Research Report

Overcoming Limitations in Elbow Movement in the Presence of Antagonist Hyperactivity

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Background and Purpose. *A traditional perspective on rehabilitation of patients with abnormal muscular hyperactivity presumes that relaxation should be facilitated prior to recruitment of antagonists, if effective movement about a joint is to occur. The purpose of the study was to determine the effect of training weak triceps brachii muscles, with hyperactivity present in the opposing biceps brachii muscles, on elbow function in individuals at least 1 year poststroke.* ***Subjects.*** *Sixteen patients with chronic stroke were randomly assigned to receive electromyographic biofeedback to retrain the triceps muscle (n=8) or to receive conventional movement training (n=8).* ***Methods.*** *Both groups participated in 5 baseline and 10 training sessions involving tasks requiring elbow extension. Preintervention and postintervention measurements included elbow extension range of motion, triceps and biceps muscle electromyographic activity during performance of elbow extension, resisted elbow extension, and a reaching task.* ***Results.*** *Two-sample t-test results of between-group comparisons for each variable were not significant. One-sample t-test results of within-group comparisons showed significant increases in triceps muscle mean electromyographic activity during two of the three tasks for the feedback group, but not for the nonfeedback group. Passive and active range of motion in both groups increased significantly, although biceps muscle co-contraction persisted.* ***Conclusion and Discussion.*** *These results suggest that functional improvements at the elbow may have been due to biomechanical (peripheral) rather than neuromuscular (central) changes about the joint. Furthermore, these preliminary data indicate that patients with stroke may be trained to increase movement without first being trained to specifically inhibit hyperactivity in muscles. [Wolf SL, Catlin PA, Blanton S, et al. Overcoming limitations in elbow movement in the presence of antagonist hyperactivity. Phys Ther. 1994;74:826–835.]*

Key Words: *Biofeedback, Cerebrovascular disorders, Elbow, Electromyography.*

Approximately 5% of people older than 65 years will sustain a stroke, making cerebrovascular accident (CVA) a leading cause of disability, as well as a major health and economic problem.[1,2] One factor traditionally thought to limit recovery of function after CVA is hypertonicity, muscular hyperactivity resulting from an exaggerated, velocity-dependent stretch reflex.[3,4] In the presence of hypertonicity, the normal agonist/antagonist relationship is altered, possibly due to abnormal patterns of activity in opposing muscle groups crossing a joint.[3,5,6] Hypertonicity of an antagonist muscle group, however, has been shown to be unrelated to decreased isometric strength of the agonist muscles.[7] Therefore, inadequate recruitment of the agonist, not the increased activity of the antagonist, may be the primary limitation to movement poststroke.[8]

Abnormal posturing over a period of time may affect the viscoelastic properties of joints or the connective tissue components of soft tissue. These changes may contribute to dysfunctional movements. Alterations in body or limb posturing may be associated with rigidity (abnormally increased non–velocity-dependent muscle activity), rather than hypertonicity.[3,9] After prolonged static length changes imposed by chronic muscle hyperactivity, shortening of muscles occurs,

Table 1. Summary of Subject Characteristics

	Age (y)	Gender	Time Since Stroke (mo)	Side of Hemiplegia	Site of Lesion
Feedback group (n=8)	74	Male	26	Right	Unknown
	57	Male	12	Left	Frontal
	75	Male	48	Left	Lacunar
	41	Female	36	Right	Parietal
	67	Male	36	Left	Internal capsule
	66	Male	27	Right	Frontal
	68	Male	15	Left	Parietal, frontal, temporal
	63	Female	61	Left	Frontoparietal, frontotemporal
	$\bar{X}$=63.88±10.88		$\bar{X}$=32.63±16.37		
Nonfeedback group (n=8)	79	Male	24	Left	Unknown
	50	Female	60	Right	Unknown
	77	Male	36	Left	Unknown
	41	Female	49	Left	Unknown
	61	Male	136	Right	Unknown
	77	Female	29	Right	Parietotemporal
	51	Male	93	Right	Brain stem
	60	Male	97	Right	Unknown
	$\bar{X}$=62.00±14.39		$\bar{X}$=65.50±39.54		

In this study, the following dependent variables were used to assess the effectiveness of two treatment strategies: elbow extension AROM and PROM, speed of movement for functional task completion, mean EMG activity of the triceps muscle during isotonic contraction, and mean EMG activity of the biceps muscle during isotonic contraction of the triceps muscle.

