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"ANXIETY IS NOT MANIFESTED BY ELEVATED HEART RATE AND BLOOD PRESSURE IN ACUTELY ILL CARDIAC PATIENTS"

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• BACKGROUND Patients with acute myocardial infarction (AMI) and heart failure (HF) are often anxious. Anxiety after AMI may cause in-hospital complications and increased mortality. Clinicians often use heart rate and blood pressure as indicators of anxiety; however, little is known about whether these measures accurately reflect anxiety in acutely ill patients.

• OBJECTIVES The purpose of this study was to determine whether heart rate and blood pressure were related to level of anxiety at the time of measurement in patients with chronic advanced HF, patients with AMI, and healthy individuals.

• METHODS For purposes of this descriptive, correlational investigation, we combined data from two studies: 1) study of anxiety among patients experiencing AMI and 2) study of the impact of a biofeedback-relaxation intervention in patients with HF. Anxiety, heart rate, and blood pressure were measured in the same manner in each group of participants. State anxiety was measured in all participants using the anxiety subscale of the Brief Symptom Inventory. Heart rate and blood pressure data were collected immediately prior to the anxiety assessment.

• RESULTS There were no correlations between anxiety and heart rate or diastolic blood pressure. Higher anxiety was associated with lower systolic blood pressure in patients with AMI ($r = -.23$, $P < .05$) and in healthy individuals ($r = -.27$, $P < .05$). Mean systolic blood pressure, diastolic blood pressure, and heart rate were similar for patients in high and low anxiety subgroups among all types of patients.

• CONCLUSION Elevated heart rate and blood pressure do not accurately reflect level of anxiety as reported by patients with HF or AMI and healthy individuals, and thus cannot be used to assess anxiety in acutely ill patients. Clinicians who use changes in heart rate or blood pressure as indicators of anxiety may fail to recognize and treat anxiety, placing their patients at high risk for both immediate and long-term complications.
Historically, healthcare providers focused on how physiologic parameters, such as dysrhythmias, Killip class, and infarct size and location, affected recurrent cardiac events and mortality for patients with coronary artery disease (CHD). More recently, researchers have examined whether psychological factors, such as anxiety and depression, impact the pathogenesis of CHD and the morbidity and mortality for persons diagnosed with CHD.

Anxiety is a “psychophysiological phenomenon experienced as a foreboding dread or threat to a human organism whether the threat is generated by internal, real or imagined dangers.” Anxiety is manifested by a variety of psychological and somatic symptoms. Understandably, inpatients with acute myocardial infarction (AMI) and heart failure (HF) are often anxious. This anxiety stems from concerns such as physical symptoms, diagnostic or therapeutic procedures, the intensive care unit (ICU) environment, risk of death, cost of treatment, and their ability to resume self-care, work or recreational activities. Ten to twenty-six percent of hospitalized persons with AMI are more anxious than individuals with a psychiatric disorder. Anxiety is more prevalent than depression for patients with AMI.

Researchers from several disciplines have evaluated whether anxiety worsens the prognosis for patients with documented CHD. Moser and Dracup used the Brief Symptom Inventory to measure anxiety in 86 patients with AMI. Patients with higher levels of state anxiety were 4.9 times more likely to develop in-hospital complications including ventricular tachycardia, ventricular fibrillation, ischemia, and reinfarction. Thomas and colleagues followed 348 patients from the Cardiac Arrhythmia Suppression Trial to study the effects of psychosocial factors on survival. In a model that included physiological and psychological variables, level of state anxiety independently predicted 3 month survival after AMI. In a study by Frasure-Smith and associates, patients with AMI who had higher anxiety scores on the state portion of the
State-Trait Anxiety Inventory experienced more frequent recurrent cardiac events during the first post-AMI year. Remarkably, anxiety was the only variable from among several cardiac and psychological variables that predicted recurrent AMI. In contrast, Lane and colleagues reported that neither state nor trait anxiety predicted cardiac or all-cause mortality for 288 patients with AMI. In this study, 52% of patients had Killip class II-IV HF, leading the researchers to suggest that anxiety may best predict mortality for those with lower levels of cardiac morbidity. Similarly, patients with AMI and high anxiety who participated in the Oxford Myocardial Incidence Study did not have higher mortality rates than nonanxious patients.

Few investigators have evaluated how anxiety affects patients with HF. For patients with HF, anxiety predicted functional status at 1 year but not rehospitalization or mortality. The effects of high anxiety often persist over time. Persons with recent AMI and depressed left ventricular function who were anxious had a higher incidence of adverse cardiac events and cardiac death in the subsequent 6-10 years. In a recent study, higher state anxiety scores predicted 5-year cardiac mortality for patients with AMI. However, when the researchers adjusted for cardiac disease severity, this relationship did not remain significant. Interestingly, results from one study showed that patients with higher anxiety had lower mortality at 5 years. The investigators proposed that anxiety may motivate patients' behaviors.

