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# Impact of Heart Transplantation on the Recovery of Peripheral and Respiratory Muscle Mass and Strength in Patients With Chronic Heart Failure

Lenise C. B. C. Fernandes, Physio, PhD,<sup>1</sup> Italo M. de Oliveira, MD, PhD,<sup>2</sup> Paula Frassinetti C. B. C. Fernandes, MD, PhD,<sup>3</sup> João David de Souza Neto, MD, PhD,<sup>2</sup> Maria do Socorro Q. Farias, Physio, MS,<sup>2</sup> Nilce A. de Freitas, Physio, MS,<sup>4</sup> Nasser C. Magalhães, MD,<sup>3</sup> and Fernando Bacal, MD, PhD<sup>1</sup>

**Background.** To assess the impact of heart transplantation (HT) on the recovery of peripheral and respiratory muscle mass and strength in patients with congestive heart failure. **Methods.** The study included 23 patients with an indication for HT (patients in the waiting list [WL] group). These patients were monitored for 1.5 to 3 years after HT; 8 died before 6 months of follow-up, 15 patients completed the early follow-up period of 6 months after HT (FU6m group), 4 died between 6 months and 3 years after HT, and 11 patients completed the late follow-up period 1.5 to 3 years after HT (FU1.5-3y group). Twenty-three healthy subjects were included in the control group. The study variables included inspiratory muscle strength, expressed as the maximum inspiratory pressure (MIP); expiratory muscle strength, expressed as the maximum expiratory pressure (MEP); peripheral muscle strength, expressed as bilateral handgrip strength (bHGS); and the cross-sectional area of the bilateral psoas major muscle (CSAbPm). **Results.** The results showed a reduction in the CSAbPm ( $1238.9 \pm 312.3 \text{ mm}^2$ ), a reduction in the bHGS ( $27.0 \pm 5.7 \text{ kg/f}$ ), a reduction in the MIP ( $60.2 \pm 29.8 \text{ cmH}_2\text{O}$ ), and a reduction in the MEP ( $75.2 \pm 33.4 \text{ cmH}_2\text{O}$ ) in patients in the WL group compared with the healthy controls. In the time series comparison, for patients in the WL, FU6m, and FU1.5-3y groups, increases were found in the CSAbPm ( $1305.4$  vs  $1458.1$  vs  $1431.3 \text{ mm}^2$ , respectively), bHGS ( $27.3$  vs  $30.2$  vs  $34.7 \text{ kg/f}$ , respectively), MIP ( $59.5$  vs  $85.5$  vs  $90.9 \text{ cmH}_2\text{O}$ , respectively), and MEP ( $79.5$  vs  $93.2$  vs  $101.8 \text{ cmH}_2\text{O}$ , respectively) ( $P < 0.00$ ). **Conclusions.** Sarcopenia was observed in patients in the WL group. Patients recovered peripheral and respiratory muscle mass and strength at 3 years after HT.

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Heart transplantation (HT) is considered the best therapeutic modality for patients in the most advanced stage of heart failure (HF). After HT, significant improvements in the symptoms, hemodynamics, heart function, and sympathetic neural activity are observed.<sup>1</sup> However, skeletal muscle

abnormalities have been suggested to remain for months after HT and may contribute to the impaired exercise capacity observed in these patients.<sup>1</sup>

The loss of muscle mass in patients with HF (also known as sarcopenia, muscular atrophy, or myopenia) is a poorly

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<sup>1</sup> Heart Institute University of São Paulo School of Medicine and Heart and Lung Messejana's Hospital, Fortaleza, CE, Brazil.

<sup>2</sup> Heart and Lung Messejana's Hospital, Fortaleza, CE, Brazil.

<sup>3</sup> State University of Ceará, Fortaleza, CE, Brazil.

<sup>4</sup> Fortaleza General Hospital, Fortaleza, CE, Brazil.