Method

Design and Sample

A pretest-posttest, two-group design was used in this study. The independent variable was the strategy of facilitation of the triceps muscle, either with or without EMG biofeedback training. Sixteen subjects, all of whom gave informed consent to participate, were randomly assigned to either a feedback group or a nonfeedback group with matching for Brunnström stage[25] (Tab. 1). We did not evaluate the reliability of classifying patients according to the Brunnström staging system. Each subject met the following selection criteria: diagnosis of hemiplegia secondary to CVA, with time since onset of 1 year or longer; the involved upper extremity in Brunnström stage 3 or 4; longer than 6 months since the subject's most recent session of physical therapy or occupational therapy for the upper extremity; absence of receptive aphasia, visual field deficits, and proprioceptive deficits in the involved elbow; and ability to obtain at least 60 degrees of passive shoulder flexion and

*Model 220, Gould Inc, Instruments Division, Cleveland, OH 44114.

†Physical Health Devices Inc, 1500 W Cypress Creek Rd, Ft Lauderdale, FL 33309.

physicians gave consent for participation and, when relevant, agreed not to alter their patients' antispasmodic medications. One subject (in the feedback group) out of the total sample was taking antispasmodic medications. All subjects underwent 5 baseline sessions for measurement of the dependent variables, followed by either 10 feedback or 10 nonfeedback sessions. Within 3 days after completion of training, a postintervention session was conducted to remeasure the dependent variables.

Measurements and Instrumentation

Measurements of elbow extension AROM and PROM were performed according to a previously validated clinical protocol.[26] Speed of functional task completion and mean EMG activity of the triceps and biceps muscles during isotonic contraction of the triceps muscle were measured during performance of three functional tasks in the following order of increasing difficulty: gravity-eliminated elbow extension (extension task); gravity-eliminated elbow extension with a 0.68-kg (1.5-lb) weight (resisted extension task); and a reaching task into elbow extension with the 0.68-kg weight, which required shoulder joint stabilization and involved knocking a foam ball off a podium positioned 229 cm (9 in) above table surface (reaching task). Speed of movement was measured in degrees per second, using an electrogoniometer, with output displayed on a Gould pen recorder.* The arms of the electrogoniometer were aligned with the acromion and the midpoint between the radial and ulnar styloid processes. Change in degrees of elbow movement corresponded to change (in millimeters) of amplitude of the recording pen.

Electromyographic activity of the biceps and triceps muscles was assessed through analog to digital conversion of the rectified EMG signal, sampled every 7.8 milliseconds, using the Bioscope 3000.† The EMG signal was band-pass filtered (100–540 Hz, loss=12 dB/octive, 60 notches=25

No effort was made to normalize EMG values or to create ratios in which EMG data obtained from muscles within a hemiplegic limb would be compared with homologous contralateral muscles. The concerns expressed recently by Gowland and colleagues[6(pp627–628)] regarding the validity of procedures that quantify muscle activity in hemiplegic limbs, are in agreement with previous work,[14] but we believe their approach of comparing quantitative differences in homologous muscles does not appear more valid than its predecessors.[19,28,29] Furthermore, our own past efforts[17,24] at normalizing EMG data from hyperactive muscles in patients with neurologic disorders produced so much variance, with standard deviations often exceeding normalized values, that we seriously question the value of applying this procedure.

Interrater and intrarater reliability of measurements taken during the initial evaluation and subsequent measurement sessions were established and maintained throughout data collection at 100% agreement between researchers. Range of motion measurements were taken until three measurements within 3 degrees of each other were attained. The use of a template ensured reliability of electrode placement for EMG measurements. The electrogoniometer used for measurement of speed was calibrated before each subject's measurement session. The EMG unit was self-calibrating. Prior to engaging the unit, a standard input voltage was provided against which EMG voltages were calibrated.

Procedure

Baseline. Subjects who met the selection criteria proceeded to the first of five baseline measurement sessions. All subjects were allowed to practice three repetitions of each of the three elbow extension tasks, beginning with the least difficult (elbow extension) and progressing to the most difficult (reaching task).

Measurement of the dependent variables occurred in random order for all subjects during each baseline session and during a separate session following the last treatment. The mean of three values was recorded for each range of motion measure. Passive range of motion of the involved elbow was measured with the subject seated and with the shoulder positioned in 0 degrees of flexion and abduction. Active range of motion of the involved elbow was measured with the subject seated and with the involved shoulder abducted and flexed to approximately 60 degrees and the elbow flexed to 90 degrees.

For acquired EMG and speed data, three measurements were taken for each task, for a total of nine measurements per variable during each session. The mean value was calculated for each task. The subjects were seated during these measurements, with the involved shoulder abducted and flexed to approximately 60 degrees and the elbow flexed to 80 degrees. For speed measurements, the arms of the electrogoniometer were affixed to each subject's elbow, and the subject was then instructed to move as fast as possible for each task.

