Full Length Research Paper

Efficacy of cervical muscle spasticity control in stimulation of weight gain in spastic diaplegic cerebral palsy children

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The aim of this work was to show the efficacy of cervical muscle spasticity control on improving of weight gain in spastic diaplegic cerebral palsy children. Thirty children were enrolled in this study and randomly assigned into two groups; group A (specialized physiotherapy program for cervical muscle spasticity control plus traditional physiotherapy program), and group B (traditional physiotherapy program only). Weight measuring scale was used to detect and follow weight gain changes. This measurement was taken before initial treatment and after 12 weeks of treatment. The children parents in both groups A and B were instructed to complete 3 hours of home routine program. Data analysis were available on the 30 spastic diaplegic cerebral palsy children participated in the study. The mean value of weight gain changes of both groups at (pre-treatment) was insignificant (p>.05). By comparison of both groups at post-treatment there was significant improvement in weight gain changes (p<.05), the difference between pre and post treatment results was significant in both groups in favor of the study group (p=0.0002). According to the results of this study it can be concluded that the combined effect of specialized physiotherapy program for cervical muscle spasticity control plus traditional physiotherapy program can be recommended in controlling of weight loss in spastic diaplegic cerebral palsy children.

Keywords: cervical muscles spasticity- vagus nerve-weight gain

INTRODUCTION

Because of the proximity of the vagus to the musculoskeletal structures in the suboccipital region, it is plausible that local inflammation, edema, muscle hypertonicity or spasm, or other somatic dysfunctions could cause either a chemical or compressive effect on the vagus, thereby affecting its optimum function. Since the vagus plays a significant role in the autonomic control of stomach function, it is therefore also plausible that if optimum performance of the vagus is impeded by dysfunction in the surrounding structures, its ability to contribute effectively to the autonomic control of stomach function might also be affected. The vagus nerve helps manage the complex processes in your digestive tract, including signaling the muscles in your stomach to contract and push food into the small intestine. An entrapped vagus nerve can’t send signals normally to your stomach muscles. This may cause food to remain in your stomach longer, rather than move normally into your small intestine to be digested leading to loss of appetite for eating and loss of weight is the result (Law Office of Michels and Watkins, 2012).
There are two main types of vagus nerve disorders. One is caused by an under-active or inactive vagus nerve, while the other is caused by a vagus nerve that overreacts to ordinary stimuli. Vagus nerve disorders that stem from an under-active vagus nerve often lead to a condition known as gastro-paresis which is a frequent and severe complication of diabetes. Patients suffering from this disorder may experience pain in the stomach, nausea, heartburn, stomach spasms, and weight loss. Patients with under-active vagus nerves often experience severe gastrointestinal problems. Those with overactive vagus nerves may faint (Snowdrop, 2009).

Because the vagus nerve supplies motor parasympathetic fibers to every organ from the neck down to the second segment of the transverse colon (except the adrenal glands), its effect can be far reaching. The vagus nerve is used to regulate the heartbeat and the muscle movement necessary to keep you breathing. This nerve also regulates the chemical levels in the digestive system so that the intestines can process food and keep track of what types of nutrients are being gained from the food that is taken in (appetite for food) (JAMA and Archives Journals, 2010).

The anatomical relationship of the efferent vagus nerve to the musculoskeletal structures at the occiput calls for manual therapy techniques such as suboccipital decompression which affect vagal functions. Manual therapies can affect vagal control, however none have targeted these particular anatomical structures and these particular therapeutic techniques. Manual therapy may have direct rather than indirect effects on the parasympathetic nervous system via manipulation of these cervical structures. Specifically, decompression of the occipito-atlantal (O-A) junction, a technique that focuses on treating an articular compression between the occiput and the atlas, may improve conditions relating to the path of the vagus as it exits the skull (Ghia et al., 2006).

