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Effects of a Dynamic Progressive Orthotic Intervention for Chronic Hemiplegia: A Case Series

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Abstract

Study Design: Case series design on a single subgroup of participants

Introduction: Management of hypertonicity and resistance to passive movement (RTPM) in the upper extremity is an intractable problem for persons with stroke and the therapists who provide their care. Therapists have limited options for assessment and treatment of this condition which can profoundly limit functional performance and quality of life.

Purpose of the Study: To evaluate the effect of a 12-week dynamic progressive orthotic intervention in persons with chronic stroke exhibiting wrist flexion contracture who are residents of a long term skilled nursing facility.

Methods: A custom fitted dynamic progressive wrist extension orthotic was applied to 6 residents with chronic stroke. The orthotic was worn for 4 hours daily, 4X/week for 12 weeks. Wrist passive range of motion (PROM) and RTPM were measured weekly and after discontinuation of the orthotics using the Modified Ashworth Scale and the Tardieu Scale of Spasticity. Signal analysis of electromyographical (EMG) flexor response to extensor stretching was also measured before and after orthotic intervention using maximal root mean square (RMS) values and EMG burst onset time.

Results: A moderate effect was found for changes in PROM with the orthotic intervention. Progress made diminished following discontinuation. Moderate effects were also seen in EMG measures which indicated increases in amount of resistive-free movement following intervention.

Conclusions: A dynamic progressive orthotic intervention can be an effective option for increasing wrist PROM and reducing RTPM in persons with chronic stroke.

Level of Evidence: 4

INTRODUCTION

One of the most debilitating aspects of stroke is hemiplegia. Half of all stroke survivors report persistent hemiplegia six months after a stroke¹. Loss of arm function and fine motor skill creates dependency in activities of daily living and may necessitate assistance from caregivers or institutionalization².

Weakness and prolonged immobility of the upper extremity can present additional problems. Muscle tissue may become denervated and flaccid, and secondary metabolic changes can occur concomitantly with paralysis. A decrease in the overall cross sectional area of muscle combined with a shift toward a fast twitch muscle fiber composition is frequently seen in post-stroke muscle; this can limit force output and increase fatigability³. Resting blood flow may be lowered and a loss of lean tissue mass may occur in the hemiplegic limb as well⁴. Overall bone mineral density can decline, further reducing muscle strength⁵. These post-stroke conditions can contribute to decreased range of motion, pain or edema. With pain and edema, inflammatory processes can begin which often progress to shoulder hand syndrome or complex regional pain syndrome⁶.

Post-stroke motor recovery is further complicated when flexor hypertonicity or spasticity is present in the arm. The definition frequently used for spasticity is "a motor disorder characterized by a velocity dependent increase in muscle tone..."⁷. The condition can be observed clinically as a disproportionate response to a quick muscle stretch⁸. This recovery presentation has been associated with greater impairment in activities of daily living (ADLs), poorer rehabilitation prognoses⁹ and increased medical costs¹⁰.

Current management of post-stroke upper extremity spasticity has been largely ineffective. Therapists have traditionally applied thermoplastic orthotics or plaster casts to various joints of the upper extremity to reduce hypertonicity or increase extensibility, but several studies cite inadequate evidence to support or discourage these practices^{11,12,13}. Passive stretching has been used as a means to counteract both biomechanical joint restrictions and spasticity but recent evidence suggests that this may not result in an appreciable increase in joint mobility¹⁴. Oral medications that reduce tone often also cause somnolence, lethargy, or disorientation in stroke patients¹⁵. Botulinum toxin and local neurolytic agents provide only temporary reduction of spasticity and require regular inoculations¹⁶. Intrathecal baclofen is invasive and achieving an appropriate level of tone reduction is often difficult; occasionally, administration can reduce tone so considerably that functional abilities are compromised and skills previously regained in therapy can be lost¹⁷. Therapists therefore, are faced with the quandary of providing spasticity intervention that may not be effective or *not* providing intervention that could potentially improve passive movement and function.

The most practical issue facing rehabilitation therapists is the resistance to passive movement (RTPM) that occurs as a result of spasticity and *quantifying* this resistance in an objective measurable way. Most therapists assess spasticity using scales or measures that describe the resistance felt when passively ranging the affected joint or during performance of a quick stretch of the muscle to elicit a stretch reflex response. Research scientists have used various instruments and methods to quantify spasticity or measure the stretch reflex response including electromyography (EMG)¹⁸, torque output¹⁹, kinematic measures²⁰, robotics²¹ and portable electronic devices²².

