

The Effect of Postsurgical Edema of the Knee Joint on Reflex Inhibition of the Quadriceps Femoris

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The purpose of this case study was to investigate reflex inhibition of the quadriceps femoris in a subject with postsurgical edema of the left knee. The subject was a 45-year-old male with a traumatic knee injury with resultant edema who underwent elective arthroscopic surgery. Reflex inhibition was assessed by H-reflex elicitation in the femoral nerve and surface electromyography of the quadriceps. To assess the degree of edema, direct circumferential measurements were taken. On the first presurgical visit, the left knee demonstrated mild edema with a decrease in H-reflex amplitudes. Two days after surgery, a further reduction in amplitudes and more swelling were demonstrated followed by an increase in amplitudes and a reduction in edema on the 28th postoperative day. These findings document a relationship between reflex inhibition and joint swelling that was previously described in experimental models where joint edema was simulated.

Reflex inhibition of the quadriceps femoris (quadriceps) has been demonstrated in the knee joints of patients in clinical populations (8) and in normal subjects in whom joint pathology has been simulated using experimental preparations (12, 19). Decrease in voluntary quadriceps function has been attributed to pain secondary to knee injury (14) or the trauma of surgical repair (15, 18); immobilization (14); and joint effusion secondary to ligamentous injury (11, 18), chronic arthritis (8), or simulations where the knee joint was infused with saline (19) or plasma (12).

The time period during which knee joint edema persists may influence the ability of quadriceps to recover from the inhibiting effects of disease, injury, or surgery. Kennedy et al. (12) and Spencer et al. (19) simulated acutely swollen knee joints and found that reflex inhibition of quadriceps was immediately reversible when the joints were aspirated. Others (8) have made similar observations in patients with chronic arthritis of the knee. In contrast, however, Newham et al. (15) stated there was no relationship between reflex inhibition and the degree of effu-

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sion in patients with chronically swollen knee joints and found no reduction in inhibition when the joints were aspirated.

Geborek et al. (8) studied 17 knee joints in 13 patients with chronic arthritis. The knee cavities were injected with sterile saline until intra-articular pressures measured 50 mm Hg. Surface electromyographic (EMG) recordings of vastus medialis and vastus lateralis revealed decreased activity levels with a concurrent reduction in extensor torque. H-reflexes, elicited by subthreshold electrical stimulation and recorded using EMG, have been shown to be reliable and valid measures of reflex activity in muscle (12, 17, 19, 21). Spencer et al. (19) injected up to 60 ml of 0.9% physiological saline solution into the knee joints of 10 healthy subjects. All subjects showed a significant decrease in H-reflex amplitudes of the quadriceps after injection of saline. Threshold inhibition in vastus medialis occurred when 20 to 30 ml of saline was injected, while rectus femoris and vastus lateralis achieved threshold at 50 to 60 ml.

Kennedy et al. (12) performed a similar experiment in the knee joints of 3 normal and 6 arthritic subjects using the H-reflex as the primary evaluative tool. Thirty to 50% reflex inhibition of quadriceps was noted after plasma was infused into the knee. Vastus medialis demonstrated the most dramatic reduction in reflex activity. In one subject, 60 ml of fluid was maintained in the knee cavity for 30 min, and reflex inhibition remained constant over this period. The authors concluded that joint capsule mechanoreceptors responded to capsular edema by evoking reflex inhibition of quadriceps.

Interestingly, while normal (12, 19), arthritic (8), and joint-injured subjects (11, 18) have been studied using aspiration (8, 12, 15, 19), dynamometry (18), and electrical stimulation (16), a review of the literature revealed few investigations that studied reflex inhibition secondary to edema in postsurgical knee patients using electrophysiological techniques. Postsurgical patients appear to be an obvious cohort of subjects who would demonstrate reflex inhibition. By coincidence, a subject known to one of the investigators was to undergo elective knee surgery and agreed to participate in this investigation. The purpose of this single-subject study was to document reflex inhibition in three muscles of the quadriceps secondary to postsurgical edema of the knee using surface EMG and elicitation of H-reflexes.

Methods

Subject

The subject was a 45-year-old male, known to one of the investigators, who was to undergo elective knee surgery and agreed to participate in this study. The subject's knee was injured approximately 9 months prior to this investigation; the subject's seasonal occupation precluded surgical treatment until 9 months after his injury. The left knee sustained a torn medial meniscus as confirmed by clinical examination and nuclear magnetic resonance imaging. The subject demonstrated intermittent symptoms including pain, locking and clicking, and occasional episodes of the knee "giving way" (buckling). The subject reported that compared with the right knee, the left knee had been moderately swollen since the injury occurred. Prior to testing the subject provided written informed consent.

