Immediate Effects of Positioning Devices on the Normal and Spastic Hand Measured by Electromyography
(volar splint, cone, finger spreader, CVA, hypertonus)

Virgil Mathiowetz

There is a continuing controversy in the literature and in clinical practice about whether positioning devices are effective in decreasing spasticity and, if so, which type is most effective. In this study, the immediate effects of a volar resting splint, a finger spreader, a firm cone, and no device were compared on eight normal and four hemiplegic subjects. Electromyography (EMG) was used to measure flexor muscle activity of the tested forearm while the subjects squeezed a grasp meter with the contralateral hand and during the following relaxation period. Results showed significantly greater EMG activity for the finger spreader compared to no device in the flexor carpi radialis of normal subjects during the grasping period. Hemiplegic subjects did not show significantly less EMG activity when using positioning devices compared to no device. In fact, the volar splint appeared to increase the EMG activity while the subjects were grasping.

Deborah J. Bolding

It has been estimated that 2 million individuals in the United States have spasticity as a result of strokes (1). The most common pattern of spasticity in the upper extremity includes retraction and depression of the scapula, internal rotation of the shoulder, flexion of the elbow and wrist, as well as flexion and adduction of the fingers and thumb (2). Spasticity in the impaired arm is often increased by the active use of the unimpaired arm (3); according to Bobath (4, p 50) this "serves to maintain spasticity and thus counteracts the recovery of hand and fingers." Therapists use a variety of methods to decrease spasticity in the impaired upper extremity (5), one of which is the use of a positioning device. But are positioning devices effective in decreasing spasticity and, if so, what type of device is most effective?

Traditionally, volar resting splints have been used for the prevention of contractures and deformities caused by flexor spasticity (6-9). In 1959, Brennan (10) studied the effects of prolonged stretch of spastic muscles using a well-padded volar splint. Both the wrist and finger flexors of 4 of his 14 subjects
were splinted from 8 to 18 weeks. At the end of the stretching period, the experimental group of splinted wrist and finger flexors had an average increase of 59 degrees in range of normal tone (range of passive movement from full flexion toward full extension during which no resistance to stretch could be felt). The adjacent, unsplinted elbow flexors were used as controls and showed only a 2.5 degree increase.

The adjacent, unsplinted elbow flexors were used as controls and showed only a 2.5 degree increase in range of normal tone. He concluded that "tonic muscle spasms shown by certain flexor muscle groups in patients with residual hemiplegia can be abolished by submitting the muscle to a long period of stretch." (10, p 1506)

By 1962, therapists and physicians were becoming increasingly aware of the neurophysiological effects of splinting. Kaplan (11) designed a dorsal splint for the stimulation of the extensor muscles and for the reciprocal inhibition to the spastic flexor muscles. Although his long-term study lacked a comparison group, Kaplan concluded that dorsal splints were considerably more favorable than volar splints in diminishing spasticity in flexor muscles.

Two studies compared the effects of dorsal versus volar splinting. Zislis (12) compared the immediate effects of dorsal, volar, and no splint on the same hemiplegic subject. Using raw EMG recordings, he found that flexor activity was greatest with the dorsal splint and least with the volar splint. Zislis concluded that volar pressure may cause flexor inhibition, whereas dorsal pressure increases flexor spasticity. Charait (13) compared ten hemiplegic subjects with volar splints and ten with dorsal splints. The subjects wore their splints from 2 to 23 hours per day, from 2 months to 2 years in duration. Various physicans and therapists evaluated the subjects' spasticity by clinical observation. Charait concluded that pressure on the volar surface of the hand appeared to increase spasticity in the flexors, whereas pressure on the dorsal surface of the hand appeared to facilitate the extensors and inhibit the spastic flexors. From very different methods of study, Zislis and Charait drew contradictory conclusions regarding the effects of volar and dorsal splinting.

As neurophysiological approaches to treatment became more accepted, splinting was increasingly questioned and alternatives were proposed. Rood stated: "A critical analysis of braces and splints frequently indicated that their application defeats their purpose by activating sensory stimuli of touch, pressure, and stretch which results in undesirable contraction of muscle." (14, p 444) She and others (15-18) recommended the use of a firm cone to provide constant pressure over the entire flexor surface of the fingers (15) and to put pressure on the insertions of the spastic wrist and finger flexors (16). This pressure was thought to have an inhibitory effect on the long flexors of the hand.

