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Strategies used to stabilize the elbow joint challenged by inverted pendulum loading

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Abstract

The stiffness of activated muscles may stabilize a loaded joint by preventing perturbations from causing large displacements and injuring the joint. Here the elbow muscle recruitment patterns were compared with the forearm loaded vertically (a potentially unstable inverted pendulum configuration) and with horizontal loading.

Eighteen healthy subjects were studied with the forearm vertical and supinated and the elbow flexed approximately 90°. In the first experiment EMG electrodes recorded activity of biceps, triceps, and brachioradialis muscles for joint torques produced (a) by voluntarily exerting a horizontal force isometrically (b) by voluntarily flexing and extending the elbow while the forearm was loaded vertically with 135 N. The relationship between the EMG and the torque generated was quantified by the linear regression slope and zero-torque intercept. In a second experiment a vertical load increasing linearly with time up to 300 N was applied.

In experiment 1 the EMG–torque relationships for biceps and triceps had an intercept about 10% of maximum voluntary effort greater with the vertical compared to the horizontal force, the inverse was found for Brachioradialis, but the EMG–torque slopes for both agonist and antagonistic muscles were not different. In experiment 2 there were 29 trials with minimal elbow displacement and all the three muscles activated on the order of 11% of maximum activation to stabilize the elbow; 19 trials had small elbow extension and 14 trials small flexion requiring altered muscle forces for equilibrium; 7 trials ended in large unstable displacement or early termination of the test. An analysis indicate that the observed levels of muscle activation would only provide stability if the muscles' short-range stiffness was at the high end of the published range, hence the elbow was marginally stable. The stability analysis also indicated that the small elbow extension increased stability and flexion decreased stability.

Keywords: Upper extremity; Elbow; Joint instability; Muscle force

1. Introduction

Although much is known about how muscles maintain static equilibrium of joints, little is known about how they maintain stability. A joint or limb segment is unstable when a small (or virtual) disturbance from a position of static equilibrium produces large displacements and potentially injurious deformations. The spine and the digits are examples of potentially unstable joints that resemble a series of inverted pendulums. The restoration of equilibrium after a perturbation can be achieved by active (CNS-mediated) adjustment of muscle tensions, but with inherent neuromuscular delays. Alternatively, small perturbations might be accommodated without such active responses, provided there is sufficient muscular stiffness and damping. Muscle stiffness increases with activation, so coactivation of agonistic and antagonistic muscles can be used to increase a joint's stability. The biomechanics of such passive stabilization that requires no active CNS-mediated adjustment of the preset muscle force or stiffness following a perturbation was demonstrated theoretically for the trunk by Bergmark (1989), and has been further explored by Crisco and Panjabi (1991), Crisco and Panjabi (1992), Cholewicki and McGill (1996), Gardner-Morse et al. (1995), Milner et al. (1995).

In complex systems such as the spine it is difficult to investigate joint stabilizing mechanisms experimentally. This paper reports experiments to determine the role of muscles in stabilizing the elbow when loaded to make the forearm potentially instable in an inverted pendulum configuration. The elbow was chosen because it could be

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considered as a simple single-degree-of-freedom system. Inverted pendulum loading was chosen because the external load produces no torque about the joint, and therefore no muscle activation is required to maintain equilibrium. Any muscle activation that is present can be interpreted as a joint-stabilizing action. In the first experiment, we recorded the muscle activation pattern when the forearm was loaded by an external torque produced by a vertical force, and compared it with that when the torque was caused by a horizontal force. The null hypothesis was that the pattern of elbow muscle activation would be the same under both conditions. In a second loading experiment, a vertical load, increasing linearly with time up to 300 N was applied to the forearm via a wrist splint. This experiment was designed to measure the muscle activation and other strategies used by subjects to maintain stability of the elbow and forearm when loaded with no torque about the joint, but in a potentially unstable configuration. Finally, an analytical model was employed to determine the degree of muscle stiffness required for elbow joint stability with muscle forces based on the measured muscle activation of the subjects.