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L.C.B.C.F. collected the data; conceived of the design of the study; conducted the analysis and interpretation of the data, drafted the article or critically revised it for important intellectual content, provided final approval of the version of the article to be published, agrees to be accountable for all aspects of the work, ensuring that

questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. I.M.d.O. collected the data, conceived of the design of the study; conducted the analysis and interpretation of the data, critically revised the article for important intellectual content, provided final approval of the version of the article to be published, agrees to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. P.F.C.B.C.F. collected the data, conceived of the design of the study; conducted the analysis and interpretation of the data, critically revised the manuscript for important intellectual content, provided final approval of the version of the article to be published, agrees to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. J.D.d.S.N. collected the data, conducted the analysis and interpretation of the data, critically revised the manuscript for important intellectual content, provided final approval of the version of the article to be published, agrees to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. M.d.S.Q.F. collected the data, conducted the analysis and interpretation of the data, critically revised the article for important

investigated comorbidity. The first study<sup>2</sup> to evaluate the clinical prevalence and impact of muscle mass loss in patients with stable HF was published in 2013 as part of a landmark study<sup>3</sup> that investigated the presence of diabetes mellitus, cachexia, and obesity as factors that aggravated HF. The study showed the presence of muscle loss in 19.5% of the patients and concluded that muscle mass loss was a frequent comorbidity in HF.<sup>2</sup>

Cachexia is a serious complication that may be present in patients with chronic diseases. Similar to sarcopenia, cachexia presents a loss of muscular mass during the evolution of the disease. Cardiac cachexia is a serious complication that is associated with a poor prognosis, which increases the morbidity and mortality of patients.<sup>4</sup>

In view of these findings, the importance of the topic, and the lack of studies in this area, we assessed the impact of HT on the recovery of the peripheral and respiratory muscle mass and strength in HF patients.

## CASUISTIC AND METHODS

This study was an observational, prospective, cohort study with a control group (CG) that was performed at a teaching hospital specializing in heart and lung diseases in the state of Ceará, Brazil.

The Research Ethics Committee of Messejana's Heart and Lung Hospital approved the present investigation under number 823/11, and all subjects provided written informed consent.

Forty patients 18 years or older were selected during the period from August 2011 to March 2013. Patients on the waiting list (WL) for HT and healthy subjects without heart disease participated in the study. Fifteen of the patients on the WL for HT were excluded for the following reasons: motor sequelae in the upper and lower limbs, hemodynamic instability, clinical improvement, and refusal to participate. Therefore, 25 patients were eligible for the study. One subject died while on the WL for HT. Twenty-four subjects underwent the transplant surgery, although 1 subject did not complete the second assessment due to technical problems. Ultimately, a total of 23 patients were investigated before HT and followed-up for 3 years after surgery.

We examined the data of patients on the WL before transplantation (WL group), during the early follow-up period performed 6 months after HT (FU6m group), and during the late follow-up period conducted from 1.5 to 3 years after transplantation (FU1.5-3y group).

The CG comprised healthy subjects without heart disease who were matched for age, sex, weight, and height and were employees of the participating hospital. The participants in this group underwent an interview to obtain data concerning hypertension, diabetes, smoking habits, and use of medication, which was followed by the selection of subjects for inclusion in the CG (n = 23).

After HT, 8 patients died before reaching the 6-month follow-up, and 15 patients survived and completed the early follow-up period (FU6m group). Between 1.5 and 3 years after HT, 4 patients died, and 11 survived to complete the late follow-up period (FU1.5-3y group) (Figure 1).

Data were analyzed from the population, including the following categorical and continuous variables: age; sex; weight; height; HF causes; hypertension; diabetes; time on the WL; time of hospitalization after HT; measurement of inspiratory muscle strength, expressed as the maximum inspiratory pressure (MIP); expiratory muscle strength, expressed as the maximum expiratory pressure (MEP); dominant handgrip strength (dHGS); nondominant handgrip strength (ndHGS); cross-sectional area of the left psoas major muscle (CSAIPm); cross-sectional area of the right psoas major muscle (CSArPm); body mass index (BMI); and creatinine concentration. All variables were assessed in the WL group and reassessed in the same patients during follow-up after transplantation. One observer technician who specialized in radiology performed the cross-sectional area of the bilateral psoas major muscle (CSAbPm) measurements, and all other parameters were measured by a single observer (the author of this paper: LCBCF) to minimize measurement bias.

