Neuromuscular Processes in the Control of Posture in Children with Developmental Coordination Disorder: Current Evidence and Future Research Directions

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Abstract
Purpose of review: The ability to control body position in space is fundamental to all daily activities. This review article summarizes the neurological and muscular deficits that are associated with poor postural control in children with developmental coordination disorder (DCD) and includes suggestions for future research work.
Recent findings: The atypical neuromuscular processes in the control of posture in children with DCD include abnormal structures and functions of the central nervous system, sensory organization disorders, abnormal recruitment of postural muscles, slow muscle contraction and lower maximum muscle strength of the legs. Inattention can further compromise neuromuscular processes and postural control performance in these children.
Summary: Atypical neuromuscular processes in static, reactive and anticipatory postural control have been reported in children with DCD. These include atypical structures and functions of the central nervous system, sensory organization disorders, abnormal recruitment pattern of postural muscles, slow muscle contraction speed and lower maximum muscle strength of the legs. Functional movement-power training is a recommended treatment strategy as it can address both the neuromuscular deficits (lower peak force and longer time to reach peak force in the knee muscles) and atypical standing balance responses of children with DCD. Future studies could explore the neuromuscular processes associated with adaptive postural control, an under-examined area, and effective rehabilitation interventions to improve functional balance control and the associated impairments in children with DCD.

Keywords: developmental disorder; motor deficit, neurological deficit; body balance; future research; review

Introduction

Among the many motor deficits of children with developmental coordination disorder (DCD) [1], poor postural control (balance) is probably the most concerning because it predisposes children to falls [2], affects their motor skills development [3●], reduces physical activity, increases the risk of obesity, and compromises physical fitness and wellbeing [4]. Between 73% and 87% of children with DCD experience balance difficulties in their daily lives [5]. DCD per se and the associated balance difficulties are quite disabling and may persist throughout life [6]. Therefore, understanding the atypical neuromuscular processes underlying the control of posture in these children is essential for the design and prescription of appropriate rehabilitation treatments [7].

This review article summarizes the growing research base from around the world that examines the neurological and muscular deficits that are associated with poor balance performance in these children and includes suggestions for future research directions to address the knowledge gaps. The neurological and muscular impairments can be categorized into two main types: central nervous system (CNS) deficits and peripheral neuromuscular system deficits. Research bearing on these categories is critiqued below.

Central nervous system deficits

Abnormal structures and functions of the CNS

Recent neuroimaging studies have revealed that motor impairments, including those to balance, in children with DCD are associated with microstructural abnormalities and dysfunction in various parts of the brain. The exact neurological underpinnings include lower activation in the left superior and inferior parietal lobules [8], reductions in functional connectivity in a number of brain regions involved in motor circuitry (e.g., the primary motor cortex, inferior frontal gyri, striatum and angular gyri) [9], atypical patterns of electroencephalographic cortical activation [10-14], lower axial diffusivity of the corticospinal tract and posterior thalamic radiations on diffusion tensor imaging [15], abnormalities in the parietal regions of the corpus callosum that connect to both primary and somatosensory motor areas [16], cortical thinning in the frontal, parietal and temporal lobes [17] and a disruption of the cerebello-cerebral network [18●,19●●]. Among these atypical patterns of neural activation and structure, cerebellar dysfunction seems to be the major contributor to poor motor control including balance control [20●,21●], as children with DCD perform poorly on traditional cerebellar function tests (e.g., finger-to-nose touching test and rapid alternating hand movements) [22] and demonstrate poor balance performance [18●,20●,21●], which corroborates the neuroimaging results. Indeed, a recent fMRI study show that children with DCD demonstrated under-activation in right cerebellar crus I, left cerebellar lobule VI and left cerebellar lobule IX during skilled motor tasks [23]. Given the cerebellum's pivotal role in motor control including postural control, cerebellar dysfunction is likely a key contributor to atypical postural control of children with DCD [21●].

Sensory organization disorder

The ability to maintain body balance in different postures or during movement requires optimal reception, processing and integration of sensory signals from the somatosensory, visual and vestibular systems. Using a computerized dynamic posturography machine, our research team has confirmed that the sensory organization of balance control is compromised in children with DCD. That is, their ability to integrate and appropriately select visual, vestibular and somatosensory information in the CNS to control posture is below the age-matched norm [24●●-27●●]. Children with DCD show difficulty in utilizing visual and vestibular inputs, and in re-weighting (increasing the use of) somatosensory inputs to

maintain body balance [24●●,25●●,27●●,28]. Sensory re-weighting refers to the process of integrating visual, vestibular and somatosensory information utilized for postural control that is dynamically regulated in response to changing environmental conditions and the availability of the senses. Re-weighting of sensory information is particularly important under challenging environmental conditions (e.g., standing in a moving bus) or when there is a loss of one or two senses (e.g., when the vision is blocked) [29]. Therefore, children with DCD demonstrate greater postural sway when standing in challenging sensory environments such as conditions 5 and 6 of the EquiTest sensory organization test (only the vestibular input was accurate and useful) [24●●,25,27●●]. The six test conditions of the sensory organization test and the sensory signals are described in table 1.

