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Influence of visual dependence on inter-segmental coordination during upright stance in cerebral palsy

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Abstract

The presence of visual dependence as an influential factor on the development of functional stability in ambulatory individuals with cerebral palsy (CP) was studied in 22 adults with spastic bilateral CP, 11 of whom were considered visually dependent, and 18 healthy adults. Participants stood upright during pitch plane disturbances of the visual field and support surface.

Intersegmental coordination behaviors were assessed by fitting trajectories of adjacent body segments to an ellipse. Mixed-model repeated measures ANOVAs were performed on ellipse orientation angle and area. Dissimilar stabilizing strategies adopted by the two groups with CP imply that visual dependence impacts postural control. Postural reorganization in response to visual flow in all groups indicates that we cannot ignore perceptual aspects of postural control when designing therapeutic interventions.

Keywords

balance; optic flow; virtual reality; rehabilitation

INTRODUCTION

Maintaining balance during upright stance is a challenging task for individuals with cerebral palsy (CP) (Rosenbaum et al., 2007). Factors contributing to the challenge include musculoskeletal changes and sensory deficits or deficits in sensory integration among primary sensory systems including the visual, somatosensory, and vestibular systems (Pavão, Silva, Savelsbergh, & Rocha, 2014). Children with spastic diplegic CP exhibited difficulties resolving intersensory conflict for maintenance of standing balance, but were able to adopt a compensatory strategy by exploiting visual signals to improve postural stability (Saxena, Rao, & Kumaran, 2014) and upright posture (Lidbeck et al., 2016). However, spatial disorientation (Previc, 1992) and postural instability (Lackner & DiZio, 1988) occur when postural responses are matched to the external visual surround rather than to interoceptive

balance demands (e.g., somatosensory and proprioceptive signals). In this case, intact vestibular information would be critical when relying principally on visual inputs in order to identify sensory conflict and an appropriate vertical orientation in space.

Stabilizing the head in space to promote reliability of vestibular feedback and gaze stabilization during active motion is crucial to postural control (Grossman, Leigh, Abel, Lanska, & Thurston, 1988; Pozzo, Berthoz, & Lefort, 1990). Separating control of the head in space and control of the trunk and lower limbs would be the most effective strategy for dealing with balance challenges (Keshner, Allum, & Pfaltz, 1987). The ability to stabilize the head is established throughout development (Thelen & Spencer, 1998). Children do not develop the adult-like repertoire of actively stabilizing the head to global space during locomotion or when balance is challenged until the age of eight years (Assaiante & Amblard, 1993; Keshner & Chen, 1996). Older adults with neurological deficits are also commonly observed to stabilize their head to trunk (Keshner, 2003). Thus, head stabilization to trunk appears to emerge when sensorimotor control is immature, impaired, or when in a challenging environment.

Postural control strategies also depend upon the perceptual preference of healthy adults (Isableu et al., 2010). Individuals who are visually independent (i.e., place greater weight on somatosensory/interoceptive inputs) tend to stabilize their head to global space and their hips to ankles. This response pattern suggests they exploit vestibular and somatosensory information when they discern a misleading visual frame of reference for maintaining upright stance (Isableu et al., 2010). In contrast, visually dependent individuals (i.e., place greater weight on visual/exteroceptive inputs) stabilize their head to the upper body and hips to global space. This response pattern suggests that the visual frame of reference rather than interoceptive information was relied upon for postural control (Isableu et al., 2010).

Sensorimotor experiences necessary for the progression of stabilizing strategies may not occur with a developmental disability such as CP. Diminished sensorimotor integrity could cause individuals with CP to rely predominantly on visual feedback (Barela et al., 2011; Slaboda, Lauer, & Keshner, 2013). While maintaining the upright stance, children and adolescents with CP coupled their postural sway to an oscillatory visual environment (Barela et al., 2011). When exposed to larger visual motions, the coupling effect remained prominent in individuals with CP but less strong in individuals without CP, indicating that postural behavior in individuals with CP could be affected by the presence of visual dependence. A Rod-and-Frame-test (RFT) supported that adults with CP overall are more visually dependent than their peers without CP (Slaboda et al., 2013). However, a high inter-individual variation in the RFT results suggested that not all individuals with CP were visually dependent. Visual-perceptual impairment affects approximately half of the CP population but physiological and psychophysical attributes contributing to visual-perceptual impairment are unknown (Ego et al., 2015). Visual dependence may be one category of visual-perceptual impairment resulting from somatosensory deficit. It remains unclear whether individuals with CP exhibit different postural control strategies as a function of visual dependence.

Human postural control in the sagittal plane while standing can be described by ankle and hip strategies. The ankle strategy stabilizes the center of mass by moving the entire body as a single inverted pendulum centered around ankle with in-phase motion above and below the hip (Nashner, Woollacott, & Tuma, 1979). The hip strategy stabilizes the center of mass by producing large and rapid motion at the hip with out-of-phase motion above and below the hip (Horak & Nashner, 1986). An ankle strategy is usually elicited in response to base of support perturbations with a subtle stimulation and a flat and firm support surface. Hip strategy, however, is elicited in response to faster and larger perturbations, or when the support surface is compliant or smaller than the base of support (Creath, Kiemel, Horak, Peterka, & Jeka, 2005). A hip strategy emerges when the postural disturbance is greater than the compensatory motion elicited by an ankle strategy, and is commonly observed in individuals with impaired balance such as older adults (Lin, Woollacott, & Jensen, 2004) and those with sensory dysfunction (Diener, Dichgans, Guschlbauer, & Mau, 1984). For adaptive human postural control in natural conditions, however, concurrent contributions from the ankle, hip, and many other joints along the longitudinal axis of the body are likely. For example, joint variability increased, but overall center of mass and head position were maintained after removing visual information in healthy human subjects maintaining upright stance (Hsu, Scholz, Schoner, Jeka, & Kiemel, 2007). This collaborative contribution of multiple joints to achieve the desired outcome is representative of motor abundance (Latash, 2012), and addresses the importance of simultaneous contribution from multiple joints to accomplish a motor task.

