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# Elastic, Viscous, and Mass Load Effects on Poststroke Muscle Recruitment and Co- contraction During Reaching: A Pilot Study

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## Elastic, Viscous, and Mass Load Effects on Poststroke Muscle Recruitment and Co-contraction During Reaching: A Pilot Study

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**Background.** Resistive exercise after stroke can improve strength (force-generating capacity) without increasing spasticity (velocity-dependent hypertonicity). However, the effect of resistive load type on muscle activation and co-contraction after stroke is not clear.

**Objective.** The purpose of this study was to determine the effect of load type (elastic, viscous, or mass) on muscle activation and co-contraction during resisted forward reaching in the paretic and nonparetic arms after stroke.

**Design.** This investigation was a single-session, mixed repeated-measures pilot study.

**Methods.** Twenty participants (10 with hemiplegia and 10 without neurologic involvement) reached forward with each arm against equivalent elastic, viscous, and mass loads. Normalized shoulder and elbow electromyography impulses were analyzed to determine agonist muscle recruitment and agonist-antagonist muscle co-contraction.

**Results.** Muscle activation and co-contraction levels were significantly higher on virtually all outcome measures for the paretic and nonparetic arms of the participants with stroke than for the matched control participants. Only the nonparetic shoulder responded to load type with similar activation levels but variable co-contraction responses relative to those of the control shoulder. Elastic and viscous loads were associated with strong activation; mass and viscous loads were associated with minimal co-contraction.

**Limitations.** A reasonable, but limited, range of loads was available.

**Conclusions.** Motor control deficits were evident in both the paretic and the nonparetic arms after stroke when forward reaching was resisted with viscous, elastic, or mass loads. The paretic arm responded with higher muscle activation and co-contraction levels across all load conditions than the matched control arm. Smaller increases in muscle activation and co-contraction levels that varied with load type were observed in the nonparetic arm. On the basis of the response of the nonparetic arm, this study provides preliminary evidence suggesting that viscous loads elicited strong muscle activation with minimal co-contraction. Further intervention studies are needed to determine whether viscous loads are preferable for poststroke resistive exercise programs.

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Stroke is the leading cause of serious, long-term disability in the United States because of sensorimotor impairments that affect functional ability.<sup>1</sup> Physical therapists are faced with the clinical challenge of designing rehabilitation programs that address the activity limitations and resultant impairments associated with stroke. Of the cluster of upper motor neuron impairments that affect movement production after stroke, weakness is most strongly correlated with activity limitations.<sup>2,3</sup> For the upper extremity, activities that include reaching are commonly affected.<sup>4,5</sup> Upper motor neuron weakness is primarily associated with poor agonist muscle recruitment.<sup>6,7</sup> However, impaired timing of agonist and antagonist muscle activation can result in co-contraction because of overlapping and opposing muscle activation; this co-contraction also can contribute to weakness during dynamic tasks.

The effect of impaired coordination associated with co-contraction after stroke remains controversial, in part because of the variety of tasks and analytical approaches used as well as the operational definitions of co-contraction. For example, abnormal co-contraction in hemiparetic muscles has been described as “markedly altered timing”<sup>8</sup> and as a “delay in initiation and termination.”<sup>9</sup> Inappropriate co-contraction has been reported during dynamic reaching af-

ter stroke<sup>9,10</sup> or in association with certain stages of recovery.<sup>11,12</sup> In contrast, others have reported normal co-contraction levels during isometric contractions<sup>13</sup> and appropriate sequential activation during reaching after stroke.<sup>14</sup>

Clinical studies have demonstrated that resistive exercise programs after stroke can increase strength (force-generating capacity)<sup>15-19</sup> and improve the performance of functional tasks<sup>20-22</sup> with no increase in spasticity (velocity-dependent hypertonicity).<sup>17,23</sup> In addition, improvements in strength have been associated with increased accuracy and timing (ie, coordination) in dynamic upper-extremity tasks.<sup>20,24</sup> Our research hypotheses were formulated, in part, on the basis of the potential of resistive exercise to improve the strength and coordination of agonist and antagonist muscle groups impaired after stroke.

Although physical therapists commonly use weights, elastic bands, pneumatic or hydraulic exercise machines, or pools for resistive exercise, no systematic studies have been conducted in people after stroke to investigate the effects of various resistive load types during strength training. The kinetic properties of resistive loads place unique demands on muscles during movement: the force required to elongate an elastic load increases with the distance the material is stretched; the force required to move against a viscous load (such as water) increases with movement speed; and for weights (mass loads), balanced and appropriately timed acceleration and deceleration forces are required to move and stop the load. Studies comparing these types of resistance have investigated adaptations to load type,<sup>25</sup> muscle responses to unexpected loading,<sup>26</sup> and variations in load magnitude<sup>27</sup>—but only in people who were neurologically intact. These subjects did

not demonstrate co-contraction with any resistive load.<sup>8,27</sup> It is not clear how these load types would affect muscle activation during resistive exercise in people after stroke.

A growing body of literature has consistently demonstrated that force production and coordination (ie, speed and accuracy) are impaired in both the paretic and the nonparetic arms after stroke.<sup>28-31</sup> The purpose of this pilot study was to investigate the effects of commonly used resistive load types (mass, elastic, and viscous) on muscle activation and timing in both arms after stroke. The significance of this study is that it contributes important clinical insights related to the effects and, potentially, the effectiveness of specific physical rehabilitation interventions for people after stroke. Thus far, there has been insufficient evidence to guide therapists in selecting the resistive load type that can result in both an increase in strength and an improvement in muscle coordination.

We hypothesized that the level of muscle activation during reaching would be higher against the viscous load because the peak force requirements of viscous loads coincide with the peak velocity profile of the movement, arguably the “weakest” part of the reach, on the basis of the force-velocity relationship of muscle contraction. In contrast, we hypothesized that muscle timing for the mass load would be associated with the largest amount of abnormal co-contraction in both the nonparetic and the paretic arms after stroke because the mass load requires appropriately synchronized agonist and antagonist muscles to successfully move (accelerate) and stop (decelerate) the load.

