Review

Hypertonicity in Children with Cerebral Palsy: a New Perspective

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Objectives: Hypertonicity is a major problem in children with cerebral palsy that has not been explained effectively to address clinical decision making. Therefore, this article aims to provide a theoretical framework about hypertonicity for clinicians, rehabilitation and medical practitioners to be used in their practice.

Methods: Literature was reviewed to examine the new perspectives towards the hypertonicity and its signs and symptoms. Then, these symptoms were scrutinized to identify various aspects of the phenomena.

Results: The results of this review revealed various components of hypertonicity, including neural and biomechanical. Neural component was also classified into positive and negative symptoms. These components altogether influence gross and fine motor function and consequently disturb children in their daily activities.

Conclusion: Using term "spasticity" is not definitively enough to explain various aspects of the affected persons. Therefore, the term "hypertonicity" appears to be much appropriate to be used by professions in their daily practices. Furthermore, to have a very effective intervention, practitioners should consider all various signs and symptoms of hypertonicity that are explained in this review.

Key words: Hypertonicity, Cerebral palsy, Children

Introduction

As individuals with cerebral palsy (CP) present a large group of clients visited by occupational therapists, understanding clinical decision making in this context is very important. Moreover, the literature suggests that both pathology and diagnosis can influence clinicians' data collection and clinical reasoning, therefore it is necessary to understand these children in depth. Hypertonicity is the most prevalent type of CP (85%) and, therefore, clinical decision making occurs more frequently for this type of diagnosis. Various factors may influence clinical decision making including individual (e.g., range of movement, amount of spasticity), familial (e.g., family income) (1), and contextual factors (e.g., work place, hospital or school) (2). Research involving therapists with various level of expertise demonstrated that hypertonicity is the most important influential factor on their decision making (3-5). Therefore, this review aims to investigate the most prevalent type of CP - the hypertonic (spastic) -including its neural and biomechanical components. Prior to this review, CP will be explained and its classification will be discussed.

Cerebral Palsy

Cerebral palsy is the most common cause of movement disorders in children (6-8). As such, people with CP constitute a major client group for occupational therapists as both outpatients and those receiving short and long term rehabilitation services (9). CP is an umbrella term, used to describe a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain, arising in the early stages of its development (10). CP is a lifelong condition (11), usually associated with various other disabling abnormalities including: seizures, learning disabilities, communication and intellectual impairments, behavioural problems, feeding, visual, speech and hearing difficulties, as well as sensory impairments (6, 12-15). The severity of CP varies from person to person and constitutes a continuum. At one end of the continuum all muscles and body movements are severely affected (16), making the person functionally dependent. At the other end, the influence of the disorder is minimal and only mild neurological signs are evident.

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Classification of children with CP

Cerebral palsy is most commonly classified according to: (1) central control and area of brain involvement; (2) functional motor abilities; (3) nature and typology of motor disorder; and (4) anatomic distribution (17). The area of central control involves selective and automatic processes (18). Selective control refers to the pyramidal tracts in the central nervous system (CNS) that control selective movements and its lesions causes spasticity. Automatic control is ruled by extrapyramidal tracts and their lesions results in athetosis and ataxia. Damage to both tracts can cause a mixed presentation.

Classification on the basis of functional motor abilities aims to address the importance of evaluating the functional consequences of disabilities emphasised by the World Health Organization International Classification of Functioning, Disability and Health (ICF) (19). Two scales of objective function for upper and lower limb have been recently developed: the Manual Ability Classification System (MACS) and the Gross Motor Function Classification System (GMFCS). MACS aims to classify children with CP on the basis of how to use their hands when manipulating objects in daily activities and rank them into five levels on the basis of proficiency (20). The GMFCS is based on the concepts of abilities and limitations in gross motor functions. This classification is a reliable and valid system and similar to MACS classifies children with CP based on their age specific gross motor activity into five levels (21).

