Journal of Orthopaedic & Sports Physical Therapy
Official Publication of the Orthopaedic and Sports Physical Therapy Sections of the American Physical Therapy Association

The Use of Neuromuscular Electrical Stimulation to Improve Activation Deficits in a Patient With Chronic Quadriceps Strength Impairments Following Total Knee Arthroplasty

Stephanie Petterson, MPT, PhD[1]
Lynn Snyder-Mackler, PT, SCS, ATC, ScD, FAPTA[1]

Study Design: Case report.
Background: Long-term deficits in quadriceps femoris muscle strength and impaired muscle activation are common among individuals with total knee arthroplasty (TKA). Failure to address strength-related impairments results in poor surgical and functional outcomes, which may accelerate the progression of osteoarthritis in other lower extremity joints. The purpose of the current case report was to implement a neuromuscular electrical stimulation (NMES) treatment protocol in conjunction with an intense weight-training program, with the aim of reversing persistent quadriceps muscle impairments after TKA.
Case Description: The patient was a 62-year-old male cyclist 12 months following simultaneous, bilateral TKA with impairments in left quadriceps strength and volitional muscle activation. His left quadriceps strength was 26% weaker than his right and central activation ratio (CAR) of his left quadriceps was 13% lower than his right quadriceps CAR. NMES to the left quadriceps was implemented for 6 weeks, in addition to an intense volitional weight-training program with emphasis on unilateral lower extremity exercises.
Outcomes: The patient demonstrated a 25% improvement in left quadriceps femoris maximal volitional force output following 16 treatments of combined NMES and volitional strength training over a 6-week period. The patient's volitional muscle activation improved from a CAR of 0.83 before treatment to 0.97 after treatment. At discharge from physical therapy and at his 18-month postoperative follow-up, the patient's left quadriceps strength was only 4% lower than his right quadriceps strength. At the 24-month follow-up, the patient's left quadriceps strength was 6% stronger than his right quadriceps strength.
Discussion: The patient was able to achieve symmetrical quadriceps strength and complete muscle activation following 6 weeks of NMES and volitional strength training. An intense strengthening program may have the potential to reverse persistent strength-related impairments following TKA. *J Orthop Sports Phys Ther 2006;36(9):678-685.* doi:10.2519/jospt.2006.2305

Key Words: *joint replacement, muscle strength, rehabilitation*

Osteoarthritis (OA) and its physical manifestations are a global issue. In the cohort of individuals older than 60 years of age, an estimated 10% of the world's population suffers from limitations attributable to OA.[38] OA-related impairments are responsible for over 18 million physician visits each year.[3]

Knee OA is characterized by degenerative radiographic findings, including joint space narrowing, the presence of osteophytes, and subchondral bone sclerosis.[1,6] Such findings can contribute to pain with weight-bearing activities, restricted joint range of motion (ROM), and functional decline. Conservative measures (ie, corticosteroids, hyaluronic acid, aquatic therapy) are often implemented to alleviate pain and minimize functional limitations; however, when conservative management fails to alleviate OA-related symptoms, total knee arthroplasty (TKA) is often indicated.

TKA is one of the most common and successful orthopedic surger-

[1] *Department of Physical Therapy, University of Delaware, Newark, DE.*
Funding provided by the National Institutes of Health (R01HD041055-01 and T32 HD07490). The methods described in this manuscript were approved by the Human Subjects Review Board of the University of Delaware.
Address correspondence to Lynn Snyder-Mackler, Department of Physical Therapy, University of Delaware, 301 McKinly Laboratory, Newark, DE 19716. E-mail: smack@udel.edu

ies in the treatment of end-stage knee OA. Over 400 000 TKA surgeries were performed in the United States in 2003.[2] Incidence trends are predicted to steadily rise over the next few decades with the growth of the older adult population.[2] The most commonly used surgical approach is the medial parapatellar approach. This surgical approach involves an incision through the extensor mechanism for visualization and access to the knee joint. The disease-affected joint surfaces are then replaced with prosthetic components designed to mimic normal knee ROM.

Dramatic reductions in knee pain[7,10,22] and increased functional performance[22,39] enhance quality of life after TKA.[10] Acute impairments in pain, swelling, joint ROM, and patellar mobility are common following TKA. In addition, patients exhibit a 60% reduction in quadriceps femoris muscle force production from preoperative baseline levels 1 month after TKA.[37] While improvement of these impairments appears likely, residual deficits, primarily in quadriceps muscle function, prevent functional recovery to the levels of healthy cohorts.[4,39]

