Muscle activity in rapid multi-degree-of-freedom elbow movements: solutions from a musculoskeletal model

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Abstract. The activity of certain muscles that cross the elbow joint complex (EJC) are affected by forearm position and forearm movement during elbow flexion/extension. To investigate whether these changes are based on the musculoskeletal geometry of the joint, a three-dimensional musculotendinoskeletal computer model of the EJC was used to estimate individual muscle activity in multi-degree-of-freedom (df) rapid (ballistic) elbow movements. It is hypothesized that this model could reproduce the major features of elbow muscle activity during multi-df elbow movements using dynamic optimal control theory, given a minimum-time performance criterion. Results from the model are presented and verified with experimental kinematic and electromyographic data from movements that involved both one-df elbow flexion/extension and two-df flexion/extension with forearm pronation/supination. The model demonstrated how the activity of particular muscles is affected by both forearm position and movement, as measured in these experiments and as previously reported by others. These changes were most evident in the flexor muscles and least evident in the extensor muscles. The model also indicated that, for specific one- and two-df movements, activating a muscle that is antagonistic or noncontributory to the movement could reduce the movement time. The major features of muscle activity in multi-df elbow movements appear to be highly dependent on the joint’s musculoskeletal geometry and are not strictly based on neural influences or neuroanatomical substrates.

1 Introduction

While isometric muscle interaction at various elbow and forearm positions have been studied, few investigators have examined how the muscles crossing the elbow joint contribute to the dynamics involved in elbow flexion/extension (f/e) and forearm pronation/supination (p/s). For example, previous studies of rapid (i.e. ‘as quickly as possible,’ ‘ballistic’) movements at the elbow (Angel 1974; Brown and Cooke 1981; Darling and Cooke 1987; Kilmer et al. 1982; Nahvi 1989; Wierzbicka and Wiegener 1992; Yamazaki et al. 1995) have not shown how the elbow and forearm position and its movement indirectly influence the neural control of the actuators crossing this joint. They have shown, however, that the electromyographic (EMG) patterns of both the agonist and antagonist muscles have a triphasic behavior. This triphasic process has been described by the acronym ABC (A = acceleration burst in the EMG signal, B = braking burst in the EMG signal, C = clamping burst in the EMG signal; Hannaford and Stark 1985) and occurs in ballistic movements which require rapid initiation and an abrupt voluntary stop at a specified position. Isometric contractions at the elbow have been studied for the two-df motions of elbow f/e and forearm p/s (Buchanan et al. 1989; Caldwell et al. 1992; Funk et al. 1987) to understand the activation strategies of muscles that cross the EJC. Other studies (Sergio and Ostry 1994, 1995; Steward et al. 1981) have found that forearm rotation plays a notable role in determining the recruitment of muscles during elbow f/e movements, and vice versa. Sergio and Ostry (1994, 1995) investigated the activity of muscles in multi-df elbow movements and reported that the activity of two joint muscles is amplitude-dependent on the joints’ motion and that the magnitude of activity between muscles changes with motion in a second df. In addition, studies of flexor muscle synergy during static movements conclude that the idea of fixed flexor synergies, or a ‘flexor equivalent,’ should be approached cautiously (Buchanan et al. 1986; Howard et al. 1986).

Because several investigations have shown that the neural control of muscles that cross the EJC is influenced by both the position and movement of the forearm, it is hypothesized that the major features and changes in muscle activity during multi-df elbow movements could be predicted with a three-dimensional musculotendinoskeletal computer model. This model

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mathematically describes the muscle dynamics, musculoskeletal geometry, and skeletal motions pertaining to f/e and p/s. If this hypothesis is true, then predictions from the model would demonstrate that the major changes of muscle activity are primarily the result of the EJC’s musculoskeletal geometry rather than the underlying neural circuitry.