### Method Participants

A convenience sample of 10 right-handed adults with hemiparesis attributable to stroke and 10 age-



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**Table 1.**  
Participants' Demographic Data<sup>a</sup>

Group	Participant	Age, y	Sex	Month After Stroke	Paretic Arm	MAS Score	UE-FM Score	Hand Opening <sup>b</sup>	Grip (kg)		Maximum Isometric Push (N)	
									Paretic Arm	Nonparetic Arm	Paretic Arm	Nonparetic Arm
Stroke	1	77	F	118	L	2	46	Yes	14	16	87	181
	2	62	M	11	L	3	39	Yes	8	45	212	351
	3	59	F	42	L	2	61	Yes	20	23	183	227
	4	56	F	11	R	2	55	Yes	10	32	106	295
	5	54	M	60	L	0	60	Yes	10	23	132	249
	6	53	M	18	R	3	31	No	21	NT	146	338
	7	52	M	316	L	2	24	No	13	43	318	407
	8	51	F	37	L	2	24	No	9	43	73	236
	9	48	M	39	L	3	21	No	17	NT	102	353
	10	31	M	276	L	3	21	Yes	2	41	97	435
$\bar{X}$ (SD)		54 (11)		93 (112)			38 (16)		12 (6)	33 (11)	145 (75)	307 (83)
Group	Participant	Age, y	Sex						Grip (kg)		Maximum Isometric Push (N)	
									Left Arm	Right Arm	Left Arm	Right Arm
Control	1	80	F						26	29	214	216
	2	60	F						32	29	240	195
	3	60	M						46	46	517	550 <sup>c</sup>
	4	58	F						25	23	211	191
	5	58	F						34	33	249	228
	6	55	M						48	52	550 <sup>c</sup>	459
	7	56	M						63	61	550 <sup>c</sup>	501
	8	54	F						43	48	373	354
	9	46	M						57	50	550 <sup>c</sup>	550 <sup>c</sup>
	10	27	M						68	71	529	394
$\bar{X}$ (SD)		55 (13)							44 (15)	44 (15)	398 (155)	364 (148)

<sup>a</sup> F=female, M=male, L=left, R=right, MAS=Modified Ashworth Scale, NT=not tested, UE-FM=upper-extremity portion of the Fugl-Meyer Motor Assessment (maximum score=66).

<sup>b</sup> Hand opening=active opening of the fingers to grasp an object.

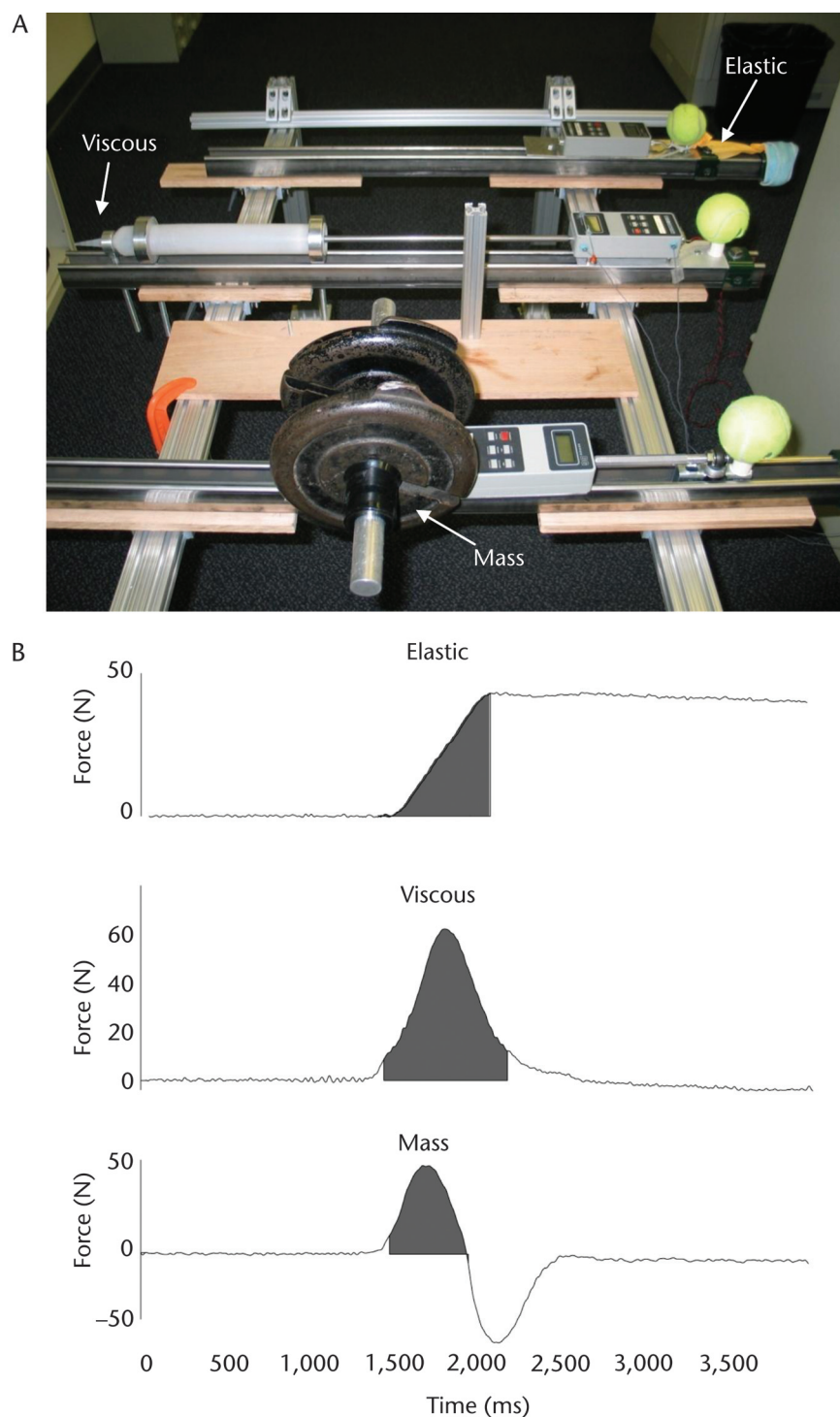
<sup>c</sup> Upper limit of force transducer.

matched right-handed control participants took part in this study. Participants were recruited from local rehabilitation centers and by use of posted flyers. Participants with hemiplegia had had a stroke at least 6 months before testing and had residual unilateral upper-extremity hemiparesis but retained the ability to push a handle at waist level away from their bodies while seated. Exclusion criteria included non-stroke-related neurologic deficits, tremor,

and inability to follow instructions. Control participants had no history of neurologic disease or injury or upper-limb injury and were age matched to the participants with stroke ( $\pm 5$  years) (Tab. 1). Written informed consent was obtained for each participant, in compliance with policies established by the institutional review boards of Marquette University and Rocky Mountain University of Health Professions.

