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Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex

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Summary. The onset latency and discharge amplitude of preprogrammed postural responses were evaluated in order to determine if the structure of synergistic activation could be altered by ligamentous laxity at the knee joint. Twelve subjects with unilateral and one subject with bilateral anterior cruciate ligament (ACL) insufficiency were tested while standing on a moveable platform. External balance perturbations (6 cm anterior or posterior horizontal displacements of the platform) were presented at velocities ranging from 15 to 35 cm/s. Perturbations were presented under the following experimental conditions: unilateral and bilateral stance, knees fully straight or flexed, and with ankle motion restricted or free. These stance, knee position, and ankle motion conditions were introduced to alter the stress transmitted to the knee joint during movement of the support surface. The automatic postural response was recorded from the tibialis anterior (T), quadriceps (Q), and medial hamstrings muscles (H) bilaterally. The normal response to an externally induced backward sway involved the automatic activation of T and Q at latencies of 80 ms and 90 ms respectively. Activation of the hamstrings in the non-injured extremity was not coupled with the postural response. Hamstrings are not typically involved in the correction posterior sway because H activation would tend to pull the center of mass further backwards. However, when the response in the ACL-deficient extremity was compared to the non-injured limb: (1) the automatic postural response in the ACL-deficient extremity was restructured to include hamstrings activation (100 ms latency), (2) H activation time was faster and less variable in the ACL-deficient limb, and (3) the ratio of H/Q discharge amplitude integrated over 100 ms and 200 ms from the onset of EMG activation showed a dominance of hamstring activity during unilateral stance on the lax limb. In addition, H/Q ratios integrated over 200 ms showed dominant hamstring activity in the ACL-deficient limb during bilateral stance. (4) Cross-limb comparisons showed greater normalized IEMG amplitudes for T, H, and Q during unilateral stance on the lax limb. These results suggest that a capsular-hamstring reflex is integrated into the existing structure of a preprogrammed postural synergy in order to compensate for ligamentous laxity. Furthermore, the generalized increase of response gain observed during perturbations of unilateral stance on the lax limb indicates that joint afference can modulate central programming to control localized joint hypermobility. A concept of postural control is discussed with respect to the capsular reflex, joint loading and displacement of the center of gravity.

Key words: Balance – Joint laxity – Ligament – Motor programming – EMG

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Introduction

The control of body sway in humans requires a complex interaction of afferent information from visual, vestibular and proprioceptive systems (Di Fabio et al. 1990; Dietz et al. 1988; Diener et al. 1988; Keshner et al. 1987; Allum et al. 1988; Diener et al. 1984a; Nashner et al. 1982). Experimental paradigms using a moveable standing platform to induce body sway have provided a method to quantify temporal and spatial characteristics of automatic neuromuscular responses which occur prior to a voluntary reaction and which are characterized by groups of muscles acting in synergistic patterns to stabilize upright stance (Woollacott et al. 1988; Nashner 1977). When a platform surface is translated in the anterior direction for example, backward sway is induced in the posterior direction. In this case, tibialis anterior, quadriceps, and abdominal muscles act as synergists to prevent a backward fall (Nashner 1977).

The activation of these synergistic patterns cannot be explained on the basis of a simple stimulus-response relationship because the same size and direction of postural perturbation can produce different muscle patterns when stance or expectancy conditions are altered (Dietz

et al. 1989a; Diener et al. 1983; Horak and Nashner 1986; Marsden et al. 1981). The mechanism which creates an altered neuromuscular response to the same input stimulus is currently a matter of speculation. However, the type and degree of load-bearing in the lower extremities may have an important influence on the characteristics of the postural response. Horstmann and Dietz (1990) utilized a water-immersion procedure (simulated micro-gravity) and applied different loads to the body axis through a weighted vest worn by the subjects. These authors demonstrated an approximate linear relationship between the contact force and the electromyograph (EMG) amplitudes of postural muscles and it was concluded that a gain control mechanism for postural reflexes was dependent on body weight. Dietz et al. (1989a) maintained that a force-sensitive receptor system was needed to monitor the position of the center of gravity with respect to the feet so that the output parameters of a triggered postural response could be modified in a graded functional way.

In addition to the gain of the automatic postural response, the structure of a centrally preprogrammed synergistic action appears to be modified by peripheral inputs. Layne and Spooner (1990) found that anticipatory postural activity in the biceps femoris was eliminated in 75% of the trials during arm flexion tasks performed in a micro-gravity environment induced by parabolic flight. The elimination of lower extremity postural activity when gravitational influences were reduced supports the view that lower extremity load-bearing provides afferent inputs that are necessary to recruit postural muscles. It is also known that applying a stretch to postural muscles while subjects are sitting (Gottlieb and Agarwal 1979) produces a recruitment pattern which differs from conditions where subjects are standing during the stretch stimulus (Di Fabio et al. 1986; Nashner 1977). Body sway and EMG response magnitude during upright stance appear to be scaled by peripheral sensory information that codes the velocity and amplitude of support surface displacements (Di Fabio et al. 1990; Diener et al. 1988). Therefore, it is possible that the mechanism controlling the gain and structure of the automatic neuromuscular response may be peripherally driven and contribute to the adaptation of centrally triggered responses through prior sensorimotor experience (Horak et al. 1989).

The sensory afference produced by distinct loads on ligaments and on the joint capsules of load-bearing joints has not received adequate attention with respect to influencing the temporal and spatial aspects of postural muscle synergies in humans. Wyke (1967) reported that all mammalian synovial joints have encapsulated corpuscles that serve as mechanoreceptors and that these end-organs may exert facilitatory and/or inhibitory influences on muscle tone through the fusimotor-muscle spindle loop (Freeman and Wyke 1967; Stener and Petersen 1962). A more recent discussion indicates that several alternative pathways may exist for joint afference as well as other sources of peripheral sensory input to facilitate or inhibit motor activity (Dietz et al. 1988; Lundberg et al. 1987).

It is thought that loss of normal afference from vertebral mechanoreceptors may contribute to balance deficits (Tinetti et al. 1986; Wyke 1979). However, the relative importance of inputs from knee joint mechanoreceptors in creating a functional balance response in humans is unknown. The knee joint is the focus of our interest because the torques about the knee during balance reactions are stabilized by sequential pre-programmed contractions of the ankle, thigh and lower trunk muscles (Nashner and McCollum 1985). The use of postural "strategies" involving horizonal shear floor reaction forces (Horak et al. 1990) will also create load-bearing on both active and passive (non-contractile) restraints about the knee joint. Pope et al. (1979) suggested that the onset of ligament-muscle reflexes in the knee was not fast enough or sustained long enough to prevent ligamentous injury. When the knee ligaments become deficient, it is not known how central and peripheral inputs are altered to compensate for the hypermobility at the tibial-femoral junction.

