

FLOPPY INFANT SYNDROME: OVERVIEW

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ABSTRACT

Floppy infants exhibit poor control of movement, delayed motor skills, and hypotonic motor movement patterns. Weak infants always have hypotonia, but hypotonia may exist without weakness. Some indications of CNS abnormality are because of poor state of alertness, lack of response to visual and auditory stimuli, inability to manage co-ordinated functions like swallowing and sucking noted that the earlier the onset, the more severe and precipitous the course. , and spinal muscular atrophy. Parents commonly complain to physicians that their baby is very passive, that it does not move its limbs like others, that its breathing pattern is abnormal or inquire why the baby cannot be weaned off the ventilator. Several studies have shown that central causes account for 60% to 80% of hypotonia cases and that peripheral causes occur in 15% to 30%.^{7,9}. Conditions where central and peripheral hypotonia may coexist are Familial dysautonomia, Hypoxic-ischemic encephalopathy, infantile neuroaxonal degeneration, Lipid storage diseases, Lysosomal disorders, mitochondrial disorders. Treatment of the infant who has hypotonia must be tailored to the specific responsible condition. In general, therapy is supportive. Rehabilitation is an important therapeutic consideration, with the aid of physical and occupational therapists.

KEY WORDS: Floppy Infant Syndrome, Hypotonia, Weakness.

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INTRODUCTION

The floppy infant syndrome is a well-recognized entity for pediatricians and neonatologists and refers to an infant with generalized hypotonia presenting at birth or in early life. An organized approach is essential when evaluating a floppy infant, as the causes are numerous [1].

There are approximately 65,000 children in the UK who have profound and multiple learning disabilities, many of which are manifested as physical disabilities (Mind, 2006). Children with physical disabilities often develop secondary

impairments such as an abnormal level of muscle tone (Goldstein 2005). Muscle tone can be defined as the tension in a relaxed muscle due to involuntary contractions of its motor units (Tortora and Grabowski, 2003). It is our muscles that exert sufficient force against gravity to pull us upright; and it is our muscles that enable our postural reactions to maintain this upright posture and so prevent us from being injured [2].

Appropriate muscle tone enables a child to respond quickly to an outside force either

through balance responses/righting reactions or protective reactions. It also allows a child's muscles to quickly relax once the perceived change is gone. A child with hypotonia has muscles that are slow to initiate a contraction against an outside force, and also cannot sustain a muscle contraction as long. In other words, the muscles "relax" quicker despite that the outside force may still be present [2].

Sarah O'Conner, 2014 defined as a reduced tension in a muscle at rest, is a relatively frequent presentation in both the infantile and childhood period. It must be differentiated from muscle weakness (a reduction in muscle power, that may or may not accompany hypotonia), and laxity (an increase in joint range of motion that often accompanies hypotonia) [3]. Neurologic muscle tone is a manifestation of periodic action potentials from motor neurons as it is an intrinsic property of the nervous system; it cannot be changed through voluntary control, exercise, or diet. True muscle tone is the inherent ability of the muscle to respond to a stretch. The child with low tone has muscles that are slow to initiate a muscle contraction, contract very slowly in response to a stimulus, and cannot maintain a contraction for as long as normal peers. Because these low-toned muscles do not fully contract before they again relax (muscle accommodates to the stimulus and so shuts down again), they remain loose and very stretchy, never realizing their full potential of maintaining muscle contraction overtime [4,5].

However, in early infancy, contrary to the expected increase in muscle tone, the response to an UMN lesion in the early stages is flaccidity and loss of muscle tone [4]. This pattern of hypotonia is usually associated with preserved or hyperactive reflexes and later evolves into spasticity [6,7].

It is important to distinguish weakness from hypotonia. Hypotonia is described as reduced resistance to passive range of motion in joints; weakness is reduction in the maximum power that can be generated. A more useful definition of hypotonia is an impairment of the ability to sustain postural control and movement against gravity. Thus, floppy infants exhibit poor control

of movement, delayed motor skills, and hypotonic motor movement patterns. Weak infants always have hypotonia, but hypotonia may exist without weakness. Some indications of CNS abnormality are because of poor state of alertness, lack of response to visual and auditory stimuli, inability to manage co-ordinated functions like swallowing and sucking noted that the earlier the onset, the more severe and precipitous the course [8].

