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Antagonist muscle coactivation during isokinetic knee extension

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The aim of the present study was to quantify the amount of antagonist coactivation and the resultant moment of force generated by the hamstring muscles during maximal quadriceps contraction in slow isokinetic knee extension. The net joint moment at the knee joint and electromyographic (EMG) signals of the vastus medialis, vastus lateralis, rectus femoris muscles (quadriceps) and the biceps femoris caput longum and semitendinosus muscles (hamstrings) were obtained in 16 male subjects during maximal isokinetic knee joint extension (KinCom, ROM 90–10°, 30° · s^{-1}). Two types of extension were performed: [1] maximal concentric quadriceps contractions and [2] maximal eccentric hamstring contractions. Hamstring antagonist EMG in [1] were converted into antagonist moment based on the EMG-moment relationships determined in [2] and vice versa. Since antagonist muscle coactivation was present in both [1] and [2] a set of related equations was constructed to yield the moment/EMG relationships for the hamstring and quadriceps muscles, respectively. The equations were solved separately for every 0.05° knee joint angle in the 90–10° range of excursion (0°=full extension) ensuring that the specificity of muscle length and internal muscle lever arms were incorporated into the moment/EMG relationships established. Substantial hamstring coactivation was observed during quadriceps agonist contraction. This resulted in a constant level of antagonist hamstring moment of about 30 Nm throughout the range of motion. In the range of 30–10° from full knee extension this antagonist hamstring moment corresponded to 30–75% of the measured knee extensor moment. The level of antagonist coactivation was 3-fold higher for the lateral (Bfcl) compared to medial (ST) hamstring muscles. The amount of EMG crosstalk between agonist–antagonist muscle pairs was negligible (R_{XY}^2<0.02–0.06). The present data show that substantial antagonist coactivation of the hamstring muscles may be present during slow isokinetic knee extension. In consequence substantial antagonist flexor moments are generated. The antagonist hamstring moments potentially counteract the anterior tibial shear and excessive internal tibial rotation induced by the contractile forces of the quadriceps near full knee extension. In doing so the hamstring coactivation is suggested to assist the mechanical and neurosensory functions of the anterior cruciate ligament (ACL).

Isokinetic dynamometry has been widely used to obtain the maximal muscle moment generated at specific joints. However, if antagonist muscles are coactivated the measurement reveals only the *net* moment generated at the joint, i.e. the moment generated by the agonist muscles minus that of the antagonist muscles. In the present study a method is described by which it is possible to estimate the *gross* moments produced by the agonist and antagonist muscle synergies crossing the knee joint. In essence the method is based on the processing of muscle electromyography (EMG) to derive contractile moment of force.

During isokinetic knee extension the contractile forces of the quadriceps muscle may create an anteriorly directed shear of the tibia relative to the femur, especially at more extended knee joint angles and at high levels of muscle force (Beynnon et al. 1992, Hirokawa et al. 1992, Kaufman et al. 1991, Nisell et al. 1989). Antagonist coactivation of the knee flexors (hamstrings) has been suggested to counteract this shear, thereby providing stability to the knee joint (Baratta et al. 1988, Draganich & Vahey 1990, Renström et al. 1986, Solomonow et al. 1987). Interestingly, a neural reflex pathway elicited by anterior cruciate ligament (ACL)-stress and activating the hamstring muscles has been demonstrated in animal preparations (Solomonow et al. 1987). Similar ACL-mediated reflex pathways were recently observed in humans (Dyhre-Poulsen & Krogsgaard 1999). However, the capacity for joint stabilization by means of antagonist hamstring coactivation may seem limited since the hamstring muscles appear not to be nearly as strong as the quadriceps muscle. Thus, hamstring/quadriceps strength ratios (H/Q) of 40–50% typically have been observed across velocities and contraction modes (Aagaard et al. 1995, 1996). However, we have

suggested that the potential for muscular knee joint stabilization during forceful dynamic knee extension may be more appropriately described by the ratio of maximal eccentric hamstring strength to concentric quadriceps strength (Agaard et al. 1995, 1996, 1998). H/Q strength ratios of 90–130% have been reported based on this functional concept, indicating a significant capacity of the hamstring muscles to provide joint stability during dynamic knee extension (Aagaard et al. 1995, 1996). This capacity for muscular joint stabilization is progressively augmented in those situations where potentially high ACL stresses are expected, i.e. at more extended knee joint positions and at high angular velocity (Aagaard et al. 1998). Evidence of hamstring muscle coactivation during forceful quadriceps contraction has been reported for isokinetic knee joint extension (Amiridis et al. 1996, Baratta et al. 1988, Bobbert & Harlaar 1992, Hagood et al. 1990, Kellis & Baltzopoulos 1996a, 1997) as well as for more complex movements (More et al. 1993, Walla et al. 1985). Only few of these studies have estimated the actual muscle forces and/or moments generated by antagonist coactivation of the hamstrings. Based on EMG measurements, Baratta and coworkers (Baratta et al. 1988) estimated that during slow isokinetic knee extension the magnitude of antagonist hamstring moment may correspond to 5–8% of the flexor moment produced during maximal agonist contraction of the hamstring muscles. However, in their study as in several others (Amiridis et al. 1996, Osternig et al. 1986), antagonist hamstring EMG was normalized relative to the agonist EMG obtained during *concentric* contraction. Thereby, eccentric antagonist moments were estimated from concentric EMG-moment relationships. Since a given level of EMG is associated with significantly larger muscle moments during eccentric compared to concentric contractions (Kellis & Baltzopoulos 1997), antagonist hamstring moments were underestimated in the studies mentioned above. In consequence, eccentric moment-EMG calibrations should be employed to estimate the eccentric antagonist hamstring moments exerted during isokinetic knee extension. Based on such an approach, we have earlier presented data (Aagaard et al. 1994) using the specific methodology reported in the present study, where dual sets of EMG-moment relations are derived separately throughout the range of knee joint motion to account for the presence of any antagonist coactivation in both the hamstring and quadriceps muscles. These early data were recently confirmed by Kellis & Baltzopoulos (1997) using a similar approach. However, in their study the possible involvement of quadriceps coactivation during the procedures of hamstring EMG-moment calibration was not accounted for.

The aim of the present study was to quantify the agonist moment generated by the knee extensors and the antagonist moment generated by the knee flexors during slow isokinetic knee extension.

Subjects and methods

Subjects

Sixteen sedentary males (body mass 73.2±5.8 kg, height 179±4 cm, age 23.5±3.4 yr, mean±SD) participated in the study. All subjects gave their informed consent to the conditions of the experiments. No subjects had any history of previous knee injury.