We used mass and muscle strength measurements in the present study as criteria for the diagnosis of sarcopenia according to the recommendations of the European Consensus on Sarcopenia.<sup>5</sup>

Notably, the patients in the study did not undergo any heart rehabilitation program after HT.

### Assessment of Handgrip Strength

Peripheral muscle strength was measured through handgrip strength (HGS) using the Jamar digital dynamometer (Sammons Preston, Mississauga, ON, Canada) according to the protocol of Moreira et al<sup>6</sup>

### Assessment of Respiratory Muscle Strength

Inspiratory muscle strength, expressed as MIP, and expiratory muscle strength, expressed as MEP, were measured using an analog CRK model manovacuumeter. The patient was seated with the trunk straight to form a 90° angle with

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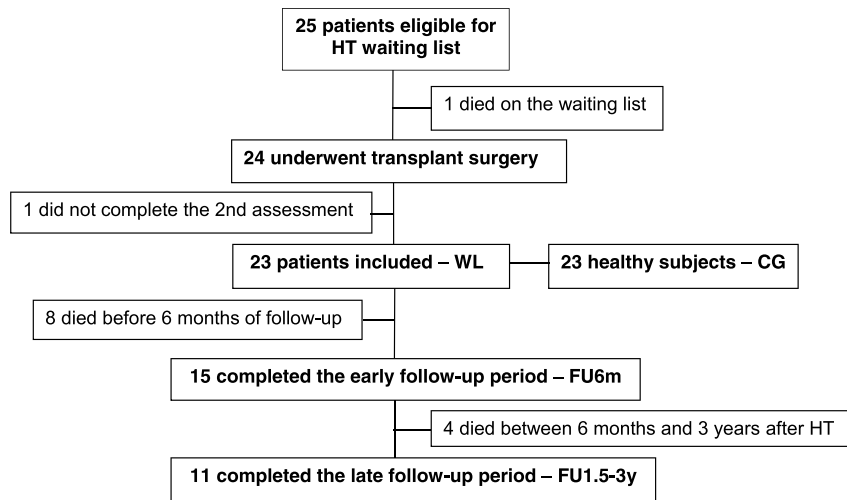
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Correspondence: Lenise Castelo Branco Camurça Fernandes, Rua Silva Paulet, 2020, Apt 1401, Aldeota, Fortaleza, Brazil. (lenisefernandes@gmail.com).

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**FIGURE 1.** Flowchart of the study population.

the thighs and feet supported on the ground.<sup>7</sup> A nasal clamp was used during the measurements, and a nozzle was applied as an interface. The MIP was measured at functional residual capacity (ie, the patient performed normal expiration followed by maximum inspiration). The MEP was measured at total lung capacity (ie, the patient performed a maximum inspiration followed by a maximum expiration).<sup>7</sup>

### Assessment of Muscle Mass

Pelvic computed tomography (CT) was used to assess the muscle mass by quantifying the CSA<sub>AbPm</sub> at the fourth lumbar vertebra level (L4).<sup>8</sup> The CT scanner used was the Multi Slice BrightSpeed Elite 16 Channels General Electric.

The patient was positioned supine on the CT scanner table with semi-inflected knees to minimize the lordotic curvature.<sup>9-10</sup> The point delineated to analyze the psoas major muscle measurement in all patients was the upper edge level of L4.<sup>8</sup> The resulting area of the outlined regions (right and left) was calculated to determine the CSA<sub>AbPm</sub>. Measurement are shown in mm<sup>2</sup>.

### Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences software version 20.0.