In this study we explored changes in the inter-segmental coordination of adults with and without CP resulting from combined perturbations of the visual field and support surface. Four conditions of platform motion and visual field motion were developed from previous work from our laboratory. We presented trials in the dark and with a stationary visual scene to serve as reference measures of the postural responses to base of support disturbances with and without visual feedback. Previous studies (Dokka, Kenyon, Keshner, & Kording, 2010; Keshner, Kenyon, & Langston, 2004; Wang, Kenyon, & Keshner, 2010) indicated that response magnitudes to visual flow significantly increased, and responses to somatosensory signals reflected stimulus amplitude, only when the two inputs (visual and platform motion) were combined. Response magnitudes dropped off, however, as the velocity of the visual scene increased beyond that of the platform (Slaboda & Keshner, 2012). Through pilot data we determined that 30°/sec was the fastest platform dorsiflexion velocity tolerated by individuals with CP without producing a fall, thus that value was selected for both the scene and platform disturbance. Visual field motion was presented in both the pitch upward and downward directions. Pitch up visual field motion was matched to the direction of the support surface disturbance which should produce a backward pitch of the head and trunk. Thus, we expected decreased intersegmental variability because of the limited motion available before the center of mass moved beyond the base of support (i.e., the heel). Pitch down visual flow was opposite the direction of the base of support disturbance and should produce forward pitch of the head and trunk. We expected increased intersegmental variability because of the greater motion available for postural compensation as center of mass moved over the foot.

Participants with CP were further grouped according to an assessment of visual dependence in order to explore whether a greater reliance on visual inputs would alter the organization of postural behaviors. We hypothesized that individuals who were visually independent would align their head to gravitational vertical regardless of visual field motion, irrespective of a diagnosis of CP. Furthermore, we hypothesized that individuals with CP who were also visually dependent would match motion of the head and trunk to motion of the visual world. We would then expect that inter-segmental variability would increase with combined pitch up motion of the support surface and visual scene even though such a compensatory response would result in increased instability.

METHODS

Subjects

Twenty-two individuals with spastic CP with bilateral involvement at GMFCS level I or II (11 females; 31.0 ± 10.5 years) and 18 individuals without CP (CONTROL; 7 females; 31.9 ± 11.5 years) were enrolled in this study. Subjects with CP were separated into CPVI (CP with visual independence, $n=11$) and CPVD (CP with visual dependence, $n=11$) groups based on their performance on the Rod and Frame Test (RFT, details in the clinical tests section). Inclusion criteria included: minimum of 20/40 corrected vision; ability to follow instructions; stand independently without assistive devices or support for >2 minutes; $>15^\circ$ of sagittal plane passive motion at ankle. Exclusion criteria were neurological disorders other than CP. No subjects were treated with Baclofen or Botox in the past 3 months. No subjects wore an ankle foot orthosis at the time of participation even in their daily life. Temple University institutional review board approved this study. Informed consent was obtained from subjects older than 18 years and parents of minors with assent from minors.

Clinical Tests

Range of motion (ROM), muscle strength, and muscle tone of lower limbs were measured and senses of light touch, pain, and vibration tested on participant's feet (Table 1). The non-instrumented *Dynamic Visual Acuity (DVA) test* assessed vestibulo-ocular reflex (VOR) function (Longridge & Mallinson, 1987). Impaired VOR was identified when ability to read the visual chart worsened by 2 lines on the eye chart when the head was turning side to side at 2 Hz. A computer-based *RFT* assessed the presence of visual dependence (VD) while seated by testing accuracy of vertical perception in the absence of visual cues to vertical (Bagust, Docherty, Haynes, Telford, & Isableu, 2013). VD was assumed if the mean or standard deviation of the angular deviations was greater than $\pm 4.5^\circ$ (Bagust et al., 2013).

Postural Tasks

Subjects were instructed to look straight ahead and maintain an upright posture while standing barefoot with feet placed a comfortable distance apart, arms resting at the sides, on a dynamic platform (Neurocom, Clackamas, OR) with integrated force plates (AMTI, Watertown, MA). Initial foot position was reproduced across trials. The virtual environment consisted of 3-walls with graphical projection synchronized through CAVELib software (MechDyne, Virginia Beach, VA). After 5-sec quiet stance, the supporting platform rotated at $30^\circ/\text{sec}$ to 3° of dorsiflexion. The platform remained in the toe-up position for 30-sec and

then returned to neutral across another 30-sec at $0.1^{\circ}/\text{sec}$ which is considered sub-threshold for vestibular stimulation (Figure 1A) (Keshner et al., 1987; Slaboda, Lauer, & Keshner, 2011). The visual scene was stationary during the first 5 sec, then followed by a 60-sec period with one of the following six conditions: a stationary visual scene (EO), eyes closed (EC), continuously rotating visual scene at $30^{\circ}/\text{sec}$ in pitch-up (PU), and continuously rotating visual scene at $30^{\circ}/\text{sec}$ in pitch-down (PD) (Figure 1B). Onset of visual field motion and support surface tilt was synchronized. All participants completed four conditions, one trial per condition, in a pseudorandom order. All participants verbally confirmed the detection of visual field motion.