AETIOLOGY

There are two categories - Central and peripheral disorders. Several studies have shown that central causes account for 60% to 80% of hypotonia cases and that peripheral causes occur in 15% to 30%^{7,9}. Conditions where central and peripheral hypotonia may coexist are Familial dysautonomia, Hypoxic-ischemic encephalopathy, infantile neuroaxonal degeneration, Lipid storage diseases, Lysosomal disorders, mitochondrial disorders [10].

CENTRAL CAUSES:

- Cerebral insult -Hypoxic ischemic encephalopathy, intracranial haemorrhage
- Brain malformations
- Chromosomal disorders - Praderwilli syndrome, Down syndrome
- Peroxisomal disorders – cerebrohepato renal syndrome (Zellweger's syndrome), Neonatal adrenoleukodystrophy
- Other genetic defects - familial dysautonomia, oculocerebrorenal syndrome (Lowe syndrome)
- Neurometabolic disorders – Acid maltase deficiency, infantile GM1 gangliosidosis
- Drug effects (ex Maternal Benzodiazepines)
- Benign congenital hypotonia

PERIPHERAL CAUSES:

- A) Due to anterior horn cell
- Infantile spinal muscular atrophy
 - Traumatic myelopathy (esp following breech delivery)
 - Hypoxic ischemic myelopathy
 - Infantile neuronal degeneration
- B) Neuropathies
- Congenital hypomyelinating neuropathy
 - Giant axonal neuropathy
 - Charcot marie tooth disease
 - Dejerine sottas disease

C) Due to neuromuscular junction

- Myasthenia gravis (Transient acquired neonatal myasthenia ,congenital myasthenia)
- Infantile botulism
- Magnesium toxicity
- Aminoglycoside toxicity

D) Myopathies

- Congenital myopathy
- Nemaline myopathy
- Central core disease
- Myotubular myopathy
- Congenital fiber type disproportion myopathy

E) Muscular dystrophies

- Congenital muscular dystrophy with merosin deficiency
- Congenital muscular dystrophy without merosin deficiency
- Congenital muscular dystrophy with brain malformations or intellectual disability
- Dystrophinopathies
- Walker Warburg disease
- Muscle – eye – brain disease
- Fukuyama disease
- Congenital muscular dystrophy with cerebellar atrophy / hypoplasia
- Congenital muscular dystrophy with occipital agyria
- Early infantile facioscapulohumeral dystrophy
- Congenital myotonic dystrophy

F) Metabolic and multisystem disease

- Disorders of glycogen metabolism (ex Acid maltase deficiency)
- Severe neonatal phosphofructokinase deficiency
- Severe neonatal phosphorylase deficiency
- Primary carnitine deficiency
- Peroxisomal disorders
- Neonatal adrenoleukodystrophy
- Cerebrohepatorenal syndrome (zellweger)
- Disorders of creatine metabolism
- Cytochrome c oxidase deficiency

CLINICAL PRESENTATION:

Parents commonly complain to physicians that their baby is very passive, that it does not move its limbs like others, that its breathing pattern is abnormal or inquire why the baby cannot be weaned off the ventilator.

1) Floppy babies in early infancy may present with abnormal posturing of limbs and body, diminished resistance of limbs to passive movement, abnormal range of joint movement and/or ventilator dependency [7,11].

2) The delay in the motor milestones.

3) Weakness. Useful indicators of weakness are: Inability to cough and clear airway secretions ('cough test'), Poor swallowing ability, weak, Paradoxical breathing pattern(intercostals muscles paralyzed with intact diaphragm) [6,11].

4) Frog-like posture and quality of spontaneous movements, excessive head lag will be evident on 'pull to sit'.

5) Examine the tongue for size and fasciculation. Fasciculation's, irregular twitching movements, generally indicate an abnormality of the anterior horn cells. Do not examine the tongue while the infant is crying. The co-existence of atrophy would strongly favour a denervative aetiology.

