2010

COGNITIVE THERAPY FOR THE TREATMENT OF DEPRESSIVE SYMPTOMS IN PATIENTS WITH HEART FAILURE

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ABSTRACT OF DISSERTATION

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ABSTRACT OF DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Nursing
at the University of Kentucky

By
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Lexington, KY

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Lexington, KY

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ABSTRACT OF DISSERTATION

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Depressive symptoms are common in patients with heart failure (HF) and adversely affect mortality, morbidity, and health-related quality of life. Cognitive therapy (CT) has been proposed as a non-pharmacological treatment for depressive symptoms in patients with HF. However, there is currently little evidence to support use of CT in patients with HF.

The purpose of this dissertation was to develop and test a brief, nurse-delivered CT intervention for the treatment of depressive symptoms in patients with HF. Prior to testing the intervention, preliminary work was conducted resulting in four manuscripts: 1) a review of the evidence for CT in treating depressive symptoms in patients with cardiovascular conditions, 2) a description of living with depressive symptoms in patients with HF and strategies that could be used to manage these symptoms, 3) a review of measures of negative thinking and the identification of a measure of negative thinking that can be used in patients with HF, and 4) an evaluation of the psychometric properties of this measure. Based on information from these manuscripts, a randomized, controlled pilot study was conducted to test the effects of a brief CT intervention on outcomes of hospitalized patients with HF who report depressive symptoms.

Forty-two hospitalized patients with HF with mild-moderate depressive symptoms were randomized to a brief CT intervention focused on reducing negative thoughts with thought-stopping and affirmations, or to usual care control. Both groups experienced improvements in depressive symptoms, health-related quality of life, and negative thinking at one week and three months. However, the intervention group experienced longer cardiac event-free survival and fewer cardiovascular hospitalizations and emergency department visits at three months when compared to the control group.

This dissertation has fulfilled an important gap in the evidence base for depression treatment in patients with HF by demonstrating that a nurse-delivered, brief CT intervention may improve cardiac event-free survival in patients with HF. This brief CT intervention is replicable, practical, can be delivered by acute care nurses, and may improve clinical outcomes in patients with HF. Additional research is needed to determine the effects of the intervention on long-term outcomes in patients with HF

KEYWORDS: heart failure, depression, depressive symptoms, cognitive therapy, negative thinking

Rebecca Dekker
Student's Signature

May 6, 2010
Date

COGNITIVE THERAPY FOR THE TREATMENT OF DEPRESSIVE SYMPTOMS IN PATIENTS WITH HEART FAILURE

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For Dan and Clara

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CHAPTER ONE:

Introduction

1. Depression in patients with heart failure

Heart failure (HF) is a chronic syndrome which affects 5.8 million people in the United States (U.S.).[1] Age-adjusted five-year survival rates for individuals with HF have improved from 43% in 1979-1984 to 52% in 1996-2000.[2] Unfortunately, despite improved survival, patients with HF continue to experience high morbidity in the form of frequent hospitalizations. The diagnosis of HF is the most common reason for hospitalization among elderly adults in the U.S.,[3] and HF currently accounts for 1.08 million hospital discharges per year—a 171% increase since 1979.[1]

In addition to high morbidity rates, patients with HF frequently experience major depressive disorder and depressive symptoms. In a meta-analysis of data from 23 studies with over 10,000 participants with HF, researchers estimated that the prevalence of major depressive disorder, as determined by a diagnostic clinical interview, was 20% in outpatients and inpatients with HF. When researchers used a more liberal estimate (self-reported depression questionnaire), the prevalence rate of depressive symptoms was 30%.[4]

Both major depressive disorder and self-reported depressive symptoms have significant clinical consequences in patients with HF. Patients with HF who have depressive symptoms are twice as likely to die and two and a half times more likely to be re-hospitalized than patients who do not have depressive symptoms.[4] Researchers have also demonstrated a dose-response relationship between depressive symptoms and long-term survival in patients with HF. Patients with mild, moderate, and severe depressive symptoms as measured by the Beck Depression Inventory are respectively, 21%, 53%, and 83% more likely to die during a seven-year follow-up period than patients who have no depressive symptoms.[5]

The reasons for the association between depressive symptoms and shortened survival in patients with HF are unclear. Depression and HF are two conditions that share important pathophysiological and behavioral links. Researchers have suggested that depression in patients with HF may be associated with proinflammatory cytokines, arrhythmias, sympathetic nervous system activation, and increased platelet aggregation, all of which may worsen the course of HF. Furthermore, depression may contribute to poor outcomes in HF via behavioral links such as non-adherence and poor social support.[6]

Researchers have found that the presence of depressive symptoms is an independent predictor of worsened health-related quality of life (HRQOL) in patients with HF.[7, 8] HRQOL is a subjective, patient-centered outcome that refers to how a health condition affects a person's total well-being. Researchers are beginning to recognize that HRQOL may be more important than quantity of life,[9] given that patients with HF are experiencing longer survival and must adjust to life with a chronic health condition.