More than half the subjects with CP stood in equinus and had limitations in passive dorsiflexion (Table 1); however, the 3° dorsiflexion displacement was tolerated and stability was maintained well enough to acquire kinematic measures. Individuals who were unable to remain upright during initial-tilt were allowed to repeat the trial. If a successful trial without stepping or support from the experimenter could not be achieved from the second trial, the subject was removed from the analyses.

Data Collection and Analysis

Three-dimensional kinematic data were collected at 60Hz using a Hawk System (Motion Analysis Inc., Santa Rosa, CA). In order to understand how the body segments were coordinated in responding to the perturbation from the supporting surface and visual field in the sagittal plane, reflective markers were placed on anatomical landmarks. Sagittal orientation was computed for the: *head* (defined as the vector between the forehead and external occipital crest); *thorax* (vector between manubrium and C7); *hip* (vector between L4/L5 joint and midpoint of bilateral ASIS); *ankle* (midpoint of bilateral head of 1st metatarsal bone and the midpoint of bilateral malleolus).

Sagittal orientation after 5-sec quiet stance was assumed to reflect a neutral position; offset of this neutral position was removed from the entire trial. Following the 5-sec quiet stance, sagittal orientation was divided into 4 windows presenting different postural demands for analysis (Figure 1C): initial-tilt (0–2sec), post-tilt (2–10sec), sustained-tilt (10–30sec), and return (30–60sec).

Angle-angle diagrams from adjacent body segments (head to thorax, thorax to hip, and hip to ankle) were created using a sagittal orientation (Figure 1E). An ellipse was fitted to each angle-angle diagram for each subject using the least-squares criterion separately for each window. Area and orientation angle of the fitted ellipse were extracted to determine intersegmental variability and control strategy, respectively (Figure 1F).

Statistical Analyses

Differences between CPVI and CPVD on GMFCS level, static visual acuity, spasticity and muscle strength in the lower extremities were examined with Mann-Whitney U tests. Dorsiflexion ROM differences were examined with an independent t-test. Fisher exact test was used to examine whether the occurrence of VOR dysfunction and the occurrence of visual dependence were related in individuals with CP.

Mixed-model repeated measures ANOVAs with group (*CONTROL*, *CPVI*, *CPVD*) as between-subject factors, and visual field conditions (*EO*, *EC*, *PU*, *PD*) as within-subject factors were performed separately on ellipse orientation *angle* and *area* derived from each segmental pair during each window (SPSS 25.0, SPSS Inc., Chicago, IL). *F*-values based on Wilks' Lambda were adopted ($\alpha=0.05$) (Howell, 2007). If a significant difference was found across groups or visual field conditions, then *post hoc* pairwise comparisons were conducted. If an interaction effect between group and visual field condition was found significant, then the data was split by group or condition before carrying out *post hoc* analyses using one-way ANOVA.

RESULTS

Clinical Testing

No significant differences between the two CP groups ($p>.05$; Table 1) were revealed by any of the clinical tests including GMFCS level, static visual acuity, ROM, muscle tone, and strength. A trend emerged ($p=.08$) suggesting that visual dependence in individuals with CP was likely associated with VOR dysfunction.

Inter-Segmental Coordination (Ellipse Orientation Angle)

During *initial-tilt*, a significant group effect emerged in the thorax-to-hip motion ($F(2,37)=5.376$, $p=.009$; Figure 3B). Pairwise group comparisons revealed that the CPVD group adopted a significantly different strategy than CPVI ($p=.048$) or CONTROL ($p=.002$); no difference emerged between CPVI and CONTROL. Individuals with visual independence (i.e., CPVI and CONTROL) primarily exhibited greater motion at the hip than the ankle ($\sim 0^\circ$ ellipse orientation angle) confluent with in-phase thorax and hip motion ($>30^\circ$ ellipse orientation angle) indicative of an ankle strategy. In contrast, CPVD individuals exhibited greater motion at the hip than ankle ($\sim 0^\circ$ ellipse orientation angle) combined with a slightly greater thorax motion than hip ($<30^\circ$ ellipse orientation angle) suggesting a hip strategy.

Transitioning into *post-tilt*, a slight increase in thorax motion with respect to hip motion ($<30^\circ$ ellipse orientation angle) emerged in individuals with visual independence (i.e., CPVI and CONTROL; Figure 3B). However, no group effect or visual field motion effect was observed, thus we conclude that a similar inter-segmental control strategy was adopted by all groups regardless of motion of the visual surround.

During *sustained-tilt*, participants continued to adopt similar inter-segmental control strategies with some discrepancy in the CPVD group (Figure 3B). A significant interaction effect emerged between group and visual field motion in thorax-to-hip motion ($F(6,70)=2.336$, $p=.041$) as well as a significant main effect of group ($F(2,37)=4.617$, $p=.016$) and a significant main effect of visual field motion ($F(3,35)=4.360$, $p=.010$). Individuals with visual independence (i.e., CPVI and CONTROL) continued to exhibit inter-segmental control strategies similar to post-tilt, but CPVD individuals adopted a significantly different strategy from the CONTROL group ($p=.004$). Pairwise comparisons between conditions revealed that the thorax-to-hip ellipse orientation angle was significantly higher than when the visual field continuously pitched downward than when static ($p=.017$),

dark ($p=.001$) or pitching upward ($p=.044$). Post hoc analyses after splitting the data by group showed that the influence of visual field motion on the thorax-to-hip motion was only significant in CPVD ($p=.041$) with in-phase thorax and hip motion ($>30^\circ$ ellipse orientation angle) when the visual field pitched downwards, and greater thorax than hip motion ($\sim 0^\circ$ ellipse orientation angle) when in the dark ($p=.004$) or during pitch upwards ($p=.019$) of the visual field.

