

Reflex Inhibition of the Quadriceps Femoris Muscle after Injury or Reconstruction of the Anterior Cruciate Ligament*

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ABSTRACT: A burst-superimposition technique was used to assess the strength of the quadriceps femoris muscle in three groups of patients. Group 1 comprised twenty patients who had had a torn anterior cruciate ligament of the knee and had a reconstruction of the ligament one to six months after the injury. Group 2 comprised twelve patients who had had a torn anterior cruciate ligament for an average of three months (a subacute tear). Group 3 comprised eight patients who had had a torn anterior cruciate ligament for an average of two years (a chronic tear). The patients in Groups 2 and 3 had not had an operation for the torn ligament.

The patients in Groups 1 and 3 had no evidence of failure of activation of the involved quadriceps, but nine of the twelve patients in Group 2 had reflex inhibition of contraction of the muscle.

Patients who have had a reconstruction of the anterior cruciate ligament demonstrate substantial weakness of the quadriceps femoris muscle.^{1,21,23,28} Although disuse atrophy is most commonly cited as the cause of the weakness in these patients, others have attributed the weakness to an inability to voluntarily activate the quadriceps femoris muscle fully.¹⁷ Deficits in the performance of the quadriceps femoris muscle have been observed in the absence of any morphological changes that are usually associated with atrophy.^{7,16} This has led some investigators to suggest that rupture of the anterior cruciate ligament disrupts the drive of the quadriceps femoris muscle by the central nervous system because of a change in the firing of afferent impulses from joint receptors.^{16,24-26}

Failure of voluntary activation of the quadriceps

femoris muscle has been demonstrated in the presence of a painful knee, a knee in which there is a chronic effusion, and a normal knee in which there is an experimentally induced effusion.^{4,10,14,27} Failure of voluntary activation has also been demonstrated in normal muscle after fatigue.¹⁹ Some investigators have suggested that the absence of an anterior cruciate ligament interferes with the patient's ability to activate the quadriceps femoris muscle fully,^{2,24,26} and others have demonstrated persistent failure of voluntary activation of the quadriceps femoris muscle for years after an injury and rehabilitation.^{12,25} Although most of these studies were done in patients who had a rupture of an anterior cruciate ligament that had not been reconstructed, the results have been extrapolated to apply to the postoperative population, in whom persistent weakness of the quadriceps femoris is also a major problem.

Investigators have called this failure of voluntary activation, reflex inhibition or arthrogenous muscle inhibition.^{14,17,18} If reflex inhibition is a cause of weakness of the quadriceps femoris muscle after rupture of the anterior cruciate ligament, with or without operative reconstruction, then traditional, volitional exercise would not be expected to remedy the deficit. Hurley et al. demonstrated that a rehabilitation program of exercise of the quadriceps femoris muscle did not affect the measured quadriceps inhibition in patients who had had a rupture of the anterior cruciate ligament.¹²

Our interest in neuromuscular stimulation after reconstruction of an anterior cruciate ligament has led us to investigate the presence of reflex inhibition of the quadriceps in this population. The purpose of this study was to determine whether patients who have a rupture of the anterior cruciate ligament can fully activate the quadriceps femoris muscle before an operation and in the early phase of rehabilitation after reconstruction of the ligament. We hypothesized that there would be significant failure of voluntary activation associated with weakness of the quadriceps femoris muscle in patients who had a torn anterior cruciate ligament and in those who had had a reconstruction of the ligament.

Methods

Subjects

Group 1 comprised twenty patients who had had an arthroscopically assisted intra-articular reconstruction

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of the anterior cruciate ligament and who were tested within three months (average, eight weeks) after the operation. In all twenty patients, the reconstruction had been done between one and six months after the injury. There were five female patients and fifteen male patients, who ranged in age from fifteen to thirty-four years (average, twenty-five years). In ten patients, the reconstruction was done with an Achilles tendon allograft; in five, with a patellar ligament allograft; and in five, with an autologous semitendinosus-gracilis graft.

