

Commentary

A Functional Explanation for a Proprietary External Head Weighting Device: A Commentary on Proposed Neurophysiological Mechanisms

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Abstract

Objective: The purpose of this commentary is to provide theoretical constructs as to how a head weighting device may work in human populations.

Discussion: A patented headweight device has been previously tested and published in the scientific literature. While these studies show the ability of this headweight device to make postural changes, the mechanisms behind this headweight device remain largely unknown. Theoretical constructs as to how this device may work in human populations are discussed.

Conclusion: The neuromotor control of posture may be affected by repeated external stimulus such as the headweight device described here. In the future, dosage, weight, and placement guidelines should be created to help physicians appropriately apply this postural headweight device.

Key Words: *Neurophysiology, Posture, Pettibon Technique, Rehabilitation, Subluxation, Spine*

Introduction

An external head weighting device patented by Pettibon has been used to improve abnormalities of the sagittal cervical spine.¹ Specific spinal disorders, such as scoliosis,²⁻⁵ lumbar kyphosis,⁶ and hunchback⁷ have been treated using this external head weighting device as part of a comprehensive active rehabilitation plan. Although the resultant changes in posture and cervical sagittal structure are readily apparent on lateral cervical radiographs,^{8,9} to this point explanations as to why these changes occur have not been proposed.

Neurologically, this device could be one of the most effective means for causing sustained input to the central nervous system. This sustained input has been shown to alter neuromuscular function and thereby promote lordotic and sagittal spinal correction evaluated by radiographic studies.^{8,9} According to the device's creator, one possible explanation for the corrections measured on radiograph is that the righting reflexes stimulated by the head weight device corrected the abnormal postures. A closer look at the mechanisms involved reveals that this statement is grossly true, but that further clarification is needed. This commentary serves to propose and explain some of the complex neurophysiologic relationships that may be responsible for the improvements demonstrated in plain film studies.

Review of Neuromuscular Function

To understand the weighting system we must first have an understanding of normal neuromuscular function. The muscular system can be broken down into the extrafusal system and the intrafusal system, indicating the duality of muscle function.¹⁰ The motor part of the muscle is known as the extrafusal system, and the sensory function of muscle is known as the intrafusal system.

Skeletal muscle originates embryologically from mesoderm. The motor part of a muscle is made up of contractile tissue, myofibrils which contain sarcomeres, made up of actin and myosin. When an action potential travels down a nerve to a muscle, calcium is released causing the actin and myosin bonds to rotate and cause contraction of the muscle. This is a gross oversimplification of muscle physiology, however; for the purposes of this paper, we are more concerned with both the afferent and efferent inputs to and from muscle rather than the actual muscle physiology itself.

The central integrative state of all neurons in the anterior horn of the spinal cord is dependent upon all of its pre-synaptic excitatory and inhibitory input. There are multiple inputs, but very importantly there is both inhibitory and excitatory input

from the cortex into the anterior horn.¹¹ It is well known that the resting length of muscle is under the influence of cortical drive through the gamma motor neuron's effect on the polar ends of muscle spindle cells. The gamma motor neuron's cell body, like the alpha motor neuron's cell body, resides in the anterior horn of the spinal cord. There is also evidence of some alpha motor neuron connections with the intrafusal system.¹²

The muscle spindle, which is located in the belly of the muscle and in parallel with the extrafusal system, is connected to the extrafusal system through a glyco calyx. The spindle is innervated by 1a afferents that are an extension of the annulospiral ending receptors found mostly on the nuclear bag, but with connections to the nuclear chain as well.¹³ The nuclear chain of the muscle spindle cell is also innervated by the 1a afferent, but in addition has a type II afferent input. The nuclear bag has no contractile tissue in it, but the polar ends of each muscle spindle are contractile and are innervated by gamma motor neurons.

According to Hall,¹⁴ the muscle spindle is believed to be more involved in the dynamic stretch reflex, and the nuclear chain is believed to be mostly involved in the static stretch reflex. However, Gandevia and Burke¹⁵ explain these pathways in greater detail than Hall. The intrafusal system is comprised of three histochemically different fibers: the static nuclear bag, the dynamic nuclear bag, and the nuclear chain. The human muscle spindle contains 2-14 intrafusal fibers (1-4 nuclear bag fibers and 3-10 chain fibers).¹⁵ Kandel et al¹⁶ also explain that there is both a dynamic gamma motor neuron and a static gamma motor neuron.

