Letters to the Editor

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Wrist Splint for Upper Motor Neuron Paralysis

To the Editor:

There are a number of concerns with the study published by Stroke written by Lannin et al.1

The concerns are: (1) the randomization; (2) the title; (3) the sample of stroke participants; (4) the volar splint; and (5) the splinting groups with the wrist and fingers in a “neutral” and in an “extended position.”

Randomization was based on the diagnosis and time from onset, not the degree of spasticity. Therefore, it is obvious that change in flexor tendon extensibility and possible effectiveness on onset, not the degree of spasticity. Therefore, it is obvious that an “extended position.”

The study states the splints used were “volar.” This has implications because it disregards how a spastic muscle responds. The splints in Figures 1 and 2 are not “volar” splints, because there are 4 large straps applied over the “dorsal” surface. In addition, the splints were molded to include additional “dorsal” surface for the dissipation of forces and for maintaining the intended position. The correct terminology for such a splint is “a predominantly volar splint.”2

There was no rationale for the selected positions of “neutral wrist” and “45° extended wrist.” Both positions are inappropriate for splint effectiveness for the stated level of spasticity. To show changes in the extensibility of fingers and wrist flexors and to prevent contractures, it is vital to position the hand just beyond the point of stretch reflex. Every group of spastic muscles (in this case wrist flexors) has its own range of motion that it could be moved before a hypertonic reaction “clasp-knife” phenomenon occurs.2,3 This is the point where a hypertonic stretch response begins to be felt from 1A afferents when the joint is moved passively. The “wrist neutral” is thus invalid. The “45° extended wrist” is also invalid as it is beyond the point at which the stretch reflex melts away from the homonymous inhibitory Golgi tendon organ. After this point or wrist angle, there is no resistance to passive stretch. Such randomly selected wrist positions are inappropriate for determining splinting effectiveness. Those participants in whom the hypertonic response began to be felt early, which are those with moderate to severe spasticity, would benefit from a smaller wrist splint angle, whereas those with mild to moderate spasticity would benefit from a greater wrist angle.

The purpose in preventive splinting is to push the point of stretch reflex as far back into wrist extension as possible to encourage tone neutralization and develop emerging volitional control.

In conclusion, the authors should be commended for their attempt to investigate the effectiveness of wrist splints in preventing contractures; however, the results are questionable because of the flaws in randomization, splint construction, splint application angle, and arbitrary selection of the angle of the wrist.

Disclosures

None.

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