2 Methods

A series of one- and two-df ballistic elbow movements was modeled and verified using kinematic and electromyographic data both from this study and from previously reported studies. One-df elbow flexions were evaluated with the forearm supinated (F@S), neutral (F@N), and pronated (F@P) to investigate how the forearm position affected the activation of the flexor muscles biceps brachii (BIC), brachialis (BRA), brachioradialis (BRD), and pronator teres (PRT). Similarly, one-df extensions were evaluated with the forearm supinated (E@S), neutral (E@N), and pronated (E@P), to investigate the activation of the extensor muscles triceps brachii (TRI) and anconeus (ANC). Two-df movements (f/e and p/s) were also evaluated to investigate their dynamic interactions on the elbow muscles, which included the supinator (SUP) and pronator quadratus (PRQ) muscles. Four different two-df movements were experimentally performed and modeled. Protocols consisted of an elbow flexion with forearm pronation (F + P) and with forearm supination (F + S), and an elbow extension with forearm pronation (E + P) and with forearm supination (E + S). Subjects were asked to move through a pre-selected angular range of motion (approximately 50°–70° of f/e and/or 70°–100° of p/s) as quickly as possible, with speed emphasized over accuracy of movement, and to hold the final position. Three trials of each protocol were measured to insure repeatable performance. In total, ten ballistic movements were evaluated and are summarized in Table 1.

2.1 Human experiments

2.1.1 Data collection

An electromyometer which measures elbow f/e, forearm p/s, and forearm abduction/ adduction (Chao et al., 1980) was used to measure the time-varying joint position (Fig. 1). The two subjects studied were right-handed men (28 and 25 years old; weight 70.4, and 74.1 kg; height 1.73 and 1.70 m, respectively). EMG surface and wire electrodes were placed over or into the relevant muscles (Delagi 1980) and adjusted so that the best electrical activity could be recorded. Individual EMG signals were checked by short isometric bursts to confirm proper recording and minimization of 60 Hz noise. The electromyograph was then positioned on the right arm and joint axes aligned with respective potentiometers. The subject was strapped to a rigid chair, and his right upper arm was secured in a horizontal position to ensure that motion of the humerus was minimized during the movement. The movement protocols were performed while keeping the arm in a sagittal plane (Fig. 1).

Two type of surface electrodes were used. BIC (short head), TRI (long head), and PRT EMGs were recorded using preamplified bipolar surface electrodes (IOMED; 5 by 1.8 by 0.7 cm, three 1.0 cm in diameter electrode surfaces 1.3 cm apart, 22 g, gain of 375, impedance 1 MOhms, CMRR greater than 100 dB at 60 Hz, filtering bandwidth 8 Hz–32 kHz, quiescent current 0.12 mA). ANC and PRQ EMGs were recorded using small surface bipolar electrodes (Beckman; Ag-AgCl, 4 mm in diameter). Due to the depth and/or density of the forearm muscles, fine wire intramuscular bipolar electrodes (50 μm platinum alloy wire with polyurethane insulation) were used for the BRA, SUP, and BRD muscles. Rubbing alcohol and abrasive cloths were used to prepare the skin before the surface electrodes were applied. The small surface and wire electrodes were amplified using the IOMED electrode amplifiers. A computer-based data-acquisition system (Ariel) was used to record the position and EMG signals at a sampling rate of 1000 Hz.

2.1.2 Data processing

The measured EMG signals were digitally filtered with 4th-order IIR filters (Barr and Chan 1986) using two procedures. The first was to determine the overall strength of the signal by zero-mean, band-pass filtering (50–200 Hz), full-wave rectifying, and low-pass filtering (10 Hz). The second was identical to the first except that the low-pass filtering was eliminated. Both signals were

<table>
<thead>
<tr>
<th>Protocol</th>
<th>Experimental final time (s)</th>
<th>Solution final time (s)</th>
<th>% difference of final times</th>
<th>Mean absolute error (deg)</th>
<th>Mean error (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>F@P</td>
<td>0.488</td>
<td>0.377</td>
<td>22.7</td>
<td>8.3/0.6</td>
<td>8.3/-0.5</td>
</tr>
<tr>
<td>F@N</td>
<td>0.518</td>
<td>0.262</td>
<td>49.4</td>
<td>8.8/2.7</td>
<td>8.8/2.2</td>
</tr>
<tr>
<td>F@S</td>
<td>0.437</td>
<td>0.299</td>
<td>31.5</td>
<td>5.8/5.2</td>
<td>5.8/5.2</td>
</tr>
<tr>
<td>E@P</td>
<td>0.448</td>
<td>0.336</td>
<td>25.1</td>
<td>5.8/3.8</td>
<td>-5.8/3.8</td>
</tr>
<tr>
<td>E@N</td>
<td>0.604</td>
<td>0.340</td>
<td>43.6</td>
<td>10.0/3.3</td>
<td>-9.4/-3.2</td>
</tr>
<tr>
<td>F+P</td>
<td>0.441</td>
<td>0.348</td>
<td>21.2</td>
<td>11.7/1.1</td>
<td>-11.7/0.3</td>
</tr>
<tr>
<td>F+S</td>
<td>0.437</td>
<td>0.349</td>
<td>20.1</td>
<td>4.7/3.5</td>
<td>4.7/-2.1</td>
</tr>
<tr>
<td>E+P</td>
<td>0.388</td>
<td>0.310</td>
<td>20.2</td>
<td>3.5/3.3</td>
<td>3.0/2.9</td>
</tr>
<tr>
<td>E+S</td>
<td>0.362</td>
<td>0.336</td>
<td>7.1</td>
<td>14.4/26.7</td>
<td>-14.4/-26.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.9/5.1</td>
<td>0.5/5.0</td>
</tr>
</tbody>
</table>
normalized to 70% of a maximum EMG value for each muscle determined from all the protocols measured. This 'maximum' EMG value was obtained by performing the identical processing procedure on each EMG signal in each protocol and then recording the resulting maximum value for each muscle across all the protocols. Seventy percent of the maximum EMG value was chosen to be the normalizing unit so that small muscle activity would not be excessively suppressed and to better visualize the relative EMG burst differences. The kinematic data were low-pass filtered (3 and 10 Hz for f/e and p/s, respectively) to eliminate high frequency noise.