Group 2 comprised twelve patients who had had a unilateral rupture of the anterior cruciate ligament for an average of three months (range, one to six months) (a subacute tear). There were four men and eight women, who ranged in age from eighteen to forty-one years (average, twenty-five years).

Group 3 comprised eight patients who had had a unilateral rupture of the anterior cruciate ligament for an average of two years (range, six months to five years) (a chronic tear). There were three men and five women, who ranged in age from twenty-four to forty-five years (average, thirty-one years).

A diagnostic arthroscopy and stress tests had been performed on all patients, while they were under anesthesia, to rule out any concomitant ligamentous injury, fracture, meniscal repair, or effusion of the knee; patients who had any of these conditions were excluded from the study. The study protocol was approved by the institutional review board of the University of Delaware, and all patients gave informed consent.

Procedure

The analysis of muscle performance consisted of tests of the strength of the quadriceps femoris muscle on the involved side and on the uninvolved, contralateral side. The testing began with a determination of the maximum voluntary isometric contraction of the quadriceps femoris muscle of the uninvolved extremity. The patients were seated and were stabilized in an electro-mechanical dynamometer (KinCom; Chattanooga Corporation, Chattanooga, Tennessee) with the knee in 60 degrees of flexion. A burst-superimposition technique was used to measure maximum voluntary isometric contraction^{20,22}. Carbon-rubber electrodes and water-soaked sponges were placed over the proximal part of the vastus lateralis and the distal part of the vastus medialis and were secured with Velcro straps.

A ten-pulse tetanic electric train of 100 pulses per second and 600-microsecond duration was delivered to the resting quadriceps femoris muscle. The stimulator (model S88; Grass Instruments, Quincy, Massachusetts) was set to deliver a supramaximum stimulus. (During previous testing of healthy subjects and of those in whom the anterior cruciate ligament had been reconstructed, we had determined that a stimulator setting of 13 and a stimulus-isolation-unit setting of 20 was supramaximum for all subjects tested.) Approximately two

seconds after application of the burst, the patient attempted to contract the quadriceps femoris muscle maximally; during the attempt, the patient was encouraged by the tester and was able to see the result on the dynamometer. A second burst was superimposed on the voluntary contraction 5000 milliseconds after the resting burst to be certain that the patient had performed a maximum contraction.

The force from each contraction was recorded from the dynamometer force-transducer and was sampled at 200 hertz. If the voluntary isometric contraction was a true maximum, no increase in force (or a slight decrease in force) from that already produced by the voluntary contraction was seen. If a patient had produced less than a maximum contraction, a tetanic contraction was seen superimposed on the voluntary contraction. If the amplitude of the superimposed tetanic contraction was more than 5 per cent of the gravity-corrected, voluntary contraction, strength-testing was repeated until the measured values of the force began to decline. An adequate rest period of one to three minutes was allowed between efforts to avoid fatigue.

Similar tests were conducted on the involved side.

Management and Analysis of the Data

The maximum isometric strengths of the involved and uninvolved quadriceps muscles were compared with the use of a two-tailed, paired *t* test for dependent measures. The level of significance (α) was 0.05. The recorded measurements of force were compared with the use of a one-way repeated-measures analysis of variance. The maximum voluntary quadriceps force was measured as the maximum force generated before the application of the superimposed burst. The burst augmentation was measured as the change in muscle

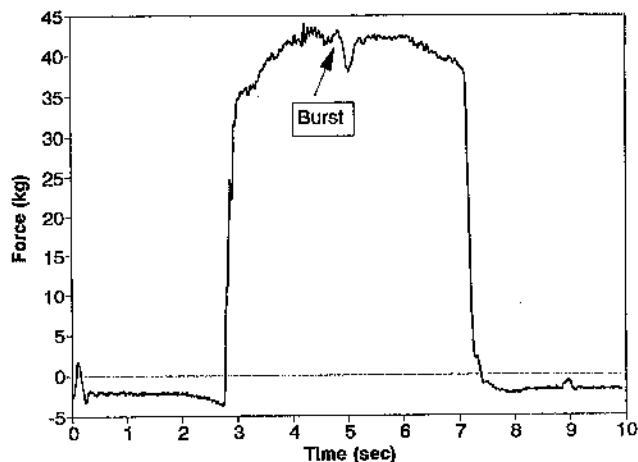


FIG. 1

Graph of a typical isometric contraction of the quadriceps femoris muscle in response to burst-superimposition testing in a patient who had had a reconstruction of the anterior cruciate ligament (Group 1). Five seconds after the resting twitch, a burst is applied. The output force decreases and then returns to peak after the burst has been completed.

force output during the 100 milliseconds after the application of the stimulus. Inhibition was defined as the difference between the two forces multiplied by 100 per cent divided by the peak force.