Electromyographic readings of the triceps and biceps muscles during the functional tasks were taken separately from speed measurements to minimize the possibility that an obtrusive electrogoniometer setup might impair natural limb movement when strapped to subjects simultaneously with EMG recording equipment. Once the electrodes were in place, the measurement protocol was initiated on the Bioprompt program.[†] The subjects were instructed to perform three repetitions of each task. The experimenter cued the subject to initiate each repetition "as fast as you can" while simultaneously prompting the computer to begin measurement of the triceps and biceps muscle activity. The contraction phase ended when the subject reached the end range of active elbow extension or the target (ball). This contraction phase was the interval across which the EMG activity was averaged at a rate of 128 samples per second. Speed of movement within subjects was consistent; therefore, intrasubject EMG measurements were probably not affected by variations in movement for each task. Intersubject movement speed and thus EMG recordings, however, were variable. Subjects rested 1 minute following a series of three repetitions of each task.

Treatment. Two to three days following the completion of the fifth baseline session, each subject began the first of 10 training sessions in either the feedback or nonfeedback group. Each training session for both groups lasted 25 minutes, and included three 1-minute rest periods. Subjects were seated with the involved upper extremity relaxed on a powder board, in a similar position as during baseline sessions.

In the feedback group, the subjects began uptraining of the triceps muscle during the elbow extension task, using one EMG channel of the Bioprompt program. Subjects were encouraged to increase the output of the triceps muscle based on the visual and auditory feedback provided by the computer.

Within the context of each of the tasks of elbow extension, the subjects worked to increase the range of elbow extension and EMG output of the triceps muscle. During the first session, all subjects trained only the elbow extension task for the full session. Subjects progressed in subsequent sessions to tasks of greater difficulty based on ability to complete the task. Regardless of ability, all subjects advanced to the task of next highest difficulty after three sessions.

The nonfeedback group's training sessions mirrored the feedback condition for both task performance and progression, except that EMG biofeedback was not used. To mimic the feedback condition, electrodes were secured to the triceps muscle, but the monitor was turned off and the program was not engaged.

All baseline and training sessions for each subject were scheduled over a 6-week period at approximately the same time of day. All subjects partici-

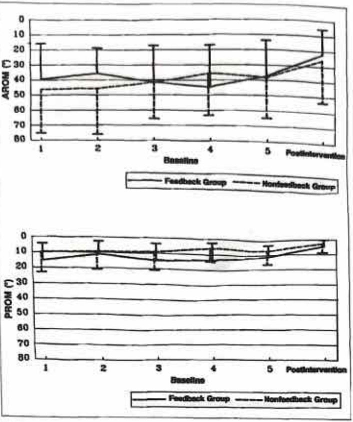

Figure. *Mean active range of motion (AROM) and passive range of motion (PROM) (in number of degrees lacking full elbow extension) across baseline and postintervention sessions for the feedback and nonfeedback groups. Error bars represent 1 standard deviation.*

vention sessions are shown in Table 2. Mean EMG activity for the triceps muscle during the elbow extension task and the resisted extension task improved significantly within the feedback group (Tab. 4). The remaining variables—mean biceps muscle EMG activity for all tasks and mean triceps muscle EMG activity for the reaching task—did not yield statistically significant changes. Significant correlations for this group were an increase in mean biceps muscle EMG activity with mean triceps muscle EMG activity during the extension task and gains in PROM (approaching 0° of extension) with increases in triceps muscle EMG activity for the resisted extension task (Tab. 5).

Nonfeedback group. Mean baseline to postintervention comparisons resulted in significant improvement in AROM and PROM (Tab. 4). Range of motion data are displayed in the Figure. Mean triceps muscle EMG activity increased for the extension and resisted extension tasks (Tab. 3); however, these changes were not significant (Tab. 4). No other significant differences were found for the remaining variables. Significant correlations seen in this group were between mean biceps muscle EMG activity, which increased with mean triceps muscle EMG activity for the task of resisted extension; increased PROM with increases in mean triceps muscle EMG activity for the reaching task; and improved AROM with increases in mean biceps muscle EMG activity for the resisted extension task (Tab. 5).

To ensure that the variability during baseline measurements within each group was not greater than the change occurring after intervention, comparisons were made between the magnitude of the differences in the fifth and first baseline measurements and the magnitude of the differences from postintervention to fifth baseline measurements. Only three significant differences were found out of 16 comparisons. The feedback group's mean triceps muscle EMG activity for the extension task and the nonfeedback group's mean biceps muscle EMG activity for the extension and the resisted extension tasks increased significantly, as expected, after intervention.

Discussion

The possibility of overcoming elbow extension restrictions in the presence of hypertonus through agonist muscle activation was examined. By exploring the original question concerning the prospects of increasing triceps muscle activity regardless of biceps muscle hypertonicity, the results provide a framework for analyzing changes in certain neuromuscular variables.