2.2 Model calculations

2.2.1 Musculoskeletal modeling

The biomechanical model of the EJC described the system's neural, muscular, and skeletal components. The articulations modeled were the two-df movements of elbow f/e, forearm p/s, and the passive motion of abduction/adduction. Eight muscles were included in the model: BIC, TRI, BRA, BRD, PRT, PRO, SUP, and ANC (see earlier sections for abbreviations). For muscles with multiple heads (BIC, TRI), muscle parameters were lumped together by summing, averaging, or scaling anatomical values reported in the literature. The components included in this model were (a) excitation-contraction dynamics, (b) musculotendon dynamics (force-length and force-velocity), (c) skeletal dynamics (equations of motion), and (d) an optimal controller. To account for the subject’s strength, the model’s musculotendon parameters of maximum isometric muscle force (active component) and tendon slack length were calibrated to maximal isometric trials of both f/e and p/s. A complete description of this model can be found in Gonzalez et al. (1996).

2.2.2 Optimal control

A dynamic optimal control algorithm (Powell 1978; Pandy et al. 1992) was used to determine individual muscle activations\(^1\) based on minimum time solutions for the one- and two-df ballistic EJC movements starting from rest and ending at rest (i.e., zero velocity and zero acceleration). The performance criterion (J) was:

\[
J = \min \int_{t_0}^{t_f} dt
\]

under the equality and inequality constraints:

\[
q_i = q_{exp}^i; \quad \dot{q}_n = 0; \quad \ddot{q}_n = 0
\]

\[
0 \leq u_i \leq 1; \quad i = 1, 64
\]

where for each df, \(q_{exp}^i\) was the final experimental position and \(q_n, \dot{q}_n, \ddot{q}_n\) were the model's final position, velocity, and acceleration, respectively. Eight control points (\(u_i\)) were used for each of the eight muscles, with the 65th control point as the movement time (MT) (\(t_f\)). The initial guesses for \(u_i\) and \(t_f\) were based on inspection of the EMG and movement data. That is, given the processed EMG data, the initial activation conditions were matched, as closely as possible, to the EMG of each muscle. Consideration was given to the final experimental position, which was based on the forward integration of the controls (Gonzalez et al. 1996). No emphasis was placed on the final velocity or acceleration when establishing these initial activation conditions.

The one-df elbow movements (no p/s movement) had an additional equality constraint to penalize any p/s movement outside the initial and final experimental p/s positions. This constraint was implemented as follows:

\[
\text{Penalty} = \int_{t_0}^{t_f} \lambda \cdot dt
\]

where \(\lambda\) was determined by a series of qualifiers as follows:

\(^1\)The term 'muscle activation' is meant to be synonymous with the model's muscle control signal \(u(t)\) (i.e., muscle stimulation). Only a small difference exists between the control signal \(u(t)\) and the active state of the muscle (i.e., an exponential rise and decay). Nonetheless, because our comparisons are to muscles' EMG, we have termed our control signals as 'muscle activation.'
if $q_t^{p/s} > q_{up}^{p/s}$ then $\lambda = |q_t^{p/s} - q_{up}^{p/s}|$ else
if $q_t^{p/s} < q_{lw}^{p/s}$ then $\lambda = |q_t^{p/s} - q_{lw}^{p/s}|$ else
\[ \lambda = 0 \]

where: $q_{up}^{p/s}$ was the upper bound of the experimental p/s position,
$q_{lw}^{p/s}$ was the lower bound of the experimental p/s position, and
$q_t^{p/s}$ was the time varying p/s position predicted by the model.