Realistically, most clinicians do not have access to laboratory-grade equipment for precision measurement or biomechanical analysis tools for use in clinics. In the clinic, commonly used tests of spasticity include the Modified Ashworth Scale (MAS)²³, which uses numerical scoring from 0-4 to describe muscle tone increases to movement, or the Modified Tardieu

Scale of Spasticity (MTS)²⁴, which defines a range of resistance-free movement. Some authors report lack of reliability and measurement inconsistency associated with using the MAS²⁵⁻²⁶, while others report moderate reliability when used with specific populations²⁷. Despite the controversy, these tests are two of the limited options practitioners have for rapid and feasible in-clinic assessment.

In the past several years, specialized orthotics have been designed as interventions for hypertonicity and spasticity. One device, popular across a variety of treatment settings, is the Dynasplint orthotic (www.dynasplint.com; Severna Park, MD, USA). This device is classified as a dynamic progressive orthotic, used for tissue remodeling and contracture management. The Dynasplint facilitates a low-load prolonged duration stretch reported to reduce contractures²⁸⁻²⁹, improve range of motion (ROM)³⁰⁻³¹ and reduce pain³². A fixed, extended joint position can be set and maintained for several days, facilitating surrounding structures to accommodate and remodel to the longer length. As the tendons and muscle tissue lengthen, the orthotic can be adjusted to larger angles until full excursion of the joint is obtained.

Recent studies have examined the effects of the Dynasplint orthotic on orthopedic-related joint limitations of the ankle³³ and knee³⁴. However, evidence regarding the effects of prolonged stretching for increasing joint ROM after stroke is mixed. Investigators have reported positive gains in passive ROM when the fingers³⁵ and the ankle³⁶ of persons with post stroke hypertonicity are stretched for extended periods, but others report less success¹⁷. One study has shown that using the Dynasplint had a positive effect on maintaining elbow extension gains following botulinum toxin injections in 30 post-stroke individuals³⁷.

The current research suggests that the application of a Dynasplint orthotic can be beneficial in improving recovery following orthopedic injury and in selected neurological lower extremity cases; however, more investigations are needed to determine if devices such as the Dynasplint are effective for the neurologically involved upper extremity. No investigation to date has been performed to examine effects that the Dynasplint orthotic may have on increasing ROM of the neurologically involved wrist. This is critical information for therapists and consumers who are required to use healthcare dollars wisely in the current economic environment.

We chose to examine the effect of 12 weeks of Dynasplint use in persons with chronic stroke who are no longer receiving therapy and are long-term residents in a skilled nursing facility. Motor recovery is less predictable in the chronic population and few viable interventions exist for these individuals³⁸. Although persons with chronic stroke can still make gains months and years after onset, benefits are typically greater during the acute phase of recovery³⁹. Therefore, residents such as these are typically not receiving therapy, often restricted in mobility or function, but most in need of an intervention that may facilitate a positive change in functional status. The purpose of this investigation was to determine if the Dynasplint wrist orthotic could have an effect on improving the passive ROM (PROM) and reducing RTPM of the hemiplegic wrist in this population.

Because clinical presentation varies so extensively in the chronic stroke population, we chose to conduct a case series where we would apply the Dynasplint wrist orthotic over a period of 12 weeks and monitor changes in wrist PROM and RTPM in six clinically-different individuals. We hypothesized that tissue modification and reduction in passive movement limitations could be accomplished irrespective of the time post stroke or extent of deformity.

METHOD

Participants

This study used a pretest/posttest case series design with blinded data collection and analysis. This type of research design is excellently suited for practitioners or clinical investigators in that it enables them to assess and evaluate behavioral changes occurring with a particular intervention in a small number of clinical cases. The principles used allow causation to be inferred as a result of multiple observations over the time course of an application and/or removal of an intervention⁴⁰.