Isokinetic dynamometry

Maximal concentric and eccentric knee joint moments were measured by use of a Kin-Com dynamometer (Kinetic Communicator, Chattecx Corp., Chattanooga, USA) (Farrell & Richards 1986). Knee joint angular velocity was $30° \cdot s^{-1}$ while joint range of motion (ROM) was 90° to 10° (0°=full knee extension). Two types of isokinetic knee extension were performed: [1] concentric contraction of the knee extensors (quadriceps agonist, hamstrings antagonist) and [2] eccentric contraction of the knee flexors (hamstrings agonist, quadriceps antagonist). Both contraction conditions involved knee extension movements. In [1] the quadriceps actively generated an extension moment and in [2] the hamstring muscles tried to oppose the motordriven movement of the dynamometer lever arm by actively generating a flexion moment.

Subjects were seated in a rigid chair with a 10° reclined backrest and firmly strapped to the seat at the hip and distal thigh. The dynamometer rotation axis was visually aligned to the lateral femoral condyle. The lower leg was attached to the lever arm of the dynamometer 1 cm above the medial malleolus with no static fixation of the ankle joint. A 15-min warm up and preconditioning to the testing device was performed prior to data sampling. On separate occasions subjects were familiarized with the dynamometer and the measuring procedures. Successive trials were performed at each contraction mode until the subject was unable to increase peak moment any further. To fulfil this criterion 6 or 7 trials typically had to be performed. The final three trials performed at each contraction mode were used for analysis. All recorded force and moment signals were corrected for the effect of gravity on the lower leg according to procedures described previously (Aagaard et al. 1995).

EMG recordings and analysis

EMG signals were obtained by use of bipolar surface electrodes (Medicotest Q-10-A) placed 2 cm apart at the m. vastus lateralis (VL), m. vastus medialis (VM), m. rectus femoris (RF), m. biceps femoris caput longum (Bfcl, lateral hamstrings), m. semitendinosus (ST, medial hamstrings). For the VL muscle electrode pairs were placed 15 cm above the patella, for VM 13 cm above the patella, for RF 20 cm above the patella and for ST and Bfcl 25 cm above the popliteal fossa. The skin of the subject was cleaned with abrasive swabs and subsequently rinsed with alcohol to increase conductivity and reduce electrode-skin impedance. The EMG electrodes were connected directly to small custom-built preamplifiers taped to the skin. The EMG signals were led through shielded wires to custom-built amplifiers with a frequency response of 10 Hz to 10 kHz and common mode rejection ratio >100 dB. The preamplifiers lowered the impedance, which effectively prevented movement artifacts. Neither passive movements nor tapping the leg produced any visible artifacts. No significant cross-talk was observed between EMG channels (see details below).

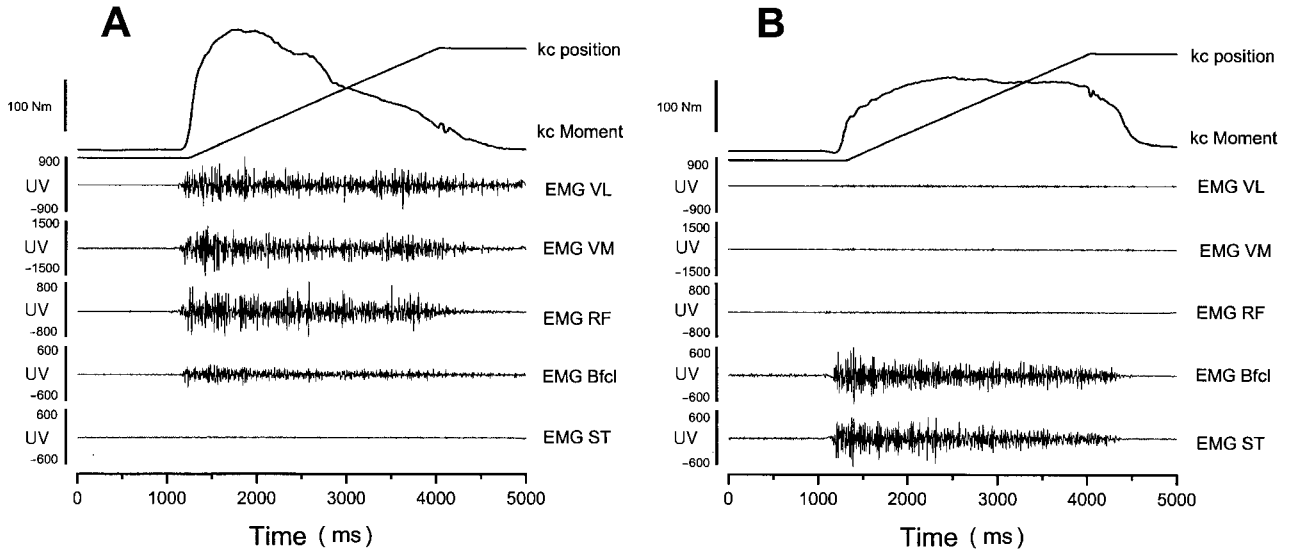

Fig. 1. Representative isokinetic knee extension trials obtained during agonist action of the quadriceps (A) and hamstring (B) muscles, respectively. Top graphs show net knee extension moment (kc moment) (A) and net flexion moment (B) as well as knee joint angle (kc position, from 90° to 10°, units not shown). Note the identical contraction modes between conditions A and B for each muscle (quadriceps concentric, hamstrings eccentric). Bottom graphs show the EMG activity of the quadriceps (VL, VM, RF) and hamstring (Bfcl, ST) muscles. In A, during agonist quadriceps contraction, the EMG of the antagonist Bfcl muscle corresponded to about 30% of its agonist EMG (shown in B). In the trial of agonist hamstring contraction (B), only slight coactivation was seen for the antagonist VL muscle.

Signal treatment

Synchronous sampling of the KinCom strain gauge signal, KinCom lever arm position and EMG signals were performed at 1 kHz analog-to-digital conversion rate (dt2801-A converter, Data Translation, Marlboro, MA). The dynamometer signal was converted to Newton and multiplied by the individual lever arm length to calculate moment of force. All recorded moments were corrected for the effect of gravity on the lower limb (see above). All EMG signals were digitally highpass filtered at 5 Hz and lowpass filtered at 200 Hz (4th order, zero-lag Butterworth filter) and subsequently smoothed by a moving RMS average with a time constant of 20 ms.

