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## GAIT ANALYSIS IN CHRONIC HEART FAILURE PATIENTS POINTS TO THE CALF AS THE SOURCE OF REDUCED FUNCTIONAL CAPACITY

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### SUMMARY

The aim of the present study was to analyze gait mechanics in patients affected by chronic heart failure (CHF).

Results indicate that the amount of power and work produced by this population is not different from that recorded from a healthy age-matched control group during walking. However, because of the smaller calf muscle mass in CHF patients, the similar plantar-flexion work output requires higher mass-specific work and relative effort.

### INTRODUCTION

CHF is a severe debilitating pathology in which the ability of the left ventricle to fill with or eject blood is compromised [1]. Although the syndrome initiates in cardiac muscle, there is poor correlation between CHF and cardiac output. The 'skeletal muscle hypothesis' of CHF [2] proposes instead that skeletal muscle dysfunction is more directly responsible for the reduced exercise capacity and functional intolerance present in CHF. Previous studies supporting this theory have reported altered metabolic, histological and morphological characteristics in CHF muscles [3,4]. Among these, our previous work [5] has indicated minimal muscle loss in proximal muscle, but a prominent reduction in the triceps surae mass and cross-sectional area of Achilles tendon in CHF patients.

Although clear evidence of skeletal muscle abnormalities in CHF has been identified from muscle-level analyses, it remains unclear how these changes translate to functional tasks. For example, are *in vivo* muscle mechanics altered to accommodate for the loss of muscle mass and function, and/or do muscles operate at a higher relative effort in CHF? The aim of this study was to investigate the functional effect of CHF (in terms of joint mechanical power and work production) during walking, among the most common form of daily activity.

### METHODS

CHF patients and age-matched control subjects free from other musculoskeletal disorders and lower limb musculoskeletal injuries within the previous 6 months were recruited for this study. The CHF group was composed of 8 subjects (4 men, 4 women; age: 62.0±10.4 yo; height:

1.65±0.10 m; weight: 67.2±15.9 Kg). The CHF patients belonged to the classes II-III of the New York Heart Association (NYHA) classification. The control group was composed of 10 healthy subjects recruited from the local community (8 men, 2 women; age: 64.1±4.8 yo; height: 1.74±0.06 m; weight: 70.9±8.8 Kg).

Subject's peak exercise rate of oxygen consumption (VO<sub>2</sub> peak) was assessed using an incremental walking protocol.

Subjects were asked to walk on an instrumented split-belt treadmill measuring ground reaction forces (Bertec, Columbus, OH, USA; 2000 Hz) at their preferred walking speed and at a speed 20% faster and slower than their preferred speed. The subject's kinematics was collected by means of an 8-camera VICON motion capture system (Oxford Metrics, UK; 100 Hz). 26 retro-reflective markers were placed on specific bony anatomical landmarks of the shoulders, pelvis and lower limbs; marker placement and consequent joint modeling were performed in accordance with the UWA lower body model [6].

Markers and force trajectories were filtered using a zero-lag 4<sup>th</sup> order low pass Butterworth filter with a 5-7 Hz optimal cut-off frequency that was selected using a custom residual analysis algorithm (MATLAB, The MathWorks Inc., USA).

A subject-specific scaled model composed of 23 degree of freedom was obtained from each subject's static trial from a generic musculoskeletal model [7].

Markers and forces trajectories were used to drive a simulation of the subject-specific model in Opensim 2.0.2 [8] in order to compute inverse kinematics and inverse dynamics, including net joint moments and power. Joint work was calculated by integrating joint power over discrete periods of time using the trapezoid method. Work and power were calculated for each joint and for each leg; values of the right and left limb were summed and normalized by leg lean body mass (recorded from DEXA [5]) for the hip, knee and total limb and by the triceps surae mass for ankle plantarflexion work (recorded from 3D ultrasound [5]). A minimum of five non-consecutive strides were used for analysis; data were first averaged within subjects and then between groups. A two tailed unpaired Student's t-test at a significance level of p<0.05 was used to compare difference in power and work between groups.

## RESULTS AND DISCUSSION

CHF preferred speeds were not statistically different than the control group ( $0.9 \pm 0.1$  and  $1.0 \pm 0.2$  m/s, respectively).