### Instruments

The experimental task involved pushing the ball handle of a light cart-and-rail apparatus 15 cm away from the body in the horizontal plane at waist level against 3 different types of resistance: mass, viscous, and elastic (Fig. 1A). The mass load consisted of disk weights mounted on the cart, the viscous load comprised a pneumatic plunger with nozzle orifices of various sizes, and the elastic load was provided by



**Figure 1.** Experimental loads. (A) Cart-and-rail apparatus used for the experimental task. Participants reached 15 cm by pushing forward against the elastic load (far), the viscous load (center), and the mass load (near). Each cart was fitted with infrared markers and a force transducer. (B) Representative data for force profiles collected from a single control participant (x-axis shows time in milliseconds; y-axis shows force in newtons) during forward reaching against elastic, viscous, and mass loads. Shaded areas under the acceleration components of the curves were used to determine equivalent loads.

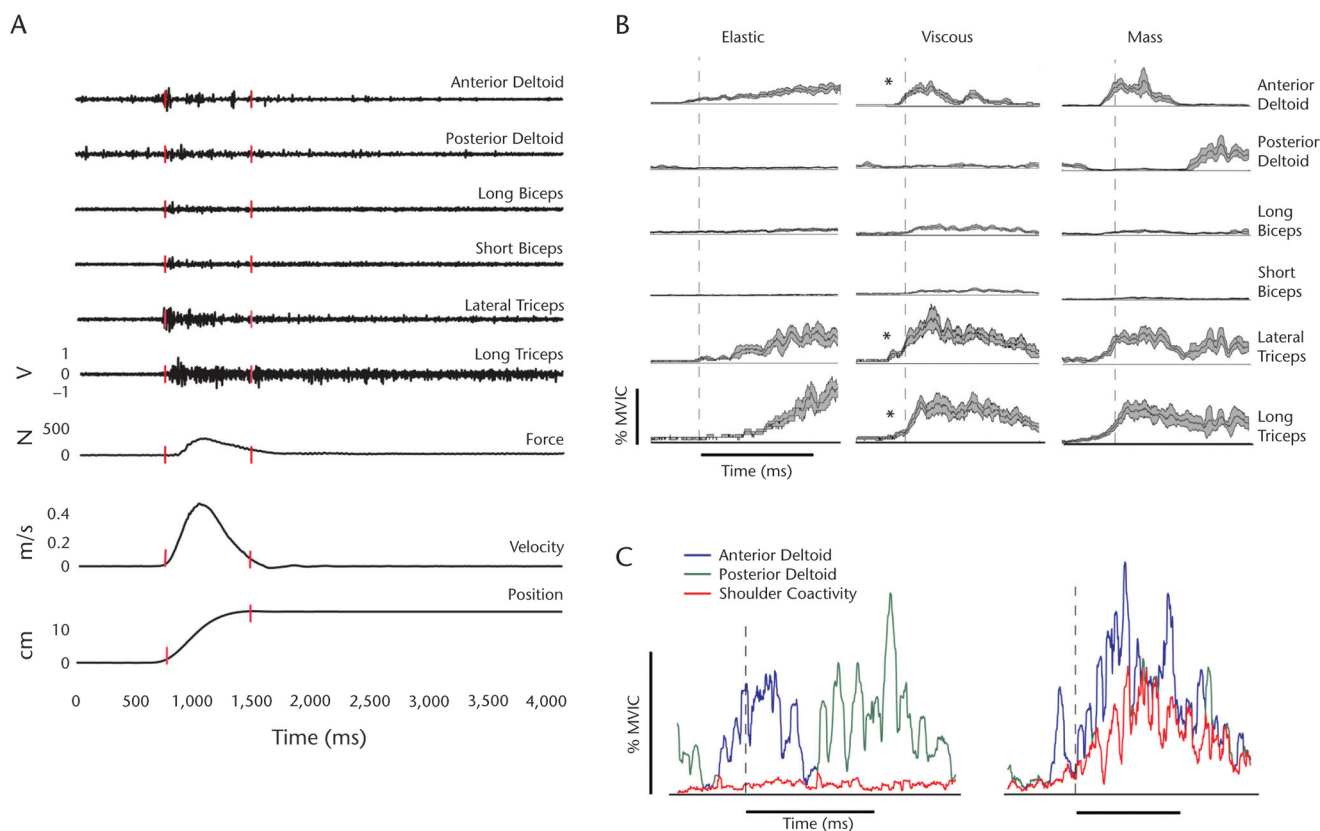
Thera-Band\* materials. Each cart was fitted with infrared markers and a force transducer (model BG 100<sup>†</sup>) mounted in series with its respective load. An OPTOTRAK 3020 motion analysis system<sup>‡</sup> recorded force, kinematic, and electromyography (EMG) data for this study at a rate of 1,000 samples per second (sample data from one trial are shown in Fig. 2A).

**Experimental Procedure**

Each participant took part in a single experimental session. After providing informed consent, participants with stroke were evaluated for motor impairment and the severity of the impairment by a licensed physical therapist. Baseline assessments for participants with stroke included the Modified Ashworth Scale (MAS) and the upper-extremity portion of the Fugl-Meyer Motor Assessment. The grip strength of both arms was assessed for all participants.

Participants were required to perform 10 successful reaches for all 3 loads with each arm. They were instructed to wait for the “go” signal and then push the cart forward until the cart marker touched the target post and remained at the target briefly before returning to the start position. A successful reach involved moving the cart forward to the spatial target (criterion: 16±1 cm) in about 0.5 second (criterion: 700±100 milliseconds). Data collection for each trial lasted 4 seconds from the time of the auditory “go” signal. Participants were informed of the criteria for a successful reach, and feedback about reach time and distance was provided after each trial. Participants were allowed to practice until they were successful

\* The Hygenic Corp, 1245 Home Ave, Akron, OH 44310.  
<sup>†</sup> Mark-10 Corp, 11 Dixon Ave, Copiague, NY 11726.  
<sup>‡</sup> Northern Digital Inc, 5555 Business Park, Ste 100, Bakersfield, CA 93309.