Calculation of hamstring and quadriceps muscle moments

Two types of knee extension movements were performed: [1] maximal concentric quadriceps (Q) contraction (see Fig. 1A) and [2] maximal eccentric hamstring (H) contraction (Fig. 1B). Based on the moment and EMG recorded in [2], the relation between hamstring EMG and its resulting flexor moment could be established for any given joint angle throughout the ROM. However, in situation [2] some antagonist muscle activity was also present (i.e. slight quadriceps coactivation, as illustrated in Fig. 1B). Therefore, the conversion of antagonist EMG into moment of force had to be established by transforming the two knee extension conditions into a set of dual equations (equations (Aagaard et al. 1994) and (Aagaard et al. 1994) related to conditions [1] and [2], respectively) with two mutual unknowns (the 'EMG-to-force' conversion factors for the quadriceps and hamstring muscles, respectively). It was assumed that at any knee joint angle the measured *net* joint moment was determined by the moment generated by the agonist muscles minus that of the antagonist muscles. Accordingly, the following relationships were established for the EMG and the net extension moment M_1 obtained in [1] (Q agonist, H antagonist) and the EMG and the net flexion moment M_2 obtained in [2] (H agonist, Q antagonist), with EMG-to-moment constants K1 and K2:

$$M_1 = K_1 \cdot EMG_{Q,\,agonist} - K_2 \cdot EMG_{H,\,antagonist} \quad [1]$$

$$M_2 = K_2 \cdot EMG_{H,\,agonist} - K_1 \cdot EMG_{Q,antagonist} \quad [2]$$

For any given knee joint angle (Θ), solving eqs. [1] and [2] to yield K_1 and K_2 allowed the antagonist and agonist muscle moments to be determined (see *Appendix*):

$M_Q(\Theta) = K_1(\Theta) \cdot EMG_Q(\Theta)$; gross quadriceps extension moment

$M_H(\Theta) = K_2(\Theta) \cdot EMG_H(\Theta)$; gross hamstring flexion moment

For each subject, K_1 and K_2 were calculated at specific contraction modes (K_1, Q: concentric; K_2, H: eccentric), at identical joint angular velocity ($30° \cdot s^{-1}$) and separately for every 0.05 knee angle between 10° and 90°. This ensured that the specificity of contraction mode, muscle length, contraction velocity and lever arm length were incorporated into the EMG-to-moment relationships.

Typically, 6 or 7 trials were performed by each subject at each of the two contraction modes. Antagonist and agonist muscle moments were calculated and averaged for the final three trials. The VL, VM and RF muscles were assumed to each contribute one-third of the total quadriceps knee extensor moment and the Bfcl and ST muscles assumed to each contribute one-half of the total hamstring flexor moment. Prior to being fed to the set of equations the EMG signal of each muscle was normalized relative to its average EMG amplitude obtained during agonist contraction, thereby avoiding that the various muscle EMG signals should contribute to eqs. [1] and [2] in proportion to their numeric EMG amplitude size (in mV).

EMG crosstalk between agonist–antagonist muscle pairs

Antagonist EMG signals may potentially have been contaminated by the EMG of adjacent agonist muscles, as a result of EMG crosstalk between electrode pairs (Koh & Grabiner 1992, 1993). To examine if this was the case, the amount of EMG crosstalk between hamstring and quadriceps EMG recordings were quantified by use of cross-correlation analysis (Basmaj-

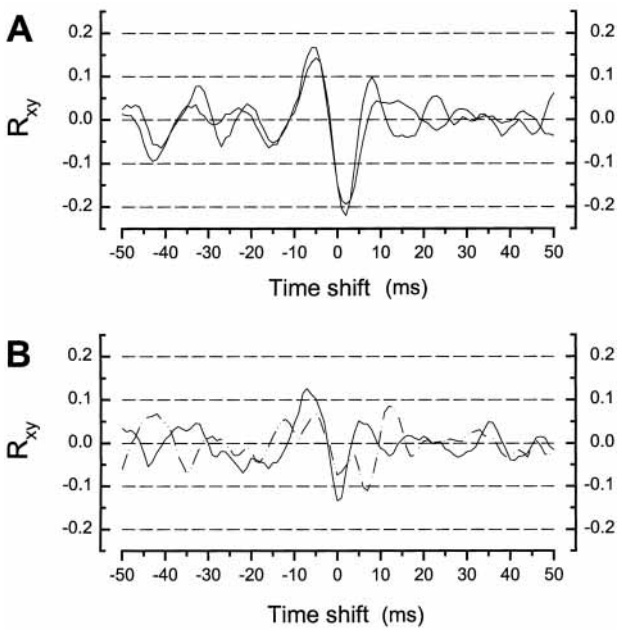

Fig. 2. EMG cross-correlation analysis. A: cross-correlating EMG signals of the VL and Bfcl in two successive trials of agonist quadriceps contraction (subj FH). Peak R_{XY} were 0.220, 0.194 and squared R_{XY} were 0.048, 0.038. Peak R_{XY} values averaged for all subjects are given in *Results*. B: cross-correlation functions shown for subject with a high degree of antagonist hamstring coactivation (subj MM, full line) and low hamstring coactivation (subj TR, broken line). Peak and squared R_{XY} were 0.134 and 0.018 (MM), 0.110 and 0.012 (TR).

ian & DeLuca 1985, Winter et al. 1994). The peak cross-correlation coefficient, $R_{XY}(\tau)$, raised to the second power represents the percentage crosstalk between electrode sites (Winter et al. 1994), this including the presence of any true synchronization between motor units. $R_{XY}(\tau)$ was calculated for all analyzed VL-Bfcl and VM-ST EMG signals, respectively, using a wide range of time shifts (from $\tau=0$ to $\tau=\pm 50$ ms, $d\tau=1$ ms) and long record lengths (phase of active contraction: about 2500 data points) as recommended by Winter et al. (1994). Average values were calculated for peak R_{XY} and R_{XY}^2. Representative cross-correlation functions are shown in Fig. 2.

Statistics

The Friedman two-way analysis of variance by ranks for related, multiple samples was used to test the variation in agonist–antagonist muscle activation (EMG) and muscle moments across knee joint positions.

Results

Antagonist muscle coactivation

The amount of antagonist coactivation always was greater in the lateral hamstring muscle (Bfcl) compared to the medial hamstring muscle (ST), corresponding to approximately 30% (Bfcl) and 10% (ST) of the EMG obtained during agonist contraction (Tables 1 and 2, Fig. 1 show illustrative trials). Overall antagonist hamstring coactivation was 15–35% when normalized relative to its agonist EMG (Fig. 3, 4). Averaged in 10° intervals, antagonist hamstring coactivation was 23.1% (80–90° knee angle), 22.5% (70–80°), 21.1% (60–70°), 21.1% (50–60°), 21.8% (40–50°), 23.4% (30–40°), 26.0% (20–30°) and 30.6% (10–

Table 1. Average muscle EMG (iEMG/intergration time in 10° intervals, ±SEM) during trials of agonist quadriceps contraction, hamstrings antagonist

Knee joint angle	EMG vast lat (μV)	EMG vast med (μV)	EMG rect fem (μV)	EMG bic fem (μV)	EMG semit (μV)
10–20°	323.0±35.6	398.8±52.7	284.9±38.3	77.5±8.1	22.5±3.0
20–30°	354.3±37.6	468.1±64.1	335.9±41.8	84.8±9.1	23.7±3.1
30–40°	373.0±40.3	474.7±61.7	357.6±40.1	87.5±9.0	23.5±3.1
40–50°	358.8±34.1	446.6±59.8	357.2±40.2	86.5±8.8	23.7±3.2
50–60°	350.8±32.0	437.9±55.8	344.0±37.9	88.0±8.9	25.0±3.3
60–70°	364.8±27.7	470.2±61.4	340.0±36.3	93.6±10.0	28.7±4.3
70–80°	396.0±38.7	478.0±57.4	333.4±35.9	107.8±12.9	31.2±4.9
80–90°	391.4±45.6	491.0±59.7	314.1±36.0	115.7±14.5	35.7±5.0

