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Novel muscle patterns for reaching after cervical spinal cord injury: a case for motor redundancy

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Abstract A fundamental issue in the neuromotor control of arm movements is whether the nervous system can use distinctly different muscle activity patterns to obtain similar kinematic outcomes. Although computer simulations have demonstrated several possible mechanical and torque solutions, there is little empirical evidence that the nervous system actually employs fundamentally different muscle patterns for the same movement, such as activating a muscle one time and not the next, or switching from a flexor to an extensor. Under typical conditions, subjects choose the same muscles for any given movement, which suggests that in order to see the capacity of the nervous system to make a different choice of muscles, the nervous system must be pushed beyond the normal circumstances. The purpose of this study, then, was to examine an atypical condition, reaching of cervical spinal cord injured (SCI) subjects who have a reduced repertoire of available distal arm muscles but otherwise a normal nervous system above the level of lesion. Electromyography and kinematics of the shoulder and elbow were examined in the SCI subjects performing a center-out task and then compared to neurologically normal control subjects. The findings showed that the SCI-injured subjects produced reaches with typical global kinematic features, such as straight finger paths, bell-shaped velocities, and joint excursions similar to control subjects. The SCI subjects, however, activated only the shoulder agonist muscle for all

directions, unlike the control pattern that involved a reciprocal pattern at each joint (shoulder, elbow, and wrist). Nonetheless, the SCI subjects could activate their shoulder antagonist muscles, elbow flexors, and wrist extensor (extensor carpi radialis) for isometric tasks, but did not activate them during the reaching movements. These results demonstrate that for reaching movements, the SCI subjects used a strikingly different pattern of intact muscle activities than control subjects. Hence, the findings imply that the nervous system is capable of choosing either the control pattern or the SCI pattern. We would speculate that control subjects do not select the SCI pattern because the different choice of muscles results in kinematic features (reduced fingertip speed, multiple shoulder accelerations) other than the global features that are somehow less advantageous or efficient.

Keywords Electromyography · Multijoint arm movement · Kinematics · Neurological disorders

Introduction

The nervous system demonstrates an amazing ability to select and quickly adjust muscle activities to meet the requirements of multijoint movement (Cooke and Virji-Babul 1995; Dounskaia et al. 1998; Gribble and Ostry 1999; Koshland et al. 2000; Pigeon et al. 2003; Sabes 2000; Sainburg et al. 1995; Shadmehr and Mussa-Ivaldi 1994). What is less known is whether the nervous system makes use of available, redundant muscles by selecting uniquely different muscle patterns to deal with the mechanics of the arm. Historically, Bernstein established the classic issue that there are redundant degrees of freedom inherent in the neuromuscular plant (Bernstein 1967, 1996; Soechting and Flanders 1991), such that there are multiple joint and muscle solutions for a given arm movement. Since then, many studies have demonstrated kinematic invariances (for instance,

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straight hand paths, planar motion, negligible wrist motion) that would reduce the degrees of freedom and so the choice of muscles would become restricted (Admiraal et al. 2002; Atkeson and Hollerbach 1985; Koshland et al. 2000; Morasso 1981). On the other hand, there are multiple synergistic muscles, as well as various combinations of agonist/antagonists, that can contribute to the same net joint torque (Buchanan et al. 1986; van Bolhuis and Gielen 1999). This motor redundancy suggests that multiple muscle solutions are possible for the same kinematic movement. Computer simulations have demonstrated a theoretical range of muscle or torque solutions (Au and Kirsch 2000; Hirashima et al. 2003a; Koshland et al. 1999), but cannot show that the nervous system actually uses alternative muscle patterns.

One way to demonstrate the nervous system's use of different muscle patterns could be to show a change in selected muscles for a given set of kinematics. Latash and Jaric (1998) have shown that different patterns of muscle activation at the elbow or wrist can be used to produce the same isometric force at the hand when subjects are given explicit instructions to change the primary muscle group. However, under everyday circumstances and during actual movement, neurologically normal subjects do not change their selection of muscles. That is, subjects demonstrate the same choice of muscles for movement to any given direction; for instance, all subjects initiate and accelerate a movement to the same direction with the same shoulder, elbow, and wrist agonists, and decelerate the movements with the same antagonist muscles (Almeida et al. 1995; Buneo et al. 1994; Gribble and Ostry 1999; Karst and Hasan 1991; Koshland et al. 2000). In fact, any trial-to-trial or intersubject differences that occur are revealed as small variations in timing and amplitude of the muscles. The fact that there are these consistent findings does not negate the possibility that the nervous system has the capacity for alternative muscle patterns but suggests that the nervous system may not employ alternative muscle patterns unless there are unusual circumstances.

Studies of patients who have recovered from neurological insult offer the opportunity to investigate how the selection of muscles can be reorganized under the pressure to regain arm function. Indeed, changes in muscle activations and muscle torques have occurred after cerebral vascular accidents and cerebellar damage, but kinematic features were also altered and abnormal, such as inaccurate and curved finger paths, inaccurate endpoint positions, and multi-peaked fingertip velocity profiles (Bastian et al. 2000; Beer et al. 2000; Topka et al. 1998). Another patient population, subjects with cervical spinal cord injury (SCI) at C5–C6, could provide an alternative case for examining the choice of muscles, and they were used in this study. For the SCI population, the arm is still subject to mechanical interactions, but the person has a reduced repertoire of muscles to call upon to deal with the mechanics. That

is, the classic SCI patient with a complete C5–C6 lesion retains the ability to activate most shoulder muscles, but can only activate elbow flexors and one wrist extensor muscle. Nonetheless, studies of arm motions in SCI subjects have shown that kinematic features were generally similar to non-injured subjects except for minor changes, such as increased scapula winging and slower speeds (Acosta et al. 2001; Kulig et al. 2001; Laffont et al. 2000; Reft and Hasan 2002; Sarver et al. 1999). These results imply that, in order to achieve normal kinematics, SCI subjects must change their muscle pattern more than simply using a normal pattern that has missing muscles. Some studies investigating isometric strength tests (Gronley et al. 2000; Zerby et al. 1994), single joint elbow movements (Wierzbicka and Wagner 1992, 1996), and reaching (Marciello et al. 1995) have proposed that SCI subjects used alternative muscle patterns. For example, Gronley et al. (2000) showed that the C5–C6 SCI subjects produced an isometric elbow extensor torque, even though they could not activate the triceps muscle. They speculated that the SCI subjects employed a different pattern of muscles, such as use of shoulder muscles to create a passive elbow torque. Although these studies suggest alternative muscle strategies may exist, they did not specifically report alternative electromyography (EMG) patterns, and no study to date has comprehensively examined both muscle activities and kinematics in multijoint arm reaching with SCI subjects. As a result, the present study addressed two questions:

1. Are the kinematics of reaching movements of SCI subjects similar to control subjects?
2. Are the muscle patterns at shoulder and elbow joints during the reaching movements of SCI subjects different from control subjects? For the reaching task, all subjects performed the center-out task in the horizontal plane because the center-out task encompasses a full range of directions as well as many combinations of shoulder vs. elbow and flexor vs. extensor muscle activities.

For the C5–C6 SCI subject, the primary motor deficit is the lack of elbow extensor muscle force and torque. The question is: what predictions can be made for changes in intact muscle activities in the center-out task given this loss of elbow extensor muscles? Although triceps always creates an extensor torque at the elbow, the role of triceps activity varies across directions in the center-out task and can change from agonist to antagonist, shortening to lengthening contraction (Gribble and Ostry 1999; Karst and Hasan 1991; Koshland et al. 2000). For almost all directions of the center-out task, elbow muscles are important to resist or assist intersegmental effects arising from shoulder motion (Galloway and Koshland 2002; Galloway et al. 2004; Koshland et al. 2000). Hence, if elbow extensor torque were absent, one would predict one of two possibilities: (1) intact muscles could be activated as usual, and then kinematics, particularly at

the elbow, would be severely altered due to unmodified intersegmental effects, or (2) muscle activity, particularly at the shoulder, would be adjusted to reduce the intersegmental effects at the elbow, resulting in relatively normal kinematics. Based on empirical findings for EMG activities (Karst and Hasan 1991) and simulation work from this laboratory (Koshland et al. 1999) this latter option would predict differing increases vs. decreases in shoulder muscle activity for different regions of the center-out task. The findings from this study were closer to the second prediction, but were still surprising. That is, our results showed that the SCI subjects did adjust shoulder muscle activities in order to achieve some normal kinematic features. Surprisingly, the same adjustment (activating only shoulder agonist muscles) was utilized for all directions. Preliminary results of this study have been published in abstract form (Koshland and Galloway 1998).