No differences were found in the muscle mass-specific hip, knee or total joint work between CHF and the control group, nor was the rate of performing this work during walking (overall power; work/stride time) different. The muscle mass-specific work and overall power at the ankle was, however, greater in CHF patients (52.8% and 47.9% respectively) compared to the control group at preferred speed. When normalized to body mass, no significant differences were found in work or power at any joint or in the total values from all joints combined.

These findings indicate that CHF walk without adjusting their total (body mass-specific) work or power output and with no redistribution of work between different joints. Importantly, however, because of the reduced volume of the triceps surae muscle group in the CHF [5], this population must produce significantly more plantar-flexion work for a given triceps surae muscle mass. This higher relative effort sustained by their triceps surae muscles might be one of the causes leading to an increased fatigability and reduced exercise capacity, which is a hallmark characteristic of the CHF syndrome [4]. In support of this theory, we observed a positive correlation between peak aerobic exercise capacity ( $\text{VO}_2$  peak) and ankle joint work during walking in CHF ( $r^2=0.62$ ). It remains unclear why CHF patients do not reduce their speed, and thus work output, to lower the muscle mass-specific work required of their triceps surae muscles. We are currently addressing this question by assessing whether their speed selection is made to optimize locomotor economy (energy per distance travelled).

## CONCLUSIONS

The amount of joint mechanical work and power produced by CHF patients during walking is the same as that of an age-matched healthy population. Nevertheless, considering that the triceps surae muscle size is reduced in CHF patients,

in order to sustain a similar plantar-flexion work output, the relative work produced by the triceps surae muscle mass in this population is significantly higher. Elevated mass-specific triceps surae muscle work may be one of the causes of the reduced exercise intolerance in CHF.

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## REFERENCES

1. Selig SE, et al., *Journal of Science and Medicine in Sport*.**13**:288-294, 2010.
2. Clark AL, et al., *Journal of the American College of Cardiology*. **28**:1092-1102, 1996.
3. Mancini DM, et al., *The American Journal of Cardiology*. **62**: 1234-1240, 1988.
4. Rehn TA, et al., *Heart failure reviews*. **17**: 421-436, 2012.
5. Panizzolo FA, et al., *XXIV Congress of the International Society of Biomechanics*, International Society of Biomechanics, Natal, Brazil, 2013
6. Besier TF, et al., *Journal of Biomechanics*. **36**:1159-1168, 2003.
7. Arnold EA, et al., *Annals of Biomedical Engineering*. **38**:269-279, 2010.
8. Delp SL, et al., *IEEE Transactions on Biomedical Engineering*. **54**:1940-1950, 2007.
9. Toth MJ, et al., *The American Journal of Cardiology* **79**:1267-1269, 1997.

**Table 1:** Mean and standard deviation values of positive and negative power and work in control and CHF calculated at their preferred speed

Pref. speed	Control				CHF			
	Hip	Knee	Ankle	Total	Hip	Knee	Ankle	Total
Pos. Power [W/Kg]	$2.5 \pm 1.1$	$0.7 \pm 0.4$	$1.5 \pm 0.7$	$4.8 \pm 1.9$	$2.3 \pm 0.9$	$0.9 \pm 0.6$	$1.8 \pm 0.7$	$4.7 \pm 1.0$
Pos. Work [J/Kg]	$2.9 \pm 1.1$	$0.8 \pm 0.4$	$1.7 \pm 0.7$	$5.5 \pm 1.7$	$2.9 \pm 0.9$	$1.1 \pm 0.4$	$2.3 \pm 1.0$	$5.8 \pm 1.3$

**Table 2:** Mean and standard deviation values of positive and negative power and work in control and CHF calculated at a speed 20% faster than their preferred

Pref. +20% speed	Control				CHF			
	Hip	Knee	Ankle	Total	Hip	Knee	Ankle	Total
Pos. Power [W/Kg]	$3.2 \pm 1.5$	$1.2 \pm 1.0$	$1.9 \pm 1.1$	$6.4 \pm 3.4$	$3.0 \pm 1.5$	$1.2 \pm 0.8$	$2.2 \pm 1.2$	$5.8 \pm 2.4$
Pos. Work [J/Kg]	$3.4 \pm 1.3$	$1.2 \pm 0.9$	$2.0 \pm 1.0$	$6.8 \pm 3.0$	$3.5 \pm 1.4$	$1.2 \pm 0.6$	$2.4 \pm 1.2$	$6.7 \pm 2.4$