Typical physical therapy management following TKA focuses on performance of activities of daily living and ROM exercises, which begin during the acute care, inpatient hospital stay. These exercises are continued following discharge from the acute care hospital in either a rehabilitation hospital or home physical therapy setting. More strenuous strengthening exercises are often not initiated until the commencement of outpatient physical therapy; however, outpatient physical therapy is not always the standard of care. While some advocate for more substantial strengthening on an outpatient basis,[4,19,22,36,37] others advocate for home physical therapy only,[14,28] or argue that rehabilitation is not necessary following TKA.[27,30]

Regardless, discharge from formal physical therapy typically occurs when ROM and pain impairments have been resolved and function has improved. Strength benchmarks are rarely used for discharge guidelines and in determining outcomes after joint replacement. However, follow-up studies indicate that, despite improved self-reports of function[11,13,22] and improved functional performance,[13,22,24] quadriceps weakness is persistent in the short and long term.[4,21,37] Weakness is present 6 months postoperatively, as evidenced by reduced quadriceps strength in the operated knee that received surgery, compared to the knee that did not receive surgery, among patients following unilateral TKA.[16] These deficits are as high as 39% compared to healthy control subjects 1 year postoperatively[39] and at least 30% 2 years following TKA.[32] Longer-term follow-up studies demonstrate persistent quadriceps weakness[4] and self-reported functional decline 3 years postoperatively.[31] In light of current physical therapy practice and compounded by the nature of the surgical procedure, it is not surprising that patients often exhibit short- and long-term impairments in quadriceps femoris function.

Recent evidence suggests strength impairments correspond to asymmetries in gait and completion of functional tasks such as sit-to-stand transfer. Specifically, interlimb differences in strength are associated with interlimb differences in knee excursion during gait and decreased vertical ground reaction force, peak knee extension moment, and EMG activity during a sit-to-stand transfer.[20] The noted asymmetries result in increased joint loads in the contralateral hip and knee, which may lead to the emergence or progression of symptomatic OA in other joints. Failure to address and reverse strength impairments may result in a greater likelihood of multiple total joint and revision arthroplasties. In fact, the next most likely joint to be replaced following TKA is the contralateral knee, followed by the contralateral hip and then the ipsilateral hip.[31] The risk of contralateral TKA is 37% within 10 years of the first operation.[17]

The existing body of literature focuses on the acute and immediate subacute management of patients with TKA.[14,22,27,28] Volitional exercise and neuromuscular electrical stimulation (NMES) are 2 effective modes of strengthening following knee injury or surgery. According to published reports, strengthening protocols require the performance of an intense exercise program at a minimum intensity of 60% of an individual's 1-repetition maximum, 3 times per week, over a minimum period of 6 to 8 weeks.[29] Greater intensities of up to 80% of 1 repetition maximum are necessary to elicit strength improvements in previously trained individuals.[29]

NMES has also been shown to be effective as an adjunct modality in the treatment of quadriceps weakness and activation impairments following knee surgery.[15,33,36] NMES uses electrical current to induce muscular contraction by preferentially activating the greater force-producing, type II muscle fibers. NMES treatment intensity is defined as the electrically elicited contraction force reported as a percentage of maximum volitional quadriceps strength. Treatment intensities of 30% to 50% of a patient's maximum volitional quadriceps strength have been shown to result in substantial gains in quadriceps strength within 4 to 6 weeks.[33,36]

No current evidence supports the physical therapy management of chronic quadriceps strength impairments following TKA. The current case report, which focuses on the physical therapy management of a patient 12 months post-TKA, demonstrated that chronic strength impairments can be successfully reversed with physical therapy interventions including aggressive strengthening and NMES.

CASE DESCRIPTION

Patient Characteristics

The patient was a 62-year-old male (height, 1.96 m; mass, 88.4 kg; body mass index, 23.1 kg/m^2), who came to our physical therapy clinic preoperatively as part of a clinical trial of rehabilitation following bilateral, primary TKA for knee OA. The patient was an avid cyclist, cycling an average of 1667 km per month prior to surgery. A battery of tests, including a burst superimposition test to measure quadriceps strength and activation, the timed up and go (TUG),[25] a stair-climbing test (SCT),[18] the 6-minute walk test (6MW),[12,26] and self-report questions were administered preoperatively for inclusion into the clinical trial. His right quadriceps maximum volitional isometric contraction force (MVIC) was 997 N with 92% activation, while the left knee strength was 793 N with 85% activation. His TUG and SCT scores were 5.1 and 7.5 seconds, respectively, and his 6MW distance was 915 m (Table 1). Scores on the functional tests were considerably better even than age-matched healthy subjects; however, the patient had considerable pain, substantial quadriceps weakness, and quadriceps activation deficits bilaterally (left greater than right).