Then their connections as follows: the 1a afferent has connections with the dynamic nuclear bag, static nuclear bag, and nuclear chain fibers. Type II fibers innervate the static chain and bag, but not the dynamic bag.¹⁷ The 1a afferent system can respond to all inputs and is one of the fastest nerve fibers found in the body, and the type II fibers which are slower, are associated only with the static stretch reflex.¹⁸ For the purpose of simplifying this to a degree we will say that when we refer to the nuclear bag we are referring to the dynamic reflex and when we refer to the chain, we mean the static reflex.

Hall¹⁴ explains that when a muscle is stretched the dynamic stretch reflex is initiated immediately and ends very quickly. Its main purpose is to resist the stretch of the muscle being stretched. This is seen as a protective mechanism. If a stretch is maintained on the muscle, i.e. it is now at a new length, the static stretch reflex is maintained for minutes if need be. It is important to note that per Guyton, the static stretch reflex is only maintained in the presence of stretch. Therefore, when an adjustment/ manipulation is performed the dynamic stretch reflex would most likely be activated, but immediately would go quiet, and the static stretch reflex would likely not be activated as there is no longer a stretch being maintained on the muscles involved after the spinal manipulation.

The Pettibon headweight device acts as a prolonged slow stretch on the cervical spinal extensor muscles. To understand this better, a look at the peripheral and central effects of muscle stretching is required. When a muscle is stretched the

1a afferent of the muscle spindle is excited and sends an excitatory signal to the alpha motor neuron in the anterior horn of the spinal cord to the original muscle from which it came. This is the basis for the monosynaptic myotatic reflexes commonly checked on musculoskeletal patients.

In addition, a primary branch fires up through the dorsal spinocerebellar tract through the inferior peduncle to excite the ipsilateral cerebellum, and another branch fires to an inhibitory interneuron to inhibit the antagonist muscle. This latter reflex is known as reciprocal inhibition.¹⁹

When a muscle is stretched or lengthened, its tone increases and its inherent resistance to stretch is activated by this mechanism. There is no real lengthening of the muscle as this would require a break in the myofilament bonds, and this would cause pathological tearing of the muscle. Other elements that are a part of passive loading of the series elastic element may be stretched, such as blood vessels, connective tissue, capsular ligaments etc. This may be a part of what we see when we think that a muscle is lengthening. We may also see a partial inhibition, and firing off of the golgi tendon organ through prolonged stretch and therefore increased tension on the musculotendinous junction.

An important element of normal muscle physiology is the way in which the nervous system monitors muscle tension. This is accomplished through the 1b afferents found in the Golgi tendon organs (GTO), which are found at the musculotendinous junctions. When stimulated, the GTOs fire an excitatory signal to an inhibitory interneuron in the anterior horn which has an inhibitory effect on the same muscle from which the signal originated. In addition, it sends an inhibitory signal through the dorsal spinocerebellar tract, which reduces the central integrative state of the ipsilateral cerebellum to which it fired.²⁰ For neuro-anatomical accuracy there is also a double decussating pathway through the ventral spinocerebellar pathway that synapses on the ipsilateral cerebellum through the superior cerebellar peduncle.²¹

Chiropractors can appreciate that the neuromuscular system has developed a highly sophisticated system for monitoring baseline muscle tone and length, as well as changes in muscle length and tension. This ability of the nervous system to monitor both length and tension in muscles is of enormous consequence when we consider joint position sense, coordinated movements, balance, and many other functions. The importance of this proprioceptive system is further demonstrated by virtue of the fact that the nervous system uses some of the fastest nerves in the human neuraxis to process this information.

Discussion

When a patient is fitted with a headweight device on the anterior aspect of the head, we are activating the muscle stretch reflex immediately. The weight causes a slow stretch on the spinal extensors that must now function to oppose the force of the added weight. If the appropriate amount of weight is used, then the excitation of the muscle spindle will cause an increased frequency of firing of the spindle cell, and therefore an increased frequency of firing of the alpha motor neurons to the very muscles being stretched increasing tone and resisting

the stretch. This will in turn also facilitate the ipsilateral cerebellum, through the pathways outlined earlier. As the weights are placed on the anterior skull, the center of mass of the skull effectively shifts anteriorly. Once this occurs, the dynamic stretch reflex is elicited in the cervical extensor musculature, responding to the added weight opposite the cervicothoracic lever arm. As the new position is maintained, the static stretch reflex remains activated to resist the ongoing muscle stretch via the headweight device. The static stretch reflex works to maintain a frequency of firing to resist the muscle stretch and increase tone of the spinal extensors.