The problems associated with depressive symptoms in patients with HF have been well-described. However, there is a lack of research testing interventions for depressive symptoms in patients with HF.[10] Given the significant clinical consequences of depressive symptoms in patients with HF, it is imperative that researchers develop and test interventions for the treatment of depressive symptoms in patients with HF.

2. Treatment of depressive symptoms in patients with heart failure

The 2009 guidelines for HF management from the American College of Cardiology and the American Heart Association provide no direction for the treatment of depression in patients with HF. The guidelines state, "Good evidence exists for the critical importance of delivering comprehensive supportive care to these patients, including the assessment and treatment of… depression."[11] The lack of specificity in these guidelines reflects the state of the science regarding the treatment of depression in patients with HF, as there has been insufficient research on treatments for depressive symptoms in patients with HF.

In the general population, evidence suggests that current pharmacological treatments for depression are only modestly effective compared to placebo. In 2008, Kirsch et al.[12] conducted a meta-analysis examining antidepressant efficacy using clinical trial data submitted to the Federal Food and Drug Administration and obtained with the Open Records Act. The authors found that among patients with moderate depression, there was a statistically significant difference between antidepressant and placebo use, but this difference did not meet criteria for clinical significance. Both drug and placebo resulted in modest improvements in depression. Among patients with the most severe levels of depression, antidepressant use resulted in a statistically and clinically significant improvement in depression compared to placebo. However, the researchers proposed that this improvement could have been due to a reduced placebo effect among the severely depressed, rather than an antidepressant effect.

Likewise, in the only randomized, controlled trial to date to test the effects of antidepressant therapy on depression in patients with HF (SADHART-HF), O'Connor et

al.[13] found that depressed patients with HF who received antidepressants did not experience a statistically significant improvement compared to placebo. However, both the intervention group and the placebo group received a nursing intervention as a part of the study protocol, and both groups experienced considerable reductions in depressive symptoms. The nursing intervention consisted of one-hour telephone counseling sessions that took place every other week for sixteen weeks and were delivered by a psychiatric nurse (Personal communication, C. O'Connor, March 3, 2010). The authors suggest that the nursing intervention was so powerful that the effects of the nursing intervention obscured any effect of the antidepressant.

The results of the SADHART-HF trial[13] suggest that non-pharmacological interventions carried out by nurses may be effective for the treatment of depressive symptoms in patients with HF. Non-pharmacological interventions have several advantages over pharmacological treatments such as a lack of drug-drug interactions, immediate short-term relief of symptoms, and greater involvement of patients in their own self-care.

Cognitive Therapy (CT) is a non-pharmacological intervention for depression that was developed by Beck in 1967,[14] has been used successfully to treat depression in multiple populations,[15] and may be useful for treating depression in patients with HF. Nurses can be trained to administer CT as a nursing intervention. Cognitive Therapy is compatible with common nursing interventions such as teaching patients to accurately appraise stressors, determining the best coping method, and increasing perceived control.[16]

Beck's cognitive model of depression provides the theoretical basis for CT. The main tenet of the cognitive model is that negative thinking influences the emotional, behavioral, and somatic symptoms associated with depression.[17] Negative thinking can be defined as cognitions about the self, world, future, and interpersonal relationships.[18] Negative thinking originates in childhood and dominates the perceptions of the depressed individual.[19] Examples of negative thoughts include, "I'm worthless," "Everything I do is a failure," and "Nothing's ever going to work out for me."[18] Negative thinking occurs automatically and persists over time. Although the content of negative thoughts is irrational, these thoughts are believable to the person experiencing them.[14]

The goal of CT is to reduce depressive symptoms by redirecting negative thinking. During CT, the therapist teaches the client to identify, analyze, and question negative thoughts. For example, the therapist helps the client identify negative thoughts

such as “I’m a burden to my family.” Together, the therapist and the client explore the evidence for the negative thought. After evaluating the thought, the therapist guides the client to challenge the thought. By challenging and changing thoughts, the client is able to modify negative emotions and behaviors.[17] Although CT holds promise as a potential treatment for depressive symptoms in patients with HF, research is needed to demonstrate its efficacy as a treatment for depressive symptoms in patients with HF.

Therefore, the purpose of this dissertation was to develop and test a CT intervention for managing depressive symptoms in patients with HF. The chapters of this dissertation represent the systematic development of my initial program of research. First, a review of the literature was performed to establish the evidence for CT as a treatment for depressive symptoms in patients with HF. Second, a qualitative research study was conducted that identified negative thinking as an important target for a CT-based intervention that would be appropriate for patients with HF. Third, a critical review of the literature was conducted to identify the best instrument for measuring negative thinking in patients with HF. Fourth, evidence was provided for the reliability and validity of the selected measure of negative thinking in patients with HF. And finally, a clinically feasible, brief CT intervention for depressive symptoms targeting negative thinking in patients with HF was tested in a randomized clinical trial.