Early animal experiments using the cat have shown that surgical resection of the sensory articular nerves to the knee leads to deficits in posture, gait, positive supporting and placing reactions (Freeman and Wyke 1966). Reflex muscle activity observed during passive knee motion following resection of the knee ligaments in the cat provided evidence that receptors in the joint capsule may, in part, be responsible for triggering muscle action (Freeman and Wyke 1967; Stener 1959).

Recent studies on humans have reported the existence of Ruffini and Golgi receptors in the anterior cruciate ligament (ACL) and these observations provide a basis for suggesting that reflex muscle inhibition can be mediated by non-contractile joint structures (Schultz et al. 1984; Zimney et al. 1986). Receptors in the joint capsule have been implicated as end-organs in a "capsular-hamstring" reflex vis-a-vis a report that hamstring EMG onsets during treadmill ambulation under load (incline) were significantly early with respect to heel-strike in patients with ACL rupture (Kalund et al. 1990). This early hamstring activation theoretically provides a compensatory mechanism to control for impending displacement of the tibia learned through repeated stimulation of a joint capsular-hamstring reflex. Furthermore, Solomonow et al. (1987) suggested that the hamstring muscles were activated in a "reflex arc" involving knee joint mechanoreceptors because subluxation of the ACL-deficient knee during isokinetic extension (while sitting) resulted in a distinct burst of hamstring activity which appeared to inhibit quadriceps discharge amplitude.

Previous work focusing on modifications of long-loop postural synergies in humans with respect to knee joint stress was not found. Although evidence for a capsular-hamstring reflex has been reported (Kalund et al. 1990; Solomonow et al. 1987), the characteristics of this response have not been defined in terms of onset latency, antagonist interaction (co-contraction, reciprocal inhibition), or functional significance with respect to balance behavior. The purpose of this study was to determine the temporal and spatial parameters, and synergistic

Table 1. Patient sample characteristics and arthroscope summary

Subject	Age (years)	Duration of injury		KT 1000 Results Displacement[a] (mm)		Arthroscope or MRI summary
		R Leg	L Leg	R Leg	L Leg	
S_1	32	–	3 m	5.0	18.5	AP instability of *L* knee and posterior medial meniscus tear
S_2	35	19 m	–	19.5	6.0	Deficiency of *R* Anterior cruciate
S_3	30	–	2 y	–	–	*L* anterior cruciate ligament tear, no associated meniscal pathology
S_4	31	–	18 y	4.3	13.5	*L* ACL deficient knee with lateral meniscal tear
S_5	36	2 y		17.7	6.8	Isolated ACL instability bilaterally with right greater than left
S_6	26		2 y	–	–	*L* anterior cruciate ligament tear with medial meniscal tear
S_7	28	–	4 y	4.8	15.6	*L* ACL deficient knee with possible medial meniscal tear
S_8	27	2 m	–	12.7	8.5	*R* ACL tear with medial meniscal tear
S_9	16	1 y	–	14.3	7.7	*R* ACL deficient knee with medical meniscal tear
S_{10}	31	6 y	–	13.2	7.8	*R* anterior cruciate ligament tear, S/P subtotal medial and partial lateral menisectomies
S_{11}	38	9 y	8 y	14.5	10.5	*R* ACL deficient knee with degerative joint disease in left knee, S/P left ACL reconstruction
S_{12}	19	–	6 w	6.3	12.8	*L* anterior cruciate ligament tear
S_{13}	22	8 y	4 y	13.0	13.0	Bilateral ACL deficient knee with *L* medial meniscal tear

w = weeks; m = months; y = years

[a] Knee displacement measured during a 134 newton upward pull

structure of a postural response in humans with unilateral ACL-insufficiency.

Methods

Subjects and procedure

Twelve patients with unilateral anterior cruciate ligament laxity (mean age 29 years, range 16–38 years) volunteered to participate in this study (Table 1). All subjects were tested prior to receiving surgical treatment for correcting joint instability. Informed consent was obtained from each subject before beginning data collection. An unusual subject with a bilateral ACL deficiency (S_{13}) was included for descriptive comparisons.

Subjects participating in this study had no known neurological deficits. Aside from their knee injuries, these subjects were healthy, active adults. Ligament insufficiency was measured clinically using a MEDmetric Arthrometer (model KT-1000, MEDmetric Corporation, San Diego, Calif., USA) and the loss of ligament integrity was verified by arthroscopic examination or through magnetic resonance imaging (MRI). The arthrometer measured displacement of the tibia on the femur (in mm) during a manual pull on the tibia of 134 N (Fig. 1). In order to insure reliability, one author (KJ) with 3 years of experience using this apparatus, obtained all measures of knee joint laxity[1]. A summary of these measures is included in Table 1.

Subjects stood independently on a force platform with each foot placed in a standardized position. The apparatus has been fully described elsewhere (Di Fabio 1987; Di Fabio et al. 1986). Anterior linear displacements of the platform were presented with the amplitude of displacement fixed at 6 cm. The velocity of platform displacements ranged from 15 cm/s to 35 cm/s and 4 perturbation speeds were randomly presented. The duration of perturbations ranged from 170 ms (at 35 cm/s) to 400 ms (at 15 cm/s).

The effects of anticipation were minimized by randomly reversing the direction of platform displacement for selected trials and by using a variable fore-period ranging from 15 to 45 s prior to initiating a trial. Perturbation direction was reversed in 12 trials (20% of the total). These reverse perturbations were 6 cm posterior linear platform displacements delivered at a velocity of 23 cm/s (duration of perturbation = 285 ms).

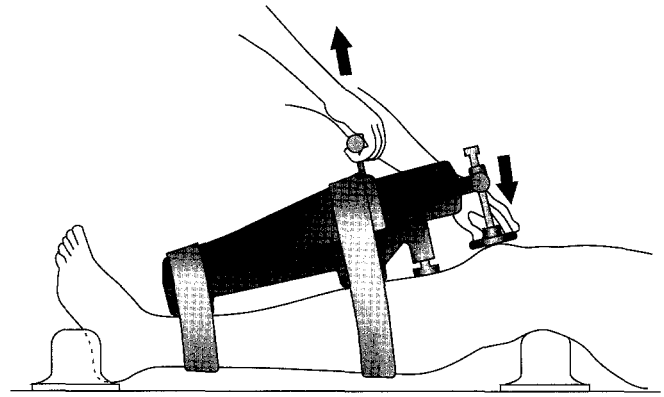

Fig. 1. Device used to quantify knee joint laxity. Subjects were placed in a standardized position and the amount of tibial displacement was measured during an upward pull of 30 lbs in the direction of the black arrow (modified from Daniel et al. (1985) Amer J Sports Medicine 13:402)

The testing sequence for each subject included bilateral stance and single leg stance (Table 2). The knees were fully extended in a normal stance position and trials were repeated with knees slightly bent (approximately 20° of flexion) to place the knee in a position allowing greater A/P translation of the tibia on the femur. In order to provide a maximal stress to the knee joint capsule, the subjects were fitted with a boot during selected trials to immobilize the ankle joint (Orthotech walking boot, Orthotechnology, 2453 Verna Ct., San Leandro, Calif., USA).