Table 2. Average muscle EMG (iEMG/intergration time in 10° intervals, ±SEM) during trials of agonist hamstring contraction, quadriceps antagonist

Knee joint angle	EMG vast lat (μV)	EMG vast med (μV)	EMG rect fem (μV)	EMG bic fem (μV)	EMG semit (μV)
10–20°	18.1±2.5	50.3±9.5	11.5±1.4	226.5±34.3	327.2±51.9
20–30°	20.1±2.6	56.2±9.4	12.7±1.5	267.4±37.0	411.2±49.9
30–40°	20.3±2.3	58.5±8.9	13.5±1.4	289.3±38.8	440.0±50.4
40–50°	20.5±2.4	56.3±8.2	14.0±1.3	292.6±39.7	461.1±49.3
50–60°	20.3±2.2	58.0±8.3	14.1±1.3	305.8±39.2	484.2±57.0
60–70°	21.1±2.4	58.8±8.7	13.9±1.5	321.4±39.7	492.8±50.9
70–80°	21.6±2.6	58.3±8.7	13.6±1.6	340.1±39.3	504.2±51.6
80–90°	20.6±2.4	57.4±8.3	13.7±1.7	355.2±45.3	517.9±50.8

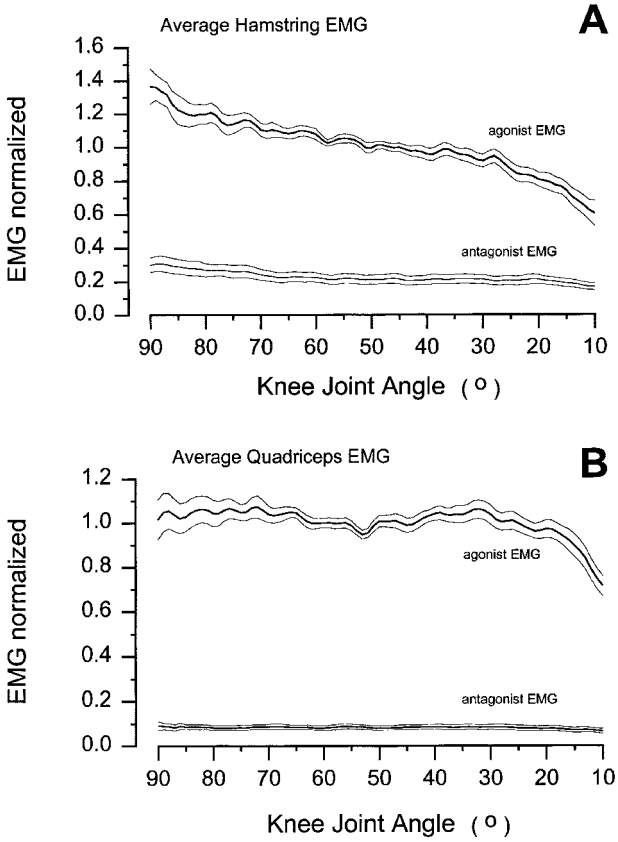

Fig. 3. Normalized agonist and antagonist EMG. A: mean hamstring EMG (Bfcl, ST) normalized relative to the average agonist hamstring EMG (iEMG/integration time) in the 90° range of motion (±SEM, n=16). For details see *Methods*. B: mean quadriceps EMG (VL, VM, RF) normalized to the average agonist quadriceps EMG in the 90–10° range of motion (±SEM, n=16).

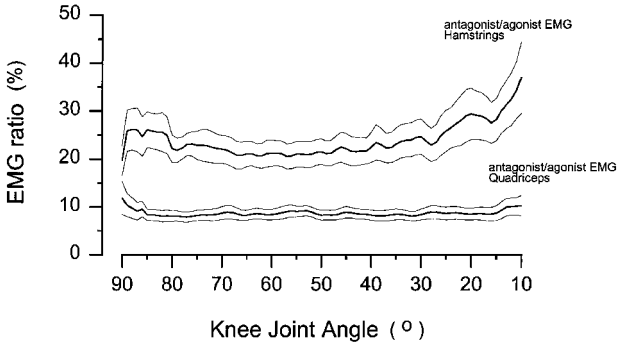

Fig. 4. Ratios of antagonist-to-agonist EMG (±SEM, n=16) for the quadriceps (VL, VM, RF) and hamstring (Bfcl, ST) muscles. All hamstring EMG signals were obtained during muscle lengthening (eccentric hamstring contraction) whereas all quadriceps EMG signals were obtained during muscle shortening (concentric quadriceps contraction).

20°) (Fig. 3, 4). Antagonist hamstring coactivation was greater towards full knee extension (10–30°) than in the midrange of joint movement (40–60°)

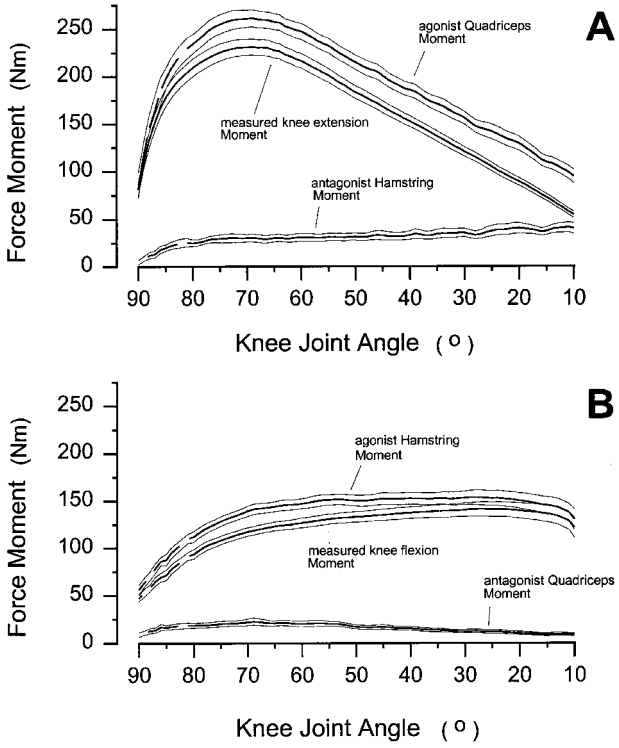

Fig. 5. Agonist and antagonist muscle moments averaged for all subjects (n=16, broken lines n=13). A: for knee extension trials with the quadriceps contracting as agonist and the hamstrings as antagonist. Graphs show the measured extension moment (±SEM), agonist muscle moment generated by the quadriceps muscle (±SEM) and antagonist muscle moment generated by the hamstring muscles (±SEM). B: knee extension trials with the hamstrings contracting as agonist and the quadriceps as antagonist. Graphs show measured flexion moment (±SEM), agonist muscle moment generated by the hamstring muscles (±SEM) and antagonist muscle moment generated by the quadriceps muscle (±SEM).

