Muscle coordination following rupture of the anterior cruciate ligament

Electromyographic studies of 14 patients

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In an electromyographic (EMG) study, the coordination in muscles acting on the knee joint was assessed in 14 patients with an arthroscopically verified complete rupture of the anterior cruciate ligament and in 16 controls. EMG and heel-contact signals were recorded while walking on a treadmill at walking gradients from 0 to 25 percent. There was an earlier onset of EMG bursts in the patients, especially in the lateral hamstrings and medial gastrocnemius; and the duration of EMG bursts also tended to be prolonged in the patients. Normalized root mean squares of amplitudes, which correlate with muscle tension, were higher in the gastrocnemius in the patients. EMG profiles, outlining the averaged muscle activity throughout the gait cycle, showed a displacement of peak activity in the hamstrings from the late swing phase into the stance phase, with increasing gradients in both the patients and the controls. Our study indicates that the gastrocnemius muscle contributes to functional stability in the anterior cruciate ligament deficient knee, and more attention should be paid to this muscle.

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In the treatment of rupture of the anterior cruciate ligamen, muscle training is essential (Giove et al. 1983, Vegso et al. 1985, Tegner et al. 1986). Contraction of the hamstring muscles exerts a posterior drawer force on the tibia and decreases the strain within the anterior cruciate ligament regardless of knee flexion angle (Renström et al. 1986, Yasuda and Sasaki 1987). Further, histologic studies have shown the presence of mechanoreceptors and free nerve endings in the anterior cruciate ligament (Schultz et al. 1984, Zimny et al. 1986, Schutte et al. 1987, Sjölander 1989). Finally, an electromyographic study has indicated a reflex arc between the thigh muscles and the anterior cruciate ligament or the knee joint capsule (Solomonov et al. 1987). Recently, we have demonstrated an earlier onset of hamstring activity in the late swing phase during walking in anterior cruciate ligament deficient subjects compared with normal controls (Kaalund et al. 1990). Knowledge of the compensatory mechanisms appears essential for planning rehabilitation: How is optimal functional stability obtained when various degrees of static instability is present?

Our purpose in this study was to obtain a detailed knowledge of coordination and activity of the muscles acting on the anterior cruciate ligament deficient knee.

Patients and methods

Eleven men and 3 women, mean age 30 (21–48) years, with an arthroscopically verified complete rupture of the anterior cruciate ligament were investigated 46 (10–102) months after their injury. The mean Lysholm score was 74 (55–95; Tegner and Lysholm 1985). The mean anterior laxity was 7.2 (3–14) mm in injured and 4.5 (1–8) mm in nonaffected knees ($P < 0.02$, Stryker's knee laxity tester; Boniface et al. 1986). By clinical examination, a slight valgus instability in semiflexion was found in 3 patients; 2 of these also had mild varus instability. Sixteen age- and sex-matched subjects with no history of knee trauma and no knee complaints were selected as controls. Testing was performed on a treadmill (Power Jog, Sport Engineering Ltd., UK) at 5 km/h and with six different gradients: 0, 5, 10, 15, 20, and 25 percent (0°–11.25°).

For EMG recording, bipolar surface electrodes (silver/silver chloride, Medicotest a/s, DK) with 2 cm of center-to-center spacing and orientation along the muscles were used. Signals were amplified (DISA 05A02) and filtered by a first-order bandpass filter (20–500 Hz). EMGs were collected from five muscles: vastus lateralis, vastus medialis,