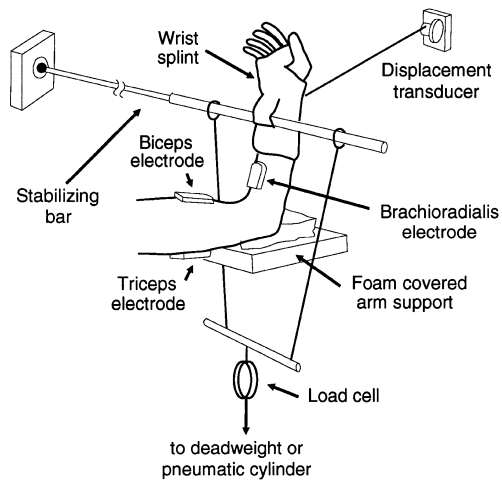

Fig. 1. Diagrammatic representation of the apparatus used to load the forearm vertically. The loading is transferred from the deadweight or the pneumatic cylinder via the load cell and cables to the wrist splint. EMG electrodes were placed over three elbow muscles.

2. Methods

Eighteen healthy subjects (10 female, 8 male) were studied with each arm in turned positioned in an apparatus with the elbow in approximately 90° flexion and the forearm vertical and supinated. The method of applying vertical load at the wrist via a custom-molded plastic splint bound to the wrist with elastic bandages is illustrated in Fig. 1. The forearm was constrained to move only in parasagittal plane by the use of a 1 m long stabilizing bar connected to a ball and socket joint at a nearby wall. For experiment 1, subjects (1) voluntarily exerted maximum isometric extension and flexion efforts against a measured horizontal resistance force, (2) voluntarily flexed and extended their elbow while the forearm was loaded vertically with a deadweight of 135 N via cables attached to a splint at the wrist. For experiment 2, a vertical load increasing linearly with time up to 300 N was applied to a wrist splint. Each arm (right and left) of the subjects was tested twice in each loading configuration. Experiments and procedures were approved by the University of Vermont Committee on Human Research.

For the first (horizontal force) test condition subjects were asked first to push (extension effort) then pull (flexion effort) against a stirrup. A load cell measured the magnitude of the horizontal force generated. Subjects were instructed to increase the force up to their maximum in about 5 s, then to release the force slowly over a further period of about 5 s. The generated force was multiplied by the measured distance from the wrist to the elbow joint to measure the torque generated at the elbow joint.

For the second (vertical deadweight load) test subjects were first asked to find the natural position in which the deadweight load did not generate any perceived flexion or extension torque. This was the reference position in which theoretically no muscle action was required for joint equilibrium because the external load produced no torque about the elbow. Subjects then voluntarily flexed and extended the elbow from a reference position about 20° in each direction to produce a torque about the elbow due to the vertical load. Horizontal displacement of the wrist was measured by a displacement transducer consisting of a multi-turn potentiometer with a string wrapped around its axle, and a coiled return spring. This transducer had a resolution of 0.2 mm, and frictional/elastic hysteresis of ± 1 mm. The horizontal movement of the wrist was then multiplied by the applied load to give the flexion or extension torque at the elbow.

For the third (ramped vertical load) test a vertical load that increased linearly with time up to 300 N in 30 s was applied to the wrist splint via cables in series with a spring connected to a pneumatic cylinder into which compressed nitrogen was bled through a valve (Fig. 1). The maximum load (300 N) was controlled by an appropriate setting of the pressure regulator on the nitrogen gas supply bottle, and the rate of load increase was set by adjusting the bleed valve. The subject was instructed to maintain the reference position of the forearm while the increasing load was applied. The applied force was recorded simultaneously by means of a load cell in the cable. Any horizontal displacement of the wrist was again recorded by the displacement transducer. The load could be released prematurely by a safety switch that exhausted

the pressure in the pneumatic cylinder. The subject held one such switch in the contralateral hand, and a second switch could be operated by the investigator. As an additional safety measure, there was a soft-stop rest for the forearm to prevent excessive movement. The end of each test was defined by either the load reaching 300 N, or the subject's arm touching a padded stop, or the voluntary termination of the test.

Electromyography (EMG) was used to record and quantify the activity of the three major muscles (biceps, triceps, and brachioradialis) considered as flexors or extensors of the elbow in the forearm supinated position. Pre-amplified, isolated EMG surface electrodes (Motion Control, Salt Lake City, Utah) were placed over the bellies of these muscles. The EMG signals, together with the signals from the force and displacement transducers were recorded by a digital data logging system consisting of an analog to digital converter (Computer Boards, Type CBI-1602) interfaced to a personal computer running custom software. The sampling frequency was 1024 Hz. Subsequently the root-mean-square (RMS) of the EMG signals was computed with a moving window of 400 ms width.