Surgical Procedure

The subject received general anesthesia, and three arthroscopic punctures were performed. The knee was irrigated and examined. Multiple loose bodies were removed from the knee cavity and a subtotal meniscectomy was performed. There was also evidence of Grade III chondromalacia over the articular surface of the patella and cartilaginous erosion of the intercondylar notch and medial femoral condyle. A partial synovectomy was also performed secondary to a hypertrophic synovial capsule along the medial aspect of the knee. After surgery, the incision sites were closed with sutures and the knee was wrapped with gauze and a 6-in. foot-to-groin elastic compression bandage. When the patient's vital signs stabilized (approximately 2 hr according to the patient's outpatient discharge summary), he was discharged to his home. The subject was permitted to bear weight on the left lower extremity as tolerated and did not require an assistive device while walking. Acetaminophen was given to the subject to control pain immediately after surgery and was discontinued by the end of the first postoperative day.

Instrumentation and Procedures

Both of the subject's knees were tested 7 days prior to surgery to establish baseline conditions as well as 2 and 28 days postsurgically. The subject was positioned supine on a treatment table with his head supported by a small foam rubber wedge. His arms and forearms were comfortably supported by the table at his sides (1), and his head was maintained in a neutral position (20). His knee was positioned in 20–22° of flexion by an underlying foam-covered wedge during all three testing sessions. The knee was positioned and tested in slight flexion in anticipation of postsurgical edema that would prevent the knee from being completely extended due to capsular distension. Joint angles were confirmed before each trial using a plastic hand-held goniometer. Circumferential measurements (14) of the knee were taken at two points: directly over the midpoint of the patella, and at a point 2 cm proximal to the superior pole of the patella.

The vastus medialis, vastus lateralis, and rectus femoris muscles were initially identified for EMG electrode placement via palpation during submaximal isometric knee extensions. The skin overlying the three muscles was shaved and lightly abraded using emery cloth to reduce electrical impedance. Motor points for the three muscles were determined using a low-voltage stimulator. A stimulating probe was moved over the muscle until a twitch contraction was elicited. The site was assumed to be the motor point and the skin was marked. One set of self-adhering, pre-gelled bipolar electrodes, 1.8 cm in diameter, spaced 2.8 cm apart were placed over the belly of each muscle. One electrode was placed directly over the motor point and the second distal to that point. One reference electrode for each EMG channel was placed over the crest of the tibia. The use of bipolar electrodes with a small interelectrode spacing (2.8 cm) confined the pick-up area of the electrodes to 2.8 cm (13) and thus minimized potential cross-talk. Interelectrode impedance was measured with an ohmmeter and maintained below 2,000 Ω .

The femoral nerve was initially located by palpating the femoral arterial pulse in the femoral triangle. An electrical stimulator probe was used to confirm the location of the femoral nerve lateral to the femoral artery at an intensity high enough to elicit a minimal twitch contraction of the quadriceps. After identification of the femoral nerve and skin preparation over the site, a self-adhering, pre-

gelled stimulating electrode (cathode) 1.8 cm in diameter was affixed to the skin overlying the nerve. The anode was placed 2.8 cm distal (center-to-center) to that point. A small pad of foam rubber (5 × 8 × 12 cm) was placed over the stimulating electrodes and held in place with a 4-in. elastic compression bandage to ensure adequate contact between the electrodes and skin (4). Stimulating and recording electrode sites were marked with a permanent marker on the left side to ensure accurate electrode placement on subsequent test days (4). The subject was given a marker to “touch-up” the electrode sites as required for the duration of the experiment.

A Grass (Grass Instrument Co., Quincy, MA) square pulse electrical stimulator (Model S48) with a stimulus isolation unit (Model SIU5) was used to elicit H-reflexes. The stimulation rate was set at 0.30 pulses per second with a duration of 0.50 ms (9, p. 332). Stimulus intensity was high enough to elicit an M-wave and accompanying H-reflexes (17). M- and H-reflex waveforms were confirmed by visual inspection on an oscilloscope connected in parallel with the recording computer. A typical M-wave and H-reflex elicited from one of the investigators during pretrial testing is depicted in Figure 1. When the waveforms were confirmed, the stimulus intensity was decreased in 10-V increments until the M-wave decreased in amplitude while the H-reflex remained constant, thus confirming the presence of the H-reflex (7; 9, p. 332). The resulting stimulus intensities, 40 and 80 V for the right and left limbs, respectively, were held constant during all three visits to the laboratory. These stimulus intensities were adequate to elicit twitch contractions of the quadriceps without causing pain.