Data collection

All data collection was completed in a fully shielded room (USC-26 RFI/EMI Shielding System, Modular Design by Universal Shield-

[1] Two subjects listed in Table 1 (S_3 and S_6) were not available for arthrometer testing prior to surgical intervention. For these two patients, the arthroscopic and MRI summaries provided the basis for evaluating knee joint laxity

Table 2. Summary of the perturbation stimulus sequence presented to each subject

Number of trials[a]	Trial description
5	Bilateral stance/Knee straight/No boot
5	Bilateral stance/Knee bent/No boot
10	Unilateral stance/Knee straight/No boot
10	Unilateral stance/Knee bent/No boot
5	Bilateral stance/Knee straight/Boot
5	Bilateral stance/Knee bent/Boot
10	Unilateral stance/Knee straight/Boot
10	Unilateral stance/Knee bent/Boot
60	Total number of trials/Subject

[a] Perturbation velocity was varied randomly within each block of 5 trials. In addition, a reversal of perturbation direction occurred once in each 5-trial block and was nested randomly in the trial sequence. Unilateral stance included trials for each leg

ing Corp., Old Bethpage, New York, USA). Surface electromyographs were recorded from the tibialis anterior (T), quadriceps (Q), and the medial hamstring muscles (H) bilaterally. The EMG signals were preamplified (common mode rejection of 87 dB at 60 Hz), full wave rectified, band pass limited from 40 Hz–4 kHz, and filtered with a time constant of 5.5 ms. Muscle discharge activity was synchronized to the onset of platform displacement and was digitized at 500 Hz for a 2-s interval. Electrode preparation and placement were standardized for each muscle. The EMG electrode assemblies (Therapeutics Unlimited, Iowa City, Iowa, USA) contained the circuitry for preamplification with a gain of 35 and each assembly held two silver-silver chloride electrodes (8 mm diam.) spaced at a standard distance of 20 mm between the centers. The on-site preamplification of muscle activity eliminated the capacitive effects caused by the leads from standard surface electrodes.

In order to verify initial loading conditions during bilateral stance, data from three force channels (fore/aft, vertical right, and vertical left sway) were filtered with a corner frequency of 22 Hz at 3 dB and were digitized online for two seconds following platform displacement. Subsequent analysis revealed no significant difference between weight-bearing on the ACL-deficient leg (51%, s.d. 7% of b.w.) compared to the normal lower extremity (49%, s.d. 9% of total b.w.) at the beginning of each trial.

Statistical design and data analysis

Data from each trial were analyzed interactively to determine the latency of muscle discharge. Three dependent variables- (1) EMG onset latency, (2) the *ratio* of hamstrings/quadriceps integrated EMG amplitudes (IEMG) over 100 ms and 200 ms from burst onset, and (3) T, Q, H muscle discharge amplitudes (100 and 200 ms IEMG from burst onset) were analyzed with an analysis of variance (ANOVA) to determine if statistically significant differences were present. Each dependent variable was analyzed using a [2 × 2 × 2] multifactorial design with the following factors: stance (unilateral or bilateral), knee position (straight or flexed), and ankle stability (boot or no-boot). The data were "collapsed" across perturbation velocities to provide an averaged block of four directionally similar trials for each condition (Table 2). In order to make comparisons across subjects and between deficient and normal limbs, the amplitude of muscle discharge was normalized to the subject's maximum amplitude obtained during the entire test for a given muscle. Also, in order to eliminate the possibility of a pain-mediated response, we verified that the subjects did not feel pain or spasm in the knee during the course of data collection.

Results

Postural muscle latencies: normal vs ACL-deficient lower extremity

When all conditions (knee position, ankle stability, and perturbation velocity) were combined, the onset latencies of T and Q were on the order of 80 ms and 90 ms (respectively) whereas the onset latency of the H muscles averaged 100 ms (Fig. 2; left). The variability of the H muscle onset latencies in the *ACL deficient limb* were reduced by 30 and 34% for bilateral and unilateral stance respectively when compared to the H latency in the normal lower extremity (Fig. 2; right). This decrease in variation suggested that a reflex response involving the hamstring muscles was organized selectively in the ACL deficient extremity. Figure 3 illustrates the integrated EMG patterns (IEMG) of postural muscles during induced backward body sway and for the control trials

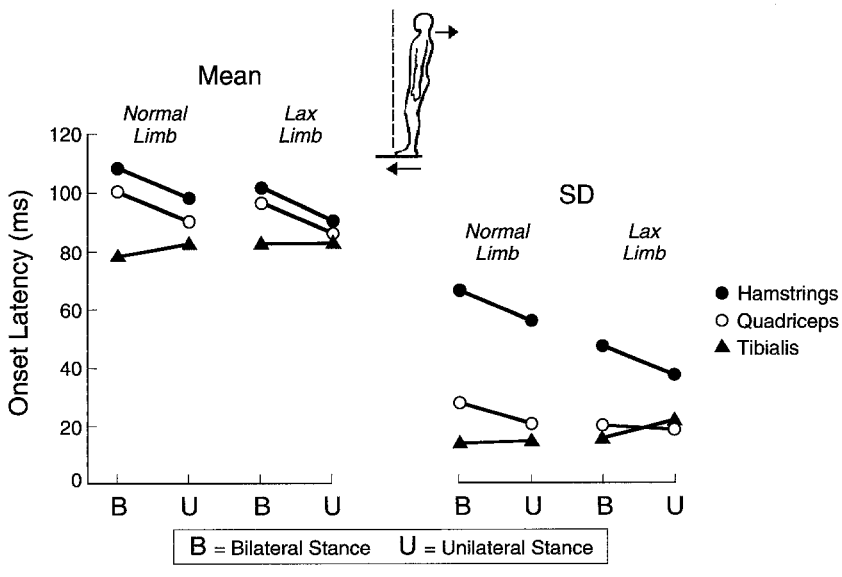

Fig. 2. Mean onset latencies and standard deviations (SD) for medial hamstrings, quadriceps, and tibialis anterior muscles in non-injured (normal) and ACL-deficient (lax) limbs during perturbations of bilateral and unilateral stance. Values were averaged across perturbation velocity, knee position, and ankle stability conditions (see 'Methods')

