Dynamics of Postural Control in the Child with Down Syndrome

ANNE SHUMWAY-COOK
and MAJORIE H. WOOLLACOTT

We examined the development of neural control processes underlying stance balance in both developmentally normal children and children with Down syndrome to test the hypothesis that motor deficiencies in children with Down syndrome are associated with deficits within the automatic postural control system. We compared children with Down syndrome and developmentally normal children in two age groups (1–3 and 4–6 years) by using displacements of a platform and measuring electromyograms from leg muscles. The automatic muscle response pattern in both normal children and children with Down syndrome were directionally specific, although the pattern were more variable than in adults. Responses in children with Down syndrome showed no adaptive attenuation to changing task conditions. Onset latencies of responses in children with Down syndrome were significantly slower than in normal children. Presence of the monosynaptic reflex during platform perturbations at normal latencies suggests that balance problems in children with Down syndrome do not result from hypotonia, which researchers have defined as decreased segmental motoneuron pool excitability and pathology of stretch reflex mechanisms, but rather result from defects within higher level postural mechanisms.

Key Words: Child development, Down syndrome, Muscle hypotonia, Posture.

When motor development of the child with Down syndrome is compared with that of the developmentally normal child, a consistent delay is observed in the acquisition of both postural and voluntary components of motor control. Cowie and Molnar studied motor control in a group of mentally retarded children and found both a delay in emergence of postural adjustments and increased variability in age of onset of postural adjustments. In tests of gross motor skills, children with Down syndrome performed consistently below their normal peers; their worst performance was in static and dynamic balance. Neuromuscular abnormalities in children with Down syndrome, which have been observed to be coincident with developmental delays, include generalized muscular hypotonia, the persistence of primitive reflexes beyond their normal disappearance with age, and slowed reaction times during voluntary movement.

Cowie and Molnar have hypothesized that the child with Down syndrome has a predominance of primitive, spinally controlled muscle-response patterns over more centrally integrated and coordinated movement patterns for the following reasons: poor myelination of the descending cerebral and brain-stem neurons and a reduction in both the number and connections of neurons in the higher nervous centers, such as motor cortex, basal ganglia, cerebellum, and brain stem. Based on behavioral observations of developmental delay, clinicians have conducted a number of research projects involving early therapeutic intervention for children with Down syndrome. These studies have attempted to facilitate normal mental and motor development through a variety of stimulation techniques with mixed results.

Although these and other studies accurately describe the behavioral deficits and delays in children with Down syndrome, few studies have explored the specific motor control deficits that could underlie postural instability and subsequent developmental delay in motor coordination. Many researchers attribute deficits in motor skills to underlying hypotonia. The physiological basis for hypotonia has been defined as decreased segmental motoneuron pool excitability and pathology of the stretch reflex mechanism. Recent work by Davis and Kelso casts doubt on this hypothesis. In tests of the ability of subjects with Down syndrome and with normal development to set or modulate voluntarily muscle stiffness, they found the two groups comparable. Although the subjects with Down syndrome did show altered movement characteristics, such as increased movement times and oscillation about the final end position, the mechanism underlying stiffness was normal.

Studies on the organization of stance postural control have shown that normal children (14 months to 10 years) and healthy adults compensate for externally induced body sway in the sagittal plane through the activation of automatic postural responses. These responses are characterized by stereotyped patterns of muscle contractions in the leg and trunk with onset latencies on the order of 100 msec. These longer latency postural responses have been shown to be more effective than the normally suppressed monosynaptic stretch reflex in returning the center of mass to within the base of support. In addition, perceptual processing associated with...
postural stability involves the integration of redundant sensory inputs from support surface somatosensory, visual, and vestibular systems. During normal development, the controlling inputs to posture apparently shift from visual dependence to a more adult-like dependence on a combination of somatosensory and visual inputs at 4 to 6 years of age. Processes responsible for resolving multimodal sensory conflict are not fully developed before the age of 7 years.