The femoral nerve was stimulated 10 times in each extremity with 10 s between stimulations (1, 2). The resulting EMG activity was stored on computer disks for subsequent analysis. Data were collected and analyzed using programs written with LabVIEW® (National Instruments, Austin, TX) programming language. EMG signals were preamplified with a Grass Model 7P3 preamplifier and then amplified downstream on a Grass Model 7DA driver amplifier (band pass = 10–500 Hz with a 60-Hz band stop filter) and fed to an analog-to-digital converter (Lab PC+, National Instruments, Austin, TX) at a sampling rate of 2000 Hz per channel. H-reflex amplitudes were measured from the baseline to peak upward (positive) deflections (1, 10) using a LabVIEW® program and recorded on a spreadsheet for subsequent analysis.

Data Analysis

Descriptive statistics (means and standard deviations) and percentage difference of EMG amplitudes were calculated.

Results

Data and descriptive statistics are presented in Table 1. M-waves and H-reflexes were consistently evoked in all three muscles of the quadriceps studied in both knees. Prior to recording, H-reflexes were confirmed by reducing the stimulus intensity to the femoral nerve causing a reduction in M-wave amplitudes while H-reflex amplitudes remained constant (7, 9). The overall shape of the H-reflex was polyphasic (19) with an initial upward deflection followed by a downward then final upward deflection until the signal returned to the baseline (Figure 2). In the left (involved) quadriceps, all initial H-reflex amplitudes remained negative (downward) with respect to the baseline (Table 1). In the right quadriceps, the vastus

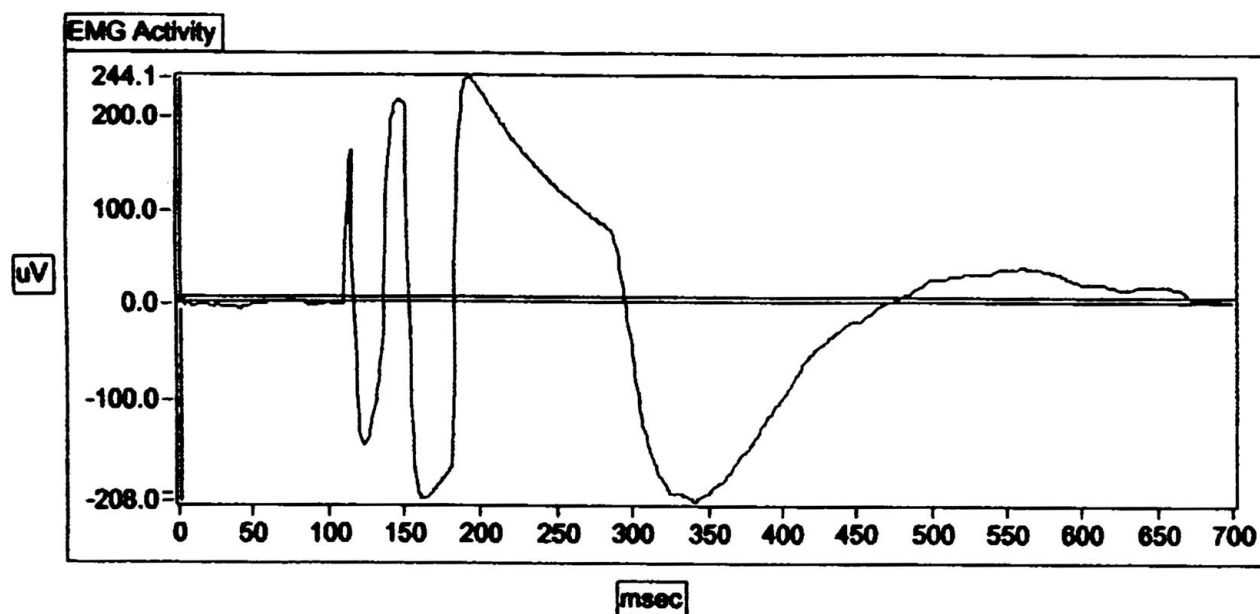


Figure 1 — M-wave (second upward deflection) and H-reflex (begins at third upward deflection). First upward deflection is stimulus artifact.