**Figure 2.**

Electromyography (EMG) data processing. (A) Representative data collected in one trial from a single control participant reaching against a viscous load. From top to bottom: channels of raw surface EMG data collected from the anterior deltoid muscle, posterior deltoid muscle, long head of the biceps muscle, short head of the biceps muscle, lateral head of the triceps muscle, and long head of the triceps muscle and force, velocity, and position data collected during forward reaching against a viscous load. Red cursors indicate the onset and offset of movement. (B) Representative data collected from a single control participant for the 6 muscles of interest plotted as the average of 10 trials of normalized, full-wave-rectified EMG data ( $\pm 95\%$  confidence interval) at 30% maximum voluntary isometric contractions (MVIC) for elastic, viscous, and mass loads. The dependent measure of agonist muscle activation (EMG impulse) was calculated as the area under the curve. Trials were aligned at movement onset (vertical dashed line). Scale bars (solid black lines) represent 500 ms for time on the x-axis and 20% MVIC on the y-axis. For traces indicated by asterisks, the vertical scale bar corresponds to 50% MVIC. (C) Representative example of normalized full-wave-rectified EMG data for anterior deltoid (blue trace) and posterior deltoid (green trace) muscle activation from a control arm (left panel) and a paretic arm (right panel) during reaching against a mass load. The red trace indicates the lowest EMG signal between the 2 muscles at each time point; the area under the red curve represents the measure of co-contraction. The left panel represents little co-contraction; the right panel represents significant co-contraction. The vertical dashed line indicates the onset of movement, when the forward reach velocity exceeded 0.02 m/s. Scale bars (solid black lines) represent 500 ms for time on the x-axis and 10% MVIC on the y-axis.

but generally needed only a few trials. Each participant reached against one type of resistance until 10 successful trials were achieved. This process was repeated for each of the remaining load types, resulting in 30 trials of data. Load order was randomized across subjects. Participants were allowed to rest as often as needed, although no subject requested a rest. Participants with

stroke started with the nonparetic limb so that they could learn the task before performing it with the less-coordinated arm<sup>32</sup>; control participants started with the dominant (right) arm.

Before and after the reaching trials, maximum-effort force data were collected from 3 isometric pushes against a cart locked in the midreach

position. The pretrial pushes were used to determine how much resistance to use for the subsequent experimental reaches. Pre- and postexperimental maximum-effort pushes also were compared to assess for fatigue; all participants exceeded their initial efforts by a small amount (<7%) in postexperimental testing, suggesting a modest learning effect and no fatigue.

### Data Collection and Analysis

**Load equivalency.** Because the different load types placed substantially different kinetic demands on the subjects during reaching, we developed a method to determine resistive loads that would require approximately equivalent efforts across the 3 load types. On the basis of previous protocols of resistive movement after stroke,<sup>12,23</sup> our goal was for each participant to reach against loads requiring approximately 30% of the preexperimental maximum isometric push. Resistive loads for each arm were matched to create “triplets” of equivalent force impulses, so that  $I_{\text{mass}} = I_{\text{viscous}} = I_{\text{elastic}}$  with  $I$  being calculated as follows:

$$I = \int_{t=0}^{t=t_f} F(t) dt$$

In this equation,  $F$  is the measured force,  $t$  is time, and  $t=0$  and  $t=t_f$  represent the start time and the finish time for the movement, respectively. Thus, each participant was assigned a mass load whose peak acceleratory force requirement was closest to (but not more than) 30% of the subject’s preexperimental maximum isometric push, and the elastic and viscous loads with impulse values equivalent to that of the mass load completed the triplet.

### EMG data collection and analysis.

Surface EMG data were collected from the anterior and posterior deltoid muscles, both heads of the biceps muscle, and the lateral and long heads of the triceps muscle (Fig. 2A). The prime movers for our task were identified as the anterior deltoid and triceps muscles in a pilot study of adults who were healthy; the antagonists were identified as the posterior deltoid and biceps muscles.

The EMG signals were preamplified with a gain of 1,000 and band limited

to frequencies between 10 and 500 Hz before sampling with a Noraxon MyoSystem 1200.<sup>§</sup> Postprocessing within MATLAB<sup>||</sup> included calculating root-mean-square amplitudes of the full-wave-rectified EMG signals with a 25-millisecond sliding-window root-mean-square filter and a 50-millisecond sliding-window low-pass filter. To capture the onset of contraction that precedes movement, we analyzed the EMG data beginning 300 milliseconds before the onset of movement. Movement onset was identified as the point at which the cart velocity exceeded 0.02 m/s, and onset ended when the velocity returned to less than 0.02 m/s (Fig. 2A, red cursors).

The EMG data were recorded during preexperimental maximum voluntary isometric contractions (MVICs) for shoulder and elbow flexion and extension and quiet resting baseline trials.<sup>28,33</sup> The lowest average EMG value for 3 resting baseline trials was subsequently subtracted from all experimental EMG values (including MVIC EMG values). The highest of the 3 resulting MVIC EMG values for each muscle group was used for normalization during postprocessing data analysis.<sup>34,35</sup>

All dependent outcome variables were calculated from the average EMG impulse data (V·ms)—expressed as a percentage of the MVIC—and included the average impulse for each agonist muscle and the co-contraction impulse between agonist and antagonist muscles. For each resistance type, the EMG signals from its 10 trials were averaged for each muscle, and the area under the curves was calculated (Fig. 2B). Our co-contraction measure estimated the amount of EMG overlap

for agonist and antagonist pairs, calculated as the area under the curve created by the lower of the 2 EMG values at each moment in time (Fig. 2C, red trace).<sup>36</sup> This co-contraction impulse value reflected how much the least-active muscle was firing throughout the movement. In this way, agonist and antagonist pairs could be active at different times without any co-contraction, a distinction that was critical for the acceleration and deceleration of mass loads (Fig. 2B, right column).

### Statistical Testing

The EMG recordings from one participant with hemiplegia were corrupted by a faulty ground electrode. The EMG data for this participant and the corresponding matched control participant were excluded from further analysis.

Given the multiple comparisons of this study, a multivariate analysis of variance (MANOVA) was used to assess for an effect of arm type or load type on task performance. Separate analyses were completed for the paretic and nonparetic arms, each paired with the respective right or left arm of the age-matched control participant. To determine the effect of specific load types (mass, viscous, and elastic) on our dependent measures of agonist muscle activation and co-contraction, we used separate *post hoc* 2 (arm type) × 3 (load type) mixed-design, repeated-measures analyses of variance (ANOVAs). In all cases, effects were considered statistically significant at  $P=.05$ , as determined with Tukey  $t$  tests, when appropriate. Statistical tests were performed with the Minitab<sup>#</sup> statistics package.

<sup>§</sup> Noraxon USA Inc, 13430 N Scottsdale Rd, Ste 104, Scottsdale, AZ 85254.

<sup>||</sup> The MathWorks Inc, 3 Apple Hill Dr, Natick, MA 01760-2098.

<sup>#</sup> Minitab Inc, Quality Plaza, 1829 Pine Hall Rd, State College, PA 16801-3008.

