Thirty-Day Analysis of Dyspnea and Edema in Heart Failure Subjects

A Senior Honors Thesis Presented in Partial Fulfillment of the Requirements for the Degree of Bachelor of Science in Nursing with Distinction
College of Nursing of The Ohio State University

By
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Description of the Problem

Heart failure (HF) is a growing epidemic in the United States. Currently, there are about 5 million diagnosed cases of heart failure in the United States; there continues to be approximately 400,000 new cases diagnosed each year (American Heart Association, 2004; Reigel & Carolson, 2002). HF primarily affects the elderly, specifically 10 out of every 1000 individuals over the age of 65 (Haldeman, Croft, Giles & Rashidee, 1999). Given this, as the population of America ages, there will undoubtedly be an increase in the number of patients diagnosed with HF. HF is the most common reason for hospitalization in the US. Exacerbations are associated with a number of symptoms. There is increasing research focused on the causes and effects of delay-time when experiencing cardiac symptoms. However, there is little research that examines the relationship between daily fluctuations in the symptoms of heart failure and treatment-seeking delays.

Pathophysiology of Heart Failure

Heart failure is the inability of the heart to satisfy the metabolic demands of the body. There are two broad types of heart failure: acute and chronic. Acute heart failure has a rapid onset of ventricular muscle failure, usually caused by a primary acute event such as a myocardial infarction. In acute heart failure, there is an abrupt decrease in the contractile ability of the heart to eject blood, which causes an abrupt decrease in cardiac output.

The second type of heart failure is chronic heart failure. Chronic heart failure most often results from such diseases as: myocardial infarction/ ischemic heart diseases,
hypertension, valvular disease, cardiomyopathies and arrhythmias. Myocardial systolic dysfunction most often results from ischemic heart disease. When the cardiac cells become ischemic, there is a loss of the vital elements of the cardiac “pump” and the remaining cells are overworked. This stimulates an increase in myocyte size, a decrease in capillary density, an increase in intercapillary distance, cardiac dilatation and a deposition of fibrous tissue (Weber, 1992). As the heart begins to dilate, the cardiac cells stretch and increase the force of contraction and subsequent stroke volume. This is known as Starling’s Law. The increased stretching of the cardiac cells stimulates further hypertrophy. Furthermore, myocardial hypertrophy increases wall tension and increases myocardial oxygen consumption, which reduces oxygen availability for vital organs.

Decreases in cardiac output stimulate the sympathetic nervous system, which will increase heart rate and induce vasoconstriction as a compensation mechanism to maintain normal blood pressure and perfusion of the vital organs. The decrease in contractility causes the ventricles to be unable to empty efficiently. In turn, blood is sequestered in the lungs or the systemic veins where an increase in hydrostatic pressure moves fluid into the interstitial space, alveoli or tissues causing pulmonary edema and an acute shortness of breath, generalized edema or organmegaly (Gordan & Child, 2000).

As cardiac output falls and blood flow to the kidneys is decreased, there is an increase in secreted renin as a response. Renin catalyzes the conversion of angiotensinogen to Angiotensin I, which in the presence of angiotensin-converting enzyme (ACE), is converted to Angiotensin II, a powerful vasoconstrictor. The formation of Angiotensin II, primarily in the pulmonary vessels, produces an increase in blood pressure provided by vasoconstriction and stimulates the release of aldosterone from the
adrenal cortex. Aldosterone increases the retention of sodium (and thus fluid) and results in increasing blood volume or preload, which adds an additional burden to the heart (Gorden & Child, 2000).

Hormonal alterations are common in HF. Catecholamines, epinephrine and norepinephrine, are released from the adrenal medulla and the sympathetic nervous system. The release of epinephrine leads to skeletal muscle vasodilatation, whereas norepinephrine stimulates peripheral vasoconstriction, an increased mean arterial pressure and an increase in afterload. Antidiuretic hormone (ADH) is a hormone released from the posterior pituitary gland when stimulated by an increase in plasma osmolality, stress, or decreased atrial stretch receptor firing. ADH causes vasoconstriction and retention of water by the kidneys. Atrial natriuretic peptide (ANP) is synthesized and stored in the cardiac muscle and released when the cardiac chambers are stretched by increased blood volume or venous return, often due to fluid overload. ANP produces diuresis and natriuresis to reduce excessive blood volume. However, as HF develops over time this effect is overwhelmed by the activity of the renin-angiotensin-aldosterone system. The result is volume overload, increased afterload and further reductions in cardiac output and blood pressure.

