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ORIGINAL ARTICLE

Peripheral Nerve Function and Lower Extremity Muscle Power in Older Men

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Abstract

Objective: To assess whether sensorimotor peripheral nerve function is associated with muscle power in community-dwelling older men.

Design: Longitudinal cohort study with 2.3±0.3 years of follow-up.

Setting: One clinical site.

Participants: Participants (n=372; mean age ± SD, 77.2±5.1y; 99.5% white; body mass index, 27.9±3.7kg/m^2; power, 1.88±0.6W/kg) at 1 site of the Osteoporotic Fractures in Men Study (N=5994).

Interventions: Not applicable.

Main Outcome Measures: A nerve function ancillary study was performed 4.6±0.4 years after baseline. Muscle power was measured using a power rig. Peroneal motor nerve conduction amplitude, distal motor latency, and mean f-wave latency were measured. Sensory nerve function was assessed using 10-g and 1.4-g monofilaments and sural sensory nerve conduction amplitude and distal latency. Peripheral neuropathy symptoms at the leg and feet were assessed by self-report.

Results: After adjustments for age, height, and total body lean and fat mass, 1 SD lower motor (β=−.07, $P<.05$) and sensory amplitude (β=−.09, $P<.05$) and 1.4-g (β=−.11, $P<.05$) and 10-g monofilament insensitivity (β=−.17, $P<.05$) were associated with lower muscle power/kg. Compared with the effect of age on muscle power (β per year, −.05; $P<.001$), this was equivalent to aging 1.4 years for motor amplitude, 1.8 years for sensory amplitude, 2.2 years for 1.4-g monofilament detection, and 3.4 years for 10-g detection. Baseline 1.4-g monofilament detection predicted a greater decline in muscle power/kg. Short-term change in nerve function was not associated with concurrent short-term change in muscle power/kg.

Conclusions: Worse sensory and motor nerve function were associated with lower muscle power/kg and are likely important for impaired muscle function in older men. Monofilament sensitivity was associated with a greater decline in muscle power/kg, and screening may identify an early risk for muscle function decline in late life, which has implications for disability.

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Lower extremity muscle power is an important determinant of late-life physical function.[1,2] Muscle power, a measure of contractile force and shortening speed, has been linked to risk of falls,[3] mobility loss measured by physical performance tests such as walking, chair stands, and stair climbing,[4-11] and self-reported functional status[2,12] in older adults. Compared with strength,

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muscle power declines more steeply with age[4,13] and may be more strongly associated with certain measures of mobility.[6,9,11,12] Moreover, training programs designed to improve muscle power and velocity of movement may be more effective at improving physical performance than those that solely incorporate basic resistance training.[14-16]

Poor muscle power in late life and its unique relationship with mobility may be due, at least in part, to impairments in peripheral nerve function.[17-22] The components of power, force and velocity production, are likely dependent on the number and firing rate of motor units.[23] In addition, afferent input and impaired sensory nerve function may play an important role in muscle and physical function[24-26]; this is believed to occur through loss of proprioception.[24,26-28] Like muscle power, peripheral nerve function declines with age,[29-33] and has similarly been linked with physical function limitations and impairments[32,34] and an increased risk of falls.[35-37] The 1999−2000 National Health and Nutrition Examination Survey showed that 35% of adults 80 years and older had impaired nerve function measured using simple screening for reduced sensation at the foot.[33] Additionally, both poor motor and sensory peripheral nerve function have been related to reduced lower extremity quadriceps strength in the Health Aging and Body Composition Study; muscle power was not assessed in this study.[25]

Despite the independent relationships of muscle power and nerve function with mobility-related outcomes, whether peripheral nerve function loss is a determinant of muscle power decline has not been assessed. In a longitudinal cohort study of older men, we evaluated whether sensory and motor peripheral nerve function measures, commonly used in clinical evaluations and neurologic studies, are related to lower extremity muscle power cross-sectionally and longitudinally. We hypothesize that worse and declining nerve function is associated with poor and declining muscle power.