Materials and methods

Subjects

Five adult male subjects with a diagnosis of complete cervical spinal cord lesion (25–37 years of age, 11–18 years post-injury), and four subjects without neurological or musculoskeletal injuries (25–37 years of age, two males and two females) participated under informed consent. Protocols were approved by the Institutional Review Board of the University of Arizona and conducted in accordance with the Helsinki Declaration. For the subjects with cervical SCI, motor and sensory deficits were consistent with a complete (class A) lesion at C5–C6, as outlined by the American Spinal Injury Association (Waters et al. 1991). This meant that subjects demonstrated normal strength for all shoulder motions and elbow flexion. Subjects were unable to activate elbow extensors (such as triceps muscles) as verified by EMG. Subjects could activate the wrist extensor muscle, extensor carpi radialis (ECR), and could extend the wrist against gravity but against minimal resistance only. Subjects were unable to activate any other wrist and finger muscles and could not produce any finger movement. Cutaneous sensation was intact following dermatome boundaries for C5 and sometimes part of C6. Proprioception was intact for shoulder, elbow, and wrist motions. Most subjects typically used anti-spasticity drugs, including a daily dose of baclofen and a nightly dose of Valium.

Task and kinematic measurement

Subjects performed point-to-point arm movements to targets in the horizontal plane, similar to previous reports on neurologically normal subjects (Koshland et al. 1999, 2000). That is, subjects sat in front of a table with

the dominant right arm supported by a mechanical apparatus, which rolled on the table. The apparatus allowed horizontal flexion and extension at the shoulder, elbow, and wrist joints. The hand was restrained in an orthoplast splint in a comfortable posture. All SCI subjects sat in their wheelchairs with a strap wrapped around their chest and the chair back. In addition, they rested their left forearm on the table to provide additional trunk stability. Both SCI and control subjects minimally moved the wrist joint and trunk segment during the reaching movements.

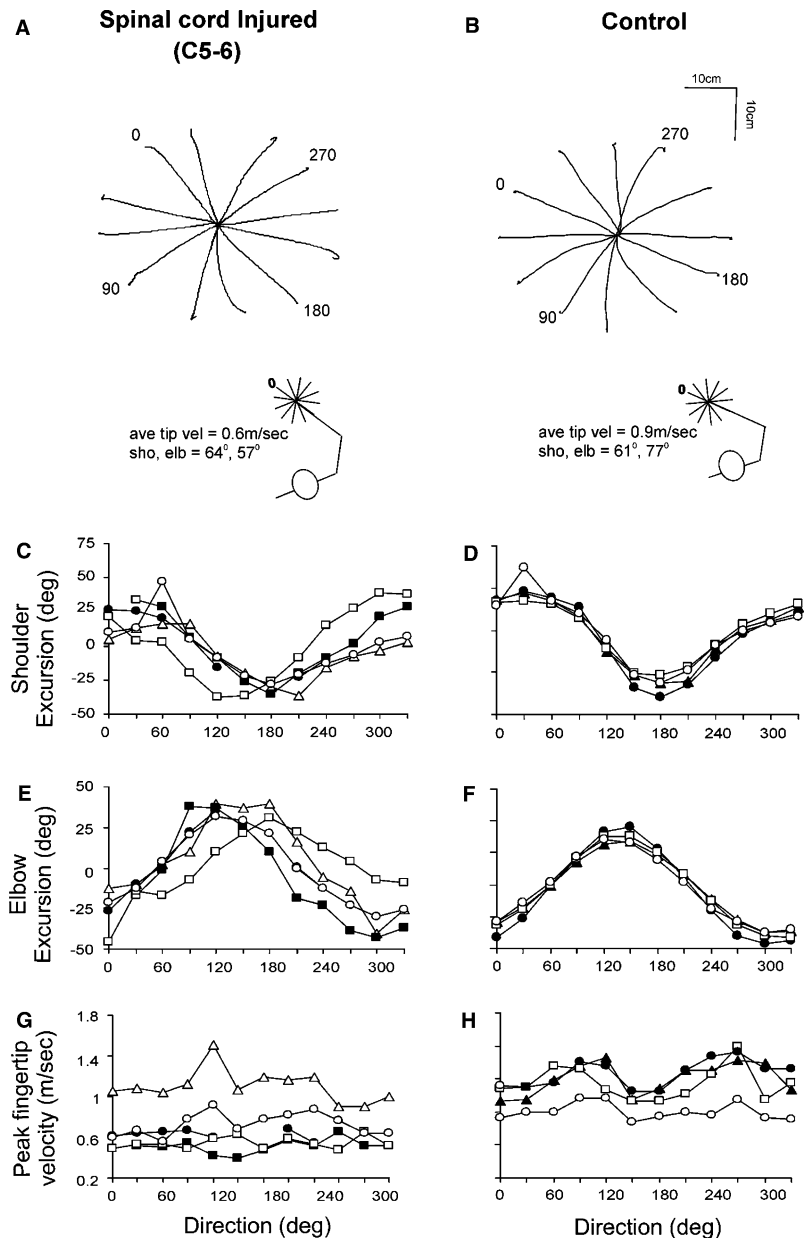
For the reaching movement, each subject was instructed to make one quick movement to each of 12 targets (20 cm distance) that were within arm's reach and evenly spaced at 30° intervals (Fig. 1A,B). The convention for the target direction was set at 0°, which was determined by a line extending from the forearm (and hand segment), and subsequent directions followed in a counterclockwise order. A small Plexiglas plate held above the table indicated target locations. A light-emitting diode (LED) inside the target plate was illuminated to indicate when to start movement towards the target. Subjects used a similar initial configuration of the arm. Initial shoulder and elbow positions were, on average, for the SCI subjects, $63 \pm 12^\circ$ and $72 \pm 14^\circ$, respectively (0° was full extension) and for the control subjects, $71 \pm 10^\circ$, $68 \pm 10^\circ$ (see stick figures in Fig. 1A,B). Previous reports have demonstrated that the choice of muscles and joint excursions follow rules based on the orientation of the target relative to the forearm, without significant effects from small differences in the initial arm configuration (Karst and Hasan 1991; Sainburg et al. 2003). Given this result and the 10–14° standard deviation in joint position of this study, the differences in initial arm configuration would not be expected to affect the EMG findings of this study. SCI subjects repeated three movements to each target, whereas control subjects repeated six movements to each target.

Reflective markers were placed at locations along the right arm of the subject (index finger, wrist, elbow, and shoulder) and on the left shoulder. Movements were videotaped (120 Hz) and digitized (Peak Performance Technologies). Coordinates of the shoulder, elbow, and wrist joints were filtered using a fourth-order critically-damped filter at 5-Hz cutoff, and angular joint displacements, velocities, and accelerations were calculated. Linear velocities of the fingertip were also calculated and peak velocity for each trial was determined. To compare differences in kinematics, a two-factor ANOVA was used in which one factor was between groups (SCI vs. control) and the other factor was a repeated measure (target directions).