A two-way (group by contraction) repeated-measures analysis of variance was used to determine if there was a significant difference between the initial force and the force produced by the superimposed burst, and if there was a difference in the response between the groups.

Results

The patients who had had a reconstruction of the anterior cruciate ligament (Group 1) were able to produce a force from a maximum voluntary isometric contraction such that the force from the additional superimposed twitch was increased less than 5 per cent (corrected for gravity, in a single test session of no more than four trials) (Fig. 1). Group data show that when the stimulation was applied, there was no significant augmentation of muscle force compared with the voluntary activity of either the involved or the uninvolved quadriceps ($p < 0.05$). In fact, the group data show a measurable, significant decline in force when the stimulation was applied ($p < 0.05$) (Fig. 2). The maximum force from a voluntary isometric contraction on the involved side was 77 per cent of that on the uninvolved side.

The patients who had a chronic rupture of the anterior cruciate ligament (Group 3) had results that were similar to those of the group that had had the reconstruction in that there was a measurable decline, rather

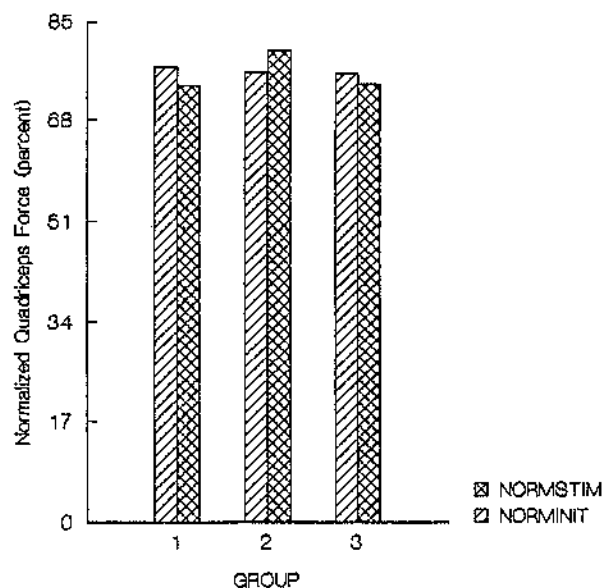


FIG. 2

Graph of the peak forces of the involved quadriceps femoris muscle, normalized to the peak force of the muscle on the uninvolved, contralateral side before (NORMINIT) and during (NORMSTIM) the application of the burst, for Group 1 (reconstructed ligament), Group 2 (subacute tear of the ligament), and Group 3 (chronic tear).

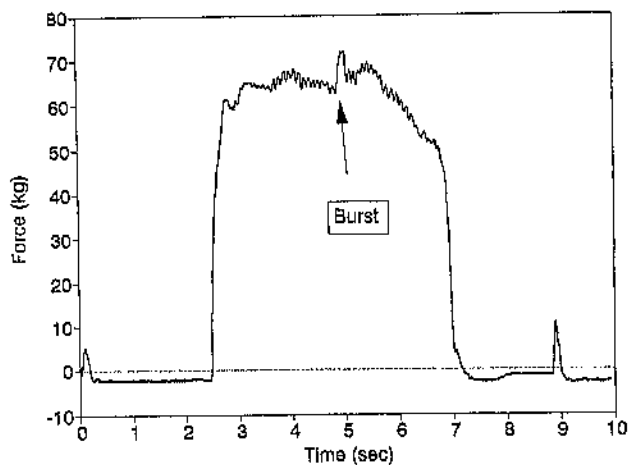


FIG. 3

Graph of a typical isometric contraction of the quadriceps femoris muscle in response to burst-superimposition testing in a patient who had a subacute tear of the anterior cruciate ligament (Group 2). Five seconds after the resting twitch, a burst is applied. The output force increases and then returns to peak after the burst has been completed.

than an increase, in force when the stimulation was applied (Fig. 2). The force of the maximum voluntary isometric contraction on the involved side was 76 per cent of that on the uninvolved side.