6) There is a paucity of antigravity movements in the weak and hypotonic infant. In central hypotonia; axial weakness is a significant feature. Preservation of muscle power with hypotonia and hyperreflexia favours a central origin to the hypotonia, while the combination of weakness in the antigravity limb muscles and hypo/areflexia together favour a neuromuscular disorder [6,7,24].

7) The presence of a typical 'myopathic' faces and paucity of facial expression are common in hypotonic infants. A high arched palate is often noted in infants with neuromuscular disorders.

8) Examination of eye movements may provide clues to the presence of ptosis and external ophthalmoplegia may suggest a Myasthenic syndrome.

9) Examination of the limbs and joints may show presence of arthrogryposis. Arthrogryposis can be a feature encountered in both neurogenic and myopathic disorders. Cowie (1870) links this hypotonia with the lack of posture regulation, as evidenced in testing the Landau reaction and traction tests. Haley (1886) concludes that posture reactions (righting, balance and supporting reactions) develop later in these children than in normal children. Mezzomo (1885)

concluded that heels-down squatting of floppy children could be a compensatory mechanism due to insufficient balance or insufficient agonist and antagonist muscle activation around the ankle. Steele's (1879) study indicates that there is an abnormal leg position in nearly 50% of the children involved. The sitting position thus becomes static in nature, while, particularly for young children, it should be the ideal play and transitional posture.

10) Wardnig and Hoffman (1840) described main clinical features as head lag, slip through or shoulder suspension test and U shape posture in their study [8].

11) There are two types of floppy baby.

Floppy strong	Floppy weak
Increased tendon reflexes	Hypo- to areflexia
Extensor plantar response	Selective motor delay
Sustained ankle clonus	Normal head circumference and growth
Global developmental delay	Preserved social interaction
Microcephaly or suboptimal head growth	Weakness of antigravitational limb muscles
Obtundation convulsions	Low pitched weak cry
Axial weakness a significant feature	Tongue fasciculations
Upper motor neuron disorder	Paradoxical chest wall Movement
Central hypotonia	Lower motor neuron disorder
	Peripheral hypotonia

DIFFERENTIAL DIAGNOSIS

Central Hypotonia: Clinical features suggestive of hypotonia of central origin are social and cognitive impairment with motor delay, dysmorphic features like fisting of hands, normal or brisk tendon reflexes ,features of pseudobulbar palsy, brisk jaw jerk, crossed adductor response or scissoring on vertical suspension, features that may suggest an underlying spinal dysraphism, history suggestive of hypoxic-ischaemic encephalopathy, birth trauma or symptomatic hypoglycemia, seizures [9].

Peripheral Hypotonia: Indicators of peripheral hypotonia are delay in motor milestones with relative normality of social and cognitive development ,family history of neuromuscular disorders/maternal myotonia ,reduced or absent spontaneous antigravity movements, reduced or absent deep tendon jerks and increased range of joint mobility, frog-leg posture or 'jug-handle' posture of arms in association with marked

paucity of spontaneous movement ,myopathic faces (open mouth with tented upper lip, poor lip seal when sucking, lack of facial expression, ptosis and restricted ocular movements) ,muscle fasciculation (rarely seen but of diagnostic importance when recognized) [9].

INVESTIGATION

If the hypotonia is considered to be central, the patients should be investigated with magnetic resonance imaging or computer tomography (CT) brain. These are helpful in the identification of structural malformations, neuronal migration defects, altered signal and characteristics of white matter. Molecular genetic testing provides the advantage of diagnostic specificity. These tests should be chosen according to the clinical presentation of the infant.

To evaluate causes of peripheral hypotonia, creatine kinase concentrations should be measured which is elevated in muscular dystrophy. Specific DNA testing can be performed for myotonic dystrophy and for spinal muscular atrophy [10].