The above findings emphasize that it is essential for clinicians to accurately assess and promptly manage anxiety in patients with CHD. Yet, healthcare professionals often fail to consider anxiety and other psychosocial factors when caring for their patients. Furthermore, clinicians often diagnose anxiety by using their clinical judgment instead of a reliable or valid instrument that is designed to measure anxiety. Results from a recent study indicated that the majority of critical care nurses believed that anxiety could be life threatening or harmful. Yet,
in another study, critical care nurses documented an anxiety assessment in only 39% of patients with AMI while physicians assessed only 6% of patients for anxiety.19,21 Additionally, clinicians did not use objective indicators to describe anxiety and documented a follow-up anxiety assessment in only 24% of patients that they had identified as being anxious. The most striking finding from this study was that there was no relationship between clinician generated and patient generated anxiety ratings.

In a recent survey, critical care nurses indicated that agitation, increased blood pressure, increased heart rate, patients’ verbalization of anxiety, and restlessness are the most important indicators of anxiety.22 In the qualitative arm of the same study, nurses reported that restlessness, increased heart rate, agitation, increased blood pressure, increased respiratory rate, and increased diaphoresis were the defining attributes of anxiety.23 These findings are concerning because physiologic symptoms of anxiety may not be as useful when assessing acutely ill patients for anxiety.2 Furthermore, it can be difficult to differentiate between signs and symptoms of anxiety and indicators that reflect deteriorations in the patient’s physiologic status. Clinicians who rely on changes in heart rate or blood pressure signs to diagnose anxiety may underestimate the presence of anxiety in their patients.22,23 Clinicians may more accurately recognize anxiety by actively listening to patient reports of anxiety22 and by assessing psychological symptoms that are associated with anxiety.2

Although critical care nurses use heart rate and blood pressure as indicators of anxiety, little is known about whether these measures accurately reflect anxiety in acutely ill patients. The purpose of this study was to determine whether heart rate and blood pressure were related to level of anxiety at the time of measurement in three groups of participants: 1) patients with chronic advanced HF; 2) patients with AMI; and 3) healthy individuals.
Methods

Design

In this descriptive study we determined the relationship between heart rate and blood pressure, and anxiety level in three groups of individuals. Blood pressure and heart rate were assessed at the same time as anxiety level. Data from two separate studies are presented in this paper.

Sample and Setting

For purposes of this study, we combined data from two studies: 1) study of anxiety among patients experiencing AMI; and 2) study of the impact of a biofeedback-relaxation intervention in patients with HF. In the AMI study, patients were enrolled based on the following criteria: 1) diagnosis of AMI using typical electrocardiogram changes and enzyme levels to confirm AMI; 2) pain free and hemodynamically stable at the time of interview; 3) AMI not experienced in an institutional setting; 4) cognitively able to participate in a short interview; 5) free of serious debilitating co-morbidities such as cancer or renal failure; and 6) able to speak English. The study was conducted in patients' hospital rooms, which were typically in the critical care unit or telemetry unit.

In the biofeedback-relaxation study, patients with HF and healthy individuals were enrolled. Inclusion criteria for the patients included the following: 1) diagnosis of advanced chronic HF with New York Heart Association (NYHA) functional classification II to IV, and left ventricular ejection fraction < 30%; 2) have undergone evaluation of HF and optimization of medical therapy, and have not been referred for heart transplantation; 3) no history of cerebral vascular accident; 4) no history of major extremity vascular problems; 5) no recent (within 6 months) myocardial infarction; and 6) not receiving sedatives, narcotics, or hypnotics. Healthy
individuals were included if they had no history of coronary heart disease, diabetes, or other major illnesses. Participants from the biofeedback-relaxation study were assessed in an outpatient clinical research center of a major academic medical center.

**Measurement**

Anxiety, heart rate, and blood pressure were measured in the same manner in each group of participants. The following sociodemographic and clinical data were collected: age, gender, ethnicity, marital status, education level, smoking status, NYHA classification, heart rate, blood pressure, and history of AMI, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty, diabetes, and hypertension.

**Anxiety.** State anxiety was measured using the anxiety subscale of the Brief Symptom Inventory. This six-item anxiety subscale is especially useful when studying acutely ill patients because it is sensitive, brief, reliable and valid, and does not rely on clinical symptoms to indicate feelings of anxiety. Instruments that include clinical indicators of emotions have been criticized for overestimating the level of the emotion in acutely ill patients. For each of the six items on the scale, patients rate their level of distress from 0 ("not at all") to 4 ("extremely"). The scores are summed and averaged. The averaged score reflects the patient’s overall level of state anxiety.