Participants were long-term residents at a 117-bed skilled nursing facility. Inclusion criteria were that residents must have 1) been discharged from and not actively receiving occupational or physical therapy services at the time of the study; 2) sustained a stroke at least one year prior to enrollment in study; 3) demonstrated intact cognitive awareness and ability to follow commands, 4) demonstrated sensory awareness and the ability to verbalize/gesture discomfort; 5) approval from guardian or responsible family member for involvement in the study if appropriate, and 6) medical clearance for participation from his or her attending physician. Residents were excluded from participation in the study if full PROM were present, or, if severe/fixed (ossified) deformities were present with less than 10% of PROM.

Eleven residents were listed by the facility as having a diagnosis of cerebrovascular accident (CVA). All eleven were screened for participation; of those, one resident elected not to participate, one resident had no residual deficits and two residents had profound fixed joint deformities at the wrist and fingers that excluded them from participation. Seven residents were initially enrolled; however, one participant withdrew from the study at the end of the third week. Data are presented on the six remaining residents. Demographics are presented in Table 1.

Clinical Measures

Passive range of motion (PROM) and the Modified Tardieu Scale (MTS)—An occupational therapist with over 30 years of clinical experience, blinded to study objectives, was enlisted to perform the clinical measures. Reliability of measures was calculated; the intraclass coefficient (ICC) established was 0.76, indicative of good reliability, and p-value was <0.001, indicative of differences between subjects⁴¹. Measures were performed weekly, at the same time of day, for all participants. PROM measures of wrist flexion and extension were taken using a standard clinical stainless steel wrist goniometer (Preston Medical, Inc.) and the MTS was also administered at each measurement session. The MTS yields two

angle positions, R1: the angle at which the muscle "catches" during a quick stretch or fast velocity maneuver; and R2: the maximum joint angle obtained during a slow controlled stretch or the maximum PROM angle. The R2-R1 difference is suggested to represent the "dynamic component of spasticity"⁴².

Modified Ashworth Scale (MAS)—The MAS uses a graded 6-level scale with varying descriptors ranging from "0 - no increase in muscle tone" to "4 - affected part(s) rigid in flexion or extension."²³ See Table 2. All participants began the study graded as a "1", indicating a slight increase in muscle tone, including a catch and release or exhibiting minimal resistance at the end of the range of motion when the wrist was moved into extension. The Ashworth Scale is commonly used by therapists as a clinically meaningful tool for measurement of resistance to passive motion (RTPM).

Surface electromyography (sEMG)—In order to produce a constant velocity of movement while measuring resistance to passive stretch, a commercial continuous passive motion (CPM) device was used (Kinetec Model 8080, USA; www.orthoexpress.com). Areas on the dorsum and ventral aspects of the forearm were prepped with alcohol wipes. A preamplified sEMG sensor housing two silver (Ag) 1mm × 1cm electrodes spaced 1cm apart (Delsys, Inc. Boston, MA; DE-2.1 surface EMG sensor) was placed on the dorsal aspect of the forearm over the extensor carpi ulnaris and on the ventral aspect over the flexor carpi radialis muscles. Both were secured with double-sided adhesive interfaces. A 2.5cm diameter round surface electrode was used as a ground electrode placed on the over the olecranon. Participants were seated in their wheelchair with the armrest of the affected upper extremity and both footrests removed to allow the feet to rest comfortably on the floor. The CPM device was placed on a height-adjusted table such that the resident was sitting erect with affected elbow, hips, knees, and ankles in a 90° neutral position. The CPM was then height adjusted so that the 90° position of the elbow could be maintained during passive motion. The affected forearm was placed in the forearm trough of the CPM machine and held in place with two Velcro straps located slightly proximal to the ulnar styloid and at midshaft of the forearm. The hand was positioned over a cylindrical metal rod with a soft foam overlay that rested slightly below the metacarpal heads on the palmar surface of the hand; the rod supported the weight of the hand and was adjusted so that the wrist was in a pronated, neutral (0°) position with digits relaxed over the cylinder. After being positioned in the apparatus, the participant was asked to sit quietly for approximately 3-4 minutes to reduce any spasticity that emerged during the positioning process and to relax muscles prior to movement. The CPM device then moved the wrist at a constant speed of 6.66°/sec into 30° of extension, followed by 30° of flexion back to neutral, continuing flexion to 60°, then back upward past neutral to 30° of extension and flexing 30° back to the starting neutral position at 0°. Total wrist excursion was 240°. See Figure 1. sEMG signals from extensor and flexor muscles were collected with the Delsys Myomonitor IV wireless sEMG system (Delsys, Inc., Boston, MA, USA). Both channels were amplified, high-pass filtered at 20 Hz and digitally converted. The sEMG signal was sampled at 2000 Hz using the Delsys EMGWorks Acquisition software program and recorded on a PC for later analysis.