Following surgery, the patient could not participate in the clinical trial. His surgeon did not permit quadriceps strengthening exercise to begin until 3 months after surgery. This timeframe well exceeded entry into our clinical trial postoperatively. The clinical trial involves 6 weeks of resistive quadriceps exercise beginning 3 to 4 weeks postoperatively. Only ROM exercises, modalities for pain and edema control, and active exercise were permitted by the surgeon. Consequently, the patient was referred to another physical therapy facility considerably closer to his home, where he received 31 visits of outpatient physical therapy over a 10½-week period before being discharged to a home exercise program. His outpatient physical therapy program began 2½ weeks postoperatively and initially focused on knee flexion and extension ROM, quadriceps setting exercises, terminal knee extension Thera-band exercises, and lower extremity proprioceptive neuromuscular facilitation (PNF) patterns. Weight-bearing lower extremity strengthening exercises, including, stability ball hamstring curls, calf raises, stool walking, and Stairmaster, were initiated around postoperative week 7, and ball squats and step-ups were initiated around postoperative week 9. Per physician orders, at no time during his outpatient physical therapy were exercises implemented to specifically overload and strengthen the quadriceps muscle group.

The patient returned for testing 9 and 12 months after surgery to receive feedback on his recovery following surgery (Table 1). Nine months postoperatively, the patient reported that he was able to lift increasingly heavier weights with his hamstrings but he was having difficulty progressing in a similar fashion with his quadriceps strengthening program. At the 9-month follow-up, his gym program consisted of double-leg knee extensions, calf raises, leg press, and bilateral hamstring curls. His gym program was modified at this time to include single-knee extension exercises.

At the 12-month period, the patient was not satisfied with his progress. His training program consisted of biking and a gym-based weight-training program similar to his modified program at the 9-month follow-up. He felt his cycling was most limited by muscle weakness and fatigue, primarily with hill and sprint interval programs. The patient reported that he was unable to exercise to the point of feeling a ''deep, achy fatigue'' in his quadriceps with resistance training. His goal was to return to competitive cycling.

Evaluation

A clinical picture of the patient 12 months postsurgery indicated that he performed at a high functional level, but had significant quadriceps strength and activation impairments (left quadriceps more impaired than the right quadriceps). The

TABLE 1. Strength, activation, and functional performance progression and referenced normative data for healthy adults.

	Preoperative	9 mo	12 mo	18 mo	24 mo	Healthy Norms, Adults Aged >60 y
TUG (s)	5.1	4.7	4.4	4.6	4.3	8.4 ± 1.7[36]; 8.5[30]; 10.9[27]
SCT (s)	7.5	7.1	7.0	7.5	7.4	*
6MW (m)	915	*	944	1022	1059	570 ± 23[14]; 448[27]
MVIC left (N)	793	508	624	913	910	574.4 ± 156[42]
MVIC right (N)	997	637	847	947	853	574.4 ± 156[42]
CAR left	0.85	0.73	0.83	0.97	1.0	0.96 ± 0.02[42]
CAR right	0.92	0.76	0.96	0.98	1.0	0.96 ± 0.02[42]

Abbreviations: 6MW, 6-minute walk; CAR, central activation ratio; MVIC, maximal volitional isometric contraction; SCT, stair-climbing test; TUG, timed up and go.
* Data are not available.

patient performed the TUG and SCT approximately a half a second faster than before surgery and walked 30 m farther on the 6MW. These functional performance times and distances well exceed values from individuals of similar sex and age who do not have OA (Table 1). The results of functional testing gave the impression that the patient's TKA had been a complete success. However, the patient's self-reports of function on the Activity of Daily Living Scale of the Knee Outcomes Survey (KOS/ADLS)[9] and a global knee rating score (GRS) were inconsistent with his functional performance. While his questionnaire scores did improve from preoperative levels, the patient only scored a 77% on the KOS/ADLS (a 0-to-100 scale with 0 representing complete impairment and 100 representing normal knee function). In addition, he rated his knee to be 90% of its full potential during activities of daily living on the GRS. These self-report scores mirrored the patient's dissatisfaction of his physical abilities after surgery.

Strength testing revealed less favorable outcomes at the 12-month testing period (Table 1). A burst superimposition test was used to measure the patient's maximal volitional force output (MVIC) and muscle activation level of the quadriceps femoris muscle group.[34] Results from the burst superimposition test include the patient's MVIC as well as the patient's central activation ratio (CAR). CAR is calculated by dividing the MVIC by the MVIC plus the augmented force from the burst (Figure 1). CAR is reported on a continuum from 0 to 1.0, where 1.0 indicates complete muscle activation.

Twelve months postoperatively, the patient's right knee extension force was 150 N lower than its preoperative baseline measure. Knee extension strength of his left quadriceps was 169 N lower than his preoperative strength and 26% weaker than his right quadriceps muscle. Volitional muscle activation of his right quadriceps muscle was 0.96 out of a possible 1.0, comparable to similar aged individuals without knee OA (mean CAR, 0.96).[35] However, the left quadriceps muscle had substantial activation deficits exhibiting a CAR of 0.83. The burst superimposition test results indicated that the reduced muscle activation levels were contributing to his left quadriceps weakness (Figure 2).