Involuntary movement disorders and tonality abnormalities provide the third concept for classification of CP (22). Tone is described as spastic (hypertonic), hypotonic, ataxic, athetoid, and/or mixed. Involuntary movement disorder is defined as any involuntary and uncontrolled movement that appears in the affected part of body. Muscle tone refers to the amount of resistance felt against passive movement, while an individual is attempting to be relaxed (23). This resistance is produced by neural and biomechanical mechanisms (24). Neural factors refer to the amount of contraction that is present naturally in the muscles as a result of tonic stretch reflex (24, 25). Biomechanical factors include physical inertia of the extremity as well as the elastic properties of tissues, joints, blood vessels, muscles etc (25, 26). Any resistance present in a relaxed person is mostly biomechanical and only some is related to neural factors (26).

The last classification of CP is based on anatomic distribution (22) that is traditionally called topography. This refers to the pattern and extent of motor involvement (27) regarding different anatomic areas including all body regions (i.e., trunk and limbs) (22). Such a classification can therefore be referred to as monoplegia in which one limb is affected (usually one arm); hemiplegia when one side of the body is affected (left or right side); diplegia where both upper and lower limbs are affected, but lower limbs are affected more severely than upper limbs; and quadriplegia where all limbs are affected (27).

Both the nature and typology of motor disorder and its anatomic distribution are usually employed to describe a person with CP, however, there is disagreement in sub-classifications, for example, between those used in Europe and Sweden. The European classification (28) was introduced by different teams from European countries in which CP was classified into three types: spastic, ataxic, and dyskinetic. Although this classification tries to distinguish and define different types of CP based on affected areas, it does not completely addresses the impact of CP in terms the number of limbs involved or the extent to which they are affected (i.e., diplegic versus quadriplegia).

Hypertonicity

Hypertonicity (spasticity) is the most prevalent estimated as high as 85% of CPcases, in Europe and Australia, and 94% in Northern Ireland (6, 29, 30). For the purpose of this article the term 'hypertonicity' will be used, because it is an umbrella term which subsumes a number of signs and symptoms consistent with central nervous system (CNS) lesions. These signs and symptoms usually occur together and seem to influence each other during task performance, for example when a person with hemiplegia wants to reach out to grasp a glass of water, she/he may experience resistance against the initial starting movement caused by spastic dystonia affecting the upper limb in a flexor pattern. When the movement starts, the agonist muscles trigger antagonist stretch reflexes and therefore, spasticity develops (e.g., elbow extension triggers stretch reflex in elbow flexors causing spasticity). Associated reactions may also occur in the other affected limbs. The clasp knife phenomenon (i.e., sudden muscle relaxation following initial resistance to movement in spastic muscles) manifests during movement usually at the end range.

Reciprocal inhibition may cause increased tone in finger muscles and prevent finger extension. These signs and symptoms cannot all be covered and defined by the term 'spasticity', thus requires a more comprehensive term such as 'hypertonicity'. Furthermore, the term hypertonicity promotes a more accurate assessment and observation covering all known signs and symptoms.

A wide variety CNS lesions may cause Hypertonicity- from proximal to distal - including: the motor or premotor cortex, the periventricular white matter, internal capsule, midbrain or pons, and the descending spinal tract (31, 32). Lesions to the central white matter are the most common cause of hypertonicity in CP (33). Lesions or any injury to the spinal cord do not come under the umbrella term of CP.

Following a CNS lesion a person usually moves through three phases: shock, a transition phase, and hypertonic state (26, 34). During shock, the spinal reflexes (e.g., stretch reflex and flexor reflex) are suppressed and flaccid muscle tone occurs for a period of time (26, 31, 34). This phase is followed by a transition stage, when reflexes gradually return. An increase in the excitability of tendon reflexes and in muscle tone is then observed. The third hypertonic phase is characterized by hyperexcitability of reflexes (stretch reflexes), increased muscle tone, exaggerated tendon reflexes and muscle spasm (34). These phases can be observed in some children with CP when hypertonicity becomes exaggerated in the first year of life (9, 27). Hypertonicity can occur in various distributions including monoplegia, hemiplegia, diplegia, and quadriplegia.