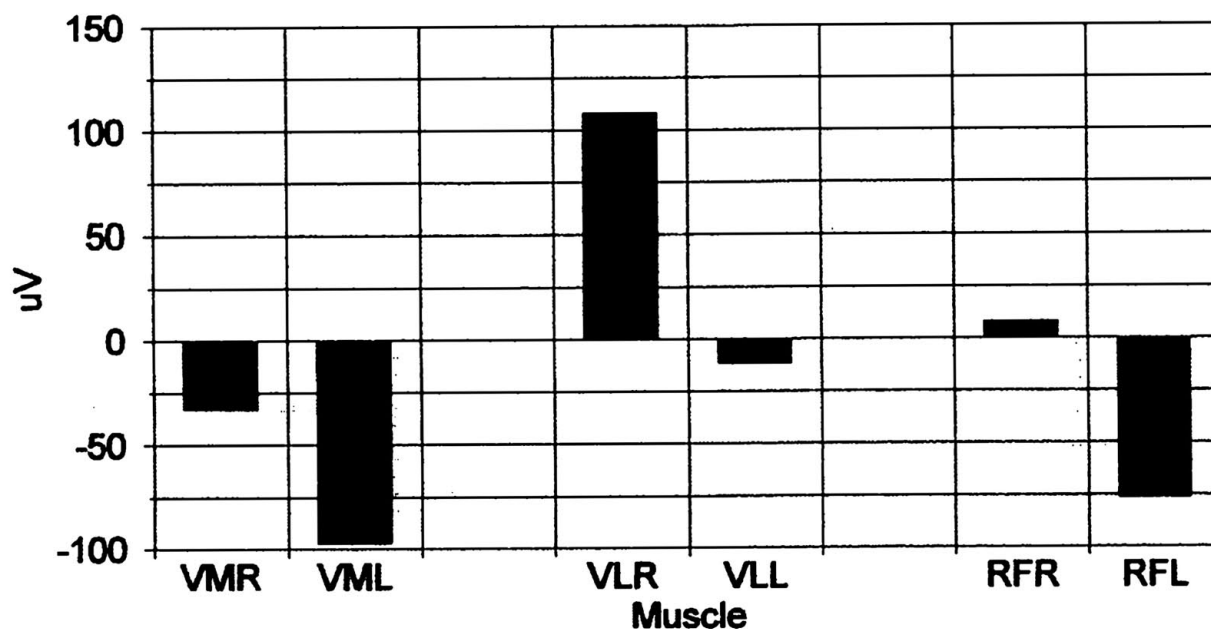


Figure 2 — Mean EMG amplitudes for H-reflexes during the preoperative visit for the left (involved) and right vastus medialis (VML and VMR), vastus lateralis (VLL and VLR), and rectus femoris (RFL and RFR).

lateralis and rectus femoris demonstrated positive amplitudes above the baseline while the vastus medialis amplitude was negative.

The mean EMG amplitude for H-reflexes across all muscles studied in the left (involved) knee was $-65.7 \mu\text{V}$ on the second postoperative day (POD 2) compared with a preoperative (Pre-Op) mean value of $-61.8 \mu\text{V}$. This was a 10% decrease in mean amplitude between Pre-Op and POD 2 (Figure 3a). Vastus medialis and rectus femoris demonstrated absolute decreases in mean amplitudes of $-108.0 \mu\text{V}$ and $-87.3 \mu\text{V}$, respectively, compared with preoperative values of $-97.3 \mu\text{V}$ and $-76.4 \mu\text{V}$. Vastus lateralis showed an absolute increase in mean amplitude from $-11.7 \mu\text{V}$ preoperatively to $-1.9 \mu\text{V}$ on POD 2. Circumferential measurements of the left knee increased on POD 2 to 40.0 cm compared with 38.5 cm Pre-OP (Figure 3b).

