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Contributions of Comorbid Diabetes to Sleep Characteristics, Daytime Symptoms, and Physical Function among Patients with Stable Heart Failure

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Abstract

Background—Diabetes (DM) and heart failure (HF) are often comorbid. Sleep disturbances, poor physical functioning, and high levels of daytime symptoms are prevalent and contribute to poor quality of life in both populations. However, little is known about the independent and additive effects of comorbid DM on sleep, physical function, and daytime symptoms among patients with HF.

Objective—To investigate the extent to which comorbid DM confers independent and additive effects on sleep disturbance, physical functioning, and symptoms among patients with stable HF.

Methods—This secondary analysis was conducted on a sample of 173 stable Class II–IV HF patients. Self-report and polysomnography were used to measure sleep quality, objective sleep characteristics, and sleep disordered breathing. Physical function measures included wrist actigraphy, the six minute walk test, and the SF-36 Physical Component Summary Score. Fatigue, sleepiness, and depression were also measured. Univariate analyses and hierarchical regression models were computed.

Results—The sample included 173 ($n = 119/68\%$ HF and $n = 54/32\%$ HF plus DM) patients (age 60.4 ± 16.1 years). In analyses adjusted for age, gender, BMI, and NY Heart Association classification, HF patients with DM had longer sleep latency and spent a greater percentage of time awake after sleep onset (WASO %) than the HF patients who did not have DM (all $p < .05$). There were no statistically significant differences in respiratory disturbance index (RDI) or self-reported sleep quality. Sleep duration was low in both groups. Patients with DM had shorter 6MWT distance, lower ratio of daytime to nighttime activity, and lower general health and self-reported physical function. Hierarchical regression models revealed that age and DM were the only significant correlates of the sleep variables, while age, gender, NYHA class, and DM were all associated with 6MWT distance.

Conclusions—Comorbid DM contributes independent and additive effects on sleep disturbances and poor physical functioning in patients with stable HF.

Keywords

Actigraphy; heart failure; diabetes; sleep; symptoms; functional status

Background

Heart failure (HF) affects approximately 5.1 million adults in the United States and is closely associated with severe disability and early mortality¹ and an incidence rate of type 2 diabetes of 3.20 per 100 HF patient years.² Sleep disturbances, sleep-related symptoms, and reduced physical functioning are common in HF and contribute to poor health-related quality of life. Diabetes (DM) is a predictor of HF^{3,4} and a prevalent comorbid condition in patients with HF, with an estimated incidence 2.5 times higher than in matched, healthy controls.⁵ Patients with DM develop HF at a younger age, are sicker, and have more comorbid conditions than their non-diabetic counterparts.^{2,6}

Sleep disturbance, including poor sleep quality, reduced sleep time, fragmented sleep, and sleep disordered breathing, as well as negative sleep-related daytime consequences, are common in patients with HF,^{7,8} as well as those with DM.⁹ Therefore, patients with HF and comorbid DM are likely to have even further reductions in health-related quality of life outcomes, such as higher symptom burden, poor sleep, and reduced physical function.¹⁰

In patients without HF, glucose intolerance is closely associated with sleep disturbance, including short sleep duration, poor sleep quality, sleep fragmentation, and sleep apnea, even when analyses are adjusted for covariates such as age, gender, and body mass index (BMI).¹¹ The relationship seems to be bi-directional. Short sleep time, poor sleep quality, and sleep disordered breathing contribute to impaired glucose tolerance and diabetes in a non-diabetic population and poor glycemic control in patients with diabetes.¹²⁻¹⁵ On the other hand, DM may contribute to poor sleep through a variety of mechanisms, such as nocturnal hypoglycemia,¹⁶ and peripheral neuropathy-related pain,^{17,18} among others.

Both patients with HF and others with DM experience decrements in physical function. Impaired glucose tolerance, new-onset DM and existing DM were linearly associated with lower resting left ventricular ejection fraction and reduced contractile reserve among people with both DM and HF, compared with non-diabetic HF patients. Distance walked on the six-minute walk test (6MWT) was shorter,¹⁹ and Minnesota Living With Heart Failure Questionnaire total scores, the physical function subscale, and the SF-12 physical function scale were all lower among HF patients with comorbid DM than those without.²⁰ In 2,013 patients with stable coronary heart disease (including HF), those with DM had higher levels of symptom distress and lower scores on the SF-36 Physical Component Summary score than those without DM.¹⁰ Taken together, these findings suggest the likely additive influence of DM on physical function, symptoms, and sleep characteristics among patients with HF. However, no studies have previously addressed the possible contributions of DM to polysomnographic measures of sleep among patients with HF.