These hemodynamic changes activate baroreceptors in the carotid sinuses and the aorta to maintain cardiac output. This stimulation of baroreceptors results in increased sympathetic outflow stimulation and a further increase in cardiac contractility and vasoconstriction of the peripheral vasculature thus increased blood pressure and heart rate. Unfortunately, this response also increases the pressure that the heart has to work against (afterload) in order to pump the oxygen-rich blood out to other vital organs,
which makes it more difficult to get the blood to those organs (Gordon & Child, 2000). This cyclical decrease in cardiac output will continue until the individual decompensates and multiple organ systems malfunction. Death is the eventual outcome without effective intervention.

**Signs and Symptoms of Heart Failure**

Given the extensive pathophysiology of heart failure, the signs and symptoms one can expect to see in a patient with heart failure include pulmonary congestion and edema, systemic venous congestion and peripheral edema, organomegaly, ascites, anorexia, confusion, pallor, orthopnoea, dyspnea (at rest and on exertion), tachycardia, tachypnea, extra heart sounds, abnormal breath sounds, jugular venous distention and weight gain (Gordon & Child, 2000).

**New York Heart Association Classifications**

Signs and symptoms develop over time and may vary from individual to individual or from day to day. The affect of these symptoms on daily life is used to classify patients. The New York Heart Association established a classification scale for patients with cardiac disease based on functional status. Classifications are as follows:

Class I. Patients with cardiac disease but without resulting limitation of physical activity.

Class II. Patients with cardiac disease resulting in slight limitation of physical activity

Class III. Patients with cardiac disease resulting in marked limitation of physical activity.

Class IV. Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort.
This classification system is primarily utilized to classify severity of functional restrictions, to establish treatment regiments for patients with heart failure and has been widely used over the past 50 years (Hurst, Morri & Alexander, 1999).

**Review of Literature**

**Delay of Treatment**

Excessive delay time has been found to contribute to an increase in morbidity and mortality in cardiac patients (Cooper, Simmons, Casstanar, Prasad, Franklin & Freelinz, 1986; Johnson & King, 1995). If there is a time window between cardiac symptom exacerbation and hospital admissions, during which health care providers could intervene earlier, this might prevent hospitalization and other serious physiological consequences for the patient (Schiff et al., 2003). Although HF is a serious and potentially deadly disease, there is very little consistency in the amount of time it takes for a patient experiencing cardiac symptoms to seek treatment.

In a sample of heart failure patients (n= 753) at a Veterans Administration facility, Evangelista et al. (2000) found that the mean delay time between a patient’s first awareness of their cardiac symptom and their arrival at the hospital was $2.93 \pm 0.68$ days. The detection of dyspnea and edema nearly doubled the time to treatment when compared with those who did not have these symptoms. A higher New York Heart Association class and care by a primary care physician also prolonged delay time. The factors associated with a decrease in delay time were presence of chest pain and a previous history of HF admissions. Therefore, there are many factors that affect when a patient with heart failure will seek care for a HF exacerbation.
Schiff, Fung, Sperof and McNutt (2003) studied a sample of patients (n = 87) admitted to a public general hospital with a diagnosis of heart failure. These investigators found that the worsening of cardiac symptoms was an observed mean of 12.4 ± 1.4 days before admissions for edema, 11.3 ± 1.6 days for weight gain and 8.4 ± 0.9 days for dyspnea. They identified a period of days to weeks between the onset of worsening cardiac symptoms and hospital admissions for heart failure decompensation. This study demonstrated that patients can detect changes in their HF symptoms and that those changes may be signs of an impending HF exacerbation. However, these subjects did not act on their symptom changes for more than a week.