Table 1 Descriptive Data for EMG Amplitudes of H-reflexes (in μV) for the Left (Involved) and Right Knees During the Three Test Sessions

No.	Pre-Op						POD 2			POD 28		
	Right			Left			Left			Left		
	VM	VL	RF	VM	VL	RF	VM	VL	RF	VM	VL	RF
1	-34.0	118.00	9.0	-98.0	-12.0	-76.0	-110.0	-1.0	-87.0	-34.0	-32.0	-73.0
2	-30.0	112.00	8.0	-96.0	-11.0	-75.0	-110.0	0.0	-91.0	-35.0	-32.0	-72.0
3	-33.0	124.00	1.0	-98.0	-12.0	-78.0	-108.0	-3.0	-91.0	-38.0	-29.0	-72.0
4	-32.0	133.00	7.0	-97.0	-13.0	-76.0	-110.0	-3.0	-83.0	-33.0	-33.0	-73.0
5	-31.0	115.00	8.0	-98.0	-11.0	-75.0	-104.0	-1.0	-85.0	-35.0	-35.0	-74.0
6	-31.0	110.00	7.0	-98.0	-11.0	-75.0	-110.0	-1.0	-93.0	-30.0	-29.0	-74.0
7	-35.0	78.00	7.0	-98.0	-11.0	-76.0	-109.0	-3.0	-83.0	-35.0	-28.0	-73.0
8	-34.0	94.00	8.0	-98.0	-11.0	-78.0	-107.0	-3.0	-87.0	-31.0	-30.0	-75.0
9	-33.0	94.00	8.0	-97.0	-12.0	-79.0	-107.0		-90.0	-33.0	-34.0	-77.0
10	-35.0	101.00	9.0	-99.0	-13.0	-76.0	-109.0		-83.0	-34.0	-30.0	-73.0
Mean	-33.0	108.0	8.0	-97.3	-11.7	-76.4	-108.4	-1.9	-87.3	-33.8	-31.2	-73.6
SD	1.8	16.3	2.3	0.8	0.8	1.4	2.0	1.2	3.8	2.0	2.0	2.0