Objective

The purposes of this study were to examine: (1) the differences in self-reported and objectively measured sleep disturbance (sleep quality, duration, and continuity) between stable HF patients with and without comorbid DM; (2) differences in self-reported daytime symptoms (depression, fatigue, daytime sleepiness) and objective and self-reported physical function (six minute walk test, daytime activity, daytime to nighttime activity ratio, SF-36 general health and physical function scales) between stable HF patients with and without comorbid DM; and (3) whether the presence of comorbid DM explained a unique proportion of the variance in sleep disturbance characteristics, physical functioning, and symptoms among patients with stable HF.

Methods

Study Design

This descriptive study employed secondary analysis of data obtained from an observational study of sleep, sleep disorders, and functional performance among patients with stable HF. Full details of the study methods have been previously reported.^{7,8,21} Details are repeated here as relevant.

Sample

The sample included participants with stable HF recruited from five specialized HF disease management programs in the Northeastern United States. Participants were considered to have stable HF if they had no hospital admissions or emergency department visits within the prior month and had no change in vasoactive medications within the prior two weeks. Participants were excluded if they had cognitive impairment, end-stage renal disease, neurologic diseases or any musculoskeletal conditions affecting mobility of their non-dominant arm (due to use of a wrist-worn actigraph to measure activity levels).

Institutional review board approval was obtained, and participants provided written informed consent. Medical records were reviewed, and participants completed a six-minute walk test (6MWT) in the clinic setting. Participants underwent a one-night unattended ambulatory polysomnographic (PSG) study at home and completed a packet of questionnaires to evaluate self-reported sleep quality, daytime symptoms (excessive daytime sleepiness, depression, fatigue, and self-reported functional performance). They wore a wrist actigraph continuously for 4 days for measurement of physical activity and sleep under free-living conditions and completed sleep/symptom diaries. They were paid \$50.00.

Variables and Measures

Sleep disturbance—Both objective and subjective measures of sleep were obtained in order to elicit its biobehavioral characteristics. Sleep was objectively measured using unattended full home polysomnography (PSG) for one night in participants' homes with the Sapiro sleep recorder (Compumedics, Inc., Charlotte, NC). Full polysomnography includes electro-encephalography, electro-oculography, chin electron myography (all used to determine sleep stages, latency, duration, and time awake); electro-cardiography; and

respiratory effort, respiratory airflow, and pulse oximetry (used to score sleep disordered breathing). Home PSG monitoring is more ecologically valid than in-lab testing because the patient sleeps in her or his own bed; further, it was preferred by patients when compared to laboratory-based PSG testing and is a valid and reliable alternative to lab-based PSG monitoring.²²

PSG measures were computed using standard methods as previously reported in detail.⁸ Briefly, PSG data were downloaded and manually scored on a high-resolution monitor, using 30-second epochs for sleep stages and 3-minute epochs for respiratory and leg movement data. PSG variables used in the analyses included: total sleep time during the major sleep period; sleep latency (the length of time from lights out to sleep onset); and the percentage of wake after sleep onset (WASO [%]), a measure of sleep continuity. Respiratory-related sleep variables included the Respiratory Disturbance Index (RDI: total of apneas, hypopneas, and respiratory-event related arousals/hour of sleep); mean oxygen saturation during sleep; and the length of time with an oxygen saturation < 90% during sleep (measures of sleep disordered breathing).

Self-reported sleep quality was measured with the Pittsburgh Sleep Quality Index (PSQI),²³ a 19-item instrument that measures perceived sleep quality with a Global Sleep Quality Score (the sum of seven component scores). The seven components that comprise the global sleep quality score include: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction over the past month. Higher scores indicate worse sleep quality. A Global Sleep Quality Score greater than 5 indicates poor sleep quality and difficulties with sleep in at least two areas. Reliability and validity were demonstrated in healthy elderly²⁴ and populations with DM²⁵ and HF²⁶ and in the current data.