Electromyography

Bipolar surface electrodes were used to record electromyographic (EMG) activity of arm muscles. For control

Fig. 1A–H Kinematics of reaching for SCI and control subjects. The left column shows data for SCI subject(s) and the right column for control subject(s). In **A** and **B**, the finger paths of individual trials for one SCI (*ss#2*) and control subject are illustrated, with their arm configurations shown below as *stick figures*. **C–H** show kinematic data averaged across all subjects for each movement direction. In **C**, **D**, **E**, **F**, positive values indicate flexion excursions while negative values indicate extension excursions



subjects, six muscles were recorded, including a flexor and extensor at each joint; pectoralis major (PEC, clavicular portion), posterior deltoid (PDL), biceps brachii (BIC), the lateral head of the triceps (TRI), the flexors of the wrist and fingers (FWF), and the extensors of the wrist and fingers (EWF). The electrodes for the finger and wrist muscles were placed on the ventral and dorsal surfaces of the forearm (respectively) and they detected EMG activity in the group of wrist–finger extrinsic muscles. For the SCI group, three muscles were recorded similarly to control subjects, including the shoulder muscles PEC, PDL, and the elbow flexor, BIC. Electrodes were also placed over the dorsal surface of the forearm and they recorded the activity of the single innervated wrist–finger extrinsic muscle, ECR. Electrodes were placed over the lateral head of the triceps to

verify that no EMG was recorded. For two of the five SCI subjects, the elbow flexor, the brachioradialis (BRD), was also recorded in addition to the BIC. All EMG signals were amplified (1,000× gain) and analog band pass-filtered (5–45 Hz).¹ Signals were then sampled at 500 Hz and stored to computer. EMG analysis was primarily qualitative (EMG was absent or present), but burst duration was measured for some muscles. Onsets and terminations of EMG activity were determined by visual inspection of individual EMG records on the computer display, similar to previous reports

¹Inadvertently, the high cut-off was unusually restricted. However, EMG power spectral analyses and testing of our equipment demonstrated that this band pass did not alter our measures of EMG (presence or absence of EMG signal and timing of bursts).

(Koshland et al. 2000). Criteria for onset were: (a) EMG amplitude increased above baseline amplitude (measured during 100 ms when the arm was at rest on table before a reach), and (b) it remained above this threshold for more than 20 ms. Burst termination was determined as EMG that dropped below baseline amplitude and stayed below this threshold for more than 20 ms. To compare differences in burst duration between control and SCI subjects, the non-parametric test, Mann–Whitney rank sum, was performed.

Prior to reaching trials, SCI subjects were asked to isometrically hold their arm against resistance while the EMG activity of the muscles was recorded. In this manner, the SCI subjects' capacity to activate specific muscles was confirmed. Subjects first positioned their arm in their initial configuration with the arm resting on the table. EMG activity was recorded while they sat quietly with muscles at rest (left column in Fig. 2). Subjects then produced isometric horizontal shoulder flexion or extension while a hand-held dynamometer was manually applied to the middle of the upperarm, and the force measured by the dynamometer was recorded (right column of Fig. 2). Subjects also produced isometric elbow flexion while the dynamometer was applied to the middle of the forearm segment. As shown in Fig. 2, substantial EMG occurred for all muscles during the isometric tasks in comparison to the resting EMG levels. Figure 2 includes data for two subjects whose data were used in later figures of arm movement; nonetheless, the data in Fig. 2 were representative of all SCI subjects. For the two subjects, BRD was recorded as well as BIC and was coactive with BIC, as shown for the one subject in Fig. 2B.

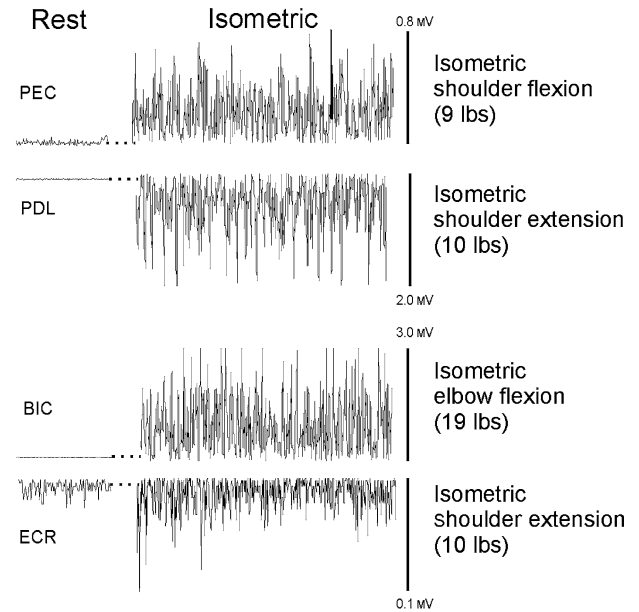
Results

Kinematics across directions

SCI subjects easily moved their arms in the apparatus, and after 3–5 practice trials, all subjects were ready to begin recording data. SCI subjects were able to reach all directions and produced straight finger paths, similar to control subjects (Fig. 1A,B). In addition, SCI subjects ended their finger path within 3.4 ± 0.8 cm of the target, similar to normal subjects who ended their finger paths at 2.9 ± 3.1 cm ($p=0.78$). Average shoulder and elbow joint excursions showed a similar pattern across target directions for both SCI and normal subjects (Fig. 1C,D,E,F). That is, the excursions varied significantly across direction (shoulder $p < 0.0001$; elbow $p < 0.0001$) with the largest elbow excursion at 120–150° and the largest shoulder excursion at 60°, 300°. There was no significant difference in the amount of joint excursion between SCI and normal subjects (shoulder $p=0.48$; elbow $p=0.31$), despite the greater intersubject variability of the SCI subjects.

SCI subjects generally produced bell-shaped velocity profiles of fingertip trajectories, illustrated in Figs. 3 and

A SCI subject #1



B SCI subject #2

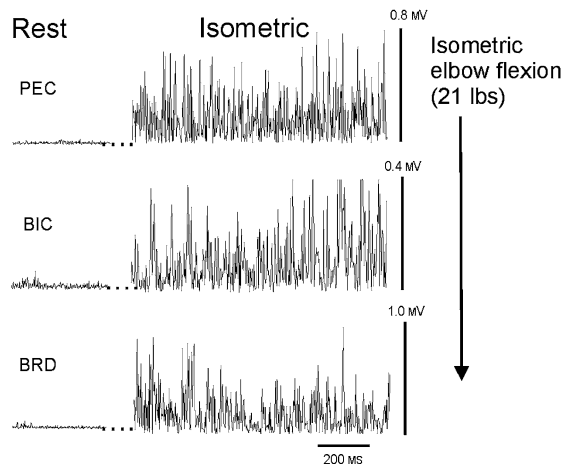
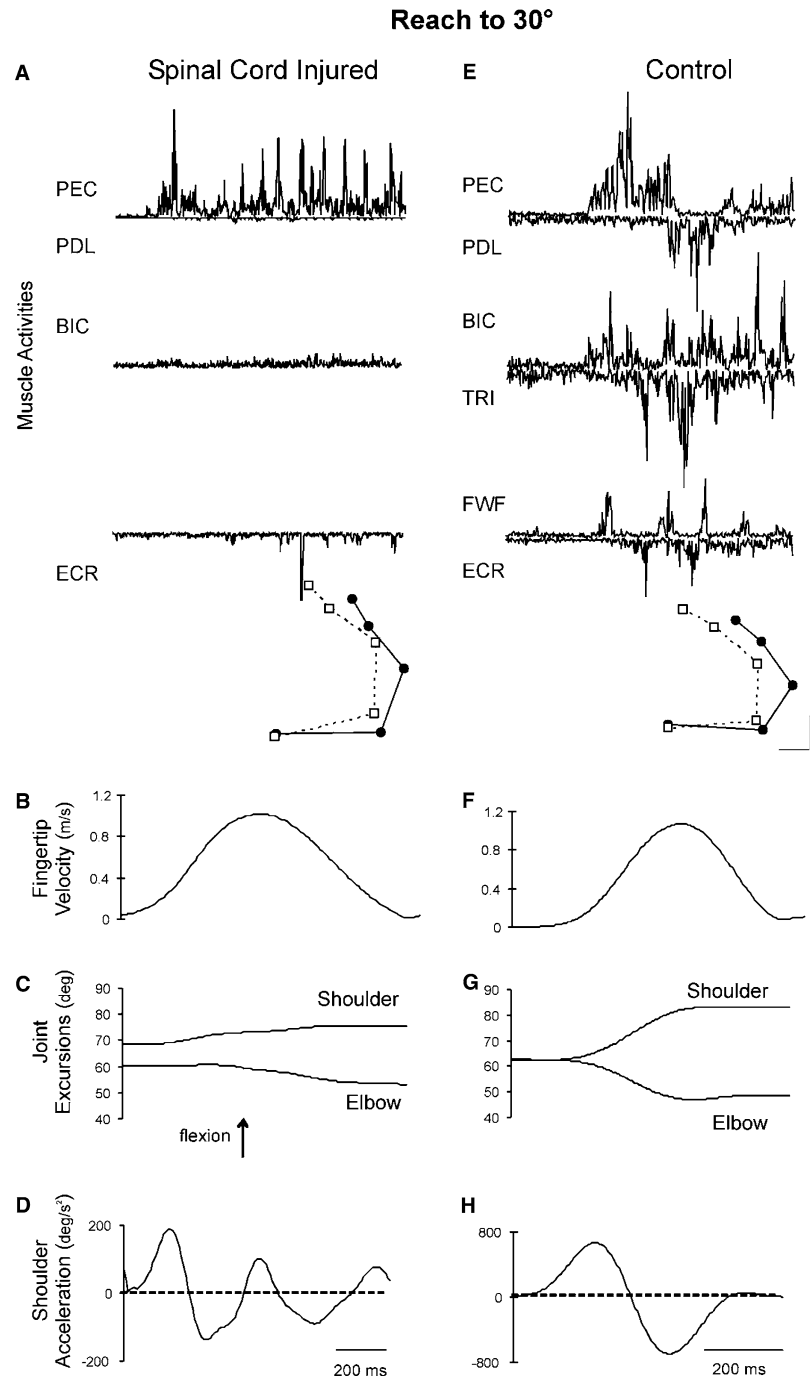