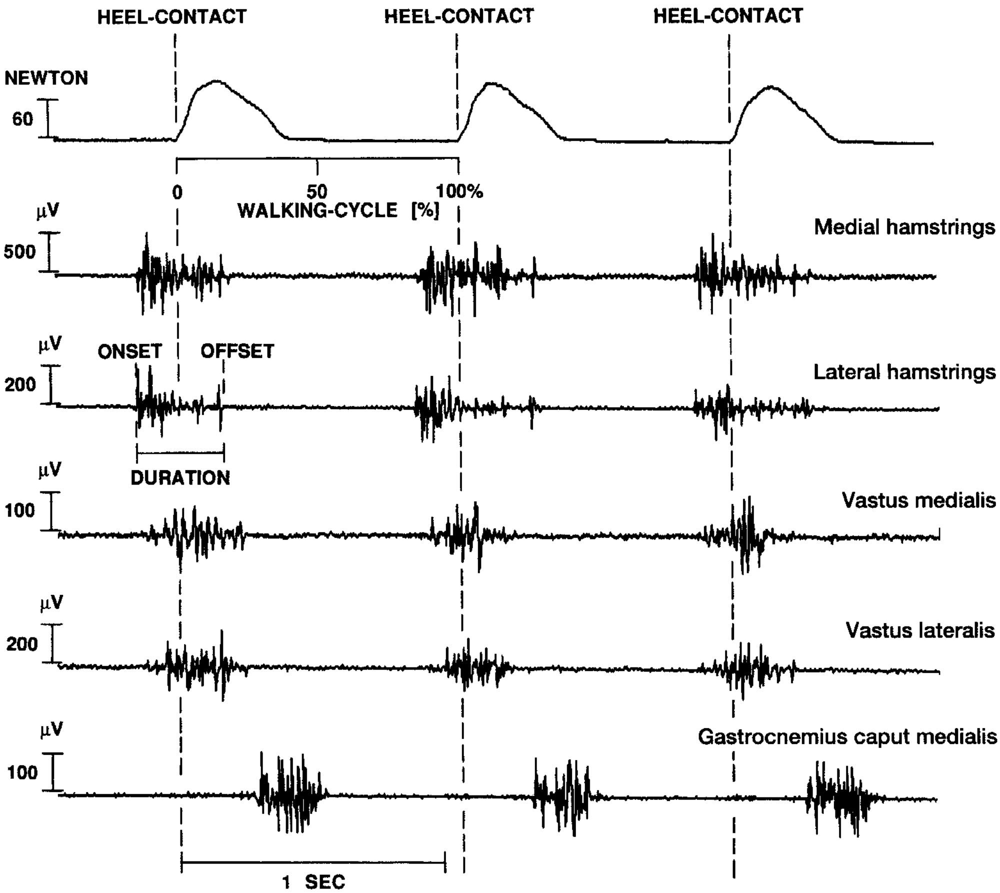

Figure 1. Heel-contact and EMG recording. Raw signals from 1 person showing normalization of the walking cycle and onset, offset, and duration of individual EMG bursts.

medial hamstrings, lateral hamstrings, and gastrocnemius caput medialis.

To determine the stride characteristics, a pressure sensible transducer (operating voltage: 0–5 V, sensitivity 17 mV/newton, assessed under conditions using solid materials) was placed inside the shoe under the heel of the subjects. EMG and heel-contact signals were recorded on a personal computer with a sampling rate of 512 Hz. Signals were displayed to assure proper amplification and to avoid artefacts.

Recording was done in one sequence, 15 sec per gradient and about 3 min totally, including time for elevation of the platform. Recording was preceded by a period of habituation.

Analysis of data

Onset of the heel-contact signal was determined by a successive integration method (Jansen 1988). A detection window of five samples, approximately 10 msec and a threshold of 70 mV, was used. Onset time (T_{on}) and offset time (T_{off}) of EMG bursts were determined 1) by calculating the variance (V_n) of the EMG signal during a period with no muscle activity and 2) by calculating the variance (V_b) of the EMG bursts. Thresholds for detection of T_{on} and T_{off} were given by $V_b > C_{on} \times V_n$ and $V_b < C_{off} \times V_n$, respectively. C_{on} and C_{off} were initialized to 3.0, and were adjusted automatically by a simple algorithm.

