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Quadriceps strength and volitional activation before and after total knee arthroplasty for osteoarthritis

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Abstract

Introduction: Patients with osteoarthritis (OA) of the knee have quadriceps weakness and arthrogenous muscle inhibition (AMI). While total knee arthroplasty (TKA) reliably reduces pain and improves function in patients with knee OA, quadriceps weakness persists after surgery. The purpose of this investigation was to assess contributions of AMI to quadriceps weakness before and after TKA and to assess the effect of pain on AMI.

Methods: Twenty-eight patients with unilateral, end-stage, primary knee OA were tested an average of 10 days before and 26 days after TKA. The mean age at time of operation was 63 years (range 49–82 years). Measurements on the involved and uninvolved knees were performed using the burst-superimposition technique, where supramaximal electrical stimulation is superimposed on a voluntary contraction. Knee pain during contraction was measured using a numeric rating scale.

Results: The involved quadriceps were significantly weaker than the uninvolved prior to TKA ($p < 0.05$). Quadriceps strength decreased by 60% ($p < 0.001$) and activation decreased 17% ($p < 0.001$) after TKA. Changes in muscle activation accounted for 65% of the variability in the change in quadriceps strength ($r^2 = 0.65$) ($p < 0.001$). Knee pain during muscle contraction accounted for a small, but significant portion of the change in voluntary activation ($r^2 = 0.22$) ($p = 0.006$).

Discussion: Exercise regimens that emphasize strong muscle contraction and clinical tools that facilitate muscle activation like biofeedback and neuromuscular electrical stimulation may be necessary to reverse the quadriceps activation failure and weakness in the patients with knee OA that worsens after TKA. The failure of current rehabilitation regimens to directly address activation deficits within the first months after surgery may explain the persistent quadriceps weakness in patients after TKA.

Keywords: Knee arthroplasty; Quadriceps; Osteoarthritis; Prospective; Aging

Introduction

Total knee arthroplasty (TKA) is one of the commonest operations performed for end-stage osteoarthritis (OA) in older individuals [10]. While TKA reliably reduces pain and improves function in patients with knee osteoarthritis, quadriceps weakness persists even years after surgery [9]. Quadriceps femoris weakness has profound functional consequences especially in older individuals resulting in disability including limitations in activities of daily living and an increased risk of falls [5,29]. The few studies that have measured quadriceps strength early after TKA report levels of quadriceps weakness that are greater than can be accounted for by muscle atrophy alone [21,22]. Recent studies have implicated impairments in central nervous system activation (failure of voluntary activation or arthrogenous muscle inhibition (AMI)) as a cause of muscle weakness that occurs with aging and in those with knee OA [6,7,12,20,25,26]. Although similar investigations have not been undertaken in patients after TKA, it is likely that AMI plays a significant role in the weakness they display. Two recent investigations have underscored the importance of AMI to surgeons and rehabilitation specialists who treat OA [13,17]. Hurley and Scott demonstrated that physical therapy resulted in a significant improvement in quadriceps muscle

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performance in a group of patients with OA and quadriceps weakness which was largely a result of reduced AMI [13]. Machner et al. suggested that surgery itself (in their case unicondylar arthroplasty) could result in improvements in voluntary activation of the quadriceps, perhaps to a greater degree than physical therapy [17].

Understanding the source of the quadriceps weakness that plagues this patient population and impairs function after surgery is critical to positive outcome after TKA. Quadriceps muscle activation deficits must be quantified and their contribution to quadriceps weakness after TKA identified so that improvements in outcomes can be made through rehabilitation that effectively targets the etiology of quadriceps weakness. The purpose of this study was to assess quadriceps femoris strength and voluntary activation before TKA and 3–4 weeks after TKA (when initial impairments are resolved, but before formal outpatient strength training begins) to better understand the role of OA and TKA on voluntary muscle activation deficits and persistent quadriceps weakness. We hypothesized that (1) voluntary activation and strength of the involved quadriceps would be less than the uninvolved quadriceps before and after surgery, (2) voluntary activation and strength of the involved quadriceps would decrease significantly after surgery, (3) voluntary activation would account for a significant amount of the loss of strength that occurs early after TKA, and that (4) knee pain would account for only a small part of the deficit in voluntary activation after surgery.

Methods

This prospective study included 28 patients who were scheduled to undergo, primary, unilateral, tricompartmental, TKA for primary OA of the knee. All prostheses were implanted according to a standard procedure with a medial parapatellar approach and cemented insertion of the patellar, tibial and femoral components. The indications for arthroplasty were clinical OA of the knee and all patients reported pain and functional disability during activities of daily living prior to surgery. Patients who showed evidence of musculoskeletal impairments that limited function in the unoperated lower extremity or the operated lower extremity other than the total knee arthroplasty were excluded. Patients who were diagnosed with diabetes mellitus, uncontrolled blood pressure, neoplasms, neurological disorders, or a body mass index (weight in kg/(height in m)^2) of greater than 40 (morbidly obese) were also excluded. Subjects' ages ranged from 49 to 82 years (avg. $= 63 \pm 8.8$ years).

