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MUSCLE AND TENDON MORPHOLOGICAL CHARACTERISTICS IN CHRONIC HEART FAILURE PATIENTS

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SUMMARY

The aim of the present work was to investigate triceps surae and Achilles tendon morphology in patients affected by Chronic heart failure (CHF).
The reduction in volume, physiological cross sectional area (PCSA) and functional muscle lengths found in CHF might be help explain the loss of motor performance in CHF. Further studies into functional tasks will be able to better clarify the link between morphology and exercise capacity in this population.

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, the 'skeletal muscle hypothesis' of CHF [2] proposes that skeletal muscle dysfunction is more directly responsible for the reduced exercise capacity and functional intolerance present in CHF patients.
While metabolic and biochemical adaptations in CHF skeletal muscle have been previously investigated [2], much less information exists on higher-level morphological and architectural adaptations in whole muscle in humans. This gap between lower-level properties of the muscle and whole-body exercise performance is important given that whole-muscle architecture is a strong predictor of skeletal muscle functional capacities [3].
The aim of the present study was, therefore, to assess the muscle morphology of triceps surae (calf muscles) and Achilles tendon in patients with CHF compared to healthy age- and exercise-matched control participants. The triceps surae (soleus, medial and lateral gastrocnemius) was chosen as a muscle group of choice because of its accessibility to imaging and because the plantar flexor muscles represent the main source of power during walking [4] and are thus among the most important muscles for relating muscle morphology to exercise capacity.

METHODS

Chronic heart failure patients and age- and exercise-matched control subjects free from musculoskeletal disorders and lower limb musculoskeletal injuries within the previous 6 months were recruited for this study. The CHF group was composed by 10 subjects (6 men, 4 women; age: 62.0 ± 10.6 yo; height: 1.67 ± 0.10 m; weight: 69.7 ± 15.6 Kg). The CHF patients were in class II-III of the New York Heart Association (NYHA) classification. The control group was composed of 15 healthy subjects recruited from the local community (9 men, 6 women; age: 60.7 ± 6.2 yo; height: 1.72 ± 0.07 m; weight: 69.9 ± 8.6 Kg).
Muscle volume was computed using a three-dimensional (3D) ultrasound technique [5] based on a combination of B-mode ultrasound imaging and 3D motion data (Fig. 1). Briefly, ultrasound images (Telemed, Sonoblaster128, Lithuania) and 3D markers trajectories (Vicon MX, OxfordMetrics, UK) of a probe-mounted marker cluster that defined the probe position in space were collected to calculate muscle volume while the foot was kept at a neutral angle (0°) within a water bath. Manual segmentation of the muscles was then performed in Stradwin 4.4 [6] to calculate muscle volume (Figure 1). Ultrasound measurements of fascicle lengths and pennation angles, were taken across the ankle range of motion. Fascicle length and pennation angle were calculated in ImageJ [7]. Fascicle length was defined as the distance between the superior and inferior aponeurosis along the collagenous structures visible on the selected image. Pennation angle (θ) was defined as the angle between the inferior aponeurosis and the muscle fascicle (as described above) for soleus, and as the angle between the superior aponeurosis and the fascicle for medial and lateral gastrocnemius. Physiological Cross Sectional Area (PCSA) was calculated as (muscle volume x cos(θ)) / fascicle length for each muscle under investigation.

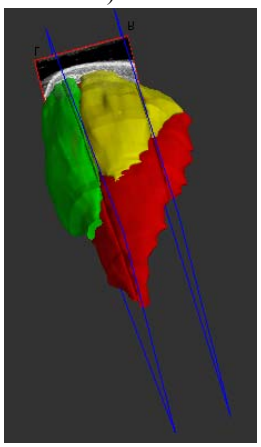

Figure 1: 3D rendering of triceps surae volume realized in Stradwin 4.4. Soleus (red), MG (green) and LG (yellow)

A scaled subject specific musculoskeletal model in OpenSim 2.0.2 [8] was used to obtain predictions of muscle-tendon unit lengths.
Tendon length was calculated as the difference between the muscle-tendon unit length of the soleus muscle predicted by the OpenSim model and the measured soleus muscle length from ultrasound (fascicle length x cos(θ)).
Tendon cross-section area (CSA) was determined by manual segmentation of ultrasound images taken in the cross section plane at the level of the medial malleoli.
A one-tailed unpaired Student's t-test at a significance level of $p<0.05$ was used to compare all the morphological parameters described above.

RESULTS AND DISCUSSION

Muscle volume of soleus and medial gastrocnemius were significantly smaller (~25%) in CHF group compared to the control group, while only a trend for a reduction in muscle volume was found for the lateral gastrocnemius. No differences in fascicles length or pennation angle were found at a neutral ankle angle (0°) in any muscle,. The PCSA of the medial gastrocnemius and soleus was sigificantly lower in CHF. Muscle parameters are presented in Table 1
Because PCSA is directly proportional to the maximum isometric force generated by the muscle, the ability to produce force in the two main plantaflexors may be substantially reduced in CHF. The capacity to generate triceps surae muscle work in CHF may be affected not only by a reduction in muscle volume, but also in a reduction in the soleus muscle's functional fascicle lengths.
Differences only in Achilles tendon CSA were found between groups (Table 2). A change in CSA but not in length, for a given value of Young's modulus, seems to indicate the presence of a more compliant tendon in CHF. These findings suggest that the reduced volume and PCSA have a remodeling action on the tendon that adapts itself to transmit lower loads. Nevertheless, it still remains possible that a remodeling of the tendon occurs in the material properties of the tendon collagen itself. Only further studies on material properties of the Achilles tendon in CHF will clarify this hypothesis.

CONCLUSIONS

This study showed a reduction in muscle volume and PCSA in patients affected by CHF. Because fiber length is not affected (at a neutral ankle angle) indicates the reduced muscle size results from a loss of PCSA only. Together, the reduction in muscle volume and PCSA can help explain the functional loss of motor performance in CHF. Future work will focus on the production of joint torque and power development in the CHF population to further investigate how these morphological properties impact on functional tasks.

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Table 1: Mean and standard deviation values of the muscle parameters investigated. * Significant differences ($p<0.05$)

	Control			CHF		
	Soleus	**MG**	**LG**	**Soleus**	**MG**	**LG**
Volume[ml]	380.3 ± 105.9	160.7 ± 39.9	91.1 ± 28.1	283.5 ± 104.3*	119.8 ± 38.3*	78.1 ± 28.7
Fascicle length[mm]	42.7±10.0	35.2±10.7	37.8±8.2	37.6±14.1	38.4±10.6	37.7±10.1
Pennation angle[°]	22.7±5.1	25.9±7.9	22.0±8.9	20.8±3.5	29.5±6.4	22.0±6.7
PCSA[cm^2]	87.5±18.0	38.6±7.7	23.5±7.7	68.4±19.2*	28.6±5.7*	19.5±6.6

Table 2: Mean and standard deviation values of tendon length and cross-section area. *Significant differences ($p<0.05$)

	Control	**CHF**
Length[cm]	28.0 ± 1.5	27.4 ± 2.9
CSA[mm^2]	73.4 ± 19.6	59.2 ± 16.1*