The time period of interest chosen for sEMG analysis was the phase of movement from the 60° flexed position of the wrist to the 30° extended position ("extension phase"), as noted in Figure 2. EMG in the flexor muscle was recorded throughout this time period and the maximal sEMG signal or "burst" that occurred was measured. Time of burst onset and power, as noted by root mean square (RMS) of the sEMG burst, was measured. The sEMG burst indicates the point at which a maximal resistance of the flexor muscle occurred in response to the extensor stretch.

Dynasplint Intervention

Residents were custom-fit with dynamic progressive orthotics manufactured by Dynasplint worn on the forearm of the affected upper extremity that stretched the wrist into extension. The Dynasplint representative adjusted the orthotic at the appropriate level of tension following the initial fitting and intermittently throughout the wearing period to obtain the maximal comfortable extended wrist position. Participants were encouraged to maintain their normal daily activities as much as possible while wearing the Dynasplint, therefore most of the participants were in wheelchairs attending facility activities and engaging in routine tasks during the time period when the Dynasplint was applied and worn. In order to ensure compliance with wearing the orthotic 4X/week, the investigators personally donned and doffed the orthotics with each participant on each day of wear. During this procedure participants were encouraged to relax the affected upper extremity as much as possible. The investigator positioned the limb into extension at their side, removing the wheelchair armrest as needed to allow gravity to assist. As the limb relaxed into extension, wrists and fingers were gently stretched and placed in the orthotic. Straps were secured and the limb was repositioned comfortably in the resident's lap or on a table if the resident were participating in an activity.

The first week of the study was used for baseline measures and intervention did not take place. The next week was an acclimation week where the orthotic was worn for progressively longer periods until the resident could tolerate wear for 4 hours. All residents tolerated a 4-hour on time by the end of the first week. For the next 12 weeks, residents wore the orthotic 4 hours daily, 4X/week. PROM, MTS and MAS measures were taken weekly by the blinded data collector; the CPM sEMG test was given again at the end of the 12 week intervention period. At the end of the 12-week intervention period, the orthotics were discontinued. Initial post-treatment measures were taken 2 days after removal of the orthotic. Retention of any changes that occurred with the intervention was measured at 2, 4 and 6 weeks following discontinuation of the orthotics. This was done to determine sustainability of any gains made. Measures of PROM along with the MTS and MAS tests were performed at these time points .

Data Analysis

The mean and standard error were calculated for each of the four measures (PROM values, TSS score, MAS rating, sEMG burst RMS value and sEMG burst onset time). The percent change from baseline on each measure was calculated for pre-post values and retention values.

All sEMG analyses were performed by a blinded investigator. sEMG signals were full-wave rectified. The sEMG burst was defined as the 2s segment during the extension phase (18.0 - 32.0s) with the greatest sEMG activation, indicating the greatest resistance to the passive movement. To measure sEMG during the burst, the root mean square (RMS) sEMG of the rectified signal was calculated with a moving window (window length 0.125 s, window overlap 0.0625 s). To smooth the signal, a moving average (2 s window, window overlap 0.25 s) of the sEMG RMS was calculated to determine the 2 s segment during which the greatest EMG power occurred (the sEMG "burst"). sEMG RMS during the burst and time of burst onset were measured. sEMG burst onset time was noted as the time at which the peak activation occurred during the sEMG burst in the extensor phase. To compare across experimental sessions, RMS sEMG values were expressed as a percentage of baseline sEMG⁴³.

Effect sizes were calculated using Cohen's *d* equation for correlated designs suggested by Dunlap et al.⁴⁴ where Cohen's $d = [(\text{Mean}_{(\text{post})} - \text{Mean}_{(\text{pre})}) / \text{SD}_{(\text{pooled})}]$. Pre-intervention values were compared to post-intervention values and where applicable, pre-intervention and post-intervention values were also compared to retention values. For clinical research, an effect size of 0.2 is considered small, 0.5, a medium or moderate effect, and 0.8, a large effect⁴¹.