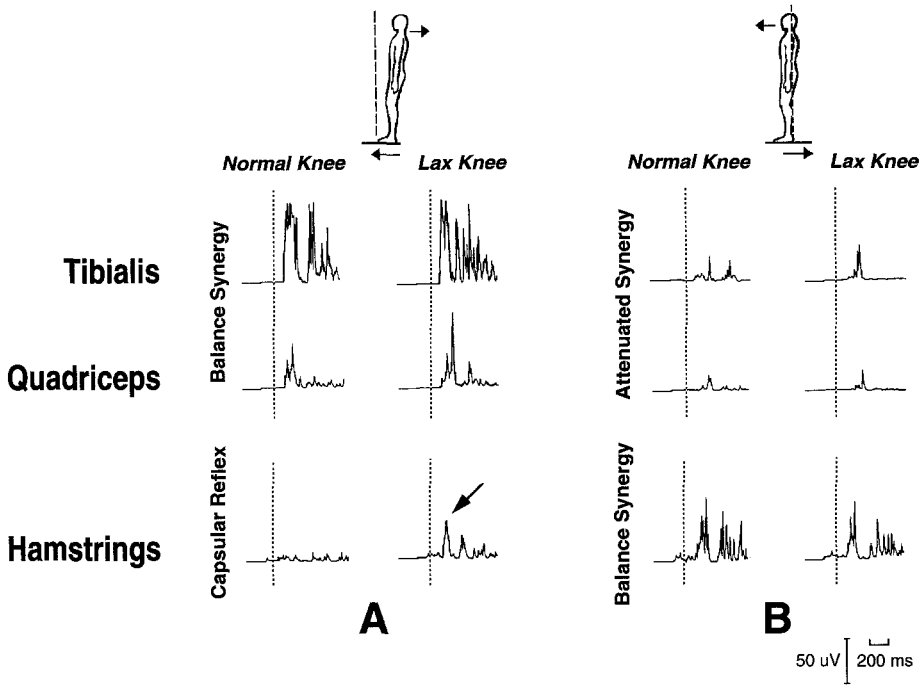

Fig. 3A, B. Unilateral capsular reflex. Each trace is an ensemble average of 4 trials from one subject who was representative of the sample. The vertical lines indicate the onset of platform displacement. **A** Externally induced backward sway during bilateral stance (knees straight and ankles free to move) produced a recruitment of tibialis anterior and quadriceps muscles bilaterally to correct body alignment. Medial hamstrings were selectively recruited in the lax limb (as indicated by the *arrow*). **B** During perturbations inducing a forward body sway under the same experimental conditions outlined for (A), tibialis and quadriceps muscle discharge amplitude was attenuated and hamstrings muscles were recruited bilaterally in a balance synergy to correct body alignment

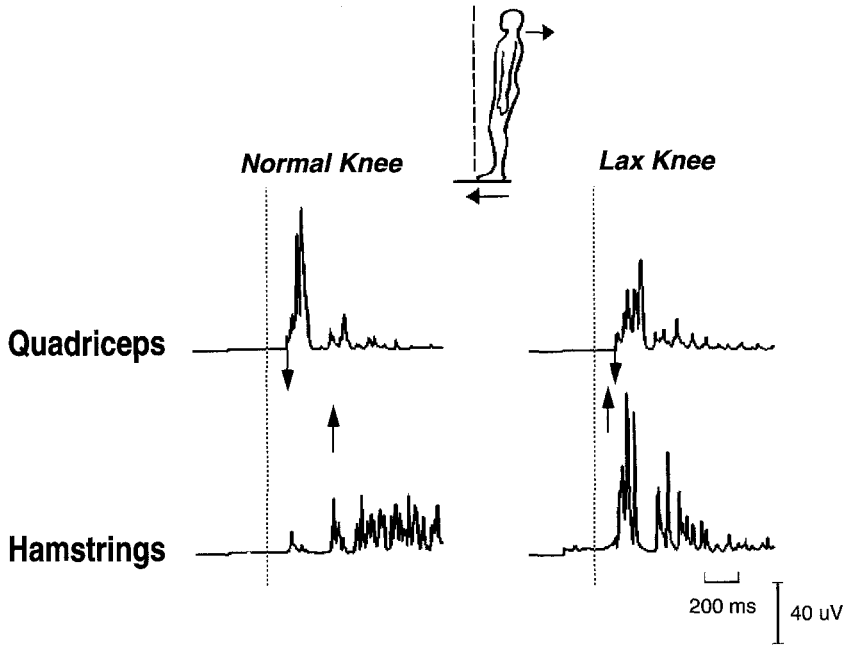

Fig. 4. Relative knee muscle onset latency. Each trace is an ensemble average of 4 trials from one subject who was representative of the sample. Trials were collected during externally induced backward sway during bilateral stance with knees straight and ankles free to move. The vertical lines indicate the onset of platform movement. Medial hamstrings (H) onset time (upward pointing arrows) was more rapid with respect to quadriceps (Q) onset time (downward pointing arrows) in the ACL-deficient (lax) extremity

inducing forward body sway. During backward sway, T and Q muscles were activated as part of a normal postural synergy which served to correct body alignment to the vertical position (Fig. 3A; top). The hamstrings were selectively activated in the ACL-deficient (lax) lower extremity as an apparent addition to the synergistic structure of the lower extremity response (Fig. 3A; bottom). In contrast, during platform-induced forward body sway the discharge amplitude of the T and Q muscles was attenuated presumably because these muscles were not needed to correct sway in the forward direction (Fig. 3B; top). During forward sway hamstrings muscles were activated bilaterally in a normally executed balance synergy (Fig. 3B; bottom).