Spasticity may be as mild as the feeling of tightness in muscles or may be severe enough to produce painful, uncontrollable spasms of the extremities; most commonly the legs, trunk and arms. Spasticity may also create feelings of pain or tightness in and around joints causing Muscle stiffness leading to movements to be less precise and making certain tasks difficult to perform. Muscle spasms leading to uncontrollable and often painful muscle contractions, involuntary crossing of the leg, Muscle fatigue, Inhibition of longitudinal muscle growth, inhibition of protein synthesis in muscle cells, Urinary tract infections, Chronic constipation, Fever or other systemic illnesses, Pressure sores (Bonaz, 2007).

As the vagus lies almost in immediate contact with the transverse process of the atlas, rotary subluxation of the atlas may induce pressure that can produce a broad range of vagus symptoms. The syndrome may include nasal and sinus congestion, swallowing and speech difficulties, cardiac arrhythmias, functional coronary artery spasm, gastric and intestinal colic, and other symptoms of vagal disturbance (Owyang and Heldsinger, 2011).

It is our vagus nerve that provides the gateway between the two parts of the autonomic systems. The vagus acts as a bio-informational data bus that routes impulses going in two directions. Since the vagus nerve acts as the central switchboard it should come as no surprise that impaired functioning of this one nerve can lead to so many different conditions and problems. Some neurological diseases actually come up from the gut spreading to the brain via the vagus nerve. Vagus nerve stems pass from the base of the brain stem, moving down the neck to the abdomen. Damage or pain in the vagus nerve can be caused by pressure from muscles or tendons that are too tight or impede on the nerve. Neck extension exercises will help reduce pain and pressure on the nerve (Kreier et al., 2002).

MATERIAL AND METHODS

Subjects

Thirty children from both sexes are characterized by low weight spastic diaplegic cerebral palsy children according to BMI (body mass index) scale were enrolled for this study, aged 6 to 10 years at time of recruitment because the children in this age are able to participate in ADL activities, children are able to stand alone and walk in crouch gait. Children who otherwise met the inclusion criteria were excluded if they had: previous BoNT-A injections in the lower limb in the past 12 months or prior lower limb surgery (i.e. tendon transfer/tendon lengthening).

Children randomized to the experimental group (A) received specialized physiotherapy program for cervical muscle spasticity control plus traditional physiotherapy program. Children randomized to the control group (B) received traditional physiotherapy program only.

The individual-based specialized physiotherapy program for cervical muscle spasticity control sessions of 30 minutes were conducted three times weekly for 12 weeks in an physiotherapy treatment room after the traditional physiotherapy session for group (A). In addition, children in the experimental group were exposed to home routine program 3 hours daily for the 12 week treatment period. Control group (B) received a traditional physiotherapy program only.

Outcome measurements

BMI Percentile Calculator for Child and Teen

-BMI is interpreted differently for children and teens even though it is calculated as weight ÷ height. Because there are changes in weight and height with age, as well as
their relation to body fatness, BMI levels among children and teens need to be expressed relative to other children of the same sex and age.

-Information needed in BMI in children
1. Birth Date. 2. Date of Measurement
3. Sex 4. Height, to nearest 1/8 inch
5. Weight, to nearest 1/4 (.25) pound

Weight Status Category Percentile Range
Underweight Less than the 5th percentile
Normal or Healthy Weight 5th percentile to less than the 85th percentile
Overweight 85th to less than the 95th percentile
Obese Equal to or greater than the 95th percentile

Intervention

For all children, the programs were conducted three times weekly, for 12 weeks. Each session lasted for 45 to 60 minutes in an physical therapy room, in addition to 3 hours of home program, 7 days a week during the treatment period.

Both groups (A and B) received a traditional physiotherapy program, as the following

