INTRODUCTION

Peripheral Arterial Disease (PAD) is a manifestation of atherosclerosis of the leg arteries that affects 10 million people in USA (Antignani, 2003). For these people, walking is a difficult task because the increased metabolic demands of the leg muscles are constrained due to the decreased blood flow. The result is claudication, defined as pain of the leg muscles during ambulation and is present in 40% of all PAD patients (Schainfeld, 2001). With rest, adequate blood flow eventually returns and the pain subsides. Claudication symptoms are treated with behavioral modifications, surgery and pharmacologically (Shainfeld, 2001). The two most prevalent pharmacological therapies use different approaches; the first (Treatment 1; T1) acts primarily by decreasing blood viscosity, while the second (Treatment 2; T2) acts primarily as a vasodilator (Dawson, 2001). Recently, research has examined PAD and the associated claudication as a primary gait disability (Gardner et al., 2001). However, this research has included only temporal and spatial parameters, such as stride length and step time, without exploring joint kinematics and kinetics. Similarly, studies investigating the effects of T1 and T2 also used the same limited approaches (Dawson et al., 2000). Thus, Gardner et al. (2001) suggested that these previous evaluations have been incomplete in their ability to describe the true gait handicap of PAD. Our goal was to further understand PAD gait and the influence of pharmacological therapies on the elimination of gait abnormalities.

METHODS

Nine PAD patients with diagnosed occlusion, which resulted in claudication, participated in the study. Five patients underwent T1 and four underwent T2. Because some patients were bilateral and some unilateral, we had nine total claudicating limbs affected by T1 and six total claudicating limbs affected by T2. Patients were asked to walk through a 10 meter walkway at self-selected normal walking pace, while ground reaction forces were collected by a Kistler force plate (600Hz). The patient performed five trials while pain free, or with no claudication (C1). The patient rested between each trial to ensure pain free data collection. Patients then walked on a treadmill at a 10% grade and at a speed of 0.67 m/s until the onset of pain. This protocol is common in PAD clinical examination. After pain was induced (C2), patients completed five more trials. Data collection was performed before (PRE) and after (POST) the use of each treatment. Treatment lasted a minimum of three months. Statistical analysis using 2x2 ANOVAs were performed on selected ground reaction force parameters.

RESULTS AND DISCUSSION

Regarding T1, the local minimum of vertical force experienced at midstance (Fzmin) was significantly decreased from PRE to POST. This indicates a higher positioning of the center of gravity and possibly a straighter leg at single support during the POST test. The braking impulse (IB) was significantly increased from PRE to POST indicating an
increase in forward momentum after touchdown. No other main effects were found for PRE to POST for T1. The second active loading vertical peak (F2) significantly decreased from C1 to C2 which could illustrate a tendency to better distribute loading between the legs during terminal stance when pain is present. Fzmin significantly increased from C1 to C2, indicating a lower positioning of the center of gravity and possibly a more flexed leg at single support when the patient was walking with claudication. The maximum propulsion force was significantly increased from C1 to C2 in the anterior-posterior direction which could be an adaptive mechanism employed to help carry the leg into swing when pain is present. There was a significant interaction for IB where the braking impulse increased significantly more for C2 than C1 from the PRE to the POST test. This possibly suggests an improvement in the patients’ gait under claudication after T1.

Regarding T2, the peak braking force significantly increased from PRE to POST which indicates that after treatment, patients were able to increase the braking force as necessary and possibly control forward momentum. The increased blood flow to the leg could enable improved muscular response leading to increased forward momentum. F2 significantly decreased from C1 to C2, which is similar to the effect seen as a result of T1 and could illustrate a tendency to distribute the vertical force during terminal stance when pain is present. The first active loading vertical peak force showed a significant interaction, where from PRE to POST, an increase was observed for the pain condition while a decrease was observed for the pain free condition. Thus, it seems that T2 promoted a change in this parameter. It is possible that during the PRE test, the patient was trying to adapt to the decreased blood flow and increased loading by possibly lowering the center of gravity with increased step distance during the first double support. However after the introduction of the vasodilator, the patient promoted a higher positioning of the center of gravity during claudication. Finally, the braking impulse showed significant interaction, which was similar with T1.

SUMMARY/CONCLUSIONS

The above results indicated that T1 affected a larger number of kinetic gait parameters than T2. The similar results found between the two treatments for F2 and IB indicated that their effects are reproducible for at least these two variables. However, the number of differences found in other variables indicated that the two treatment mechanisms could result in differential effects on kinetic gait parameters on PAD patients. These preliminary findings need to be substantiated with larger samples sizes, and examination of the corresponding kinematic and joint moment data.

REFERENCES