Components of Hypertonicity

As previously mentioned, hypertonicity has two main components, neural and biomechanical. (see Table 1) (25). The neural component consists of two types of symptoms which can be classified as positive and negative. Positive symptoms comprise those features that are not normally present, for example, spasticity, flexor spasms, clasp knife phenomenon, clonus, associated reactions, spastic dystonia, and pathological co-contractions (8, 26, 35-37). Negative symptoms comprise features that have been lost and result in muscle weakness; problems in making selected or isolated movements (dexterity), and fatigability (8, 26, 36, 37). Positive symptoms are mostly caused by the release of more or less intact motor subsystems from precise proximal control (32). Spinal reflex activities are normally controlled and inhibited by the upper centres of CNS. When this inhibition is lost following injury, the balance is destroyed and excitation can be seen (26). Most negative symptoms are direct results of disconnecting lower motor centres from higher ones (32). In other words, positive symptoms are present in the lower motor centres and are controlled by higher motor centres, while negative symptoms are caused directly by damage to the higher motor centres and loss of connections to lower motor centres (32). It seems that negative symptoms (e.g., weakness) are more contributed to motor dysfunction, than positive symptoms (e.g., spasticity) (38-40).

Table Error! No text of specified style in document.. Clinical Features of Hypertonicity

Neural Components: **Positive symptoms**:
Spasticity
Flexor spasm
Clasp knife phenomenon
Clonus
Associated reaction
Spastic dystonia
Pathological co-contraction
Negative symptoms:
Weakness
Loss of selective control of muscles and limb segment
Fatigability
Biomechanical Components:
Muscle shortness
Muscle contracture
Fibrosis
Atrophy

Based on Barens, 2001; Burke, 1988; Katz &Rymer, 1989; Mayer, 1997; O'Dwyer et al., 1996; Sanger, 2003b; Sheean, 2002; Young, 1989.

The biomechanical components of hypertonicity include shortness occurring in soft tissues, muscles, joints, and blood vessels as well as contractures causing limitation in passive range of motion (26, 36). There is also restriction of passive and active range of motion. The inertia of the limb that produces resistance in normal muscle tone does not change in hypertonicity(25). The different components (i.e., neural and biomechanical) of hypertonicity will now be addressed.

Spasticity is defined as a problem in muscle tone due to a velocity-dependent increase in tonic stretch reflexes. Velocity-dependence refers to the speed of the stretch impacting on resistance resulting in greater stretch reflex activity. The tonic stretch reflex is a sustained muscle stretch, and has a long-term response, rather than a phasic one, similar to a tendon reflex (26). Spasticity is mediated by Ia afferent fibres situated in muscle spindles. Any stretch to Ia causes muscle contraction via the spinal cord. Spasticity is also length-dependent which means that the excitability of the tonic stretch reflex depends on the length of the muscle which is being stretched (26), The shorter the muscle, the greater the resistance.

Flexor spasm is another type of positive symptoms resulting from the dis-inhibited normal "flexor withdrawal reflex" that shows itself in a flexor pattern (26). Flexor withdrawal reflex is a nociceptive reflex normally elicited in response to a painful stimulus and resulting in a withdrawal reaction. This reaction helps a person to protect his/her body against harmful situations such as a sharp object. In the presence of an upper motor neuron lesion, this reflex becomes exaggerated resulting in a flexor pattern in upper limb (26).

Clasp knife phenomenon accompanies spasticity and is characterized by sudden muscle relaxation following initial resistance to passive movement around a joint. The underlying mechanism is different from spasticity. Because spasticity is length-dependent, in stretching the spastic muscle, the tonic stretch reflex is greater when the muscle is short. As the stretch is continued and the muscle lengthens, the excitability of the tonic stretch reflex reduces. At the same time, the resistance to the stretch slows movement, and it reduces the spasticity due to the velocity-dependent mechanism. The combination of velocity-dependent and length-dependent mechanisms lead to a point where the stretch is so slow and the muscle so long that it results in a reduction of the excitability of the tonic stretch reflex to such an extent that resistance suddenly disappears (26).

Spastic dystonia is a sustained (tonic) flexor posture that can affect elbow, fingers and wrist flexors and leg extensors. Hemiplegic posture is one example of spastic dystonia in which the person stands or walks while her/his arm is in high flexion and leg in increased extension. The mechanism underlying spastic dystonia is not clear. It seems to come from a supraspinal drive to the alpha motor neurones (26). The dis-inhibition of vestibulospinal tract may cause spastic dystonia, because one of the responsibilities of the vestibulospinal tract is the maintenance of antigravity muscles (i.e., the extensor muscle group in the lower limb and flexor muscle group in the upper limb) (41).