The purpose of this study was to test the hypotheses that deficits in static and dynamic balance skills found in children with Down syndrome are, in part, the result of abnormalities within the automatic postural control system. Experiments were designed to 1) quantify deficiencies in the long latency automatic postural response system, specifically those processes that determine spatial and temporal structure within stereotyped muscle synergies; 2) determine if deficiencies exist in the sensory integration mechanisms underlying the organization of orientation information from somatosensory, visual, and vestibular systems; and 3) determine the existence of higher center modulation over spinally mediated stretch reflex action. Based on a review of pertinent literature, we generated the following hypotheses: 1) Contrary to developmentally normal children, children with Down syndrome will show major disorganization in the automatic postural control system as shown by an inability to structure appropriately spatial and temporal aspects of muscle action and will not show appropriate inhibition of spinal reflex activity and 2) consistent with developmentally normal children, children with Down syndrome will be more dependent than adults on visual orientation inputs than support surface somatosensory inputs and will be unable to solve problems of postural stability resulting from incongruent orientation inputs.

METHOD

Subjects

We tested a total of 17 children, aged 15 months to 6 years (11 developmentally normal children and 6 children with Down syndrome). Our criteria for selection of the children with Down syndrome was presence of trisomy-21 or mosaic Down syndrome, normal vision and hearing, absence of congenital heart defects, no history of seizures, absence of current medications, and independence in stance or ambulation. The researchers explained the purpose, procedures, risks, and benefits of the study to parents, who gave their informed consent.

We divided the normal children and children with Down syndrome into two groups: 1) a group of older children, aged 4 to 6 years, four children with Down syndrome and six normal children, who were given all experimental tests and evaluation procedures; and 2) a group of younger children, aged 15 to 31 months, two children with Down syndrome and five normal children, who were evaluated on only the simpler experimental procedures. We previously studied postural response patterns in normal children aged 7 to 10 years and healthy adults.

Initial Clinical Evaluations

After selection, but before experimental sessions, each child with Down syndrome in the older group was independently, clinically assessed to determine the presence of developmental delays by two pediatric therapists experienced in evaluating and treating children with developmental disabilities. They used the Bayley Scales of Infant Development and the Brigance Diagnostic Inventory of Early Development (birth to 7 years) as their developmental and motor control tests. The children with Down syndrome functioned 18 to 24 months behind their age level with significant performance decrements in both static and dynamic balance tests. All children were judged moderately hypotonic by clinical assessment based on tests of resistance to passive stretch, joint extensibility, muscle consistency, and antigravity posturing.

The Table presents a summary of the clinical evaluation results. When we normalized the data for variability in chronological age, all four older children with Down syndrome (three trisomy-21 and 1 mosaic Down syndrome) performed at the same overall developmental level, which demonstrated a surprising homogeneity of behavioral performance across these children.

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* Years

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Apparatus

We used a hydraulically controlled platform capable of both horizontal (anterior-posterior [AP]) and rotational movements (about an axis colinear with the ankle joint) to elicit postural responses. The platform consisted of a base plate (50 cm x 52 cm) suspended at the four corners on strain gauge sensors, which measured total load (± 0.005 kg/m linearity ± 5%) and torque (the difference between the forces detected by the anterior and posterior strain gauges). Anterior or posterior platform displacements were one-half sine waves.
We measured AP sway through the use of a rod attached to the subject’s hips with a strap and connected to the base of the platform by a potentiometer.

We completed high speed (100 frames/sec) film analysis using a Kodak camera placed 7 m from the primary sagittal plane of action with the optical axis set at a height to maximize the subject within its field of view. We smoothed cinemographic data using a cubic spline smoothing process and digitized every fifth frame. Our biomechanical analysis included quantification of joint angles at hip, knee, and ankle during initial stance and subsequent sway. Sway motion was separated into two components: 1) platform-induced motion and 2) compensatory motion. The data were represented graphically with stick figures constructed from digitized data points.

Procedures

We tested each child during at least three separate 45-minute sessions, which were conducted during different weeks to assure replicability of observations over time. The first two sessions were used for evaluation of postural responses to platform translations and rotations. Ten anterior and 10 posterior horizontal platform translations were sequenced randomly during the session. In addition, we used three linear sequences of five ankle dorsiflexing perturbations to study the adaptation mechanisms of each child.