Effect of changing stance conditions on muscle latency

Activation latencies during bilateral stance. Hamstring activation was significantly faster in the ACL-deficient extremity (Fig. 4) during bilateral stance perturbations (ACL-deficient H mean onset = 101 ms; normal mean H onset = 115 ms; $F_{1,94} = 3.97$, $p < 0.05$). The onset latencies for T and Q did not differ statistically between

Table 3. Mean (SD) and trial count (n) for neuromuscular response latencies (ms) in both lower extremities (combined) and averaged across 4 perturbation velocities for ankle stability and knee position conditions

Condition	Tibialis anterior	Quadriceps	Hamstrings
Bilateral stance			
Ankle stability:			
Ankle boot	79 (18)	95 (23)	114 (68)
(n)	184	184	169
No boot	82 (11)	101 (20)	102 (45)
(n)	192	191	184
Knee position:			
Knee straight	81 (12)	101 (21)	110 (63)
(n)	186	186	176
Knee bent	78 (17)	94 (27)	108 (51)
(n)	190	189	177
Unilateral stance			
Ankle stability:			
Ankle boot	84 (22)	86 (15)	102 (57)
(n)	191	192	181
No boot	81 (13)	90 (24)	89 (33)
(n)	189	190	181
Knee position:			
Knee straight	84 (17)	93 (20)	101 (53)
(n)	191	191	180
Knee bent	81 (19)	84 (19)	90 (40)
(n)	189	191	182

normal and ACL-deficient extremities. When data from normal and lax lower extremities were combined, Hamstring latencies were more rapid during bilateral stance perturbations without ankle stabilization ($F_{1,94}=3.81$, $p<0.05$; Table 3; top). In contrast, T and Q onsets were more rapid during support surface displacements when the ankle was stabilized by a boot (T latencies, $F_{1,94}=7.99$; Q latencies, $F_{1,94}=6.91$, $p<0.01$). Knee position also altered the T and Q onset latencies. When the knee was flexed, T and Q onsets were more rapid compared to the straight knee position (T latencies, $F_{1,94}=9.48$, $p<0.01$; Q latencies, $F_{1,94}=10.06$, $p<0.001$; Table 3, top). However, variations in knee position did not statistically alter the H latencies.

Activation latencies during unilateral stance. Unilateral stance perturbations produced a statistically non-significant trend toward more rapid H muscle onset latencies in the ACL-deficient extremity (ACL-deficient H mean onset = 90 ms; normal limb mean H onset = 100 ms; $F_{1,94}=3.70$, $p=0.057$). The onset latencies for T and Q did not differ statistically between normal and ACL-deficient lower extremities. When data from both lower extremities were combined, the H onset times (Table 3, bottom) were more rapid without ankle stabilization ($F_{1,94}=8.42$, $p<0.005$) and with the knee flexed ($F_{1,94}=4.31$, $p<0.05$) compared to ankle stabilization and knee-straight conditions respectively. The T onset latencies were also slightly more rapid without stabilization of the ankle during perturbations of unilateral stance compared to trials with application of the ankle

Table 4. Mean (SD) and trial count (n) for 200 ms and 100 ms IEMG amplitudes for comparison of homologous muscle response intensity (expressed in percent of maximum amplitude): unilateral stance perturbation

	ACL-deficient limb			Normal lower limb		
	200 ms	100 ms	n	200 ms	100 ms	n
Ankle stability:						
Ankle boot						
Tibialis anterior	74 (21)	73 (22)	95	61 (23)	66 (21)	96
Quadriceps	68 (20)	61 (20)	96	63 (21)	62 (21)	96
Hamstring	57 (18)	52 (20)	95	50 (22)	48 (24)	86
No boot						
Tibialis anterior	84 (12)	86 (13)	95	75 (17)	81 (15)	94
Quadriceps	66 (18)	59 (20)	96	56 (22)	52 (21)	94
Hamstring	70 (22)	61 (24)	94	58 (25)	54 (23)	87
Knee position:						
Knee straight						
Tibialis anterior	80 (17)	81 (16)	96	69 (20)	74 (19)	95
Quadriceps	71 (20)	65 (23)	96	64 (22)	61 (22)	95
Hamstring	60 (20)	53 (21)	96	51 (23)	51 (21)	84
Knee bent						
Tibialis anterior	79 (17)	79 (19)	94	69 (19)	75 (18)	95
Quadriceps	63 (18)	56 (18)	96	56 (20)	54 (20)	95
Hamstring	67 (20)	61 (23)	93	57 (24)	58 (26)	89

boot ($F_{1,94}=4.09$, $p<0.05$). The Q onset latencies were more rapid during perturbations of single stance with the knee flexed compared to conditions with the knee straight ($F_{1,94}=21.16$, $p<0.001$).

Amplitude of postural muscle discharge

Hamstrings amplitudes were compared to quadriceps amplitudes in the same lower extremity for each trial. A ratio of 1 indicated identical discharge amplitudes for each muscle whereas a ratio exceeding 1 represented a disproportional increase in the hamstring discharge amplitude for the 100 or 200 ms integration period following burst onset. During bilateral and unilateral stance perturbations the 200 ms H/Q ratio showed a dominant hamstring activation in the ACL-deficient limb. For bilateral stance perturbations (*inducing backward sway*), the mean H/Q ratio for the ACL-deficient limb was 1.46 compared to a mean H/Q ratio of 1.08 for the normal lower extremity ($F_{1,94}=5.38$, $p<0.05$). During unilateral stance perturbations the mean H/Q ratio for the lax lower extremity was 1.32 compared to a mean H/Q ratio of 0.87 for the normal limb ($F_{1,94}=8.70$, $p<0.01$).

The H/Q ratios for IEMG amplitudes integrated over 100 ms did not show any significant differences between lax and normal lower extremities during bilateral stance. However, unilateral stance perturbations evoked a significantly larger contribution from the Hamstrings muscles in the ACL-deficient lower extremity. The mean H/Q ratio for the ACL-deficient limb was 1.40 compared to a mean H/Q ratio in the normal lower extremity of 1.02 ($F_{1,94}=5.68$, $p<0.05$).

Muscle discharge amplitudes were normalized to the maximum amplitude obtained during the entire test for that muscle (within a given subject) in order to study relative changes in discharge amplitude for *each muscle pair* (across limbs). Tibialis and H showed significantly greater normalized discharge amplitude (100 and 200 ms IEMG) during unilateral stance perturbations on the ACL-deficient lower extremity compared to the normal lower extremity. Quadriceps discharge amplitude was also greater in the lax lower limb but only for the 200 ms integration. All muscles except for quadriceps increased discharge amplitude when the ankle was free to move (Table 4). Knee position influenced the Q and H discharge amplitude in different ways. The Q demonstrated significantly larger amplitudes when the knee was straight whereas H amplitude was significantly larger when the knee was flexed prior to support surface displacement. There were no statistical differences between the ACL-deficient lower extremity and the normal lower extremity discharge amplitudes (100 or 200 ms IEMGs) when homologous muscle pairs were compared for bilateral stance perturbations.

Trial-by-trial latency and IEMG amplitude comparisons

The aggregate summary of all conditions indicated a delayed hamstring onset compared to quadriceps (Fig. 2). However, the averaging process obscured the response profile for a majority of the subjects. A trial-by-trial analysis for each subject revealed that hamstring activation *preceded* quadriceps activation in the ACL-deficient limb more frequently for 8 of 12 subjects during bilateral stance (Fig. 5; top). The increased frequency of early hamstring activation (with respect to Q) was observed in only 4 of 12 subjects during unilateral stance (Fig. 5; bottom). The reduction of the frequency of early hamstring onsets during unilateral stance perturbations may have been due to the increased limb stabilization provided by T and Q muscles for some patients.