Associated reactions occur when there is an increase in spasticity in involved areas when a person with hypertonicity attempts to do a task (voluntary movement). The greater the effort, the more exaggeration of the response of the associated reaction (26). It may manifest in an exaggerated flexor pattern in the left affected arm and extensor pattern in the left affected leg, for example, when the person tries to comb his/her hair with the sound right hand. Associated reactions are not due to any stretch or nociceptive reflex, but appear to be the result of tonic efferent drive to the alpha motor neurons of muscles (26).

Pathological reciprocal inhibition: Reciprocal inhibition occurs normally in groups of muscles in two ways. First, when agonist muscles contract, their antagonist muscles relax simultaneously to allow a smooth movement to occur (26) (for example, in reaching out elbow extensors contract and inhibit elbow flexors). Second, in some circumstances, both flexor and extensor muscles work together to fix the joint for a movement (26), such as, fixing elbow and wrist during writing.

Reciprocal inhibition may be disordered in two ways. It may be reduced leading to inappropriate co-contraction. For example, during elbow extension, flexors are not inhibited and oppose the movement. This may be explained by two mechanisms: the triggering of the tonic stretch reflex and/or out-of-phase activation of antagonists (in the previous example, elbow flexors) (26, 36). Excessive

reciprocal inhibition is the second type of disorder in which weakness of antagonists may occur, e.g., excessive inhibition of muscle elbow extensors by the elbow flexors may cause weakness in the extensors (36).

Negative symptoms caused by upper motor neuron (UMN) lesions in hypertonicity may include weakness that is characterized by an inability to generate force adequately, and slow movement (8, 26, 36). Two problems in the motor units may contribute to weakness. First, an inability to recruit sufficient motor units and difficulties sustaining their discharge rate (Hoefer, & Putnam, 1940 cited in Mayer, 1997). Secondly, an inability to orderly recruit and modulate motoneurons within a given motoneuron pool can lead to insufficient muscle activation (25). Another negative sign is impairment in making **selected or isolated movements** across specific joints (36). Clients with hypertonicity usually show stereotypic whole limb movements in which they demonstrate mass flexor or extensor patterns. The last negative symptom is **fatigability** (32, 42) resulting from inefficient muscle activation due to loss of orderly recruitment and rate modulation of motoneurons (25).

Biomechanical changes in muscles may be largely responsible for hypertonicity (25) and may contribute to resistance to passive movements more than tonic stretch reflexes (37). Biomechanical changes involve muscle contracture and shortening in soft tissue. Contracture is defined as an increased resistance to passive stretch due to shortening of muscle length resulting from a decrease in the number of sarcomeres in series along the myofibrils (43). Contractures in children with hypertonicity may happen over time through three mechanisms. Firstly, a cerebral lesion, associated with paresis, causes a transformation of motor units resulting most probably in shortening of muscle fibres leading to contracture (44). Muscle and soft tissue changes compensate for the loss of supraspinal drive and contribute essentially to hypertonicity in both active and passive movements (45). Secondly, the limited and stereotypical movements as well as weakness typical for most people with hypertonicity tends to result in shortening of muscle fibres, a reduced range of movement, and the onset of contractures, as they get older (26, 36, 46). Thirdly, when a CNS lesion occurs in childhood, the race between bone and muscle growth in the child may accelerate development of more contractures and deformities (47). The presence of soft tissue changes apart from contractures is controversial, requiring further research. While some believe that degenerative changes (fibrosis) and muscle atrophy may also occur following hypertonicity (25, 36), others argue that it results from shortening in muscle fibres rather than atrophy and fibrosis (48).

The development of contractures in CP appears to progress through three stages. First, deformities are flexible and reversible. This occurs typically in the younger child with hypertonicity. In the second stage, contractures become fixed and may require surgical intervention. In the third stage, in addition to contractures, there are changes in the structure of bones and joints (7).