During the *return* period, participants exhibited in-phase hip and ankle motion, suggesting that, regardless of group, individuals “passively” traveled on the moving support surface as it gradually returned to neutral. However, a significant group effect appeared in thorax-to-hip motion ($F(2,37)=6.455$, $p=.004$; Figure 3B). Pairwise group comparisons showed that the CONTROL group exhibited in-phase thorax and hip action ($>30^\circ$ ellipse orientation angle), whereas greater thorax than hip motion ($<30^\circ$ ellipse orientation angle) was observed in CPVI ($p=.004$) and CPVD ($p=.006$). Thus, both CP groups produced greater upper body motion when exposed to a dynamic visual environment than when in a static or dark visual field; the upper body strategy of the CONTROL subjects was similar strategy regardless of visual field motion.

Also, the interaction effect between group and visual field motion was not significant during the return period, although visual field motion had a significant effect on head-to-thorax motion ($F(3,35)=3.020$, $p=.043$; Figure 3A) across all groups. Pairwise comparison between conditions revealed that in-phase head and thorax motion ($>30^\circ$ ellipse orientation angle) appeared when the visual field was dark; more pronounced head-to-thorax motion was observed when the visual field motion was static ($p=.028$) or continuously pitched upward ($p=.007$).

Inter-Segmental Movement Variability (Ellipse Area)

During *initial-tilt*, a significant interaction between group and visual field motion emerged in head-to-thorax motion ($F(6,70)=3.567$, $p=.004$; Figure 4A) with significant main effects of group ($F(2,37)=8.044$, $p=.001$) and visual field motion ($F(3,35)=5.208$, $p=.004$). A significant interaction between group and visual field motion also emerged in thorax-to-hip motion ($F(6,70)=2.698$, $p=.021$; Figure 4B) with significant main effects of group ($F(2,37)=10.355$, $p<.001$) and visual field motion ($F(3,35)=5.396$, $p=.004$). Significant effects of group ($F(2,37)=4.902$, $p=.013$) and visual field motion ($F(3,35)=3.432$, $p=.027$) were also observed in hip-to-ankle motion (Figure 4C). Thus, the groups were observed to respond differentially to the initial balance disturbance: all individuals with CP (i.e., CPVI and CPVD) exhibited a greater ellipse area (i.e., higher movement variability) in the upper body above the hip, especially when exposed to continuous visual field motion; the highest variability was observed during pitch up visual flow.

Intersegmental variability when transitioning into *post-tilt* remained the same for individuals with visual independence whereas intersegmental variability decreased in CPVD individuals suggesting a greater impact of visual motion on the compensatory behavior for visually dependent individuals. A significant interaction between group and visual field motion emerged in head-to-thorax motion ($F(6,70)=2.860$, $p=.015$; Figure 4A) with significant main effects of group ($F(2,37)=4.378$, $p=.020$) and visual field motion ($F(3,35)=7.653$, $p<.001$).

Pairwise comparisons of the specific visual field motion during the post-tilt period showed significantly higher head-to-thorax variability during a static visual field ($p=.043$) and in both PU ($p=.020$) and PD ($p=.048$) than in the dark. Moreover, *post hoc* analyses after splitting data by visual field motion showed that CPVI exhibited significantly higher head-to-thorax variability than CPVD and CONTROL when the visual field was pitching upwards or downwards; these group differences were not found when the visual field was static or dark. Significant group effects also emerged in thorax-to-hip ($F(2,37)=6.661$, $p=.003$; Figure 4B) and hip-to-ankle motion ($F(2,37)=4.827$, $p=.014$; Figure 4C); variability was significantly higher in individuals with CP than that in the CONTROL group.

Intersegmental variability in the CPVI group reduced from post-tilt as they transitioned into *sustained-tilt*. A significant main effect of visual field motion emerged in head-to-thorax motion ($F(3,35)=2.887$, $p=.049$; Figure 4A) and pairwise comparisons revealed greater head-to-thorax variability during PU than PD ($p=.016$) and EO ($p=.005$). A significant group effect emerged in hip-to-ankle motion ($F(2,37)=4.605$, $p=.016$; Figure 4C) with pairwise group comparisons revealing that the CPVD group exhibited greater hip-to-ankle variability than CONTROL ($p=.005$) or CPVI ($p=.037$). A significant effect of visual field motion also emerged in hip-to-ankle motion ($F(3,35)=4.143$, $p=.013$; Figure 4C) with pairwise comparisons indicating that greater hip-to-ankle variability during PD than EO ($p=.002$) and EC ($p=.002$).

During *return*, significant effects of visual field motion were observed in head-to-thorax ($F(3,35)=2.893$, $p=.049$; Figure 4A), thorax-to-hip ($F(3,35)=3.233$, $p=.034$; Figure 4B), and hip-to-ankle ($F(3,35)=5.111$, $p=.005$; Figure 4C) motion. Greater variability was observed when the visual field continuously pitched up or down compared to static and dark. A significant group effect also emerged in hip-to-ankle ($F(2,37)=4.003$, $p=.027$; Figure 4C) where CPVI exhibited greater hip-to-ankle variability than CONTROL ($p=.008$).

DISCUSSION

As previously reported in the literature (Ego et al., 2015; Yu, Lauer, Tucker, Thompson, & Keshner, 2018), visual dependence was found in half of participants with bilateral spastic CP and this study was designed to determine whether the presence of visual dependence needs to be considered as an influential factor in the development of functional stability. The combination of visual flow with a support surface disturbance in matched and opposing directions has been shown to be an effective approach for visual-vestibular mismatch and analyzing the relative weighting of these inputs (Keshner et al., 2004; Slaboda & Keshner, 2012).