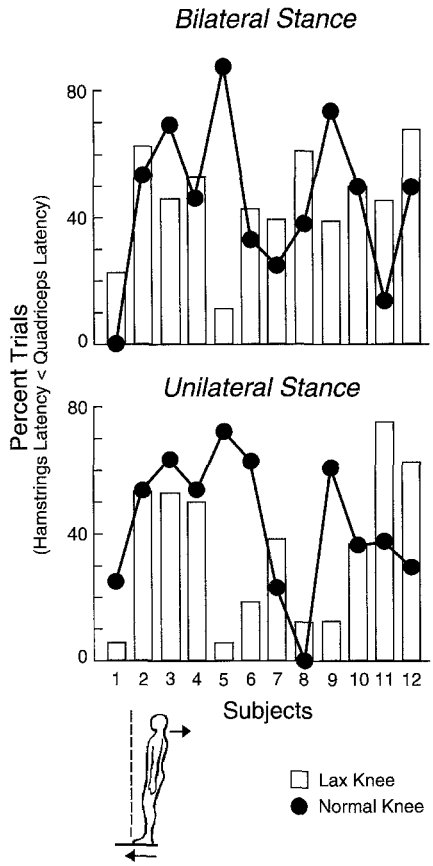

Fig. 5. Percent of trials for each subject in which medial hamstrings onset was more rapid than quadriceps onset (lax vs normal knee)

The IEMG amplitude of the hamstrings dominated the H/Q ratio for approximately half of the subjects (Fig. 6). When hamstrings amplitude was compared to quadriceps amplitude for each trial (by subject), the frequency of trials with a larger hamstring amplitude was greater in the lax lower limb for five to eight subjects during bilateral and unilateral stance (depending on condition).

Bilateral ACL-deficient subject

During the course of this study we discovered an unusual patient with bilateral ACL insufficiency. Prior injury created an equal amount of tibial displacement (13 mm) in each knee. The bilateral instability had not been surgically corrected. This patient is listed as S_{13} in Table

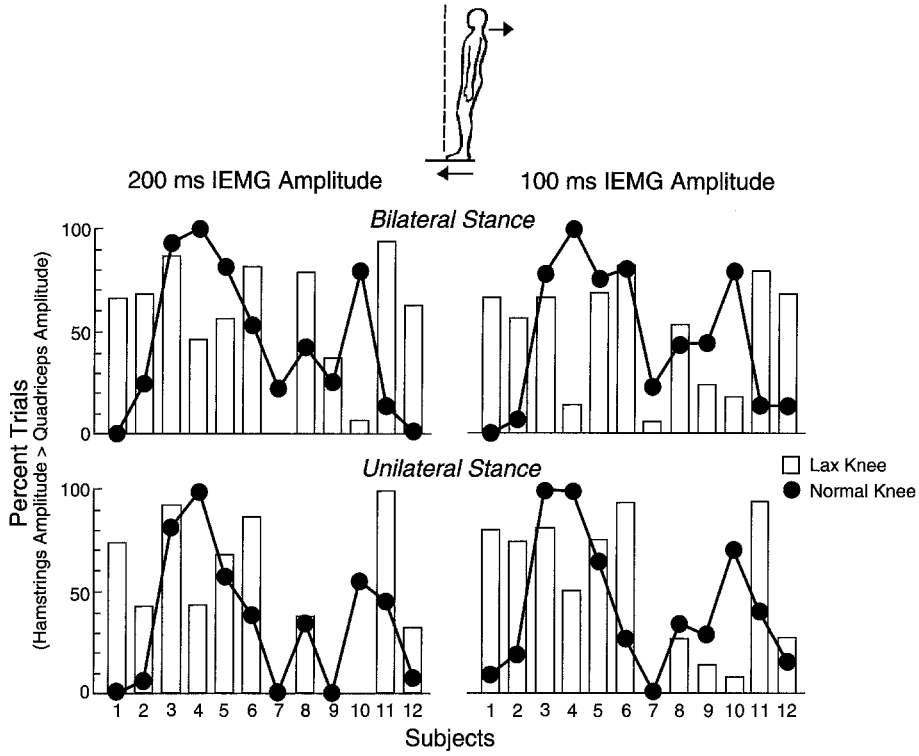

Fig. 6. Percent of trials for each subject in which medial hamstrings IEMG discharge amplitude was greater than quadriceps amplitude (lax vs normal knee)

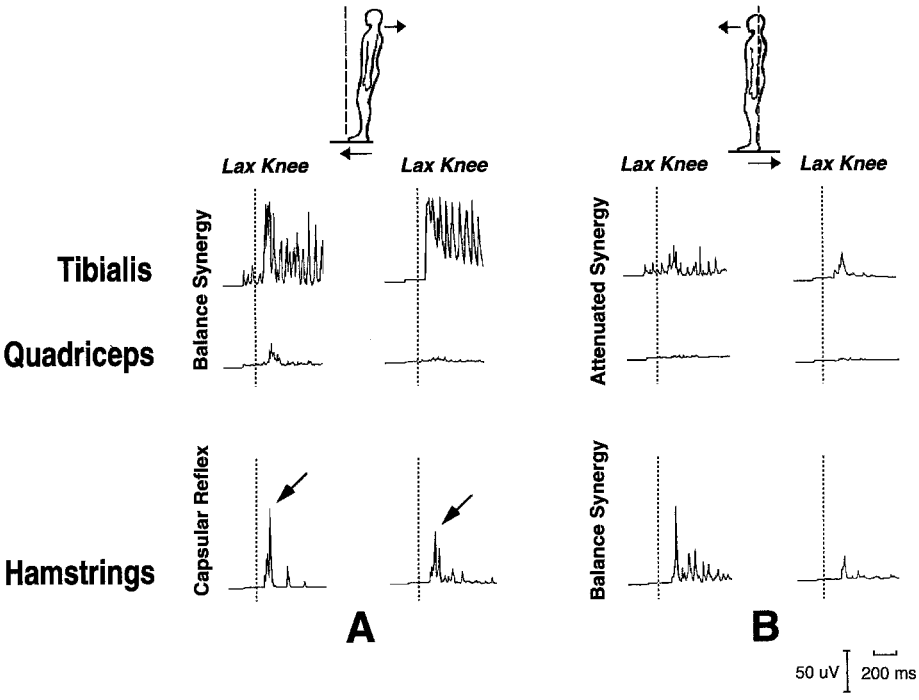

Fig. 7A, B. Bilateral capsular reflex. Each trace is an ensemble average of 4 trials from a subject who had bilateral ACL-deficiency (S_{13}). The vertical lines indicate the onset of platform displacement. **A** Externally induced backward sway during bilateral stance (knees straight and ankles free to move) produced a recruitment of tibialis anterior muscle bilaterally to correct body alignment. Medial hamstrings were recruited in both lax limbs (as indicated by the *arrows*). **B** During perturbations inducing a forward body sway under the same experimental conditions outlined for (A), tibialis muscles discharge amplitudes were attenuated and hamstrings muscles were recruited bilaterally in a balance synergy to correct body alignment