Physical function is the “ability to carry out activities that require physical actions, ranging from self-care (activities of daily living) to more complex activities that require a combination of skills, often within a social context,” as defined by the Patient-Reported Outcomes Measurement Information System (PROMIS) (<http://www.nihpromis.org>). To elicit its multiple dimensions, we used the 6MWT, SF-36 global health and physical function components, and daily wrist actigraphy.

The 6MWT²⁷ is reliable, valid, and safe in elderly adults with a variety of medical conditions, including HF and DM.¹⁹ The 6MWT correlated with treadmill exercise tests in patients with stable coronary heart disease²⁸ and was a significant and independent predictor of morbidity, rehospitalization, and mortality in patients with left ventricular dysfunction,²⁹ stable coronary heart disease, and HF.^{28,30} The 6MWT was highly correlated with quality of life,³¹ making it a good measure for associations with functional status and ability to perform daily work.

Self-reported physical function and overall health status were measured with the Physical Component Summary (PCS) score and the General Health (GH) subscale from the SF-36 Medical Outcomes Study (version 2).^{32,33} The SF-36 is composed of eight scales, all of

which have demonstrated high levels of reliability in the general population and among patients with DM^{34,35} or stable HF.⁷

Daytime physical activity was measured over four consecutive days and nights with the Actiwatch-64 wrist actigraphy monitor (Respironics Mini Mitter, Inc., Bend, OR). Wrist actigraphy reflects the combination of whole body activity and arm activity and is sensitive to motion in all directions. Wrist actigraphy has been used to quantify physical activity and sleep disturbances in patients with DM,^{36,37} and reliably discriminates between levels of activity intensity in adults with type 2 DM.³⁸ In HF patients, wrist actigraph movement scores were correlated with peak oxygen consumption during treadmill testing.³⁹

Participants wore the actigraphs on their non-dominant wrists. Actigraph data were collected over 4 consecutive days (Day 4 included PSG night). Only the first three days were used in the current study. Actigraphy was recorded in 30-second epochs. Daytime summary data were computed using the Actiware Sleep v5 Program (Respironics Mini Mitter, Bend, OR), based on the period from lights on in the morning to lights out in the evening, determined by sleep diary recordings and actigraph event markers. Activity-related variables included four-day measures of the average daily activity counts per minute, and the ratio of minutes of daytime activity to minutes of nighttime activity. This measure was chosen as a surrogate measure of the amplitude of the circadian rhythm of activity-rest, with higher values indicating a stronger circadian rhythm.

Self-reported symptoms included measures of depression, fatigue, daytime sleepiness, and pain. Depression was measured using the Center for Epidemiologic Studies Depression Scale (CES-D). The CES-D is a self-report, 20-item questionnaire used to screen for depressive symptoms in the general population. The instrument uses a 0-3 response scale, with higher scores suggesting higher depressive symptoms.⁴⁰ The CES-D has been used in many large DM epidemiological studies,⁴¹ and was found to be sensitive for predicting sub-clinical depression in patients with both type 1 and type 2 diabetes.^{42,43} In patients with stable HF, the CES-D had a Cronbach's alpha of 0.89, supporting its reliability in HF patients.⁷

Fatigue was measured using the global fatigue index score from the Multidimensional Assessment of Fatigue (MAF) scale. The MAF is a 16-item multidimensional questionnaire about the severity and effects of fatigue. Internal consistency and convergent and divergent ability have been supported.⁴⁴ In patients with stable HF, the Cronbach's alpha was 0.91.⁷

Daytime sleepiness was measured using the Epworth Sleepiness Scale (ESS).⁴⁵ The ESS asks patients to rate how easily they would fall asleep in eight routine daily situations on a 0-3 Likert-type scale. Scores range from 0-24, and a score of 10 or higher indicates abnormal sleepiness. The ESS has strong test-retest reliability and internal consistency in healthy⁴⁶ and HF populations.⁷ The ESS is able to discriminate between patients with narcolepsy and healthy controls.⁴⁷

Average nightly pain was measured by self-report, by asking participants how often they were awakened by pain during the past month, using a 0 (never) – 5 (everyday or almost

everyday) rating scale. This item was taken from the Sleep Heart Health Study Sleep habits scale.⁴⁸

Statistical Analyses

Descriptive statistics included measures of central tendency for continuous variables and frequencies for categorical variables. Unadjusted *t* tests were computed to evaluate differences in self-reported and PSG-derived sleep variables. Significance levels were set at $p < 0.05$.