2.2.3 Computational implementation

A $5^\circ, 75^\circ/s$, and 1000$^\circ/s^2$ difference from the experimental final kinematic conditions was considered sufficient for convergence in either f/e or p/s for the final joint position, velocity, and acceleration, respectively. The tolerance was determined by visual inspection of the raw position data. Time was normalized to MT. The model’s performance was evaluated by calculating the mean and absolute mean error for the time-varying joint position in both dfs.

The strength of the triphasic bursts was calculated by averaging the values of the processed-normalized EMG signal and predicted activations over normalized time intervals. These intervals were: A burst, 0%-43%; B burst, 43%-86%; and C burst, 86%-100% of MT. Interval lengths were determined based on visual inspection of the EMG signals in the ten protocols, the length of the interval between activation nodes used by the model (one-seventh or 14% of MT), and the findings of Brown and Gillear (1991) on triphasic burst durations.

3 Results

Both one-df movements of elbow f/e and two-df movements of elbow f/e with forearm pronation or supination are presented for subject no. 1. The experimental data and the model’s minimum time solution for both the joint kinematics and activations for the eight muscles are shown for two protocols (F@P and F+S; Fig. 2–5). These are representative of the performance by both subjects. These two protocols were also chosen because they denote how activating a muscle that is antagonistic or noncontributory to the movement further minimizes the MT.

MTs for the model were always smaller than for the subject. Differences ranged between 7% and 49% with the majority between 20% and 32% (Table 1). The mean and absolute mean errors were under 10$^\circ$ for 85% of the two dfs (omitting f/e: E@S, E+P; p/s: E+P) and 60% of these trajectories were under $5^\circ$ (Table 1).

3.1 Individual protocol solutions

Only 9% of the constraints imposed on the movement protocols were not met by the model’s final solutions.

The constraints violated were the final f/e velocity in F@N, E@N, E@P, E+P, final p/s velocity in E+P, and final f/e acceleration in E@S. The model’s predicted movements which could not meet the constraints evidently occurred because the starting point on the solution space violated the velocity and acceleration constraints. Due to the large number of protocols and the substantial computational requirements for each solution, consistent initial values for muscle activations and weighting factors were chosen despite the resulting 9% of violations. Conclusions are not drawn from the modeled protocols for which the solution failed to meet the constraints.

3.2 Flexion in a pronated position (F@P)

The experimental and model position, velocity, and acceleration of elbow flexion with the forearm held in a pronated position (F@P) are shown in Fig. 2. The normalized-time position difference between the model and the subject averaged 8.3$^\circ$ for f/e and 0.6$^\circ$ for p/s. The model’s peak f/e velocity was larger than the subject’s, indicating a smaller MT. Note that the acceleration for the model’s f/e was asymmetrical, while the subject’s was symmetrical.

Predicted muscle activations and experimental EMGs are shown in Fig. 3. The EMG A burst is seen in BIC, BRA, and BRD; the B burst in ANC; and the C burst in BRA. The model also indicated three bursts. A good fit between the model activation and the EMG is seen in the first half of the movement in BIC and BRD. BRA activity was predicted throughout the movement because of the BRA pure f/e movement resulting in a minimized MT. The model predicted a C burst in BIC, BRA, and BRD to clamp the final f/e position. TRI and ANC activity were only predicted in the last half of the movement to brake the flexion movement (B burst), yet an increased amount of EMG activity is seen in ANC versus TRI. Nonetheless, because of the size and mechanical advantage of TRI, the extension movement of TRI was larger than that of ANC. The C burst was more delayed in the model than in the subject, which produced the model’s asymmetrical f/e acceleration. The reason for the delayed C burst in the model was the optimal controller’s exploitation of the extension moment produced by gravity and lack of practice of the subject. SUP activity was predicted by the model yet was not seen in the subject. This SUP activity in the model allowed for an increased PRT activity, which led to an increased amount of flexor moment. The model substantially increased the PRT activity, a flexor and pronator, to accelerate the flexion movement. The PRT’s pronation moment was opposed by the supination moment of the SUP as represented by the SUP activity in the latter half of the movement. The PRQ activities were similar throughout.