Between-Group Comparisons

Lack of any significant differences in the between-group comparisons of the change over baseline measurements confirmed the homogeneity of the two groups prior to intervention. Improvements may therefore be more confidently attributed to the interventions and not to group differences present before intervention. Comparing the changes that occurred from baseline to postintervention revealed that feedback and nonfeedback training were both effective in improving elbow extension, but did not produce significantly different results.

required active shoulder elevation as well as elbow extension, there was convergence of the biceps and triceps muscle EMG values. The triceps muscle EMG values comparatively decreased, and the biceps muscle values increased, indicating more co-contraction during the reaching task. In each situation, responses were consistent within individuals or repeated efforts. Electromyographic variability occurred in the magnitude of responses across individuals. Co-contraction in this instance refers to the presence of simultaneous activity in opposing muscles and does not depend on latency or timing (not measured in this study). Consequently, these measurements examine the overall activity, not the sequencing of activity.

The effect of co-contraction on movement is controversial. Under normal circumstances, co-contraction provides the joint with greater stability, especially when distal limb segments are free to move.[35] After stroke, however, co-contraction about the elbow is often abnormal with biceps muscle contraction while patients attempt elbow extension.[19] A probable mechanism for the dysfunction is the delayed ability to cease muscle activity during reciprocal movements, due to impaired reciprocal inhibition.[6]

Studies that have attempted to downtrain biceps muscles and uptrain triceps muscles simultaneously[5,6,15,24] have demonstrated that co-contraction persists even after training. More recently, inadequate recruitment of the agonist, not the presence of abnormal co-contraction, has been shown to be the limiting factor for completion of a reaching task.[8]

An increase in co-contraction may be due to the proximal stabilization that must be attained to extend the elbow toward a target in space. This finding supports previous work using primates that showed the presence of co-contraction versus reciprocal activation for proximal stability during distal movement.[36]

Mean biceps muscle EMG activity during elbow extension remained relatively static in both groups; however, correlations revealed that mean biceps muscle EMG activity increased with mean triceps muscle EMG activity during the first two tasks (Tab. 5). These correlations indicate that abnormal co-contraction may have increased with training. Increases in biceps muscle EMG activity also correlated with improvements in AROM for the nonfeedback group. This increase in range of motion without a reduction in the hypertonicity of the biceps muscle further delineates the components contributing to movement restrictions. Changes may have occurred peripherally in the viscoelastic properties of the joint rather than in central control over disinhibited biceps muscle motoneurons. Similar findings were seen by Thilman and co-workers[37] in subjects poststroke.

Limitations

The study had several limitations, including the limited generalization of the findings to other joints and to other patient populations with neurological problems. Homogeneity between groups with regard to diagnosis was impossible to confirm because information available regarding lesion sites was nonspecific (Tab. 1). The power of the *t* tests was low, only .30 based on a probability value of ≤.05, due to the small sample size and high variability of the measurements. Because the power was so low, further studies are needed on larger samples to determine whether biofeedback is a viable method for increasing triceps muscle output in individuals with chronic stroke. In addition, five baseline measurement sessions may not have been adequate to allow subjects' improvement to plateau, usually a necessary prerequisite before initiation of the experimental protocol. One postintervention measurement session may not be adequate to reflect improvements that may have occurred throughout the treatment. Explanations regarding correlations should be guarded because the finding of significant correlations across tasks within each group was inconsistent.

Given the sample size and intersubject variability in elbow movement speed when subjects were instructed to "move as fast as they can," the important relationship of movement speed to changes in EMG activity over time or within trials for each group cannot be interpreted. Meaningful correlations will require a far more sophisticated data acquisition and analysis system than that used in this study. Only then can movement speeds truly be related to EMG activity as a function of the specific training paradigm.

Future Directions

In future studies, researchers may consider comparing the performance of patients with acute versus chronic stroke to better delineate the roles of hypertonicity and viscoelastic properties in the development of movement restrictions. Additional alternatives for future observations should include the study of other single joints or functional combination of joints. Measurement of joint stiffness, which refers to the amount of torque required to passively move a joint at a specific angle, would help to further delineate neuromuscular versus biomechanical changes associated with hypertonicity. This measurement would also procure information relevant to the specific origins of the central components associated with the phenomena of hyperactivity, specifically, which aspect of the reflex is altered and the resulting contributions to limitations of movement.[38-40]

Clinical Implications

Clinically, the results of this study indicate that functional training may facilitate improvements in AROM and PROM; however, decreases in hyperactivity may not accompany this increase in movement. Consequently, treatment efforts to increase arm function may be more effective when focusing on improving motoneuron recruitment rather than reducing activity in antagonists.[8] This approach

restricting the range and fluidity of joint movements.[9] Biomechanical changes around the joint may follow, including adhesions of tissues, decreased tendon and soft tissue extensibility, and decreased sarcomere number and length.[10,11] Consequently, post-CVA movement deficits may at first be dependent on disinhibition of select muscle groups, but ultimately may result primarily from biomechanical changes and impaired recruitment of weakened muscles.