Although magnetic resonance images of the patient's thigh musculature to assess muscle cross-sectional area were not acquired, girth measurements were taken at midthigh (half the distance between the greater trochanter and the superior border of the patella) and 2 cm above the superior border of the patella. No side-to-side difference in girth was measured in the patella measurement. However, the left thigh was 1.6 cm smaller at the midthigh measurement, a potential indication of muscle atrophy of the left quadriceps femoris muscles.

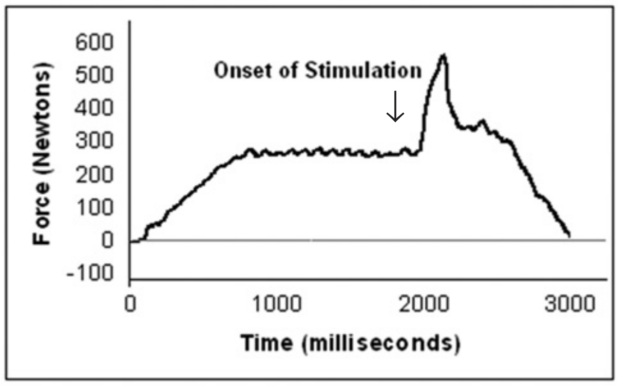

FIGURE 1. An example of a quadriceps force trace obtained from a burst superimposition test. The superimposed burst of electrical stimulus was applied at approximately 2000 milliseconds, denoted by the black arrow. Central activation ratio (CAR) is calculated by dividing the maximal volitional force (MVIC) just prior to the black arrow by the augmented force output after the arrow. In this example, MVIC was 269 N and the augmented force was 565 N, representing a CAR of 0.48.

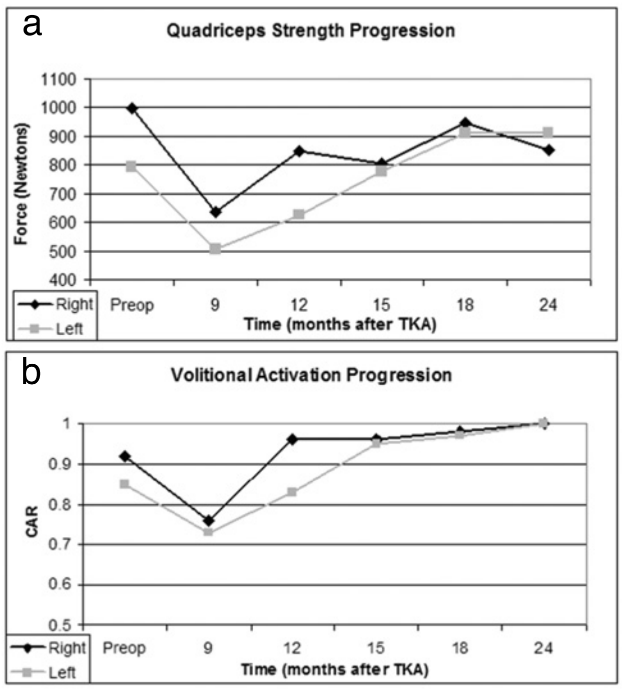

FIGURE 2. The time of quadriceps strength (a) and volitional activation (b) recovery from preoperative status through the 24 months after total knee arthroplasty (TKA). High-intensity resistive strength training and neuromuscular electrical stimulation (NMES) occurred between 12 and 15 months postoperatively. NMES was only applied to the left quadriceps muscle. Abbreviations: MVIC, maximal volitional isometric contraction; CAR, central activation ratio.

Diagnosis

In comparison to age-matched healthy individuals and patients 1 year after TKA, the physical therapy evaluation indicated that the patient had superior functional performance in walking and stair climbing. The patient's primary impairment was quadriceps weakness, with more substantial impairment in the left compared to the right quadriceps muscle. It seemed reasonable to infer that both muscle atrophy and muscle activation deficits were primary underlying causes of the patient's left quadriceps weakness, as indicated by smaller left quadriceps girth measurement and a lower CAR in the left quadriceps muscle.

Interventions

Based on the finding of bilateral quadriceps weakness and left quadriceps muscle activation deficit, a decision was made to implement a strengthening program consisting of high-intensity resistance training and NMES. The patient began treatment 13½ months after his surgery. The resistance program consisted of lower extremity exercises performed at the patient's local gym in conjunction with his cycling training program. Exercises included unilateral knee extensions in sitting, unilateral leg press in a semi-reclined position, unilateral wall squats in standing, prone hamstring curls, standing calf raises, and hip abduction and adduction in sitting performed on nautilus equipment. The patient was instructed to perform 3 sets of 10 repetitions, 3 times per week, at approximately 75% of his 1 repetition maximum for each leg, with the goal of reaching fatigue upon completion of each set of each exercise. The patient was instructed to retest his 1-repetition maximum weekly and increase the resistance accordingly. The patient logged his lower-extremity–strengthening program, which was monitored weekly by a physical therapist.