Rule out sepsis	If central hypotonia is Suspected	If peripheral hypotonia is suspected
Blood culture	MRI, CT Scan	Electromyography (EMG)/Nerve conduction studies (NCS)
Urine culture	Karyotyping	Muscle biopsy for staining with different reagents and electron microscopy
CSF culture	Molecular genetics	Creatine kinase (CK) level
Full blood count	Very long chain fatty acids	Toxin assay i.e. Botulism
C reactive protein	Serum/Urine amino acids	Auto antibody levels
ESR	Urine organic acids	
Serum electrolyte,calcium, Magnesium	Blood/CSF lactate	Specific DNA testing
Serum glucose	Carnitine/acylcarnitine levels	
TORCH Test		

ASSESSMENT

Obstetric history

- Identify cause and the timing of onset
- Maternal exposures to toxins or infections suggest a central cause
- Information on fetal movement in utero, fetal presentation, and the amount of amniotic fluid.
- Low Apgar scores may suggest floppiness from birth
- Breech delivery or cervical position – cervical

cervical spinal cord trauma

- Maternal disease – myotonic dystrophy
- Perinatal history – drug or teratogen exposure
 - Decreased fetal movements
 - Abnormal presentation
 - Polyhydramnios/ oligohydramnios
 - Apgar scores
 - Resuscitation requirements
 - Cord gases
- **Postnatal History**
 - Respiratory effort
 - Ability to feed
 - Level of alertness
 - Level of spontaneous activity
 - Character of cry

Course of illness

- A term infant who is born healthy but develops floppiness after 12 to 24 hours – suspect inborn error of metabolism
- Infants suffering central injury usually develop increased tone and deep tendon reflexes.
- Central congenital hypotonia does not worsen with time but may become more readily apparent

Development history

- Motor delay with normal social and language development decreases the likelihood of brain pathology.
- Loss of milestones increases the index of suspicion for neurodegenerative disorders

Dietry/feeding history

- A dietary/feeding history may point to diseases of the neuromuscular junction, which may present with sucking and swallowing difficulties that 'fatigue' or 'get worse' with repetition.

Family history

- Developmental delay (a chromosomal abnormality)
- Delayed motor milestones (a congenital myopathy) and
- Premature death (metabolic or muscle disease).
- Any significant family history – affected parents or siblings, consanguinity, stillbirths, childhood deaths

General examination

- When lying supine, all hypotonic infants look much the same, regardless of the underlying cause or location of the abnormality within the nervous system.

- Lack spontaneous movement
- Full abduction of the legs places the lateral surface of the thighs against the examining table, and the arms lie either extended at the sides of the body or flexed at the elbow with the hands beside the head.
- Pectus excavatum indicates long standing long-standing weakness of the chest wall muscles
- Infants who lie motionless eventually develop flattening of the occiput and loss of hair on the portion of the scalp that is in constant contact with the crib sheet.
- Hip subluxation or arthrogryposis suggest hypotonia in utero .
- High-pitched or unusual-sounding cry - suggests CNS pathology
- A weak cry - diaphragmatic weakness
- Fatigable cry - congenital myasthenic syndrome.
- Assessment for dysmorphic features
- Detailed neurologic assessment - tone, strength, and reflexes
- Assessment of tone – begin by examining posture, and movement. A floppy infant often lies with limbs abducted and extended.
- Test traction response (most sensitive measure of postural tone). In this grasp the hands and pull the infant toward a sitting position. A normal term infant lifts the head from the surface immediately with the body. When attaining the sitting position, the head is erect in the midline for a few seconds. During traction, the examiner should feel the infant pulling back against traction and observe flexion at the elbow, knee, and ankle. The traction response is not present in premature newborns of less than 33 weeks' gestation. The presence of more than minimal head lag and of failure to counter traction by flexion of the limbs in the term newborn is abnormal and indicates hypotonia.
- Explains vertical suspension. By 1 month, normal infants lift the head immediately and maintain it in line with the trunk. The examiner places both hands in the infant's axillae and, without grasping the thorax, lifts straight up. The muscles of the shoulders should have sufficient strength to press down against the examiner's hands and allow the infant to suspend vertically without falling through. Normal response – Head

erect in the midline with flexion at the knee, hip, and ankle joints. When a hypotonic infant is suspended vertically, the head falls forward, the legs dangle, and the infant may slip through the examiner's hands because of weakness in the shoulder muscles

- Explains horizontal suspension with inverted U posture Other findings are Decreased resistance to flexion and extension of the extremities Exaggerated hip abduction & ankle dorsiflexion, Oral-motor dysfunction, Poor respiratory efforts , Gastroesophageal reflux, Note the distribution of weakness ex .face is spared versus the trunk and extremities.
- Deep tendon reflexes (DTRs) often normal / hyperactive in central conditions
- Clonus and primitive reflexes may persist DTRs - normal, decreased, or absent in peripheral disorders.