In the final session, we examined postural responses under varying sensory conditions in the children above 3 years of age. Children below 3 years of age would not tolerate the unstable support surface and eye closure without crying. We asked the older children to stand for five seconds under four different sensory conditions. First, they stood on a normal stable platform surface with eyes open (S
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\(_n\)). Second, they stood on a normal platform surface with eyes closed (eye closure was monitored by one of the researchers) (S
\(_n\)V
\(_c\)), a situation representing loss of redundancy among sensory inputs but not intersensory conflict. Third, they stood on a platform surface in a servomechanism state, vision normal (S
\(_v\)V
\(_n\)), a condition in which the platform was rotated in direct proportion to AP sway motion as measured by the potentiometer. This procedure minimized orientational input from ankle and feet by maintaining a fixed 90-degree ankle joint angle despite body sway; as a result, support surface inputs were perceptually correct but incongruent with orientationally correct visual and vestibular inputs and resulted in an intersensory conflict situation. Fourth, we asked children to balance with their eyes closed (S
\(_v\)V
\(_c\)), platform surface in a servomechanism state. This condition provided orientationally incorrect somatosensory inputs in conjunction with an absence of visual inputs; left only orientationally correct vestibular inputs to mediate balance; and, thus, represented both a measure of vestibular input efficacy and a multimodal sensory conflict.

Data Analysis

We analyzed muscular responses to platform tests by using rectified and filtered EMGs. We determined the EMG onset latency by visual inspection and EMG amplitude by numerically integrating the first 76 msec of the EMG response, which provided a relative measure of muscle contractile activity. We used the Pearson product-moment correlation (significance level of .05) to express the stability of the amplitude relationship between distal and proximal muscles.

To define stability objectively in response to postural perturbations, we created a “Stability Index” from body sway measures. We did this by numerically integrating potentiometer data and scaling it to each child’s theoretical limit of sway so that increasing sway excursions corresponded to increasing scores on the Stability Index with a score of 100 representing loss of balance. We determined theoretical limits of sway by first measuring the height and length of the support base of each child and calculating the center of mass. Then, we calculated the maximum angle of sway possible by using an arc tangent function to relate height of the center of mass to base of support.

Because of the small sample size and the lack of homogeneity of variance, we used the Mann-Whitney U test on all tests of statistical significance.

RESULTS

To provide a perspective for the results of this study, selected responses of our children will be compared throughout this section with the responses of adults and children from our previous studies. Gurfinkel et al have shown that in healthy adults, the excitability level of the myotatic reflex is considerably reduced in the upright, standing posture. In our study, four developmentally normal children, ages 4 to 6 years, showed incomplete suppression of the monosynaptic stretch reflex in response to dorsiflexing rotational perturbations in 40% of the trials. The four children with Down syndrome also showed incomplete inhibition of the stretch reflex during stance in 40% of the trials.

In response to platform translations, both normal children and children with Down syndrome produced postural response synergies, which were appropriately directionally specific, although more variable than the postural synergies of adults. Figure 1 displays EMG activity from four lower extremity muscles in a normal child and a child with Down

![Fig. 1. Electromyographic activity in response to a forward sway translation.](image-url)
TEMPORAL DELAY
DISTAL-PROXIMAL MUSCLES

Fig. 2. Comparison of temporal delay between distal and proximal muscles in normal children and children with Down syndrome. Zero millisecond represents time of onset for distal muscles.

MUSCLE AMPLITUDE CORRELATIONS

Fig. 3. Distal-proximal muscle amplitude correlations for adults, normal children, and children with Down syndrome.

Both children with Down syndrome and normal children displayed an increased temporal delay in the recruitment of proximal muscles in comparison with adults. Figure 2 shows the temporal delay between distal and proximal muscles in adults, normal children, and children with Down syndrome in response to a forward sway (gastrocnemius-hamstring muscles) and backward sway (tibialis anterior-quadriceps muscles) perturbation. In adults, proximal muscles were activated 10 to 25 msec later. In normal children, proximal muscle delays of 36 ± 18 msec in forward sway and 58 ± 16 msec in backward sway were common. Children with Down syndrome showed a delay of 57 ± 13 and 36 ± 17 msec for forward and backward sway, respectively. The children's delays were significantly slower in comparison with the adult data (p < .01) but not with respect to one another.