The left quadriceps femoris muscle was also treated with NMES. The decision was made to administer NMES only to the left quadriceps because it was the weaker of the 2 quadriceps muscles. In addition, the left quadriceps muscle had significant activation deficits, whereas the right quadriceps had full activation. The patient came to the physical therapy clinic 3 times per week for 6 weeks. The treatment protocol was previously published by Snyder-Mackler et al.[33] The patient was seated in an electromechanical dynamometer with the knee flexed and stabilized at 60° of knee flexion. Two stabilizing straps were secured around the patient's waist and left thigh. Following appropriate skin preparation, two 7.6 × 12.7-cm self-adhesive electrodes (CONMED Corporation, Utica, NY) were placed over the muscle bellies of the rectus femoris muscle proximally and the vastus medialis muscle distally (Figure 3). A clinical neuromuscular electrical stimulator (Versastim 380; Electro Med Health Industries, Miami, FL) was used to provide the electrical current. Stimulation was characterized by a 2500-Hz, sinusoidal, alternating waveform current at a burst rate of 75 bursts per second for 10 seconds (Table 2). The current amplitude was raised to the patient's maximum tolerance with the goal of achieving a minimum electrically elicited force output of 30% of the patient's daily

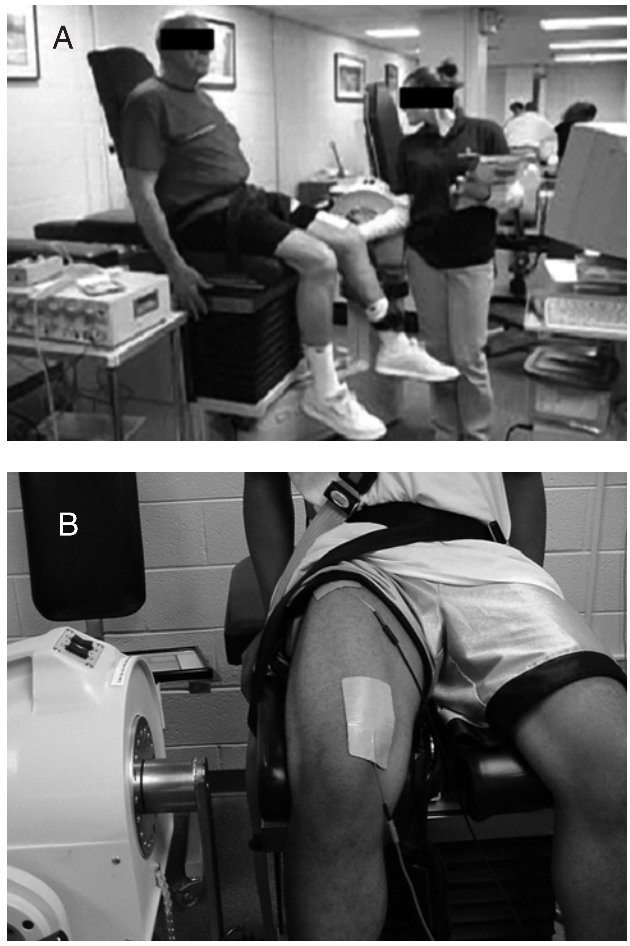

FIGURE 3. The patient setup for the neuromuscular electrical stimulation treatment. (A) The patient was seated in an electromechanical dynamometer with the knee flexed and stabilized at 60° of flexion; (B) two 7.62 × 12.25-cm self-adhesive electrodes were placed over the muscle bellies of the rectus femoris muscle proximally and the vastus medialis muscle distally.

MVIC. Each treatment session consisted of 10 contractions with an 80-second rest period between contractions. The patient's left thigh was assessed for redness and skin irritation following each daily NMES session.

The plan of care developed for the patient was to perform both the volitional strengthening program and the NMES treatment 3 times a week for 6 weeks. The length of the proposed treatment period was chosen based on previous research, which demonstrated that the performance of strengthening program for at least 6 weeks, is necessary to achieve significant strength gains especially in previously trained individuals such as this patient.[29] NMES treatments and volitional strength training typically occurred on the same day. NMES treatments were administered prior to volitional strengthening.