**Table 2.**  
Participants' Strength Values and Analysis<sup>a</sup>

Parameter	Control Participants						Participants With Stroke			Paretic vs Control Arms, P <sub>4</sub>	Nonparetic vs Control Arms, P <sub>5</sub>
	Right	Left	P <sub>1</sub>	(Paretic) Control Arm	(Nonparetic) Control Arm	P <sub>2</sub>	Paretic Arm	Nonparetic Arm	P <sub>3</sub>		
$\bar{X}$ (SE) grip strength, kg	44.2 (3.9)	44.2 (3.9)	1.0	44.4 (3.9)	44.0 (43.9)	.95	12.4 (2.4)	33.3 (3.4)	<.001	<.001	.11
$\bar{X}$ (SE) maximum isometric push, kg	363.8 (12.2)	398.3 (12.4)	.62	367.2 (12.4)	374.9 (12.4)	.91	145.6 (8.6)	307.0 (9.1)	<.001	.001	.24

<sup>a</sup> P<sub>1</sub>=one-way analysis of variance (ANOVA) for comparison of right and left arms of control participants, P<sub>2</sub>=one-way ANOVA for comparison of (paretic) control and (nonparetic) control arms, P<sub>3</sub>=one-way ANOVA for comparison of paretic and nonparetic arms, P<sub>4</sub>=one-way ANOVA for comparison of paretic and control arms, P<sub>5</sub>=one-way ANOVA for comparison of nonparetic and control arms.

**Results**  
**Baseline Participant Characteristics**

Participants with stroke and participants who were neurologically intact (control participants) were well matched with respect to age and sex (Tab. 1). As expected, values for both grip and isometric strength assessments for the paretic arm were significantly lower than those for both the nonparetic and the control arms ( $P \leq .001$  for each). There were no significant strength differences between the right and the left arms of the control participants ( $P$  values for grip and push were 1.0 and .62, respectively) (Tab. 2). Although the lower mean values of both strength measures for the nonparetic arm than for the control arm were not significantly different ( $P$  values for grip and push were .11 and .24, respectively), these differences reflected medium to large effect sizes<sup>37</sup> (.79 and .55 for grip and push, respectively), consistent with previous reports indicating mild impairment of the nonparetic arm.<sup>28,30,31,38</sup>

**Paretic Arm**

The MANOVA provided evidence for a difference in arm type ( $P < .001$ ) but not in load type ( $P = .74$ ) across our paretic arm performance measures. A *post hoc* repeated-measures ANOVA indicated that the level of

normalized muscle activation was significantly higher in the paretic arm than in the control arm for the agonist muscles (anterior deltoid and triceps muscles) as well as for coactivity at the shoulder and elbow ( $P < .001$  for all comparisons), reflecting a marked effect of stroke on muscle recruitment and selection (Tab. 3, Fig. 3).

Although the MANOVA did not provide compelling evidence of an effect of load type, visual inspection of the data in a comparison of the paretic and control arms in Figure 4 (histogram pairs in top row, right) suggested that the anterior deltoid muscle recruitment of the control arm was affected by load type. A separate *post hoc* one-way ANOVA revealed that the level of anterior deltoid muscle recruitment for the elastic and viscous loads was significantly higher than that for the mass load in the control arm ( $P = .004$ ), whereas there were no differences between the load types in the paretic arm ( $P = .92$ ). We suspect that this interaction between load type and arm type did not reach statistical significance because of the similar trending of the data for both the paretic and the control arms in combination with the large variability of the data for the paretic arm.

**Nonparetic Arm**

A second MANOVA provided compelling evidence for significant effects of arm type ( $P = .007$ ) and load type ( $P = .002$ ) in a comparison of the nonparetic and control arms. A *post hoc* analysis revealed that normalized muscle activation was significantly higher in the nonparetic arm than in the matched control arm for all outcome measures ( $P < .05$ ) except anterior deltoid muscle recruitment (Tab. 3, Fig. 3).

Both agonist muscle recruitment and co-contraction were affected by load type at the shoulder but not at the elbow (Tab. 3, Fig. 4). Elastic and viscous loads resulted in significantly higher levels of muscle recruitment than the mass load for the anterior deltoid muscle in both the nonparetic and the control arms (Tab. 3 [Tukey *post hoc* analysis], Fig. 4). Although the elastic load elicited higher levels of shoulder co-contraction than the mass load in the nonparetic arm, the viscous load did not (Tab. 3 [Tukey *post hoc* analysis], Fig. 4). A marginal interaction between arm type and load type for anterior deltoid muscle recruitment was also demonstrated ( $P = .06$ ). Despite the different magnitudes of the responses, both arm types demonstrated a sensitivity to load type at the shoulder, reflecting a blend of



## Poststroke Muscle Recruitment and Co-contraction During Reaching

**Table 3.**

Muscle Impulse Data From Repeated-Measures Analysis of Variance for Nonparetic and Paretic Arms<sup>a</sup>

Parameter	Load	V-ms for:				Load (2 df)		Arm (1 df)		V-ms for:				Load (2 df)		Arm (1 df)	
		Nonparetic Arm (n=9)		Control Arm (n=9)		F	P	F	P	Paretic Arm (n=9)		Control Arm (n=9)		F	P	F	P
		$\bar{X}$	SE	$\bar{X}$	SE					$\bar{X}$	SE	$\bar{X}$	SE				
Anterior deltoid muscle activation						12.93	<.001 <sup>b</sup>	1.92	.17					0.84	.44	15.54	<.001
	Mass	28.6	9.3	24.4	4.0					130.3	36.4	34.8	8.9				
	Elastic	88.4	14.8	67.2	13.6					150.9	45.2	81.6	15.2				
	Viscous	80.7	14.1	67.8	7.5					149.9	33.5	84.0	20.7				
Triceps muscle activation						0.93	.40	4.48	.04					0.88	.42	17.69	<.001
	Mass	235.2	31.6	169.4	30.8					322.9	72.5	201.1	22.9				
	Elastic	169.2	37.8	123.0	19.2					259.1	43.7	141.2	26.8				
	Viscous	233.8	79.4	129.4	19.9					282.3	46.7	163.1	36.8				
Shoulder coactivity						3.49	.04 <sup>c</sup>	4.83	.03					0.10	.91	19.4	<.001
	Mass	6.9	3.9	8.8	2.3					39.7	11.1	8.9	2.0				
	Elastic	24.2	6.1	9.4	1.7					44.2	13.2	8.9	2.0				
	Viscous	15.6	2.2	10.3	1.9					35.8	10.8	9.0	2.2				
Elbow coactivity						0.47	.63	11.47	.001					0.59	.56	43.46	<.001
	Mass	28.8	6.5	15.1	3.8					74.2	16.5	14.3	4.1				
	Elastic	23.2	5.9	11.3	2.6					56.7	8.8	11.2	3.1				
	Viscous	27.1	6.1	12.2	2.6					65.6	14.0	11.5	3.3				

<sup>a</sup> Bold type indicates comparisons that achieved statistical significance ( $P < .05$ ).

<sup>b</sup>  $P$  values determined with Tukey *post hoc* analysis for comparisons of mass with elastic, mass with viscous, and elastic with viscous were .0001, .0003, and .95, respectively.