1. The experimental protocol used for S_{13} was identical to the protocol described for unilateral ACL-deficient subjects. However, the data from S_{13} were not pooled with the remainder of the sample. We observed a capsular response bilaterally which corresponded to joint laxity in each knee (Fig. 7A; bottom). In addition, latencies in both lower extremities were similar to the latencies observed in the *lax limb* of subjects with unilateral ACL deficiency (Table 5). Specifically, hamstring onsets were generally more rapid compared to Q onset latencies in each ACL-deficient extremity. The H/Q ratios for muscle discharge amplitudes (100 and 200 ms IEMG) showed a dominance of hamstring activity in each lax lower limb during all stance perturbations.

Table 5. Muscle onset latencies, and H/Q amplitude ratios for right and left lower extremities during perturbations of bilateral and unilateral stance in one subject with bilateral ACL-deficiency[a]

	Bilateral stance perturbation		Unilateral stance perturbation
Onset latencies (ms) [mean (SD)]:			
Right			
Tibialis anterior	71 (9)		72 (8)
Quadriceps	93 (22)		81 (16)
Hamstrings	89 (11)		86 (11)
Left			
Tibialis anterior	69 (11)		75 (11)
Quadriceps	96 (20)		102 (17)
Hamstrings	80 (8)		81 (10)
Percent trials hamstring onset < quadriceps onset:			
Right	56%		33%
Left	75%		87%
Hamstrings/Quadriceps IEMG ratio:		[200 ms IEMG]	
Right	2.84 (0.55)		2.27 (0.42)
Left	4.13 (0.72)		3.47 (0.58)
		[100 ms IEMG]	
Right	3.39 (0.88)		2.85 (0.67)
Left	5.22 (1.13)		3.74 (1.12)

[a] Data averaged across velocities, knee position, and ankle stability conditions. Sixteen trials were analyzed for each muscle in each stance condition

Discussion

Damage to the anterior cruciate ligament creates instability in the knee joint. The joint laxity resulting from ACL insufficiency increases the tensile stress on secondary capsular restraints (Daniel et al. 1985; Solomnow et al. 1987). Previous studies have suggested that mechanoreceptors in the human and cat knee joint capsule provide end-organs for a reflex arc that facilitates muscle action to control joint instability (Kalund et al. 1990; Solomnow et al. 1987; Freeman and Wyke 1966). However, direct measurement of the spatial and temporal aspects of such a reflex has not been previously reported in humans. In the present study we analyzed the onset latencies and discharge amplitudes of postural synergists during reactions to support surface displacements in subjects with unilateral knee joint laxity. Our goal was to describe the effect of hypermobility in the knee on the organization of the postural response.

Postural muscle onset latencies in the ACL-deficient limb: restructured preprogrammed response

External perturbations inducing a backward body sway normally activate postural synergists (T and Q) which function to realign the body axis to the vertical position (Diener et al. 1983; Nashner 1977). The results of the present investigation showed that:
1) subjects with unilateral ACL-deficits had normal synergistic activation of T and Q in the non-injured limb (Fig. 3A),
2) the postural synergy in the ACL-deficient lower extremity was restructured to include hamstring muscle activation with onset latencies that reflected long-loop processing (Figs. 2 and 3A), and
3) hamstrings activation occurred more rapidly in the lax limb compared to the non-injured limb (Fig. 4).
Previous studies (Horak and Nashner 1986; Horak et al. 1990; Macpherson et al. 1989) have proposed that the activation of postural synergists is preprogrammed by central mechanisms using various combinations of a finite set of response strategies. It was thought that a limited number of motor output patterns could be combined (without altering the essential elements of the original motor patterns) in order to optimize the response to novel task conditions. For example, Horak and Nashner (1986) described two distinct balance responses (a hip and an ankle strategy) that were combined to varying degrees while subjects practiced standing on a narrow beam. The complex responses observed during practice on a narrow support surface had all the identifiable elements of a pure ankle strategy interdigitated with a pure hip strategy (see Fig. 5; Horak and Nashner 1986). In contrast, our findings indicated that the sensory input from stimulation of a hypermobile joint was sufficient to alter the structure of the preprogrammed balance response in a manner not previously defined (Figs. 3 and 7). Our results indicate that postural responses can be unilaterally restructured and pre-programmed to compensate for joint hypermobility. Given the number of joints (and degrees of freedom) involved with the control of upright stance, the restructuring of synergistic activity may be derived from an array of possibilities much larger than previously proposed.

The recruitment of postural synergists, to some degree, is controlled in advance of a balance perturbation (Di Fabio et al. 1990; Di Fabio and Badke 1989; Diener

et al. 1988). The selection of postural muscles to correct body sway is largely dependent upon inputs that provide knowledge of the intrinsic and extrinsic mechanical constraints of the balance task (Macpherson et al. 1989). During perturbations inducing backward sway, the activation of T (at an 80 ms latency) results in a forward pull on the shank because the feet are fixed on the supporting surface. The onset of hamstring EMG activity in the ACL-deficient limb at 100 ms indicates that the addition of this muscle to the postural synergy was programmed in advance of the perturbation in order to restrict excessive anterior motion of the tibia on the femur.

The relatively stable recruitment latency of H (Fig. 2) and the early recruitment of H in the lax limb (Fig. 4) would be consistent with the view that hamstrings activation was learned as part of a functional response to restrict excessive anterior motion of the tibia (with respect to the femur). Our findings fit nicely with those of Kalund et al. (1990) who reported a more rapid onset of hamstrings EMG during inclined treadmill walking for subjects with ACL insufficiency. Alteration of the structure and latency of a preprogrammed postural response could be based on a cognitive set developed from prior sensorimotor experience (Evarts and Tanji 1974; Horak et al. 1989). In addition, our evidence suggests that the onset of hamstrings EMG activation in the ACL-deficient lower extremity was somewhat "invariant" (and thereby pre-programmed by central mechanisms) whereas the activation of H in the non-injured extremity was highly variable and not tightly coupled within the postural synergy (Fig. 2).

