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Electromyographic patterns suggest changes in motor unit physiology associated with early osteoarthritis of the knee[1,2]

S. M. Ling M.D.†*, R. A. Conwit M.D.‡, L. Talbot Ph.D.§, M. Shermack M.D.‖, J. E. Wood M.D.¶, E. M. Dredge M.D.†, M. J. Weeks B.S.†, D. R. Abernethy M.D., Ph.D.# and E. J. Metter M.D.†

† *Clinical Research Branch, National Institute on Aging Intramural Research Program (NIA-IRP), National Institutes of Health, MD 21225, United States*

‡ *National Institute of Neurological Diseases and Stroke Extramural Research Program, Bethesda, MD 20894, United States*

§ *Uniformed Services University of Health Sciences, Bethesda, MD 20894, United States*

‖ *The Johns Hopkins Medical Institutions, Baltimore, MD 21224, United States*

¶ *Department of Orthopedic Surgery, Harbor Hospital, Baltimore, MD 21225, United States*

Laboratory of Clinical Investigation, NIA-IRP, Baltimore, MD 21224, United States

Summary

Objective: To assess characteristics of active motor units (MUs) during volitional vastus medialis (VM) activation in adults with symptomatic knee osteoarthritis (OA) across the spectrum of radiographic severity and age-comparable healthy control volunteers.

Methods: We evaluated 39 participants (age 65 ± 3 years) in whom weight-bearing knee X-rays were assigned a Kellgren & Lawrence (KL) grade (18 with KL grade = 0; four each with KL grades = 1, 2 and 4; nine with grade 3). Electromyography (EMG) signals were simultaneously acquired using surface [surface EMG (S-EMG)] and intramuscular needle electrodes, and analyzed by decomposition-enhanced spike-triggered averaging to obtain estimates of size [surface-represented MU action potentials (S-MUAP) area], number [MU recruitment index (MURI)] and firing rates [MU firing rates (mFR)] of active MUs at 10%, 20%, 30% and 50% effort relative to maximum voluntary force [maximal voluntary isometric contraction (MVIC)] during isometric knee extension.

Results: Knee extensor MVIC was lower in OA participants, especially at higher KL grades ($P = 0.05$). Taking the observed force differences into account, OA was also associated with activation of larger MUs (S-MUAP area/MVIC × %effort; $P < 0.0001$). In contrast, the estimated number of active units (MURI/MVIC × %effort) changed differently as effort increased from 10% to 50% and was higher with advanced OA (KL = 3, 4) than controls ($P = 0.0002$).

Conclusion: VM activation changes at the level of the MU with symptomatic knee OA, and this change is influenced by radiographic severity. Poor muscle quality may explain the pattern observed with higher KL grades, but alternative factors (e.g., nerve or joint injury, physical inactivity or muscle composition changes) should be examined in early OA.

Key words: Knee osteoarthritis, Quadriceps muscle, Motor unit, Electromyography, Mobility.

Introduction

Osteoarthritis (OA) of the knee is a leading cause of mobility limitations in late life and is often accompanied by quadriceps muscle weakness[1,2]. Although quadriceps weakness appears to be both a consequence[3–5] and a risk factor for knee OA[6], muscle strengthening has not been proven to protect individuals from OA[7]. Moreover, recent evidence suggests that greater knee extensor strength increases risk of OA progression in knees that are malaligned[8,9]. Thus, the role quadriceps muscle strength plays in the natural history of knee OA appears to be complex and worthy of further investigation.

Electromyography (EMG) measures the electrical activity of contracting skeletal muscle and can provide a unique physiologic assessment of the motor unit (MU) – the functional unit of skeletal muscle comprised a single lower motor neuron and all the muscle fibers it innervates. The force generated during a muscle contraction is determined by the number of and size of active MUs, as well as the rate and timing of their discharge. Since all fibers in a given MU contract synchronously in an all or none fashion, the precision with which muscles are engaged in action is largely dependent on MU size, firing rate and activation

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*Address correspondence and reprint requests to: Dr Shari M. Ling, M.D., Staff Clinician, ASTRA Unit at Harbor Hospital, 3001 S. Hanover Street, Baltimore, MD 21225, United States. Tel: 1-410-350-3934, 1-410-350-3950 (office); Fax: 1-410-350-3957, 1-410-350-3963; E-mail: lingsh@grc.nia.nih.gov

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strategy used to generate force. According to Henneman's principle[10], MUs are recruited into action beginning with smaller units and adding progressively larger units as force increases. In large muscles, such as those engaged during knee extension, force increases are achieved by progressive recruitment of MUs. Additional force is generated at higher levels by increasing the rate at which the MUs fire. In contrast, the strategy employed by small muscles, e.g., intrinsic hand or ocular muscles, firing rate increases prior to recruitment of additional units. Thus, the MU activation pattern and strategy employed by large muscles during knee extension allows for exertion of greater force, but with less precise control than that which is achievable by smaller muscles, e.g., intrinsic hand or ocular muscles.