The onset, offset, and duration times were normalized to percentages—100 percent representing

Table 1. Mean onset time (*SE*) and duration(*SE*) of EMG bursts given as a percentage of gait-cycle duration. Negative values of onset time means onset before heel contact (0 percent)

	Onset			Duration		
Elevation (%)	ACL	Control	*P*-value	ACL	Control	*P*-value
Medial hamstring						
0	–20 *2*	–17 *2*	< 0.01	35 *7*	33 *8*	NS
5	–19 *2*	–18 *2*	< 0.05	40 *6*	39 *6*	NS
10	–20 *2*	–18 *2*	NS	45 *8*	43 *8*	NS
15	–20 *2*	–18 *2*	NS	46 *9*	45 *8*	NS
20	–20 *2*	–18 *2*	NS	47 *10*	44 *8*	NS
25	–19 *2*	–19 *2*	NS	46 *8*	46 *8*	NS
Lateral hamstrings						
0	–20 *2*	–18 *3*	< 0.05	34 *10*	31 *7*	NS
5	–19 *3*	–16 *2*	< 0.05	44 *8*	40 *8*	NS
10	–18 *3*	–15 *3*	< 0.05	49 *7*	46 *6*	NS
15	–18 *3*	–15 *3*	< 0.05	53 *8*	50 *7*	NS
20	–17 *3*	–15 *2*	< 0.05	53 *6*	53 *6*	NS
25	–17 *2*	–16 *2*	NS	53 *7*	55 *7*	NS
Vastus medialis						
0	–12 *3*	–10 *1*	NS	29 *9*	27 *4*	NS
5	–12 *3*	–10 *2*	NS	31 *7*	29 *5*	NS
10	–11 *3*	–9 *2*	NS	36 *7*	33 *7*	NS
15	–9 *2*	– 8 *2*	NS	45 *6*	37 *7*	< 0.05
20	–10 *2*	–8 *2*	< 0.01	49 *6*	44 *7*	NS
25	–10 *1*	–8 *2*	< 0.05	52 *6*	47 *7*	NS
Vastus lateralis						
0	–12 *4*	–10 *2*	NS	29 *9*	27 *4*	NS
5	–10 *3*	–9 *2*	NS	29 *8*	29 *7*	NS
10	– 9 *3*	–8 *2*	NS	34 *7*	32 *9*	NS
15	– 9 *2*	–8 *2*	NS	42 *6*	39 *10*	NS
20	–10 *2*	–8 *2*	< 0.01	48 *6*	44 *8*	NS
25	–10 *2*	–8 *1*	< 0.05	50 *5*	49 *6*	NS
Gastrocnemius medialis						
0	9 *8*	15 *4*	< 0.05	38 *7*	35 *6*	NS
5	12 *5*	18 *4*	< 0.05	37 *6*	32 *4*	< 0.05
10	12 *5*	18 *4*	< 0.05	38 *5*	33 *3*	< 0.05
15	12 *5*	16 *4*	< 0.05	39 *6*	36 *4*	NS
20	9 *5*	12 *4*	NS	42 *6*	40 *4*	NS
25	8 *4*	10 *4*	NS	44 *6*	42 *4*	NS

ACL Anterior cruciate ligament deficient cases.

one gait cycle (Arsenault et al. 1986, Winter and Yack 1987, Shiavi et al. 1988, Figure 1). The root mean square (RMS) amplitude of the individual bursts, which represents the EMG activity and correlates with the muscular force under isometric conditions, was calculated (Milner-Brown and Stein 1975). Normalization was achieved by setting the mean RMS amplitude for each person and each muscle at 100 percent at a 0 percent gradient.

EMG profiles were calculated for each muscle and for each of the six gradients on the basis of 10 to 15 gait cycles per gradient using the rectified EMG signals divided into 25 intervals, with each interval covering 4 percent of the gait cycle (Arsenault et al. 1986, Winter and Yack 1987). Normalization was done by setting the mean value of the EMG amplitude at 100 percent (Winter and Yack 1987).

The Student's *t*-test and linear regression analysis were used.