3.3 Flexion and supination (F+S)

A simultaneous ballistic flexion and supination movement trajectory (F+S) is shown in Fig. 4. The absolute
differences between the model and subject for the f/e and p/s position trajectories were 3.5° and 3.4°, respectively. The peak velocities for the model were larger for f/e and p/s, and occurred approximately at the same relative time for p/s and slightly later for f/e. Note that the acceleration for the model’s f/e is asymmetrical, while the subject’s is symmetrical.

Model activations and EMGs are shown in Fig. 5. The EMGs and the model activations contain a triphasic pattern in flexion and supination, with the respective B and C bursts being much larger in the model. EMGs and predicted activations are similar in BIC, BRA, BRD, SUP, and PRQ. The differences between BIC, BRA, and BRD EMGs and model activations were due to model effects on the p/s moment (i.e., the model is minimizing the MT by maximizing the flexion moment at the expense of the pronation moment, see Discussion). For example, because BRA does not produce any p/s moment, BRA activation was present throughout the movement, in the model and subject, whereas BIC and BRD shut off in the mid-range of the movement. Likewise, in the model and subject, SUP activity was also present throughout the movement. This SUP activity generated a supination moment to attain a forearm supinated position. Activity was also predicted in the pronators (PRT, PRQ) but to a larger degree than measured in the subject. This increased PRT and PRQ activity in the model was used to counteract some of the supination torque produced by BIC, BRD, and SUP. The model here
indicates how a co-contraction can assist in reducing MT (see Discussion).

3.4 Strength of triphasic bursts

The relative intensities of the triphasic bursts for the subject and model are shown for BIC and TRI (Fig. 6). To demonstrate the relative burst of muscle activity, each burst’s average was normalized to the largest corresponding burst observed in the muscle across all ten protocols. This normalization was performed to demonstrate more clearly the relative differences in the strength of the signals between bursts and between movement protocols (Brown and Gilleard 1991). In general, the model predicted the same relative activations as the experimental data in all three bursts. The subjects generally had smaller B and C EMG bursts because the indwelling electrodes and experimental setup limited the amount of practice the subjects could have (Darling and Cooke 1987; Moore and Marteniuk 1986; Mustard and Lee 1987).

The A burst in the subject and model compare favorably for BIC (Fig. 6). This was especially evident in the large activation averages associated with F@N, F@S, and F+S and the mid-level averages of E@S, E+S, F@P, and F+P given by the model. The average BIC activation was larger with forearm supination and smaller with forearm pronation for both flexion and extension in the subject and the model. The level of BIC’s B burst activity generally corresponds to where the subject also shows activity. Clearly in the E@S and E+S protocols, BIC was braking the movement, and the lower BIC activities of the other extension protocols are illustrative of the final f/e velocity constraint going unmet. The lower B bursts in the subject’s E@P and E+P indicate that a strong BIC braking activity would create an indirect supination moment and affect the forearm pronated position. The level of BIC’s C burst activity shows less agreement between the model and subject.

The averages of the TRI A and B bursts for the subject and model are alike. The subject had small amounts of co-contraction in the flexion protocols, while the model showed none (Fig. 7), which is in agreement with the findings of Moore and Marteniuk (1986) that with practice, co-contraction decreases. Both the subject and model showed that the magnitude of the TRI A burst was not notably affected by forearm position or movement (Sergio and Osty 1994, 1995). The large B burst of the model in E@N was due to the model failing to find a minimum time solution. C bursts showed less agreement.
The A bursts for six of the eight muscles are shown in Fig. 7 and indicate the extent to which the model and subject initially activated each muscle in each protocol. BIC envelopes are very similar except that the model’s BIC activity was twice as large in F@N. The indirect supination moment produced by the model’s BIC in F@N was countered by increased PRT activity. Lower BIC activities for pronated positions or movements are seen in both the subject and model. BRA patterns were also similar, but the model’s magnitudes were higher, except for F@P. The increase in the model’s BRA magnitude in F+P was used to increase the flexion moment and therefore reduce the MT. The magnitude of the model’s BRA A burst at F@N was lower than in other flexion movements because of the initial activation conditions. BRD patterns for the model and subject were similar and fairly consistent for flexion activities but were larger for the model. BRD activity in F@S and F+S were larger in the model because it opposed the secondary supination moment of BRD with PRT and PRQ moment. The envelopes of TRI were similar, yet in contrast to the other muscles, the model’s magnitudes were slightly smaller. The SUP activity patterns showed one major difference: The amount of activity produced by the subject in E@S. To maintain a fully supinated forearm position during extension, SUP was active in the subject to oppose the passive pronation moment which limits the forearm’s range-of-motion. Supination moment was not needed in the model because the passive joint moments created by nonmuscular structures were not included. PRT activity was always directed toward flexion. To minimize the MT in F+S, the model

