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Gait deficiencies associated with peripheral artery disease are different than chronic obstructive pulmonary disease

John D. McCamley[a], Eric J. Pisciotta[a], Jennifer M. Yentes[a,*], Shane R. Wurdeman[a,b], Stephen I. Rennard[c,d], Iraklis I. Pipinos[e,f], Jason M. Johanning[e,f], Sara A. Myers[a,c]

[a] Center for Research in Human Movement Variability, University of Nebraska at Omaha, Omaha, NE, USA
[b] Hanger Inc, Houston, TX, USA
[c] Division of Pulmonary, Critical Care, Allergy and Sleep Medicine, University of Nebraska Medical Center, Omaha, NE, USA
[d] Clinical Discovery Unit, AstraZeneca, Cambridge, UK
[e] Department of Surgery, Omaha Veterans' Affairs Medical Center, Omaha, NE, USA
[f] College of Medicine, Department of Surgery, University of Nebraska Medical Center, Omaha, NE, USA

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ABSTRACT

Objective: Previous studies have indicated that patients with peripheral artery disease (PAD), display significant differences in their kinetic and kinematic gait characteristics when compared to healthy, aged-matched controls. The ability of patients with chronic obstructive pulmonary disease (COPD) to ambulate is also limited. These limitations are likely due to pathology-driven muscle morphology and physiology alterations establish in PAD and COP, respectively. Gait changes in PAD were compared to gait changes due to COPD to further understand how altered limb muscle due to disease can alter walking patterns. Both groups were independently compared to healthy controls. It was hypothesized that both patients with PAD and COPD would demonstrate similar differences in gait when compared to healthy controls.

Methods: Patients with PAD (n = 25), patients with COPD (n = 16), and healthy older control subjects (n = 25) performed five walking trials at self-selected speeds. Sagittal plane joint kinematic and kinetic group means were compared.

Results: Peak values for hip flexion angle, braking impulse, and propulsive impulse were significantly reduced in patients with symptomatic PAD compared to patients with COPD. After adjusting for walking velocity, significant reductions ($p < 0.05$) in the peak values for hip flexion angle, dorsiflexor moment, ankle power generation, propulsion force, braking impulse, and propulsive impulse were found in patients with PAD compared to healthy controls. No significant differences were observed between patients with COPD and controls.

Conclusions: The results of this study demonstrate that while gait patterns are impaired for patients with PAD, this is not apparent for patients with COPD (without PAD). PAD (without COPD) causes changes to the muscle function of the lower limbs that affects gait even when subjects walk from a fully rested state. Altered muscle function in patients with COPD does not have a similar effect.

1. Introduction

Peripheral artery disease (PAD) and chronic obstructive pulmonary disease (COPD) are diseases that produce substantial exercise limitation in the affected patients [1,2]. Patients with either disease generally present with reduced muscular strength [3–6]. Previous studies have suggested that PAD significantly alters locomotor function [7–10] whereas, functional changes are not as pronounced in patients with COPD [11,12]. The impairments arising from these pathologies at a functional level, challenges the ability of affected patients to maintain independent living. Therefore, a primary focus for patients with PAD and patients with COPD is the assessment and rehabilitation of physical function. Physical activity produces a higher metabolic energy demand that requires an increased supply of oxygenated blood to the muscles compared to resting conditions. PAD substantially affects the delivery of blood to the legs even at submaximal exercise levels [13]. The effect of COPD differs with the delivery of oxygenated blood not limiting in patients at submaximal exercise levels [14]. Whether the deficiencies arising from these diseases impair gait biomechanics in a similar manner is not known, but it is important for exercise rehabilitation

* Corresponding author at: Biomechanics Research Building, University of Nebraska at Omaha, 6160 University Drive South, Omaha, NE 68182-0860, USA.
E-mail address: samyers@unomaha.edu (S.A. Myers).

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protocols.

1.1. Peripheral artery disease

PAD is a disease characterized by atherosclerosis, which is blockage of arteries due to plaque accumulation, and causes reduced supply of oxygenated blood to peripheral tissues. The most common symptom associated with PAD is intermittent claudication, defined as ischemia induced discomfort, pain, or cramping, which causes the patient to stop walking [10]. Advanced biomechanical analyses have determined the kinetic and kinematic alterations in patients with PAD [7–10]. Prior to claudication onset, patients with PAD have a significantly decreased propulsive (anterior-posterior) and vertical components of ground reaction forces (GRF) compared to healthy controls. Patients with PAD also demonstrate functional impairments through decreased walking speed and cadence [10]. In addition, patients with PAD present with greater ankle plantarflexion angle during early stance, reduced time to peak plantarflexion, and increased time to peak dorsiflexion [10]. These kinematic changes result in an altered rollover shape of the foot, which interferes with the optimal transfer of energy that typically occurs in healthy individuals [15]. Patients with PAD have significantly reduced peak ankle power generation at push-off, hip power generation at toe off, hip power absorption in mid stance, and knee power absorption in early and late stance [7,8]. Changes to the optimal gait result in less efficient walking patterns that corroborate with insufficient oxygen delivered to the leg muscles; increasing claudication pain and decreasing quality of life overall.

