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Quantifying Neuromuscular Electrical Stimulation Dosage after Knee Arthroplasty

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Abstract

Recovering functional ability after total knee arthroplasty (TKA) requires recovery of strength and voluntary activation. Short-term recovery of strength and activation are enhanced following a protocol combining strength training with neuromuscular electrical stimulation (NMES). The purpose of the study was to determine if a dose response curve could be constructed for patients who received NMES as part of their treatment after TKA. NMES dosage was quantified as the electrically evoked knee extensor torque, expressed as a percentage of the subject's maximal voluntary contraction. Dose-response curves were generated, with the associations between NMES training intensity and quadriceps strength, voluntary activation, and lean muscle cross-sectional area examined using Pearson Product-Moment Correlation Coefficients. Significantly, linear correlations were observed between NMES training intensity and both quadriceps strength and voluntary activation, but not lean muscle cross-sectional area. These results suggest that maximizing the elicited training force during rehabilitation will enhance short-term recovery following TKA.

Keywords

Quadriceps strength; voluntary activation; total knee arthroplasty; neuromuscular electrical stimulation; rehabilitation

1. Introduction

Total knee arthroplasty (TKA) procedures are expected to increase substantially over the next 20 years [1]; however, consensus on treatment regimens following TKA remains equivocal. Quadriceps strength is a primary determinant of lower extremity functional ability [2]; with a larger portion of the quadriceps weakness observed in patients following TKA attributable to reductions voluntary activation than to muscle atrophy [3]. Improvements in both quadriceps strength and voluntary activation have been demonstrated after surgery with the use of progressive quadriceps strength training combined with neuromuscular electrical stimulation (NMES) [4]. Therefore, it is important to understand how the training dosage of NMES used for patients who have undergone TKA influences quadriceps strength, size and voluntary activation. The purpose of this study was to establish a dose-response curve and determine how the intensity of NMES treatment after TKA influences the improvement in quadriceps strength, atrophy, and activation. We hypothesized a positive dose-response relationship for all.

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2. Materials and Methods

2.1 Subjects

Seventy individuals (29 women; 51–82 yrs) scheduled for unilateral TKA who were included in the NMES arm of the trial were the subjects for this study [4]. Exclusion criteria included severe obesity (BMI ≥ 40 [5]), history of diabetes, uncontrolled high blood pressure, cardiovascular disease, and symptomatic osteoarthritis in the contralateral knee or history of other function-limiting lower extremity pathologies. Subjects who received NMES were evaluated for changes in quadriceps strength and voluntary activation at initial evaluation (IE; ~4 weeks after surgery), midway through the intervention (Mid), and after completing the intervention (End). Atrophy was assessed before and after the intervention with magnetic resonance imaging (MRI). The study was approved by the University of Delaware's Human Subject Review Board and subjects provided informed consent.

2.2 Neuromuscular Electrical Stimulation Training

Subjects received 6-weeks of progressive strength training [6] with neuromuscular electrical stimulation (2–3 times/week) beginning 3–4 weeks after surgery [4]. The NMES was performed on an electromechanical dynamometer (KinCom; Chattanooga Corporation, Chattanooga, TN), with the knee flexed to 60° and the hip flexed to ~90°. The protocol comprised 10 electrically evoked contractions of the quadriceps delivered through two self-adhesive electrodes (7.62 × 12.70 cm; ConMed; Utica, NY) for 10 s, with a 2.5 kHz sinusoidal wave at 50 Hz (Electro Med Health Industries; Miami, FL). Stimulus intensity was determined by each subject's maximum tolerance, with target dosage of at least 30% of the subject's maximal voluntary contraction (MVC). The dose of each contraction was recorded as a percentage of the involved knee MVC. Dose was defined as the average recorded dose over the 18 sessions.