Nine of the twelve patients who had a subacute rupture (Group 2) had inhibition of more than 5 per cent that could not be eliminated within a single test session (Fig. 3). The group data show a measurable, significant increase in force ($p < 0.05$) when the stimulation was applied to the involved quadriceps femoris (Fig. 2). The force of the maximum voluntary isometric contraction on the involved side was 76 per cent of that on the uninvolved side.

Discussion

Four types of neuroreceptors have been described in the capsule of the knee joint and in the intra-articular structures³⁰. All four are responsible for the primary afferent discharges from the joint and all affect function¹⁵. Distension of the knee joint — either experimentally induced or chronic — has been implicated as a cause of diminished voluntary activation of the quadriceps femoris muscle^{4,10}. Some investigators have suggested that an effusion distends the joint capsule and the resultant activation of afferent impulses reflexively inhibits the quadriceps femoris muscle. The failure of voluntary activation of the quadriceps femoris muscle that results from experimentally induced effusion is reversed when the effusion is removed^{4,27}.

However, the results from studies of chronic knee effusions are conflicting. Some have shown reversal of the reflex inhibition after aspiration; others have not^{10,14}. Therefore, distension of the joint may not be the most critical factor in the failure of voluntary activation of the quadriceps femoris.

Mechanoreceptors have been identified in the anterior cruciate ligament, and attempts have been made to estimate the specific contribution of the anterior cruciate ligament to movement and position sense¹³. Injury to the anterior cruciate ligament and laxity of the joint may affect receptors from which afferent impulses arise and may alter the motor messages to the quadriceps-femoris and hamstring-muscle groups. Some investigators have reported substantial impairment of proprioception in knees in which the anterior cruciate ligament was torn, compared with proprioception in the uninjured knees, but others have reported no such difference^{3,6,15}. Solomonow et al. presented evidence for a direct reflex arc from the anterior cruciate ligament to the hamstring muscles^{24,26}.

The joint capsule contains a dense aggregate of mechanoreceptors that are believed to play a role in proprioception and protection of the joint³⁰. In the knee joint, Ruffini endings (mechanoreceptors) are concentrated in the superficial, anterior and posterior aspects of the capsule; they detect changes in pressure and deformation caused by displacement of the joint¹¹. In a patient in whom the anterior cruciate ligament is ruptured, there is increased deformation of the joint capsule as the tibia subluxates anteriorly; this may cause inhibition of the quadriceps femoris muscle. When a torn anterior cruciate ligament has been reconstructed, and there are also no mechanoreceptors in the graft, perhaps there is no failure of voluntary activation of the quadriceps femoris muscle because the joint stability has been restored and there is less capsular deformation.

The patients in Group 1 were able to activate the weak quadriceps femoris muscles fully after reconstruction of the anterior cruciate ligament. The group data show that there was actually a decrease in force in the 100 milliseconds after the application of the superimposed burst. This represents a collision of the antidromic electrical and orthodromic voluntary action potentials that have come to be recognized as the hallmark of full muscle activation (Fig. 1). The amplitude and shape of the resting response are equal and opposite to the superimposed burst response.

Hurley et al.¹² and Newham et al.¹⁸ used a twitch-superimposition technique, which is similar to the burst-superimposition technique, to investigate inhibition of the quadriceps femoris muscle in patients who had a torn anterior cruciate ligament. The authors demonstrated substantial inhibition of the quadriceps femoris^{12,18} that was most profound during isometric contractions and at slow isokinetic velocities and that persisted even after a period of rehabilitation¹². If the patients truly have a failure of voluntary activation of the quadriceps, then logically a volitional training regimen would be ineffective in increasing muscle strength.

