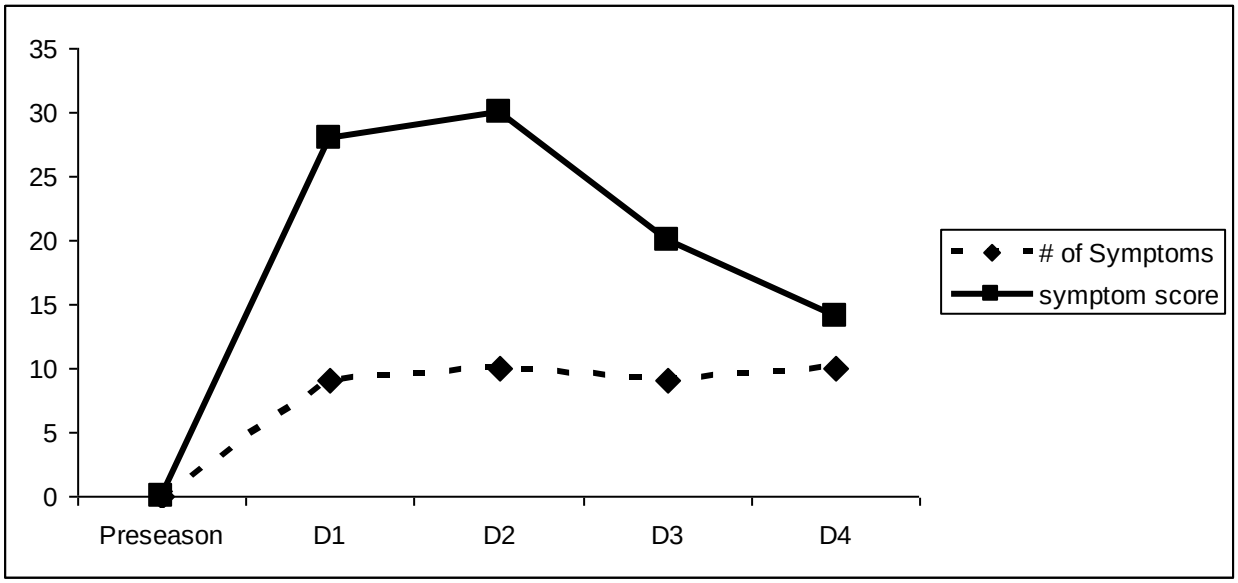

35
30
25
20
15
10
5
0
Preseason
D1
D2
D3
D4
# of Symptoms
symptom score

Figure 8a

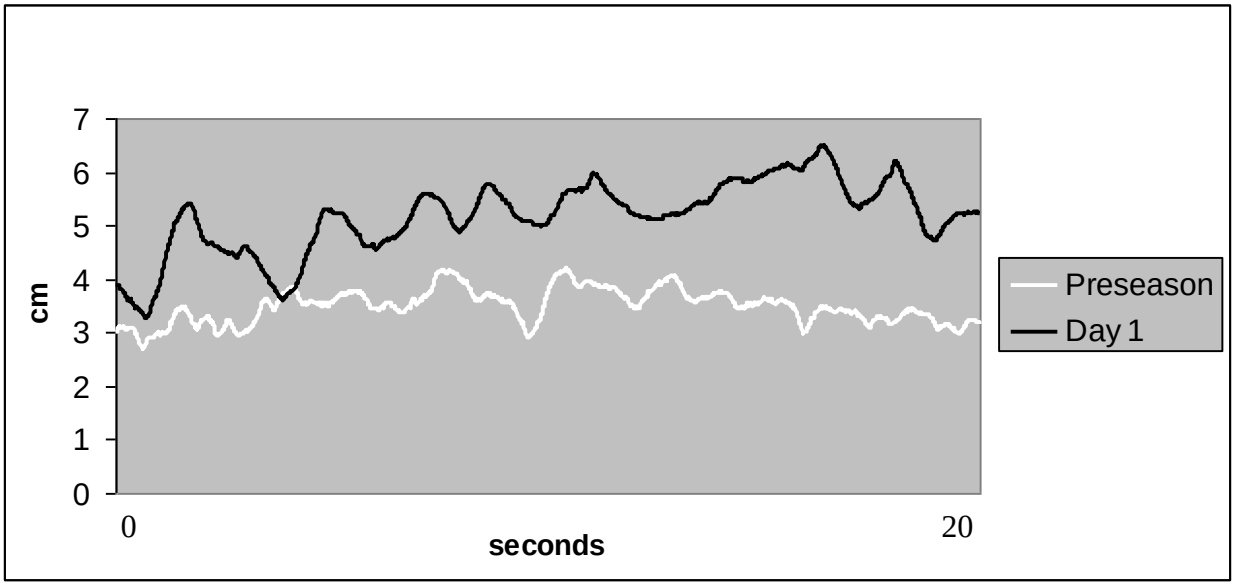


Figure 8b