Fig. 4. F+S measured and predicted trajectory. Experimental (thin) and model predicted (bold) position, velocity, and acceleration trajectories are shown for elbow f/e (solid) and forearm p/s (dashed) normalized with respect to its final time. Zero degrees is full elbow extension and full forearm pronation.
created a larger flexion acceleration moment by activating PRT (a flexor and pronator) and opposed the pronation moment with increased SUP activity.

4 Discussion

The purpose of this study was to provide a detailed comparison between minimum-time model-based muscle activations and processed EMG during multi-df ballistic movements at the elbow. The model was able to predict both the relative magnitudes and the timing of the agonist-antagonist muscle pairs along with the major features of muscle activity associated with two-df movements at the elbow.

4.1 Limitations

There are several limitations to this study. Large variations of the model’s p/s accelerations were due to low p/s inertial characteristics of the forearm and the intermediate constraint used in the one-df elbow flexions to keep the p/s position inside the imposed bounds. The discontinuities seen in the predicted p/s accelerations were also caused by the introduction of via points in the computed moment arms of most of the pronator and supinator muscles (Gonzalez et al. 1996). However, these discontinuities did not alter the major features of the joint’s acceleration. Passive moments created by nonmuscular structures at the limits of the joint’s range of motion were not included in the model. This is because the movements modeled did not approach these limits (with the one exception E@S).

The model consistently delayed the onset of the braking burst for TRI and ANC in flexion movements and thus developed an asymmetrical f/c acceleration. In contrast, Darling and Cooke (1987) reported that with increased movement speeds, the onset of antagonistic or braking bursts comes earlier in the movement, and Moore and Marteniuk (1986) reported that with increased practice, the acceleration profiles become symmetrical. Several explanations could account for these differences. First, while the model was calibrated to subject strength via maximum isometric contractions, the model appears to be ‘dynamically’ stronger in extension when compared with flexion, and could brake the flexion more quickly and thus closer to the end of the movement. Second, ballistic elbow movement studies have been performed in the horizontal plane, while this study was performed in the vertical plane. Therefore, the model uses gravity in braking the flexion movements.
Third, it is possible that the onset of the braking burst occurs earlier in faster movements with the same amplitude because of the elbow’s agonist-antagonist neural coupling, which was not meant to be represented in the EJC model.

4.1 One-df movements

Both the subjects and the model adjusted the activity of the flexors (BIC, BRA, BRD) with forearm position. These results are consistent with the findings of other investigators of muscle activity with forearm position changes (Buchanan et al. 1989; Funk et al. 1987; Jamison and Caldwell 1993; Sergio and Ostry 1994; Stewart et al. 1981). Both subject and model strongly activated the BIC in F@S and F+S. The subject reduced the BIC activation for F@N, F@P, and F+P where a large BIC activity would have produced a secondary unmatched supination torque. The model was able to keep a large BIC activation in F@N while opposing the secondary supination moment of BIC with PRT and PRQ activity.

Although Buchanan et al. (1989) found that the activity in BRA and BRD decreased with increased BIC activity, the model did not indicate such a decrease for both BRA and BRD. Funk et al. (1987) reported that the flexor muscles are most active in the neutral forearm position, yet this was not evident in this study’s model or experimental results, nor according to Buchanan et al. (1989). The BRA EMG magnitude order was similar to Stewart et al. (1981), but the model predicted that BRD is equally active in all forearm positions.

In the F@P protocol, it was observed that SUP activity allows more activation of PRT, which provides increased flexion moment and therefore reduces the MT. The large co-contraction of SUP and PRT enhanced the performance by reducing the MT. We are confident of this because during preliminary trials, without the p/s constraint, the model activated the PRT muscle to increase flexion moment, causing the forearm to pronate, then returned the forearm to the initial position at the end of the movement. This protocol shows that a muscle that is noncontributory (SUP) to a movement can shorten the MT of a ballistic movement.

