A NEUROLOGICAL RATIONALE FOR INJURY RECALL TECHNIQUE

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Abstract: It is well known that muscle spindle mediated stretch reflexes are commonly modified by descending suprasegmental pathways arising from cerebellar and cerebral adaptations. These concepts are reviewed and a model is presented for understanding the effects of injury recall technique (IRT) in light of suprasegmental, primarily cerebellar, adaptation.

INTRODUCTION

The three major sensory inputs for postural control are the eyes, the inner ears (the vestibular mechanism), and mechanoreceptors from the ankle joint. Postural adaptation depends on cerebellar integration of all three areas and its efferent supply to brainstem centers for descending pathways to motor neuron pools. This paper proposes that the clinical findings of injury recall technique (IRT) [1] [2] are associated with cerebellar adaptation and habituation to areas of injury and trauma.

DISCUSSION

The cerebellum receives primary inputs from the cerebral cortex (through the corticopontocerebellar tracts via the inferior olive,) the inner ear (both directly from the vestibular nerve and indirectly via vestibular nuclei,) and ipsilateral mechanoreceptors (MR) (via the dorsal and ventral spinocerebellar tracts and the cuneocerebellar tracts.) The most important MR inputs to the cerebellum arise from muscle spindle afferents, from both primary and secondary muscle spindle cell receptors.

The stretch reflex has local spinal cord effects to increase muscle tone proportionally to the amount of stretch. The stretch reflex afferent information is also carried to the cerebrum via the thalamus and to the cerebellum as previously mentioned. These higher levels allow for adaptive changes (plasticity) in the stretch reflex modulated by descending pathways.

These adaptive changes are most often explained in neurology texts by the example of stretch reflexes associated with dorsiflexion of the foot. [3] [4] When the gastrocnemius and soleus are stretched, this causes a reflexive contraction of both muscles to bring the foot back to its original position. If a standing person leans or sways forward, these muscles reflexively contract to bring the person back to original upright position. However, if the floor under toe toes tilts upward causing the same amount of stretch to the gastrocnemius and soleus, the reflex contraction of these muscles causes the person to fall backward. If the floor is repeatedly tilted in this fashion causing dorsiflexion of the feet, there is an adaptive response where the stretch reflex response becomes decreased, that is, altered to meet the new environmental input.

A good example of this phenomenon is how a person's stretch reflexes adapt when on a rolling boat. After there is a new, habituated decrease in the dorsiflexion stretch reflexes, the person better tolerates the boat's movement. However, when the person returns to dry land, there is a period of instability until the stretch reflexes adapt back to their original environment. As a result of this changing in adaptation, most people report that they feel like they are still on the boat for a period of time after they are on land.

Such adaptations depend on projections from cerebral cortex to the cerebellum. The cerebral cortex itself is activated in this regard by afferents arising from muscle spindle cells. People with cerebellar disorders do not have the ability to adapt to the rolling boat. In other words, there is no plasticity of the stretch reflex due to the cerebellum's inability to adapt. [3]

Following injury, there are various and numerous alterations in stretch reflexes as they adapt to flexor reflex afferent pathways arising from nociceptors. The secondary effects of nociception become the new normal as the person adapts to the trauma, just as the person adapts to the rolling boat. It is proposed that, if the trauma is significant enough, an adaptation of cerebellar modulation of stretch reflexes, which must take place, becomes the new norm as nociceptors continue to fire and change stretch reflex responses while the injury heals.

If the injury is one which will require IRT, the synthesis of local, cerebellar, and corticocerebellar adaptations creates an alteration in posture, part of which is reflected in ankle proprioceptive adaptations to the injury. It is interesting that most texts explain stretch reflex adaptation using the example of ankle dorsiflexion, and this is the same pattern we see associated with injury and IRT. (Note how a supine person's feet dorsiflex when they are exposed to nociceptive input such as a hard or painful manipulation.)

One can see that IRT is associated at least in part with cerebellar adaptation by applying functional neurological assessment of cerebellar function in parallel with IRT. There are always inputs to the cerebellum from the vestibular complex in the inner ear. The three bilateral semicircular canals are arranged such that any head movement will be associated with activation of at least one, and usually two or three of them on each side. The semicircular canals cause reflex changes in postural muscle activity. Putting the head in a distinct position to activate primarily one of the canals will elicit reflex responses which are characteristic of that canal.

The six head positions which will activate specific semicircular canal activity are:

1) rotating the head to the right (right lateral canal)
2) rotating the head to the left (left lateral canal)
3) tilting the head anterior and left (left anterior canal)
4) tilting the head posterior and right (right posterior canal)
5) tilting the head anterior and right (right anterior canal)
6) tilting the head posterior and left (left posterior canal)

The positive challenge for IRT is when a conditionally facilitated (strong) muscle becomes conditionally inhibited (weak) when the area of previous injury is activated by patient touch or doctor stimulus (usually pinching) and the talus is challenged in a cephalward direction. To demonstrate the cerebellar relationship to a positive IRT pattern, simply activate the area of injury by patient touch or doctor pinching, but rather than add the talus challenge, place the head in one of the six positions to activate one of the semicircular canals as mentioned above. This will result in a strong muscle weakening just the same as the cephalward talus challenge does.

Correction of IRT is by micro-manipulation of the talus in a distal direction while the area of injury is activated. Following this correction activation of the injury with the positive semicircular canal related head position is now negative. This suggests that the IRT problem was associated with plastic adaptation in the cerebellum which is no longer present following IRT correction.

CONCLUSIONS

A normalization of all of the accompanying concomitants to plasticity-altered (adapted) cerebellar and cerebral functions explains the far-reaching and dramatic effects often achieved by IRT. This includes changes in sensory and motor functions, autonomic concomitants, and improved cognitive function. The cerebellar adaptation theory of IRT also opens the door to understanding how to integrate IRT with other procedures; specifically why this author recommends that, when indicated, IRT be performed prior to any other therapies which may affect cerebellar functions. This includes, of course, all other manipulations.

The cerebellum is the integrator of all motor functions. If there is plasticity in the cerebellum causing adaptation away from optimal function, such as an adaptation to a major trauma, this must become the applied kinesiologist's first treatment step in the overall goal of muscle balancing and AK procedures.

REFERENCES

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