We computed univariate general linear models to assess differences between groups on all sleep, physical function, and symptom variables while controlling for covariates known to be associated with the variables of interest, including BMI, NYHA classification, age, and gender. Use of these models also allowed us to adjust for uneven sample sizes between groups. Hierarchical linear regression analyses were used to assess the independent contributions of diabetes to the variance in those dependent variables that were significantly different between groups (sleep latency, WASO, 6MWT), after controlling for clinical and demographic variables. All statistics were run with IBM SPSS 19 (SPSS, Chicago, IL).

Results

The sample characteristics were reported previously,^{7,8,21} but are repeated here for clarity. The sample included 173 participants (age 60.4 ± 16.1 years) with stable HF, of whom 54 also had DM. Within the total sample, 65.3% were male, 5.8% Latino, and 63.3% Caucasian. The average ejection fraction $32.63 \pm 15.2\%$, and BMI 30.72 ± 8.0 . The participants with DM reported more comorbid conditions than the HF participants without DM (3.6 ± 1.7 vs. 1.9 ± 1.1 ; $p < 0.05$).

There were no statistically significant differences between HF patients with and without DM on age, gender, or race. Although the mean BMI indicated that both groups were obese, BMI was significantly higher in the participants with DM (32.15 ± 6.6 vs. 30.1 ± 8.6 , $p < 0.05$), and a higher percentage of HF patients with DM were classified as NYHA Class III or IV than those without DM (Table 1).

PSG findings revealed that the participants with DM had longer sleep latency (38.23 ± 36.34 vs. 26.69 ± 34.64 minutes) and a higher percentage of WASO (28.11 ± 18.47 vs. 21.78 ± 13.61) than their non-diabetic counterparts (all $p < 0.05$). Although the HF patients with DM slept an average of 30 minutes less than non-DM patients, the difference in total sleep time was not statistically significant (Table 2).

The HF patients with DM had a higher unadjusted respiratory disturbance index (RDI) and spent more minutes with a mean nocturnal oxygen saturation $< 90\%$ than the patients without DM. However, these findings were not statistically significant after adjustment for age, gender, BMI, and NYHA classification. After adjusting for age, gender, BMI, and NYHA classification, self-reported sleep quality was low in both groups, but did not differ between groups in the global PSQI score. The participants with DM reported more nightly

pain during the prior month than the non-DM HF participants, but again this was not statistically significant. (Table 2).

To evaluate the explanatory contributions of comorbid DM to sleep characteristics, we performed a series of hierarchical linear regression models for each of the sleep variables that were significantly different between the groups with and without DM (sleep latency, WASO). In the first model of each analysis, we included covariates that were correlated to the dependent variable and might confound the findings, such as age, gender, and BMI. In the second model, we included DM to evaluate its independent effects. In the model in which WASO was the dependent variable, age was the only significant predictor in the first step of the model. Diabetes explained an additional 2% of the variance when included in the second step (Table 3). Diabetes was the only statistically significant variable that explained sleep latency, explaining an additional 3% of the variance, when included in the second step. (Table 4)

We used general linear models to examine DM-related differences in objectively measured and self-reported physical function while controlling for NYHA classification, age, gender, and BMI. The participants with DM had shorter 6MWT distances (by an average of 70 meters) and poorer self-reported physical function, as well as poorer global health (all $p < 0.05$) (Table 5). We performed a hierarchical linear model, with age, gender, NYHA class, and BMI included in the first step and diabetes added in the second step. Diabetes explained an additional 4.9% of the variance in 6MWT distance. (Table 6)

Average daily activity counts per minute were lower in participants with DM, although not statistically significant when data were controlled for age, NYHA classification, BMI, and gender. However, the ratio of daytime to nighttime activity was significantly lower in the patients with DM (7.9 vs. 10.3, $p < 0.05$).

There were no statistically significant differences between the patients with and without DM on depression, fatigue, or excessive daytime sleepiness. However, the mean depression score among the patients with DM (19.61 ± 1.42) was well above the clinically meaningful cutpoint of 16, which is used to identify people at risk for clinical depression.⁴⁹ Those without diabetes had mean scores of 15.54 ± 0.94 . The patients with DM also reported higher nocturnal pain levels, but the differences were not significant (Table 5).