Bobath questioned the use of cock-up splints. She suggested the use of a foam rubber finger spreader that abducts the fingers and thumb. She stated, "Abduction not only facilitates extension of the fingers but also reduces flexor spasticity throughout the whole arm." (2, p 107) More recently, Doubilet and Polkow (19) and Snook (20) described static splints based on this Bobath concept.

The controversy in the literature over whether to splint and the most effective positioning device for the spastic hand is reflected in present clinical practice. Neuhaus, et al. (21) found that most therapists considered the effects of splinting on spasticity of moderate to major importance in deciding whether to splint. Within this group, some believed splints inhibited spasticity; others believed they facilitated it; and still others responded as if the effects of splinting were dependent on the severity of the spasticity. For example, 75 percent of therapists chose to splint when presented with a hemiplegic patient with moderate-to-severe spasticity, but only 24 percent would splint a patient with minimal spasticity.

The controversy continues because there has been no definitive study comparing the effects of various positioning devices and no device on the spastic hand. In addition, data on normal hand response to positioning devices are lacking. The purpose of this study is to compare the immediate effects of a volar resting splint, a finger spreader, a firm cone, and no device on the normal and spastic hand as measured by EMG.

Because there is little-to-no EMG activity in normal and spastic hand muscles at rest (22), a method of indirectly eliciting flexor muscle activity in the tested forearm was needed. The method chosen for this study was similar to the one used by Hopf, et al. (23), who studied "irradiation" to the contralateral side by having subjects squeeze a grasp meter with one hand while...
recording EMG activity in the contralateral unexercised forearm. For the purposes of this study, irradiation, or flexor muscle activity, is defined as the cumulative, integrated EMG (IEMG) activity measured in mm/second from the surface electrodes placed over the flexor capri radialis (FCR) and the flexor digitorum profundus (FDP) of the unexercised forearms of the normal and hemiplegic subjects.

The following hypotheses were proposed: First, for normal subjects, there would be no significant difference in flexor muscle activity of the unexercised FCR and FDP when using a volar resting splint, a finger spreader, a firm cone, or no device during the grasping period. Second, for hemiplegic subjects, there would be significantly less flexor muscle activity of the unexercised FCR and FDP when using a volar resting splint, a finger spreader, or a firm cone than when using no device during the grasping period. Third, for both normal and hemiplegic subjects, there would be no significant difference in flexor muscle activity of the unexercised FCR and FDP when using a volar resting splint, a finger spreader, a firm cone, or no device during the post-grasp periods.

The Volar Resting Splint was Rolyan's functional position hand splint (Rolyan Medical Products, Menomonee Falls, WI 53051) constructed from rigid polyform with three Velcro straps: 2.5-cm (1-inch) straps over mid-forearm and wrist, and 5-cm (2-inch) strap over the fingers. The Firm Cone was Rolyan's preformed cone with a single 5-cm (2-inch) wide Velcro strap. The Finger Spreader is pictured in Figure 1.

### Methods

**Subjects.** Eight normal and four hemiplegic subjects volunteered to participate in the study. Normal subjects were free of neuromuscular or orthopedic dysfunction and ranged from 25 to 40 years of age. The hemiplegic subjects (Table 1) had moderate to severe spasticity in the impaired wrist and fingers. Stretch reflex was elicited in the first two-thirds range as wrist and fingers were rapidly and passively extended (5, p 38). All hemiplegic subjects were medically stable with no cardiac problems or uncontrolled hypertension.

**Instrumentation.** A Grass model 7 polygraph (Grass Instrument Co., 100 Old Colony Avenue, Quincy, MA) was used to record EMG activity in the FCR and FDP of the unexercised forearms. The EMG activity was amplified and displayed on a Grass polygraph and a Grass digital display. The EMG activity was also recorded on a magnetic tape for later analysis. The EMG activity was recorded at a paper speed of 100 mm/second and a time constant of 0.1 seconds. The EMG activity was recorded for four time periods: during the grasping period, and during the first, second, and third post-grasp periods.

### Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Age/Sex</th>
<th>Age/Post- Onset (yrs)</th>
<th>Involved Side</th>
<th>Spasticity Wrist/Fingers</th>
<th>Cause of Hemiplegia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26/F</td>
<td>8</td>
<td>R</td>
<td>Moderate/Moderate</td>
<td>Aneurysm in Anterior Cerebral Artery</td>
</tr>
<tr>
<td>2</td>
<td>69/F</td>
<td>7</td>
<td>R</td>
<td>Severe/Severe</td>
<td>CVA (uncertain cause)</td>
</tr>
<tr>
<td>3</td>
<td>19/M</td>
<td>4</td>
<td>L</td>
<td>Moderate/Severe</td>
<td>CVA due to Embolus</td>
</tr>
<tr>
<td>4</td>
<td>61/M</td>
<td>9</td>
<td>R</td>
<td>Moderate/Moderate</td>
<td>CVA due to Carotid Artery Occlusion</td>
</tr>
</tbody>
</table>

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MA 02169) and Beckman silver/silver chloride, 2-mm diameter, surface electrodes (Beckman Instruments, Inc., Schiller Park, IL 60176) recorded the EMG activity in the FCR and FDP of the unexercised forearm. Because only a general measure of flexor muscle activity was needed, surface, rather than indwelling, electrodes were used. Raw data were recorded by 7P3 differential pre-amplifiers with input impedance of 14 megohms, with half amp frequency range of 10-75 Hz to pens and 10-20,000 Hz to 7P10 cumulative integrator, with sensitivity calibrated at 33 μV/cm, and with chart speed of 25mm/second. IEMG activity was recorded by 7P10 cumulative integrators with an input impedance of 500 K ohms and half amp high frequency of 35 Hz. No artifact was detected on the raw data so all IEMG data collected were analyzed. The signal from the grasp meter was recorded by a 7P1 low level DC amplifier with an input impedance of 2 K ohms and simultaneously displayed on a Tektronix 511 Oscilloscope (Tektronix, Inc., Box 500, Beaverton, OR 97005) to enable subjects to visually monitor percent maximal voluntary contraction (MVC) of grip. The motor point of each muscle was located by a Teca (Teca Corporation, White Plains, NY 10603) SP2 AC electrical stimulator. The recording electrodes were attached 1 cm on each side of the motor point, parallel to the long axis of the muscle. Skin preparation included brisk rubbing with alcohol pad, light abrading with emory paper, and massaging electrode cream with a stiff brush over the electrode site to decrease skin impedance to less than 10 K ohms. A ground electrode was clipped to the subject's ipsilateral ear.

Procedure. All subjects were seated on an armless wooden chair with their feet flat on the floor, their heads in midline, and their arms hanging freely at their sides. Before testing began, each normal subject was asked to give a MVC of grip of the unimpaired hand from which 80 percent MVC was calculated. The 80 percent MVC of grip was selected for the normal subjects based on a pilot study that found the amount of overflow increased as percent of MVC of grip increased. As a consistent but large amount of overflow was desired, 80 percent ± 10 percent MVC of grip was selected. Normal subjects were able to consistently hold this percent of grip for only 10 seconds. Before testing, subjects practiced holding 80 percent ± 10 percent MVC grip for 10 seconds while watching the visual display for feedback.

As recommended by Gregg, et al. (24), a 2-minute rest period was provided to avoid the effects of fatigue from the practice. After the first positioning device was put on, subjects were allowed a 2-minute period to adapt to the positioning device and to allow the EMG activity to return to a baseline level. During the first 10 seconds of the test recording, each subject squeezed 80 percent ± 10 percent MVC of grip that caused overflow to the impaired side. During the following 45 seconds of the test recording, the subjects relaxed with the grasp meter still in their hand. After the test recording was completed, the positioning device was removed. After another 2-minute rest period, the next positioning device was put on and the subject was again allowed a 2-minute period to adapt to the device before the next test recording began. The same procedure was repeated for all four test conditions.

The procedure for the hemiplegic subjects was identical except that they squeezed 50 percent ± 10 percent MVC of grip (one half of their maximal effort) for 15 seconds to avoid undue cardiovascular stress. The assumption was made that less effort was needed to elicit overflow in the hemiplegic subjects. To obtain a more complete view of the immediate effects of the positioning devices over time on hemiplegic subjects, the EMG was recorded starting with a 15-second baseline before the devices were put on and continuing through the test recording.

Intervening variables such as fatigue, order of trials, the normal variability of flexor muscle activity over time, and the consistency of overflow were controlled by using a balanced design sequence for devices. The variability of flexor muscle activity between subjects was controlled by having each subject act as his or her own control. Efforts were made to minimize the effects of environmental factors such as temperature and visual and auditory distractions by using the same room for all subjects. Other factors such as changes in medications and medical and emotional status were partially controlled by the relatively short (20-30 minutes) testing time.