1. Hot packs to improve circulation and relax muscle tension applied on the LL for 20 minutes.
2. Facilitation of anti-spastic muscles of LL: tapping followed by movement, quick stretch, triggering mass flexion, biofeedback, weight bearing, clenching to toes, compression on bony prominence, rapping the muscle, approximation, vibration, irradiation to weak muscles by strong muscles, and ice application for brief time.
3. Prolonged stretch to spastic LL muscles to gain relaxation via techniques used as prolonged stretch (positioning, night splint, reflex inhibiting pattern, Bobath technique) for 20 minutes.
4. Passive stretching to tight muscles (both tendo-achillis muscles, hamstring, hip adductors, hip flexors) to destruct adhesions in muscles and sheath. It must be decent gentle gradual stretch not over stretch at all, lasting 20 second then relaxation 20 second 3-5 times per session then maintain the new range by using adjustable ankle foot orthoses with knee immobilizers splint after session and during sleep.
5. Graduated active exercise for upper limb muscles and trunk muscles.
6. Gait training using aids in closed environment using obstacles, side walking followed by pass walking to stimulate protective reaction.
7. Balance training program which include static and dynamic training.
8. Faradic stimulation for both ant-tibial groups to modulate muscle tone, for triggering mass flexion of lower limb aiming for modulate extensor tone spasticity. To prevent cross electricity to reach cult muscles because these spastic muscles are more sensitive to electric stimulation than weak muscles. Mother was asked to support both ankles during electrical stimulation for 15 minutes.

The experimental group (group A) received specialized physiotherapy program for cervical muscle spasticity control as following:

This technique is a form of manual therapy that may directly affect the baroreceptors of the neck in addition to effects on other sensory afferent nerves. interventions designed to increase parasympathetic control

1- Prolonged stretch for back and neck extensors(by positioning and by complete flexion of both LL with flexion of trunk)
2- Suboccipital and paraspinal muscles myofascial release
3- Scapular mobilization for both sides to release adhesions in peri-scapular muscles
4- Mild, decent, gentle traction of cervical region from supine and head outside plinth
5- Hot packs on back and cervical region
6- Skin traction for upper fiber of trabizius
7- Passive stretch to SCM muscles
8- Deep sustained manual pressure on trigger points with short-duration pressure (1–2 seconds). common trigger points involve levator scapulae, the splenius cervicus lateral to the C4–C6 spinous processes, and the splenius capitis over the C1–C2 laminae. Peripheral inhibitory afferent impulses can be generated to partially close the pre-synaptic gate by acupressure

RESULTS

Patients characteristics

Table 1 shows the demographic and clinical characteristics of all patients. There were 14 boys (46.66%) and 16 girls (53.33%), and in term of Right hand dominance reported in 14 patients (46.66%), and also 16 patients (53.33%) were left hand dominance. There were no significant difference between the two groups in terms of age (p=0.2019), in term of sex (p=0.4814) and in term of hand dominances (p=1.0000).
Table 1. Patients’ characteristics.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group N=15</th>
<th>Control group N=15</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>7.67±1.45</td>
<td>8.27±1.03</td>
<td>0.2019</td>
</tr>
<tr>
<td>Sex N%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>6(40%)</td>
<td>8(53.33%)</td>
<td>0.4814</td>
</tr>
<tr>
<td>Girls</td>
<td>9(60%)</td>
<td>7(46.66%)</td>
<td></td>
</tr>
<tr>
<td>Hand dominance N%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>7(46.66%)</td>
<td>7(46.66%)</td>
<td>1.0000</td>
</tr>
<tr>
<td>Left</td>
<td>8(53.33%)</td>
<td>8(53.33%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. The average test of weight gain level in both groups.

<table>
<thead>
<tr>
<th>Weight gain level</th>
<th>Study group Mean±SD</th>
<th>Control group Mean±SD</th>
<th>P-value (within groups)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-treatment</td>
<td>17.73±2.34</td>
<td>18.20±2.70</td>
<td>0.6175</td>
</tr>
<tr>
<td>Post-treatment</td>
<td>18.67±2.47</td>
<td>18.47±2.64</td>
<td>0.6942</td>
</tr>
<tr>
<td>Improvement%</td>
<td>5.3%</td>
<td>1.48%</td>
<td>0.8861</td>
</tr>
<tr>
<td>P-value (within groups)</td>
<td>0.0002</td>
<td>0.0406</td>
<td></td>
</tr>
</tbody>
</table>

Changes in weight gain

Mean test scores and standard deviations for both groups are shown in the table 2. The mean value of weight gain level in both groups (assessed by weight scale) at baseline measurement (pre-treatment) was insignificant (p>0.05), while both groups had a significant improvement in weight gain post-treatment (p<.05). The average improvement of weight gaining level tended to being highly significant in the study group (17.73±2.34 versus 18.67±2.47, p=0.0002) than in the control group (18.20±2.70 versus 18.47±2.64, p=0.0406). The percentage of improvement of weight gain level was (5.3%) in the study group compared to the (1.48%) in control group.