In a study by Friedman (1997) of elderly HF patients (n = 181) in an acute care facility, 91% of the subjects reported experiencing dyspnea for an average of 3 days before seeking treatment. He compared this with the 4 to 12-day duration of cardiac symptoms in a younger cohort of patients and concluded that more intensive patient education addressing prompt health care seeking was necessary.

**Reasons for delay**

Delays in seeking treatment are very costly and can result in death of a patient. When examining why patients with heart failure have difficulties in self-care, specifically the recognition of symptoms, Carlson, Riegel & Moser (2001) found most patients (n = 139) had a very difficult time recognizing the symptoms of heart failure. They also reported that patients with experience addressing heart failure symptoms recognized them easier than those who are newly diagnosed. These investigators found several factors limiting their subject’s ability to recognize their symptoms. These include comorbidities, lack of confidence in their ability for self-care, and patient education. It is difficult to
monitor the symptoms of one illness, but trying to identify and differentiate between several chronic conditions is much more challenging. These investigators also identified that confidence in one’s ability to effectively treat their cardiac symptoms was low in this sample. The investigators speculated that might be why patients wait so long with worsening symptoms before seeking treatment, often in an emergency department (ED).

Another potential reason for delays in seeking treatment for one’s heart failure is self-care. Self-care management includes general health maintenance activities that are imperative to maintaining a high quality of life in patients with heart failure. Carlson et al. (2001) found that while patients with heart failure were following general health maintenance recommendations, such as smoking cessation and alcohol avoidance, less than half of these patients exercised regularly. These researchers speculated that this may be due to a lack of education about the safety and necessity of regular exercise among patients with heart failure. Additionally, Rockwell and Riegel (2001) found that patients with higher education and those who are symptomatic are more likely to engage in self-care more often than those patients who are uneducated and asymptomatic. The finding that patients with higher education perform self-care more often than those patients with less education has been supported in other research (Rockwell & Riegel, 2001). However, the finding that patients experiencing more cardiac symptoms were more knowledgeable about symptoms requiring self-care differs from previous investigations. Rockwell and Riegel (2001) concluded that patients with more disease-related symptom experience have engaged in self-care more often than those who do not have this history and that experience promoted a better understanding of change from baseline health status.
In an additional investigation, Carlson, Riegel and Moser (2001), found that misconceptions and lack of knowledge are barriers to self-care. Also, patients with heart failure often do not seek any treatment until re-hospitalization is imminent. They found that most of these patients were unable to evaluate the meaning and importance of their cardiac symptoms and/or did not believe that their self-care could help relieve their symptoms. Carlson et al. (2001) also identified personal struggles, negative emotions, co-morbid conditions, physician support and control as potential barriers to self-care in patients with heart failure. While all of these studies explored possible reasons why there is a large variation in the amount of time it takes for a symptomatic patient with heart failure to seek treatment, none looked at the daily variation of symptoms.

**Dyspnea**

Dyspnea is the most common symptom of heart failure and is the primary reason individuals seek treatment in an emergency department (Parshall, Welsh, Brockopp, Heiser, Schooled & Cassidy, 2001). Dyspnea and chest pain are the most common symptoms reported upon admission to the ED for HF patients (Evangelista, et al, 2000). According to Parshall et al. (2001), there variations in intensity of dyspnea and distress experienced during a heart failure exacerbation. Dyspnea is one of the hallmark symptoms of heart failure and very often contributes to a patient seeking treatment for their heart failure exacerbation and there are documented correlations in the relationship between dyspnea variation and treatment-seeking (Parshall et al., 2001).

Parshall et al. (2001) found that there is a general increase in distress related to dyspnea over the week before deciding to seek treatment in the ED (n = 52). Specifically, these investigators found that about half (54%) of their sample recalled their dyspnea as
having been severe for one day or less before presenting to the ED, that slightly more than one third (35%) indicated their dyspnea had been that severe for more than 6 days, and that only a few (11%) fell in between. Parshall et al. (2001), identified two distinct groups of patients who experienced dyspnea secondary to heart failure: those who experienced an increase in dyspnea intensity and distress over a short period of time (3 days or less) and those who experienced no change in intensity and distress up to a week before seeking treatment. They concluded that those with less tolerance for symptoms had a more dynamic conceptualization of their dyspnea and those with a greater tolerance for dyspnea had a more static conceptualization of it and thus a longer duration prior to seeking care.