**Heart rate and blood pressure.** Heart rate and blood pressure data were collected using two methods to establish the validity of the measurement. The same methods were used for participants from both studies. Trained research assistants assessed heart rate by auscultating the apical pulse for a full minute, and determined blood pressure using the technique outlined by the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood
Heart rate and blood pressure were also measured using a calibrated noninvasive blood pressure monitor.

**Procedure**

The appropriate Institutional Review Boards approved this study and all participants gave informed, written consent. Healthcare providers referred their patients to the respective studies. We used flyers to recruit healthy participants. Trained research assistants who were all cardiovascular nurses explained the study requirements to potential participants and collected all data.

Data in the AMI study were collected within 72 hours of patients' admission to the hospital with AMI symptoms. The AMI study included no intervention and a one-time assessment of anxiety during a patient interview. Heart rate and blood pressure data were collected immediately prior to the anxiety assessment. In the biofeedback-relaxation study, anxiety, heart rate, and blood pressure measurements were collected in an outpatient setting during a two-hour data collection session. Data used in the present analyses were collected at baseline, before the intervention was instituted.

**Statistical Analyses**

All data were entered into a personal computer and analyzed with SPSS software, version 11.5. Data are presented as frequencies and means ± standard deviations. Using Kendall's tau-b, correlation coefficients were computed between the mean anxiety level of each group and heart rate, systolic blood pressure, and diastolic blood pressure. We also subdivided each of the three groups into a “high anxiety group” and a “low anxiety group” based on the median split of the anxiety scores. We performed two-tailed t tests to evaluate whether the high and low anxiety
groups manifested differences in mean heart rate, systolic blood pressure, and diastolic blood pressure at the time of measurement. A $P$-value of $< .05$ was considered statistically significant.

**Results**

**Sample Characteristics**

The sample of 117 patients was composed of 32 (27.4%) patients with HF, 54 (46.1%) patients with AMI, and 31 (26.5%) healthy individuals. The sociodemographic and clinical characteristics of the sample are summarized in Table 1. The mean age of the sample was 56.8 years; patients in the AMI group were older than patients in the HF and normal groups ($P < .05$). Over half (56.4%) of the participants were married, 59.8% were female, and the majority (79.5%) were Caucasian.

**Anxiety Level**

The mean anxiety scores for the HF, AMI, and healthy groups were .98, .52, and .58, respectively, which in each group is substantially greater than the norm referenced anxiety level of $0.35 \pm 0.45$. In this study, 62.5% of patients with HF, 38.8% of patients with AMI, and 54.9% of healthy individuals reported higher anxiety than the norm reference.

**Relationship Between Anxiety and Physiologic Variables**

The results of the correlational analyses presented in Table 2 show that there were no correlations between anxiety and heart rate or diastolic blood pressure. There were only two significant correlations. Interestingly, higher anxiety was associated with lower systolic blood pressure in healthy individuals ($r = -.27, P < .05$) and patients with AMI ($r = -.23, P < .05$).

As previously described, the patients were divided into high and low anxiety subgroups. As shown in Table 3 mean systolic blood pressure, diastolic blood pressure, and heart rate were
similar for patients in the high and low anxiety subgroups regardless of the presence or absence of CHD.

**Discussion**

Results from this study show that heart rate and blood pressure do not accurately reflect level of anxiety as reported by patients with AMI or HF and healthy individuals. Traditionally, clinicians have assessed acutely ill patients for high anxiety by detecting changes in heart rate and blood pressure; however, findings from this study do not support this practice. From a physiologic perspective, it would seem that increased anxiety should be associated with an elevated heart rate and blood pressure. Although the purpose of this study was not to test the mechanisms linking anxiety with poor outcomes, we found that heart rate and blood pressure were not elevated in this sample of participants regardless of their anxiety level. In fact, an unexpected finding was that higher anxiety was associated with lower systolic blood pressure in healthy individuals and in patients with AMI. We propose several explanations for our findings.