Fig. 2A–B Comparison of EMG amplitude at rest (*left column*) vs. isometrically resisted motions (*right column*). In **A**, the EMG trace for each available muscle is shown for one SCI subject (#1). Different isometric tests were selected based on the best EMG response in that muscle. Extensor muscles (PDL, ECR) are illustrated inverted. In **B**, data from another SCI subject (#2) illustrates simultaneous flexor EMG at the shoulder and elbow during one isometric task (elbow flexion). Force recorded by the dynamometer (lbs) and EMG calibration bars are included to the right of each EMG trace

6. SCI subjects reached with slower speeds, as average fingertip velocities were significantly less than those of normal subjects (Fig. 1G,H; $p=0.01$). Not only were SCI subjects slower, but post hoc analyses also showed that fingertip velocities did not vary across direction for SCI subjects ($p=0.3$), whereas fingertip velocities significantly increased and decreased across direction for normal subjects ($p=0.0001$). Control subjects reached

Fig. 3A–G Differences in EMG muscle patterns between SCI and control subjects for moving to one direction (30°). EMG records are plotted for an individual trial from one SCI (A; *ss#3—fast SCI*) and one control subject (E). Flexor EMG traces are depicted upwards with extensor EMG traces downwards. The initial (*solid line*) and final (*dashed line*) arm configurations for each trial are shown as *stick figures* below the EMG traces, and the perpendicular calibration bars each represent 10 cm. The fingertip velocities of the trial are depicted in **B** and **F**, with corresponding joint angle and shoulder joint acceleration traces in **C**, **D**, **G** and **H**. All traces in a column are shown on the same timescale indicated by the horizontal calibration bar at the bottom of the figure



with greatest fingertip velocities for directions perpendicular to the forearm (90°, 270°), consistent with earlier reports for healthy subjects (Gordon et al. 1994). One SCI subject was able to move with peak fingertip speeds equal to control subjects. This SCI subject (indicated by triangular symbols in Fig. 1G) demonstrated an average peak speed of 1.1 m/s, similar to the average for control subjects of 1.3 m/s. The other four SCI subjects moved slower, at an average peak speed of 0.6 m/s. In general, then, the kinematics of reaching movements of the SCI subjects displayed straight paths, endpoint accuracy, bell-shaped velocities, and joint excursions similar to

control subjects, while moving more slowly than control subjects.

Muscle activities to one representative target direction (30°)

Differences in elbow and wrist muscle patterns

Patterns of muscle activities were remarkably different for the SCI subjects from those of control subjects. To illustrate this point, the muscle activities for movements

to one target direction (30°) are compared in Fig. 3. This figure shows an individual trial for a control subject compared to a trial from the SCI subject who produced normal speeds. For this point-to-point movement, control subjects typically activated muscles at the three joints (shoulder, elbow, and wrist), as shown for the one subject in Fig. 3E. In contrast, muscle activities for the SCI subject occurred almost exclusively at the shoulder with very little or unmodulated distal muscle activity (Fig. 3A). In this particular trial (Fig. 3A), the biceps muscle (BIC) exhibited low-level tonic activity before and during the movement, with very little change in modulation. The wrist extensor muscle activity (ECR) was also tonically active (one large spike near the end of movement; either a large motor unit or an artifact). Hence, minimal muscle activity occurred at the elbow and wrist joints. This pattern was observed for all SCI subjects; that is, BIC EMG was absent in 86% and ECR in 67% of trials ($n=42$ trials of reaches to 30°). In contrast, for normal subjects, BIC EMG was absent in 8% and ECR in 4% of trials ($n=24$).

Differences in shoulder muscle patterns

Although muscle activity was present at the shoulder joint for both the SCI and control subjects, the pattern of shoulder activity for SCI subjects was very different from that of control subjects. For the control subject in Fig. 3, muscles were activated in a reciprocal pattern at the shoulder joint (as well as at the elbow and wrist joints). For this direction, the first muscle activated at the shoulder joint was the flexor muscle (PEC). Later in the movement, the antagonist muscle, the extensor (PDL), was activated with a second agonist (PEC) burst at the end of movement. In contrast, for the SCI subject, only the flexor muscle (PEC) was active with no reciprocal activity of the antagonist extensor muscle (PDL). Moreover, even though PEC activity was present for both the SCI and control subject, the shape of PEC activity for SCI subject was very different from that of the control subject. For the control subject, a typical interference burst occurred in which PEC EMG amplitude gradually increased to a peak and then decreased. For the SCI subject, however, PEC activity occurred throughout the entire duration of the movement with repetitive bursts rather than an interference pattern. The PEC activity at the beginning of the particular trial in Fig. 3A may represent an interference pattern, but was immediately followed by repetitive bursts that were atypical of control subjects.

The differences in shoulder EMG shown for the one trial in Fig. 3A was consistent for all SCI subjects moving to the 30° target. Duration for the PEC EMG was significantly longer for SCI vs. control subjects ($p=0.009$) with an average duration of 1221 ± 527 ms for SCI subjects vs. 160 ± 29 ms for control subjects. In fact, the burst duration of PEC occupied, on average, 180% of movement time for SCI subjects (PEC EMG

continued after movement termination as in Fig. 3A), in contrast to 54% of movement time for the control subjects. For the antagonist shoulder muscle of control subjects, PDL burst duration averaged 160 ± 45 ms. For the few trials that PDL was present for SCI subjects ($n=8\%$ or 5 trials), the duration was short at 32–86 ms. In general then, the shoulder muscle pattern for reaching to 30° by all SCI subjects showed very long bursts in PEC, with no or minimal activity in PDL.

Differences in kinematics for the 30° movement

The very different EMG patterns between the two trials in Fig. 3 would be expected to produce different kinematic consequences. Indeed, there were joint kinematic differences in Fig. 3, while global reaching features were maintained nonetheless. For instance, in Fig. 3, shoulder and elbow excursions were less for the SCI subject than the control (SCI—7°, 7°sho, elb in Fig. 3C vs. control—20°, 15° in Fig. 3G), and movement duration was prolonged for the SCI subject (SCI—925 ms vs. control—406 ms; see different timescales in Fig. 3B,F). Nonetheless, for both trials, subjects moved the same distance (20 cm) with bell-shaped fingertip velocities. Although the peak velocities were similar for both trials (Fig. 3B 1.02 m/s, Fig. 3F 1.07 m/s), the bell-shaped velocity curve was much broader and slower to develop for the SCI trial, consistent with the reduced magnitude of shoulder angular accelerations (see different scales in Fig. 3D,H). The discrepancy in joint excursion might be explained by the fact that, for this trial, this SCI subject showed 11° of trunk rotation, in contrast to 8° of trunk rotation for the control subject (look at stick figures of initial and final arm configurations below Fig. 3A,E). Rotation at the most proximal point has a large effect on the fingertip movement, and for instance, 11° of trunk rotation alone would move the fingertip 75% of the distance to the target, in contrast to 8° which would move the fingertip 53% of the distance. This means that considerably less shoulder and elbow excursion was needed to reach the target for the SCI trial. On average, however, SCI subjects did not demonstrate significantly more trunk rotation than normal subjects (SCI— $11 \pm 6^\circ$ vs. control— $9 \pm 2^\circ$, $p=0.91$), and as previously reported, they did not show significant differences in average joint excursions (Fig. 1C,D,E,F). Moreover, post hoc analysis showed that at the 30° direction, joint excursions for SCI subjects did not significantly differ from controls (sho $p=0.24$, elb $p=0.41$). Hence, the differences in joint excursion and trunk rotation that occurred for Fig. 3C seem to be specific to these trials.