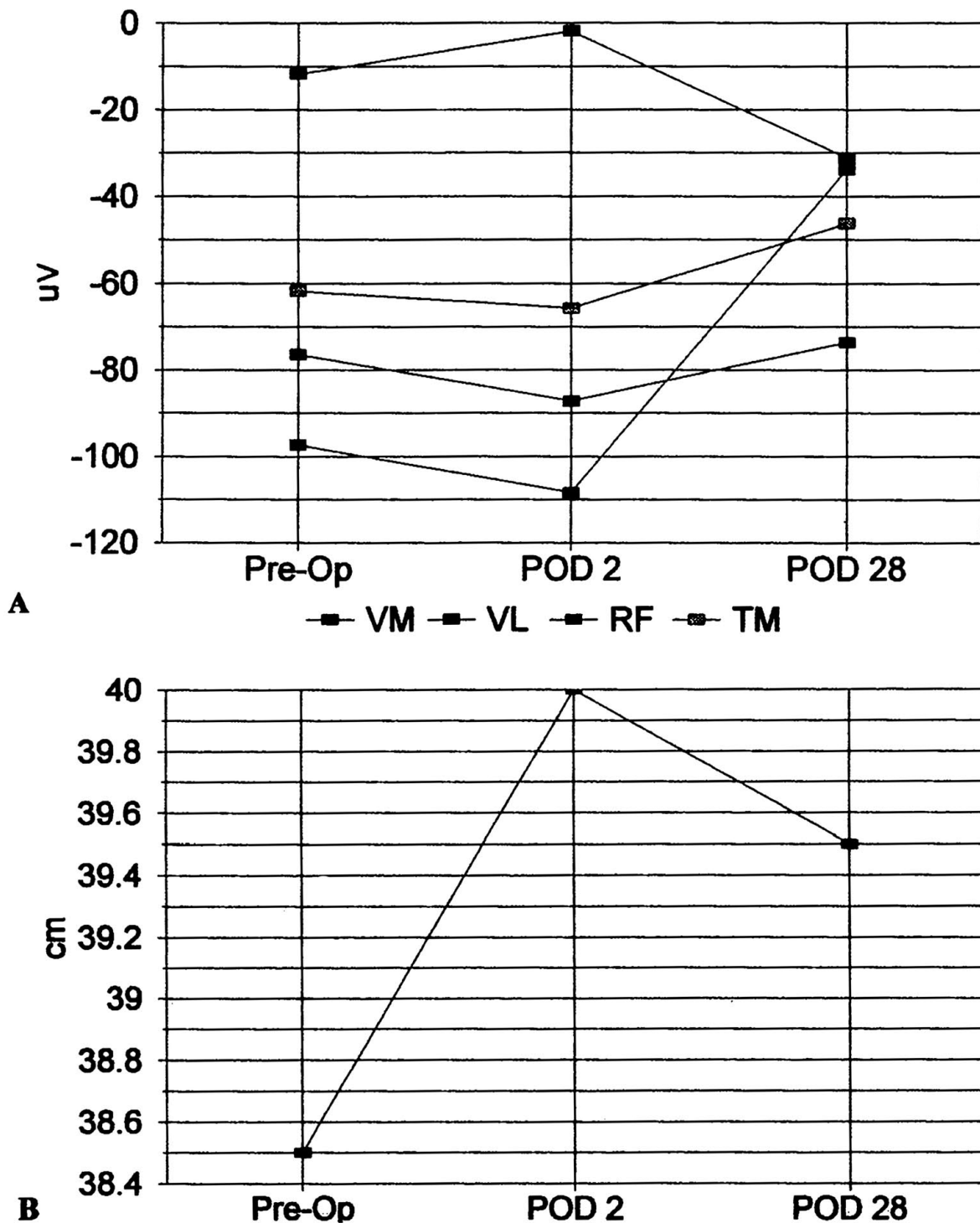


Figure 3 — (a) Mean EMG amplitudes of H-reflexes for the left (involved) quadriceps during the three test sessions. TM refers to the total mean amplitude value on each test session collapsed across muscle. (b) Circumferential measurements of the left (involved) knee during the three test sessions. Note scale.

On the 28th postoperative day (POD 28), mean EMG amplitudes for H-reflexes across all muscles increased 30% compared with POD 2 (Figure 3a). Vastus medialis and rectus femoris absolute mean amplitudes increased to $-33.8 \mu\text{V}$ and $-73.6 \mu\text{V}$, respectively, compared with POD 2 values, while the mean amplitude for vastus lateralis decreased to $-31.2 \mu\text{V}$. An increase in total mean H-reflex amplitudes for the three muscles on POD 28 was accompanied by a decrease in circumference of the left knee to 39.5 cm (Figure 3b).

The right (uninvolved) knee, which was also studied on the preoperative visit to establish baseline conditions, demonstrated a mean H-reflex amplitude

across all muscles of 27.7 μV (Table 1). The vastus medialis mean absolute amplitude was $-33.0 \mu\text{V}$, while amplitudes for rectus femoris and vastus lateralis were 8.0 μV and 108.0 μV , respectively (Figure 2). Comparative values for the left knee are described above. The preoperative circumference of the right knee was 38.0 cm compared with 38.5 cm for the left.

Discussion

Previous investigations by Kennedy et al. (12) and Spencer et al. (19) in human experimental models, where the knee joints of normal subjects were infused with sterile saline to simulate joint edema, showed evidence of quadriceps alpha motoneuron reflex inhibition as joint capsules were distended. H-reflex amplitudes were used to monitor motoneuron excitability levels. That is, increases in amplitudes were indicative of increased efferent discharge and vice versa. deAndrade, Grant, and Dixon (3) reported decreased voluntary activation of the quadriceps secondary to joint distension in both experimental preparations as well as in subjects with chronically distended knees. Joint capsular mechanoreceptors of the Ruffini type (18), which respond to changes in joint capsule pressure (6, 19) and deformation (17), appear to be the principal mediator of this response and were not confounded by pain (19). Snyder-Mackler et al. (18) indicated that patients with subacute or chronic rupture of the anterior cruciate ligament that was not repaired by surgery demonstrated joint laxity that led to joint deformation. The authors speculated that mechanoreceptor discharge brought on by deforming forces triggered reflex inhibition of the quadriceps. Patients who underwent ligament repair before becoming chronic (time frame not stated) had essentially the same force production in quadriceps when the involved and uninvolved sides were tested and compared.

In the present study, a 45-year-old male subject underwent elective arthroscopic surgery approximately 9 months after sustaining an injury to the medial side of his left knee. On the first preoperative visit to our laboratory, the subject presented with mild edema in the left knee compared with the right knee, which increased 2 days after surgery and subsided by the 28th postoperative day (Figure 3b). Findings from the present study were in general agreement with investigations by Kennedy et al. (12) and Spencer et al. (19) and demonstrated decreased H-reflex amplitudes in the left quadriceps on POD 2 that increased by POD 28. Mechanoreceptors within the joint capsule (6, 12) and in other tissues (18) respond to deformation and pressure changes due to distension (3), triggering afferent signals that reflexively inhibit the quadriceps. Findings at the time of surgery did not suggest changes in the integrity of supporting ligaments; however, the torn medial meniscus, loose bodies, and erosion of articular cartilage probably accounted for the postinjury edema experienced until the time of surgery. Thus, joint distension appears to explain the observed quadriceps inhibition. This is further supported by the fact that inhibition decreased when swelling decreased after surgery.