It could be argued that the long hamstring latencies were due to delays in the mechanical stimulation of joint receptors following loss of ligamentous integrity. It would follow that an alternate response could then be "unmasked" revealing an actual latency more characteristic of a segmental stretch reflex. In order for this argument to be credible, at least two conditions would have to be met; (1) the muscle response would have to be localized to the knee and (2) the pattern of activation would have to be dependent on an adequate stimulus rather than on the context of stimulus delivery. It was not possible to fully answer this question given the limited number of muscles studied in our investigation. However, Lass et al. (1991) reported early onset latencies for five different knee muscles (including knee flexors, extensors, and the gastrocnemius) in subjects with complete rupture of the anterior cruciate ligament during treadmill gait at different grades of inclination (see Table 1 in Lass et al. 1991). While this observation would meet the "localized-response" criteria for a establishing a segmental reflex mechanism, not all of the muscles studied by Lass et al. (1991) fired early in all conditions. For example, medial hamstring onset was earlier in ACL-deficient subjects, but only for low grades of inclination. It can be assumed that higher grades of traedmill tilt would continue to provide an adequate physiological stimulus for depolarization of joint receptors. Changes in the distribution of early onset latencies across different muscles as the inclination was augmented seemed to suggest that the recruitment pattern in ACL-deficient subjects was context-dependent and that some central mechanism was coordinating the compensatory pattern. A generalized response deficit involving posture and gait following resection of the sensory articular nerves in the cat knee joint provides further evidence of a centrally mediated response dependent upon inputs from articular receptors (Freeman and Wyke 1966).

The hamstrings in the ACL-deficient extremity did not appear to be co-activated with the quadriceps to provide joint stability. While the mean onset latency of the hamstrings appeared longer with respect to the quadriceps onset time when all conditions were averaged (Fig. 2), this aggregate summary obscured the response profile for a majority of the subjects (see Fig. 5). The lack of simultaneous activation of antagonist muscles has been previously interpreted as evidence for a central pattern generator (Horak and Nashner 1986). However, the existence of a pattern generator (with a finite set of responses) for restructuring postural synergies did not provide an adequate explanation for the results observed in the present study because a unique postural pattern was created by the presence of joint hypermobility (Figs. 3 and 7). One could speculate that alterations in mechanoreception following ligamentous lesion in *any* weight-bearing joint might provide the necessary afferent input to uniquely restructure the postural synergy as a way to compensate for ligamentous laxity.

Contribution of joint afference to long-loop reflexes

Mechanoreceptors in the ACL-deficient knee joint capsule provided a likely source for the afferent input necessary to restructure the postural synergy. Horstmann and Dietz (1990) suggested that force-dependent receptors distributed along the vertical axis of the body in the joints and vertebral column provided a gain control mechanism for postural muscle discharge. However, studies addressing the contribution of lower extremity joint afference to various components of long-loop postural reflexes have been focused on the ankle and have not addressed the issue of capsular mechanoreception following the loss of ligamentous support. Several reports indicated that skin receptors from the foot and joint receptors from the ankle did not contribute to medium latency (50 ms) long loop reflexes (Ilces 1977; Chan et al. 1979; Diener et al. 1984b). The literature is equivocal with regard to sensory modulation of the late EMG component (100 ms) of long loop reflexes. Diener et al. (1984b) found that the latency and amplitude of late antagonistic response in the tibialis anterior was independent of afferences from the foot and ankle. Others have shown that the amplitude of this late response in sitting was attenuated by blocking joint afferents (Katz and Rondot 1978). Cutaneous, joint, and pressure receptors around the foot and ankle have been found to contribute significantly to postural stabilization in the lower frequency domain but a threshold stimulus duration of 75 ms was required before sensory information could influence balance behavior (Diener et al. 1984a, 1988).

From a functional perspective, Freeman et al. (1965) noted that patients with previous ankle injury had persistent functional instability. However, these findings were not replicated in a subsequent investigation (Tropp et al. 1984). The equivocal results are difficult to reconcile because objective measures of joint laxity were not reported.

Inhibitory influence of joint capsule afference on postural muscle discharge in the ACL-deficient limb

It is possible that afferent information from the knee-joint capsule contributed to the inhibition of quadriceps muscle activity. The 100 ms and 200 ms H/Q ratios showed a dominance of hamstring activity during perturbations of *unilateral stance*. In addition, the discharge amplitude of hamstrings dominated the H/Q ratio (200 ms IEMG) for *bilateral stance* (Fig. 6). The notion that mechanoreceptors in the anterior-joint capsule can inhibit quadriceps muscles agrees with earlier findings involving cat limb-muscle reflexes (Freeman and Wyke 1966). In addition, loads directly applied to the ACL of chloralosed anesthetized cats were previously reported to facilitate hamstrings discharge (Solomonow et al. 1987). If similar neural networks exist in humans, the loss of ACL inhibitory influence on the quadriceps following ligament rupture might be compensated by a capsular reflex potentiated through hypermobility of the knee joint. One must consider other sources of peripheral sensory afference as having an influence on quadriceps muscle activity. However, the precise weighted influence of joint mechanoreceptors on the automatic postural response mechanism could not be determined in the present study.

Capsular-hamstring reflex: restructuring of the postural synergy

Previous work has shown that polysynaptic activation of postural muscles is functionally directed and is not dependent on muscle stretch alone as an adequate stimulus (Gollhofer et al. 1989; Di Fabio et al. 1986; Diener et al. 1983; Nashner 1976). The coordination of leg-muscle activation during perturbations of gait (Dietz et al. 1989b), during parabolic flight (Layne and Spooner 1990), or under conditions of simulated micro-gravity (Horstmann and Dietz 1990; Dietz et al. 1989a) indicated that reflex patterns in postural muscles were strongly linked to joint afference.

In the present study, T and Q muscles represented a pair of postural muscles acting synergistically to correct a posterior sway of body mass. The activation of H muscles would normally be antagonistic to the correction of posterior body sway (e.g.; causing the body to fall further backwards). The selective restructuring of a postural synergy to include unilateral hamstring activation suggests that central balance mechanisms may utilize input from joint mechanoreceptors to correct for localized joint hypermobility. The dominance of rapid hamstring onset and enhanced H amplitudes (compared to Q) in the lax lower extremity suggests that joint afference in humans can modify postural responses to that antagonistic muscles are converted to functional synergists when it is necessary to maintain balance. Collectively, these results suggest that the critical variable for regulating upright stance is related to controlling the placement of the center of gravity. Further study is needed to determine (1) how the *degree* of ligamentous laxity correlates with alterations in the synergistic structure of automatic postural responses and (2) if anesthetic injection of the ACL-deficient lower extremity will alter the temporal and/or spatial characteristics of the synergistic patterns utilized to counter-act ligamentous laxity.

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