**Weight Gain and Edema**

The activation of the renin-angiotensin-aldosterone system leads to the retention of fluid and a systemic increase in blood volume with subsequent pulmonary and systemic congestion. In patients with heart failure, significant weight gain is one sign that may predict a heart failure exacerbation. Accordingly, when patients are diagnosed with heart failure, they are educated to weigh themselves daily and to call their practitioner’s office with any significant changes, i.e., any change ≥ 2-3 lbs overnight or ≥ 3-5 lbs in one week (Heart Failure Society of America, 2003).

A review of the current literature yielded several articles addressing weight gain in patients with HF, however most of these are descriptive articles examining the relationships between fluid dynamics, thiazolidinediones and heart failure (Giles, 2003; Hollenberg, 2003; Tang, Francis, Hoogwerf & Young, 2003). Thiazolidinediones are insulin-sensitizing agents that are widely used in patients with Type 2 diabetes mellitus.
These studies do indicate that when a patient is diagnosed with HF and diabetes mellitus, there is a potential for weight gain as a result of fluid retention or fat accumulation (Giles, 2003). Hollenberg (2003) suggested that in new-onset heart failure, edema is uncommon, and it is even less common in patients taking thiazolidinediones to control their diabetes mellitus. A study by Tang et al. (2003) concluded that a direct association between the risk of fluid retention and the baseline degree of severity of HF has yet to be established and that patients with HF who are being treated with thiazolidinediones experienced a pattern of edema opposite to a pattern during direct myocardial depression (i.e., there is more peripheral edema than pulmonary edema). A patient may experience dyspnea and weight gain simultaneously. As there is an increase in fluid retention, pulmonary congestion may result and it becomes harder for a person to breathe. Thus, in this study we looked at weight gain and self-reported edema as they fluctuated from day to day and determine the association with dyspnea in a group of HF patients.

**Conclusion**

The most commonly recognized symptoms of a HF exacerbation are dyspnea and edema. Although there are significant changes in the signs and symptoms of patients experiencing a HF exacerbation, research shows that there is a large variation in when these patients seek medical treatment. This may occur because some patients may think that a slight increase in dyspnea, edema or weight is a normal fluctuation when in reality it is an early sign of a HF exacerbation. These patients may tend to rely on past experiences to guide their self-care, and may feel that their symptoms will improve in 1-2 days without any medical intervention. Most HF patients are taught to notify their health care provider if they experience a significant change in dyspnea, edema or weight gain,
but there are no studies that investigate normal fluctuations in signs and symptoms that may influence the decision to seek treatment.

**Study Purpose**

The purpose of this study was to characterize daily fluctuations in self-reported dyspnea, edema, and body weight in subjects with the New York Heart Association class I through IV HF for one month. Our specific aims were:

1. To describe daily fluctuations in self-reported dyspnea, edema and body weight;
2. To examine relationships between self-reported dyspnea, edema and body weight in this cohort; and
3. To evaluate the relationship between symptom stability and emergency department visits and hospitalization.

**Methods**

**Design**

This was a descriptive correlational study. Data were collected daily for 30 days from a cohort of heart failure subjects (n = 48). Association between self-reported dyspnea, edema and body weight were determined.

**Sample & Setting**

This investigation was a sub-study of a larger investigation titled “Appetite, nutrition, and inflammation in patients with heart failure”. For this investigation, we studied a sample of 48 subjects who met the following inclusion criteria: (a) diagnosis of HF with a left ventricular ejection fraction of = 40%, (b) stable on medication regimen for at least 3 months, (c) no history of myocardial infarction in previous 3 months, (d) able to speak and write English, (e) no obvious cognitive deficit and (f) have a telephone.
The exclusion criteria included (a) valvular heart disease, (b) history of cerebral vascular accident, (c) co-existing terminal illness or other illness known to be associated with decreased appetite and weight loss or (d) have been referred for heart transplantation. Subjects were recruited from the three Midwestern HF clinics, two in central Ohio and one in central Kentucky. For this sub-study, a convenience sample of 48 subjects with NYHA classification I-IV HF was selected.