First, although it is a long-held belief that anxiety is associated with increased heart rate and blood pressure, examination of the existing evidence reveals several points that counter this belief: 1) individuals with even extreme anxiety do not respond in a homogenous manner, given that some persons exhibit an increase in heart rate and blood pressure while others show no heart rate or blood pressure response; 2) when investigators reported that anxiety was associated with an increased heart rate and blood pressure, the increases often were minimal and not clinically important; 3) many investigators failed to show any change in heart rate or blood pressure in response to acute anxiety; and 4) the physiologic response to anxiety is far more complex than originally thought and involves differential responses depending on which brain hemisphere is activated.
Second, patients with AMI or HF who are anxious and on beta blockers may not manifest a rapid heart rate or elevated blood pressure. In our sample, 93% of the AMI patients were on beta blockers. This does not, however, explain the lack of relationship between anxiety and blood pressure and heart rate in patients with HF (HF patients on beta-blockers were excluded from the biofeedback-relaxation study) and in healthy participants.

Third, heart rate and blood pressure are gross indicators of sympathetic tone and therefore may not be accurate indicators of anxiety, especially when it is sustained. Particularly among cardiac patients, whose autonomic nervous system tone is altered acutely and chronically, adaptation to sustained sympathetic nervous system activation may include blunting of the heart rate and blood pressure response. For example, among patients with HF, baroreceptor function is altered early and contributes to the development and progression of HF. Consequently, heart rate and blood pressure responses to changes in autonomic nervous tone may be distorted.

Fourth, acutely ill patients who are anxious may exhibit anxiety in a variety of ways and either may not be able to mount an overt physiologic response to anxiety or the response may be masked by the complex pathophysiologic responses of the presenting illness. For example, volume status abnormalities, pain, or changes in patient activity all affect heart rate and blood pressure. Additionally, while some healthy or ill individuals who initially experience intense, acute anxiety may exhibit tachycardia and hypertension, this physiological response may not persist as the individual experiences chronic anxiety that persists for hours or days.

We recommend that clinicians assess anxiety using an instrument designed for that purpose. The principle finding of this study indicates that heart rate and blood pressure are not reliable indicators of anxiety for patients with AMI and HF. Although one could argue that heart rate tended to be higher in the high anxiety groups, the difference in heart rate was neither
statistically nor clinically significant. Clinicians who rely on changes in heart rate and blood pressure as indicators of patient anxiety may fail to recognize and treat anxiety.

Further prospective research is needed to identify the most efficient and effective method of assessing anxiety in acutely ill patients. Clinicians need a brief, valid, and reliable instrument that is easy to administer. However, some critically ill patients are not capable of either completing such an instrument or subjectively reporting anxiety. Therefore, future research is needed to explore whether any physiologic measures are reliable indicators of patient anxiety.

A limitation of this study is that we measured anxiety at one time point. More research is needed to evaluate whether our findings would persist over time. A second limitation is that healthy individuals had higher than expected levels of anxiety. Interestingly, even healthy individuals did not manifest an elevated heart rate or blood pressure.

In summary, elevated heart rate and blood pressure do not accurately reflect level of anxiety as reported by patients with either AMI or HF and healthy individuals. Results of this study indicate that use of an anxiety assessment instrument will enable clinicians to more accurately assess patient anxiety.
References


16. Frasure-Smith N, Lesperance F. Depression and other psychological risks following myocardial infarction. *Arch Gen Psychiatry.* 2003;60:627-636.