The studies of Hurley et al.¹² and of Newham et al.¹⁸ did not include patients in whom the anterior cruciate ligament had been reconstructed. Our patients who had

a reconstruction of the ligament (Group 1) were studied a much shorter period of time after the injury, but our patients who had not had a reconstruction (Groups 2 and 3) were quite similar to those of Hurley et al. and of Newham et al. Nevertheless, the patients in our Groups 2 and 3 had much less inhibition than the patients of Hurley et al. and of Newham et al. Several differences in the procedures in the studies may explain the difference in the extent of inhibition. The electrical stimulation in the present study was much more powerful; this may have motivated the patients to make a stronger maximum voluntary isometric contraction. There is some brief discomfort associated with supramaximum stimulation. The patients were told that if they contracted the quadriceps femoris muscles forcefully and there was no significant electrical superimposition, they would not be stimulated again during the session. Additionally, in the studies of Hurley et al. and of Newham et al., all practice contractions were voluntary; none included the electrical set-up. We found that some very large forces were generated by the burst superimpositions during the first, and sometimes the second and third, contractions. However, all patients in whom the anterior cruciate ligament had been reconstructed were able to eliminate the effect of the superimposition almost completely by the fourth trial. This may indicate that practice alone is sufficient to overcome any inhibition after reconstruction of the anterior cruciate ligament. Studies of early gains in voluntary strength showed large increases in muscle performance before the development of hypertrophy^{8,9}. Although the neural mechanisms of these gains in strength are not well understood, some authors have maintained that practice alone (even mental practice) can change the programming in the central nervous system for the production of a maximum voluntary contraction²⁹.

The findings in the patients in Group 1 are similar to those of isometric contraction in healthy subjects: the maximum force of voluntary contraction could be predicted from submaximum efforts. In the present study, as the force of the contraction increased from trial to trial, the tracing of the height of the superimposed twitch decreased in a complementary fashion. In fact, the behavior of the involved quadriceps femoris muscles in patients who had had a reconstruction was identical to that of the uninvolved muscles except that the involved muscles were weaker.

Clinically, it is extremely pertinent that the weak muscle could be fully activated by patients in whom a reconstruction had been done from one to six months after the injury. If the involved quadriceps femoris muscle is not inhibited, then volitional exercise should have the potential to strengthen the muscle to a level that is equal to that of the uninvolved, contralateral muscle; thus, these findings shed little light on the reason why weakness of the quadriceps femoris muscle persists after reconstruction of the anterior cruciate ligament.

Perhaps irreversible muscle atrophy or an alteration in muscle cells occurs, or patients are not encouraged to contract the muscles vigorously enough during exercise. Provision of verbal and visual feedback may improve the performance of an exercise and increase the gain in strength after reconstruction of an anterior cruciate ligament. We, as well as others, have demonstrated that neuromuscular electrical stimulation after reconstruction increases the strength of the quadriceps more than a similar regimen of volitional exercise^{5,23}. Perhaps the neuromuscular electrical stimulation overcomes the patient's tendency not to contract the quadriceps fully.

Our finding that the patients who had a chronic tear (Group 3) demonstrated full voluntary activation is puzzling in light of the findings in Group 2 and those of previous investigators^{15,24}. It is possible that the joint receptors in the capsule that fire in response to stretch, thereby inhibiting the quadriceps femoris muscle, accommodate to the new (stretched) capsular length and no longer fire. However, the fact that the involved quadriceps femoris muscles of Group 3 continued to be weaker than the uninvolved muscles tends to discount this explanation. It is more likely that the affected muscle fibers atrophy as a result of the subacute failure of voluntary activation. In the chronic condition, then, the fibers can no longer be activated by any means, even superimposed supramaximum electrical stimulation. If this is the case, patients who have a reconstruction long

after a chronic tear of the anterior cruciate ligament will probably never regain full strength of the quadriceps. However, in this study, we did not test patients who had had a reconstruction for a chronic tear.