Visual dependence is the term used to represent the degree to which an individual relies on visual stimuli for spatial orientation (Roberts, Da Silva Melo, Siddiqui, Arshad, & Patel, 2016). Although usually considered a perceptual trait or cognitive style mediated by central reweighting of the sensory inputs (Lambrey & Berthoz, 2003), it has also been demonstrated that visual dependence increases with vestibular disorders (Agarwal et al., 2012; Cousins et al., 2014; Guerraz et al., 2001) and is modulated by eye movements, in particular nystagmus (Roberts et al., 2016). Thus, visual dependence is likely a category of visual perceptual

impairment resulting from a vestibular and/or somatosensory deficit. Traditional clinical sensory tests for light touch and vibration were intact across all subjects in this study, which might imply that visual dependence was a perceptual choice (Lambrey & Berthoz, 2003) emerging through sensorimotor experiences rather than reweighting due to sensory deficit. However, based on the DVA results in this study, we infer that visual dependence was more prevalent in those with a reduced VOR function (Table 1) implicating vestibular dysfunction with the appearance of visual dependence.

Dissimilar postural control strategies adopted by the CPVI and CPVD groups supports the concept that visual dependence impacts the control of postural responses. The principle difference was observed in the CPVI group who adopted strategies similar to the individuals without CP when encountering sudden disturbances in the environment (i.e., initial-tilt), but then adopted strategies similar to CPVD when receiving diminished vestibular feedback and a changing orientation in space (i.e., return period). When the sensory array was dynamic, all three groups adopted similar strategies for postural stabilization (i.e., post-tilt). During initial-tilt, both vestibular and visual information contributed to knowledge of orientation in space. With the presence of continuous visual field motion, efficient control of upright stance would rely on down-weighting the visual motion cues in order to identify self-motion versus global orientation (Dokka et al., 2010). Individuals with CPVD, however, adopted a hip strategy rather than the ankle strategy exhibited by individuals with CPVI and those without CP. This suggests that, with visual dependence, visual field motion was perceived to augment the initial tilt and present a greater balance challenge requiring the response accommodation observed for a fast perturbation with a large amplitude (Horak & Nashner, 1986). Additionally, the human-environment dynamic in individuals with CPVD was differently perceived under the influence of different visual field motion; which is evident by the different control strategies adopted by CPVD when exposed to different visual field conditions. From these results we would recommend that treatment interventions for individuals with CP and visual dependence be designed to address vestibular as well as motor impairment.

Biomechanical factors likely contributed to group differences elicited by the initial tilt. We attempted to control for these by minimizing differences between CPVI and CPVD in range of motion, muscle strength, and spasticity of the lower limbs. All subjects with CP were GMFCS level I or II signifying that standing and walking are functional with minimal assistance. The displacement and velocity parameters have been shown to have little effect on the stretch reflex in individuals with spasticity (Hornby, Kahn, Wu, & Schmit, 2006) and were chosen in order to minimize perturbation impact on lower limb tonus. Prolonged latencies of muscle onset observed with a crouched posture (Burtner, Qualls, & Woollacott, 1998) could preclude a reliance on increased upper body motion to counter environmental disturbances (Hicks, Schwartz, Arnold, & Delp, 2008) and an increased reliance on ankle somatosensory information. However, a crouched posture was observed in individuals with both CP groups but CPVD did not demonstrate the ankle strategy. Consequently, we deduce that a crouched posture does not supersede the effect of visual dependence.

While individuals with CPVI adopted an ankle strategy similar to those without CP in response to initial tilt, their greatest inter-segmental variability emerged throughout the post-

tilt period. There were no falls or loss of balance during this period, thus the increased inter-segmental movement variability was unlikely indicative of impaired functional posture control. The increased intersegmental variability observed across multiple segmental pairs may have a minimal effect on the position of center of mass (Hsu et al., 2007). However, further study would be needed to confirm this proposition. The increased variability could also represent exploratory movements that provide continuous, dynamic self-motion feedback (Murnaghan, Carpenter, Chua, & Inglis, 2017; Yu, Bardy, & Stoffregen, 2011). Under the influence of dynamic visual field motion, it may take a longer period for individuals with CPVI to explore the human-environment dynamic and re-orient themselves after the support surface perturbation. The initial tilt combined with a continuously pitching upward visual field at the same velocity creates a disorienting situation, during which either a reliable visual or vestibular signal would be necessary to return to a vertical orientation in space (Kluzik, Peterka, & Horak, 2007). Continuous visual field motion reduced the reliability of those signals. Increasing self-motion following initial support surface perturbation would serve the purpose of increasing somatosensory feedback gains in order to facilitate distinguishing between self and environmental motion. There are numerous reports of increased sway during quiet stance when sensory feedback becomes unreliable (Horak & Nashner, 1986; Keshner et al., 1987; Murnaghan, Horslen, Inglis, & Carpenter, 2011). For individuals with CPVI, increased head-to-thorax variability in the moving visual environment would suggest the utilization of self-motion for exploratory behavior to resolve the sensory conflicts. In contrast, for individuals with CPVD, such exploratory behavior may have diminished quickly because of their reliance on vision.

Visual field motion continued to impact the postural behavior of individuals with CPVD into the sustained-tilt period, as evident in the emergence of in-phase thorax-to-hip motion during pitching-down visual flow and pronounced thorax-to-hip motion during pitching-up visual flow even though the support surface was kept tilted and stationary, which was not notable in individuals with visual independence (i.e., CPVI and CONTROL). Visual field motion induced self-motion, also known asvection, emerges approximately 10 seconds into immersive virtual environment experience (Guerraz & Bronstein, 2008; Wang et al., 2010). In corroborate with our previous finding that the deviation of the center of mass into the same direction as visual field motion was most notable in individuals with CPVD (Yu et al., 2018), thevection phenomenon was more noticeable in individuals who are visually dependent.

