MECHANICS AND ENERGETICS OF POST-STROKE WALKING: TOWARDS A MUSCLE-LEVEL UNDERSTANDING

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INTRODUCTION

Many stroke survivors have an asymmetrical walking gait that causes altered lower-limb joint mechanics and elevated metabolic energy consumption. By combining center-of-mass (COM) level mechanical analyses with measurements of oxygen consumption, Detrembleur et al. showed that increased total positive mechanical work done by the muscles closely mirrored increased metabolic energy consumption in post-stroke walkers [1]. More recently, Jonkers et al. used inverse dynamics analyses to examine how COM energy is broken down across individual joints during hemiparetic walking. They showed a decrease in positive mechanical work performed by the paretic ankle and a saturation of positive mechanical work done by the paretic hip but did not relate these changes to differences in metabolic cost [2]. It is possible that due to differences in muscle-tendon architecture (i.e. tendon compliance) of the ankle versus hip muscle-tendon units (MTUs), the elevated metabolic cost of asymmetric walking could be explained by a shift in mechanical work from more efficient ankle MTUs to less efficient hip MTUs [3].

Surprisingly, few studies have linked differences in metabolic cost with changes in positive mechanical work performed by the joints (ankle+knee+hip). We hypothesize that when comparing post-stroke walkers to healthy controls at the same speed: (1) the summed total joint positive mechanical work of the paretic plus non-paretic limbs will be higher than in control limbs (2) there will be a shift in positive mechanical work from the ankle joint to the hip joint in the stroke limbs and (3) these mechanical differences will be reflected by an increase in metabolic cost. Furthermore due to weak, uncoordinated force generation, we expect altered length change patterns of medial gastrocnemius (MG) fascicles in stroke versus control muscles during walking.

METHODS

Thus far, we have recruited one subject with post-stroke hemiparesis (mass=89kg) and one healthy control (mass=90kg). During treadmill walking at 0.75 m/s, we collected kinematic (Vicon Inc. motion capture, 120 Hz), kinetic (Bertec Inc. instrumented treadmill, 980 Hz), real-time muscle fascicle image (Telemed Inc. ultrasound probe, 50 Hz), and oxygen and carbon dioxide flow rate (Jaeger Inc., 1/30 Hz) data. The trials lasted four minutes, which ensured steady-state metabolic measurements. We combined standard inverse dynamics (C-Motion Inc., Visual 3-D) to assess joint mechanical performance and indirect calorimetry to assess metabolic energy expenditure. We calculated the average positive mechanical power (W/kg) for the ankle, knee, and hip joints over a walking stride for both paretic and non-paretic limbs, as well as for both legs in the healthy control. We summed the average powers of the individual joints of each leg to yield total average power (W/kg) performed by the lower-limb joints on the COM. To calculate the net metabolic power (W/kg), we converted oxygen consumption and carbon dioxide production flow rates using standard physiology equations. To examine the length trajectory of the MG during walking, we tracked the origin and insertion points of a single fascicle, frame by frame, in the recorded digital image over a multiple strides.

RESULTS

As expected, we found a marked increase in net metabolic power when comparing the stroke walker to a size-matched healthy control (Figure 1). At the same walking speed (0.75 m/s), the net metabolic power doubled from 1.56 W/kg in the healthy control to 3.29 W/kg in the hemiparetic walker. The average positive joint mechanical power summed across the paretic and non-paretic joints was 76%

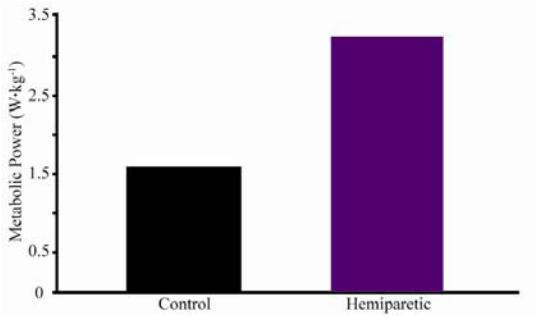

Figure 1: Bar graph showing differences in net metabolic power (W/kg) between the healthy control and the stroke walker for treadmill walking at 0.75 m/s.

higher (0.72 W/kg verses 0.41 W/kg) than the sum across the control joints (Figure 2).