<< Table 1 >>

Peripheral neuromuscular deficits

Apart from CNS deficits, children with DCD also exhibit peripheral neuromuscular deficits. These deficits include abnormal recruitment of postural muscles, slow muscle contraction and lower maximum muscle strength of the legs, which can affect different types of postural control. The following paragraphs describe the various neuromuscular deficits that have been found in children with DCD during different types of balance task.

Static postural control and the associated neuromuscular deficits

It is widely acknowledged that postural stability in a fixed stance not only requires reliable visual, vestibular and somatosensory information, but also appropriate motor (balance) responses including hip and ankle strategies to maintain anterior–posterior postural stability [30●,31]. Our research team first discovered that children with DCD tend to use a hip strategy excessively when forced to rely on vestibular inputs to maintain postural stability when standing [24●●]. This is considered an ineffective balance response because it will increase energy consumption for postural control and increase the risk of falling [32]. We found that the primary neuromuscular cause of the increased reliance on the hip strategy in children with DCD is prolonged hamstring muscle force production [27●●]. When the muscles of the hamstring (a major hip extensor) cannot quickly produce enough force to control the forward sway of the trunk in standing, excessive hip flexion (hip sway) can result, as is observed in children with DCD [27●●]. Other possible neuromuscular explanations for overreliance on a hip strategy for balance could be lower knee muscle peak force and increased knee flexor and extensor co-activation [33●]; less steady muscle force production in the legs [34]; and increased and prolonged activation or co-contraction of the ankle muscles when standing [35,36●]. All these neuromuscular deficits may alter the lower-limb kinematics of standing balance control and might result in excessive hip sway in children with DCD. Certainly, more physiological data should be collected to confirm these neuromuscular causes of atypical postural control patterns in children with DCD.

Reactive postural control, functional balance performance and the associated neuromuscular deficits

Previous studies of reactive postural control (the ability to maintain postural stability when balance is unexpectedly challenged [37]), have revealed that inconsistent timing of postural muscle activation is the major neuromuscular event underlying problems in reactive balance performance in children with DCD [38,39]. These children displayed an abnormal proximal-to-distal muscle activation pattern on electromyographic measurement in response to unexpected (platform) balance perturbations instead of the normal distal-to-proximal muscle activation pattern [40,41]. This atypical muscle activation timing will result in greater

displacement of the center of gravity before the appropriate leg muscles contract to return the center of gravity to its original position [42]. Such neuromuscular deficits significantly contribute to the inferior reactive balance performance in children with DCD, leading to postural instability and a greater risk of falling in daily life [2].

Our recent study has further revealed that prolonged muscle activation onset latency in the gastrocnemius (mean difference between DCD group and control group = 19.72ms) can adversely affect functional balance performance in DCD for activities such as throwing and catching a ball while maintaining postural stability (ball skills subscore as measured on the Movement Assessment Battery for Children (MABC)). Specifically, gastrocnemius muscle activation onset latency explained 11.4% of the variance in the MABC-derived ball skills subscore [43●●]. In addition, lower gastrocnemius peak force was associated with the poor dynamic balance performance (e.g., hopping, walking with heels raised and tandem walking) and MABC ball skills performance of children with DCD [43●●]. These results indicate that both the amplitude and timing of gastrocnemius muscle contraction were compromised in this group of children, and that could have weakened their base of support and thus influenced their balance performance during functional activities.

Anticipatory postural control and the associated neuromuscular deficits

Current evidence on anticipatory postural control (i.e., postural adjustment made in advance of planned movement) suggests that children with DCD demonstrate a delay in the onset times of the tibialis anterior, external oblique and internal oblique muscles while kicking a ball, climbing stairs and during single-leg standing compared with typically developing children [44●]. In addition, during goal-directed reaching movement, an anticipatory function was not present in most of the anterior trunk muscles in children with DCD. Altered postural muscle activation may thus explain the poor anticipatory postural control in this group of children [40]. Indeed, a recent review article written by Wilson et al. [19●●] also suggests that children with DCD have reduced automatization of muscle synergies that support postural control. This could be explained by their poor internal modeling or predictive control.