Individuals with CPVI and CPVD adopted the hip strategy in the return period when the support surface moved at a velocity below the threshold for stimulation of otolith signals (Fitzpatrick & McCloskey, 1994); individuals without CP minimized intersegmental motion and appeared to passively “ride” as the platform returned to neutral. A response matched to motion of the support surface would suggest that individuals without CP were able to up-weight ankle somatosensory information to remain in sync with the moving platform. In contrast, both CP groups increased upper body motion and minimized lower limb motion. Spasticity at the ankle might have limited their ability to compensate for platform motion, however these individuals were able to adopt an ankle strategy when the visual field rotated in upward pitch. Alternatively, individuals with CP may primarily rely on visual field feedback rather than ankle somatosensory inputs when otolith feedback is unreliable. In the

dark, ankle somatosensory inputs would be necessary, but not necessarily produce the most accurate postural restoration (Wang et al., 2010).

A limitation in this population is change in tone and range of motion at the ankle. Although we have attempted to minimize the impact of equinus stance across groups, postural strategies could be modified by availability of ankle motion. Another limitation is the lack of a comprehensive evaluation of visuo-perceptual and oculomotor functions of CP. A recent study reported approximately half of individuals with spastic CP with diplegia were accompanied by visuo-perceptual impairment and/or oculomotor dysfunction (Lidbeck et al., 2016). Only VOR responses were tested to identify vestibular function, thus we had no information about the otolith organs which contribute significantly to the stabilization of head (Blouin, Teasdale, & Mouchnino, 2007). Further studies are currently underway in our lab to assess the how postural control in individuals with CP is influenced by visual and vestibular functions.

Our results reveal that all CP participants relied less on lower limb proprioceptive information and more on visual and vestibular information than participants without CP when encountering balance challenges. Altered sensory weighting is not a singular process as it varies within the population with CP; some demonstrated visual dependence and others did not. Perceptual choice is elaborated throughout the developmental process and depends upon the available movement experiences. Possible mechanisms for increased reliance on a visual frame of reference could be the inability to suppress noisy feedback or diminished signals from vestibular feedback. Based on our results, it is unlikely that changes in functional mobility alone would explain the differences in control strategy; thus, differences in sensory-motor integration need to be considered. Reliance on visual feedback could underlie compromised postural control in adults with CP and visual dependence signifying that we cannot ignore the perceptual aspects of postural control when designing therapeutic interventions.

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Figure 1.

Protocol and analysis used in this study. **(A)** A diagram of the protocol used in this study indicates the motion of supporting surface throughout a trial in the tilted platform condition. **(B)** The six conditions of visual scene motion. **(C)** Each trial was divided into four time windows representing a different postural demand during upright stance. **(D)** A cartoon illustration showing the control strategies adopted by CONTROL, CPVI, and CPVD. **(E)** Angle-angle plot from two adjacent body segments (using thorax-to-hip motion as an example). An ellipse was fit to the data and from the fit ellipse, two parameters were computed: ellipse angle (indicative of control strategy), and ellipse area (indicative of movement variability). **(F)** A cartoon illustration identifying how orientation of the ellipse signifies relative motion between the two adjacent segments (using thorax-to-hip as an example).

Figure 2.

Angle-angle plots of head with respect to thorax (top row), thorax with respect to hip (middle row), and hip with respect to ankle (bottom row) in the sagittal plane in six visual scene conditions from left to right columns: eyes open (EO), eye closed (EC), pitch up at 30°/s (PU), pitch down at 30°/s (PD) from a representative subject (A) without cerebral palsy (Control), (B) cerebral palsy with visual independence (CPVI), and (C) cerebral palsy with visual dependence (CPVD). Trajectories from initial-tilt (blue), post-tilt (light blue), sustained-tilt (yellow), and return period (black).

Figure 3.

Orientation angle of the fitted ellipse representing (A) head to thorax, (B) thorax to hip, and (C) hip to ankle angle-angle plot in four visual conditions including eyes open (EO), eyes closed (EC), pitch up (PU), and pitch down (PD) during initial-tilt (0–2 sec), post-tilt (2–10sec), sustained-tilt (10–30sec), and return (30–60sec). Shaded area illustrates in-phase motion in the paired body segments. Asterisk (*) indicates significant difference between visual field conditions ($p < .05$). Pound (#) indicates significant group difference within the same visual field condition ($p < .05$). Pilcrow (¶) indicates significant difference emerged in the pair-wise group comparison ($p < .05$).

Figure 4.

Area of the fitted ellipse representing (A) head to thorax, (B) thorax to hip, and (C) hip to ankle angle-angle plot in four visual conditions including eyes open (EO), eyes closed (EC), pitch up (PU), and pitch down (PD) during initial-tilt (0–2 sec), post-tilt (2–10sec), sustained-tilt (10–30sec), and return (30–60sec). Asterisk (*) indicates significant difference between visual field conditions ($p < .05$). Pound (#) indicates significant group difference within the same visual field condition ($p < .05$). Pilcrow (¶) indicates significant difference emerged in the pair-wise group comparison ($p < .05$).

TABLE 1.

Participants' characteristics.