Post-operative pain management

Spinal anesthesia was used and morphine sulfate was also injected at the time of the spinal to provided pain relief for up to 24 h. For breakthrough pain, patient controlled analgesia (PCA) is also used for the first 24 h. Subsequently oral narcotic analgesics were used for up to 2 weeks followed by use of non-narcotic analgesics as required.

Post-operative rehabilitation

Post-surgical management was done according to a standard protocol including progressive weightbearing on the surgically treated leg and exercises. The post-operative regimen included inpatient hospitalization for four days followed by 2 weeks (six visits) of home physical therapy. Home physical therapy consisted of isometric quadriceps exercises, training in activities of daily living (ADLS) and transfers, walking (weight bearing as tolerated) with an assistive device and active range of motion. Patients were cleared to begin outpatient physical therapy when their incision staples or sutures had been removed.

Clinical assessment

The Activities of Daily Living scale of the Knee Outcome Survey (KOS-ADLS), a sensitive, responsive regional health status index, was used to describe the overall functional status of the patients at each time period [14,18]. The question "To what degree does pain affect your level of daily activity?" from the KOS-ADLS was used to describe the effect of pain on activity for the patients at each time period. Scoring ranges from 0 (unable to perform activity) to 5 (does not affect activity).

Quadriceps strength and activation

Assessment of strength and voluntary activation of the quadriceps muscle was completed in all patients an average of 10 days *before* surgery and 26 days *after* surgery. The Human Subjects Review Committee at the University of Delaware approved the study and all subjects gave informed consent.

Quadriceps activation was estimated by superimposing a supramaximal electrical stimulus on a maximum voluntary isometric contraction (MVIC) [24,26]. Patients were seated on an electromechanical dynamometer (KinComIII, Chattecx Corporation, Chattanooga, TN) with their hips flexed to ≈85 deg and their knees flexed to 75 deg. The axis of the dynamometer was aligned with the axis of the knee joint and the bottom of the force transducer pad was positioned against the anterior aspect of the leg proximal to the lateral malleolus. The lower leg, thigh, and pelvis were stabilized using inelastic straps with Velcro closures. Two 3.0" by 5.0" self-adhesive neuromuscular stimulation electrodes were secured to the thigh. The anode was placed over the motor point of the rectus femoris and the cathode was placed over the motor point of the vastus medialis. A Grass S8800 stimulator with a Grass Model SIU8T stimulus isolation unit (Grass Instruments, West Warwick, Rhode Island) was used to deliver 1000 μs pulses. The stimulator was driven by a personal computer that uses customized software (Labview V 4.0.1) to control the timing parameters of the stimulation protocol. Data were digitized at 200 samples s-1 and analyzed with custom written software (Labview, National Instruments).

To ensure that the patients were exerting a maximal effort, patients were familiarized with the procedure, received verbal encouragement from the tester and visual feedback from the dynamometer's real time force display. Several submaximal (perceived as 50–75% of maximal effort) contractions served to familiarize the patient with the apparatus. The quadriceps femoris muscle was then stimulated at a submaximal intensity to familiarize the patient with the electrical stimulation. After the patient was familiar with the procedure, testing began by having the patient perform a 3–5 s MVIC. During the contraction, a supramaximal, 10-pulse, 100 pulse/s train of electrical stimulation was delivered to the muscle to assess muscle activation level (i.e. burst-superimposition technique). With a maximal contraction, no increase in force is elicited by the burst. If a subject was unable to activate the quadriceps muscle fully (i.e. the burst augmented the force) (Fig. 1), MVIC testing was repeated up to two additional times. The maximal voluntary force recorded during the three tests was used in the analysis. Each attempt at achieving an MVIC was separated by at least 5 min to minimize the effects of muscle fatigue on test performance.

Pain assessment

Immediately after each maximal quadriceps contraction, patients were asked to rate their knee pain during the contraction using a 0–10 numeric rating scale where 0 represents no pain and 10 represents the "worst pain imaginable" [16]. These data were used to assess the effect of knee pain with muscle contraction on muscle activation deficits.