Discussion

Patients with HF are at considerable risk for sleep disturbances, poor physical functioning, and high levels of daytime symptoms. However, the presence of comorbid DM conferred increased levels of sleep disturbance, as documented by the polysomnographic variables and both objective and subjective measures of physical functioning. These findings underscore the added burden of diabetic comorbidity to these outcomes and their potential contributions to morbidity and mortality.

The average sleep latency among the patients with DM was longer than the 30 minutes generally considered an objective criterion for insomnia.⁵⁰ The WASO was also higher in the patients with DM, who, on average, spent approximately 28% of their time awake after

they had already fallen asleep. Although the reasons for this are not completely known, it is possible that these findings may be due to the slightly higher levels of pain in patients with DM. Although data were not available on the sources of pain, pain in diabetes may be due to neuropathic pain in the lower extremities — a condition that affects approximately 70% of patients with diabetes and has been associated with both sleep difficulties and high rates of insomnia.⁵¹ High levels of pain throughout the night may also cause increased leg movements or non-respiratory arousals that could influence sleep continuity and time to fall asleep. It is also possible that the patients with DM experienced episodes of hypo- or hyperglycemia, which might explain the difference in sleep variables. In a comparison study of patients with and without DM, patients with DM had significantly lower sleep efficiency and more fragmented sleep. Also, in the patients with DM, poor glucose control (elevated A1C) was associated with lower sleep efficiency and more nocturnal moving time after adjustment for age and gender.³⁷

Although not statistically significant, the approximately 30-minute difference in sleep duration between the HF patients with and without DM is clinically significant. In addition, the finding of short total sleep time in the entire sample (~5 hours), regardless of the presence of DM, is striking and has important implications for metabolic control and obesity in these patients. A number of cross-sectional and experimental studies show associations between sleep duration of less than six hours and obesity,^{52,53} diabetes,^{12,54} and insulin resistance and metabolic syndrome.^{12,55} In a nationally representative sample of adults, short sleep duration (< 6 hours) was linearly and inversely associated with BMI and waist circumference, after adjusting for gender, race, and ethnicity.⁵³ Di Milia (2013) reported a strong association between short sleep (6 hours or less) and obesity, even when controlling for the presence of DM.⁵² In unadjusted analyses, sleep duration was shorter in the patients with DM than those without (302 vs. 333 minutes [$p = 0.051$]). However, after controlling for BMI, sleep duration did not differ between groups, suggesting that BMI has effects on sleep duration independent of diabetes alone. These findings contrast with those of the Sleep Heart Health Study, in which sleep times of < 6 or > 9 hours were associated with an increased risk for both impaired glucose tolerance and overt diabetes when adjusted for body composition.¹² However, both the HF and HF + DM groups had very short sleep duration. Short sleep duration could potentially put all HF patients at higher risk for impaired glucose tolerance and overt diabetes, while worsening glycemic control in the patients with DM. Mechanisms for the relationship between sleep duration and altered glucose metabolism are not well understood.⁵⁶

Sleep disordered breathing is common among both patients with HF and those with DM,^{8,11,15} but is especially associated with increased BMI. In this study, HF patients with and without DM were obese, although the patients with DM had somewhat higher levels of BMI. Differences in sleep disordered breathing were explained by BMI rather than DM in the analyses that controlled for these effects. Thus, DM did not confer additional risk for sleep disordered breathing.

Self-reported physical function, general health, 6MWT distance, and day to night activity ratio were all lower in the HF patients with DM than in their non-diabetic counterparts. In concert with the findings of Deaton et al.,¹⁰ this suggests that DM confers additional

decrements in these quality of life indicators. However, the physical function and 6MWT results are also congruent with earlier studies of abnormal glucose metabolism in patients with HF that documented that those with previously existing and newly diagnosed DM had lower resting ventricular ejection fraction and contractile reserve and lower 6MWT results (349 m vs. 379 m, respectively) than HF patients with normal glucose tolerance (467 m, $p < 0.001$). The lack of differences in the cardiac parameters did not explain the lower exercise capacity but suggested that there were independent effects of diabetes on both cardiac function and exercise capacity.¹⁹ Reductions in physical functioning are associated not only with poor quality of life, but also with increased risks for mortality⁵⁷ that may be especially pronounced among HF patients with DM.