(P<0.01). For the quadriceps muscle the amount of antagonist coactivation was about 10% (Fig. 3, 4). Averaged in 10° intervals, antagonist quadriceps coactivation was 8.2% (80–90°), 8.2% (70–90°), 8.5% (60–70°), 8.8% (50–60°), 8.5% (40–50°), 8.3% (30–40°), 8.7% (20–30°) and 9.2% (10–20°) (Fig. 4).

Antagonist and agonist muscle moments

During quadriceps agonist contraction the measured knee extension moment as well as the calculated agonist quadriceps moment and antagonist hamstring moment all were observed to vary considerably throughout the range of motion (Fig. 5A). Averaged for successive 10° intervals in the joint range of motion, the measured knee extensor moment ranged between 72 Nm and 223 Nm while agonist quadriceps moment ranged between 111 Nm and 252 Nm and antagonist hamstring moment ranged between 29 Nm and 39 Nm (Table 3). During agonist hamstring

Table 3. Measured moment and calculated agonist–antagonist muscle moments obtained during quadriceps and hamstring agonist contractions (averaged in 10° intervals, ±SEM)

Knee joint angle	Quadriceps agonist			Hamstrings agonist		
	Measured moment (Nm)	Agonist Q moment (Nm)	Antagonist H moment (Nm)	Measured moment (Nm)	Agonist H moment (Nm)	Antagonist Q moment (Nm)
10–20°	72.1±3.6	111.3±6.4	39.2±4.5†	134.5±8.5	143.5±8.5	9.4±1.6‡
20–30°	104.8±4.4	141.6±7.2	36.9±4.9†	140.7±7.6	152.5±7.7	11.7±1.8‡
30–40°	137.0±5.3	171.2±7.1	34.2±4.5†	139.0±6.5	152.5±6.5	13.6±1.7‡
40–50°	168.2±6.2	200.3±7.5	32.1±4.0	135.3±5.9	151.6±6.0	16.3±2.1‡
50–60°	200.6±7.1	231.5±7.6	31.0±3.8	130.4±5.3	150.2±5.5	19.8±2.6
60–70°	226.5±7.8	256.0±8.2	29.9±3.8	122.5±4.7	143.9±5.2	21.4±3.0
70–80°	223.3±8.7	251.8±9.6	28.6±3.7	107.1±4.5	127.2±4.5	20.0±2.8
80–90°	164.9±8.6	182.8±9.6	17.9±2.8	72.3±3.8	87.7±4.3	15.4±2.3

† antagonist hamstring moments: 10–40°>40–90° ($P<0.001$).
‡ antagonist quadriceps moments: 10–20°<20–30°<30–40°<40–50°<50–80° ($P<0.001$).

contractions the measured knee flexion moment, antagonist quadriceps moment and agonist hamstring moment also varied across knee joint angles (Fig. 5B). Averaged in 10° intervals, the measured knee flexor moment ranged between 72 Nm and 141 Nm while agonist hamstring moments and antagonist quadriceps moments ranged between 88 Nm and 153 Nm, and 9 Nm and 21 Nm, respectively (Table 3).

Antagonist hamstring muscle moment was greater ($P<0.001$) at extended knee joint positions (10–40°) than at flexed positions (60–90°) (Table 3). Conversely, antagonist quadriceps moment decreased ($P<0.001$) towards full extension (Table 3). For the hamstring muscles a steep increase ($P<0.001$) in the ratio of antagonist-to-measured moment was observed towards full knee extension, whereas it remained less variant for the quadriceps muscle (Fig. 6). Averaged in 10° intervals, ratios of antagonist-to-measured moment were 10.4% and 21.3% for the hamstring and quadriceps muscles, respectively (80–90° knee angle), 12.8% and 18.7% (70–80°), 13.3% and 17.5% (60–70°), 15.5% and 15.2% (50–60°), 19.2% and 12.0% (40–50°), 25.2% and 9.8% (30–40°), 35.7% and 8.3% (20–30°), 55.9% and 7.0% (10–20°).

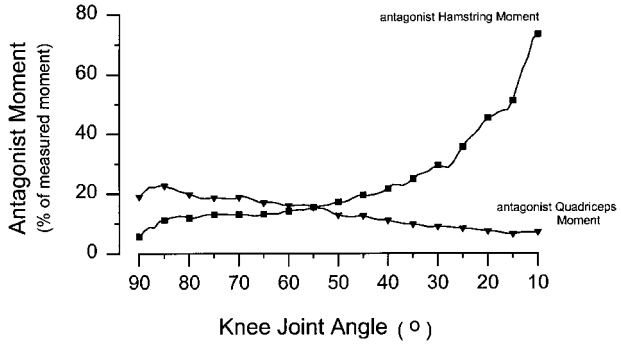

Fig. 6. Antagonist muscle moments expressed relative to the measured knee joint moment, averaged for all subjects ($n=16$). Squares: antagonist hamstring moment expressed relative to the net extension moment measured during agonist quadriceps contraction. Triangles: antagonist quadriceps moment expressed relative to the net flexion moment measured during agonist hamstring contraction.

EMG crosstalk

Cross-correlating the EMG signals of adjacent agonist–antagonist muscle pairs revealed average R_{XY} values of 0.245±0.035 (±SEM) and 0.197±0.032 for VL-Bfcl and VM-ST, respectively. The corresponding R_{XY}^2 were 0.060 and 0.039.

Discussion

Several notable findings emerged with the present study. Firstly, considerable coactivation of the antagonist hamstring muscles was observed during slow isokinetic knee extension during maximal volitional activation of the quadriceps muscle (agonist), and in consequence significant antagonist hamstring moments were generated. Secondly, it appeared that the lateral hamstring muscle (Bfcl) was coactivated to a greater extent than the medial hamstring muscle (ST).

Agonist and antagonist muscle moments

Significant amounts of antagonist hamstring EMG were observed throughout the range of joint motion (Table 1, Fig. 3A), as also reported by several previous studies (Amiridis et al. 1996, Baratta et al. 1988, Bobbert & Harlaar 1992, Hagood et al. 1990, Kellis & Baltzopoulos 1996a, Kellis & Baltzopoulos 1997). This hamstring coactivation resulted in opposing knee flexor moments of 15–40 Nm with the highest values observed near the end range of motion (Fig. 5A), where it corresponded to 45–73% of the

measured extension moment (Fig. 6, 20–10° knee joint angle). In the studies by Baratta et al. (1988) and Hagood et al. (Hagood et al. 1990), antagonist hamstring moment estimated from the amplitude of rectified antagonist EMG was essentially constant throughout the 90–10° range of joint excursion. In the present study, as in another recent study (Kellis & Baltzopoulus 1997), antagonist hamstring moment was also observed to remain constant across knee joint angles (Fig. 5A). However, when expressed relative to the maximal knee flexor moments measured during agonist hamstring contraction, antagonist hamstring moment was 2–4 fold higher in the present study compared to those of the above studies (20–25% vs 5–8%). This discrepancy can be ascribed to the fact that in these studies, as in previous others (Amiridis et al. 1996, Osternig et al. 1986), antagonist hamstring EMG was normalized relative to the hamstring EMG obtained during *concentric* agonist contraction. In consequence, eccentric antagonist moments were estimated from concentric EMG-moment relationships (Baratta et al. 1988). Since a given level of hamstring EMG yields 20–80% larger knee flexor moments during slow ($30° \cdot s^{-1}$) eccentric compared to slow concentric contraction (Kellis & Baltzopoulos 1997), the normalization procedure used in the above studies caused antagonist hamstring moments to be significantly underestimated.