If too much weight is used, or if the metabolic demands of the weight exceed the muscular ability to resist it, the weight may cause an offload from the spindle to the Golgi tendon organ (GTO). In this case, where the stretch exceeds the muscle's ability to maintain resistance to that stretch, then an increased amount of tension would be transferred to the musculotendinous junction. This may exceed the threshold of the GTO, causing a massive inhibition via 1b afferents to the spinal extensor muscles thus protecting the muscles from tearing. This may explain why some patients appear to worsen or do not change when the weights are placed on them. This could occur in absence of ligamentous damage due to a weakened metabolic state, and very likely when a patient does exhibit ligamentous damage as they may also then have decreased shunt stabilization required for proper muscle contraction and joint function.

As stated prior, the head weighting device puts a slow stretch on the spinal extensor muscles, which facilitates contraction of these muscles by the aforementioned mechanism, thus pulling the head and spine into relative extension. This relative change in head position has further impact on the central nervous system in the following ways. The change in joint position further stimulates type I mechanoreceptors found in the zygapophyseal joints and joint capsules of the spine. This has an excitatory effect upon the cerebellum bilaterally which then fires into the vestibular nuclei located in the floor of the fourth ventricle of the pons. The medial vestibular nuclei are largely responsible for cervical spine extensor tone, as a function of maintaining horizontal gaze.^{10,22}

The next consequence of the change in head position is that it activates the vestibular canal system. The most likely canal to be stimulated would be the posterior canal due to its orientation and the typical positioning of the headweights with 80% of the weight on the front and 20% of the weight rotated to the side. If the patient can maintain this it would pull the head into relative lateral flexion and extension. This would exactly oppose the anterior canal which would typically be quite plastic in a person with prolonged forward head posture. The canal system fires powerfully into the vestibular nucleus thereby stimulating the vestibulospinal pathways and the medial longitudinal fasciculus. The medial longitudinal fasciculus, being the first pathway to myelinate in the human, connects the vestibular nuclei to cranial nerves III, IV, VI, and XII. The reason for these connections is to maintain foveation on the retina during head movement and changes in head position. The fovea is concerned with directing a crisp visual image to the occipital cortex. Collectively, these are the righting reflexes to which Pettibon refers as causing the headweight to work;¹ the vestibulo-ocular reflex in particular.

This has been extensively discussed in a previous review by Morningstar et al.²³

In cases where the expected changes do not occur, there might be a loss of integrity to the ligamentous structures creating instability, which could be observed through motion x-ray, but it might also indicate a weakened vestibular system unable to cope with the input, or a trans neurally degenerating cerebellum that fatigues due to the increase stimulation and lack of oxygen or glucose or other metabolic related issues such as an autoimmune disease.

The shoulders and hip afferents fire primarily into the spinal cerebellum, the globose and emboliform nuclei located near the midline of the cerebellum. When a patient is fitted for shoulder and or hip weights, once again there is tremendous afferent input into these areas. Depending on the area of the cerebellum that is under functioning a patient might see greater improvement with the addition of these body weights then with midline stimulation alone as with the head weights. In addition, using weights on the body as well as the head affords more spatial summation to the nuclei involved and may promote c-jun, c-fos, and c-mir protooncogenic expression.

Conclusion

It seems plausible, if not already accepted as self-evident, that poor posture or postural habits may be considered a primary factor in the development of articular dysfunction. Our hopes in writing this commentary is to shed some light on the idea that specific neuromuscular rehabilitation techniques may have the ability to correct posture and thus reduce the potential recurrence of chronic spinal or extravertebral dysfunction. We simply want to propose a viable explanation as to why external asymmetrical weighting seems to have the postural modification capabilities that preliminary studies have demonstrated.

All patients fitted for these weights should have a complete neurological examination to determine the areas of weakness centrally not just peripherally and should be tested to determine the capacity for handling this type of therapy. It is important to note that not all stimulation is good, nor is all stimulation bad, It is individual to the persons own neurologic and metabolic system. A system that has lost its central integrative state and has a loss of protein replication from decreased activation along with decreased fuel supply may be weakened by an input that exceeds its metabolic capacity.

Future research into external head and body weighting should include testing those outcomes that provide the most insight into the patient's adaptive response, as well as including data on other forms of external weighting besides head weighting, such as shoulder, hip, chest, wrist, or ankle weighting.

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