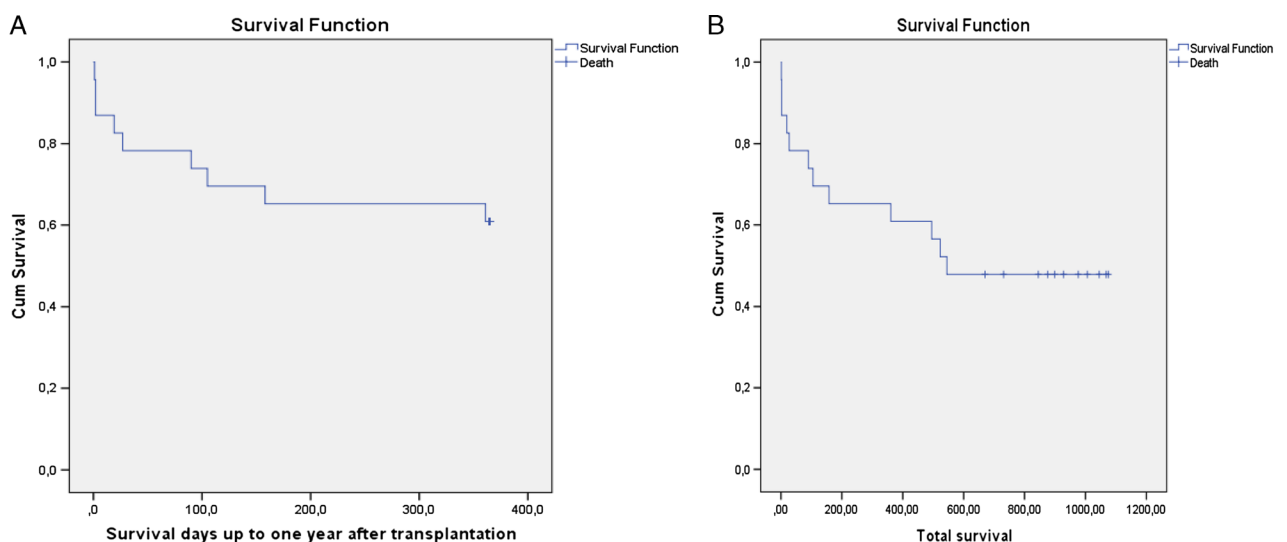
In the univariate descriptive analysis, the frequency distribution test was used for categorical variables, and measurements of central tendency and dispersion were used for numerical variables.

Pearson's (r) correlation test was used to analyze the correlations between the muscle mass and the peripheral and respiratory muscle strength variables.

Generalized linear models (ANOVA) were used to analyze the muscle mass and strength variables at the 3 time points of the study (before HT and at 6 months and 1.5-3 years of follow-up). Differences were considered significant at  $P \leq 0.05$ .

### RESULTS

We observed survival rates of 60.9% at 1 year after HT and 47.8% at the end of the study after a mean follow-up period of 2 years for the 23 patients included in this study (ie, 11 patients were alive with functioning heart grafts) (Figures 2A and B).



**FIGURE 2.** Kaplan-Meier analysis of the survival rate 1 year after transplantation (60.9%, A) and at the end of the study (47.8%, B), with a mean follow-up of 3 years.

**TABLE 1.**  
Baseline characteristics of mass measurements and muscle strength among surviving and deceased patients

Variables	Deceased (n = 12)	Surviving (n = 11)	P
	Mean ± SD	Mean ± SD	
CSAbPm, mm <sup>2</sup>	1185.12 ± 340.98	1313.33 ± 280.68	0.33
bHGS, kg/f	26.69 ± 6.59	27.29 ± 4.87	0.80
MIP, cmH <sub>2</sub> O	60.83 ± 38.36	59.55 ± 18.36	0.80
MEP, cmH <sub>2</sub> O	71.25 ± 41.67	79.55 ± 22.30	0.55

Univariate analysis for central tendency of measures (mean) and dispersion (SD).

The causes of death included 4 deaths due to septic shock, 4 deaths from heart arrest without a specific cause, 2 deaths due to sudden death, 1 death due to biventricular dysfunction, and 1 death due to acute HF. Patients in the group who died posttransplant had no baseline differences in any of the measures of muscle mass or strength compared to posttransplant survivors (Table 1).