In the first two loading configurations (Experiment 1) the relationship between the EMG and the torque generated appeared linear for all muscles (e.g. Fig. 2). Therefore, the linear regression relationship between RMS-EMG (arbitrary volts) and the torque at the elbow joint (N m) was calculated, and the zero-torque intercept (RMS-EMG) and slope (RMS-EMG/N m) were noted. Antagonistic coactivation of each flexor muscle was quantified as its RMS-EMG/torque slope for an extension torque divided by that for a flexion torque and multiplied by 100 to express it as a percentage, and vice versa for the extensor (triceps) muscle. To test the hypothesis that the muscle activation patterns were the same for torques caused by horizontal and vertical loading, the zero-torque intercepts, slopes, and percent antagonistic activation were compared for each muscle. These comparisons were made by using repeated measures analyses of variance (General Linear Models Procedure, SAS Institute Inc., Cary NC, USA). The repeated factors were first or second trials, left and right limb, and loading condition (horizontal or vertical load).

For the second experiment (ramped vertical load), each trial was first categorized as belonging to one of four groups, according to whether subjects displaced the limb: Group 1: no measurable displacement of the elbow joint (i.e. less than 5 mm displacement at the wrist); Group 2: small extension displacement (5–10 mm) of the wrist; Group 3: small flexion displacement (5–10 mm) of the wrist; Group 4 large unstable displacement (more than

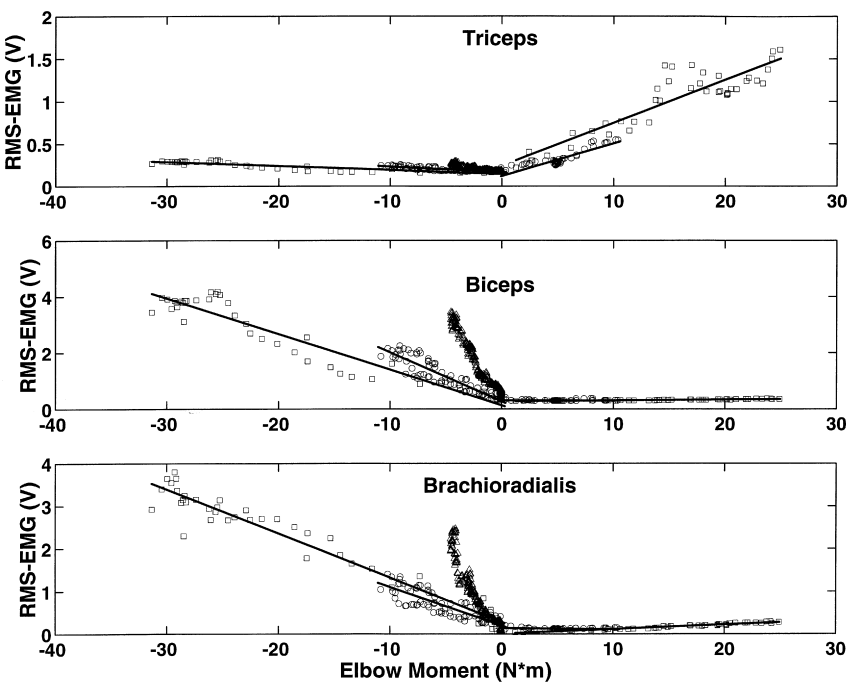

Fig. 2. Examples of RMS-EMG of the three muscles Brachioradialis, Biceps and Triceps plotted against the generated torque (in N m) about the left elbow of one subject (increasing and subsequently decreasing torque recordings). (□) for a horizontal force maximum effort trial; (○) for a vertical load flexion/extension trial; (△) for a ramped vertical load experiment. The straight lines are linear regressions fitted to the sampled data. The sign convention was positive torques tending to extend the elbow. The temporal separation of points on the graphs in 10 ms. In this case the linear regressions are similar for each muscle for the horizontal force maximum effort trial and the vertical load flexion/extension trial. However, the magnitude of the EMG in the ramped vertical load experiment for these three muscles was between 3.4 and 4.4 times greater compared to that for the other two trials, indicating an overall higher level of muscle coactivation.

10 mm at the wrist) and/or early termination of the test. The maximum RMS-EMG signals produced during each of these trials were expressed as a proportion of the maximum value from the same muscle in the horizontal force trials.

A static analytical model of the elbow joint and three of its muscles was used to investigate the required muscle stiffness for stability of the forearm loaded with 300 N at the wrist in an inverted pendulum configuration. The geometry of the model was defined by the positions of the point of application of load and the origins and insertions of three muscles (biceps, triceps and brachioradialis) all relative to an origin of axes placed at the elbow joint. These coordinates were derived from the data given by An et al. (1981) and listed in Table 1 for the vertical forearm position. These values gave moment arms of the muscles that were similar to those in Murray et al. (1995).