There was another kinematic difference that occurred between the two trials that was consistent for other trials and subjects. The SCI subject showed reduced magnitude of shoulder acceleration (note the scale differences in Fig. 3D vs. H), and multiple waves occurred in the shoulder angular acceleration trace, in contrast to the control subject who produced the typical biphasic

acceleration trace with one smooth, initial acceleration and one later deceleration (Fig. 3F). This pattern of multiple shoulder accelerations occurred in 93% of SCI trials to the 30° target but never occurred in trials from control subjects. The magnitude of the first peak in shoulder angular accelerations was $180^\circ/s^2$ for SCI subjects vs. $847^\circ/s^2$ for control subjects. All in all, for reaching to 30° direction, the unusual muscle pattern of SCI subjects produced very different shoulder accelerations and occasional differences in joint excursions with trunk rotation, but preserved the straight paths and bell-shaped velocities.

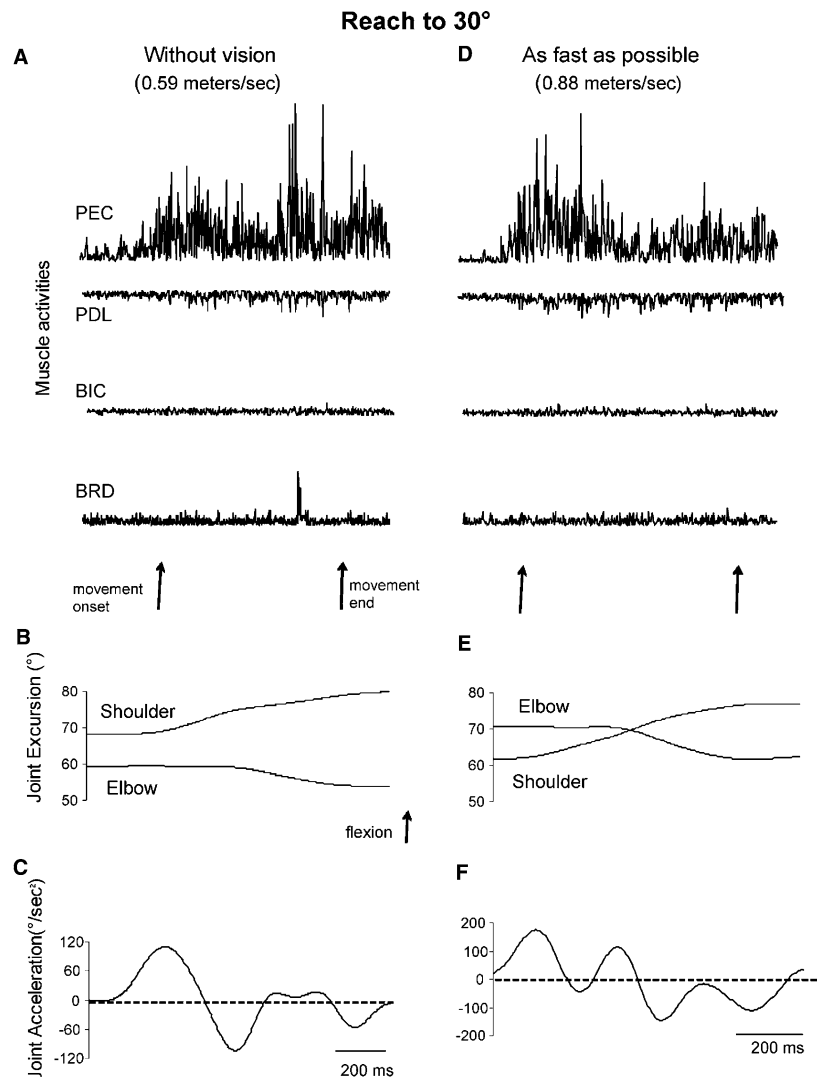
Alternative explanations for novel muscle pattern

What factors besides motor redundancy could explain the SCI subject's novel muscle pattern? First, SCI subjects may have relied more heavily on visual feedback throughout the movement. To address this issue, we asked SCI subjects to move towards the 30° target with their vision blocked (by wearing special glasses). The

same pattern of muscle activities occurred; namely, flexor muscle activity was present at the shoulder but no shoulder extensor (PDL) or elbow flexor (BIC) activity occurred (Fig. 4A). For all SCI-injured subjects, the EMG patterns did not change with vision blocked, suggesting that the use of visual feedback during the reaching did not cause the subjects to use their atypical muscle pattern.

Another explanation for the change in muscle patterns could be that the slower speed of movement of SCI subjects required a different EMG pattern. Even though one SCI subject moved at a normal speed, the other four SCI subjects tended to move slower. To test this idea, we recorded additional trials in which the slower SCI subjects ($n=4$) were instructed to move as quickly as possible. In Fig. 4B, the EMG traces for a movement produced by one SCI subject are displayed. This subject increased his speed to 0.88 m/s with instructions to move as quickly as possible, in contrast to an average of 0.5 m/s when previously instructed to make one quick movement. He showed the same pattern of muscle

Fig. 4A–F Same format as Fig. 3. The EMG pattern (shoulder and elbow muscles) is shown when the subject's vision was blocked (*left column A–C*) and with increased speed when instructed to move as fast as possible (*right column D–F*). The SCI subject was the one who moved slower and is the same subject whose finger paths were depicted in Fig. 1A (*ss#2*) and whose average joint excursions were represented by the open squares in Fig. 1C



activities with either set of instructions. That is, substantial EMG occurred only in the shoulder flexor muscle (PEC). The same findings occurred for the other SCI subjects when asked to move as quickly as possible.

Even though the unusual EMG pattern did not change with the faster movements, the SCI subjects still did not reach speeds comparable to control subjects. Average speed of the four SCI subjects when asked to make one quick movement was 0.6 ± 0.12 m/s and this increased to 0.7 ± 0.08 m/s when asked to move as quickly as possible. It was therefore still possible that the different patterns of muscle activities for the SCI subjects could have reflected patterns typical of slower control movements. As a result, we asked control subjects to move more slowly, paced with a metronome. Figure 5 illustrates the EMG patterns for shoulder agonist and antagonist muscles across a range of speeds. All trials were movements to the 30° target, and trials of similar speeds were matched for the SCI and control subjects. The range of speeds included trials with fingertip velocities from 0.64–0.94 m/s. For the shoulder agonist (PEC), the control subject exhibited an initial burst that was followed by a quiet period. This quiet period usually started at 100–200 ms after movement

onset (Fig. 5A, right). A second burst occurred towards the end of movement and continued for the 100 ms after movement termination; that is, until the end of records shown in Fig. 5A, right. This pattern occurred consistently across all speeds, even for the slowest movement at 0.64 m/s. In contrast, the SCI subject exhibited continuous PEC activity for all trials with irregular modulations (Fig. 5A, left). For the antagonist muscle (PDL), the control subject showed a burst whose onset always occurred in the quiet period of the agonist burst (Fig. 5B, right). In contrast, for the SCI subject, all trials but the fastest trial showed no or very minimal tonic PDL activity (Fig. 5B, left). For the fastest trial in Fig. 5B, left, an antagonist burst was visible, and a corresponding quieter period was apparent in the PEC EMG trace for this same trial. In general, the findings from this figure suggest that for movements to the 30° target, the shoulder EMG pattern of the SCI subject was indeed unlike control subjects moving at similar slow speeds.