OUTCOMES

Following the completion of 16 NMES treatment sessions over a 6-week period, the patient's quadriceps strength and muscle activation were reassessed. The results revealed a 155-N improvement in maximum force production in the left quadriceps femoris muscle. This represents a 26% improvement in strength over the course of a 6-week physical therapy program. His left quadriceps muscle was now only 4% weaker than his right quadriceps muscle. This is a considerable improvement since he was 25% weaker prior to the formal physical therapy program. Improvement in left quadriceps muscle activation also occurred during the combined physical therapy intervention. Left quadriceps volitional activation levels progressed from a CAR of 0.83 before physical therapy to a CAR of 0.95 over the treatment period. Left thigh girth 2 cm above the patella increased 2.2 cm from the initial evaluation and left midthigh girth increased 2.1 cm. Right quadriceps strength also improved 11% over the 6-week period of physical therapy; however, no change was noted in the volitional activation levels.

The patient was discharged from formal physical therapy following the completion of 16 NMES treatments. The patient was instructed to continue his cycling and lower extremity strengthening program. Two additional follow-up evaluations were conducted to assess quadriceps strength, muscle activation, and functional performance (Table 1). The first occurred 3 months following discharge from formal physical therapy, 18 months after his TKA. The second occurred 24 months following his TKA. While his functional performance was similar to his 12-month assessment, the patient demonstrated improvement in his self-perceived function, as indicated by improved scores on the KOS/ADLS and GRS. The patient also demonstrated further increases in right and left quadriceps strength. Left quadriceps strength and volitional muscle activation exceeded preoperative levels and levels achieved at all other subsequent testing intervals. Left quadriceps strength was within 40 N of right quadriceps strength at the 18-month follow-up and, by the 24-month follow-up, left quadriceps strength surpassed that of the right (Figure 2).

DISCUSSION

This case report provides evidence for the successful management of chronic strength impairments following TKA for primary knee OA. The use of 2 intensive strengthening protocols, NMES and a unilateral lower extremity weight-training program, for 6 weeks resulted in improved quadriceps strength, thigh girth, and activation levels in addition to improved patient satisfaction. The measured improvements were maintained following conclusion from formal physical therapy.

Quadriceps strength is an important determinant of functional performance.[5,23] The patient's functional performance outcomes far exceeded values of individuals of similar sex and age who do not have OA even prior to the commencement of the combined NMES and high-intensity resistive exercise program. However, the patient was unsatisfied with his surgical outcome and functional recovery 12 months postoperatively (Table 1). The patient attributed his persistent, residual quadriceps strength deficits to his perceived limitations with his cycling activities.

TABLE 2. Neuromuscular electrical stimulation treatment protocol used for quadriceps strengthening. A Versastim 380 clinical neuromuscular electrical stimulator was programmed with the following stimulation parameters to induce muscle contraction.

Parameter	Setting
Carrier frequency	2500 Hz
Burst rate	75 bursts per sec
On time	10 seconds plus 2-sec ramp time
Off time	80 sec
Current amplitude	To patient tolerance; goal of 30% of patient's daily MVIC
Dose	10 contractions
Treatment frequency	3 times per wk
Treatment duration	6 wk

Abbreviation: MVIC, maximal volitional isometric contraction.

Volitional activation deficits and muscle atrophy are 2 main contributors to the dramatic quadriceps weakness associated with TKA.[4,21,37] If activation deficits and atrophy are not specifically targeted in rehabilitation interventions, these impairments will persist. Additional adverse consequences may include progression of OA in other lower extremity joints, additional arthroplasty surgeries, reduced function, and reduced quality of life. In the current scenario, the patient was only referred to physical therapy services for stretching and active exercise. The lack of sufficient resistive exercises most likely contributed to the persistence of quadriceps weakness. Traditional rehabilitation programs with similar trends of thought may not be sufficient to reverse strength-related impairments, especially if these impairments are chronic as with the current patient.

Aggressive strength training at 75% of 1-repetition maximum can increase force-generating capacity and induce muscle hypertrophy.[29] However, weakness combined with activation deficits presents even greater challenges.[8] If a muscle can not be activated to its full potential, it can be argued that strengthening solely through volitional exercise will not be sufficient to overload the muscle and enhance strength. The current patient exhibited significant activation deficit in his left quadriceps, which was hypothesized to be the main contributor to the patient's strength deficit. Therefore, NMES was used as an adjunct modality in the treatment of the patient's left quadriceps weakness and activation deficits.

NMES induces muscle contraction to overload the muscle via random recruitment of muscle fibers. NMES also targets a greater proportion of the larger type II, greater force-producing muscle fibers. During volitional exercise, recruitment of muscle fibers occurs according to the Henneman size principle (ie, recruitment progresses from small to large motoneurons). Therefore, the larger type II muscle fibers, activated in NMES protocols, are rarely if ever activated with volitional exercise, especially in the presence of activation deficits. Previous research in patients with bilateral TKA has demonstrated the positive effect of NMES in the subacute postsurgical period.[36] Quadriceps muscles treated with NMES demonstrated improved muscle activation levels with concomitant increases in strength. The resolution of activation deficits appeared to be the underlying mechanism for associated strength gains.[36]

When treated with 6 weeks of physical therapy, the current patient achieved similar benefit as patients treated with NMES in the immediate subacute period. The patient demonstrated a 25% increase in quadriceps strength and a 16% increase in quadriceps muscle activation levels. While a cause-and-effect relationship cannot be drawn from this case report, it is possible that improved activation levels may have contributed to improvements in quadriceps strength. Subjective reports also substantiated the impact of strengthening on functional performance and patient satisfaction indicated by his increased ability to perform hill and sprint cycling workouts.