Synergic muscles in adults are tightly coupled and, despite trial to trial amplitude variations, contract in fixed proportion to one another to yield a high correlation (r = 85) between amplitudes of related muscles. Significantly lower (p < .001) intermuscle amplitude correlations were found for both forward and backward sway synergies for normal children (r = .45) compared with adults and for children with Down syndrome (r = .22) compared with normal children. Figure 3 presents the correlation data for gastrocnemius and hamstring muscles in response to forward sway perturbations from six adults previously studied, six normal 4- to 6-year-old children, and four 4- to 6-year-old children with Down syndrome.

The onset latencies of postural muscles of children with Down syndrome were significantly slower (p < .001) than normal children in response to externally produced perturbations to balance. Normal children aged 4 to 6 years were slightly slower than adults (112 ± 12 msec in comparison with 100 ± 4 msec, p < .01). Children with Down syndrome were significantly slower than adults (136 ± 32 msec in forward sway and 162 ± 40 msec in backward sway, p < .001). Figure 4 displays the average distal muscle onset latency for all three groups to both forward and backward sway perturbations.

Figure 5 compares 3 sets of muscle response patterns involving gastrocnemius and hamstring muscles in two children syndrome in response to a platform perturbation producing forward sway. The movement backwards of the platform caused the child to sway forward; a resultant primary response in gastrocnemius and hamstring muscles brought the center of mass back to a point of equilibrium within the base of support. A secondary response in the antagonist muscles, the tibialis anterior and the quadriceps femoris, occurred because of an initial overcorrection. Gross sequencing of muscles in response to platform movements was comparable in both normal children and children with Down syndrome, although children with Down syndrome showed subtle differences in timing and force relationships between synergic muscles. In addition, both groups of children showed low levels of tonic background EMG activity in the muscles recorded.
with Down syndrome (aged 22 months) who had been walking one week and one month, respectively, to two normal children. The normal children consisted of a chronologically age-matched child (22 months old) and a developmentally age-matched child (walking for one month). In the child with Down syndrome who had been walking one week, postural responses to platform-induced sway were poorly organized. No response was found in four of the eight trials (falls resulted); in the remaining trials, the response was limited to activation of the distal muscle of the appropriate synergy. Latencies were also quite slow (gastrocnemius muscle = 160 msec, tibialis anterior muscle = 190 msec). The 22-month-old child with Down syndrome who had been walking one month showed postural response patterns that were only slightly better organized. Distal muscle activation of the appropriate synergy was consistently present at latencies between 140 and 160 msec. Proximal muscle coupling was only occasionally present. These results were in clear contrast with both normal children who showed consistent synergic activation of both distal and proximal leg muscles at significantly (p < .01) faster latencies (100 ± 12 msec).

Figure 6 is a graphic representation constructed from digitized data points that present body motion associated with platform-induced backward sway and compensatory muscular action. Our biomechanical analysis of joint angle changes associated with platform-induced body motion indicated that both normal children and children with Down syndrome, like adults, sway initially like inverted pendulums, with motion principally about the ankle joints (Fig. 6A). Unlike adults, normal children and children with Down syndrome, aged 4 to 6 years, showed considerable differential motion at knee and hip during compensatory postural adjustments. Figure 6B displays body motion associated with compensatory muscle action in response to backward sway in a normal child. It illustrates the muscle action that brings the center of mass from its backward position forward within the base of support buckling at both knees and hips. Similar responses were seen in the children with Down syndrome.

Figure 7 compares average sway to a forward and backward postural perturbation across the two age groups of normal children and children with Down syndrome. In response to both forward and backward platform translations, the normal children aged 15 to 31 months swayed on the average closer to their limits of stability than did normal children aged 4 to 6 years (p < .01). The children with Down syndrome aged 4 to 6 years swayed on the average closer to their limits of sway than did normal children aged 4 to 6 years (p < .01).