1.2. Chronic obstructive pulmonary disease

In 2010, COPD was reported to be the third leading cause of death in the United States with over 137,000 reported cases [16]. COPD is characterized by progressive and persistent expiratory airflow limitations associated with chronic inflammation of the airway [17]. COPD limits ventilation causing dynamic hyperinflation when expiratory time is insufficient to permit lung emptying [18]. This can occur during physical activity and imposes constraints on tidal volume, and leads to dyspnea [12,19]. Patients with COPD may experience exacerbations, or acute instances of disease worsening [20]. One-third of patients with severe COPD experience 15 min or less of physical activity each day [21]. Studies have indicated that slow-twitch oxidative (type-1) muscle fibers decrease in favor of fast-twitch (type-2) anaerobic muscle fibers in this population [22]. This shift suggests decreased endurance during physical activity in patients with COPD. The severity of COPD symptoms is correlated with gait abnormalities [11], with severe COPD linked to slower walking speeds [23,24], reduced cadence [25], reduced step length, increased double support time [26] and more altered step time and width variability [27]. These may be associated with balance deficiencies detected for patients with COPD [28]. Muscular deficiencies have also been observed with a reduction in push-off force following a no-rest condition [5,29], and patients with COPD found to have weaker dorsiflexor and plantar flexor muscles and greater fatigability of distal leg muscles [4].

The mechanisms of impairment differ between the pathologies. In PAD, physical activity induced ischemia restricts blood supply. In COPD, airflow is restricted due to altered lung structure. Our group and others have previously demonstrated that oxygen delivery was not the only factor limiting function in PAD patients, but that mitochondrial dysfunction restricts the efficient use of the already limited nutrients and oxygen further lowering the energy levels of pathologic muscle [13,30–33]. Patients with COPD also exhibit altered muscle mitochondria function with evidence of decreased mitochondrial density and biogenesis, impaired mitochondrial respiration, and increased mitochondrial production of reactive oxygen species in biopsies of the vastus lateralis [34,35].

Limitations in blood supply to the muscles can be reversed by surgery for patients with PAD, however these interventions do not enable patients to return to the activity levels of healthy subjects [36,37]. Medication can be used to improve the endurance in patients with COPD who do not develop muscle fatigue during exercise, but may not be effective when contractile fatigue is present [38]. It is important to understand how the different disease mechanisms, alter the ability of lower limb muscles to contribute to efficient gait. Currently, there are no guidelines on whether exercise intervention should be prescribed to patients with COPD and if so, what those exercise programs should entail. Those programs that do exist for patients with PAD [39] are not standardized and the effectiveness may vary. The understanding of how disease mechanisms in PAD and COPD alter lower limb function is useful information in determining how to mitigate the effect of these diseases [40,41]. It can potentially facilitate the design of specific interventions or recommendations for supervised exercise programs aimed to restore gait and independence to patients with PAD and patients with COPD.

The aim of this research was to differentiate the functional alterations associated with each disease by investigating changes in gait kinetics and kinematics associated with PAD prior to the onset of pain and compare them to the changes in gait of patients with COPD while at rest. We hypothesized that after accounting for reduced walking velocity, both PAD and COPD patient groups would have similarly altered kinematic and kinetic gait characteristics compared to healthy individuals during a rested condition.

2. Methods

2.1. Participants

Three groups of subjects were recruited for the analysis: 1) patients with bilateral PAD (fontaine stage II), 2) patients with COPD, and 3) healthy elderly control subjects. The University's Institutional Review Board and the Institutional Review Board at the Omaha VA Medical Center approved all study procedures. Informed consent was obtained for each individual involved in the study. All subjects were able to understand instructions and independently perform the required experimental tasks, such as walking on a treadmill.

Twenty-five patients with PAD were recruited from the vascular surgery clinics of the Veterans' Affairs Medical Center of Nebraska and Western Iowa, and the University of Nebraska Medical Center in Omaha. Patients were screened and evaluated by two board-certified vascular surgeons. The screening procedure included a detailed medical history, physical examination, computerized tomographic angiography, hemodynamic assessment and direct evaluation and observational analysis of walking impairments. Ankle-brachial index levels below 0.9 and symptomatic claudication were inclusion criteria. Patients were excluded from participating in the study if they had gait deficiencies caused by comorbidities, such as cardiac, pulmonary, neuromuscular, or musculoskeletal disease. Additionally, patients with PAD who experienced pain or discomfort during walking for reasons other than claudication pain such as arthritis, low back pain, peripheral neuropathy, or musculoskeletal pain were excluded from this study.

Sixteen patients with COPD were recruited from the Pulmonary Clinical Studies Unit at the University of Nebraska Medical Center and the general clinics of the Veterans' Affairs Medical Center of Nebraska and Western Iowa to participate in the study. All patients with COPD were screened by a board-certified nurse practitioner. The patients were diagnosed with COPD by a combination of history, clinical exam, and lung function testing. All patients had a measured FEV1/FVC (represents the proportion of a person's vital capacity that they are able to expire in the first second of forced expiration) less than 0.7 [42]. Patients were free from other co-morbidities that affect gait including musculoskeletal problems, PAD (all patients had an ankle-brachial index greater than 0.9), or neurologic disorders.

Twenty-five, age, height, and body mass matched, healthy