Although there was no statistically significant difference in depression between patients with and without DM, the observed higher levels in the patients with HF and DM are clinically meaningful; the lack of a significant difference may be due to the small sample size. Depression is common in both HF and DM. However, prevalence rates differ among studies and are confounded by related psychological and physiological variables. Pouwer reported that among 3,107 adults with type 2 DM, the prevalence of depression was only associated with having DM and a comorbid condition; not with DM alone.⁴³

Results from this study suggest that HF patients with comorbid DM are at higher risk for sleep disturbance and decrements in physical function. While previous studies have documented that HF patients have poor sleep and physical function,²⁶ both HF specialists and endocrinology clinicians should be particularly cognizant of the potential increased risk among HF patients who also have DM.

Future studies are needed to evaluate the biological and behavioral factors that lead to poor sleep among the patients with DM and to evaluate the extent to which strategies designed to improve sleep duration and reduce time awake after sleep onset may improve sleep and related outcomes for these patients. Interventions may focus on disease-related factors for poor sleep (e.g., pain, hypoglycemia), but may also include the use of hypnotic medications or cognitive behavioral strategies to promote sleep extension and continuity. Increased physical activity and exercise are important to both groups, but especially to patients with these comorbid conditions.

Limitations

The sample size and data used in this study were not designed a priori to address the effects of DM on patients with HF. Therefore, an important limitation is the lack of measures of glycemic control. Because of the older age and high BMI of the total sample, many patients likely had impaired glucose tolerance but did not qualify for the DM group. Because of the cross-sectional analyses, it was not possible to evaluate the natural history of DM and HF over time or their combined effects on the sleep, symptoms, and function variables. Although we found quite large and clinically meaningful differences in several outcome variables (e.g., sleep latency and 6MWT results), the study may have lacked statistical power because of the small number of patients with DM. Nevertheless, the approximate 30% prevalence of DM in this sample is consistent with earlier studies of HF populations.^{2,6}

Future longitudinal studies with large numbers of HF patients who have DM are needed to further evaluate its contributions in patients with HF. Also, HF has a negative effect on the outcomes of DM patients. Studies of measures of glycemic control, as well as other symptom measures (e.g., pain) and biomarkers, are needed to further explain the added risk conferred by DM on sleep and physical function. Although pharmacological and behavioral interventions are available to promote sleep, and physical activity interventions are available for patients with either HF or DM, studies are needed to evaluate their effects in patients with these frequently comorbid conditions.

In summary, HF and DM are often comorbid conditions. The findings of this study suggest the independent contributions of DM to sleep and physical function among patients with stable HF — a large and growing group of patients. Future research is needed into these important relationships.

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Table 1

Demographic and Clinical Characteristics

	Total Sample N = 173	HF Patients with Diabetes n = 54	HF Patients without Diabetes n = 119
Age in years (mean $\pm$ SD)	60.4 $\pm$ 16.1	62.9 $\pm$ 14.3	59.2 $\pm$ 16.7
BMI kg/m ²	30.7 $\pm$ 8.0	32.15 $\pm$ 6.6	30.1 $\pm$ 8.6 ^a
Gender (% male)	65.3	72.2	62.2
Hispanic or Latino	(5.8%)	(1.9%)	(7.6%)
Ethnicity (%)			
Race (% non-White)	(36.7%)	(30%)	(37%)
Charlson Comorbidity	2.4 $\pm$ 1.5	3.6 $\pm$ 1.7	1.9 $\pm$ 1.1 ^a
Index (mean $\pm$ SD)			
NYHA Class (n/%)			
I No symptoms		0	5 (4.2%)
II Mild symptoms		24 (44.4%)	71 (59.7%)
III Moderate symptoms		25 (46.3%)	36 (30.3%)
IV Bedrest		5 (9.3%)	7 (5.8%)
Ejection Fraction (%)	32.6 $\pm$ 15.2	33.5 $\pm$ 15.5	32.2 $\pm$ 15.1

^a $p < 0.05$.