A pattern of multiple shoulder joint accelerations accompanied the continuous and irregularly modulated shoulder flexor EMG in Fig. 3. A similar question then arose for the pattern of shoulder accelerations as had

Fig. 5A–B EMG traces of individual trials to 30° target at different speeds for one SCI subject (*ss#2*—left column, same as Figs. 1A and 4) and one control subject (right column). Trials have been matched for speeds from 0.64 to 0.94 m/s. Shoulder agonist traces (PEC) are shown in **A**, while shoulder antagonist traces (PDL) are shown in **B**. All EMG traces for a muscle are on the same scale, except the two PDL traces at 0.86 and 0.94 m/s of the control subject that, as indicated, were three times the scale of other traces. The vertical dotted line indicates the onset of movement (time of 10% peak fingertip velocity) to which all EMG traces were aligned. All EMG traces end at 100 ms after movement termination (movement termination is the time of 10% peak fingertip velocity on deceleration)

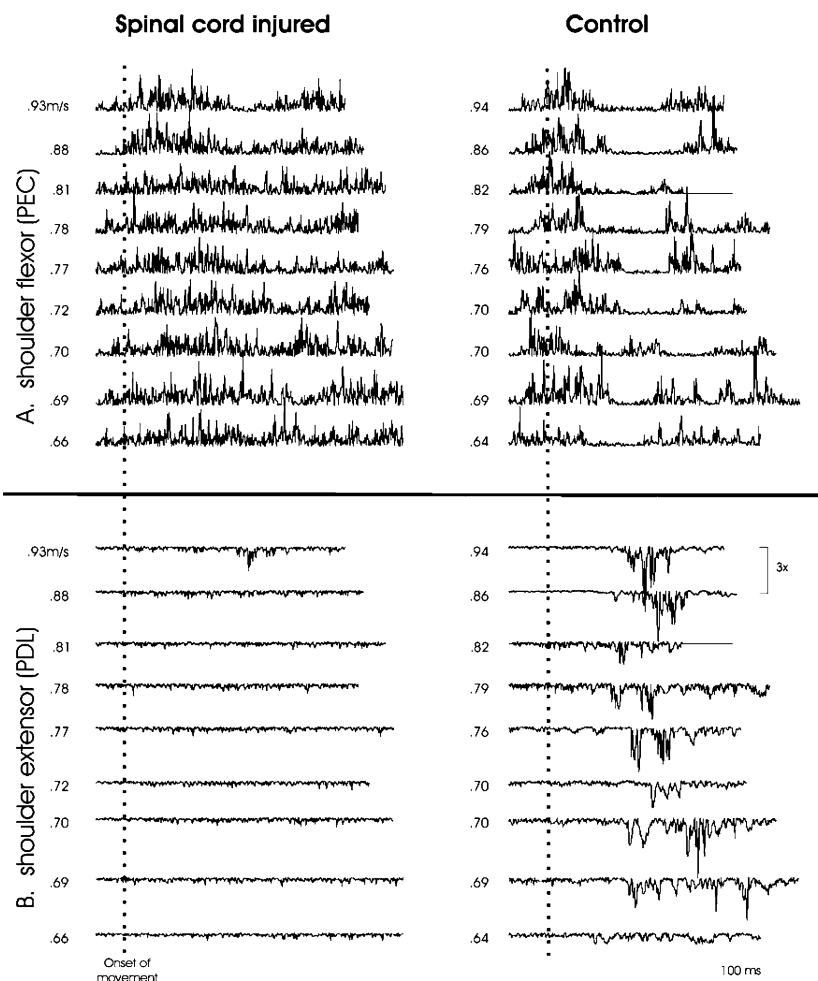
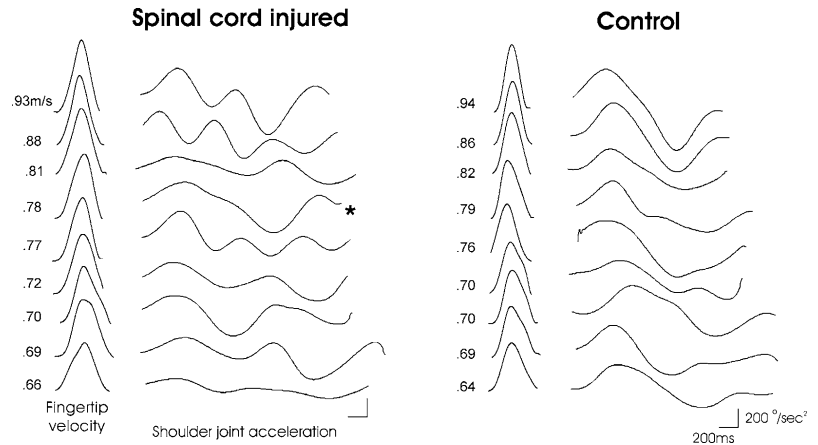


Fig. 6 Shoulder kinematics for movements at different speeds are displayed for the same EMG trials shown in Fig. 5. The bell-shaped fingertip velocity profiles are shown to the left and the accompanying numbers indicate the peak velocity for each trial. To the right, the shoulder acceleration traces are shown for the same time period as the EMG traces in Fig. 5 (100 ms before movement onset up to 100 ms after movement termination)



arisen for the shoulder muscle activity; namely, it was possible that the multiple shoulder accelerations were also a feature of normal, slower movements and were not related to the shoulder EMG pattern. In Fig. 6, shoulder angular acceleration traces are displayed for the same trials for which shoulder EMG data are shown in Fig. 5. Shoulder joint accelerations for the control subject remained relatively biphasic across the range of speeds (Fig. 6, right), although the traces were less smooth for slower movements (for example, ≤ 0.79 m/s). In contrast, the shoulder acceleration traces for the SCI subject most often showed multiple accelerations and decelerations, usually 2–3 cycles/trial (Fig. 6, left). Only one trace exhibited a biphasic profile (identified by an asterisk in Fig. 6, left). Despite the multiple peaks in shoulder joint accelerations², fingertip velocity profiles were always bell-shaped and relatively smooth, similar to control subjects (displayed to the left of each acceleration trace). This means that for the SCI subjects, the continuous shoulder flexor muscle activity was typically accompanied by multiple shoulder joint accelerations, and this pattern of EMG and joint accelerations was not a feature of slower movements of control subjects.

Muscle activities summarized for all directions

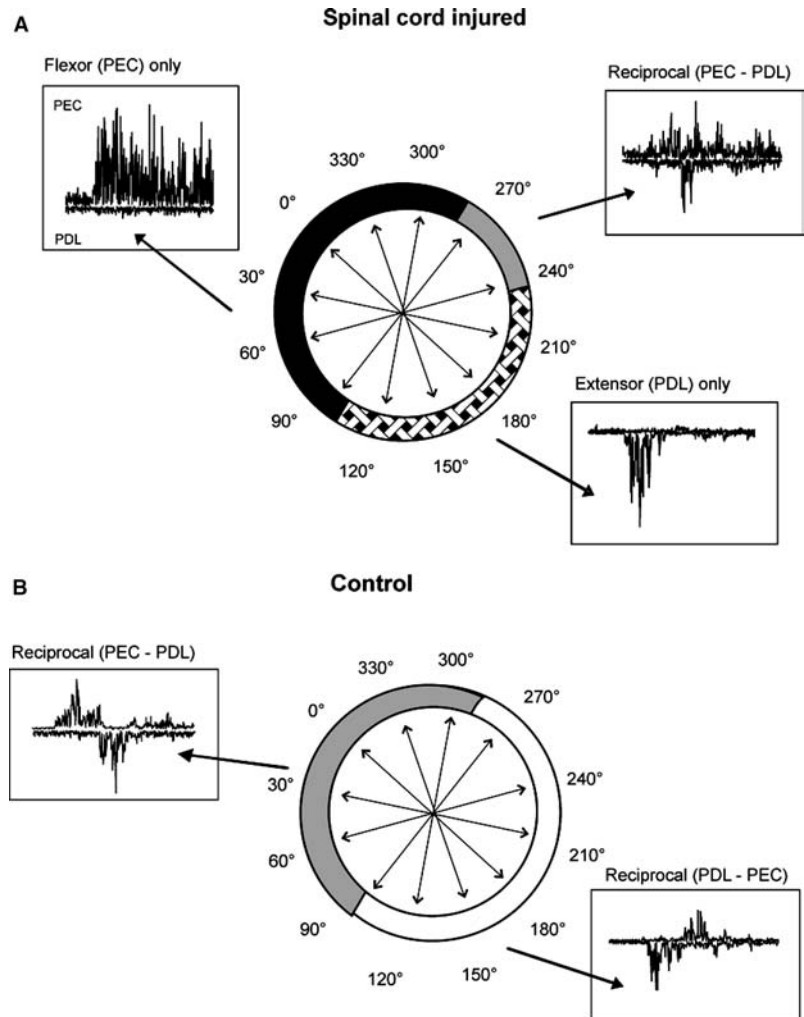
The novel pattern of muscle activities observed for SCI movements to the 30° target were also observed in movements to the other target directions. Data are summarized across directions for one SCI and one control subject in Fig. 7. The control subject used one of two reciprocal patterns of shoulder muscles to move to targets in the center-out task (Fig. 7B). For movements to targets, starting at 300° and moving counterclockwise to 90°, the control subject used a reciprocal pattern that was initiated with the shoulder flexor, followed by shoulder extensor activity (PEC–PDL, indicated by the ring with gray shading in Fig. 7B). For the remaining