Results

Onset time of EMG bursts

In all five muscles, the EMG activity started earlier in ACL patients versus controls at all the gradients (Table 1), although significant differences were not seen at all gradients. In the lateral hamstrings and

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Table 2. Root mean square (RMS) amplitudes of EMG bursts in the *gastrocnemius caput medialis* at increasing walking gradients. Values (*SE*) are normalized to 100 percent at a 0 percent gradient

Elevation (%)	ACL		Control	
0	100		100	
5	133	*21*	128	*10*
10	154	*34*	145	*15*
15	187	*61*	158	*15*
20	205	*74*	166	*16*
25	222	*92*	177	*27*

ACL Anterior cruciate ligament deficient cases.
ACL vs. Control: $P < 0.01$ (regression analysis).

the gastrocnemius caput medialis, the most marked differences were observed. In the gastrocnemius, the onset time was decreased up to 6 percent.

Duration of EMG bursts

In 28 of 30 comparisons, the mean duration of EMG bursts was numerically longer among the patients than among the controls (Table 1). However, statistically significant differences were observed in only the vastus medialis and the gastrocnemius, the EMG bursts being prolonged by 8 percent and 5 percent of the gait cycle, respectively.

RMS amplitudes of the EMG bursts

With increasing gradient, the normalized RMS of the EMG amplitudes increased in all the muscles in both the patients and the controls. In the gastrocnemius the RMS amplitudes in the patients significantly exceeded those of the controls at an increasing gradient (Table 2). The difference at a 25 percent gradient was 45 percent of the normalized initial value. The increase of RMS amplitudes in the hamstrings and the vastus muscles did not differ between patients and controls.

EMG profiles

Intersubject profiles were calculated for the patients and the controls at each gradient (Figure 2). With increasing gradient, the mean peak activity in the individual profiles in the lateral hamstrings of the patients moved from –6.9 ± 5.3 percent (± 1SD) at a 0 percent gradient to 10 ± 8.4 percent ($P < 0.001$) at a 25 percent gradient. This did not differ from the controls, in which peak activity moved from –6.8 ± 3.0 percent to 6.0 ± 11 percent ($P < 0.001$). The same phenomenon was observed in the medial hamstrings, peak activity moving from –8.8 ± 3.1 percent to 1.4 ± 6.2 percent ($P < 0.001$) in the patients and from –7.9 ± 3.4 percent to –0.6 ± 3.1 percent ($P < 0.001$) in the controls, with no significant difference between the groups. No consistent displacement of mean peak activity with increased gradient was seen in the other muscles.

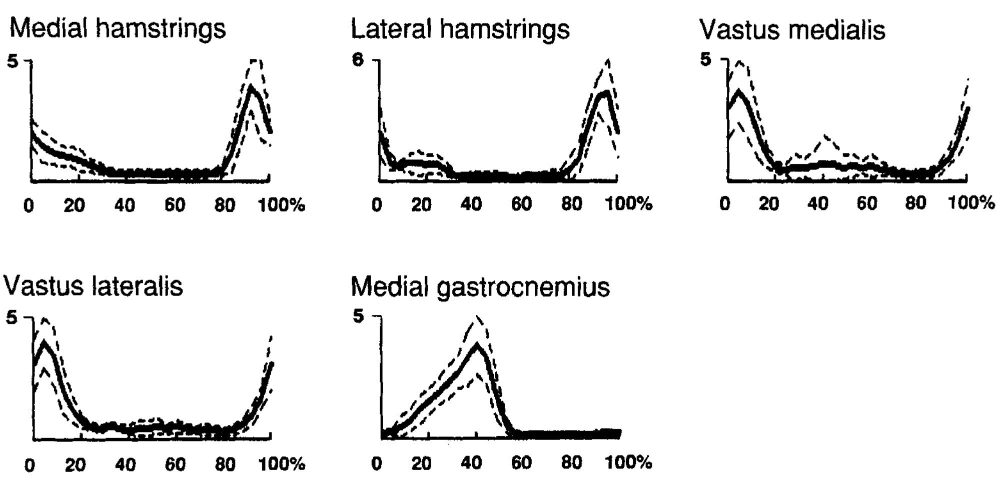

Figure 2. EMG profiles for control subjects at a 0 percent walking gradient. The walking cycle is normalized to 100 percent. Heel contact is at 0 percent. ± SE is indicated. Values on the ordinate are given in respect to mean amplitude = 1.