DISCUSSION

The most important milestone in spastic diplegic C.P is independent walking

For severely involved children the question is if they will ever walk something that cannot be answered until they reach the age of 7 or 8 years. It is also not an either-or issue; a number of children are able to walk short distances, yet do not walk for longer ones. Some factors that might inhibit walking include:
- Ataxia
- Spasticity
- Lack of muscle coordination
- Muscles working against each other

Other important milestone in spastic diplegic C.P

Diplegia is usually noticed and may be diagnosed because the child is not walking. Parents often focus on this but it is important to remember that most children with diplegia will eventually walk independently but with physical dysfunction (the most important milestone) and there are also important milestones.
- Eating well
- Weight gain
- Overall health
- Average growth
- Development of hand function (Morton et al., 2006).

The vagus nerve is one of the largest nerve systems in the body. Only the spinal column is bigger. Sometimes this nerve is referred to as cranial nerve X, the 10th cranial nerve. The vagus is used to send a variety of signals throughout the body, but will also transfer signals back to the brain. The vagus nerve is constantly sending updated sensory information about the state of the body's organs "upstream" to your brain via afferent nerves. In fact, 80-90% of the nerve fibers in the vagus nerve are dedicated to communicating the state of your viscera up to your brain (Maser et al., 2007).

Neurologic disturbances may result from muscular and fibrotic changes along the cranial nerve pathways which exit from the skull and pass intimately between and under suboccipital fasciculi. Five of the cranial nerves are thus vulnerable: the facial, glossopharyngeal, vagus, spinal accessory, and hypoglossal. In addition, circulatory impairment of major and minor nerves of the neck may alter the function of those cranial nerves that do not exit from the skull proper, such as the olfactory, optic, oculomotor, trochlear, trigeminal, abducens, and auditory, but which are contained within the cranium and remote from vertebral subluxation encroachment effects. We should not overlook the fact that it is essentially muscle which produces and maintains the subluxation. Attention must be paid to the reasons why the subluxation has been produced and is maintained (Bueter et al., 2010).

Often times, pressure in the tendons or tense muscles can press upon vagus nerve that may lead to hyper or hypoactivity of vagus nerves. Dimensional exercises like neck extension (moving your chin away from chest as far as possible), neck flexion (touching your chin with your chest) and neck retraction exercises can help in decreasing pressure on vagus nerve. Other exercises can directly involve throat, floor of the mouth and base of the skull to improve vocalization, swallowing and breathing exercises are helpful in the moderate stimulation of vagus nerve that helps in improving the systemic functions of vagus nerve. Moreover, different postures that are adopted during exercise helps in releasing pressure from the delicate nerve fibers (Pardo et al., 2007).

Hypertonia of the suboccipital muscles may cause a decided impediment of venous drainage from the suboccipital area via vertebral and deep cervical veins, resulting in a passive congestion with consequent pressure upon the vagus nerve in the area. This is perceived by the patient as unilateral or bilateral pain and a throbbing discomfort, and may be palpated as knotty lumps within suboccipital muscles. The condition appears to be of a reflex nature more common among people under mental tension or those who work closely with their eyes over long periods (Bugajski et al., 2007).

Any event that would cause constriction in the connecting area between the cerebral subarachnoid space and the vertebral canal limits the escape of cerebrospinal fluid into the inferior vertebral canal. This results in a degree of increased intracranial pressure. An atlanto-occipital subluxation may cause the dura mater of the cisterna cerebellaris to be pressed against the posterior medullary velum and partially occlude the foramina of Luschka and Magendie and interfere with the flow from the 4th ventricle. The resulting increase of intraventricular fluid accumulation may create a large variety of symptoms such as deep-seated, stubborn, "internal pressure" headaches, nausea, a tendency toward projectile vomiting, unusual visual disturbances, and protopathic ataxias (Bugajski et al., 2007).
SELECTED EFFECTS OF CERVICAL AREA HYPERTONICITY

Excessive hyper-tonicity of a muscle, confirmed by palpatory tone and soreness, will lead to an atlanto-occipital subluxation and its site of osseous attachment as vagus nerve which lead to impaired of stomach function leading to remaining of food for prolonged time in stomach delaying its passage to intestine leading to loss of appetite for eating and weight loss. Below is a listing of common problem areas in the neck.