Electromyographic (EMG) biofeedback is a modality frequently used in the treatment of neuromuscular dysfunction to provide immediate visual and auditory cues that represent specific muscle contractions.[12,13] The subject may attempt to modify these muscle contractions by controlling the feedback signals. Consequently, EMG biofeedback may be used to facilitate or reduce muscle activity to restore motor control. A traditional training strategy of relaxation of spastic musculature (downtraining) followed by facilitation of weak or paretic musculature (uptraining) has been advocated by many clinicians.[13,14] Results from other studies[6,15,16] indicate that the downtraining component may not be necessary. Downtraining alone may be effective in reducing hyperactivity, but when followed by uptraining, co-contraction often persists, resulting in little or no carryover of the downtraining effect.[17]

Data available to clinicians for evaluating the efficacy of EMG biofeedback include measurements of passive range of motion (PROM), active range of motion (AROM), and EMG activity.[12] Limits found in PROM help to determine whether limits in AROM are a result of changes in the biomechanical properties of muscle as opposed to myoneural alterations. The EMG measurements provide information about the target and/or opposing muscle during contraction. Thus, during active elbow extension, triceps brachii muscle EMG measurements will reflect agonist muscle activity, whereas biceps brachii muscle EMG measurements will show the amount of co-contraction.

Changes in speed of movement may be related to changes in functional abilities.[18] After sustaining a stroke, patients may have the capability to increase speed of movement. Although hypertonicity may be considered to be velocity-dependent, even during fast movements, spastic restraint may not be the primary motor deficit in patients with hemiplegia.[19] A key problem of control may not be reflex activity, but rather incomplete but prolonged antagonist recruitment during active reciprocal movements. Thus, the timing and magnitude of elbow extensor muscle activation may be impaired through uncontrolled recruitment in elbow flexors and prolonged activity during fast reciprocal movements (also known as abnormal co-contraction).

Controversy exists about the effectiveness of training patients with neurological impairment for functional improvements rather than first attempting to reduce hypertonus.[20-22] Traditional treatment strategies, such as neurodevelopmental treatment (NDT) and downtraining with EMG biofeedback, have focused primarily on decreasing antagonist activity before working toward agonist muscle facilitation.[14,17,23] Inadequate agonist recruitment, however, is a more consistent finding than increased antagonist activity as the major cause of decreased motor abilities.[8] Patients with stroke and traumatic brain injury have demonstrated the ability to improve in functional tasks despite enhanced activity in spastic musculature after participating in trials of one of two EMG feedback methods.[24]

In summary, the mounting evidence appears to indicate that specific "downtraining" of hyperactive muscles may not be a necessary precursor for attaining increased active joint movement or function. Alternatively, initiating treatment with functionally oriented tasks may be a better therapeutic strategy for the patient who is poststroke. In applying such a strategy, one would bypass the traditional notion of downtraining a hyperactive muscle in favor of recruiting weaker antagonist muscles within the context of task-oriented movements.

The purpose of this study was to determine the efficacy of uptraining the triceps muscle, in the presence of biceps muscle hypertonicity, using both feedback and nonfeedback paradigms during goal-oriented movements. Electromyographic biofeedback training was compared with simply practicing functional tasks in the absence of biofeedback.

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Table 2. *Mean (±SD) Electromyographic (EMG) Data (in Microvolts per Second) for Triceps Brachii and Biceps Brachii Muscles Across Baselines and Postintervention for the Feedback Group*

	Baseline						
	1	2	3	4	5	Mean	Post-Intervention
Extension task							
Triceps brachii muscle EMG	16.96±11.23	15.54±11.07	16.69±10.51	13.93±7.38	17.15±9.42	16.05±1.34	30.26±16.62
Biceps brachii muscle EMG	8.10±3.33	7.81±3.55	8.79±2.80	7.33±2.80	7.39±2.48	7.88±0.60	9.71±4.51
Resisted extension task							
Triceps brachii muscle EMG	16.71±13.32	17.35±13.52	16.00±7.25	16.25±7.58	17.55±9.62	16.77±0.67	26.63±13.15
Biceps brachii muscle EMG	8.79±4.05	8.86±4.62	8.70±1.94	8.28±3.45	7.56±2.74	8.44±0.54	9.58±3.54
Reaching task							
Triceps brachii muscle EMG	11.70±7.60	13.74±9.49	12.05±5.67	13.33±6.89	11.55±7.35	12.47±1.00	12.90±7.43
Biceps brachii muscle EMG	14.24±7.75	12.43±4.31	15.45±7.81	13.40±7.02	12.36±6.48	13.58±1.30	13.05±9.62