In the assessment of a client with recent onset of hypertonicity (before the onset of shortness and contracture), the abnormal resistance against passive movement is the result of reflexes and neural activities. However, in chronic hypertonicity, both neural and biomechanical components may provide resistance against passive movement. In other words, resistance felt by clinicians is generated by reflex activity and abnormal properties of muscle and other soft tissues (36). In active movements, nevertheless, it is very difficult to distinguish clinically between the contribution of abnormal neuronal and biomechanical components (36). Electromyographic (EMG) and biomechanical recordings show that overall muscle activity is reduced during functional movements in muscles with hypertonicity (45). Therefore, the influence of the signs and symptoms of hypertonicity on active and passive movements differs requiring more investigation for effective treatment.

Biomechanical components as well as positive and negative symptoms may affect the movement activities (movement dysfunction) of a person with hypertonicity (26, 36, 39, 46), resulting in limitations in occupational performance (i.e., activities of daily living, work and school activities, play, and recreational activities). Figure 1 illustrates the interactions between positive and negative symptoms, and biomechanical components contributing to occupational performance.

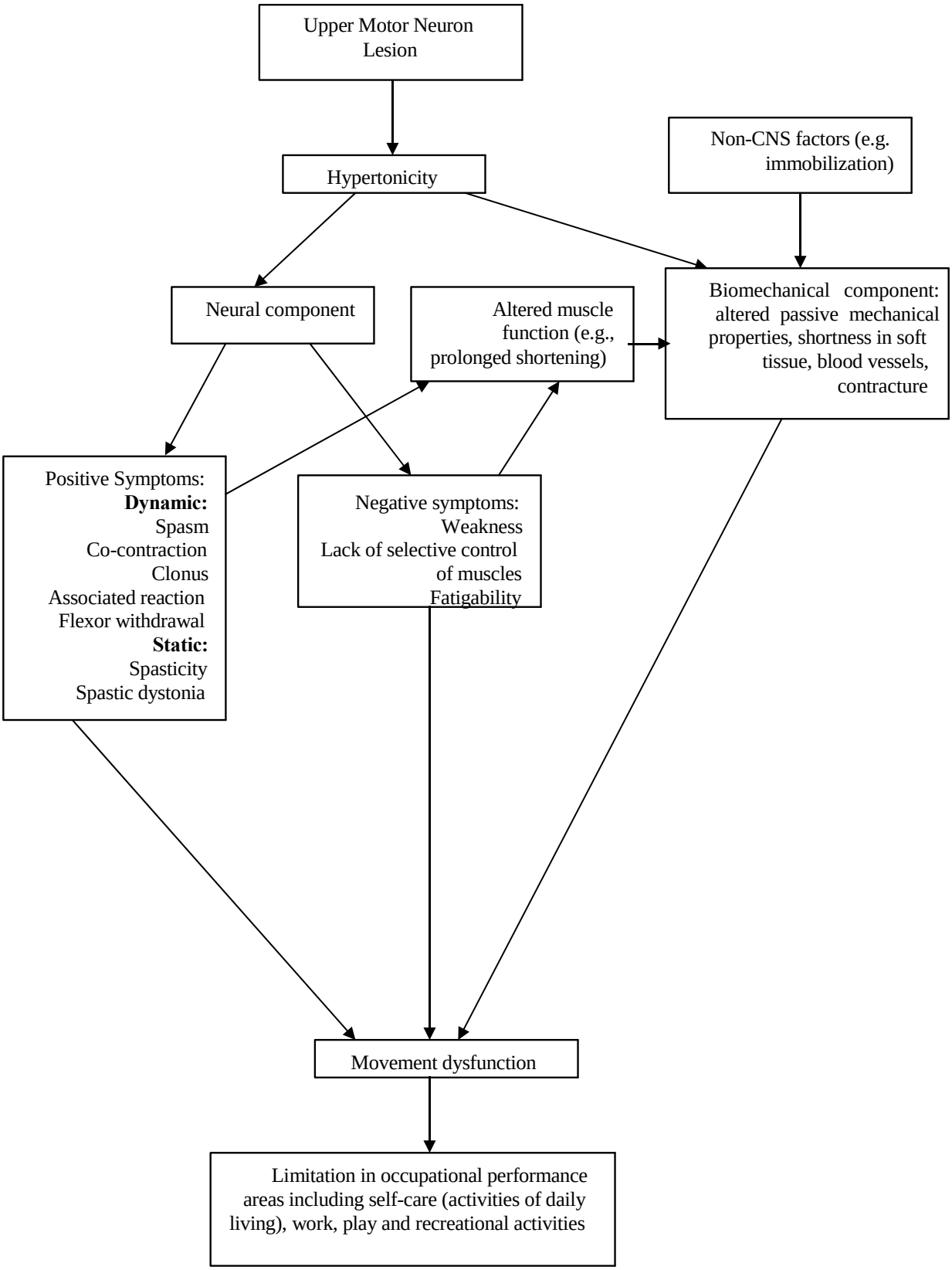

Figure 1. Interaction and relationship among positive and negative neural symptoms, and biomechanical components.