### Table 1 Comparison of baseline characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Entire Sample (N = 117)</th>
<th>HF Group (N = 32)</th>
<th>MI Group (N = 54)</th>
<th>Healthy Group (N = 31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.8 ± 13.9</td>
<td>53.5 ± 13.3</td>
<td>62.8 ± 14.4</td>
<td>49.3 ± 8.3</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>72.8 ± 12.9</td>
<td>69.5 ± 13.6</td>
<td>76.7 ± 13.2</td>
<td>69.8 ± 9.9</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>122.2 ± 19.2</td>
<td>114.4 ± 16.9</td>
<td>121.1 ± 17.2</td>
<td>131.6 ± 21.2</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>70.4 ± 16.0</td>
<td>74.3 ± 14.2</td>
<td>60.5 ± 10.4</td>
<td>83.6 ± 14.8</td>
</tr>
<tr>
<td>Education (years)</td>
<td>13.7 ± 3.1</td>
<td>13.8 ± 1.9</td>
<td>12.4 ± 2.6</td>
<td>16.0 ± 3.8</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Male</td>
<td>47 (40.2)</td>
<td>22 (68.8)</td>
<td>12 (22.2)</td>
<td>13 (41.9)</td>
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<td>Female</td>
<td>70 (59.8)</td>
<td>10 (31.3)</td>
<td>42 (77.8)</td>
<td>18 (58.1)</td>
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<tr>
<td>Marital status</td>
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<tr>
<td>Single</td>
<td>14 (12.0)</td>
<td>4 (12.5)</td>
<td>3 (5.6)</td>
<td>7 (22.6)</td>
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<td>Married</td>
<td>66 (56.4)</td>
<td>21 (65.6)</td>
<td>32 (59.3)</td>
<td>13 (41.9)</td>
</tr>
<tr>
<td>Divorced/separated</td>
<td>21 (17.9)</td>
<td>5 (15.6)</td>
<td>8 (14.8)</td>
<td>8 (25.8)</td>
</tr>
<tr>
<td>Widowed</td>
<td>15 (12.8)</td>
<td>1 (3.1)</td>
<td>11 (20.4)</td>
<td>3 (9.7)</td>
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<td>Cohabitate</td>
<td>1 (0.9)</td>
<td>1 (3.1)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Ethnicity</td>
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</tr>
<tr>
<td>Nonhispanic White</td>
<td>93 (79.5)</td>
<td>27 (84.4)</td>
<td>43 (79.6)</td>
<td>23 (74.2)</td>
</tr>
<tr>
<td>Black</td>
<td>20 (17.1)</td>
<td>4 (12.5)</td>
<td>10 (18.5)</td>
<td>6 (19.4)</td>
</tr>
<tr>
<td>American Indian</td>
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<td>Asian</td>
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<td>Other</td>
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<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>1 (3.2)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>25 (21.4)</td>
<td>6 (18.8)</td>
<td>17 (31.5)</td>
<td>1 (3.1)</td>
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<tr>
<td>History of MI</td>
<td>31 (26.5)</td>
<td>15 (46.9)</td>
<td>16 (29.6)</td>
<td>0 (0.0)</td>
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<tr>
<td>History of CABG</td>
<td>15 (12.8)</td>
<td>8 (25.0)</td>
<td>7 (13.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>History of PTCA/stent</td>
<td>14 (12.0)</td>
<td>5 (15.6)</td>
<td>9 (16.7)</td>
<td>0 (0.0)</td>
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<tr>
<td>History of HTN</td>
<td>50 (42.7)</td>
<td>12 (37.5)</td>
<td>34 (63.0)</td>
<td>4 (12.9)</td>
</tr>
<tr>
<td>History of diabetes</td>
<td>23 (19.7)</td>
<td>6 (18.8)</td>
<td>17 (31.5)</td>
<td>0 (0.0)</td>
</tr>
</tbody>
</table>

Values in table are mean ± standard deviation or actual number of patients followed by percentage in parentheses. Column percents may not equal 100% due to missing data.

HF = heart failure; MI = myocardial infarction; CABG = coronary artery bypass grafting; PTCA = percutaneous transluminal coronary angioplasty; HTN = hypertension
Table 2  Correlations between anxiety, heart rate, and blood pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>HF Group Total Anxiety</th>
<th>MI Group Total Anxiety</th>
<th>Healthy Group Total Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>.26</td>
<td>.11</td>
<td>.07</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>.10</td>
<td>-.23*</td>
<td>-.27*</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>.24</td>
<td>-.10</td>
<td>-.25</td>
</tr>
</tbody>
</table>

*P < .05 by Kendall’s tau

HF = heart failure; MI = myocardial infarction; BP = blood pressure

Anxiety assessed by the Brief Symptom Inventory subscale
<table>
<thead>
<tr>
<th></th>
<th>High anxiety group</th>
<th>Low anxiety group</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HF Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats per minute)</td>
<td>73.9 ± 13.2</td>
<td>65.7 ± 13.2</td>
<td>.09</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>117.9 ± 21.1</td>
<td>111.4 ± 11.8</td>
<td>.28</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>79.1 ± 13.9</td>
<td>70.0 ± 13.5</td>
<td>.07</td>
</tr>
<tr>
<td><strong>MI Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats per minute)</td>
<td>79.5 ± 13.9</td>
<td>73.4 ± 12.1</td>
<td>.09</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>116.8 ± 14.5</td>
<td>125.1 ± 18.8</td>
<td>.08</td>
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<tr>
<td>Diastolic BP (mm Hg)</td>
<td>59.3 ± 8.7</td>
<td>61.6 ± 11.9</td>
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<tr>
<td><strong>Healthy Group</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats per minute)</td>
<td>69.9 ± 10.8</td>
<td>69.6 ± 9.0</td>
<td>.95</td>
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<tr>
<td>Systolic BP (mm Hg)</td>
<td>126.0 ± 21.0</td>
<td>138.5 ± 19.9</td>
<td>.10</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>80.5 ± 16.0</td>
<td>87.4 ± 12.8</td>
<td>.19</td>
</tr>
</tbody>
</table>

HF = heart failure; MI = myocardial infarction; BP = blood pressure