2.3 Quadriceps Strength, Voluntary Activation, and Size

Quadriceps strength and voluntary activation were also recorded on a KinCom, with hip flexed ~90° and knee flexed ~75°. The axis of rotation was aligned with the lateral femoral condyle and the distal end of the lever arm secured to the lower leg ~2 cm above the lateral malleolus. Subjects completed submaximal and maximal knee extension contractions for familiarization and warm-up, with strength recorded as the peak force during a MVC; subjects received visual feedback and strong verbal encouragement. Analysis of changes in quadriceps strength was evaluated using the quadriceps index (QI = involved MVC/uninvolved MVC).

Voluntary muscle activation was assessed using the burst super-imposition technique [7]. A Grass S8800 stimulator delivered a 12-pulse, 100-Hz train of stimuli at 135 Volts (Grass Instruments; West Warwick, RI) through two electrodes (7.2 × 12.7 cm; CONMED Corp, Utica, NY) applied proximally over the rectus femoris and distally over the vastus medialis. A program controlled the timing of stimuli to be delivered during the plateau in force of an MVC (LabView 4.01, National Instrument; Austin, TX). Voluntary activation was quantified with the central activation ratio (CAR) [4] as the percentage increase in force after stimulation above the MVC force.

Lean Muscle Cross Sectional Area (LMCSA) of the quadriceps was quantified in 29 of 70 subjects using MRI before (IE) and after (End) the intervention. Subjects were imaged axially, from greater trochanter to tibial plateau (7 mm interslice intervals) while laying supine, knees extended, in a 1.5 Tesla body coil (Signa, General Electric, Waukesha, WI) using standard sequence imaging (GE SPGR: 2-D spoiled gradient-echo, 500-ms pulse TR, 8-ms TE with a 256 × 256 encoding matrix and 480×480-mm field of view). Images were

processed in IMOD software (The Boulder Laboratory for 3D Electron Microscopy of Cells; Boulder, Colorado) digitally outlining the quadriceps (Intuos 2; Wacom Corporation; Vancouver, Washington). Customized software scaled the outlines and removed intramuscular fat and connective tissue to calculate the enclosed LMCSA. The MRI image with the largest LMCSA was used for data analyses.

2.4 Data Analysis

Repeated measures ANOVAs were performed to examine changes in QI and CAR across time (IE, Mid, End) with Least Significant Difference used for pairwise comparisons in the presence of a main effect (SPSS v15.0; SPSS Inc.; Chicago, IL). Huynh-Feldt corrections were applied for unequal variance of the repeated measures factors. Paired samples t-test was performed to determine changes in LCSA from IE to End. The relations between training dose and response (change in QI, CAR and LMCSA) were evaluated using Pearson Product-Moment Correlation Coefficients. Significance for statistical analyses was $P \leq 0.05$.

3. Results and Discussion

The treatments produced main effects for strength and activation ($P < 0.001$). Strength improved at each time point of the intervention, evident by significant changes in the QI (IE = 49.9 ± 22.9%; Mid = 66.2 ± 24.3%; End = 81.3 ± 26.7%). Similarly, activation levels increased at each time point (IE = 78.3 ± 15.4%; Mid = 87.7 ± 12.6%; End = 89.5 ± 10.1%). The LMCSA significantly improved from IE (30.8 ± 12.7 cm^2) to the End (36.4 ± 14.6 cm^2) of treatment ($P < 0.001$).

The average dose exceeded the minimal target of 30% MVC for all but eight subjects; one subject's average dose was 10% MVC, seven subjects had average doses 23–29% MVC, with the group average of 55.6 ± 28.9% MVC (range 10%–203%).

The stimulation training dosage was significantly and positively correlated with changes in QI (r = 0.480; $P < 0.001$; Fig. 1A) and CAR (r = 0.574, $P < 0.001$; Fig. 1B), but not LMCSA (r = –0.325; $P = 0.086$).

Quadriceps strength, voluntary activation, and LMCA had statistically and clinically important improvements over the intervention period. NMES dose was significantly and positively correlated with strength and activation improvements, but not with increases in LMCSA. NMES overcomes activation deficits during application. Perhaps the main mechanism by which NMES contributes to muscle strength gain is via its effects on voluntary muscle activation.