**Procedure**

Institutional review board approval was obtained from The Ohio State University Biomedical Institutional Review Board. Subjects were referred to the investigators by cardiologists and nurse practitioners. Referred subjects were contacted by telephone, received a clear explanation of the study and were invited to participate in the study. The researchers explained the informed consent to the subjects via the telephone. Following verbal consent, a research assistant obtained signed, informed consent at the subjects’ home. During this visit, baseline data collection was completed with questionnaires that included: (a) demographic data and (b) daily symptom rating scale. Daily, for the next 30 days, the subjects recorded severity of heart failure symptoms. These included evaluation of anxiety, appetite, fatigue, depression, sleeplessness, dyspnea, edema, and weight gain, using a 10-point analog scale with zero indicating the “worst it has ever been” and ten indicating the “best it has ever been”. This reported analysis only included dyspnea, edema and body weight. Experts in the field of heart failure were used to establish face validity of the instrument. The subject evaluated and recorded symptoms and body weight was measured and recorded using a calibrated scale. All measures were made at the same time of day each day.
Results

Sample

Descriptive statistics were used to characterize the sample (see Table 1). This study had 48 participants with a mean age of 48 ± 15 years. More than half of the participants were male (54.2%) and most were married (54.2%) More than half of the subjects were retired (52.1%), one-third were on sick leave or disability (41.7%) and the remaining subjects were employed full or part time outside the home (6.3%). A majority (54.2%) of the sample was classified as NYHA class III; overall, subjects had a mean ejection fraction of 30.3 ± 14.1%. The mean yearly income was $30,827 for the participants and almost 90% of this sample had access to medical insurance. This sample had an above average educational level of 12.7 ± 2.52 years.

Daily mean dyspnea score

Subjects were asked to rate their daily shortness of breath at the same time every day using a 0 to 10 scale with zero being the “worst I have ever experienced” to ten indicating, “the best I have ever experienced”. Ratings ranged from 0 to 10. The average daily rating of dyspnea for the sample ranged from 5.2-6.9, indicating moderate daily dyspnea. There were significant daily fluctuations. For example, the average daily rating on day 3 was 5.2 and on day 6, the average rating was 5.9. There were similar fluctuations throughout the 30-day period. (see Figure 1)

Daily mean self reported edema

Subjects were asked to rate their daily perceived edema at the same time every day using a 0 to 10 scale with zero being the “worst I have ever experienced” to ten indicating, “the best I have ever experienced”. Ratings ranged from 0 to 10. The average
daily rating of self-reported edema ranged from 3.0 to 5.2, indicating moderate to severe daily self-reported edema. There were fewer daily fluctuations in self-reported edema and the ratings remained stable for this time period (see Figure 2).

**Mean daily body weight**

Subjects were asked to weigh themselves on the same calibrated scale everyday at the same time for 30-day period. Subjects recorded their daily weight in pounds. Average daily weight ranged from 176.9 lbs to 186.6 lbs. Average daily weight for the 30-day period was 180.2 ± 44.9 pounds (see Figure 3).

**Association between self reported dyspnea and edema**

The association between self-reported dyspnea and edema was evaluated using a Spearman correlation coefficient based on the ordinal level of the data. This association was significant at the p < 0.01 significance level every day during the 30-day time period. This was a positive relationship. As a participant reported more edema, there was a corresponding increase in rated dyspnea (see Table 2).

**Association between body weight and dyspnea**

The association between self-reported dyspnea and body weight was evaluated using a Spearman correlation coefficient based on the ordinal level of the dyspnea data. Few of the associations between body weight and self-reported dyspnea were significant at the p ≤ 0.05 significance level. This relationship was a negative relationship, which indicated as body weight increased, the dyspnea rating decreased (meaning greater dyspnea) and the actual dyspnea experienced increased (see Table 3).