Inattention, neuromuscular deficits and postural control

Emerging evidence indicates that postural control requires not only normal neuromuscular performance, but also (mental) attention [45-47]. Attention can also influence the neuromuscular performance associated with postural control [48-51]. For example, focusing attention on a movement effect (external focus of attention) can lead to more efficient motor unit recruitment patterns, such as reduced co-contraction of the agonist and antagonist muscles when performing a functional task [48]. The coordinated recruitment of muscle fibers can in turn enhance the timing of force production [49]. Additionally, attention modulates activity in the cortical sensorimotor areas [50] and improves the use of the sensory feedback that guides the motor responses [51]. Attention is therefore crucial in postural control as it directly influences balance and balance-related neuromuscular performance in children both with and without disabilities.

Previous studies have reported a significantly higher incidence of attention deficits in children with DCD compared with children with typical development [52,53]. Inattention in children with DCD could further compromise their neuromuscular performance and balance functions though it is often overlooked by pediatricians and psychiatrists [54]. Our recent study revealed that children with DCD have inferior motor and balance performance and are less attentive to manual dexterity, ball skills and balance movements (as measured on the

MABC) than are their typically developing peers [47]. Laufer et al. [45] also reported that children with DCD demonstrated greater postural sway (center of pressure path-length velocity) than did children with typical development during static standing while performing a cognitive task. Collectively, these findings suggest that attentional deficits (concurrent performance of cognitive and motor tasks) exacerbates the postural control issues seen in children with DCD.

Future research directions

Adaptive postural control of children with DCD

It is well known that postural control requirements vary with different tasks and environments [55]. Therefore, children with DCD need to acquire different types of balance ability including (1) maintaining balance in a static posture (static postural control); (2) maintaining balance in response to a postural perturbation (reactive postural control); (3) adjusting the posture before initiating a movement (anticipatory postural control); and (4) modifying postural muscle responses in a timely way in response to changing task demands (adaptive postural control) [55]. At present, most DCD studies have focused on the first three types of postural control. No study has yet examined adaptive postural control ability in children with DCD. As neuroimaging data suggest cerebellar dysfunction in children with DCD [18●,20●,21●] and cerebellar pathology is known to affect the grading or scaling of muscle force output to postural perturbations of varying size [56], it is logical to hypothesize that children with DCD will manifest impaired postural muscle responses and abnormal adaptive balance performance. Therefore, further studies may explore the neuromuscular processes associated with adaptive postural control in children with DCD.

Rehabilitative interventions

In addition to investigating the postural control deficits in children with DCD, designing appropriate treatment programs to remediate their impairments and balance dysfunction is also very important. Most current treatment regimens focus on the induction of neuroplastic changes in the CNS by means of task-specific training [57]. Less emphasis has been placed on the treatment of the peripheral neuromuscular impairments mentioned above that can also adversely affect postural control [15,58,59]. We postulate that balance interventions for children with DCD should address both the CNS and peripheral neuromuscular deficits to maximize the treatment effect. It is known that functional movement training can induce neuroplastic changes in the CNS and muscle power training can increase speed of muscle force production and reduce the peripheral neuromuscular deficit. Therefore, we have devised and tested a novel functional movement-power training program for children with DCD to improve both their neuromuscular deficits (e.g., lower peak force and longer time to reach peak force in the knee muscles) and balance strategies [58]. Further clinical trials could explore interventions to improve the recruitment pattern/timing of postural muscles, gastrocnemius muscle contraction force and speed and, more importantly, attention during balance tasks in children with DCD.

Conclusions

The atypical neuromuscular processes in the control of posture in children with DCD include atypical structures and functions of the CNS, sensory organization disorders, abnormal recruitment pattern of postural muscles, slow muscle contraction speed and lower maximum muscle strength of the legs. Inattention may further compromise the neuromuscular processes and postural control performance in these children. Future studies could explore the neuromuscular processes associated with adaptive postural control, and the

translation of this basic research into effective rehabilitation interventions for improving balance control and the associated impairments in children with DCD.

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Compliance with ethical standards

Not applicable.

Conflict of interest

The authors declare that they have no conflicts of interest.

Human and animal rights and informed consent

Not applicable.

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●● Of major importance

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Table

Table 1. The six test conditions of the EquiTest sensory organization test and the sensory signals [24●●-27●●]

Condition	Description	Sensory signals disrupted	Accurate sensory signals available
1	Eyes open, fixed platform	None	Somatosensory, visual, vestibular
2	Eyes closed, fixed platform	No visual signals	Somatosensory, vestibular
3	Eyes open, sway-referenced surround, fixed platform	Conflicting visual signals	Somatosensory, vestibular
4	Eyes open, sway-referenced platform	Conflicting somatosensory signals	Visual, vestibular
5	Eyes closed, sway-referenced platform	Conflicting somatosensory signals, no visual signals	Vestibular
6	Eyes open, sway-referenced surround and platform	Conflicting somatosensory and visual signals	Vestibular