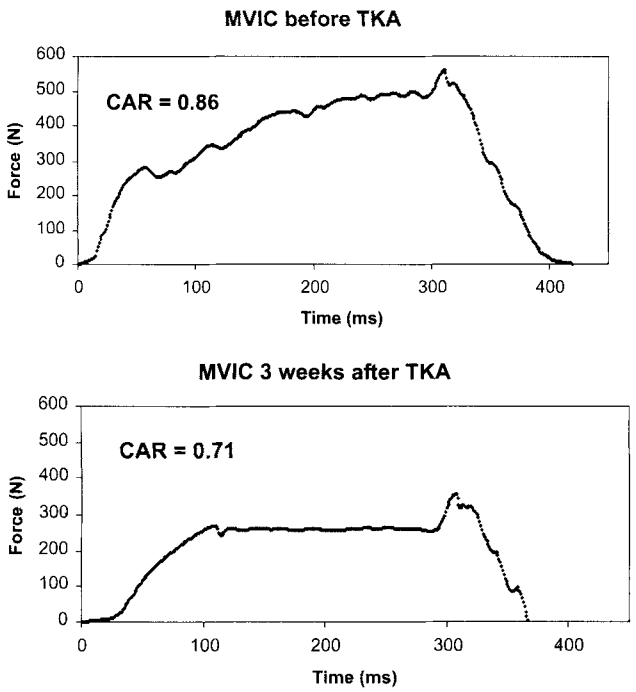

Fig. 1. Quadriceps force tracings. Force tracings from maximum voluntary isometric contraction (MVIC) testing with burst superimposition with the same individual before and 3 weeks after total knee arthroplasty.

Data management and analysis

Clinical assessment

KOS-ADLS scores and KOS pain scores were compared from before to after surgery using a paired *t*-test.

Quadriceps strength and activation

MVIC values were normalized to body mass index. The peak volitional and electrically elicited forces were used to determine the degree of voluntary quadriceps muscle activation by the patient. The central activation ratio (CAR) was used to quantify voluntary activation. The CAR is calculated by dividing the maximum voluntary force by the total force (including any force augmentation from the burst) [15]. A CAR of 1.0 signifies complete activation. In contrast, a CAR of less that 1.0 suggests incomplete activation of the muscle by the central nervous system.

Paired *t*-tests with Bonferroni correction (adjusted alpha = 0.0102) were used to compare: (1) the strength and CAR of the involved to uninvolved quadriceps before and after surgery, (2) the strength of the involved quadriceps before and after TKA, (3) the CAR of the involved quadriceps before and after TKA, (4) the strength of the uninvolved quadriceps before and after TKA, (5) the CAR of the uninvolved quadriceps before and after TKA. Linear regression was used to assess the relationship between percent change in MVIC of the involved quadriceps (MVIC before surgery – MVIC after surgery)/MVIC before surgery) and the change in CAR.

Pain and muscle activation

Linear regression was used to assess how knee pain during quadriceps contraction, as measured by the verbal rating scale, affects voluntary activation as measured by CAR. A *p*-value of less than 0.05 was considered significant for all tests.

Results

Testing was completed an average of 10 days prior to TKA and 26 days after TKA. KOS-ADLS score was $56 \pm 18\%$ prior to surgery and $59 \pm 15\%$ after surgery ($p > 0.05$). KOS pain score was better (2.8 ± 1) after surgery than before surgery (2.8 ± 1) ($p = 0.002$).

Quadriceps strength and activation

The involved quadriceps were significantly weaker than the uninvolved side before surgery ($p = 0.004$) and after surgery ($p < 0.001$). After surgery, the quadriceps femoris of the involved leg was significantly weaker than before surgery ($p < 0.001$) (Table 1). Normalized quadriceps strength of the involved leg decreased by an average of 60% after surgery. All patients were profoundly weaker after surgery as the range of strength loss after surgery was 30–85% (Fig. 2).

Voluntary muscle activation of the involved side was not significantly lower than the uninvolved side before

Table 1
Volitional activation and strength of the quadriceps before and after TKA

	CAR involved	CAR uninvolved	MVIC/BMI involved	MVC/BMI uninvolved
PRE	0.85 ± 0.17	0.91 ± 0.11	20 ± 8^{a}	25.6 ± 10
POST	0.69 ± 0.2^{b}	0.91 ± 0.11	8 ± 5^{b}	25.4 ± 9

Mean ± SD and statistical comparison of maximal values for each subject. All values are normalized units. CAR = central activation ratio; MVIC = maximum voluntary isometric contraction; BMI = body mass index.

[a] Different from uninvolved side.

[b] Different from uninvolved side and pre-operative value; ($p < 0.01$).

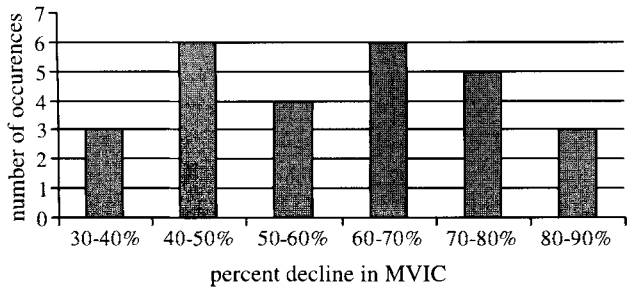

Fig. 2. Distribution of quadriceps strength loss after TKA ($n = 28$). Number of patients with strength loss in each decile.