A total of 87% of patients were male. Nonischemic cardiomyopathy was the most common HF etiology and was observed in 65.2% of the patients, followed by ischemic cardiomyopathy in 17.4% and chagasic cardiomyopathy in 17.4% of patients. Among all patients, 39.1% were categorized as functional class III, and 60.9% categorized as functional class IV. The left ventricle ejection fraction was 27.6 ± 6.9%. The mean time on the WL for HT was 50.2 ± 42.7 days. The median hospitalization time after HT was 26 (21-37) days, and the mechanical ventilation time was less than 1 (1-1) day. Before HT, only 1 patient was diabetic, and during early follow-up after HT, another patient developed transitory diabetes. However, during the late follow-up period, only 1 patient remained diabetic. Systemic hypertension was observed in 5 patients before transplantation, in 8 patients during early follow-up, and in 7 patients during late follow-up after HT. Table 2 shows the clinical and demographic characteristics of the study patients.

Univariate analysis of the WL group compared to the CG showed that sarcopenia was present based on a decrease in the peripheral muscle mass and strength. The variables (CSAbPm, bilateral HGS [bHGS], MIP, and MEP) were similar between the CG and FU1.5-3y group, demonstrating recovery of sarcopenia at 3 years after HT (Table 3).

When the muscle mass and strength variables were compared over time (in the WL, FU6m, and FU1.5-3y groups) in the 11 surviving patients, we found an increase in muscle mass and peripheral and respiratory muscle strength during the 3 years of follow-up after transplantation (Tables 4 and 5).

In the multivariate analysis, MIP and bHGS differed significantly among the WL, FU6m, and FU1.5-3y groups.

The WL group exhibited a strong correlation between bHGS and MIP ( $P = 0.00$  and  $r = 0.63$ ) and between bHGS and MEP ( $P < 0.00$  and  $r = 0.75$ ). In the FU6m group, no correlation was found between the peripheral and respiratory muscle strength. In the FU1.5-3y group, a strong correlation was found between bHGS and MEP ( $P = 0.00$  and  $r = 0.80$ ) (Figure 3).

## DISCUSSION

In the present study, the univariate analysis revealed sarcopenia in the WL group. The sarcopenia levels had recovered in the FU6m and FU1.5-3y groups compared with those

in the WL group (Table 3). These outcomes conflict with those of other studies<sup>1,11,12</sup> that have observed intrinsic abnormalities in the skeletal muscle of patients before and after HT, thereby contributing to a lower exercise capacity after transplantation. At the end of the late follow-up (FU1.5-3y group), the heart transplant patients presented muscle mass and strength levels similar to those in the CG, confirming the recovery of muscle mass and strength in the study population (Table 3). A recent study on the fragility phenotype performed in patients after HT and after ventricular assist device implantation supports the findings of the present study.<sup>13</sup>

The results of the multivariate analysis between the WL group and CG showed a reduction of peripheral muscle strength ( $P = 0.01$ ) but not in muscle mass ( $P = 0.06$ ). We can infer some explanations for this finding, such as the patients' noncachectic profile, the use of lower doses of glucocorticoids, or the small sample size.

A rapid recovery of muscle mass and respiratory muscle strength occurred in the FU6m group, whereas peripheral muscle strength progressively recovered in the FU1.5-3y group (Table 5).

Braith et al<sup>14</sup> demonstrated that lean mass was significantly decreased at 2, 5 and 8 months after transplantation in the CG members who did not participate in the training (this group was similar to our patients who did not undergo any training). These results were in contrast to our findings, which demonstrated that muscle mass increased by 11.7% in the early follow-up (FU6m) group after transplantation, even though our patients were taking a mean dosage of 15 mg/d of glucocorticoids (prednisone).

Our results might reflect the nutritional status of our patients, who presented with an average BMI of 25.1 kg/m<sup>2</sup>, which did not correspond to clinical malnutrition or cachexia but rather revealed an overweight status according to the World Health Organization. These data are in agreement with those of several congestive HF databases,<sup>15,16</sup> indicating the so-called obesity paradox (ie, patients with congestive HF

**TABLE 2.**  
Clinical and demographic characteristics of the study patients in the WL, FU6m, and FU1.5-3y groups, 2011-2015

