Dynamic muscle quality of the plantar flexors is impaired in claudicant patients with peripheral arterial disease and associated with poorer walking endurance

Stephanie King, MSc,[a,b] Natalie Vanicek, PhD,[a,c] and Thomas D. O'Brien, PhD,[b,d] *Kingston upon Hull, Liverpool, and Bangor, United Kingdom; and Sydney, New South Wales, Australia*

Objective: Peripheral arterial disease and intermittent claudication (PAD-IC) negatively affects physical activity and function. There is evidence for plantarflexor muscle dysfunction and weakness; however, the extent to which this dysfunction can be attributed to reduced muscle size or quality, or both, is not yet known. This study investigated whether in vivo plantarflexor muscle quality during static and dynamic contractions is altered by PAD-IC and whether such changes are associated with impaired walking endurance according to initial and absolute claudication distances.

Methods: The study recruited 22 participants, consisting of 10 healthy controls and 12 claudicant patients with occlusion of the superficial femoral artery (seven unilateral and five bilateral). Muscle quality of the combined gastrocnemius muscles during static contractions was calculated by normalizing the estimated maximal potential muscle force to the physiological cross-sectional area of the lateral and medial gastrocnemius. Muscle quality during dynamic contractions of the combined plantarflexor muscles was calculated as the ratio of peak voluntary concentric plantarflexor power and the summed volume of lateral and medial gastrocnemius.

Results: Dynamic muscle quality was 24% lower in the claudicating-limb and asymptomatic-limb groups compared with controls ($P = .017$ and $P = .023$). The differences were most apparent at the highest contraction velocity ($180°/s$). Dynamic muscle quality was associated with reduced walking endurance ($R = 0.689$, $P = .006$ and $R = 0.550$, $P = .042$ for initial and absolute claudication distance, respectively). The claudicating-limb group demonstrated a trend toward reduced static muscle quality compared with controls (22%, $P = .084$). The relative contribution of the soleus muscle to plantarflexion maximum voluntary contraction was significantly higher in the claudicating-limb and asymptomatic-limb groups than in controls ($P = .012$ and $P = .018$).

Conclusions: The muscle strength of the plantarflexors in those with PAD-IC appears to be impaired at high contraction velocities. This may be explained by some reduction in gastrocnemii muscle quality and a greater reliance on the prominently type I-fibered soleus muscle. The reduced dynamic capability of the plantarflexor muscles was associated with disease severity and walking ability; therefore, efforts to improve plantarflexor power through dynamic exercise intervention are vital to maintain functional performance. (J Vasc Surg 2015;62:689-97.)

Peripheral arterial disease and intermittent claudication (PAD-IC) refers to a chronic lower limb atherosclerotic disease that primarily affects the older population.[1] The disease negatively affects functional ability,[2] physical activity

levels,[3] and quality of life.[4] The most frequent site of claudication pain is in the plantarflexor (triceps surae) muscles,[5] where there are clear signs of dysfunction such as reduced ankle power generation during gait.[6,7] However, the few previous studies directly investigating plantarflexor function are inconsistent about whether strength is diminished in claudicant individuals compared with healthy controls.[8-10] Plantarflexor strength is a strong predictor of death in men with PAD-IC[11,12]; thus, it is essential to understand the nature of any strength impairments and the underlying mechanisms so that exercise interventions may be designed accordingly.

The "strength" of a muscle group, as measured externally by a hand-held or isokinetic dynamometer, depends on numerous factors, including muscle size and quality, voluntary activation level, any resistance to intended effort from coactivation of the antagonist muscle, and length of the moment arm about which the muscle is working.[13] Muscle quality during isometric contractions (often known as specific tension or specific force) is defined as the

Table I. Participant characteristics[a]