CONCLUSION

This case report supports the implementation of formal physical therapy, including NMES and a high-resistance strength-training program, in the management of chronic quadriceps strength and activation impairments following TKA. The weak and inhibited left quadriceps muscles treated with a combined NMES and volitional strength training program exhibited dramatic increases in strength and muscle activation. Although this case report demonstrates the potential benefit of NMES and high-resistance strength training in the management of persistent quadriceps strength and activation deficits following TKA, future research is necessary to determine the efficacy of this intervention in a similar cohort of patients and to determine the dose of treatment necessary to reverse adverse strength-related impairments. Furthermore, additional research may shed light on the functional carryover of the resolution of interlimb strength asymmetries to the symmetrical performance of walking and quadriceps-demanding tasks, such as stair-climbing, as well as suggest the feasibility of return to high-level recreational activities.

ACKNOWLEDGEMENTS

We would like to thank the efforts of our research assistants, Katie Clements and Sarah Hingston, and the clinicians in the University of Delaware Physical Therapy Clinic for their support of this work.

REFERENCES

1. Altman R, Asch E, Bloch D, et al. Development of criteria for the classification and reporting of osteoarthritis. Classification of osteoarthritis of the knee. Diagnostic and Therapeutic Criteria Committee of the American Rheumatism Association. *Arthritis Rheum.* 1986;29:1039-1049.
2. American Academy of Orthopaedic Surgeons. Arthroplasty and Total Joint Replacement Procedures: 2003. Available at: http://www.aaos.org/wordhtml/research/stats/arthroplasty_recent.htm#Knee.
3. American Academy of Orthopaedic Surgeons. Information about Knee Replacements: 2002. Available at: http://www.aaos.org/wordhtml/research/stats/arthroplasty_recent.htm#Knee.
4. Berth A, Urbach D, Awiszus F. Improvement of voluntary quadriceps muscle activation after total knee arthroplasty. *Arch Phys Med Rehabil.* 2002;83:1432-1436.
5. Connelly DM, Vandervoort AA. Effects of detraining on knee extensor strength and functional mobility in a group of elderly women. *J Orthop Sports Phys Ther.* 1997;26:340-346.
6. Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Ann Intern Med.* 2000;133:635-646.
7. Fitzgerald JD, Orav EJ, Lee TH, et al. Patient quality of life during the 12 months following joint replacement surgery. *Arthritis Rheum.* 2004;51:100-109.
8. Hurley MV, Newham DJ. The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees. *Br J Rheumatol.* 1993;32:127-131.
9. Irrgang JJ, Snyder-Mackler L, Wainner RS, Fu FH, Harner CD. Development of a patient-reported measure of function of the knee. *J Bone Joint Surg Am.* 1998;80:1132-1145.
10. Jones CA, Voaklander DC, Johnston DW, Suarez-Almazor ME. Health related quality of life outcomes after total hip and knee arthroplasties in a community based population. *J Rheumatol.* 2000;27:1745-1752.
11. Jones CA, Voaklander DC, Suarez-Alma ME. Determinants of function after total knee arthroplasty. *Phys Ther.* 2003;83:696-706.
12. Kervio G, Carre F, Ville NS. Reliability and intensity of the six-minute walk test in healthy elderly subjects. *Med Sci Sports Exerc.* 2003;35:169-174.
13. Konig A, Walther M, Kirschner S, Gohlke F. Balance sheets of knee and functional scores 5 years after total knee arthroplasty for osteoarthritis: a source for patient information. *J Arthroplasty.* 2000;15:289-294.
14. Kramer JF, Speechley M, Bourne R, Rorabeck C, Vaz M. Comparison of clinic- and home-based rehabilitation programs after total knee arthroplasty. *Clin Orthop Relat Res.* 2003;225-234.
15. Lewek M, Stevens J, Snyder-Mackler L. The use of electrical stimulation to increase quadriceps femoris