Data Reduction and Analysis. The test recording was divided into four time periods: the grasping period was the first 10 seconds (15 seconds for hemiplegic subjects) that the subjects were within their percent MVC of grip; the post-grasp periods 1, 2, and 3 (15 seconds each) were measured in the 45 seconds after the grasp meter returned to baseline. The average IEMG activity was calculated in mm/sec for each device, and for each muscle (FCR and FDP). Too many variables prevent the direct comparison of this unnormalized IEMG data across subjects or muscles. The
most common method of normalizing EMG data (25) was impossible to use because of the inability to differentially test spastic muscles. Consequently, the method used by Perry, Easterday, and Antonelli was selected. This method related “the individual test results to the total of all data from that electrode.” (26, p 9) In this study, the normalized score was derived by dividing each test score by the sum of all 16 test scores for that muscle. All statistical analyses, tables, and graphs were done using normalized scores that will hereafter be referred to as EMG activity.

Results

Grasping Period. For normal subjects, the one-way ANOVA showed a significant difference between devices for FCR (Table 2). A Newman-Keuls post-hoc test located significantly greater EMG activity for the finger spreader as compared to no device. A trend of higher EMG activity for the finger spreader was also noted in the FDP (Figure 2) but did not reach the p < .05 level of significance.

For hemiplegic subjects, the results of one-way ANOVAs were not significant (Table 2). This was probably due to the high variability among subjects. A trend of higher EMG activity was noted in both the FDP and FCR for the volar splint (Figure 3). Clearly, the volar splint, the finger spreader, and the cone did not evoke less EMG activity than no device.

Discussion
The results showed significantly greater EMG activity for the finger spreader compared to no device in the flexor carpi radialis of normal subjects during the grasping period. This result would reject the first hypothesis, which predicted no significant differences between devices. The initial explanation of this unexpected result was based on the fact that five out of eight subjects reported that the finger spreader seemed to stretch their fingers or seemed uncomfortable. This passive stretch could stimulate la afferents of the muscle spindle to activate alpha motor neurons (27).

### Table 2

One-way ANOVAs of Repeated Measures between Devices during the Grasping Period

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal Subjects: N = 8</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FDP Between Devices</td>
<td>3</td>
<td>1405.4</td>
<td>1.93</td>
<td>.16</td>
</tr>
<tr>
<td>Between Subjects</td>
<td>7</td>
<td>1051.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>21</td>
<td>729.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FCR Between Devices</td>
<td>3</td>
<td>729.8</td>
<td>3.37</td>
<td>.04*</td>
</tr>
<tr>
<td>Between Subjects</td>
<td>7</td>
<td>187.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>21</td>
<td>216.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hemiplegic Subjects: N = 4</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FDP Between Devices</td>
<td>3</td>
<td>7905.4</td>
<td>2.90</td>
<td>.09</td>
</tr>
<tr>
<td>Between Subjects</td>
<td>3</td>
<td>9923.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>9</td>
<td>2722.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FCR Between Devices</td>
<td>3</td>
<td>8603.7</td>
<td>3.07</td>
<td>.08</td>
</tr>
<tr>
<td>Between Subjects</td>
<td>2</td>
<td>1820.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>9</td>
<td>2789.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significant at the .05 level.
As the three other normal subjects reported no subjective differences among devices and had larger hand size, there appeared to be a relationship between the stretch provided by the finger spreader and EMG activity. However, only a low negative correlation was found in FDP ($r = .24$) and in FCR ($r = .37$) between hand size and EMG activity. In addition, if the passive stretch stimuli were responsible for the increased EMG activity in normal subjects, an even stronger effect on the hemiplegic subjects with spasticity should have occurred. This was clearly not the case as the mean score of the hemiplegic subjects on the finger spreader are relatively close to no device (Figure 3).

An alternative explanation is based on the fact that wrist flexion would assist abduction and extension of the fingers as in tenodesis release. If the subject unconsciously flexed the wrist to decrease the stretch on the fingers, high EMG activity could have been produced in the FCR. Yet one question remains: Why did hemiplegic subjects seem to show relatively less EMG activity than normal subjects for the finger spreader?

The lack of significant differences between devices for the hemiplegic subjects during the grasping period would reject the second hypothesis, which predicted that the volar splint, the finger spreader, and the cone would have significantly less EMG activity than no device. In addition, the effects of positioning devices over time (Figure 4) generally show that no device evokes the least amount of EMG activity. Considering this evidence, the belief that the volar splint, the finger spreader, and the cone decrease flexor spasticity (2, 12) needs to be seriously questioned in terms of immediate effects.