	Age (years)	Sex (F/ M)	Diagnosis	RFT (°) [Mean (SD)]	Group	Snellen (logMAR)	DVA (logMAR)	(logMAR)	VOR Function ^a	GMFCS E&R ^b	Light Touch ^c	Pain ^d	Vibration ^e	Crouch Stance ^f	Dorsiflexion ROM (R ; L)	Spasticity (R/L) ^g HAM QUAD GAS	Muscle Strength (R/L) ^h QUAD HAM DF PF
S01	23	M	Quadriplegia	0.94 (22.39)	CPVD	20/20 (0.00)	20/30 (0.20)	0.20	Impaired	I	Intact	Intact	Intact	w/out	12°; 14°	1 / 0 0 / 0 1+ / 1	5 / 5 5 / 5 5 / 5
S02	19	F	Diplegia	-4.56 (21.51)	CPVD	20/40 (0.30)	20/15 (-0.10)	-0.40	Intact	I	Intact	Impaired	Intact	w/out	14°; 10°	1+ / 1 0 / 1 1+ /	5 / 5 4 / 4 5 / 5
S03	45	F	Quadriplegia	-10.06 (21.08)	CPVD	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	II	Intact	Impaired	Intact	w/	5°; 15°	1+ / 1 0 / 0 1+ /	5 / 5 4 / 5 4 / 5
S04	45	F	Diplegia	-3.44 (6.86)	CPVD	20/20 (0.00)	20/30 (0.20)	0.20	Impaired	I	Intact	Intact	Intact	w/out	<5°; <5°	1+ / 1+ 1 / 0 2 / 2	4 / 5 4 / 5 4 / 5
S05	39	F	Diplegia	0.38 (11.28)	CPVD	20/40 (0.30)	20/70 (0.54)	0.24	Impaired	II	Intact	Intact	Intact	w/	<5°; <5°	1+ / 1+ 1 / 1 1 / 1	4 / 4 5 / 5 4 / 4
S06	35	M	Diplegia	-5.06 (4.37)	CPVD	20/20 (0.00)	20/20 (0.00)	0.00	Intact	I	Intact	Intact	Intact	w/out	5°; 10°	0 / 0 0 / 0 1 / 0	4 / 4 4 / 5 4 / 4
S07	50	M	Quadriplegia	3.81 (6.26)	CPVD	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	I	Intact	Impaired	Intact	w/	<5°; 5°	1 / 1 0 / 0 0 / 1+	4 / 5 5 / 5 4 / 5
S08	38	F	Diplegia	-6.00 (31.25)	CPVD	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	I	Intact	Impaired	Intact	w/out	<5°; <5°	1 / 2 1 / 0 2 / 2	5 / 5 5 / 5 5 / 5
S09	17	M	Diplegia	0.75 (27.04)	CPVD	20/20 (0.00)	20/20 (0.00)	0.00	Intact	I	Intact	Impaired	Intact	w/out	15°; 15°	0 / 0 0 / 0 1+ / 2	5 / 5 5 / 5 5 / 5
S10	21	F	Diplegia	-3.81 (15.39)	CPVD	20/15 (-0.10)	20/30 (0.20)	0.30	Impaired	I	Intact	Intact	Intact	w/	10°; 5°	0 / 0 0 / 0 0 / 1+	4 / 4 5 / 4 4 / 4
S11	38	F	Diplegia	0.75 (5.96)	CPVD	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	I	Intact	Intact	Intact	w/out	14°; 15°	0 / 0 0 / 0 1 / 1	5 / 5 5 / 4 5 / 4
S12	21	F	Diplegia	-0.50 (3.37)	CPVI	20/40 (0.30)	20/30 (0.20)	-0.10	Intact	I	Intact	Intact	Intact	w/	10°; 6°	1 / 0 0 / 0 1+ / 1	5 / 5 5 / 5 5 / 5
S13	17	M	Diplegia	-1.25 (1.00)	CPVI	20/15 (-0.10)	20/15 (-0.10)	0.00	Intact	II	Intact	Intact	Intact	w/out	9°; 9°	1+ / 1 0 / 0 1+ / 1	4 / 4 5 / 5 4 / 4
S14	30	M	Diplegia	-1.81 (1.98)	CPVI	20/25 (0.10)	20/25 (0.10)	0.00	Intact	II	Intact	Intact	Intact	w/out	9°; 6°	2 / 2 2 / 1+ 1 / 1	4 / 4 5 / 5 4 / 3+
S15	20	M	Diplegia	0.69 (4.33)	CPVI	20/40 (0.30)	20/30 (0.20)	-0.20	Intact	I	Intact	Intact	Intact	w/	10°; 10°	1 / 1 0 / 0 1 / 1	5 / 5 5 / 5 5 / 5
S16	28	M	Diplegia	-0.88 (2.63)	CPVI	20/40 (0.30)	20/70 (0.54)	0.24	Impaired	II	Intact	Impaired	Intact	w/	<5°; <5°	2 / 2 1 / 2 2 / 2	4 / 4 5 / 5 4 / 4
S17	43	F	Diplegia	0.00 (1.87)	CPVI	20/40 (0.30)	20/20 (0.00)	-0.30	Intact	I	Intact	Intact	Intact	w/out	16°; 15°	1 / 1 1 / 0 0 / 1	5 / 5 5 / 5 5 / 5