1. **Splenius capitis**. Increased tone tends to pull the C5–T3 spinous processes lateral, superior, and anterior and to sub-luxate the occiput inferior, medial, and posterior.

2. **Scalenus anterior**. Hyper-tonicity tends to pull the C3–C6 transverse processes inferior, lateral, and anterior and the 1st rib superior and medial.

3. **Scalenus medius**. Excessive tone tends to pull the C1–C7 transverse processes inferior, lateral, and anterior and the 1st rib superior and medial.

4. **Scalenus posterior**. Hyper-tonicity tends to pull the C4–C6 transverse processes inferior, lateral, and anterior and the 2nd rib superior and medial.

5. **Obliquus capitis superior**. Increased tone tends to roll the occiput anterior and inferior and pull the atlas posterior and superior to produce a lateral occiput tilt and condyle jamming.

6. **Obliquus capitis inferior**. Increased tone tends to produce a rotary torque of the atlas-axis motion unit.

7. **Rectus capitis posterior major**. Hyper-tonicity tends to pull the occiput posterior, inferior, and medial and the spinous of the axis superior, lateral, and anterior. Strong hyper-tonicity will lock the occiput and axis together so that they appear to act as one unit even though they are not contiguous.

8. **Interspinales**. Excessive muscle tone between the spinous processes tends to hyperextend the motion unit.

9. **Sternocleidomastoideus**. Increased tone tends to pull the sternum and clavicle posterior and superior and the occiput inferior and anterior.

10. **Upper trapezius**. Hypertonicity tends to pull the occiput postero-inferior, the C7–T5 spinous processes lateral, and the shoulder girdle medial and superior (Cui et al., 2006).

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Vagus Nerve Disorders

Symptoms of vagus nerve disorders are classified as overactive (in which symptoms are produced due to excessive release of neurotransmitters) and inactive or under-active (in which less stimulation of nerve interferes with the systemic functioning of organs). Classic symptoms of vagus nerve disorders include:

1. **Gastro-paresis**
   The under-activity of vagus nerve may interfere with the blood supply of stomach after ingestion of food that leads to improper digestion. Gastroparesis is marked by painful spasms in the stomach that affect normal food intake, heartburn, nausea and weight loss.

2. **Peptic Ulcer**
   Defects in the normal functioning of Vagus nerve may impair the normal control mechanisms that modulate the gastric acid secretion. Excessive secretion of peptic acid can lead to ulceration, dyspepsia and gastro-esophageal reflux disease.

3. **Muscle Cramps**
   Vagus nerve supply muscles of vocal cord and any defect or disorder of vagus nerve directly interfere with voice and breathing. Moreover, it may also involves other muscles that are supplied by vagus nerve.

4. **Difficulty in Swallowing**
   Glottis is normally closed when a person is swallowing in order to prevent the aspiration of food. This is managed by gag-reflex (gagging sensation if the back of throat is touched). In patients of head injury or stroke, gag reflex may get impaired leading to choking while eating and difficulty in swallowing.

5. **Organ Dysfunction**
   In some conditions, a branch or tributary of nerve is affected that leads to localized symptoms of organ dysfunction due to damage to nerve fibers or discrepancy in the synthesis of neurotransmitters.

6. **Pain**
   Pain is the most common symptom of vagus nerve disorders as a result of mechanical pressure, trauma or injury that leads to inflammatory swelling leading to neuralgia. Vagus nerve can be damaged throughout its course but most common nerve pain symptoms are due to pinched nerve (when nerve exits through tiny foramina in the skull).

7. **Fainting**
   Over-activity of Vagus nerve increases the firing rate of receptors that presents with sudden episodes of collapse and fainting (also referred to as vasovagal reflex). Although, it is not dangerous, but fainting episodes may increase the risk of accidental injuries that may prove life threatening.