Table 2
Comparison of Self-Reported and Objective Sleep Characteristics between HF Patients with and without Diabetes^a

Sleep Parameters	Patients with DM (<i>n</i> = 54)		Patients without DM (<i>n</i> = 119)	
	Unadjusted Means (SD)	Adjusted Means (SD)	Unadjusted Means(SD)	Adjusted Means (SD)
Self-Reported Night Variables				
PSQI Global Sleep Quality	9.39 (4.55)	9.38 (4.59)	8.45 (4.02)	8.41 (4.01)
Pain from Diary	1.81 (1.24)	1.83 (1.24)	1.51 (1.34)	1.48 (1.33)
PSG Sleep Variables				
Total Sleep Time (minutes)	302.35 (109.43)	302.30 (110.48)	333.32 (89.1) ^c	331.02 (87.77)
Sleep Latency (minutes)	37.85 (36.11)	38.23 (36.34)	26.67 (34.49)	26.69 (34.64) ^b
Wake After Sleep Onset (%)	28.24 (18.32)	28.11 (18.47)	21.69 (13.55) ^b	21.78 (13.61) ^b
PSG Respiratory Variables				
Respiratory Disturbance Index	29.74 (21.14)	29.2 (21.0)	21.75 (17.45) ^b	22.0 (17.51)
Mean Oxygen Saturation	92.52 (2.94)	92.49 (3.0)	93.40 (2.59)	93.41 (2.6)
During Sleep (%)				
Time at O ₂ < 90% (minutes)	15.46 (2.7)	15.54 (19.83)	10.23 (1.7)	10.27 (18.46)

^a Means adjusted for age, gender, BMI, and NYHA classification.

^b *p* < .05

^c *p* = .051

Table 3

Hierarchical Regression Analysis with Percent Wake after Sleep Onset (WASO [%]) as the Dependent Variable

Variable	Model 1				Model 2			
	B	SEB	β	P-value	B	SEB	β	P-value
Age	.333	.078	.343	.000	.305	.078	.314	.000
Gender	−2.873	2.404	−.088	.234	−2.353	2.390	−.072	.326
BMI	−.026	.156	−.013	.870	−.093	.158	−.048	.559
History of Diabetes					5.234	2.448	.157	.034
R ²	.138				.161			

Table 4

Hierarchical Regression Analysis with Sleep Latency as the Dependent Variable

Variable	Model 1				Model 2			
	B	SEB	β	P-value	B	SEB	β	P-value
Age	-.264	.190	-.119	.168	-.340	.190	-.153	.076
Gender	-2.541	5.867	-.034	.666	-1.14	5.82	-.015	.845
BMI	-.542	.382	-.119	.172	-.704	.384	-.159	.069
History of Diabetes					14.103	5.96	.185	.019
R ²	.018				.050			

Table 5

Differences in Physical Function and Daytime Symptom Measures between HF Patients with and without Diabetes^a

	Patients with DM (<i>n</i> = 54)	Patients without DM (<i>n</i> = 119)	<i>p</i> -value
6MWT (meters) ^b	251.60 (16.78)	319.81 (10.43)	0.001
Ratio of daytime to nighttime activity	7.9 (0.7)	10.3 (0.4)	0.002
Average activity counts per minute during day	216.94 (17.28)	216.51 (11.10)	0.984
SF-36 General Health	11.80 (0.58)	13.47 (0.39)	0.020
SF-36 Physical Component Summary Score	26.06 (0.20)	26.57 (0.13)	0.038
CESD Depression	19.61 (1.42)	15.54 (0.94)	0.20
Global Fatigue	31.26 (2.03)	29.3 (1.34)	0.433
Epworth Sleepiness Scale	9.11 (0.60)	7.96 (0.40)	0.119

^a Mean scores from each variable have been adjusted for age, gender, NYHA class, and BMI. Data are reported as adjusted means and standard error.

^b Fewer participants completed the 6- meter walk distance: diabetes group *n* = 45, no diabetes group *n* = 112.

Table 6

Hierarchical Regression Analysis Summary for Variables Predicting 6MWT (meters)

Variable	<u>Model 1</u>				<u>Model 2</u>			
	B	SEB	β	P-value	B	SEB	β	P-value
Age	−2.91	.638	−.346	.000	−2.594	.623	−.309	.000
Gender	−86.136	19.289	−.310	.000	−92.471	18.734	−.333	.000
NYHA Class	−66.565	14.277	−.321	.000	−58.871	13.983	−.284	.000
BMI	−1.040	1.242	−.064	.403	−.295	1.220	−.018	.809
History of Diabetes					−68.217	20.086	−.231	.001
R ²	.318				.367			