dB) and underwent a true root mean square (RMS) to DC conversion using a 25-millisecond time constant. Bio-flex fabric sensors,[†] silverized reusable surface electrodes with interelectrode distances of less than 1 cm, were secured on the bellies of the biceps and triceps muscles. The biceps muscle electrodes were placed at the midpoint between the anterior acromial surface of the scapula and the bicipital fossa. The triceps muscle electrodes were placed at the midpoint between the posterior acromial surface and the olecranon. The ground electrode, for each of the biceps and triceps muscle electrode pairs, was placed laterally over the deltoid muscle, between the biceps and triceps muscles. Skin impedance was reduced below 1 kΩ by scrubbing the skin with alcohol and spraying electrodes with Spraytrode Conductive ECG Spray[‡] prior to placement. During elbow extension, mean EMG activity of the triceps and biceps muscles was measured. For all tasks, EMG activity was present in both muscle groups (Tabs. 2 and 3); the variability in duration of activity from each muscle for each movement, however, precludes determining the relative magnitude of co-contraction or reciprocal inhibition. The antagonist muscle was always active at some point during the movement. Within a given time period, we believe mean EMG values provide information on the average activity of the muscles. Given the interelectrode distances and small (7-mm) recording surfaces, EMG activity was probably confined to the underlying muscle and not volume conducted from the antagonist.[27]

[†]Pharmaceutical Innovations Inc, Newark, NJ 07114.

Table 3. *Mean (±SD) Electromyographic (EMG) Data (in Microvolts per Second) for Triceps Brachii and Biceps Brachii Muscles Across Baselines and Postintervention for the Nonfeedback Group*

	Baseline						
	1	2	3	4	5	Mean	Post-Intervention
Extension task							
Triceps brachii muscle EMG	12.98±9.76	16.74±13.74	15.18±9.87	18.39±14.21	20.10±11.51	16.68±2.76	23.60±12.81
Biceps brachii muscle EMG	10.48±5.49	9.95±5.65	10.28±4.32	7.91±4.26	8.55±3.34	9.43±1.14	9.49±3.85
Resisted extension task							
Triceps brachii muscle EMG	13.25±9.28	15.21±15.56	18.69±13.44	16.81±12.17	19.93±10.19	16.78±2.67	22.54±12.79
Biceps brachii muscle EMG	10.59±4.87	9.26±5.81	9.86±4.23	7.74±4.05	8.61±3.70	9.21±1.10	9.50±4.39
Reaching task							
Triceps brachii muscle EMG	11.69±10.49	16.29±9.89	14.58±9.97	15.07±11.81	14.14±6.94	14.35±1.69	15.19±5.89
Biceps brachii muscle EMG	13.84±7.96	15.37±7.67	13.96±7.00	10.11±6.59	11.75±6.92	13.01±2.07	15.25±13.96

Table 4. *Results of One-Sample t Tests for Within-Group Comparisons of the Change[a] During Intervention (Postintervention Versus Mean Baseline)*

	Feedback Group			Nonfeedback Group		
	Mean Change[a]	t	P	Mean Change[a]	t	P
Range of motion						
Passive	16.95	−3.627	.008[b]	14.90	−2.973	.021[b]
Active	6.68	−3.438	.011[b]	4.70	−2.906	.023[b]
Mean triceps brachii muscle EMG						
Extension task	14.21	2.610	.035[b]	6.92	1.611	.151
Resisted extension	9.86	3.304	.013[b]	5.76	1.132	.295
Reaching task	0.43	−0.111	.915	0.84	0.495	.636
Mean biceps brachii muscle EMG						
Extension task	1.83	1.135	.294	0.06	−0.112	.914
Resisted extension	1.14	1.175	.278	0.29	0.134	.897
Reaching task	0.53	−0.205	.843	2.24	0.625	.552

[a]Range of motion units are in degrees; electromyographic (EMG) units are in microvolts per second.

[b]$P \leq .05$

pated in two to four sessions per week for both baseline and training. Postintervention measurements of the dependent variables were taken during a separate session within 3 days of the 10th treatment session. The procedure mirrored baseline measurement sessions.

Data Analysis

For each subject, mean values were calculated at each session for the following variables: AROM, PROM, biceps and triceps muscle EMG activity for each task, and movement speed for each task. All EMG measurements were quantified simultaneously for each task. Additionally, mean values for baseline and postintervention measurements were determined for all variables. Improvement was defined as a decrease in mean AROM and PROM, with 0 degrees defined as full extension; a decrease in mean EMG activity of the biceps muscle; an increase in mean EMG activity of the triceps muscle; and an increase in mean speed.