8. **Other Symptoms**
   Other symptoms include changes in the rhythm of heart, urinary difficulties and changes in vocal tone (Henley et al., 2008).

In general, the abundant proprioreceptors of the vertebral column enable the brain to know where each segment is and what it is doing at any given time without visual confirmation. More specifically, data are relayed as to the degree of muscle tension and/or the length of muscles via the muscle spindles and Golgi tendon organs. Tension messages are moved through fast-conducting
nerves from annulo-spiral endings and through higher threshold nerves from flower spray receptors in the muscle spindle. The less complex Golgi tendon organs near the musculo tendinous junctions discharge impulses initiated by either muscle contraction or stretch. Other receptors near the articular surfaces relate messages about joint speed and direction of motion.

NEUROVASCULAR IMPLICATIONS OF UPPER CERVICAL SUBLUXATIONS

The upper cervical spine has been shown to be the most concentrated area of mechano-receptors (joint position receptors) in the body. The suboccipital muscles have been also been shown to have a very dense number of muscle spindle cells and GTOs (Golgi tendon organs). Muscle spindles measure the rate of change in muscle length, monitoring joint position as it relates to the muscle. GTOs measure muscle tension. Loss of mobility of any one or more segments of the spine correspondingly influences circulation. The resulting partial anoxia has a harmful influence upon nerve function. The artery and vein supplying a spinal nerve are situated in the foramen between the nerve and the fibrous tissue in the anterior portion of the foramen. It is unlikely that circulation to the nerve would be disrupted without first irritating or compressing the nerve because the arteries and veins are much smaller, the blood pressure within the lumen makes them resistant to compression, and nerve tissue is much more responsive to encroachment irritation.

The medulla oblongata extends well into the lower reaches of the foramen magnum and the ligamentous ring that connects it with the atlas, thus any type of occipital or atlantal subluxation may produce abnormal pressure on this portion of the brain stem. Bilateral posterior shifting of the occiput or atlas may cause pressure upon the pyramids or adjacent olivary bodies producing a syndrome of upper motor neuron involvement characterized by a degree of spastic paralysis or ataxia. A lateral shifting of the occiput may cause pressure upon the tubercle of Rolando producing pain in the area of trigeminal nerve distribution, headache, sinus discomforts, ocular neuralgias, and aches in the jaw.

Cervical subluxation (particularly atlantal, axial, or occipital) producing muscle spasm may produce unilateral or bilateral constriction of the vertebral arteries resulting in circulatory impairment. A large number of equilibrium, cardiac, respiratory, cranial nerve, extra-pyramidal, vagal, visual, and auditory symptoms may follow. The vertebral nerve (sympathetic) runs along the vertebral artery within the arterial foramen of the cervical transverse processes. Irritation to this nerve is considered to occur from mechanical irritation to the vertebral artery anywhere along its course producing symptoms of a vasomotor nature; eg, headache, vertigo, tinnitus, nasal disturbances, facial pain, facial flushing, and pharyngeal paresthesias. Cailliet points out that although sympathetic fibers have not been found along the cervical roots, surgical decompression of an entrapped nerve root relieves symptoms attributed to the sympathetics. The mechanism for this effect is unclear.

Vertebral Artery Compression. Associated subluxation development may produce vertebral artery compression. Symptoms of unsteadiness, dizziness, and fainting spells will occur especially when the head is rotated to the opposite side.

Autonomic Involvement. Vague autonomic symptoms may be exhibited such as dizziness, blurred vision, and hearing difficulties. These can usually be attributed to involvement of the plexus around the vertebral artery or intermittent disruption of the blood flow (Henley et al., 2008).

CONCLUSIONS

These data demonstrate that suboccipital decompression, often used in a manipulative medicine regimen, can affect stomach function variability acutely. This effect is consistent with potential changes in the control of heart by the parasympathetic nervous system. Further investigation in patient populations to determine the acute and long-term benefits of this and other manipulative medicine treatments is needed.

REFERENCES

Snowdrop (2009). Treatment of Language and Communication in Children with Cerebral Palsy