Clinical Manifestations of Hypertonicity

When hypertonicity occurs, there are accompanying abnormalities in patterns of motor behaviour observed throughout the body in both gross and fine movements. Persistent and exaggerated postural reflexes such as tonic neck reflexes, asymmetrical and symmetrical tonic neck reflexes cause the development of abnormal movement patterns resulting in difficulties in maintaining the body in space, and restricting active and passive movements (17). Righting and equilibrium reactions might also be poor in people with hypertonicity and limit active movement patterns and upright positions. Consequently, movement and function of the upper limbs are affected and can impact on the performance of everyday tasks.

Gross movement in children with hypertonicity and CP might be affected in two ways. First, hypertonicity may cause delays in the attainment of gross movement milestones compared with normal children. The rate of delay depends on the severity of hypertonicity, the more severe, the greater the delay. Secondly, presence of abnormal patterns of movement and posture in people with hypertonicity can make this problem worse. Abnormal patterns of movement and posture manifest in all developmental milestones such as rolling over, creeping, crawling, sitting, standing up, and walking.

Postural reflexes such as asymmetrical and symmetrical tonic neck reflexes, and tonic labyrinthine reflexes (supine and prone) are essential to normal child development (49). For example, the asymmetrical tonic neck reflex normally helps a child to develop eye-hand coordination by bringing the hand towards the midline. However, these reflexes in a child with hypertonicity may be exaggerated thereby hampering developmental progress (49). In this situation, the asymmetrical tonic neck reflex may cause asymmetry in the body, prevent arms coming to midline, and restrict movement such as rolling from supine to prone. In another example, the tonic labyrinthine reflex may increase extensor tone in the trunk during sitting up from the supine position and provide excessive resistance against flexion. Exaggerated postural reflexes may also impede the maintenance of the body in space. In an upright position such as sitting, the tonic labyrinthine reflex may decrease balance and stability. Therefore, as the child moves his/her head, this reflex is activated resulting in loss of balance. All problems in gross movements and maintaining position interfere with a child's ability to carry out activities of daily living, playing and school work.

Upper limb function is also affected by hypertonicity. The upper limbs are important for the performance of daily tasks. With the help of hands and arms, a person is able to reach out, grasp, manipulate, transfer and release objects. People accomplish activities of daily living such as dressing, feeding, toilet, and showering by using their arms and hands. Moreover, hands provide the means whereby human beings manipulate their environment. Hands have a main role in every aspects of motor, social, language, and cognitive development (50). The contribution of the hands to development starts even before birth, e.g. , by grasping and releasing the umbilical cord, and sucking the thumb (Milani-Comparetti, 1980 cited in Erhardt) and continues after birth.

Hypertonicity imposes abnormal patterns on the upper limbs, causing limitation in range of movement and speed and resulting in excessive efforts in the performance of tasks (50). Different patterns may be observed in the upper limbs of people with hypertonicity, but the most prevalent one is the antigravity pattern (51), that is, scapular protraction and depression, shoulder adduction and internal rotation, elbow flexion, forearm pronation, wrist flexion and ulnar deviation, thumb adduction and flexion, and finger flexion.

Impaired upper limb function also interferes with gross motor development and impacts on actions such as rolling over, creeping, and crawling. For example, because children with hypertonicity have limited hand function (e.g., by holding walking aide) they are not able to facilitate standing and walking. These limitations result in a delay in attaining developmental milestones.

Conclusion

Hypertonicity includes two neural and biomechanical components resulting in abnormal movement patterns in both gross motor function and manual abilities in the children with CP; consequently, these children experience problems in their all activities of daily living. Focusing on all aspects of the hypertonicity will surely help to decide better for these children and have better results.

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