**Association between body weight and self-reported edema**

The association between self-reported edema and body weight was evaluated
using a Spearman correlation coefficient based on the ordinal level of the edema rating. Few of the associations between self-reported edema and body weight were significant at the $p \leq 0.05$ significance level. These were negative relationships. As body weight increased, the self-reported edema rating decreased (meaning greater edema), and the actual edema experienced increased (see Table 4).

**Symptom stability, emergency department visits and hospitalizations**

The investigators divided the sample into stable and unstable groups based upon their daily dyspnea rating. The investigators defined a stable participant as one who had daily dyspnea rating within $\pm 1$ from day 1 on the visual analog scale over the 30-day time period. An unstable participant was one whose daily dyspnea rating varied greater than a $\pm 1$ rating, from day one on the visual analog scale over the 30-day time period. This value was chosen based on the Parshall et al. (2001) investigation. Twenty-six (66.7%) participants were classified as stable and 16 (33.3%) were classified as unstable.

Mean ED visits and hospitalizations were compared for the two groups using an independent t-test. There was a significant difference in the total number of ED visits between the groups ($p \leq 0.05$). The unstable symptom group had $0.69 \pm 0.8$ visits, whereas; the stable symptom group had only $0.15 \pm 0.4$ visits (see Table 5). There was also a significant difference between the total number of hospitalizations between the stable and the unstable group ($p \leq 0.05$). During the 30-day time period, the unstable symptom group was hospitalized a mean number of $1.46 \pm 1.9$ times, whereas, the stable symptom group was hospitalized an average of $0.31 \pm 0.6$ times ($p < 0.05$). Thus, the unstable symptom group was more likely to visit the ED and be hospitalized compared with the stable symptom group.
Discussion

During the 30-day study period, subjects with heart failure reported moderate levels of self-reported dyspnea and edema. There were fewer daily fluctuations in self-reported edema than in self-reported dyspnea. There were fairly large fluctuations in weight over the 30-day period. We also found that the daily relationship between self-reported dyspnea and edema was a positive, significant relationship ($p < 0.01$). This signified that as the participants reported more edema, there was a corresponding increase in their dyspnea. The relationship between self-reported dyspnea and body weight was significant for 3 days ($p < 0.05$), which indicated that as body weight increased, self-reported edema increased. There were a few significant positive correlations between self-reported edema and body weight. As body weight increased so did self-reported edema. The sample was divided into stable and unstable groups based upon their daily dyspnea rating. Two thirds of subjects experienced stable dyspnea. There was a significant difference between the total number of ED visits and hospitalizations between the stable and the unstable group. Those subjects with more symptom variability were more likely to visit the ED and be hospitalized than those subjects with less symptom variability during the 30-day period.

This is the first study to describe the daily variations in self-reported dyspnea, edema and body weight in a group of heart failure subjects and to evaluate the associations between these variables. Even though our sample was classified as NYHA I-IV, self-reported dyspnea was rated as moderate on average for the group. Self-reported edema was also rated as moderate to severe on average for the period. This reflects the severity of daily symptoms of the group. Twenty six (54%) of the subjects were NYHA
Class III. This classification indicates marked limitation in physical activity due to HF symptoms and moderate to severe symptom ratings would be expected. However, slightly more than one third (35.5%) was class I or II, which would indicate little interference with functional ability. Overall, dyspnea and edema were self-rated as moderate to severe on a daily basis for the group as a whole. This may reflect the severity of self-reported symptoms by the majority of the sample.