The potential energy (PE) of this modeled system was calculated for small displacements from the equilibrium position. If the total PE increased with displacement, the elbow joint was considered to be stable. In this analysis two components of the PE were considered: (1) the imposed force P multiplied by its vertical height h, (2) the elastic energy stored in or released by length changes δL of the muscles, considered as Hookian springs representing the muscle short-range stiffness k_m. For each muscle this is equal to the area under the load deformation curve, which is the muscle force F_m multiplied by the length increment δL, plus the force increment multiplied by the length increment.

Hence

$$PE = Ph + \sum_{1}^{3} (F_m \, \delta L + \tfrac{1}{2} k_m \delta L^2)$$

Short-range muscle stiffness k was calculated as a function of muscle force F_m and initial length L using the equation $k = qF_m/L$ (Bergmark, 1989), where q is a dimensionless multiplier. The vertical height h of the load P was $\lambda \sin(\Theta)$ where Θ is the angular displacement of the forearm, length λ, which was set to 200 mm. The length changes of muscles δL were calculated as the change in the straight line origin to insertion distance in this small displacement model, ignoring any possible large displacement effects such as wrapping of muscles over bony structures.

The amount of activation of the flexor muscles in these analyses was based on the averaged values of the RMS-EMG (as a percent of maximum) in the ramped vertical load experiments (Table 3). The average activations were multiplied by the specific tension (maximum force per unit area) for each muscle, which was 0.7 N/mm^2 for triceps and 1.0 N/mm^2 for flexors. These specific force values were based on values given by Buchanan and Shreeve (1996), and were selected because they were compatible with elbow joint moment equilibrium using the experimentally determined muscle activation levels. To achieve moment equilibrium in the vertical arm position, first the activations of biceps and brachioradialis were set to the mean values found in the ramped vertical load experiments with vertical arm position (Group 1 in Table 3). Then the activity of triceps that was compatible with elbow moment equilibrium in the model was then calculated. Using the specific tensions 0.7 N/mm^2 for triceps and 1.0 N/mm^2 for the flexors, the calculated triceps activation was close to the mean derived from EMG measurements (10.4 and 11.2%, respectively). For modelling the flexed and extended elbow positions (Groups 2 and 3 simulations) the muscle activations were all specified based on the mean experimental values in Table 3. Then the joint angle that gave moment equilibrium was calculated by first calculating the joint torque due to the muscle forces, then finding the joint angle at which the vertical load (300 N) generated an equal and opposite torque. The muscle stiffness parameter q was treated as a variable, and the critical value necessary for stability was found empirically (see Fig. 3).

3. Results

Tests were made of 18 subjects, with two trials of each arm, yielding potentially 72 trials for each test condition. However, because of EMG electrode problems there

Table 1
Coordinates of applied loads and muscle attachments (in mm) and muscle areas used in the analytical model for the neutral position (forearm vertical). (Origin of axes = elbow joint center; x = horizontal; y = vertical)

	Muscle origin (forearm)		Muscle insertion (humerus)		Area (mm^2)	Specific max. tension (MPa)
	x(mm)	y(mm)	x(mm)	y(mm)		
Biceps	5	32	− 145	32	460	1.0
Triceps	19	− 19	− 65	− 19	1880	0.7
Brachio-radialis	− 51	174	− 51	10	150	1.0
Applied load position	0	200				

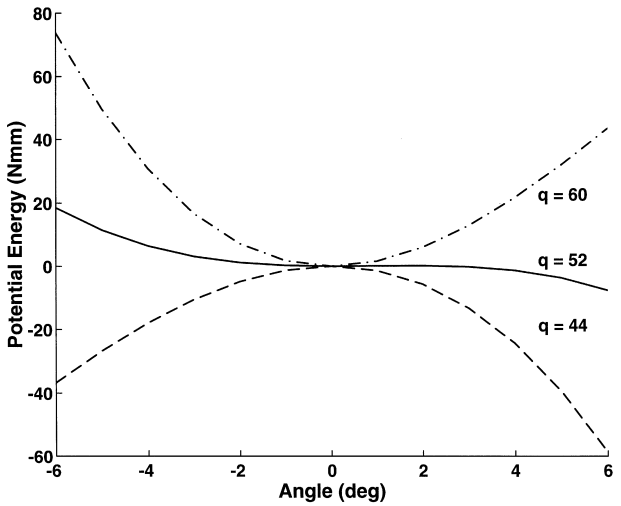

Fig. 3. Examples of calculated potential energy of the elbow when supporting a vertical load of 300 N with vertical forearm for three different values of the muscle stiffness parameter $q = 44$(– – –), $q = 52$(———) and $q = 60$(– · – · –). The elbow is stable for $q = 60$, metastable for $q = 52$ and unstable for $q = 44$.