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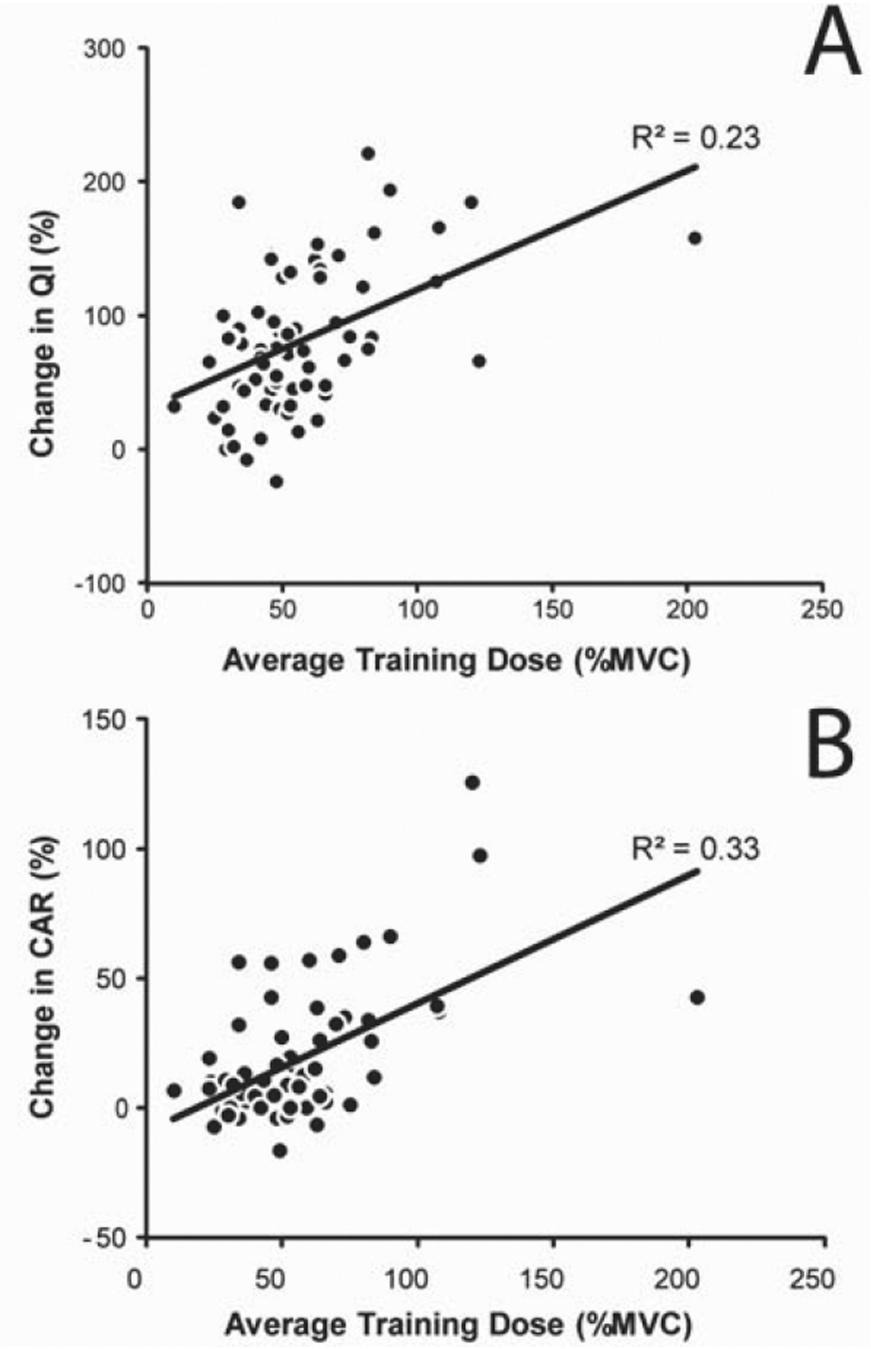

Fig. 1.

The changes in quadriceps index (QI; A) and central activation ratio (CAR; B) as a function of the average training dosage were significant, $P < 0.001$.