RESULTS

PROM

Five of the six participants increased PROM measures from week 1 to week 12. Average range gained in passive wrist extension across all participants was 15.66 ± 8.71 degrees. Weekly changes in PROM are graphed in Figure 2. Visual inspection indicates an increasing trend pattern across the 12-week period for all participants except S1. This participant became increasingly ill during the last three to four weeks of the intervention. She was subsequently hospitalized for a week after the intervention was completed; therefore her values for this measure and others show a higher variability. For S1, a stable baseline and acclimation week was present followed by an initial increase during the early intervention period, then a downward trend back to original PROM measures recorded at baseline (change pre-post, -17%). The remaining participants (S2, S3, S4, S5, S6) showed a stability in PROM during the baseline and acclimation weeks, followed by increases during the intervention period (change pre-post, 37%, 25%, 5%, 23%, and 6% respectively). Participants S1 and S6 showed a strong improvement from baseline and acclimation weeks to the first week of intervention as evidenced by the sharp discontinuity in level between these two points. Overall, results indicate an alignment of data trending toward increases in PROM outcomes for 5 of the 6 participants.

All participants lost or showed no gains in PROM during week 2, 4, and 6 after the 12-week intervention when orthotic wearing was discontinued (retention phase). Average change in passive wrist extension from discontinuation until the end of the retention phase was -9.50 ± 4.92 degrees. Values recorded during this phase indicated that two participants showed no change in PROM following discontinuation of the orthotics (S1 and S2) and the other four lost PROM in wrist extension (S3, -11%; S4, -11%; S5, -3%; S6, -26%). Retention

changes in PROM are also graphed for all participants in Figure 3 and summarized in Table 3.

Modified Tardieu Scale

MTS measures collected over the 12-week intervention for each participant are shown as scatter plots in Figure 3. Points shown are the differences calculated between the R2 and R1 angles. These values ranged from 17.50° to 36.83° (average difference, $27.81 \pm 6.85^\circ$). All participants except S1 trended downward throughout the intervention period. This suggests that the area between the maximum PROM angle (R2) and the initial "catch" during a quick stretch maneuver (R1) - was smaller after intervention. This indicates that the point at which the catch occurred was later during the movement, i.e., the available range of free passive movement without resistance - increased following intervention. During the retention phase, S1 and S6 showed stability or a slight decrease in MTS scores; the remainder of subjects' MTS scores trended upward, suggesting development of less available free passive movement and a return to baseline values.

Modified Ashworth Score

Very little change was noted in the MAS of the six subjects across the 12-week intervention period. Participants S2, S3, S4, S5, and S6 were scored as a 1 ("slight increase in muscle tone manifested by a catch and release or by minimal resistance at the end of the range of motion" per MAS guidelines) at week 1 and week 12, suggesting no notable change in this measure across the intervention. Participant S1, who became ill during the intervention, showed an increase in the MAS score. This indicates deterioration on this measure from week 1 when she scored a 1, and week 12 when she scored a 2 (more marked increase in muscle tone through most of the range of motion" per MAS guidelines). During the retention phase, S1 returned back to a score of 1; all other participants' MAS scores showed no change from week 12 through retention week 6.

sEMG RMS and sEMG burst onset time

Three participants, S2, S4, and S6, showed a decrease in EMG RMS of the maximal burst during the extension phase when pre-intervention measures were compared to post-intervention measures (pre-post change, 21%, 53%, and 54% respectively). Because RMS reflects the power or energy contained within the sEMG signal, a reduction would suggest less power during the burst, indicative of less resistance of the flexors present during the extension movement. S5 showed a minor increase in these same values (2%) while S1 and S3 showed an increase in sEMG RMS during the burst (41% and 31% respectively).

sEMG burst onset time occurred later in three participants (S1, S4, S6) from pre to post-intervention measures (S1, 7%; S4, 28%; and S6, 17%). A later time point for burst onset from pre to post-intervention measures suggests that the time period prior to the onset of the burst was longer in duration, indicating an increase of the range of resistive-free movement. The remaining three participants showed a minimal non-significant increase (S5, increase of 0.03%) or minimal decreases (S2, -2%; S3, -7%). See Figure 4.

Effect sizes (Cohen's *d*) were calculated for each measure and can be found in Table 4. Medium effects were found for the pre-post and post-retention PROM comparisons. Similarly, sEMG burst onset times showed a medium effect. Smaller effects were seen in the pre-retention PROM comparison and sEMG RMS.