Morrissey (14) stated that in addition to joint effusion and knee immobilization, pain is one of the most frequently cited causes of thigh muscle reflex inhibition. Pain did not appear to be a contributing factor to inhibition in this subject. In fact, the subject was remarkably pain-free 24 hr after surgery. Immediate postoperative knee pain was characterized by the subject in the recovery room as moderate and was controlled by oral acetaminophen, which was discontinued at the end of the first postoperative day. During testing on POD 2 and POD 28, the subject

did not complain of joint pain and had not taken any pain medication on either day. Stimulus intensities during H-reflex elicitation were purposely kept to levels (40–80 V) that were not painful, thus eliminating pain as a source of inhibition and supporting our contention that joint edema played a major role in decreasing quadriceps activation.

Interestingly, mean H-reflex amplitudes for the left knee muscles ($-46.2 \mu\text{V}$) on POD 28 fell short of mean values for the right ($27.7 \mu\text{V}$), suggesting that the left alpha motoneuron pool for the femoral nerve continued to experience an inhibitory response by joint capsule receptors (7). It is assumed that as recovery progresses, amplitudes in both quadriceps will become symmetrical.

Newham, Hurley, and Jones (15) suggested that reflex inhibition of quadriceps, as well as the potential for improvement, differs in patients with acute versus chronic knee effusion. While inhibition may resolve or at least improve in patients with acute effusion, there was no relationship between the degree of effusion and reflex inhibition in 10 patients studied and characterized as chronic. Aspiration of the knee joints did not decrease the degree of reflex inhibition. Conversely, Geborek, Moritz, and Wolheim (8) and others (5) did describe a reversal of reflex inhibition in arthritic knee patients who presumably would be characterized as having chronic disease. In the present study, the subject sustained his knee injury 9 months prior to having surgery. During the presurgical period, working often exacerbated his symptoms of pain, swelling, and clicking. It is not clear whether this subject's knee condition should be characterized as chronic. But if one accepts that the subject had a chronic injury, the increase in mean H-reflex amplitudes for the left vastus medialis and rectus femoris, coupled with a decrease in knee joint edema by POD 28, suggests improvement in quadriceps activation and disagrees with the findings of Newham, Hurley, and Jones (15).

Mean H-reflex amplitudes for vastus medialis and rectus femoris were remarkably consistent, and both muscles demonstrated a decrease 2 days after surgery followed by increases in mean amplitudes 28 days later (Figure 3). Mean H-reflex amplitudes for vastus lateralis did not respond in the same manner, demonstrating a slight increase on POD 2 and decreasing by POD 28. It should be noted that during the subject's second visit, the EMG recording channel for vastus lateralis was exceptionally "noisy," the source of which could not be identified or corrected. It is possible that the recorded amplitudes for vastus lateralis may have included significant artifact, thus obscuring the true signal. Based on the consistent responses of the other two muscles it is reasonable to speculate that vastus lateralis may respond in a similar way to vastus medialis and rectus femoris.

Kennedy et al. (12, p. 334) reported that "of all parts of the quadriceps studied, most dramatic inhibition was consistently observed in the vastus medialis." In our study, vastus medialis also demonstrated the greatest decrease in mean amplitudes on POD 2 followed by a large increase on POD 28 (Figure 3a), supporting Kennedy et al.'s observation.

While previous studies have employed human models in which joint edema has been simulated, the current study assessed a subject with actual postsurgical edema that approximated the results of others (12, 19). It is unclear whether differences in reflex inhibition exist between acutely versus chronically injured or diseased joints. Clinically, early and aggressive treatment of knee joint edema (14) appears to be warranted to prevent neurogenic atrophy of the quadriceps. In light of the present findings it seems reasonable to expect that full force production of all parts of the quadriceps will not be realized until edema is controlled.