Both normal children and children with Down syndrome aged 4 to 6 years showed postural response patterns to rotational perturbations, which indicated that somatosensory inputs in isolation were sufficient to bring automatic postural responses to threshold. Normal children, however, showed adaptive attenuation of postural responses to rotational perturbations within 15 trials but the children with Down syndrome did not adapt. The two youngest children with Down syndrome (20 months) were similar to healthy children under the age of 3 years by not showing long latency postural responses and losing balance in response to all rotational
Clinical evaluation of the four older children with Down syndrome indicated they were functioning 18 to 24 months behind age level with significant performance decrements in both static and dynamic balance tests. This evaluation is consistent with existing literature documenting delays in the longitudinal development of children with Down syndrome. All four children with Down syndrome were determined to be moderately hypotonic, a finding also consistent with existing literature. The presence of both tonic activity during standing and of short latency myotatic reflexes in response to platform rotations at latencies comparable to the latencies of normal children was not consistent with clinical findings of hypoactive deep tendon reflexes and hypotonia defined as reduced motoneuron pool excitability. Rather, these data support Davis and Kelso in suggesting that children with Down syndrome show muscle stiffness and motoneuron pool excitability comparable to that of normal children.

In addition, the presence of short latency myotatic reflexes (normally suppressed in the standing adult) in both normal children and children with Down syndrome lends little support to the hypothesis that children with Down syndrome show decreased higher center modulation of more primitive patterns controlled at lower centers.

Children with Down syndrome produced directionally specific, postural response patterns in response to external disturbances to stability. These response patterns were similar to those of normal children aged 4 to 6 years, although the patterns were more variable. Onset latencies in children with Down syndrome were significantly slower and resulted functionally in increased body sway and, in some instances, loss of balance. Delayed activation of postural responses in children with Down syndrome could not be attributed to reduced segmental motoneuron excitability in light of 1) normal myotatic latencies and 2) presence of low level tonic background activity in many trials, which was indicative of suprathreshold motoneuron excitability.

The presence of myotatic reflexes at normal latencies in conjunction with significant delays in long latency postural responses presents an interesting paradox. Currently held syndromes. Under the first condition where all three inputs to the postural control system were present (SₘVₙ), children with Down syndrome were significantly ($p < .05$) more unstable than normal children or adults, although they were still well within their limits of stability. When redundancy of inputs was reduced by removing visual inputs through eye closure (SₘVₙC), both normal children and children with Down syndrome showed significantly diminished stability ($p < .05$) when compared with adults, although again the children were well within their limits.

When the support surface was rotated in direct proportion to AP sway, the fixed ankle-joint angle and somatosensory inputs that resulted were incongruent with visual and vestibular inputs (SₘVₙ). Two of the four 2- to 4-year-old children with Down syndrome fell, and one of the six 2- to 4-year-old normal children fell. When the removal of visual inputs left only vestibular inputs and orientationally incorrect somatosensory inputs to mediate balance reactions, three of these four Down syndrome children fell. Four of these six normal children fell under the same condition.

**DISCUSSION**

The presence of myotatic reflexes at normal latencies in conjunction with significant delays in long latency postural responses presents an interesting paradox. Currently held.
views in rehabilitation literature that attribute developmental delays and balance problems in children with Down syndrome to decreased segmental motoneuron pool excitability and pathology of the stretch reflex mechanism leading to hypotonia should be questioned. Rather, our data are consistent with results from studies that examined the effect of cerebellar lesions on long latency postural responses. Those studies found normal short latency (myotatic) responses but delayed long latency postural responses in patients with cerebellar lesions and in animals with reversible cooling of the cerebellar nuclei. In addition, results from our study support Burke who critically questioned the validity of traditional views on the role of the muscle spindle in disorders of muscle tonus.