Based on previous reports, considerable variation seems to exist for the magnitude of antagonist hamstring activity during isokinetic knee extension. Much of this variation, however, is explained by differences in EMG normalization procedures between studies. Thus, normalizing the amplitude of antagonist hamstring EMG (i.e. recorded during eccentric contraction) by that recorded in maximal *concentric* agonist contraction of the hamstrings resulted in an antagonist hamstring activity of 5–10% during slow maximal isokinetic knee extension in the study by Baratta and coworkers (1988). In comparison, antagonist hamstring coactivation was much higher in the present study (i.e. 15–35%, Fig. 4), where the antagonist EMG was normalized relative to the EMG recorded during agonist contraction of identical mode (H: eccentric, Q: concentric). Using EMG normalization procedures similar to those of the present study, Kellis & Baltzopoulos (1996a, 1996b) reported antagonist hamstring coactivation to be 15–25% for isokinetic knee extension at $30° \cdot s^{-1}$. Substantially lower levels of antagonist hamstring coactivation, however, were found in an earlier study (Snow et al. 1993) which also examined slow ($30° \cdot s^{-1}$) isokinetic knee extension and where the measure of hamstring coactivation (the ratio of antagonist-to-agonist EMG) was calculated in a manner identical to that of the present study, i.e. agonist and antagonist hamstring contractions were all eccentric. In the above study about 15% antagonist hamstring coactivation was observed in the range of 40–20° from full knee extension, with greater values observed in the initial phase of extension (approx. 20–30% at 70–50° knee joint positions (Snow et al. 1993)). Since calculated by the fraction of agonist to antagonist EMG, variations in antagonist muscle coactivation would arise from a change in either antagonist or agonist EMG amplitudes or both. Thus, to interpret variations in antagonist muscle coactivation, information on the agonist and antagonist EMG also should be given (Fig. 3). However, in the study mentioned above, no details were given on the levels of agonist or antagonist muscle EMG (Snow et al. 1993). Even though the quantification and interpretation of antagonist muscle coactivation clearly is not uncomplicated, this aspect does not influence the estimation of antagonist muscle *moments* as calculated in the present study and by Kellis & Baltzopoulos (1997). Thereby, the antagonist muscle moments derived seem to constitute a more realistic measure of the degree of antagonist muscle coactivation than that provided by the EMG analysis alone.

The major advantage with the methods used in the present study and in the study by Kellis & Baltzopoulos (1997) was that antagonist muscle moments were predicted from EMG-moment relationships determined during agonist contractions of exactly the same type (H: eccentric, Q: concentric). As pointed out by Kellis & Baltzopoulos (1997), this approach likely results in a better prediction of antagonist moments because it is based on real measurements and does not require any anatomical or mathematical assumptions on moment arm lengths, muscle lengths or muscle contraction velocity.

It should be noted that the methodology used in the present study differed from that of the above study. Firstly, during hamstring agonist contraction any amount of antagonist quadriceps coactivation was accounted for in the present study, whereas it was considered to be minimal and therefore neglected by Kellis & Baltzopoulos (1997). However, the antagonist coactivation and antagonist moments observed for the quadriceps muscle were relatively low compared to those observed for the hamstring muscles (present study, Kellis & Baltzopoulos 1997). Hence, neglecting the influence of antagonist quadriceps activity was unlikely to have resulted in large errors in the calculation of agonist and antagonist muscle moments. Secondly, in the present study the fractional nature of the EMG-to-moment algorithm implied a linear relationship to exist between the muscle EMG and muscle moment at a given knee joint angle. In contrast, Kellis & Baltzopoulos (1997) used second order polynomial fitting to obtain the EMG-moment relationship over a range of submaximal and maximal contraction intensities. However, as stated in their study, the relationship between hamstring EMG and

knee flexor moment was 'almost linear' (Kellis & Baltzopoulos 1997). Accordingly, only minor errors probably were introduced by the fractional (i.e. linear) relationships derived in the present study.

Despite the methodological differences mentioned above, the antagonist hamstring moments of the present study were remarkably similar to those reported by Kellis & Baltzopoulos (1997). For the 90–80–70° angle intervals we observed values of 18–29 Nm while these authors reported values of approximately 20–30 Nm (Kellis & Baltzopoulos 1997). At 70–10° knee joint angle the antagonist hamstring moments of 30–39 Nm also corresponded closely to the 30–40 Nm reported by these authors. Moreover, expressing antagonist hamstring moment as a fraction of the measured extension moment revealed values which corresponded very closely to those displayed by Kellis & Baltzopoulos (1997). Maximal measured knee extension moments also were highly similar in the two studies.

Differentiated patterns of antagonist hamstring coactivation

The present data (Table 1, Fig. 1) show antagonist coactivation to be 3-fold greater in the lateral hamstrings (m. biceps femoris, long head; Bfcl) compared to the medial hamstrings (m. semitendinosus; ST). At extended knee joint angles, contraction of the quadriceps muscle may result in substantial anterior translation and internal rotation of the tibia, which in turn can create significant stress load on the ACL (Beynnon et al. 1992, Hirokawa et al. 1992, Kaufman et al. 1991, More et al. 1993, Nisell et al. 1989, Renström et al. 1986). Due to its lateral insertion on the tibia the Bfcl muscle is capable of creating external tibial rotation. The pronounced Bfcl coactivation observed thus may represent a protective mechanism against the internal tibial rotation induced by the contractile forces of the quadriceps muscle. In contrast, pronounced coactivation of the ST muscle would not be desirable as this would add to the amount of internal tibial rotation as a result of its medial insertion at the tibia.