Variables	WL (n = 23) mean ± SD	FU6m (n = 15) mean ± SD	FU1.5-3y (n = 11) mean ± SD
Age, y	50.8 ± 13.0	51.2 ± 13.0	50.73 ± 13.1
Height, cm	1641.64 ± 100.1	1631.63 ± 100.1	1631.63 ± 100.1
Weight, kg	67.7 ± 11.2	68.9 ± 11.6	69.7 ± 10.4
BMI, kg/m <sup>2</sup>	25.1 ± 3.6	25.8 ± 3.4	26.0 ± 3.2
CSAbPm, mm <sup>2</sup>	1238.9 ± 312.3	1351.7 ± 498.8	1431.3 ± 448.1
CSArPm, mm <sup>2</sup>	1231.4 ± 319.8	1353.8 ± 511.7	1424.6 ± 446.7
CSAIPm, mm <sup>2</sup>	1246.4 ± 313.4	1349.6 ± 490.4	1437.9 ± 456.8
bHGS, kg/f	27.0 ± 5.7	29.0 ± 6.2	34.7 ± 7.7
dHGS, kg/f	28.3 ± 6.7	30.3 ± 5.9	37.1 ± 8.1
ndHGS, kg/f	25.6 ± 5.3	27.7 ± 6.8	32.3 ± 7.8
MIP, cmH <sub>2</sub> O	60.2 ± 29.8	83.7 ± 29.6	90.9 ± 20.7
MEP, cmH <sub>2</sub> O	75.2 ± 33.4	90.3 ± 30.7	101.8 ± 22.3
Creatinine CL, mg/dL	69.4 ± 27.3	70.5 ± 24.6	66.4 ± 14.9
Creatinine, mg/dL	1.27* (0.9-1.5)**	1.20* (1.07-1.40)**	1.25*(1.15-1.39)**

Univariate analysis for central tendency of measures (mean) and dispersion (SD). For asymmetric distributions, (–) the median \* (p25-p75) \*\* was used.

CL, clearance; SD, standard deviation.



**TABLE 3.**

Comparison of strength and muscle mass among patients in the WL group, FU6m and FU1.5-3y group with CG, 2011 to 2015

Variables	GC (n = 23)	WL (n = 23)	P	FU6m (n = 15)	P	FU1.5-3y (n = 11)	P
	Mean ± SD	Mean ± SD		Mean ± SD		Mean ± SD	
CSAbPm, mm <sup>2</sup>	1533.1 ± 436.6	1238.9 ± 312.3	0.01	1351.7 ± 498.8	0.39	1431.3 ± 448.1	0.12
bHGS, kg/f	38.2 ± 10.5	27.0 ± 5.7	<0.00	29.0 ± 6.2	0.30	34.7 ± 7.7	0.23
dHGS, kg/f	39.4 ± 11.7	28.3 ± 6.7	0.00	30.3 ± 5.9	0.36	37.1 ± 8.1	0.65
ndHGS, kg/f	37.0 ± 10.0	25.6 ± 5.3	<0.00	27.7 ± 6.8	0.28	32.3 ± 7.8	0.09
MIP, cmH <sub>2</sub> O	94.4 ± 29.1	60.2 ± 29.8	<0.00	83.7 ± 29.6	0.02	90.9 ± 20.7	0.86
MEP, cmH <sub>2</sub> O	102.17 ± 28.3	75.2 ± 33.4	0.00	90.3 ± 30.7	0.16	101.8 ± 22.3	0.69

Univariate analysis; All P value are compared with the CG.

and a higher BMI may have a decreased risk of death and hospitalization compared to those with a “normal” BMI).

Mancini et al<sup>17</sup> reported the presence of muscular atrophy in patients with HF. The authors demonstrated the incidence of severe muscle atrophy in 68% of patients with HF. They suggested that these anthropometric measurements of the arm could underestimate muscular atrophy due to the presence of substantial fat infiltration into the skeletal muscle detected by nuclear magnetic resonance.

Schaufelberger et al<sup>1</sup> demonstrated that intrinsic abnormalities in skeletal muscle found before HT remained 6 and 9 months after HT and might contribute to a lower exercise capacity and muscle strength in these patients, in contrast to our findings.