targets (120° moving counterclockwise to 270°), the opposite reciprocal pattern was used, in which shoulder extensor activity was initially active, followed by shoulder flexor activity (PDL–PEC, indicated by ring with white fill). In contrast, the SCI subject in Fig. 7A used a reciprocal pattern (PEC–PDL) to only one target direction (270°). For all other directions, he used one shoulder muscle without antagonist activity. For targets 300–90°, the SCI subject used only the shoulder flexor muscle (PEC-only, indicated by the ring of black shading), whereas for the remaining target directions, 120–240°, he only used the shoulder extensor muscle (PDL-only, indicated by the ring of stippled fill).

The other subjects followed the general patterns illustrated in Fig. 7, and data for all subjects were summarized in Fig. 8. All control subjects used one of the two reciprocal patterns. This finding can be seen in Fig. 8B, in which bars are either gray, representing the PEC–PDL reciprocal pattern, or white, representing the PDL–PEC reciprocal pattern. Moreover, the four control subjects used the same reciprocal pattern for each direction, except for one direction, 270°. For the 270° direction, one control subject used a PEC–PDL reciprocal pattern while the three other control subjects used the PDL–PEC reciprocal pattern. In contrast, the five SCI subjects rarely used a reciprocal pattern. For 6/12 directions, SCI subjects used the same non-reciprocal pattern; PEC-only for five directions (300°, 330°, 0°, 30°, 60°) and PDL-only for one direction (150°). For the remaining directions, different SCI subjects used various patterns of shoulder muscles and, for example, to the 120° direction, one SCI subject used PEC-only, three SCI subjects used PDL-only, and the remaining SCI subject used a reciprocal pattern of PEC–PDL. In general, the SCI subjects used a PEC-only pattern when control subjects used a reciprocal PEC–PDL pattern (black bars occur in Fig. 8A where gray bars occur in Fig. 8B). Also, the SCI subjects tended to use a PDL-only pattern when control subjects used the reciprocal PDL–PEC pattern (stippled bars occur in Fig. 8A where white bars occur in Fig. 8B). For only one direction (180°), SCI muscle patterns seemed similar to control, as 4/5 SCI subjects used a reciprocal PDL–PEC pattern.

²Typically, multiple peaks also occurred in the traces of elbow joint acceleration but were not necessarily coincident with the multiple peaks in shoulder joint acceleration.

Fig. 7A–B Patterns of shoulder EMG across target directions for a representative SCI subject (A; *ss#1*, same as Fig. 2A) vs. control subject (B). The control subject always used a reciprocal shoulder pattern, indicated by the representative EMG traces in the boxes (PEC—upward trace; PDL—downward trace). The target directions for which the reciprocal pattern, PEC–PDL, was used are indicated by the *gray-filled ring*, whereas the directions for which the reciprocal pattern, PDL–PEC, was used are indicated by the *white-filled ring*. The SCI subject used a PEC-only pattern (*black ring*) or a PDL-only pattern (*stippled ring*). Representative EMG traces are again shown in the *boxes*. For one direction (270°), the SCI subject demonstrated a reciprocal pattern (PEC–PDL—*gray ring*)



These findings extend the results from the one direction, 30° (Figs. 3, 4, 5, and 6); namely SCI subjects used remarkably different muscle activity patterns for almost all directions of horizontal reaching (Figs. 7 and 8).

Discussion

The primary finding of this study was that SCI subjects did not activate certain arm muscles, innervated above the level of the lesion, that were typically activated by control subjects during center-out reaching movements. These findings demonstrate an unexpected change in selection of muscles by SCI subjects and what we call a fundamentally different choice of muscle pattern. The evidence that the SCI were not selecting these muscles is based on our findings that, on the one hand, the SCI subjects were capable and did activate the muscle when isometrically resisting a force and when moving to certain directions; on the other hand, they did not activate the same muscle for reaching movements to other directions. For example, the shoulder extensor, PDL, was absent for five target directions (300–90, Figs. 7 and

8) but was activated for another four target directions (120–210°, Figs. 7 and 8) and when isometrically resisting 10 lbs of force (Fig. 2). By looking at the pattern of deactivated muscles across directions, it was apparent that SCI subjects choose not to activate the shoulder antagonist muscle for almost all directions, which was sometimes the flexor (PEC) and other times the extensor muscle (PDL). At the elbow, the SCI subjects chose not to activate BIC (Figs. 3 and 4) or BRD (Fig. 4) for any reaching movements, despite the evidence that they could activate the BIC and BRD when isometrically resisting a force (Fig. 2). This means that the elbow flexors were not activated as either an agonist or antagonist muscle for any direction.

Choice of muscle patterns for reaching in C5–C6 SCI subjects

Previous studies of EMG with SCI subjects have reported properties of motor units (Davey et al. 1990; Latash 1988; Thomas and del Valle 2001; Wiegner et al. 1993) and amplitudes of muscle activities (Gronley et al.

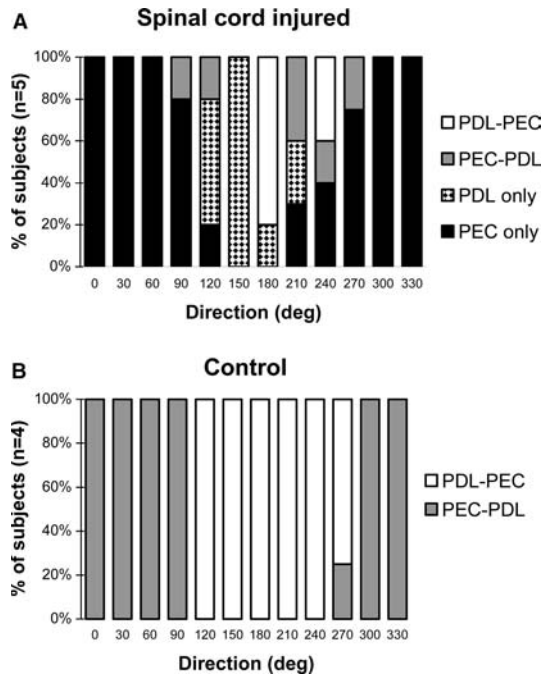


Fig. 8A–B Summary for the pattern of shoulder EMG for five SCI subjects (A) vs. four control subjects (B). The shading follows the format of the shading in Fig. 7. Control subjects always used a reciprocal pattern (gray or white), and for all but one target direction, all control subjects produced the reciprocal pattern (all gray or all white). All SCI subjects used the PEC-only pattern for 5/12 directions, from 270–60° (black shading). For the other directions, the pattern of shoulder EMG varied among the SCI subjects. For only two directions (180 and 240°), some SCI subjects used a control-like pattern of reciprocal PDL-PEC activity (white shading)

2000; Marciello et al. 1995; Seelen et al. 1998; Simard and Ladd 1971; Wierzbicka and Wiegner 1992, 1996), but none have stated that certain available muscles (those innervated above the level of the lesion) were not selected. Although several of these studies examined arm movements, the tasks were quite different from the horizontal planar reaching movements of this study, and the authors did not report temporal patterns of muscles or record similar muscles across the arm as in this study. Therefore it is unknown if a reciprocal agonist/antagonist pattern occurred at the shoulder and if activity occurred at both the shoulder and elbow joints in these previous studies.