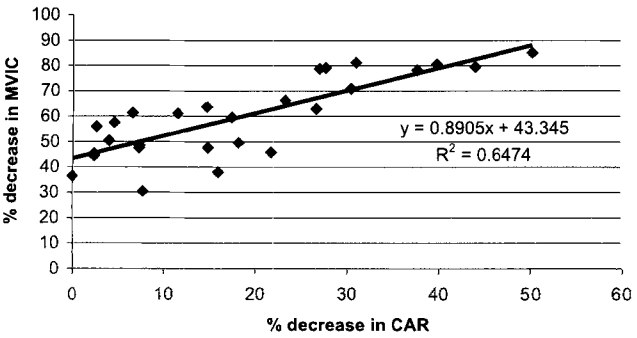

Fig. 3. Percent change in quadriceps strength and volitional activation. Linear regression analysis examining the relationship between changes in quadriceps strength (MVIC) and changes in muscle activation (central activation ratio, CAR) before and 3–4 weeks after TKA ($r^2 = 0.647$, $p < 0.001$) ($n = 28$) (mean ± SD).

surgery ($p = 0.039$), but was significantly reduced on the involved side after surgery ($p < 0.001$); CAR decreased by an average of 17% from pre-operative values. Linear regression revealed that decreased CAR accounted for 65% of the variability in the change in quadriceps strength from before to after surgery ($r^2 = 0.647$; $p < 0.001$; Fig. 3). There were no significant differences in strength or voluntary activation for the uninvolved quadriceps from before to after surgery ($p > 0.05$).

Pain and muscle activation

Pain with muscle contraction increased from 1.5/10 to 3.9/10 ($p = 0.002$) from before to after surgery. The change in pain during muscle contraction accounted for a small, but significant portion of the change in CAR ($r^2 = 0.22$) ($p = 0.006$).

Discussion

Although a variety of factors may contribute to quadriceps muscle weakness that occurs after TKA, the results of this study suggest that the inability to fully activate the quadriceps muscle is the major cause. Patients scheduled for TKA had involved quadriceps that were significantly weaker than their uninvolved quadriceps. An average of 3.5 weeks after surgery, quadriceps strength had decreased by 60%. More than half of the strength loss was explained by an increase in AMI. Pain with contraction accounted for less than 25% of the increase in AMI that occurred from before to after surgery.

Pain is common in individuals with end-stage osteoarthritis, as well as in patients during the first weeks after TKA [3,4,20]. Pain management is a focus of treatment in the early period after TKA. In addition to intraoperative and post-operative pharmacological pain management, most rehabilitation programs after TKA include treatment for pain, including cryotherapy and analgesics [3,19]. In this sample, while knee pain's effect on activity decreased from before to after surgery, the overall KOS-ADLS outcome rating was unchanged. Since pain during a maximum quadriceps contraction accounted for only a small portion of the increase in AMI that occurred after surgery in the present study, current pain management regimens may be adequate.

Muscle atrophy does not appear to be the predominate etiology of quadriceps weakness after TKA. Perhonen et al. found less than 5% quadriceps atrophy in patients 3 weeks after TKA, while healthy adults have exhibited 16–21% quadriceps atrophy with disuse and immobilization over approximately the same time frame [1,8,21,27]. This is not surprising as patients are not immobilized after TKA. In fact, beginning as early as the day of surgery, patients are encouraged to use the involved leg during ambulation and perform knee active range of motion and isometric exercises repeatedly throughout the day [3]. While healthy individuals almost always recover 100% of their muscle strength within 4–8 weeks of remobilization after a period of disuse [1,23], patients after TKA are unable to achieve comparable recovery, even years after surgery [2,9,11,28]. The results of the present study suggest that more vigorous exercise programs may be necessary to restore quadriceps strength in this population [13]. Interventions directly affecting activation, (e.g. neuromuscular electrical stimulation and biofeedback), may better address the weakness than voluntary exercise alone. More appropriate rehabilitation regimens that target inhibition rather than atrophy may actually result in shorter courses of rehabilitation that are more effective than current programs.

While a continued emphasis on pain control after surgery appears warranted, rehabilitation programs should also focus on fully restoring quadriceps muscle strength. Exercise regimens that emphasize strong muscle contraction and clinical tools that facilitate muscle activation, like biofeedback and neuromuscular electrical stimulation, may be necessary to reverse the quadriceps activation failure and weakness in the pa-

tients with knee OA that worsens after TKA. The failure of current rehabilitation regimens to directly address activation deficits within the first months after surgery may provide some explanation the persistent quadriceps weakness in patients after TKA.

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