To assess the assumption of homogeneity between the two groups at baseline, a two-sample *t* test was performed on the mean of the five baseline measurements for each variable. To test whether the change from preintervention to postintervention measurements was different between the two groups, a two-sample *t* test was performed on the difference scores (postintervention minus the mean of the five baseline values). The *t* statistic for this test is the square root of the *F* test for the interaction of group×time for a repeated-measures analysis of variance.[50]

Within-group comparisons were made for both feedback and nonfeedback groups for each variable. To assess the effect of both treatments, the postintervention measurements were compared with the mean values of baseline measurements. To determine whether postintervention changes were greater than the expected initial baseline changes, the change within baseline measurements was compared with the change after intervention for each group. One-sample *t* tests were used to determine statistical significance for these comparisons. These analyses yield the same results as a repeated-measures analysis of variance for two time points. The normality of the distributions of the data for each dependent variable in each group was tested by the Shapiro-Wilkes statistic.

Correlations of the differences from postintervention to mean baseline values were performed within both groups using the Pearson Product-Moment Correlation Coefficient. Significance was set at an alpha level of $\leq .05$ for all statistical tests.

Results

Between-Group Comparisons

No significant differences were found between groups for the mean of the baseline measurements, thus confirming the assumption of homogeneity between groups preintervention. The two-sample *t* tests of difference scores from baseline to postintervention showed no significant differences for any variable. Thus, the amount of change was not related to treatment.

Within-Group Comparisons

Comparison of the mean baseline and postintervention measurements showed significant differences for some variables (Tab. 4). Measurements of speed for both groups were highly variable across all measurement sessions because of intersubject variability. Furthermore, the slow chart speed of the pen recorder offered poor resolution for the calculation of actual degrees per second. Consequently, because the reliability of these measurements is questionable, the speed variable was eliminated from further analysis.

Feedback group. Improvements in both AROM and PROM after EMG biofeedback training were statistically significant (Tab. 4). To further illustrate improvements, the trends across the measurement sessions for range of motion are displayed in the Figure. Variability across baselines and improvement from baselines to postintervention are represented. Mean EMG values of the triceps and biceps muscles across baseline and postinter-

Table 5. *Pearson Product-Moment Correlations of Differences From Postintervention to Mean Baselines*[a]

	Mean EMG			
	Triceps Brachii Muscle Extension	Triceps Brachii Muscle Resisted Extension	Triceps Brachii Muscle Reaching	Biceps Brachii Muscle Resisted Extension
Feedback group				
AROM	−.509	−.267	+.178	−.585
PROM	−.514	−.739[b]	+.122	−.632
Mean biceps brachii muscle extension	+.858[b]	...	...	...
Mean biceps brachii muscle resisted extension	...	+.551	...	...
Nonfeedback group				
AROM	−.405	−.501	−.429	−.707[b]
PROM	−.286	−.496	−.746[b]	−.071
Mean biceps brachii muscle extension	+.669	...	...	...
Mean biceps brachii muscle resisted extension	...	+.776[b]	...	...

[a]Gains in active range of motion (AROM) and passive range of motion (PROM) are represented by values approaching 0 degrees of extension, yielding negative postintervention minus preintervention values. Gains in triceps brachii muscle electromyographic (EMG) activity are represented by increasing microvoltage; consequently, a negative correlation implies improvement in both variables.

[b]$P \leq .05$.

Several factors must be considered to explain this finding. The combination of limited training sessions, limited sample size, and the chronic nature of the patient with stroke may have contributed to why one treatment was not superior to the other. Ten training sessions may have been too few to distinguish feedback from nonfeedback effects among this chronic group based on the findings indicating that effects of neuroplasticity are limited in patients who are more than 6 months poststroke.[31,32] Although there was a tendency for the feedback group to display more significant postintervention changes in mean triceps muscle EMG activity (Tab. 4) and more quantitative EMG differences between antagonist muscles with goal-directed training (Tabs. 2 and 3), one cannot assume that increasing the sample sizes would have necessarily yielded more impressive differentiation between the groups.

Furthermore, the development of both postural adaptations and antagonist muscle imbalances after the CVA may imply that the prevailing neuromuscular status of these patients is temporally dependent. Specifically, in the earlier stages after the cerebral insult, movement and limb posturing may be primarily influenced by the central lesion site causing disinhibition of select muscle groups. The relative contributions of peripheral joint and muscle changes (eg, collagen structure of tendon, viscoelastic properties, connective tissue alterations) may have a greater influence on limb posturing and movement after a period of time. If this supposition is correct, then specific retraining techniques[14,16,24] or exercises designed to recruit weak agonist muscles[8] may yield better results early in the rehabilitation process, before structural tissue changes have occurred.