4.3 Two-df movements

The extensors (TRI, ANC) were fundamentally unaltered in the model and experimental data by forearm position or by simultaneously pronating or supinating during elbow extension, which corroborates Sergio and Ostry’s (1994, 1995) conclusion that TRI activity is not affected by forearm position or movement. However, the
flexors (BIC, BRA, BRD) were affected by the requirement to simultaneously pronate or supinate the forearm in both the model and subject. The largest change was evident in BIC. BIC was activated at less than 50% of maximum in F+P for both subject and model, while BRA and BRD were more fully activated in the model and BRD in the subject. In the F+S movement, the subject’s BRA was not activated as strongly as BIC and BRD. This result is in contrast to Sergio and Ostry’s (1994) conclusion that BRD is not adversely affected by forearm motion.

A more pronounced activity of PRT and BRD was observed for both subject and model in F+P as compared with E+P. This was probably due to the supination moment produced by BIC in the process of flexing the elbow, which therefore required pronation opposition. The activity of PRQ was similar in E+P and F+P (Sergio and Ostry 1994).

Examination of the SUP EMG in the E+S and F+S protocols showed that the subject more fully activated SUP in E+S than in F+S, while the model had similar levels of activity for SUP in both E+S and F+S. This SUP difference between the subject and model was due to the large PRT activity in the model used to minimize the MT in F+S which, therefore, required additional supination torque. Once again, the model’s F+S solution shows how a muscle antagonistic to the supination requirement (PRT) can reduce the final MT since elbow flexion is the determining factor in minimizing time. In the E+S movement, the model strongly activated BIC (as compared with the subject) to brake the extension and thus produce a secondary supination moment which was countered with some pronator activity (PRT, PRQ). Again, antagonist muscles (PRT, PRQ) helped reduce the MT by allowing more BIC braking moment. The predicted BIC activity during E+S and the mild activation of PRT during E+P was also found by Buchanan et al. (1989). They noted that BIC remained active when forearm supination torque was performed with elbow extension torque and that the BIC muscle activity increased substantially when supination was performed with elbow flexion.

5 Conclusions

The results of this musculoskeletal model of the EJC compared quite favorably with experimental data. It can be deduced that the modeling approach, when executed carefully and accurately, can be used to gain insight into important motor control and biomechanical issues. For example, this model demonstrated how muscles that cross the EJC tend to be activated based on biomechanical parameters (i.e., forearm position and movement) rather than on any particular synergistic rule. This has also been found by others in nonballistic studies (Buchanan et al. 1986, 1989; Jamison and Caldwell 1993; Sergio and Ostry 1994, 1995). In agreement with Sergio and Ostry’s (1994, 1995) results, our model’s muscle activation patterns replicated the major features

Fig. 7. Agonist muscle activation used to accelerate the forearm in both model (bold) and subject (thin) for six muscles and all ten protocols is shown. Each radial line represents a specific movement protocol, and the strength of the signal is shown by its radial magnitude. Length of radial axis is 1 unit (i.e., full activation) with hash marks at the midpoints. See Methods for abbreviations.
of their findings. First, our model, showed that changes in the amount of muscle activity for muscles that contribute to both flexion and p/s depended on the motion of the joint. Second, one-df muscles (SUP, BRA) were affected by the kinematic requirements in a secondary df. Third, activity levels of two-df muscles were larger when the motion performed by the model used the muscles as agonists in both dfs. This is due to the coupling of joint torques produced by the multi-df muscles, which sometimes produces secondary moments which can be opposed by other muscles. Fourth, the relative magnitudes of activation between muscles change with motion in a second df. Fifth, TRI muscle activity was not affected by the orientation or motion of the p/s df. Our study appears to indicate that the patterns of muscle activity in multi-df movements are primarily based on the geometry of the joint. An unanticipated finding was that the model predicted how noncontributory or antagonist muscles could reduce MTs in certain ballistic movements (F@P, F+S, E+S) by allowing multi-joint muscles to have stronger contractions (activity). While it may be tempting to assume elbow flexor muscles act in synchrony during elbow movements and therefore can be replaced by a ‘flexor equivalent’ when needed to reduce the mechanical redundancy, the synchrony concept is a poor assumption. There is little evidence to support muscle synchrony for muscles crossing the elbow joint during elbow flexion and extension, except possibly the extensors, because nonextensor muscles are affected by the position and movement of the forearm.

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