<sup>c</sup>  $P$  values determined with Tukey *post hoc* analysis for comparisons of mass with elastic, mass with viscous, and elastic with viscous were .03, .31, and .49, respectively.

both normal and impaired responses in the nonparetic arm.

### Discussion

On the basis of the kinetic properties of common resistive load types used for strengthening, reaching against elastic or viscous loads requires only agonist muscle activation, whereas reaching against mass loads requires appropriately timed agonist and antagonist muscle activation to move and stop the load (Fig. 2B).<sup>39</sup> As expected, people without neurologic involvement had higher levels of agonist muscle (ie, anterior deltoid and triceps muscles) activation during forward reaching against the elastic and viscous loads than against the

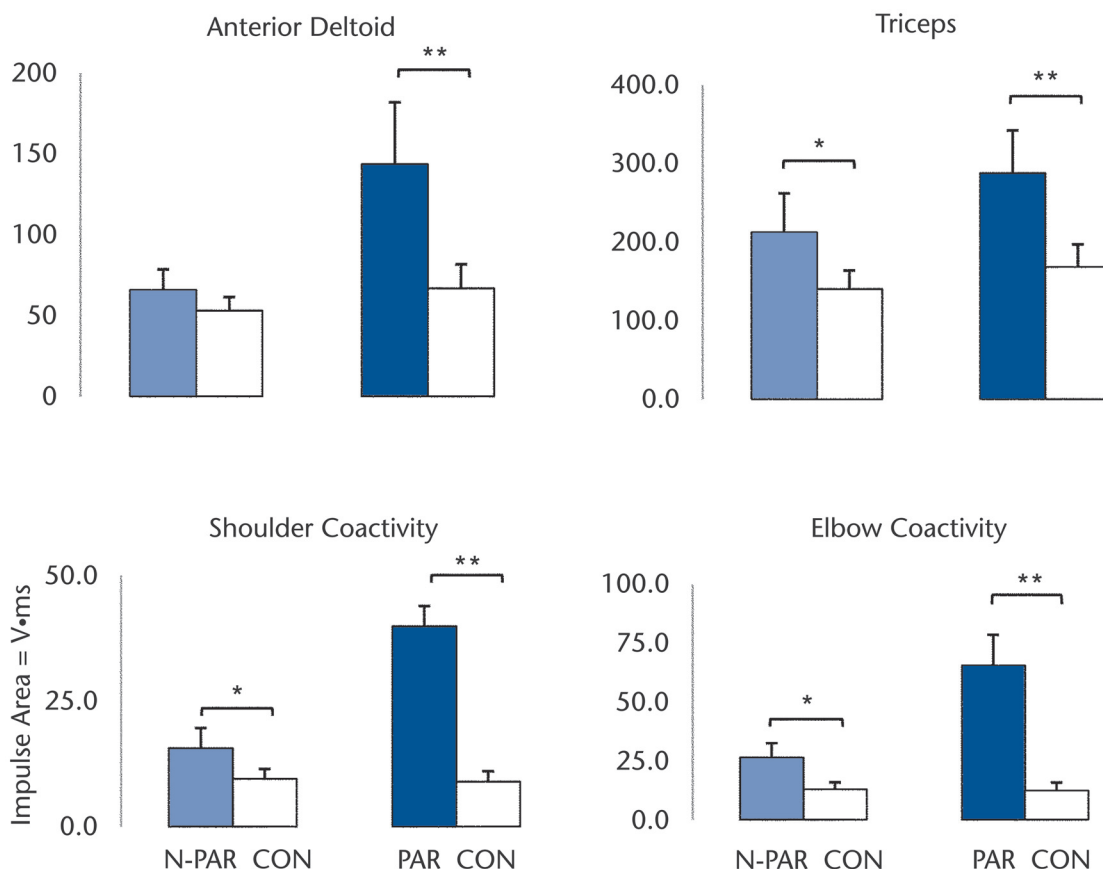
mass load, with little co-contraction for any load type.

In contrast, reaching with a hemiparetic (paretic) arm resulted in a high percentage of muscle activation and a higher level of co-contraction across all load types relative to those in people without neurologic involvement.

We also found evidence of impairment during reaching in the nonparetic arm. With the exception of shoulder agonist muscle activation, nonparetic arms also demonstrated higher percentages of muscle activation and co-contraction, which differed in response to load type. Spe-

cifically, elastic and viscous loads were associated with a higher level of agonist muscle activation (anterior deltoid muscle), and elastic loads were associated with the most co-contraction at the shoulder.

In light of our findings of differences in muscle activation patterns in the paretic and nonparetic arms of people after stroke and people who were neurologically intact (control participants), future research and clinical applications should include consideration of the types of loads being used to improve strength, and a load type should be chosen on the basis of its effect on muscle activation.



**Figure 3.**

Effect of arm type on normalized electromyography (EMG) impulses. Bar graphs show mean and standard error (SE) of the normalized EMG impulses for the agonist anterior deltoid and triceps muscles (top row) and coactivity for the shoulder and elbow (bottom row) by group collapsed across load types. Shaded bars represent the paretic arm (PAR [dark blue]) and the nonparetic arm (N-PAR [light blue]) of participants with stroke; white bars represent matched control (CON) participants. Significant differences are indicated by asterisks: \* $P < .05$ , \*\* $P < .001$ .