MANAGEMENT

Treatment of the infant who has hypotonia must be tailored to the specific responsible condition. In general, therapy is supportive. Rehabilitation is an important therapeutic consideration, with the aid of physical and occupational therapists. Nutrition is of primary importance to maintain ideal body weight for the age and sex which is often achieved through the nasogastric route or percutaneous gastrostomy [7].

RESPIRATORY CARE

Respiratory problems are a primary cause of morbidity and/or hospital admission particularly in young children. There is an increased prevalence of sleep-related upper airway obstruction and lower airway disease, but the significance of symptoms is often under-recognized. Therefore, specialist investigation and treatment are often necessary but not often sought. In a teaching hospital in Australia, a retrospective chart review of 232 admissions of children with Floppy syndrome over a 6.5-year period gave over half the causes for admission as respiratory problems. Of the total admissions, 10% were ultimately admitted to the Paediatric Intensive Care Unit [13].

Regular physiotherapy is not popular with children, especially those with lower airway problems. However, it may be useful to teach parents how to perform physiotherapy so that it

can be used when their child is unwell and so may tolerate it better. A large number of children may simply require oxygen, even if they have large airway problems. Non-invasive ventilation is relatively uncommon [13].

TRANSFER

Independent transfer has been considered the most important factor in communication skills and cognitive development. Spasticity in different forms and weak muscles may prevent normal voluntary movements and may also lead to deficient postural control, making it difficult for the children to attain an upright position and dynamic weight-bearing on the skeleton. The importance of weight-bearing for the skeleton is shown by the postnatal adaptation of the skeleton to the environment outside the womb, in which the femoral diaphysis decreases volumetrically by about 30% during the first six months, probably because the baby is no longer kicking the inside of the uterus and thus no longer engaging in frequent 'resistant training' (Rauchand Schoenau, 2001) [14].

General recommendations for time spent in the standing shell, based on presumptions of increasing bone density or reducing risk of hip dislocations, could be replaced by individual recommendations adapted to the wishes of the child. This type of vibrating platform could be a non-invasive treatment to improve BMC and give children with severe CP a more effective and enjoyable time in the standing shell [14].

BIOFEEDBACK

Different pressure-sensitive pads have been developed to feed back information on heel strike to the patient. Conrad and Bleck (1880), Dalton (1887) and Seeger and Caudrey (1883) all noted improved heel strike in children with equinus gait. However the long-term improvement in these children was either not evaluated or found to be not significant.

Miyazaki et al (1986) developed a portable limb-load monitor to limit weight bearing. Olney et al (1889) used a computer to give simple visual and auditory feedback of knee angle changes during gait.

Basaglia et al (1889) reviewed studies on correction of hyperextension of the knees with electrogoniometry feedback and noted good

results. In their research they found significant improvements in knee angle during walking at the end of treatment and at one year follow-up. However, they found no significant gains in walking speed.

Lord et al (1882) designed a video system in which the centre-of-pressure under the feet was shown to patients on a screen in front of them and it was particularly successful in motivating patients to re-establish basic motor skills by repeated and directed practice.

Sackley et al (1892) described treatment with the 'SMS balance performance monitor', which is a lightweight portable unit that gives objective visual information on weight distribution and postural sway with different coloured lights and auditory signals. The authors outline how a variety of exercises and functional skills can be practiced with a patient standing on the two footplates and receiving visual feedback from the movable monitor. They found that both staff and patients enjoyed working with the equipment, that it was easy to use, and that it gave accurate and quantitative information to both patients and therapists [15].