were 68 horizontal force trials, 69 vertical force flexion–extension trials and 69 ramped vertical force trials with usable data.

In the comparison of the EMG–torque relationships for torques produced by horizontal and vertical loads respectively (experiment 1), the EMG–torque relationships for biceps and triceps had a greater intercept (by about 10% of maximum voluntary activation) in test condition 2 than 1; the inverse was found for Brachioradialis (Table 2). These differences by test condition were significant ($p < 0.01$) in the repeated measures ANOVA, but the other repeated factors (limb, trial) were not significant factors. There were no significant differences between the agonist EMG–torque slopes for horizontal or vertical loading states for any of the three muscles. Also the antagonistic muscle activations (defined as the percentage of the EMG–torque slopes for antagonistic compared to agonistic conditions) were not different, although the standard deviations were greater in the vertical loading condition. In Table 2, torques tending to extend the elbow were considered positive, so the EMG–torque slopes are negative in Table 2 for flexion torques.

For the second experiment with ramped vertical load, there were 29 trials in Group 1 (no measurable displacement of the elbow joint), 19 trials in Groups 2 (small extension displacement of the elbow) and 14 trials in Group 3 (small flexion displacement of the elbow). There were 7 trials in Group 4 (large unstable displacement and/or early termination of the test). The muscle activations, expressed as the maximum EMG in each trial and normalized as the percentage of the maximum value observed in any test (normally a horizontal force, maximum effort test) differed according to the Group (Table 3). There was a reciprocal relationship according to the limb motion for the flexor muscles, whose mean activation was 12.3% (biceps) and 10.2% (brachioradialis) in trials in Group 1 'no motion', and the activation increased to 27.5% (biceps) and 26.1% (brachioradialis) in trials in Group 2 'extension'. It was 2.1% (biceps) and 3.9% (brachioradialis) in trials in Group 3 'flexion'. There were significant differences ($p < 0.05$ by ANOVA) between groups for biceps and for brachioradialis, but not for triceps muscle, whose mean activation was 11.2, 10.3 and 13.8%, respectively, for Groups 1,2 and 3. The same pattern was found, including significant differences, in the EMG values prior to normalization by the maximum recorded values.

For the analytical stability model simulations of the flexed and extended elbow positions (Groups 2 and 3 simulations) the joint angle that gave a triceps activation equal to the experimentally observed value was 3.2° for Group 2 (extension) and 2.6° for Group 3 (flexion). The critical values of the short-range stiffness parameter

Table 2
Means (and standard deviations) of the slopes and intercepts of linear regressions between agonist muscle EMG and elbow torque, and antagonistic activation expressed as antagonistic slope as a percentage of agonistic slope. EMG was measured in arbitrary volts. Positive torques tend to extend the elbow

		Intercept		Slope		Antagonistic slope ratio (%)	
		Horizontal force trials (V)	Vertical force trials (V)	Horizontal force trials (V/Nm)	Vertical force trials (V/Nm)	Horizontal force trials	Vertical force trials
Triceps	Mean	− 0.010	0.194	0.055	0.047	− 6.54	− 7.08
	(SD)	(0.201)	(0.106)	(0.031)	(0.037)	(6.01)	(23.3)
Biceps	Mean	0.171	0.325	− 0.059	− 0.058	− 7.01	− 6.18
	(SD)	(0.233)	(0.157)	(0.037)	(0.066)	(5.48)	(32.2)
Brachio-radialis	Mean	0.231	0.069	− 0.052	− 0.058	− 14.7	− 12.50
	(SD)	(0.209)	(0.177)	(0.031)	(0.064)	(11.1)	(16.8)

Table 3
Percentages of maximum activation of each of the three muscles during the ramped vertical load trials; mean (and SD) for each Group

	Group 1 no motion	Group 2 extension	Group 3 flexion
Biceps	12.3 (± 23)	27.5 (± 36)	2.1 (± 2)
Triceps	11.2 (± 19)	10.3 (± 12)	13.8 (± 11)
Brachio-radialis	10.2 (± 19)	26.1 (± 31)	3.9 (± 3)

q (i.e. the value that gave metastable equilibrium) were 52 for Group 1 (no displacement), 30 for Group 2 (extension) and 70 for Group 3 (flexion) (see Fig. 3). The value of q found this way was quite sensitive to the assumptions of the model, and values in the range 40–80 at vertical forearm position could be found for plausible values of the geometric and other variables in this simplified model.