EMG techniques have been developed to study MU activity during muscle activation that indicates the size and number of active MUs and the rate at which they discharge[11,12]. Neuromuscular alterations, and specifically changes in the peripheral nerve, MU, and muscle composition, are likely contributors to age-associated sarcopenia[13], and are also plausible contributors to the impact of the quadriceps on OA – an age-associated disease. We have previously demonstrated age-associated MU recruitment patterns during progressive, sub-maximal muscle activation of the vastus medialis (VM)[13–16]. Since the size of active units is directly related to muscle force generated, alterations in the MU might at least in part explain the relationship of muscle weakness with OA. Thus far, EMG techniques used to study OA have been limited to the evaluation of whole muscle activation patterns assessed by surface EMG during mobility task performance[17]. Although informative with regards to the timing and magnitude of whole muscle contraction, surface EMG cannot distinguish between neural and muscle contributions to force generation. At this time, little is known regarding the pattern of MU activation during force generation in the presence of knee OA.

In this study, we employed a technique developed to sample active MUs during knee extension at different force levels using EMG signals simultaneously acquired from surface EMG (S-EMG) and an intramuscular needle. We used this method to assess MU activation patterns, specifically MU size and firing rate, in adults with symptomatic knee OA across the spectrum of radiographic severity and healthy control volunteers of comparable age.

Methods

STUDY DESIGN

We analyzed EMG data from participants in a case–control observational study that were pooled with pre-intervention data from an electrical stimulation treatment study for OA of the knee[18]. This research was approved by the Institutional Review Boards of the Johns Hopkins Bayview Medical Center, MedStar Research Institute and the Johns Hopkins University Joint Committee on Clinical Investigation.

PARTICIPANTS

Cases from both studies comprised adults with symptomatic, radiographically confirmed OA of the knee who were recruited from the Baltimore–Washington metropolitan area by newspaper and Internet advertisements, and clinic posters. Healthy controls were recruited by the same means, and data were pooled with data from healthy adult participants of the Baltimore Longitudinal Study of Aging who denied knee symptoms or radiographic signs of OA and who underwent the same EMG evaluation. The analysis sample comprised 21 participants with OA of the knee and 18 controls.

MEASURES

Evaluation of OA

Knee symptoms were assessed by asking, "During the past year, have you had pain, aching or discomfort in your knees on most days for at least one month?" In addition, weight-bearing AP radiographs were independently assigned a Kellgren & Lawrence (KL) grade (0–4) by two rheumatologists[19]. Discordant readings were adjudicated by discussion and consensus between the two reviewers.

Strength testing

All strength measurements and testing were performed on a Kin-Com 125E dynamometer (Chattecx, Chattanooga, TN) following the same standardized protocol[20–22]. Maximal voluntary isometric force in Newtons (N) was measured at a knee angle of 60° of flexion. The average of the two best trials out of three was used as maximal voluntary isometric contraction (MVIC).

Electromyographic data collection

These data were collected using previously published methods and analyzed by decomposition-enhanced spike-triggered averaging (DE-STA)[15,16,20] as described below. The basic approach is to collect surface and intramuscular needle electrode EMG while subjects generate a fixed isometric force on the KinCom that represents a given percentage of their MVIC. The needle signal is used to identify individual MUs, while the surface EMG reflects the total muscle activity, and is decomposed to identify the surface representation of the individual MUs.

Electrical activity of the muscle was recorded using an active electrode over the VM muscle as the femoral nerve was electrically stimulated with a Teca bipolar stimulator. The inactive recording electrode was placed on the patellar tendon and ground electrode positioned between the stimulating and recording sites. The active and inactive recording electrodes were Teca 32 mm diameter discs. The recording electrode was moved several times to assure that recording was over the innervation zone and that supra-maximal stimulation resulted in a compound muscle action potential (CMAP) of maximum amplitude and minimal rise time.

After determining MVIC, the concentric needle electrode was positioned in the VM directly beneath the surface electrode configuration. Since most physical activities are achieved at relatively low force levels, rather than at MVIC, data were collected during knee extension with subjects sustaining consistent knee extension contraction for a period of 20–30 s at intensities of 10, 20, 30 and 50 percent (%) effort relative to MVIC. EMG signals were amplified, filtered and acquired using a Clarke Davis Advantage Medical A100 EMG system (London, ON). Simultaneously detected intramuscular and surface detected EMG signals were acquired using band pass filtering from 10 Hz to 10 kHz and 5 Hz to 1000 Hz, and sampling rates of 25 kHz and 2.5 kHz, respectively. Repeated efforts were made at each force level with intramuscular electrode repositioning until 15–20 MU trains were sampled at each force level (coefficient of variation of approximately 10%)[13]. MU trains and surface-represented MU action potentials (S-MUAP) were reviewed visually to