Within-Group Comparisons

Significant within-group improvements were found in both groups for AROM and PROM. Repetitive active elbow movements during functional tasks might have been sufficient to improve these objective measurements. This finding helps to differentiate between peripheral and central components of hypertonus. Improvements in PROM consistent with increases in AROM (without decreases in biceps muscle EMG activity) strengthen the explanation that biomechanical changes may be one of the primary limiting factors in this particular chronic stroke population. If increases had occurred only in AROM, then central components logically would have been affected. Conversely, if only PROM showed improvements, then primarily peripheral components would have been considered. Thus, the range of motion improvements seen during this type of training may be a result of decreased biomechanical restrictions from adhesions, capsular shortening, and muscular contracture, as well as increased muscle fiber activation.[4,12] Consequently, in the condition of hypertonus, a pathological mechanical change in muscle fibers, as well as altered muscle fiber activation, should be considered.[33,34]

The significant increases for the feedback group in mean triceps muscle EMG activity in two of the three tasks (extension and resisted extension) revealed that audio and visual feedback enhance training, whereas similar changes were not seen in the nonfeedback group (Tab. 4). For the same two tasks, empirical comparisons of the mean triceps and biceps muscle EMG data (Tabs. 2 and 3) show that with the upper extremity supported, there were greater differences between the biceps and triceps muscle values (triceps:biceps muscle ratio of approximately 2:1). In contrast, for the reaching task, which

may be most effective in the acute patient during early stages after the cerebral insult. For the patient with chronic neurologic impairment, these findings justify treatment aimed at affecting biomechanical restrictions inherent in the spastic limb, as well as neuromuscular deficits. Finally, both neuromuscular and biomechanical changes can occur via the functional treatment approach presented in this study.

Conclusion

Electromyographic biofeedback up-training of the triceps muscle and practicing functional tasks without EMG biofeedback in the presence of biceps muscle hypertonicity both appear to be effective in improving elbow function in patients with chronic stroke. Improvements within both groups support existing theories concerning biomechanical (peripheral) restrictions contributing to limitations of movement, along with the contributions from disinhibited central pathways, to the manifestations of hypertonicity. Support is given to focusing treatment on functionally improving motoneuron recruitment in a weak, paretic muscle versus initially reducing hyperactivity in the spastic antagonist muscle.

Acknowledgments

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Assignment: Students will be assigned a body region. Each student will then be responsible for choosing one article from a peer reviewed journal using the virtual library in relation to applied anatomy and kinesiology for the assigned body region. Students must have the article approved by the instructor prior to submission of the annotated bibliography. The instructor will approve the article based on a peer reviewed journal not content or relevance to topic. Students will then be required to write an annotated bibliography on the approved article.

Date: Students need to turn in a hard copy of their research article for approval on no later than **Tuesday 10/08/19.**

The Final assignment is **DUE ON November 19, 2019.**

Instructions: Students will construct an annotated bibliography. An annotated bibliography is a list of citations of articles, books, and other publications on a particular topic. Each citation is followed by a relatively brief paragraph that summarizes the source's argument and other relevant material including its intended audience, sources of evidence, and methodology. The assignment will be completed individually and out of class.

The annotated bibliography consists of two elements:

1. The citation in current **AMA** style format

2. The Annotation - The annotation should consist of one paragraph using whole complete sentences in the third person and should be approximately 150-200 words in length. The assignment should be typed, double spaced, in Times New Roman 12 font, 1" margin.

The annotation should include most, if not all, of the following:

- Explanation of main purpose
- Description of content
- Focus of article
- Relevance of topic
- Type of intended audience
- Evaluate its method, conclusion and/or reliability
- Strengths / weaknesses or biases
- Your own brief impression of the work

Assessment: The assignment will be assessed according to the criteria identified in the grading rubric on the attached page.

*Once all of the annotated bibliographies are turned in the instructor will be responsible for compiling a comprehensive annotated bibliography, which will be given to the students.

Student Name
PTA 1401: Applied Anatomy and Kinesiology with Lab

L.I.R.N. assignment: annotated bibliography
Sample

This is a sample of an annotated bibliography.

1. Waters, Eric. Suggestions From the Field for Return to Sports Participation Following Anterior Cruciate Ligament Reconstruction. J Orthop Sports Phys Ther. 2012; 42.4. 326-36. Retrieved From http://www.jospt.org/issues/articleID.2737,type.2/article_detail.asp

Eric Waters's, in his 2012 article "Suggestions From the Field for Return to Sports Participation Following Anterior Cruciate Ligament Reconstruction" concentrates on the treatment of ACL injuries. He supports this by setting examples of specific exercises that treat or prevent ACL injuries. His purpose is to show how treatment works with an ACL injury and explain how to prevent it, in order for others to educate themselves with the study that he has done. His intended audience is physical therapists, doctors, physicians, and athletes.

Water's article is relevant to my topic because he focuses on rehabilitation of athletes. Stating, "Preparing a basketball player for an effective return to play requires that the final and most functional phase of the rehabilitation program encompass a thorough protocol based on exercises that maintain proper lower extremity alignment throughout all the conceivable scenarios of a basketball game," (333) he exemplifies that it is important to perform certain exercises in order to compete at the best ability.