Coactivation of antagonist muscles

Several studies have examined antagonist hamstring coactivation and its role in providing dynamic knee joint stability (Baratta et al. 1988, Draganich & Vahey 1990, Kaufman et al. 1991, More et al. 1993, Renström et al. 1986, Solomonow et al. 1987, Yasuda & Sasaki 1987). The coactivation of antagonist muscles appears to be an inherent component in many types of joint movement (Smith 1981). At the knee joint, coactivation of the hamstring muscles has been shown to reduce the anteriorly directed shear of the tibia relative to femur that may occur during active knee extension (Draganich & Vahey 1990, Kaufman et al. 1991, Renström et al. 1986). The mechanical joint properties, such as joint stiffness, change with active joint moment (Milner & Cloutier 1993). However, this relationship may not be straightforward as the presence of agonist and antagonist muscles, which generate forces in opposing directions, allows for joint stiffness and net joint moment to be regulated separately and independently of each other (DeLuca & Mambrito 1987). While net joint moment is the difference between the moments exerted by the agonist and antagonist muscles, the joint stiffness is the sum of the individual stiffness of the agonist and antagonist muscles. Consequently, situations of both high net moment and low joint stiffness or low net moment and high joint stiffness may occur (DeLuca & Mambrito 1987). Antagonist coactivation of the hamstrings has been suggested to reduce the amount of anterior-posterior joint shear (Baratta et al. 1988). In strong support of this notion, the tensile ACL stress created by contraction of the quadriceps muscle at extended knee joint positions is reduced by simultaneous coactivation of the hamstring muscles (Draganich & Vahey 1990, Kaufman et al. 1991, More et al. 1993, Yasuda & Sasaki 1987). Furthermore, the presence of antagonist coactivation is effective in damping externally induced joint oscillation, even in situations where a myotatic reflex could create mechanical instability (Milner & Cloutier 1993). Evidence seems to suggest that the cerebellum plays an important role in switching from reciprocal activation to coactivation (see DeLuca & Mambrito (1987)). Moreover, in certain types of joint movement a common drive mechanism seems to exist by which the CNS may control the separate agonist–antagonist motoneuron pools as if they were one pool performing the same task. This common drive, or so-called proportional motoneuron activation, appears to be present during either of two states: when uncertainty exists in the required task or during anticipation of compensatory muscle forces (DeLuca & Mambrito 1987). It is not unlikely that the maximal dynamic knee extensions performed in the present study represented situations where compensatory hamstring muscle forces could be anticipated to regulate the contraction-induced ACL stresses described above. Hence, it seems reasonable that maximal isokinetic knee extension movements would involve patterns of proportional agonist–antagonist motoneuron activation.

EMG crosstalk

During active knee extension significant parts (15–25%) of the antagonist hamstring EMG signal may arise due to EMG spillover from the quadriceps muscle, i.e. as a the result of EMG crosstalk between

electrodes (Koh & Grabiner 1992, 1993). In the present study negligible amounts of EMG crosstalk were observed between antagonist Bfcl–agonist VL muscles and antagonist ST–agonist VM muscles, as indicated by peak R_{XY}^2 values of 0.060 (VL-Bfcl) and 0.039 (VM-ST). The R_{XY}^2 values (Winter et al. 1994) indicate that the amount of EMG crosstalk was on average 0.04–0.06, or 4–6%. Because of the marginal agonist–antagonist EMG cross talk it may be justifiable to conclude that the antagonist muscle moments were derived as a result of specific neural activation patterns and were not the result of EMG spillover from agonist muscles. The low level of EMG crosstalk is demonstrated in Fig. 2B where cross-correlation functions are shown for subjects with high antagonist hamstring coactivation (subject MM, full line) and low hamstring coactivation (subject TR, broken line), respectively. As can be seen, the R_{XY}^2 values were low (0.018 and 0.012), regardless of the amount of antagonist coactivation (Fig. 2B). Peak R_{XY} was 0.134 (subject MM) and 0.110 (subject TR). The low levels of agonist–antagonist EMG cross-talk observed in the present study conforms well to previous findings, also obtained during isokinetic knee extension and flexion (Bernardi et al. 1995).

Conclusions

In summary, considerable antagonist coactivation of the hamstring muscles was present during slow isokinetic knee extension involving maximal volitional quadriceps contraction. Towards complete knee extension (10–30°) this antagonistic moment corresponded to 30–74% of the net extension moment. The methodology presented may be a useful tool to examine the role of antagonist coactivation for dynamic knee joint stability. The present data suggest that during isokinetic knee extension a neural pathway exists which coactivates the flexors of the knee. This coactivation mechanism may be important for the stability of the knee joint as it potentially prevents anterior tibial displacement and assists in the mechanical and proprioceptive roles of the anterior cruciate ligament.

Key words: quadriceps; hamstrings; isokinetics; EMG; antagonist coactivation.

References

Aagaard P, Simonsen EB, Dyhre-Poulsen P. Antagonist muscle co-contraction during isokinetic knee extension. In: Blankevoort L, Kooloos J (eds.): Proc 2nd World Congr Biomech II, 1994: 295 (abstract).

Aagaard P, Simonsen EB, Dyhre-Poulsen P. A non-invasive method for measuring antagonist co-contraction force during isokinetic knee extension and flexion. Scand J Med Sci Sports 1994: 4: 286 (abstract).

Aagaard P, Simonsen EB, Trolle M, Bangsbo J, Klausen K. Isokinetic hamstring/quadriceps strength ratio: influence from joint angular velocity, gravity correction and contraction mode. Acta Physiol Scand 1995: 154: 421–7.

Aagaard P, Simonsen EB, Trolle M, Bangsbo J, Klausen K. Specificity of training velocity and training load on gains in isokinetic knee joint strength. Acta Physiol Scand 1996: 156: 123–9.

Aagaard P, Simonsen EB, Magnusson P, Larsson B, Dyhre-Poulsen P. A new concept for isokinetic Hamstring/Quadriceps strength ratio. Am J Sports Med 1998: 26: 231–7.

Amiridis IG, Martin A, Morlon B, et al. Co-activation and tension-regulating phenomena during isokinetic knee extension in sedentary and highly skilled humans. Eur J Appl Physiol 1996: 73: 149–56.

Baratta RV, Solomonow M, Zhou BH, Letson D, Chuinard R, D'Ambrosia R. Muscular coactivation. The role of the antagonist musculature in maintaining knee stability. Am J Sports Med 1988: 14: 83–7.

Basmajian JV, DeLuca CJ. Muscles alive: their functions revealed by electromyography. Baltimore: Williams & Wilkins, 1985.

Bernardi M, Solomonow M, Sanchez JH, Baratta RV, Nguyen G. Motor unit recruitment strategy of knee antagonist muscles in a stepwise, increasing isometric contraction. Eur J Appl Physiol 1995: 70: 493–501.

Beynnon B, Howe JG, Pope MH, Johnson RJ, Fleming BC. The measurement of anterior cruciate ligament strain in vivo. Int Orthop 1992: 16: 1–12.

Bobbert MF, Harlaar JQ. Evaluation of moment-angle curves in isokinetic knee extension. Med Sci Sports Exerc 1992: 25: 251–9.

DeLuca CJ, Mambrito B. Voluntary control of motor units in human antagonist muscles: coactivation and reciprocal activation. J Neurophysiol 1987: 58: 525–42.

Draganich LF, Vahey JW. An in vitro study of anterior cruciate ligament strain induced by quadriceps and hamstring forces. J Orthop Res 1990: 8: 57–63.