In addition to being not significantly different from no device, the volar splint appeared to increase the EMG activity during the grasping period and while it was being put on. One possible explanation for this increased EMG activity was that the spastic wrist and finger flexors were stretched while the volar splint was put on. This stretch stimuli may have caused the Ia afferents of the muscle spindle to fire and to have a facilitatory effect on alpha motor neurons (27). When the flexor muscles contracted and met the resistance of the splint,
there may have been increased Ia afferent firing and increased recruitment of alpha motor neurons (28). A similar mechanism is speculated for the grasping period except that the process is initiated by the irradiation or overflow instead of the stretch stimuli. It might also be speculated that the finger spreader and the cone elicited less EMG activity because they provided less initial stretch or resistance to the flexor muscles especially of the wrist. Another possible explanation is that the contact stimuli of the volar resting splint might elicit the grasp reflex (29) and thus increase the EMG activity. The assumption is made that the volar resting splint provides greater contact stimuli than the firm cone or finger spreader. For whatever reason it would appear that the volar splint is the least desirable positioning device while the hemiplegic subject is doing any activity that requires a comparable effort to squeezing 50 percent MVC of grip.

The third hypothesis, which predicted no significant difference between devices during the post-grasp periods for both normal and hemiplegic subjects, was supported by the results of the two-way ANOVAs. This hypothesis was based on the premise that there is little-to-no EMG activity in normal and spastic hands at rest (22). Thus, it would have been extremely difficult to demonstrate significant differences between devices while the subjects were at rest. In opposition to this view is the report by Zislis of EMG differences on an immediate basis between dorsal, volar, and no splint while the subject was apparently at rest (12). His results, showing the volar splint to have less EMG activity in the flexors compared to no splint, would also contradict the results of this study; however, his EMG findings were based on raw data from only one subject.

The main limitation of this study was the small number of hemiplegic subjects and the variability of their responses to positioning devices and grasping. This variance appeared to be related to level of spasticity. The two hemiplegic subjects with severe spasticity in FDP had much higher EMG scores during the grasping period. Associated with this was an increase in their scores for the volar splint. In contrast, the hemiplegic subjects with moderate spasticity had only slightly increased scores during grasping. Thus, it appeared that subjects with moderate spasticity needed a higher percent of MVC of grip to cause a significant amount of overflow.

This was confirmed by a repeat testing of Subject 1 at 80 percent MVC of grip (not included in data analysis), which caused significantly more EMG activity than 50 percent MVC of grip. Because of the small number of subjects in this study, the results and conclusions of this study must be accepted with caution. In addition, this study does not attempt to assess the long-term effects of positioning devices. Recently a new method of assessing muscle tone was described (30) and appears to have greater potential than EMG to evaluate the long-term effects of positioning devices.

Future studies could have a larger number of subjects, could test over longer periods of time, use a higher percent MVC of grip at least for sub-

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jects with moderate spasticity, and group subjects according to moderate or severe levels of spasticity. Other positioning devices now used in clinical practice such as dorsal splints (11,13), spasticity reduction splints (20), and inflatable splints (31) might also be compared. Although it is clear that grasping can cause significant overflow, it would be of benefit for therapists to know whether other activities of daily living such as cooking, dressing, or transfers cause comparable amounts of overflow in subjects with spasticity. On the assumption that increased overflow is undesirable, some activities of subjects with spasticity might be contraindicated or may have to be done in a way that does not increase the overflow.

**Summary**

This study compared the immediate effects of a volar resting splint, a finger spreader, a firm cone, and no device on the flexor digitorum profundus and flexor carpi radialis of eight normal and four hemiplegic subjects. Flexor muscle activity was measured by EMG while the contralateral hand was squeezing a grasp meter and during the follow­ing relaxation period.

The results showed significantly greater EMG activity for the finger spreader compared to no device in the FCR of normal subjects. This may have been caused by subjects unconsciously flexing their wrist to decrease the stretch on the fingers. For hemiplegic subjects, because the volar splint, the finger spreader, or the cone did not evoke significant overflow, it would be of benefit for therapists to know whether other activities of daily living such as cooking, dressing, or transfers cause comparable amounts of overflow in subjects with spasticity. On the assumption that increased overflow is undesirable, some activities of subjects with spasticity might be contraindicated or may have to be done in a way that does not increase the overflow.

**REFERENCES**