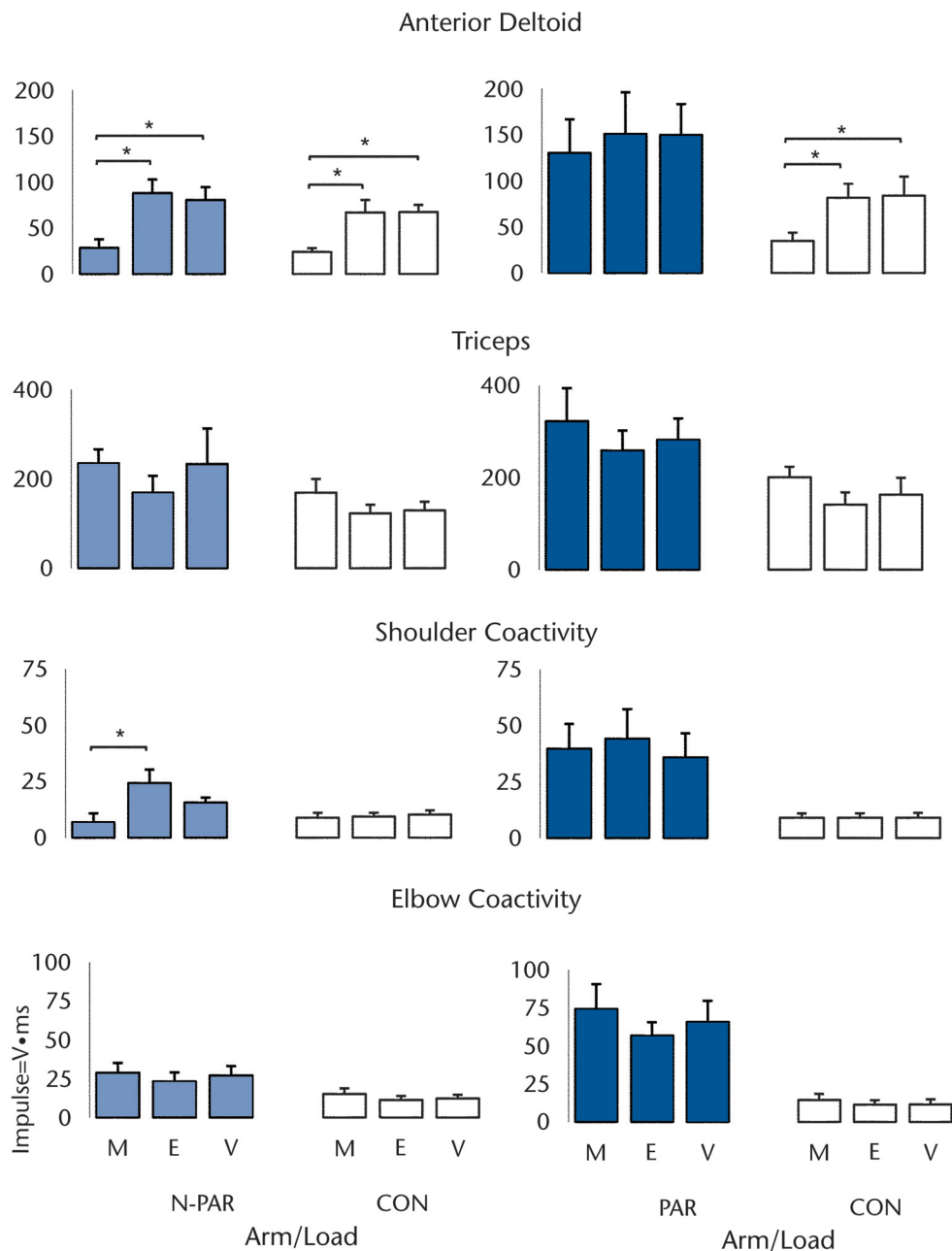
### Agonist Muscle Recruitment

Because the peak force requirement of a viscous load coincides with the peak of the velocity profile (theoretically the weakest part of the reach, on the basis of the force-velocity relationship of the muscles), we hypothesized that the viscous load would induce a higher level of agonist muscle recruitment. This hypothesis was only partially supported by our data. The elastic and viscous loads were equally effective in eliciting significantly higher levels of agonist muscle activation than the mass load—and only at the shoulder for the control and nonparetic arms. As expected for the control group,

the elastic and viscous loads elicited only agonist muscle activation, whereas the mass load elicited a brief agonist burst and then a brief antagonist burst, which coincided with the acceleration and deceleration profiles for the load, respectively. This biphasic muscle activation profile is consistent with those in other single-joint studies of subjects who were healthy and who responded to changes in these load types.<sup>19,27,39</sup> Gottlieb et al<sup>27</sup> reported synchronization of biphasic muscle torque and EMG values at both the elbow and the shoulder in response to resisted reaching, whereas our participants demonstrated this pat-

tern only at the shoulder. This difference likely was attributable to the arm configuration for the reaching task. In the study by Gottlieb et al,<sup>27</sup> the reach occurred with 90 degrees of shoulder abduction, whereas the reach occurred with 0 degrees of abduction in the present study. Such posture-dependent and task-specific effects also have been described by other authors.<sup>40–43</sup>

In contrast, the paretic arm consistently used a higher percentage of maximum voluntary effort and co-contraction across all load types. This finding is consistent with those of other stroke studies reporting that



**Figure 4.**

Effect of load type by arm on normalized electromyography (EMG) impulses. Bar graphs show mean and standard error (SE) of the normalized full-wave-rectified EMG impulses for the agonist anterior deltoid (first row) and triceps (second row) muscles, shoulder coactivity (third row), and elbow coactivity (fourth row) by group and load type. Bars are ordered by load type (M=mass, E=elastic, and V=viscous) for the paretic (PAR [dark blue]), nonparetic (N-PAR [light blue]), and respective matched control (CON [white]) groups. Significant differences are indicated by asterisks: \* $P < .05$ . Note differences in the scaling of the y-axis.

participants with more motor impairment after stroke lacked the ability to individually coordinate joints within a limb and were unable to adapt their motor responses to various upper-extremity tasks.<sup>5,44-46</sup> Lum et al associated the presence of these abnormal synergies with strength imbalances,<sup>47</sup> suggesting an association between strength and coordination in this population.

Importantly, both paretic and nonparetic muscles used a higher percentage of their voluntary capacity to reach against various types of resistance, as evidenced by increased agonist muscle recruitment and co-contraction in most conditions. Thus, although our results are consistent with the earlier findings that paretic muscles are much less efficient in producing force, requiring more EMG activity to effectively move a load,<sup>4,6,7,47</sup> our results also implicate excessive co-contraction of the antagonist muscle<sup>10-12,48</sup> as potentially contributing to the clinical presentation of weakness during our dynamic task.

### Coactivity

Some investigators have suggested that abnormal co-contraction patterns represent a reduction in the number of muscle combinations or possible synergies available in a paretic limb after stroke,<sup>18</sup> reflecting impairments in both agonist muscle recruitment and antagonist muscle inhibition.<sup>49</sup> Our second hypothesis proposed that the levels of agonist-antagonist muscle co-contraction would be elevated in the paretic and nonparetic arms in response to the mass load, the only load that inherently required appropriately timed agonist and antagonist muscle activation.<sup>39</sup> Our dynamic task, combined with the different kinetic demands for the loads tested, required a measurement of co-contraction that was sensitive to the timing of agonist and antagonist muscle activation. Simply

measuring antagonist muscle activation levels may contribute to erroneous conclusions about the presence or absence of co-contraction; it is not just *whether* antagonist muscles are on but *when* they are on that is critical. Thus, a unique contribution of this pilot study is the introduction of a temporally sensitive method of quantifying co-contraction. On the basis of this analysis, we were able to demonstrate and support our hypothesis that both the paretic and the nonparetic arms would exhibit a significant amount of co-contraction not observed in the control arms.