Dyhre-Poulsen P, Krogsgaard MR. Muscular reflexes elicited from the anterior cruciate ligament of the knee – a sensory function of the cruciate ligament. In revision, J Appl Physiol, 1999.

Farrell M, Richards JG. Analysis of the reliability and validity of the kinetic communicator exercise device. Med Sci Sports Exerc 1986: 18: 44–9.

Hagood S, Solomonow W, Baratta R, Zhou BH, D'Ambrosia R. The effect of joint velocity on the contribution of the antagonist musculature to knee stiffness and laxity. Am J Sports Med 1990: 18: 182–7.

Hirokawa S, Solomonow M, Lu Y, Lou Z, D'Ambrosia R. Anterior-posterior and rotational displacement of the tibia elicited by quadriceps contraction. Am J Sports Med 1992: 20: 299–306.

Kaufman K, Kai-nan A, Litchy WJ, Morrey BF, Chao YS. Dynamic joint forces during isokinetic exercise. Am J Sports Med 1991: 3: 305–16.

Kellis E, Baltzopoulos V. Agonist and antagonist EMG-angle relationships during isokinetic concentric and eccentric exercise. Isokin Exerc Sci 1996a: 6: 79–87.

Kellis E, Baltzopoulos V. The effects of normalization methods on antagonist activity during concentric and eccentric isokinetic knee extension and flexion. J Electromyogr Kinesiol 1996b: 6: 235–45.

Kellis E, Baltzopoulos V. The effect of antagonist moment on the resultant

joint moment during concentric and eccentric efforts of the knee extensors. Eur J Appl Physiol 1997: 76: 253–9.
Koh TJ, Grabiner MD. Cross talk in surface electromyograms of human hamstring muscles. J Orthop Res 1992: 10: 701–9.
Koh TJ, Grabiner MD. Evaluation of methods to minimize crosstalk in surface electromyography. J Biomech 1993: 26(suppl 1): 151–7.
Milner TE, Cloutier C. Compensation for mechanically unstable loading in voluntary wrist movement. Exp Brain Res 1993: 94: 522–32.
More RC, Karras BT, Neiman R, Fritschy D, Woo SL, Daniel DM. Hamstrings – an anterior cruciate ligament protagonist. Am J Sports Med 1993: 21: 231–7.
Nisell R, Ericson MO, Nemeth G. Tibiofemoral joint forces during isokinetic knee extension. Am J Sports Med 1989: 17: 49–54.
Osternig LR, Hamill J, Lander JE, Robertson R. Co-activation of sprinter and distance runner muscles in isokinetic exercise. Med Sci Sports Exerc 1986: 18: 431–5.
Renström P, Arms SW, Stanwyck TS, Johnson RJ, Pope MH. Strain within the anterior cruciate ligament during hamstring and quadriceps activity. Am J Sports Med 1986: 14: 83–7.
Smith AM. The coactivation of antagonist muscles. Can J Physiol Pharmacol 1981: 59, 733–47.
Snow CJ, Cooper J, Quanbury AO, Anderson JE. Antagonist cocontraction of knee flexors during constant velocity muscle shortening and lengthening. J Electromyogr Kinesiol 1993: 3: 78–86.
Solomonow M, Baratta RV, Zhou BH, et al. The synergistic action of the anterior cruciate ligaments and thigh muscles in maintaining joint stability. Am J Sports Med 1987: 15: 207–13.
Walla DJ, Albright JP, McAuley E, Martin RK, Eldridge V, El-Khoury G. Hamstring control and the unstable anterior cruciate ligament-deficient knee. Am J Sports Med 1985: 13: 34–9.
Winter DA, Fuglevand AJ, Archer SE. Crosstalk in surface electromyography: theoretical and practical estimates. J Electromyogr Kinesiol 1994: 4: 15–26.
Yasuda K, Sasaki S. Muscle exercise after anterior cruciate ligament reconstruction. Biomechanics of the simultaneous isometric contraction method of the quadriceps and the hamstrings. Clin Orthop 1987: 220: 266–74.

Appendix

The extension moment M_1 measured during concentric quadriceps action (quadriceps Q: agonist; hamstrings H: antagonist) is determined by the difference between the agonist extension moment M_{Qext} and antagonist flexion moment M_{Hflex} (K1 and K2 denoting EMG-to-force constants):

$$M_1 = K_1 \cdot EMG_{Q,agon} - K_2 \cdot EMG_{H,antag} \quad [1]$$

Correspondingly, the force-moment M_2 measured in knee extension movements involving eccentric hamstring action (H agonist, Q antagonist) is determined by

$$M_2 = K_2 \cdot EMG_{H,agon} - K_1 \cdot EMG_{Q,antag} \quad [2]$$

The pair of equations [1] and [2] can be solved for any knee angle Θ as it consists of two equations with two unknown variables: the EMG-to-force constants K_1 and K_2. Dividing equation [2] with $EMG_{H,agon}$ yields

$$M_2/EMG_{H,agon} = K_2 - K_1 \cdot EMG_{Q,antag}/EMGp_{H,agon} \quad [3]$$

Correspondingly, dividing equation [1] with $EMG_{H,antag}$ and rearranging gives

$$M_1/EMG_{H,antag} = -K_2 + K_1 \cdot EMG_{Q,agon}/EMG_{H,antag} \quad [4]$$

Subsequently, adding left and right sides of eqs. [3] and [4], respectively, and isolating K_1 results in the following solution for [1] and [2]:

$$K_1 = (A_1 + A_2)/(B_2 - B_1) \quad [5]$$

$$K_2 = A_1 + K_2 \cdot B_1 \quad [6]$$

Where
$A_1 = M_2/EMG_{H,agon}$ $B_1 = EMG_{Q,antag}/EMG_{H,agon}$
$A_2 = M_1/EMG_{H,antag}$ $B_2 = EMG_{Q,agon}/EMG_{H,antag}$
At any given knee angle (θ), calculating K_1 and K_2 according to [5] and [6] yields quadriceps and hamstring muscle moments M_{Qext} and M_{Hflex}:

$$M_{Qext}(\Theta) = K_1(\Theta) \cdot EMG_Q(\Theta)$$
$$M_{Hflex}(\Theta) = K_2(\Theta) \cdot EMG_H(\Theta)$$

The fact that K_1 and K_2 were determined at identical joint angular velocity, at specific contraction modes (K_1: concentric Q, K_2: eccentric H) and separately for every 0.05° knee joint angle between 10° and 90°, ensures that the specificity of 1) muscle length, 2) muscle contraction velocity, 3) internal muscle lever arm length and 4) contraction mode is inherent in the EMG-to-force relationships depicted. Prior to being fed to the set of equations the EMG signal of each muscle was normalized relative to its average EMG amplitude obtained during agonist contraction, thereby avoiding that the various EMG signals should contribute to eqs. [1] and [2] in proportion to their numeric EMG amplitude size (in mV).