Furthermore, the relative contribution of the total joint positive mechanical work within each limb was altered between control, paretic, and non-paretic limbs (Figure 2). Most notably, the hip joint positive mechanical power output of the paretic plus non-paretic limbs was more than 1.5 times higher than the hip joint mechanical power output of the healthy control limbs (0.34 W/kg versus 0.12 W/kg). Focusing only on the stroke walker, the paretic limb produced ~1/3 of the total joint average mechanical power compared to the non-paretic limb. Within the paretic limb, the hip rather than the ankle produced the majority of the mechanical power (76% vs. 13%).

Figure 3 highlights differences in MG fascicle lengthening and shortening behavior between the stroke and healthy control walkers. As expected, the control MG remained relatively isometric through most of stance before rapidly shortening during push-off (40-60% stride). The non-paretic MG initially lengthened, absorbing energy, before shortening during late-stance. The paretic MG lengthened throughout most of stance and began to shorten well after the transition into swing. These data indicate that the asymmetry in limb mechanics during post-stroke walking may manifest even at the level of the individual muscles.

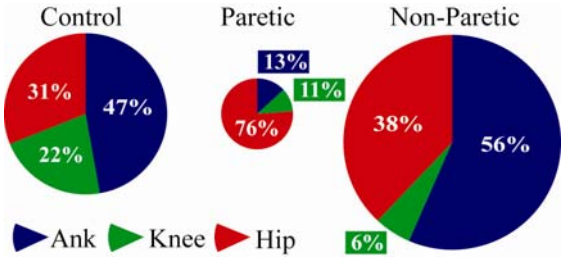

Figure 2: Pie charts showing percentage of total average positive mechanical power (W/kg) contributed by the hip, knee, and ankle during walking at 0.75 m/s. The total area of the charts is representative of the total average positive power relative to other conditions. Control pie chart is for both legs combined.

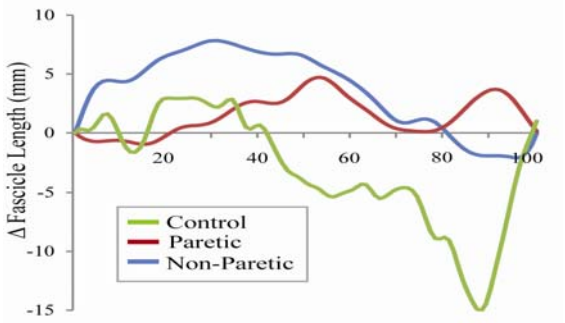

Figure 3: Length changes of MG fascicles (mm) during walking at 0.75 m/s. Length changes are shown over a stride (0% heel-strike to 100% heel-strike of same leg) and are taken relative to the recorded length at heel-strike.

DISCUSSION AND CONCLUSIONS

As hypothesized, there were increases in both joint positive mechanical work and metabolic cost for the stroke patient when compared to the healthy control. Also, there was a clear shift in mechanical power production from the ankle to the hip in both the non-paretic and paretic limbs.

These findings support the idea that elevated metabolic cost in post-stroke hemiparetic walking is due to (1) increased total workload of lower-limb joints and (2) redistribution of work to proximal MTUs. Redistribution of mechanical work from the ankle to the hip likely requires increased work done by muscle fascicle shortening rather than tendon elastic recoil. This is because hip muscles lack significant series compliant tissues that can be used to store and return elastic energy.

Furthermore, the MG length change patterns we observed in both paretic and non-paretic limbs suggest that the 'catapult mechanism' exploited during normal walking may be impaired, significantly reducing the amount of energy recycled in the Achilles' tendon and aponeurosis.

In future work, we will continue to examine the muscle-level mechanics during post-stroke walking in order to further elucidate the links between mechanics and energetics of asymmetric, hemiparetic walking.

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