We found considerable difference in the organization of postural patterns in very young children with Down syndrome in comparison with very young normal children. Unlike normal children under 3 years of age who showed reasonably consistent, though unmodulated sway synergies, in response to platform translations, the two 22-month-old children with Down syndrome showed very inconsistent, poorly organized, and quite slow sway responses. This difference in postural response organization between young normal children and children with Down syndrome is particularly surprising when one examines the ontogenesis of postural control in normal children. The normal child under 3 years of age appears to have more consistently organized and less variable postural responses than does the normal 4 to 6 year old. The young child’s inability, however, to modulate postural responses appropriately can result in instability as a result of overcompensation and subsequent body oscillation.

The finding that postural synergies in the two young 22-month-old children with Down syndrome were more poorly organized than in the 4- to 6-year-old children with Down syndrome lends support to the concept of a difference in the ontogenetic development of postural control between children with Down syndrome and normal children. Possibly, equilibrium problems in children with Down syndrome are not just the result of delayed, albeit normal, development but, in fact, represent a difference in the evolution and development of postural control. More research on the development of postural control in children under 3 years of age is needed to confirm this possibility.

Results from experiments that tested balance under altered sensory conditions demonstrated that children with Down syndrome, like normal children aged 4 to 6 years, had difficulty maintaining balance with loss of redundant sensory inputs and that situations presenting the greatest threat to stability were those with incongruent inputs representing multimodal sensory conflict. This study cannot determine, however, whether the development of organizational processes underlying resolution of multimodal sensory conflict are absent or delayed in the child with Down syndrome. To clarify this question, further work with children with Down syndrome above the age of 10 is necessary because these processes do not normally mature until 7 to 10 years of age. Platform rotations, like translations, result in ankle rotation and stretch to ankle muscles but without the concomitant shift in the center of mass and resultant body sway. As a result, although somatosensory inputs from lower extremities associated with translation are congruent with visual and vestibular inputs in signaling body sway, somatosensory inputs secondary to rotation are in conflict with vision and vestibular inputs, which do not signal sway. The postural response that occurs in the stretched muscle to ankle rotation, in the absence of actual body sway, is destabilizing and, in the normal adult and child above 7 years, attenuates within three to five trials. Lack of attenuation of inappropriate postural responses during rotational perturbation trials suggests that the organizational processes underlying adaptation of postural response patterns to changing task demands may be less developed in the 4- to 6-year-old child with Down syndrome than in the normal child. These data are consistent with previous work on abnormalities of stance balance control in patients with cerebellar pathology.

CONCLUSIONS AND THERAPEUTIC IMPLICATIONS

This study found that children with Down syndrome under age 6 years demonstrate deficits in the postural control system that may provide a partial explanation for functional balance problems common to these children. Postural responses to externally induced loss of balance were present but slow and, as a result, often insufficient for reestablishing and maintaining stability. In addition, organizational processes subsuming the adaptation of postural response patterns to changing environmental contexts were poorly developed. Because the children involved in this study were under the age of 10 years, this study cannot clarify whether processes responsible for resolution of conflict among orientation inputs to postural control are normal or abnormal in children with Down syndrome. These results suggest, however, that therapeutic remediation of balance problems in children with Down syndrome should focus on two primary areas: 1) assisting children in the development and refinement of postural synergies, specifically enhancing motor coordination by improving the spatiotemporal coupling between multiple muscle groups that act together and 2) improving the organizational processes responsible for adapting postural response patterns to changing task conditions.

We should emphasize that neither the process of formation of postural synergies or that of integration of sensory inputs is voluntary or conscious. This is an important understanding when considering therapeutic intervention. The maintenance of stability requires the execution of fast (automatic) postural responses with onset latencies below those of voluntary reaction time responses. Techniques that rely on voluntary or consciously acquired balance responses probably will not ensure stability unless the learned response becomes automated (ie, not requiring conscious processing). The same may be said for strategies for adapting to multimodal sensory conflict.

Our research suggests that hypotonia defined as decreased segmental motoneuron pool excitability and pathology of stretch reflex mechanisms may not be the controlling factor in slowed or delayed postural responses. This may explain why pharmacological and certain therapeutic approaches that focus on improving muscle tonus in children with Down syndrome, nonetheless, show little functional gains in the acquisition of developmental skills.
REFERENCES