Specifically, we found that the nonparetic shoulder showed a significantly higher level of co-contraction with the elastic load than with the mass load but not the viscous load. A *post hoc* analysis (*t* tests) confirmed that the level of co-contraction of the nonparetic shoulder was significantly higher than that of the control shoulders for the elastic load ( $P=.05$ ), but the levels of co-contraction for the viscous and mass loads were lower and comparable to those of the control shoulders ( $P=.09$  and  $P=.68$ , respectively). Therefore, the nonparetic arm showed more flexible motor strategies than the paretic arm but this variable level of co-contraction is not consistent with the consistently minimal co-contraction seen in the control arms. We believe that the higher level of co-contraction for the elastic load may reflect the need for large stabilization forces at the end of reach for the elastic load.<sup>13,50</sup>

### The “Unimpaired” (Nonparetic) Arm Also Is Affected

Consistent with the growing body of evidence that the “unimpaired” limb also shows subtle motor impairments after stroke,<sup>30,31</sup> we also found significant differences between the nonparetic and control arms in all EMG outcome measures except anterior deltoid muscle recruitment

(Fig. 3). Lower baseline strength is consistent with deficits in isometric torque production reported by other authors.<sup>28,29</sup> More sensitive kinematic and kinetic studies have consistently demonstrated motor control deficits in the “less paretic” limb after stroke, such as the impaired muscle timing represented in the present study by high levels of co-contraction.<sup>51</sup> Such deficits call into question the use of the nonparetic arm as a matched control for research or clinical practice.

### Clinical Applications and Future Studies

Because both muscle weakness and co-contraction correlate significantly with motor impairment and disability,<sup>52</sup> the results of the present study may have important implications. There is very little literature suggesting effective treatment strategies for temporal coordination deficits after stroke. With mounting evidence supporting the use of resistive strength training to reduce impairments after stroke,<sup>15,16</sup> the logical extension of the present study is to determine whether training with a particular type of resistance can preferentially benefit both muscle recruitment and coordination.

In our study, muscle activation patterns (our indicator of coordination) were specific to our task and the kinetic demands imposed on the limb by different resistive loads for both the nonparetic and the control arms. Viscous loads, in particular, appeared to place demands on the muscle that resulted in higher levels of muscle activation with less co-contraction than did elastic or mass loads. However, the paretic arm responded with high levels of muscle activation and co-contraction across all load types. Future intervention studies could investigate whether strengthening exercises with viscous loads are more effective than those with other loads for developing

strength without co-contraction after stroke.

Some investigators have suggested that weakness in the paretic elbow musculature reveals a strong task dependence that is attributed to abnormal synergy between the elbow and the shoulder muscles<sup>43</sup> and that can be modified with training.<sup>46</sup> On the basis of the specificity of the training literature,<sup>40,41,53</sup> one could extrapolate that practice accelerating and decelerating mass loads might facilitate the generation of more appropriately timed agonist and antagonist muscle activation, elastic loads might foster stability, and viscous loads might preferentially facilitate agonist muscle recruitment with minimal co-contraction. Lum et al<sup>54</sup> found that participants with stroke showed improvements in agonist muscle EMG amplitude and work output and reductions in force direction errors after guided reaching against robotically simulated viscous loads. However, the support provided during the guided reaching task reduced the need to accommodate the mass of the limb, altering the dynamic requirements of the reaching task. Cirstea et al reported that a single session spent practicing arm pointing movements led to improved elbow and shoulder muscle timing in subjects who had had a stroke and had mild to moderate levels of functioning.<sup>42</sup> Given that the arm itself acts predominantly as a mass load during reaching tasks, such findings may provide mechanistic support for randomized controlled trials that have demonstrated the effectiveness of task-specific training in people after stroke.<sup>55</sup> Further investigation is needed to determine which type of resistive training might best help subjects acquire more appropriate motor responses after stroke.

Although the optimal treatment has yet to be identified, there is a grow-

ing awareness of the need to address both strength and coordination in rehabilitation paradigms.<sup>43</sup> For example, Sullivan et al<sup>21</sup> used a combination of task-specific training and isotonic strengthening of the legs to improve ambulation; that strategy positively affected both impairment and function, with improvements continuing even 6 months later. Using a different exercise sequence, Patten and colleagues<sup>22</sup> evaluated an upper-extremity hybrid intervention comprising both resistance training and functional task practice and reported strength gains with increased EMG activation and marked improvement in all clinical and functional measures. Although further research along these lines is needed, our pilot study is a preliminary step in developing a method for directly comparing different types of resistive loads and evaluating their effects on motor control.

### Study Limitations

The present study had a few minor limitations. The side on which the lesion was located was not homogeneous across participants (8 of 10 had right-side cerebrovascular accidents). Despite the fact that our participants' Fugl-Meyer Motor Assessment scores fell within the moderate impairment category,<sup>12</sup> our participants had with a fairly wide range of impairment levels within that category. Given that muscle activation patterns may be related to the level of residual arm function,<sup>11,12,56,57</sup> these variables should be more tightly controlled in future work. Finally, although the clinically based loads were specifically chosen for their validity, matching these load triplets to each participant's strength was limited to the weight increments available. Robotic techniques have already shown potential in stroke rehabilitation for the arm<sup>19,58</sup> and would provide a means for more precisely matching resistive loads to individual abilities.

### Conclusion

The present study provides preliminary information on the effects of reaching against equivalent mass, viscous, and elastic loads on the muscle activation patterns of the paretic and nonparetic arms of people who have had a stroke. Of the 3 load types, only the viscous load resulted in increased activation with minimal co-contraction in the nonparetic shoulder. Because of the motor control deficits (including upper motor neuron weakness and co-contraction) in the paretic arm, muscle activation in that arm was less efficient and did not differ across the load types. In contrast, muscle activation patterns did differ by load type in the control and nonparetic arms. Consistent with previous reports, the nonparetic arms showed impairments in muscle activation that might not be readily detected with clinical motor control assessments.

The significance of this pilot study is that it revealed anticipated differences in muscle activation and co-contraction by load type that were not distinguishable in the hemiparetic arm. However, the nonparetic arm provided a model of mildly impaired motor response that might be more sensitive to investigations of intervention efficacy. Future studies should be conducted to determine intervention effectiveness before the initiation of an intervention trial.

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Dr Stoeckmann and Dr Scheidt provided concept/idea/research design, project management, and fund procurement. All authors provided writing and data analysis. Dr Stoeckmann provided data collection and participants. Dr Scheidt provided facilities/equipment. Dr Sullivan and Dr Scheidt provided consultation (including review of manuscript before submission).

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## Poststroke Muscle Recruitment and Co-contraction During Reaching

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