References

1. Bulbulian, R., and D.K. Bowles. Effects of downhill running on motoneuron pool excitability. *J. Appl. Physiol.* 73(3):968-973, 1992.
2. Crone, C., and J. Nielsen. Methodological implications of the post activation depression of the soleus H-reflex in man. *Exp. Brain Res.* 78(1):28-35, 1989.
3. deAndrade, J.R., C. Grant, and A.St.J. Dixon. Joint distension and reflex inhibition in the knee. *J. Bone Joint Surg.* 47-A:313-322, 1965.
4. Enoka, R.M., R.S. Hutton, and E. Eldred. Changes in excitability of tendon tap and Hoffman reflexes following voluntary contractions. *Electroenceph. Clin. Neurophysiol.* 48:664-672, 1980.
5. Fahrner, H., H.U. Rentsch, N.J. Gerber, C. Beyeler, C.W. Hess, and B. Grünig. Knee effusion and reflex inhibition of the quadriceps. A bar to effective training. *J. Bone Joint Surg.* 70-B:635-638, 1988.
6. Freeman, M.A., and B. Wyke. The innervation of the knee joint. An anatomical and histological study in the cat. *J. Anat.* 101:505-532, 1967.
7. Garland, S., L. Gerilovsky, and R.M. Enoka. Association between muscle architecture and quadriceps femoris H-reflex. *Muscle Nerve* 17:581-592, 1994.
8. Geborek, P., U. Moritz, and F.A. Wolheim. Joint capsular stiffness in knee arthritis. Relationship to intraarticular volume, hydrostatic pressures, and extensor muscle function. *J. Rheum.* 16:1351-1358, 1989.
9. Hecox, B., T.A. Mehreteab, and J. Weisberg. *Physical Agents: A Comprehensive Text for Physical Therapists.* Norwalk, CT: Appleton & Lange, 1993.
10. Hugon, M. Methodology of the Hoffman reflex in man. In *New Developments in Electromyography and Clinical Neurophysiology*, J.E. Desmedt (Ed.). Basel, Switzerland: Karger, 1973, pp. 277-293.
11. Hurley, M.V., D.W. Jones, D. Wilson, and D.J. Newham. Rehabilitation of quadriceps inhibited due to isolated rupture of the anterior cruciate ligament. *J. Orthop. Rheumatol.* 5:145-154, 1992.
12. Kennedy, J.C., I.J. Alexander, and K.C. Hayes. Nerve supply of the human knee and its functional importance. *Am. J. Sports Med.* 10(6):329-335, 1982.
13. Lynn, P.A., N.D. Bettles, A.D. Hughes, and S.W. Johnson. Influences of electrode geometry on bipolar recordings of the surface electromyogram. *Med. Biol. Eng. Comput.* 16:651-660, 1978.
14. Morrissey, M.C. Reflex inhibition of thigh muscles in knee injury. Causes and treatment. *Sports Med.* 7:263-276, 1989.
15. Newham, D.J., M.V. Hurley, and D.W. Jones. Ligamentous knee injuries and muscle inhibition. *J. Orthop. Rheumatol.* 2:163-173, 1989.
16. Newham, D.J., T. McCarthy, and J. Turner. Voluntary activation of human quadriceps during and after isokinetic exercise. *J. Appl. Physiol.* 71:2122-2126, 1991.
17. Schieppati, M. The Hoffman reflex: A means of assessing spinal reflex excitability and its descending control in man. *Prog. Neurobiol.* 28:345-376, 1987.
18. Snyder-Mackler, L., P.F. DeLuca, P.R. Williams, M.E. Eastlack, and A. R. Bartolozzi. Reflex inhibition of the quadriceps femoris muscle after injury or reconstruction of the anterior cruciate ligament. *J. Bone Joint Surg.* 76-A:555-560, 1994.
19. Spencer, J.D., K.C. Hayes, and I.J. Alexander. Knee joint effusion and quadriceps reflex inhibition in man. *Arch. Phys. Med. Rehabil.* 65:171-177, 1984.
20. Traccis, S., G. Rosati, S. Patraskakis, M. Bissakou, G.F. Sau, and I. Aiello. Influences of neck receptors on soleus motoneuron excitability in man. *Exp. Neurol.* 95(1):76-84, 1987.

21. Urbscheit, N., and B. Bishop. Effects of cooling on the ankle jerk and H-response. *Phys. Ther.* 50:1041-1049, 1970.

Acknowledgment

We would like to acknowledge Arthur J. Nelson, PhD, PT, for his assistance in interpreting our data.