Methods

Study population

We used data from a nerve function ancillary study in which 372 participants had nerve function and power measured during the first visit, and 241 participants had these repeated during a second visit. The ancillary study was performed at the Monongahela Valley site 4.6±0.4 years after the 2000 to 2002 baseline visit. The second visit occurred 2.3±0.3 years later. This ancillary study was part of the Osteoporotic Fractures in Men Study, which is a cohort of community-dwelling ambulatory men (N=5994) 65 years and older enrolled between March 2000 and April 2002 at 6 U.S. clinic sites (Birmingham, AL; Minneapolis, MN; Palo Alto, CA; Monongahela Valley near Pittsburgh, PA; Portland, OR; and San Diego, CA; n=1005 in Pittsburgh at baseline). Eligibility for the main study included the ability to walk without assistance of another person or an aide, the ability to provide self-reported data, the ability to understand and sign an informed consent, the absence of bilateral hip replacements, the absence of a medical condition that would result in imminent death, and anticipated residence near a clinic site for the duration of the study period. The primary recruitment strategy was mailing invitations to men living in the surrounding communities of clinic sites. Supplementary strategies included community and senior newspaper advertisements and presentations to community groups. The study protocol was approved by the University of Pittsburgh Institutional Review Board, and written informed consent was obtained from all participants before testing. Of 662 men with nerve function measured during the first visit of the ancillary study, 372 had muscle power measured and were included in the cross-sectional analyses. Reasons for muscle power not being measured included temporary equipment failure (n=205), refusal of participants (n=11), and inability because of participants' physical limitations (n=74). Participants with missing cross-sectional muscle power data did not differ by age but had a slightly higher body mass index (BMI) (28.6kg/m^2 vs 27.9kg/m^2, $P=.03$) and a higher prevalence of diabetes (27.9% vs 17.0%, $P<.001$). Of the participants included in the cross-sectional analysis, 279 returned for the second visit. The change analysis included data from 241 participants with complete nerve function and muscle power data from the first and second visits of the ancillary study. During the second visit, 1 participant refused muscle power testing, and 53 participants were unable because of physical limitations. Participants with missing data for the change analysis were older (78.8y vs 76.3y, $P<.001$) but had a similar BMI and prevalence of diabetes.

List of abbreviations:

BMI body mass index
SNAP sural nerve action potential

Peripheral nerve measures

Nerve conduction was measured bilaterally on the deep peroneal motor and sural sensory nerves using an automated nerve conduction study device (NC-stat[a]),[38] which has been previously validated in healthy older adults with criterion standard nerve conduction studies (correlation coefficient >95%).[39] Participants' feet were warmed to at least 30°C if they were <30°C before testing. Parameters recorded from the peroneal motor nerve included the motor amplitude of the compound muscle action potential (in millivolts), measured from baseline to the negative peak of the compound muscle action potential waveform; the distal motor latency (in milliseconds), which is the time from the stimulus to the onset of motor activity; and the mean F-wave latency (in milliseconds), which is the mean value of the time from the stimulus to the onset of F-wave activity. Sensory nerve measures included the sural nerve action potential (SNAP) sensory amplitude (in microvolts), which is the difference between the negative and positive peak of the SNAP waveform, and the distal sensory latency (in milliseconds), which is the time from the stimulus to the negative peak of the SNAP. Light (1.4-g) and standard (10-g) monofilament sensitivity were defined as the ability to detect 3 of 4 touches at the dorsum of the great toes. Insensitivity was defined as the inability to detect 3 touches. The standard monofilament was performed only if the participant could not feel the light monofilament. Sensory nerve conduction was performed on the nondominant side. Motor nerve conduction and monofilament testing were performed on both sides unless technical difficulty occurred. Self-reported peripheral neuropathy symptoms occurring within the past 12 months included (1) numbness or tingling; (2) sudden stabbing, burning, pain, or aches; and (3) an open or persistent sore, or gangrene on either the feet or legs. All measures were repeated at the follow-up visit.

Lower extremity muscle power

Muscle power was measured using a single leg press (Nottingham Leg Extensor Power Rig[b]).[40] Participants were seated with their