	Age (years)	Sex (F/ M)	Diagnosis	RFT (°) [Mean (SD)]	Group	Snellen (logMAR)	DVA (logMAR)	(logMAR)	VOR Function ^a	GMFCS E&R ^b	Light Touch ^c	Pain ^d	Vibration ^e	Crouch Stance ^f	Dorsiflexion ROM (R ; L)	Spasticity (R/L) ^g				Muscle Strength (R/L) ^h			
																HAM	QUAD	GAS	QUAD	HAM	DF	PF	
S18	35	M	Diplegia	−0.50 (2.62)	CPVI	20/30 (0.20)	20/30 (0.20)	0.00	Intact	II	Intact	Impaired	Intact	w/out	15°; 10°	1 / 0	0 / 0	1+ / 1+	5 / 5	5 / 5	4 / 4	5 / 5	
S19	22	M	Diplegia	0.94 (2.38)	CPVI	20/20 (0.00)	20/20 (0.00)	0.00	Intact	I	Intact	Intact	Intact	w/	<5°; <5°	1+ / 1	1 / 0	1+ / 1+	4 / 5	5 / 5	4 / 4	4 / 5	
S20	34	F	Diplegia	−0.56 (4.41)	CPVI	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	II	Intact	Intact	Intact	w/	<5°; <5°	0 / 1	0 / 0	1 / 1	3+ / 4	3+ / 3+	3+ / 3+	3+ / 3+	
S21	21	M	Diplegia	−2.13 (0.79)	CPVI	20/25 (0.10)	20/50 (0.40)	0.30	Impaired	II	Intact	Intact	Intact	w/out	5°; 5°	0 / 0	0 / 0	0 / 0	4 / 5	5 / 5	4 / 4	5 / 4	
S22	41	F	Quadriplegia	−0.38 (1.87)	CPVI	20/25 (0.10)	20/25 (0.10)	0.00	Intact	II	Intact	Intact	Intact	w/	5°; 12°	0 / 0	0 / 0	0 / 1	4 / 5	5 / 5	4 / 4	4 / 5	
T01	41	M	-	0.13 (2.05)	CONTROL	20/20 (0.00)	20/30 (0.20)	0.20	Impaired	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T02	27	M	-	−0.56 (1.29)	CONTROL	20/15 (−0.10)	20/20 (0.00)	0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T03	19	F	-	0.25 (2.15)	CONTROL	20/40 (0.30)	20/20 (0.00)	−0.30	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T04	22	F	-	−0.50 (1.77)	CONTROL	20/25 (0.10)	20/20 (0.00)	−0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T05	45	M	-	−1.56 (3.06)	CONTROL	20/40 (0.30)	20/40 (0.30)	0.00	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T06	48	M	-	0.81 (2.33)	CONTROL	20/40 (0.30)	20/30 (0.20)	−0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T07	27	M	-	−0.44 (1.02)	CONTROL	20/30 (0.20)	20/15 (−0.10)	−0.30	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T08	26	F	-	−0.88 (1.69)	CONTROL	20/25 (0.10)	20/20 (0.00)	−0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T09	44	F	-	0.94 (1.08)	CONTROL	20/40 (0.30)	20/30 (0.20)	−0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T10	18	M	-	0.75 (1.56)	CONTROL	20/25 (0.10)	20/30 (0.20)	0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T11	31	M	-	−0.44 (1.32)	CONTROL	20/15 (−0.10)	20/15 (−0.10)	0.00	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T12	20	M	-	−1.31 (3.27)	CONTROL	20/30 (0.20)	20/30 (0.20)	0.00	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T13	18	F	-	−0.31 (2.10)	CONTROL	20/20 (0.00)	20/15 (−0.10)	−0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	
T14	50	M	-	0.56 (3.48)	CONTROL	20/30 (0.20)	20/30 (0.20)	0.00	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-	-	

	Age (years)	Sex (F/ M)	Diagnosis	RFT (°) [Mean (SD)]	Group	Snellen (logMAR)	DVA (logMAR)	(logMAR)	VOR Function ^a	GMFCS E&R ^b	Light Touch ^c	Pain ^d	Vibration ^e	Crouch Stance ^f	Dorsiflexion ROM (R ; L)	Spasticity (R/L) ^g			Muscle Strength (R/L) ^h		
																HAM	QUAD	GAS	QUAD	HAM	DF
T15	21	F	-	-1.88 (3.01)	CONTROL	20/20 (0.00)	20/30 (0.20)	0.20	Impaired	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-
T16	43	M	-	-2.25 (2.10)	CONTROL	20/15 (-0.10)	20/20 (0.00)	0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-
T17	44	F	-	1.81 (2.80)	CONTROL	20/40 (0.30)	20/50 (0.40)	0.10	Intact	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-
T18	30	M	-	0.31 (1.16)	CONTROL	20/30 (0.20)	20/50 (0.40)	0.20	Impaired	-	Intact	Intact	Intact	-	-	-	-	-	-	-	-

Abbreviations: GMFCS-E&R (gross motor function classification system – expanded and revised); CPVD (cerebral palsy with visual dependence); CPVI (cerebral palsy with visual independence); DF (Dorsiflexion); GAS (gastrocnemius); HAM (hamstring); PF (plantar-flexion); QUAD (quadriceps); RFT (rod and frame test); SD (standard deviation); TD (typical developing); VOR (vestibulo-ocular reflex);

^AVOR function is determined based on the difference score of logMAR between the static and dynamic visual acuity tests. If d(logMAR) is equal or greater than 0.2, VOR dysfunction is suggested.

^bGMFCS-E&R is a 5-level classification system that describes the gross motor function of children with cerebral palsy up to 18 years of age on self initiated movement with the focus on walking ability in the current study. Our evaluation is based on the description made for the age band of 12–18 years of age. *Level I* represents the ability to walk without restrictions; *Level II* represents the ability to walk with some limitation especially in some of the more advanced motor skills such as running, jumping, and climbing up and down stairs.

^cLight touch sensation was tested with Semmes Weinstein monofilament examination on the plantar part of big toes and lateral border of the feet. The test force includes 2g, 4g, and 10g.

^dPain sensation was evaluated using the pinprick test on the plantar part of big toes and lateral border of the feet.

^eVibration sensation was evaluated using the 128 hertz tuning fork on the ball of both heels.

^fCrouch stance was present if the knee flexion during quiet stance exceeds 15°.

^gMuscle spasticity was evaluated using modified Ashworth scale.

^hMuscle strength was evaluated using manual muscle testing with the scaling range from 0 (none) to 5 (normal).