All patients who had had a reconstruction, regardless of the type of operation, were able to achieve full activation of the involved muscle in a single test session, even though the involved muscle was weaker than the uninvolved, contralateral muscle. No patient required more than four trials; most required only two. These data suggest that, at least in patients who have had a reconstruction, there is no reflex inhibition of the quadriceps femoris after injury to the anterior cruciate ligament.

Most of the patients who had had a torn anterior cruciate ligament for six months or less before testing — and who had not had a reconstruction — were incapable of full voluntary activation of the quadriceps muscles, and therefore may not have responded well to a rehabilitation program that relied solely on volitional exercise. If quadriceps inhibition is a direct result of capsular stress from joint laxity, and if irreversible atrophy occurs after a period of time, then a patient who has a torn anterior cruciate ligament may be able to regain full function of the quadriceps only if the reconstruction is done before such atrophy occurs.

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References

1. Arvidsson, I.; Eriksson, E.; Haggmark, T.; and Johnson, R. J.: Isokinetic thigh muscle strength after ligament reconstruction in the knee joint: results from a 5-10 year follow-up after reconstructions of the anterior cruciate ligament in the knee joint. *Internat. J. Sports Med.*, 2: 7-11, 1981.
2. Barratta, R.; Solomonow, M.; Zhou, B. H.; Letson, D.; Chuinard, R.; and D'Ambrosia, R.: Muscular coactivation. The role of the antagonist musculature in maintaining knee stability. *Am. J. Sports Med.*, 16: 113-122, 1988.
3. Barrack, R. L.; Skinner, H. B.; and Buckley, S. L.: Proprioception in the anterior cruciate deficient knee. *Am. J. Sports Med.*, 17: 1-6, 1989.
4. deAndrade, J. R.; Grant, C.; and Dixon, A. St. J.: Joint distension and reflex muscle inhibition in the knee. *J. Bone and Joint Surg.*, 47-A: 313-322, March 1965.
5. Dellitto, A.; Rose, S. J.; McKowen, J. M.; Lehman, R. C.; Thomas, J. A.; and Shively, R. A.: Electrical stimulation versus voluntary exercise in strengthening thigh musculature after anterior cruciate ligament surgery. *Phys. Ther.*, 68: 660-663, 1988.
6. Dvir, Z.; Koren, E.; and Halperin, N.: Knee joint position sense following reconstruction of the anterior cruciate ligament. *J. Orthop. and Sports Phys. Ther.*, 10: 117-120, 1988.
7. Elmqvist, L. G.; Lorentzon, R.; Johansson, C.; and Fugl-Meyer, A. R.: Does a torn anterior cruciate ligament lead to change in the central nervous drive of the knee extensors? *European J. Appl. Physiol.*, 58: 203-207, 1988.
8. Enoka, R. M.: Muscle strength and its development. New perspectives. *Sports Med.*, 6: 146-168, 1988.
9. Enoka, R. M., and Fuglevand, A. J.: Neuromuscular basis of the maximum force capacity of a muscle. In *Current Issues in Biomechanics*, pp. 215-235. Edited by M. D. Grabiner. Champaign, Illinois, Human Kinetics, 1993.
10. Fahrer, H.; Rentsch, H. U.; Gerber, N. J.; Beyeler, C.; Hess, C. W.; and Grünig, B.: Knee effusion and reflex inhibition of the quadriceps. A bar to effective retraining. *J. Bone and Joint Surg.*, 70-B(4): 635-638, 1988.
11. Freeman, M. A., and Wyke, B.: The innervation of the knee joint. An anatomical and histological study in the cat. *J. Anat.*, 101: 505-532, 1967.
12. Hurley, M. V.; Jones, D. W.; Wilson, D.; and Newham, D. J.: Rehabilitation of quadriceps inhibited due to isolated rupture of the anterior cruciate ligament. *J. Orthop. Rheumatol.*, 5: 145-154, 1992.
13. Johansson, H.: Role of knee ligaments in proprioception and regulation of muscle stiffness. *J. Electromyog. and Kinesiol.*, 1: 158-179, 1991.
14. Jones, D. W.; Jones, D. A.; and Newham, D. J.: Chronic knee effusion and aspiration: the effect on quadriceps inhibition. *British J. Rheumatol.*, 26: 370-374, 1987.
15. Krause, R.; Schmidt, M.; and Schaible, H.-G.: Sensory innervation of the anterior cruciate ligament. An electrophysiological study of the response properties of single identified mechanoreceptors in the cat. *J. Bone and Joint Surg.*, 74-A: 390-397, March 1992.
16. Lorentzon, R.; Elmqvist, L. G.; Sjöstrom, M.; Fagerlund, M.; and Fugl-Meyer, A. R.: Thigh musculature in relation to chronic anterior cruciate ligament tear: muscle size, morphology, and mechanical output before reconstruction. *Am. J. Sports Med.*, 17: 423-429, 1989.