Utilising microcomputers as therapeutic aids has been more common in occupational therapy (Pinnington and Brown, 1894). It has been shown to be an asset in employment, perceptual and cognitive training, communication and leisure (Stoneman, 1885).

Crofts and Crofts (1888) report on EMG with a BBC Microcomputer. Electrical activity of weak muscles is recorded and transferred to the computer. In this way a patient can cause display changes on the computer screen and even play games, receiving feedback from small and larger muscle contractions.

Mackey (1889) used a switch box linked into a BBC microcomputer on which cerebral palsied children pressed down with flat hands. The amount of pressure was represented by a visual display on the computer screen. The experiment showed that the children maintained the targeted pressure for much longer when they received auditory and visual feedback from the computer. Handling techniques can be used to improve the child's performance of functional tasks such as sitting, walking, and reaching by

promoting postural alignment prior to movement. Other specific sensory interventions such as tapping a muscle belly, tactile cuing, or pressure are tailored to specific impairments the child may have. Impairments include such things as difficulty in recruiting a muscle contraction for movement initiation, lack of pelvic control for midline positioning, or inability to control certain body segments during changes of position. The ultimate goal of any type of therapeutic intervention is functional movement. If an infant cannot maintain postural control in sitting without hand support, then the ability to play with toys is limited. Children with weak or uncoordinated lower extremities commonly perform a "commando crawl" using only their arms to pull them along the surface. This is also called drag crawling if the lower extremities do not assist in producing the movement but are dragged along by the pull of the arms.

The greatest challenge for physical therapists and physical therapist assistants who work with children with neurologic deficits may be to determine how to bring the world to a child who has limited head or trunk control or limited mobility.

In addition, by suggesting a variety of therapeutic play positions that can be incorporated into the daily routine of the child, make it unnecessary for the caregiver to have to spend as much time stretching specific muscles. Pictures are wonderful reminders. Providing a snapshot of how you want the child to sit can provide a gentle reminder to all family members, especially those who are unable to attend a therapy session. If the child is supposed to use a certain adaptive device such as a corner chair sometime during the day, help the caregiver to determine the best time and place to use the device [12,16].

CARRYING POSITIONS

- a) Sitting postures.
 - W sitting, this should be avoided.
 - B: Wide abducted long sitting should be avoided.
 - C: Proper sitting with legs abducted.
- b) Place the child in a curled-up position with shoulders forward and hips flexed. Place your

arm behind the child's head, not behind the neck.

c) Avoid lifting the child under his arms without supporting the legs. The child with hypertonicity may "scissor" (cross) the legs. The child with hypotonicity may slip through your hands.

d) Bend the child's legs before picking him up. Give sufficient support to the trunk and legs while allowing trunk rotation.

e) Hold the child with low tone close, to provide a feeling of stability.

f) Have the child straddles your hips to separate tight legs. Be sure the child's trunk is rotated forward and both his arms are free.

g) Use of Prone position.

h) Positions to Encourage Head Control. Positioning the child prone over a half-roll encourages head lifting and weight bearing on the elbows and forearms. Positioning the child supine on a wedge in preparation for anterior head lifting.

i) Manual approximation through the shoulders in the four-point position (Thorofare, NJ, Slack Inc., 2001)

j) Coming to stand from a squat requires good lower extremity strength and balance (Cristaralla M,1875).

k) Vertical standers support the child's lower extremities in hip and knee extension and allow for varying amounts of weight bearing depending on the degree of inclination. The child's hands are free for upper-extremity tasks, such as writing at a blackboard,

BALANCE AND GAIT TRAINING:

Body weight supported treadmill training has shown marked improvement in the gross motor function measure standing dimension and walking, running, and jumping dimension in 10 children with cerebral palsy (Schindl MR,et al 2000).