Several prior investigations have identified that HF patients have worsening symptoms for days prior to an exacerbation, which led to an ED visit or hospitalization. Schiff et al. (2003) found that worsening dyspnea was detected for 8.4 ± 0.9 days prior to a heart failure exacerbation. Evangelista et al. (2000) found that the mean delay time between a patient’s first awareness of cardiac symptoms (dyspnea, edema, fatigue and angina) and their arrival at the hospital was 2.93 ± 0.58 days. Friedman (1997) found that subjects reported worsening dyspnea for an average of 3 days before seeking treatment. In our study, unstable self-reported dyspnea was more likely to result in an ED visit or a hospitalization during the 30-day period. This indicates that patients with unstable HF symptoms like dyspnea are more likely to interpret these symptoms changes as worsening of HF and seek treatment in the ED; these subjects were also more likely to be experiencing an exacerbation that required hospitalization.

These data described the daily weight fluctuations of these subjects. We found that there were large fluctuations during the 30-day period. These changes may be due to alterations in activity, diet and medication use, which may also be related to individual adherence to the prescribed therapeutic regimen. This finding requires further
investigation and determination of factors associated with daily alterations in body weight in this population of patients.

There was a strong association between self-reported dyspnea and edema. This likely is the result of increased pulmonary congestion. There was also an association between self-reported dyspnea and body weight that indicated the increase in blood volume produced a detectable increase in dyspnea, most likely due to greater pulmonary congestion. These findings also suggest that patients can detect daily changes in their symptoms, which may help the patients modify their health behaviors and self-care, as well as provide a cue for contact their primary health care provider to report these changes in symptoms. These data also indicated some significant association between self-reported edema and body weight. This may indicate that subjects detected the redistribution of fluid, rather than a change in fluid volume status with changes in daily symptom detection.

We found that patients with stable dyspnea were less likely to visit the ED or be hospitalized than those with unstable dyspnea. This finding may be useful to health care practitioners who work with HF patients. Practitioners may have HF patients keep a written or electronic log of their daily symptom severity to evaluate individual differences in symptoms. Those with unstable symptoms may require adjustment to their therapeutic regimen or additional contact with a health care provider. Follow-up care and teaching may also be based on this evaluation to ensure continued appropriate management of HF symptoms. Practitioners may also want to regularly contact unstable patients to evaluate condition and assess the effectiveness of prescribed HF management.
Limitations

There were several limitations to this investigation. We had a small sample size \((n = 48)\), which influenced the power of the statistical analyses. The visual analog scale used to evaluate daily symptom severity was inverted from more commonly used visual analog scales (zero being the worst I have ever experienced to ten indicating the best I have ever experienced). Also, it may have been difficult for these subjects to conceptualize symptom severity with the numerical ratings on the visual analog scale. These data were subjective, self-reported ratings and lacked any objective, standardized criteria for the subjects to use to gauge their daily symptom severity. However, symptoms are subjective and self-report is the best way of evaluating the subject’s perception of those symptoms.

Conclusion

Modern health care requires patients to be active participants in maintaining a healthy, fruitful life. The patient must understand their bodies and their diseases and become the first person aware of deviations from normal. This study sought to illustrate that patients can recognize variability in their heart failure symptoms.

For the 30-day study period, subjects with heart failure reported moderate levels of self reported dyspnea and edema. The mean dyspnea rating was significantly associated with changes in body weight indicating that fluid retention increased the sensation of breathlessness. Self reported dyspnea was also associated with self-reported edema. Heart failure patients are able to monitor daily symptoms, detect small changes and may be able to modify self-care activities and therapeutic regimen based on these ratings. Those subjects with greater variability of dyspnea were significantly more likely
to have ED visits and hospitalizations. This information could be used to help practitioners to evaluate daily symptom ratings and to tailor patient monitoring and intervention to improve HF patient outcomes.
References


Table 1. Characteristics of subjects (n = 48)

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<tr>
<td>II</td>
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<td>III</td>
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<td>IV</td>
<td>1 (2.1%)</td>
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SD = standard deviation

Responses may not total 100% due to missing data points
Table 2. Associations between self-reported dyspnea and edema

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<th>Day 1</th>
<th>Day 2</th>
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<th>Day 4</th>
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<td>.599**</td>
<td>.671**</td>
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<td>.542**</td>
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</table>