It is possible that other synergistic muscles were active in the present study but were not recorded as the representative flexor and extensor at each joint. That is, anterior deltoid could have been active when PEC was silent. Scapular/shoulder muscles, such as teres major and latissimus dorsi could have been active when PDL was silent. Although a potential trade-off to other synergistic muscles cannot be refuted, evidence from this study and other studies suggest it is unlikely. That is, for two of the five SCI subjects of this study, the two elbow flexors, BIC and BRD, were coactive during the isometric task (Fig. 2B) whereas both flexors were silent

during arm movements (Figs. 3A and 4). In addition, previous EMG studies with SCI subjects have shown that shoulder synergists tend to be coactive. That is, anterior deltoid was coactive with pectoralis as well as with scapular muscles, such as latissimus dorsi (Gronley et al. 2000; Seelen et al. 1998). The combined results from this study and these other EMG studies of SCI then suggest that for SCI subjects and control subjects, synergistic muscles tend to be coactive as a group (Bouisset et al. 1977; Buchannan et al. 1986; Buneo et al. 1994; Gribble and Ostry 1999; Howard et al. 1986; van Bolhuis and Gielen 1997). Two studies explicitly instructed subjects to use elbow vs. wrist (Latash and Jaric 1998) or BIC vs. BRD muscles (Howard et al. 1986), and although subjects were able to modify the relative amount of activation, they were unable to silence a muscle of a synergistic group. The absence of PEC, PDL, and BIC activity in the present study, therefore, implies that antagonist shoulder muscles and elbow flexors as general groups were not selected for planar reaching in SCI subjects.

In contrast to the difference in muscle activities, some kinematic aspects of reaching by SCI subjects were similar to control subjects. Paths of the finger to the target were relatively straight, and velocities were bell-shaped and symmetrical (Figs. 1 and 6). The pattern of shoulder and elbow joint excursions varied across direction in the same manner as control subjects, and the average amount of joint excursion was generally similar (Fig. 1). However, there were some kinematic consequences of the different muscle pattern; namely, multiple shoulder joint accelerations and slower peak path velocities. These findings demonstrate an interesting realization about the interaction of kinematic features. Altered kinematic details such as multiphasic shoulder accelerations can nonetheless lead to unaltered general kinematic features such as straight finger paths and bell-shaped velocities. In this manner, the atypical muscle pattern of SCI subjects produced altered kinematic details but still was able to produce reaching movements that were globally similar to control subjects.

Taken together, the comparison of SCI subjects with control subjects of this study suggests that the nervous system does have a choice of muscle patterns to deal with the mechanics of the arm. The findings show that straight path movements can be achieved with a pattern of reciprocal muscles at each joint (control pattern) or with a pattern of unidirectional shoulder muscles only (SCI pattern).

Mechanical plan for reaching of C5–C6 SCI subjects

The question of why did the SCI subjects select the muscles they do to reach to targets remains, however. Previous multijoint arm studies suggest some clues (Dounskaia et al. 1998; Galloway and Koshland 2002; Galloway et al. 2004; Gribble and Ostry 1999; Ketcham et al. 2004). Control subjects typically use a reciprocal

pattern at the shoulder to accelerate and then decelerate the upperarm. This is a straightforward relationship, much like single joint control for which a flexor muscle flexes the joint, and extensors extend the joint. Reciprocal activity also occurs at the elbow but must deal with the mechanical consequences created by the upperarm motion. For example, for the 30° target described in this study, our previous work has shown that initial upperarm acceleration into flexion causes the forearm to flip into extension (Koshland et al. 1998; Figs. 3 and 5 in Galloway and Koshland 2002), and BIC is activated to limit elbow extension to the desired amount. Later when the upperarm is decelerated (when PDL is active), TRI is activated to prevent the forearm from flopping back into flexion. The SCI subjects cannot activate TRI, and hence we propose that the SCI subjects utilize a different strategy that avoids this problem. First, they reduce shoulder accelerations so that they do not need to produce large shoulder decelerations (Figs. 3 and 6). In this way, the shoulder deceleration remains minimal and they are not put in the situation of needing to prevent any ensuing elbow flexion. Second, they use multiple, small shoulder accelerations to produce small passive elbow extensions until they achieve enough total elbow extension to reach the target. The ultimate consequence of this strategy is that with the small shoulder accelerations, the SCI subjects can achieve elbow extension by passive mechanical effects and they do not need to activate their BIC or PDL muscles.

This strategy is consistent with hints from previous studies of EMG in SCI subjects, which have shown that muscle patterns change in order to compensate for missing elbow function. Marciello et al. (1995) demonstrated that C6 SCI subjects could produce isometric force in elbow extension. This extensor force was exerted despite the fact that 4/5 of the subjects produced no extensor force on manual muscle tests and only two subjects exhibited any triceps surface EMG, which was still very minimal and could not be ruled out as crosstalk. The authors proposed that the elbow extensor force was produced by the shoulder muscles (anterior deltoid and pectoralis) through the mechanical linkage of upper arm and forearm. Similarly, Gronley et al. (2000) proposed that C6 cervical SCI subjects demonstrated increased shoulder EMG in several tasks in order to control the distal motion of the elbow and wrist (raising an extended, straight arm into flexion, drinking from a cup). In the present study, similar findings are revealed. SCI subjects produced almost normal amounts of elbow motion with no elbow muscle activity at all. This means that elbow motion was passively achieved, and most likely through the action of the upperarm. Kinetic analyses of SCI reaching need to be conducted to confirm our speculation, but it is beyond the scope of this study. One of several difficult issues that would need to be addressed would be to determine the static and dynamic visco-elastic muscle contribution to the generalized muscle torque, calculated as a residual term in inverse dynamics techniques. One study has made esti-

mates of the relative contribution of passive components to the muscle torque term through the use of simulations (Hirashima et al. 2003b) and others have measured stiffness in control subjects (Koike and Kawato 1995; Lestienne and Pertuzon 1974; Mah 2001; Osu and Gomi 1999), but nonetheless, it would be an ambitious project to accurately measure or even estimate visco-elastic properties in SCI subjects.

Although kinetic analyses have not been applied to reaching of SCI subjects, previous reports have demonstrated that SCI subjects are able to decelerate the arm with passive visco-elastic properties (Wierzbicka and Wiegner 1992, 1996). SCI subjects were able to perform desired amounts of elbow flexion, which also means that they could accurately stop the elbow at desired positions. Moreover, SCI subjects reduced their agonist activation and acceleration in order to decelerate the arm by passive visco-elastic properties since they lacked the ability to activate the antagonist muscle (TRI). In fact, when the authors provided an external elbow extensor torque, SCI subjects were able to increase their acceleration and BIC activity, suggesting the decreased agonist activity and accelerations was a strategy to deal with their limited ability to decelerate the elbow. Similarly in the present study, antagonist muscle activity was not present at either the shoulder or elbow joints. Only agonist activity was generally present at the shoulder, so deceleration of the upperarm was presumably achieved through visco-elastic properties (and minimal friction of the apparatus rolling on the table, which was calculated to be 1.5 N or 0.34 lbs). Our findings add to the previous single-joint findings that deceleration can be achieved by visco-elastic properties, as long as speeds remain below some threshold level. This has been demonstrated for control subjects (Lestienne 1979) and for SCI subjects in the previously mentioned studies by Wierzbicka and Wiegner (1992, 1996). Four of the five SCI subjects could increase their speeds but could not reach normal speeds, suggesting movements of the control subjects occurred above a speed threshold that required active muscle deceleration torque. It is curious that one of the SCI subjects in this study was able to move at control speeds and still did not activate shoulder antagonist and elbow muscles. Again, kinetic analysis would be needed to investigate the possible reasons that this subject could achieve the faster speeds.

All in all, the findings of the present study add to previous studies that SCI can modify their muscle patterns to make use of the mechanical linkage of the arm. The use of shoulder muscles alone in the SCI subjects also add to hypotheses about general control of the arm, particularly the hypothesis proposed by several laboratories that a shoulder-driven strategy may be a general rule for arm reaching and throwing (Dounskaia et al. 1998; Galloway and Koshland 2002; Hirashima et al. 2003a; Ketcham et al. 2004). If true, these ideas support revised designs of neuroprostheses. Alternative muscle patterns suggest that controllers (neural or computer

software) can move a robotic arm with either fewer (shoulder only) or more actuators (all joints). Moreover, the shoulder-driven strategy suggests that just as walking in robots and prosthetics can be achieved through the regulation of a simple inverted spring, reaching can possibly be achieved through the regulations of a damped linkage that is manipulated at its most proximal joint.

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