DISCUSSION AND CLINICAL IMPLICATIONS

The objective of this case series study was to determine if the Dynasplint wrist orthotic could have an effect in increasing PROM and reducing RTPM in six persons with chronic stroke living in a skilled nursing setting. PROM of the wrist increased in 5 of the 6 participants; these gains were lost or only minimally maintained after the Dynasplint was discontinued. Following the retention phase of the study, residents were given the option to continue use of the Dynasplints if they so desired. Effect size results suggest that the residents involved showed moderate PROM changes with the intervention and additionally suggest that the changes were lost when the intervention was discontinued. MTS measures showed a downward trend when raw data were analyzed, suggesting larger ranges of resistance-free movement but non-significant effect sizes were obtained on this measure. Similarly, MAS scores used with these individuals remained constant despite the intervention. We propose that the MAS may not be sensitive enough to detect smaller increments of change in this population. Changes in sEMG burst onset time showed a moderate effect, suggesting that the range of free movement without resistance increased during the intervention. sEMG changes in RMS were variable among the six subjects and showed little appreciable effect.

The Dynasplint orthotic for the hemiplegic wrist can be an effective intervention for improving PROM and expanding the range of free movement in persons with chronic stroke. Interestingly, S2, S3 and S5 achieved the greatest gains in PROM, yet these residents exhibited the least pliable joint movement at the start of the intervention. This suggests that the Dynasplint wrist orthotic may be most effective in persons with unyielding joint stiffness, severe contracture, or high levels of spasticity. In contrast, S4 and S6 who had more extensibility at the wrist joint achieved lesser gains.

Additionally, residents of this long-term nursing facility who were no longer receiving therapy services and who exhibited long-standing shortening of the wrist flexors were still able to make gains in passive range and decrease resistance to extensor stretch. This could be extremely helpful information for therapists practicing in these settings. Consistent use of the Dynasplint orthotic has the potential to facilitate changes in recovery status so that therapists can recommend new or continued therapy services. The Dynasplint orthotic could also be used with residents who are currently participating in therapy for additional remodeling between sessions.

The success of using orthotics such as the Dynasplint in the long-term skilled nursing setting often depends on compliance and utilization of the device according to manufacturer or designer standards. Our team maximized compliance by investigators personally donning and doffing the orthotic according to our study protocol. Dynasplint representatives also

coordinated each case and worked with the residents to insure proper fitting and adequate progression of stretch.

Unfortunately RTPM and spasticity remain an intractable problem for persons with stroke and the therapists who provide care for them. Although much evidence suggests that spasticity and pathological flexor patterning in the upper extremity following stroke is a result of *cortical* disinhibition⁴⁵, the clinical tools we have currently that are practical and available for use in practice only address the problem *peripherally*. Devices such as the Dynasplint can offer a viable option in peripheral management of joint restriction due to tonal abnormalities. Most importantly, reducing tonal or ROM impairment can be the foundation for improving function and performance of daily activities. In addition, until more reliable measures than the MAS or the MTS emerge that are feasible to use in clinics, therapists will continue to use these measures as the best available tools to assess and qualify spasticity.

New therapies for spasticity are, however, on the horizon. Transcranial magnetic stimulation (TMS) is now being used as an intervention to address spasticity from the cortical vantage point. Recent evidence is suggesting that TMS may facilitate reductions in tonal abnormalities after stroke⁴⁶⁻⁴⁷. Research and clinical practice continue to be incredibly divergent on the measurement, quantification, and treatment of tonal abnormalities related to stroke, but innovative treatments and development of in-clinic devices with ease of use may eventually close this gap.

LIMITATIONS

Participants' physical and mental state throughout the 12 week intervention period often oscillated which may have had an effect on spasticity and RTPM. Tardieu measures quantifying spasticity reflected this variability. S1 had medical issues toward the end of the intervention and therefore her outcomes appear to have been impacted by her declining physical status. Spasticity has been reported to fluctuate with changing physiological or emotional status⁴⁸ which could have also contributed to the variability of the data.