17. **Morrissey, M. C.:** Reflex inhibition of thigh muscles in knee injury. Causes and treatment. *Sports Med.*, 7: 263-276, 1989.
18. **Newham, D. J.; Hurley, M. V.; and Jones, D. J.:** Ligamentous knee injury and muscle inhibition. *J. Orthop. Rheumatol.*, 2: 163-173, 1989.
19. **Newham, D. J.; McCarthy, T.; and Turner, J.:** Voluntary activation of human quadriceps during and after isokinetic exercise. *J. Appl. Physiol.*, 71: 2122-2126, 1991.
20. **Rutherford, O. M.; Jones, D. A.; and Newham, D. J.:** Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. *J. Neurol., Neurosurg. and Psychiat.*, 49: 1288-1291, 1986.
21. **Seto, J. L.; Orofino, A. S.; Morrissey, M. C.; Medeiros, J. M.; and Mason, W. J.:** Assessment of quadriceps/hamstring strength, knee ligament stability, functional and sports activity levels five years after anterior cruciate ligament reconstruction. *Am. J. Sports Med.*, 16: 170-180, 1988.
22. **Snyder-Mackler, L.; Binder-Macleod, S. A.; and Williams, P. R.:** Fatigability of human quadriceps femoris muscle following anterior cruciate ligament reconstruction. *Med. and Sci. Sports and Exerc.*, 25: 783-789, 1993.
23. **Snyder-Mackler, L.; Ladin, Z.; Schepesis, A. A.; and Young, J. C.:** Electrical stimulation of the thigh muscles after reconstruction of the anterior cruciate ligament. Effects of electrically elicited contraction of the quadriceps femoris and hamstring muscles on gait and on strength of the thigh muscles. *J. Bone and Joint Surg.*, 73-A: 1025-1036, Aug. 1991.
24. **Solomonow, M.; Baratta, R.; and D'Ambrosia, R.:** The role of the hamstrings in the rehabilitation of the anterior cruciate ligament-deficient knee in athletes. *Sports Med.*, 7: 42-48, 1989.
25. **Solomonow, M.; Zhou, B. H.; and Baratta, R.:** Coactivation patterns of the knees antagonist muscles. In *Proceedings of the IEEE — Engineering Medicine and Biology*, 1987.
26. **Solomonow, M.; Baratta, R.; Zhou, B. H.; Shoji, H.; Bose, W.; Beck, C.; and D'Ambrosia, R.:** The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *Am. J. Sports Med.*, 15: 207-213, 1987.
27. **Stokes, M., and Young, A.:** The contribution of reflex inhibition to arthrogenous muscle weakness. *Clin. Sci.*, 67: 7-14, 1984.
28. **Tibone, J. E., and Antich, T. J.:** A biomechanical analysis of anterior cruciate ligament reconstruction with the patellar tendon. A two year followup. *Am. J. Sports Med.*, 16: 332-335, 1988.
29. **Yue, G., and Cole, K. J.:** Strength increases from the motor program: comparison of training with maximal voluntary and imagined muscle contractions. *J. Neurophysiol.*, 67: 1114-1123, 1992.
30. **Zimny, M. L., and Wink, C. S.:** Neuroreceptors in the tissues of the knee joint. *J. Electromyog. and Kinesiol.*, 1: 148-157, 1991.