muscle force in an elderly patient following a total knee arthroplasty. *Phys Ther*. 2001;81:1565-1571.
16. Lorentzen JS, Petersen MM, Brot C, Madsen OR. Early changes in muscle strength after total knee arthroplasty. A 6-month follow-up of 30 knees. *Acta Orthop Scand*. 1999;70:176-179.
17. McMahon M, Block JA. The risk of contralateral total knee arthroplasty after knee replacement for osteoarthritis. *J Rheumatol*. 2003;30:1822-1824.
18. Mizner RL, Petterson SC, Snyder-Mackler L. Quadriceps strength and the time course of functional recovery after total knee arthroplasty. *J Orthop Sports Phys Ther*. 2005;35:424-436.
19. Mizner RL, Petterson SC, Stevens JE, Vandenborne K, Snyder-Mackler L. Early quadriceps strength loss after total knee arthroplasty. The contributions of muscle atrophy and failure of voluntary muscle activation. *J Bone Joint Surg Am*. 2005;87:1047-1053.
20. Mizner RL, Snyder-Mackler L. Altered loading during walking and sit-to-stand is affected by quadriceps weakness after total knee arthroplasty. *J Orthop Res*. 2005;23:1083-1090.
21. Mizner RL, Stevens JE, Snyder-Mackler L. Voluntary activation and decreased force production of the quadriceps femoris muscle after total knee arthroplasty. *Phys Ther*. 2003;83:359-365.
22. Moffet H, Collet JP, Shapiro SH, Paradis G, Marquis F, Roy L. Effectiveness of intensive rehabilitation on functional ability and quality of life after first total knee arthroplasty: A single-blind randomized controlled trial. *Arch Phys Med Rehabil*. 2004;85:546-556.
23. Moxley Scarborough D, Krebs DE, Harris BA. Quadriceps muscle strength and dynamic stability in elderly persons. *Gait Posture*. 1999;10:10-20.
24. Parent E, Moffet H. Comparative responsiveness of locomotor tests and questionnaires used to follow early recovery after total knee arthroplasty. *Arch Phys Med Rehabil*. 2002;83:70-80.
25. Podsiadlo D, Richardson S. The timed "Up & Go": a test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc*. 1991;39:142-148.
26. Poole-Wilson PA. The 6-minute walk. A simple test with clinical application. *Eur Heart J*. 2000;21:507-508.
27. Rajan RA, Pack Y, Jackson H, Gillies C, Asirvatham R. No need for outpatient physiotherapy following total knee arthroplasty: a randomized trial of 120 patients. *Acta Orthop Scand*. 2004;75:71-73.
28. Ranawat CS, Ranawat AS, Mehta A. Total knee arthroplasty rehabilitation protocol: what makes the difference? *J Arthroplasty*. 2003;18:27-30.
29. Rhea MR, Alvar BA, Burkett LN, Ball SD. A meta-analysis to determine the dose response for strength development. *Med Sci Sports Exerc*. 2003;35:456-464.
30. Ritter MA, Thong AE, Davis KE, Berend ME, Meding JB, Faris PM. Long-term deterioration of joint evaluation scores. *J Bone Joint Surg Br*. 2004;86:438-442.
31. Shakoor N, Block JA, Shott S, Case JP. Nonrandom evolution of end-stage osteoarthritis of the lower limbs. *Arthritis Rheum*. 2002;46:3185-3189.
32. Silva M, Shepherd EF, Jackson WO, Pratt JA, McClung CD, Schmalzried TP. Knee strength after total knee arthroplasty. *J Arthroplasty*. 2003;18:605-611.
33. Snyder-Mackler L, Delitto A, Stralka SW, Bailey SL. Use of electrical stimulation to enhance recovery of quadriceps femoris muscle force production in patients following anterior cruciate ligament reconstruction. *Phys Ther*. 1994;74:901-907.
34. Stackhouse SK, Dean JC, Lee SC, Binder-MacLeod SA. Measurement of central activation failure of the quadriceps femoris in healthy adults. *Muscle Nerve*. 2000;23:1706-1712.
35. Stevens JE, Binder-Macleod S, Snyder-Mackler L. Characterization of the human quadriceps muscle in active elders. *Arch Phys Med Rehabil*. 2001;82:973-978.
36. Stevens JE, Mizner RL, Snyder-Mackler L. Neuromuscular electrical stimulation for quadriceps muscle strengthening after bilateral total knee arthroplasty: a case series. *J Orthop Sports Phys Ther*. 2004;34:21-29.
37. Stevens JE, Mizner RL, Snyder-Mackler L. Quadriceps strength and volitional activation before and after total knee arthroplasty for osteoarthritis. *J Orthop Res*. 2003;21:775-779.
38. Symmons D, Mathers C, Pfleger B. Global burden of osteoarthritis in the year 2000. Geneva, Switzerland: World Health Organization; 2000.
39. Walsh M, Woodhouse LJ, Thomas SG, Finch E. Physical impairments and functional limitations: a comparison of individuals 1 year after total knee arthroplasty with control subjects. *Phys Ther*. 1998;78:248-258.