The mean values found for bHGS measurements in the CG patients of the present study are consistent with those from studies<sup>18,19</sup> conducted in the Brazilian population that evaluated HGS values in normal subjects.

When comparing the 3 follow-up periods, our study showed that bHGS was significantly different ( $P < 0.00$ ). bHGS increased by 10.5% during the early follow-up and by 5% during the late posttransplant period. These results are in contrast to those of studies<sup>1,20,21</sup> that demonstrated impaired skeletal muscle function years after HT.

Some authors<sup>22,23</sup> have identified muscle strength as a long-term cardiovascular risk factor and mortality index.<sup>24</sup> A cohort study<sup>25</sup> showed that decreased muscle strength was associated with increased all-cause mortality but that reduced muscle mass was not strongly associated with mortality.

The analysis of the respiratory muscle strength was showed a decrease in both MIP and MEP in the patients in the WL group. We found a significant difference compared to the CG. During the posttransplantation follow-up (especially in

the FU6m group), the MIP increased by 43.5% compared with that in the WL group, which indicated significant early recovery after cardiac transplantation.

Coronel et al<sup>26</sup> assessed patients before HT and reported mean MIP and MEP values of  $88.85 \pm 29.28$  cmH<sub>2</sub>O and  $122.7 \pm 42.02$  cmH<sub>2</sub>O, respectively. These values were higher than those determined in our study. This discrepancy may be attributable to the use of functional residual capacity because this measurement has the advantage of not being affected by the respiratory system elastic recoil.<sup>27</sup>

According to Meyer et al,<sup>28</sup> patients with HF have a significant decrease in MIP, which is potentially a consequence of a reduced muscle mass. This reduction of the muscle mass might alter the capillary density and the activity of oxidative enzymes, which could be the main factor causing diaphragm atrophy.<sup>29</sup> A decrease in MIP is associated with the worst prognosis in these patients.<sup>28</sup>

### Final Remarks

The results of this study revealed that patients with lower peripheral muscle strength before HT underwent a late recovery after HT.

The inclusion of a CG allowed us to compare rare data in the literature.

This study provided survival data through the Kaplan-Meier method, which permitted the analysis of patients with different follow-up periods.

This study has some limitations. For instance, studies of heart transplant patients and patients in the final stage of HF are difficult because these patients have high mortality rates. Therefore, the study had a small sample size (75% power) because it comprised surviving patients.

**TABLE 4.**

Measurements of peripheral and respiratory muscle strength and muscle mass of 11 surviving patients after HT, 2011 to 2015

Variables	WL (n = 11)	FU6m (n = 11)	FU1.5-3y (n = 11)	P
	Mean ± SdE	Mean ± SdE	Mean ± SdE	
CSAbPm, mm <sup>2</sup>	1305.4 ± 83.4	1458.1 ± 139.7	1431.3 ± 135.1	<0.00
bHGS, kg/f	27.3 ± 1.5	30.2 ± 2.1	34.7 ± 2.3	<0.00
dHGS, kg/f	28.4 ± 1.6	31.2 ± 2.0	37.1 ± 2.4	<0.00
ndHGS, kg/f	26.2 ± 1.4	29.2 ± 2.2	32.3 ± 2.4	<0.00
MIP, cmH <sub>2</sub> O	59.5 ± 5.5	85.5 ± 7.6	90.9 ± 6.2	<0.00
MEP, cmH <sub>2</sub> O	79.5 ± 6.7	93.2 ± 9.4	101.8 ± 6.7	<0.00

SdE, standard error.