Discussion

In 1968, Carlsöö and Nordstrand compared EMG signals in anterior cruciate ligament deficient patients with normal controls during level walking. They found no consistent differences in muscle coordination between the two groups. In a dynamic on/off EMG study, Tibone et al. (1986) found only minor differences between anterior cruciate ligament deficient and uninjured knees during level walking. During running, the vastus medialis had an earlier onset in the involved limb, and the activity of the medial hamstrings was prolonged as compared with the lateral hamstrings. During stair climbing, activity was present more often in the lateral hamstrings in the late swing phase in the involved limb compared with the uninvolved limb.

Branch et al. (1989) studied the effect of knee braces during side-step cutting maneuvers. They assessed the activity of the muscles from the rectified EMG signals. Without braces, the lateral hamstrings were more active in the precut swing phase in anterior cruciate ligament deficient subjects when compared with normal controls. During the stance phase, activity was increased in the medial hamstrings, while the quadriceps and the medial gastrocnemius activity were decreased.

Thus, the previous, different EMG studies have contributed to a beginning of the understanding of how functional stability is obtained in anterior cruciate ligament deficiency.

As regards the thigh muscles, our results demonstrate an earlier activation and perhaps a general tendency to prolonged activity in all the muscles. These phenomena seem compensatory for the unstable knee. The earlier onset of the hamstrings, especially the lateral hamstrings, is considered to stabilize the knee in preparation for foot contact with the ground, decreasing the risk of subluxation. This is consistent with the results of Tibone et al. (1986) and Branch et al. (1989). Thus, in the search for optimal rehabilitation programs, efforts should be made to imitate and supplement such compensatory mechanisms.

Recent EMG studies have extended the knowledge of quadriceps and hamstring interaction by the demonstration of coactivation between the two muscle groups in healthy men during flexion and extension exercises when lying on the side (Baratta et al. 1988) and during extension exercises in a seated position (Draganich et al. 1989).

Although thigh muscle activity appears most important for the functional stability following anterior cruciate ligament rupture, strengthening exercises alone probably are not sufficient. Pope et al. (1979) showed that voluntary reaction time for muscular contraction is often too slow to protect the knee in typical sports situations. Further, Walla et al. (1985) found that the Cybex strength and power estimates did not correlate with the functional status. However, the presence of hamstring control on a "reflex level"—i.e., activity present, instinctively, despite distraction and reducing a pivot shift during physical examination—was most closely associated with high functional rating scores. The fact that the differences we observed in the thigh muscles were alterations in timing, and no increase in the RMS amplitude of the EMG, is consistent with these hypotheses, and may express that compensation by coordination is most important.

Thus, rehabilitation programs should include elements of muscle-coordination training.

As regards the gastrocnemius, our study seems to be the first one to demonstrate that this muscle probably plays an important role for the anterior cruciate ligament deficient knee; substantial alterations in timing, as well as activity, were observed. The gastrocnemius acts at two joints. At the knee joint, it is primarily a flexor muscle; but, as regards its anatomy, it may also be an important muscle contributing to reduction of knee joint laxity. Markolf et al. (1978) showed that a simultaneous contraction of the knee muscles increased the stiffness of the knee joint on an average of two to four times; anterior-posterior laxity was reduced to 25 to 50 percent of the normal value. We consider the altered gastrocnemius function, together with alterations in thigh muscles, to be an attempt to stabilize the knee joint, which otherwise is susceptible to subluxation.

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