Gait speed and the gross motor function measure are the most commonly reported outcomes in treadmill training studies, and results across studies are generally positive, with moderate to large effect sizes. (Damiano DL, DeJong SL. 2009). Devices that support and assist leg movement during stepping, such as the Lokomat, have also been used in cerebral

palsy (Meyer-Heim A,et al. 2007) as well as in spinal cord injury and stroke with positive results, although their superiority over therapist-assisted training has not been established. The National Institute of Neurological Disorders and Stroke states that physical therapy can improve motor control and overall body strength in individuals with hypotonia. Physical therapists might use neuromuscular/sensory stimulation techniques such as quick stretch, resistance, joint approximation, and tapping to increase tone by facilitating or enhancing muscle contraction in patients with hypotonia. For patients who demonstrate muscle weakness in addition to hypotonia strengthening exercises that do not overload the muscles are indicated [17].

Neuromuscular Electrical Stimulation (NMES) can also be used to "activate hypotonic muscles, improve strength, and generate movement in paralyzed limbs while preventing disuse atrophy. NMES should ideally be combined with functional training activities to improve outcomes (Fenichel GM,2005) [18]. In hypotonic muscles, the muscle spindle is slack and not sensitive to changes in muscle length. This results in slow activation which makes movement difficult. Children with hypotonic muscles often have difficulty in maintaining balance because their postural tone is too low. This is especially true while performing fast and accurate movements (Tyldesley and Grieve, 2002) which can be partially attributed to the delayed activation response. Conventional physiotherapy for children concentrates on the improvement of gross motor functions such as balance, crawling, sitting, standing and walking (Hallam, 1897). Physiotherapy can be painful and is often hard work. Many adjunctive therapeutic activities have been developed as alternatives to traditional physiotherapy (Harris 1878;Chernget al., 2004) [18].

HYDROTHERAPY

Hydrotherapy has been claimed to positively impact the motor development of young children (Stein, 2004). It uses the beneficial effect of buoyancy which puts less mechanical stress on the joints than in land based exercises (Kent, 2003; White, 1895). The body's buoyancy enables children to move independently. Combined with the reduction in gravity, the

buoyancy decreases compressive forces on weight bearing joints which may aid relaxation, decrease muscle spasm and muscle tension (Koury, 1896; White, 1895) [18].

THERAPEUTIC HORSEBACK RIDING

It is hypothesized that therapeutic horseback riding benefits children with motor disorders e.g. cerebral palsy, because of the rhythmic, three-dimensional movement of the horse's walking which replicates the movement of a human pelvis during walking, thus providing a normal sensorimotor experience (Quint and Toomey, 1898; Riede 1885; cited in Pauw 2000). Furthermore, riding horseback continuously changes the relationship between the rider's centre of mass and their base of support therefore improving coordination and challenging balance (Cherndet al., 2004). This is further facilitated by changes in the horse's stride, velocity and direction which demands stimulation of righting and equilibrium responses (Cherndet al., 2004; Pauw 2000). The position of the pelvis plays an important role in efficiency of movement as it influences the position of the lumbar spine which affects thoracic and cervical alignment, thus influencing the position of the head and limbs (Quint and Toomey, 1898) [18].

REBOUND THERAPY

Rebound therapy may provide constant opportunity for sensory integration of kinesthetic, visual, and vestibular input. The vestibular sensory system, which responds to changes in head position, body movement and the pull of gravity, is heightened in rebound therapy because of the vertical motions of the body on the trampoline.

Noda et al., (2003) reported noticeable improvements in the clinical conditions of permanent vegetative state patients following musicokinetic therapy (passive trampoline bouncing to music). They attributed the improvements to stimuli simultaneously activating vestibular, somatosensory and motor pathways and functions within the brain. The pleasant and beneficial feeling of weightlessness reported in many hydrotherapy studies is also simulated in rebound therapy: the therapist can push the bed from underneath a

child thus pushing them into the air (Rollings, 2005). Feeling weightless is not only euphoric, but puts less pressure on the joints than in land-based exercises. Similarly to water, the bed allows independent movement. The stimulatory pressure to the skin, induced by the trampoline bed may stimulate nerves which may increase blood flow and lessen pain sensitivity.

Elisabeth Graham (2006) stated that Rebound therapy can increase abnormally low muscle tone by exerting a persisting stimulatory pressure to the skin and decrease abnormally high muscle tone by vibrating muscle spindles, increasingly elasticity, and accentuating the amount of laxity within the muscle fibers [18].

Conflicts of interest: None

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