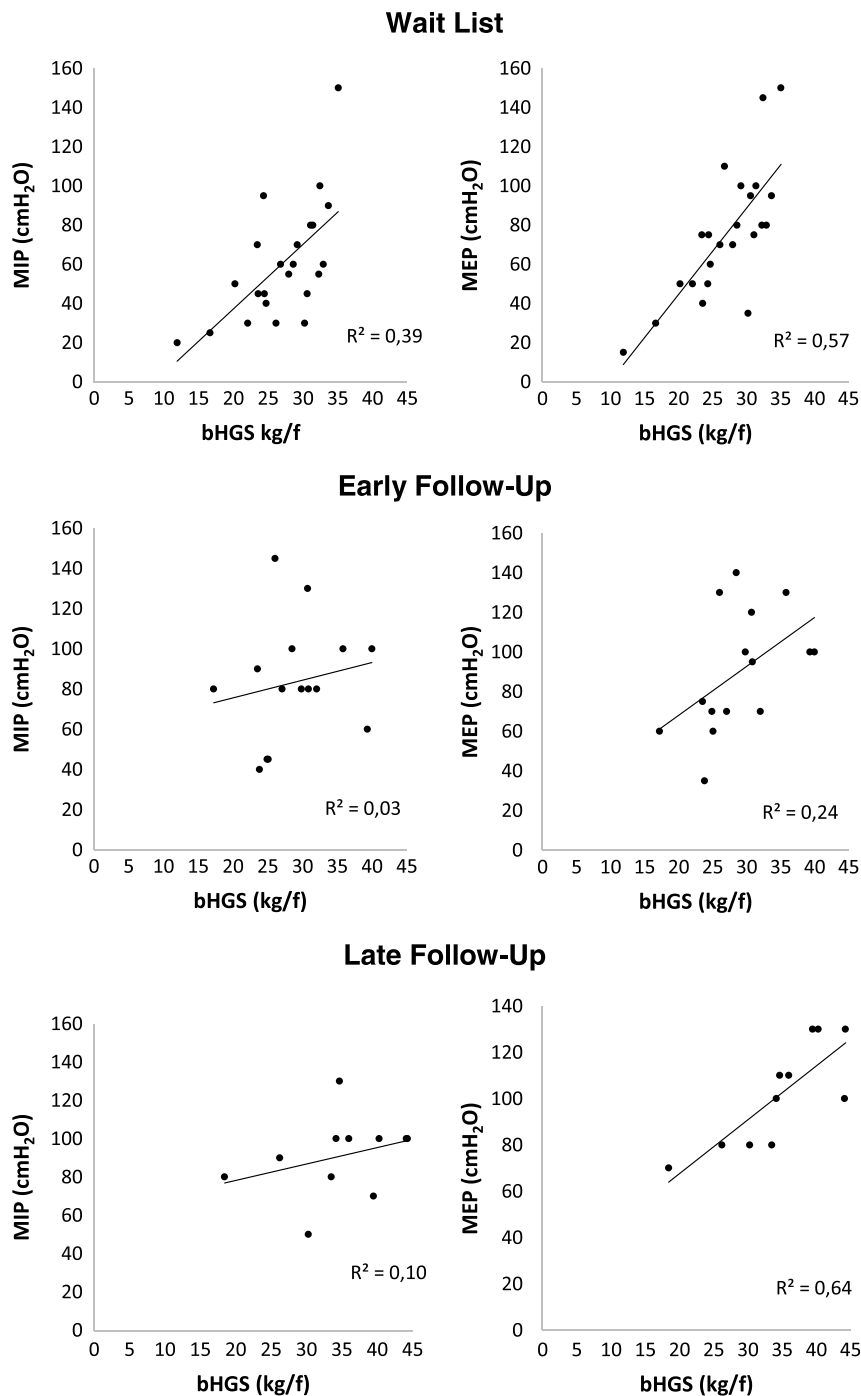
### CONCLUSIONS

The findings of this study revealed sarcopenia in patients before HT.

**TABLE 5.**

Percentage increase in muscle mass and strength over time after cardiac transplantation

	WL and FU6m, %	FU6m and FU1.5-3y, %	WL and FU1.5-3y, %
CSAbPm, mm <sup>2</sup>	11.70	-01.84	09.64
bHGS, kg/f	10.54	15.00	27.14
MIP, cmH <sub>2</sub> O	43.50	06.38	52.67
MEP, cmH <sub>2</sub> O	17.14	09.27	28.00



**FIGURE 3.** Correlation between peripheral muscle strength and inspiratory muscle strength before HT in the early and late follow-up groups, according to Pearson correlation test.

Three years after HT, the patients recovered peripheral and respiratory muscle mass and strength.

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