Spearman correlation coefficients

**p ≤ 0.01 (2 tailed)
Table 3. Associations between self-reported dyspnea and body weight

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 1</td>
<td>-.194</td>
<td>-.266</td>
<td>-.314*</td>
<td>-.287</td>
<td>-.180</td>
<td>-.349*</td>
<td>-.243</td>
</tr>
<tr>
<td>Week 2</td>
<td>-.213</td>
<td>-.225</td>
<td>-.108</td>
<td>-.249</td>
<td>-.205</td>
<td>-.164</td>
<td>-.190</td>
</tr>
<tr>
<td>Week 3</td>
<td>-.354*</td>
<td>-.269</td>
<td>-.232</td>
<td>-.227</td>
<td>-.223</td>
<td>-.161</td>
<td>-.156</td>
</tr>
<tr>
<td>Week 4</td>
<td>-.037</td>
<td>-.202</td>
<td>-.170</td>
<td>-.085</td>
<td>-.206</td>
<td>-.234</td>
<td>-.222</td>
</tr>
</tbody>
</table>

Spearman correlation coefficients

* p ≤ 0.05 (2-tailed)
Table 4. Associations between self-reported edema and body weight

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 1</td>
<td>-.172</td>
<td>-.249</td>
<td>-.225</td>
<td>-.221</td>
<td>-.235</td>
<td>-.298*</td>
<td>-.298*</td>
</tr>
<tr>
<td>Week 2</td>
<td>-.212</td>
<td>-.236</td>
<td>-.301</td>
<td>-.262</td>
<td>-.302*</td>
<td>-.195</td>
<td>-.209</td>
</tr>
<tr>
<td>Week 3</td>
<td>-.298</td>
<td>-.257</td>
<td>-.200</td>
<td></td>
<td>-.254</td>
<td>-.284</td>
<td>-.183</td>
</tr>
<tr>
<td>Week 4</td>
<td>-.121</td>
<td>-.198</td>
<td>-.173</td>
<td>-.103</td>
<td>-.191</td>
<td>-.199</td>
<td>-.202</td>
</tr>
</tbody>
</table>

Spearman correlation coefficients

* $p \leq 0.05$ (2-tailed)
Table 5. A comparison of hospitalizations and emergency department visits for stable and unstable symptom groups

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>t</th>
<th>df</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total hospitalizations: All cause</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.31</td>
<td>0.62</td>
<td>-2.13</td>
<td>13.29</td>
<td>0.05*</td>
</tr>
<tr>
<td>Unstable</td>
<td>13</td>
<td>1.46</td>
<td>1.90</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hospitalizations: For Heart Failure</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.08</td>
<td>0.27</td>
<td>-1.73</td>
<td>13.50</td>
<td>0.11</td>
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<tr>
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<td>13</td>
<td>0.46</td>
<td>0.78</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hospitalizations: Other cardiac</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.04</td>
<td>0.20</td>
<td>-1.50</td>
<td>14.45</td>
<td>0.15</td>
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<tr>
<td>Unstable</td>
<td>13</td>
<td>0.23</td>
<td>0.44</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total ER visits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.15</td>
<td>0.38</td>
<td>-2.44</td>
<td>14.95</td>
<td>0.03*</td>
</tr>
<tr>
<td>Unstable</td>
<td>13</td>
<td>0.70</td>
<td>0.75</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ER visits: Caused by heart failure</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.04</td>
<td>0.20</td>
<td>-1.04</td>
<td>15.36</td>
<td>0.32</td>
</tr>
<tr>
<td>Unstable</td>
<td>13</td>
<td>0.15</td>
<td>0.38</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ER visits: Other cardiac</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>26</td>
<td>0.04</td>
<td>0.20</td>
<td>-0.48</td>
<td>18.20</td>
<td>0.66</td>
</tr>
<tr>
<td>Unstable</td>
<td>13</td>
<td>0.08</td>
<td>0.28</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p ≤ 0.05
Figure 1. Average self-reported dyspnea for 30 days (n = 48)
Figure 2. Average daily self-reported edema for 30 days (n = 48)
Figure 3. Average daily weight for 30 days (n = 48)