Variables	Claudicating limb (n = 12)	Asymptomatic limb (n = 7)	Healthy control (n = 10)
Males, %	75	57	40
Age, years	65.0 (6.7)	66.1 (7.5)	61.6 (3.6)
Height, m	1.71 (0.08)	1.69 (0.10)	1.66 (0.09)
Mass, kg	81.5 (18.2)	82.3 (21.1)	72.3 (10.9)
BMI, kg/m^2	27.7 (5.1)	28.5 (4.8)	26.1 (3.7)
ABPI			
Before exercise	0.81 (0.23)	1.01 (0.16)	0.99 (0.10)
After exercise	0.55 (0.21)	0.90 (0.06)	1.00 (0.13)
Claudication distance, m			
Initial	105 (45)	N/A	N/A
Absolute	265 (136)	N/A	N/A
Hypertension, %	50	43	10
Hypercholesterolemia, %	58	71	20
Former smokers, %	58	57	30
Present smokers, %	42	43	0

ABPI, Ankle brachial pressure index; *BMI*, body mass index; *N/A*, not applicable.
[a]Data are presented as group mean (standard deviation) unless otherwise stated.

maximal potential muscle force (calculated using the above factors) normalized to the physiological cross-sectional area (PCSA).[13] Voluntary joint moments have been measured previously in PAD-IC,[8-10] but the factors that determine externally measured strength have not. Consequently, the underlying mechanisms explaining any disease-induced strength losses have not been identified, and muscle quality has not yet been quantified in claudicant patients. Thus, it remains unknown if and how any deleterious changes in muscle properties contribute to reduced functional ability. Therefore, exactly how exercise interventions should be optimally designed to improve physical function is not apparent.

Muscle quality is known to reduce with aging and disuse[14] and to increase in response to resistance training.[15] It is reasonable to assume that muscle quality would be altered in claudicant patients given that the disease primarily affects the elderly[1] and is associated with reduced physical activity.[3] Additional factors associated with PAD-IC that may further affect muscle quality include intramuscular fat infiltration,[16] which would reduce the quantity of contractile material within a given muscle, and altered fiber type composition, with contradicting findings of shifts towards more type II (fast-twitch)[17-19] and conversely to more type I fiber (slow-twitch).[20,21] Because the specific tension differs between fiber types,[22] any changes at the fiber level may affect whole-muscle quality and force-producing potential, and thus, functional strength. If isometric muscle quality is altered with PAD-IC, then it follows that the ability to use this force-producing potential during dynamic contraction would also be impaired, especially if the proportion of fast-twitch type II fibers is reduced. Any change in muscle quality might also reduce the responsiveness of muscles to exercise training, a vital component of treatment for PAD-IC.[5] This may contribute to the inconsistent effects reported after progressive resistance training in claudicant patients.[23]

The purpose of the study was to determine whether PAD-IC causes changes in the strength characteristics of the plantarflexors and in the quality of the gastrocnemii muscles. This was achieved by exploring relationships between static and dynamic measures of muscle quality and disease severity, as assessed through the ankle-brachial pressure index (ABPI), and comparing asymptomatic and symptomatic limbs of claudicant patients with those of healthy controls. To explore the effects of muscle quality on function, correlations were performed between the factors affecting muscle strength and walking endurance as quantified through initial and absolute claudication distances. Our first hypothesis was that increased disease severity would be associated with lower voluntary isometric plantarflexion moments and concentric plantarflexion powers and that this would be explained by smaller muscle size and reduced static and dynamic muscle quality. Our second hypothesis was that walking endurance would be associated with reduced maximum isometric plantarflexion moment, concentric plantarflexion power, and static and dynamic muscle quality.

METHODS

Ethical approval for the study was granted by the National Health Service Research Ethics Committee (REC reference: 11/YH/0335).

Participants

The study recruited 22 participants, consisting of 12 claudicant patients (seven unilateral, five bilateral) and 10 healthy controls (Table I). Claudicant patients were recruited via consultant referral from a local outpatient vascular clinic. Considered for inclusion were men and women aged between 55 and 80 years who were diagnosed with Rutherford grade 1 chronic limb ischemia[24] with an arterial narrowing of the superficial femoral artery. Those with extensive disease were also included; however, the primary stenosis identified using vascular imaging was located in the superficial femoral artery for all participants. Healthy controls were recruited from the local community via e-mail and word of mouth. Individuals deemed to have