The participating residents wore the orthotics for a maximum of 4 hours, 4X/week. Using this time and schedule we were able to achieve consistency across all participants. Dynasplint recommends a 6-8 hour wearing schedule daily; therefore our results may have been more robust if longer time periods on more days were incorporated. Combining use of the orthotics with other adjuvant methods such as neuromuscular electrical stimulation, biofeedback or functional task performance could also have optimized results. Perhaps most importantly, the orthotic is designed to address motor/biomechanical impairments only; functional treatment planning should be incorporated into any therapy program to address translating improved movement patterns or increases in ROM into successful task performance.

Finally, the residents involved were few and the study was designed as a clinical case series model. Larger more comprehensive projects or randomized control trials investigating effectiveness may confirm or refute these findings.

CONCLUSION

Resistance to passive movement or spasticity in the upper limb following stroke can lead to soft tissue tightness, fixed contracture, disuse and joint deformity. These conditions can be further magnified in the chronic stroke patient who is institutionalized or immobile. Clinicians are challenged by having few reliable tests to measure RTPM and spasticity. Devices such as the Dynasplint orthotic can have a beneficial effect on increasing PROM and decreasing RTPM in the hemiplegic wrist in persons with chronic stroke, but larger trials that can produce convincing evidence are needed so that healthcare dollars spent on the equipment are used judiciously. However, this was the first investigation to explore the benefits that can be gained with the use of the Dynasplint orthotic for improving ROM and joint tightness in the chronic hemiplegic wrist, and these outcomes can be used as preliminary work to support further study. More investigations are needed from clinical researchers to empirically test the effectiveness of this device across diverse patient populations with various neurological pathologies.

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CPM Wrist Movement

Figure 1.

Excursion of wrist flexion and extension while using CPM. Gray arrow indicates the primary area of interest (during movement from 60° of wrist flexion through 30° of wrist extension) for EMG measures of flexor resistance.

Figure 2.

Weekly changes in wrist extension PROM throughout 12-week study and 6 week retention period. B=baseline week; no intervention provided. A=acclimation week; time in splint was gradually extended until resident tolerated 4 continuous hours of wear. R=retention weeks. Measures were taken at week 2, 4, and 6 following 12-week intervention/discontinuation of wear.

Figure 3.

Changes in Modified Tardieu Scale (MTS) throughout intervention period for the six participants. Points indicate the R2-R1 angle measure. R2 represents the maximum angle possible during slow passive range of motion of the joint. R1 represents the angle at which the first resistance to passive movement or "catch" is felt during a quick stretch maneuver. All participants decreased this value (see trendlines) across the intervention period except for S1. Decreases are indicative of a closer approximation of maximum angle at PROM and first catch during a quick stretch, suggesting a smaller area of hypertonicity, spasticity or resistance to passive movement than observed pre-intervention.

EMG Burst Onset

Figure 4.

Pre and post-intervention measures for time of EMG burst onset during extension phase of CPM movement. Participants S1, S4, and S6 showed a later burst onset post-intervention indicating a larger range of free movement when compared to pre-intervention.

Table 1

Demographics of participants.

Participant	Gender	Age	Type	TPS*	Affected UE	MAS
1	F	59	Ischemic	11	L	1/5
2	F	54	Hemorrhagic	3	R	1/5
3	M	71	Ischemic	2	L	1/5
4	F	66	Ischemic	3	R	1/5
5	M	64	Ischemic	7	L	1/5
6	F	53	Hemorrhagic	8	R	1/5

* Time post stroke (years)

Table 2

Modified Ashworth Scale rankings.

Score	Description
0	No increase in tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of ROM, but affected part(s) easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension

Table 3

Changes in wrist PROM during study and subsequent retention period. (–) indicates a negative trend or loss, (+) a positive trend or gain. NC = no change.

Participant	Change in Wrist PROM wks.1-12	Trend	Change in Wrist PROM wks.2-6 after 12-wk intervention (retention)	Trend
1	– 17	–	0	NC
2	43	+	0	NC
3	30	+	– 12	–
4	7	+	– 15	–
5	23	+	– 30	–
6	8	+	0	NC
Avg	15.66		– 9.50	
SE	8.71		4.92	

Table 4

Effect size estimations.

Test	Effect size Cohen's <i>d</i>
PROM Pre-Post	0.67
PROM Pre-Retention	0.25
PROM Post-Retention	0.60
Tardieu Pre-Post	0.03
Tardieu Pre-Retention	0.13
Tardieu Post-Retention	0.06
EMG RMS	0.18
EMG burst onset	0.62