4. Discussion

The muscle activation patterns measured by the intercepts and slopes of the RMS–EMG/torque relationships showed that inverted pendulum vertical loading was associated with greater pre-activation of the biceps and triceps muscles (as measured by the intercepts) than the horizontal force condition. There was no significant difference between the slopes for vertical and horizontal load conditions of either agonists or antagonists. Vertical load challenges elbow stability, so it was expected that it would require more muscular coactivation. The absence of difference in the slopes indicates that the muscle activation strategy at the elbow that generated torque was independent of the external force direction once the initial pre-activation was set.

The results of the experiments with ramped vertical loading confirmed that this loading state is potentially unstable, since some subjects did indeed become unstable in the ramped vertical load tests, and others apparently adopted strategies that helped them to stabilize their elbows. Although some subjects (29 trials) maintained their forearms vertical in these tests, many others (43 trials) responded by making a small angular displacement of the forearm, despite the prior instruction given. The variability of these responses between and within subjects points to the possibility that these differing responses were a result of variations in the initial position of the forearm at the start of the test, despite instructions to subjects to start at the vertical forearm 'neutral position' they had found subjectively. Those subjects who maintained a vertical forearm (Group 1) coactivated all three muscles on the order of 11% of maximum activation to stabilize the elbow. In the trials with extension or flexion of the elbow (Groups 2 and 3), the triceps activation did not change significantly; instead the activity of the flexor muscles was increased and decreased, respectively. This suggests that the response was achieved through a command that modulated flexor muscle activity only, with the consequence that in flexed arm positions the overall level of muscle coactivation was reduced. This reduction in coactivation was associated with lowered stability.

It should be noted that the levels of antagonistic activity recorded were probably overestimated because of 'crosstalk' between recording electrodes (De Luca and Merletti, 1988; Koh and Grabiner, 1992; Zhou et al., 1996). In Groups 1 and 2 small angular displacements produced small muscle length changes and small displacements of the electrodes relative to the muscles, but these were considered negligible because the angular displacement were so small compared to the range of motion of the elbow.

For the horizontal force trials, subject voluntarily controlled force magnitude whereas in the vertical force flexion/extension trials it was the displacement that they controlled. The latter may have caused greater cocontraction since there is evidence that the pattern of elbow muscle activation differs in these two conditions (Tax et al., 1989; Buchanan and Lloyd, 1995), with greater cocontraction when opposing an elastic load, compared to isometric conditions (Theeuwen et al., 1994). By examining the EMG/torque relationship, and quantifying both its intercept and slope, this study points to an initial increase in coactivation, rather than a change in subsequent recruitment as the significant difference when joint stability was challenged.

The analytical model indicated that the level of muscle activation observed in Groups 1, 2 and 3 would only provide stability with short-range stiffness parameter q at the high end of the experimentally reported physiological range given by Crisco and Panjabi (1990). Those subjects who extended or flexed their forearm (Groups 2 and 3) required altered muscle activation for equilibrium. Group 2 subjects (who extended the elbow creating an extension moment of the vertical load) increased flexor (agonist) activation only, whereas Group 3 subjects (who flexed the elbow) decreased antagonist (triceps) activity with only a small, not statistically significant increase in agonist activity. According to the analytical model, this failure to increase antagonistic activity in the flexed arm position made the elbow potentially more unstable (as evidenced by the increase in the critical value of muscle stiffness). Apart from the anatomical assumptions in this model, these findings are also subject to the assumption that muscle short range stiffness is a linear function of activation and that short-range stiffness is responsible for joint stabilization.

Overall, the findings of these investigations indicate that there are certain joint loading conditions in which the muscle activation patterns are different from those

required for equilibrium, to provide adequate stiffness to stabilize the joint. For the vertically loaded forearm, there was an increase in the initial muscle activation rather than an alteration of the subsequent relationship of muscle activation to increasing joint moment, compared to the more stable horizontal load case.

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