3. Summary of subsequent chapters

Chapter Two is the critical review of the literature[20] to determine the empirical support for the use of CT in patients with HF. Because there was limited research on CT in patients with HF, the review was expanded to include research studies CT across all cardiovascular-related conditions. Insights from these studies were interpreted with respect to applicability to patients with HF. Electronic databases were searched from 1980 through 2009 using the following key words: depression, cognitive therapy, cardiovascular, diabetes, and stroke. Articles were included if they met the following criteria: randomized, controlled trial; cognitive therapy intervention; depression or depressive symptoms measured as an outcome; sample consisted of patients with cardiovascular disease, non-hemorrhagic stroke, or diabetes. Fourteen articles met the inclusion criteria and were extracted for review. Positive effects of CT on depression or depressive symptoms were reported in eight of the fourteen studies. Possible reasons for the mixed results, as well as recommendations for the design of future studies testing CT in patients with HF are provided.

Chapter Three is the qualitative research study[21] conducted to obtain information

needed to guide the development of a CT intervention for depressive symptoms in patients with HF. The research questions in this qualitative descriptive study were: How do persons with HF describe their depressive symptoms? What strategies do they use to reduce depressive symptoms? The sample consisted of ten outpatients with chronic HF who had clinically significant depressive symptoms as measured by the Beck Depression Inventory version II or a history of depression. Data were collected via taped, individual, 30-60 minute interviews which took place in the participants' homes. Content analysis was conducted using the qualitative software ATLAS ti (v5). The participants identified negative thoughts that exacerbated their depressed mood. The most frequently mentioned strategy that participants used to manage their depressive symptoms was positive thinking, which included the use of affirmations. The findings from this study were used to develop a brief CT intervention which will be presented in Chapter Six.

Chapter Four is the critical review and analysis[22] of three measures of negative thinking to identify the best instrument for measurement of negative thinking in patients with HF. Three of the most widely used instruments that measure negative thinking were selected for review: the Crandell Cognitions Inventory, Automatic Thoughts Questionnaire, and Cognitive Checklist- Depression. The three instruments were described and their strengths and weaknesses were systematically compared, with particular attention to strength of evidence for reliability and validity. The Crandell Cognitions Inventory was identified as the instrument that has the best potential for measuring negative thinking in patients with HF, and recommendations for future psychometric testing were provided.

Chapter Five is the report on the psychometric properties of the Crandell Cognitions Inventory (CCI) in patients with HF, to establish reliability and validity of this measure of negative thinking in patients with HF. The purpose of this study was to evaluate the internal consistency reliability and construct validity of the CCI in patients with HF. Cross-sectional questionnaire data were collected from 179 outpatients with HF using the CCI, Beck Depression Inventory-II, and Brief Symptom Inventory- anxiety subscale. Reliability was assessed with Cronbach's alpha; construct validity was evaluated with factor analysis and hypothesis testing. The findings provided evidence for the reliability and validity of the CCI for measuring negative thinking in patients with HF.

Chapter Six presents the findings of the randomized, controlled pilot study testing a brief CT intervention in hospitalized patients with HF. Patients were eligible to participate if they were admitted to the hospital with a diagnosis of HF and were

experiencing mild to moderate depressive symptoms as measured by the Beck Depression Inventory-II. A total of forty-two patients were randomized to the intervention and control groups. The intervention group received a single 30-minute brief CT intervention at the bedside and a one-week telephone booster, and the control group received usual care. Depressive symptoms, negative thinking, and HRQOL were measured at baseline, one week, and three months. Three-month cardiac event-free survival data were also collected by patient interview and hospital record review. The results showed that both groups experienced significant improvements in depressive symptoms, negative thinking, and HRQOL over time. However, the intervention group experienced longer cardiac event-free survival compared to the usual care group (80% alive without a cardiac event vs. 40%, $p = .048$).

Chapter Seven is an integrated discussion in which the prior chapters of this dissertation are synthesized to advance the state of the science of treatment of depressive symptoms in patients with HF and make recommendations for practice and future research.

CHAPTER TWO:

Cognitive Therapy for Depressive Symptoms in Patients with Heart Failure: A Critical Review

1. Introduction

Depression is a significant problem in patients with heart failure (HF). One in five persons with HF has clinical depression,[1] and up to 48% have clinically significant depressive symptoms.[2] According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR), a major depressive episode, sometimes referred to as clinical depression, consists of five or more symptoms which are present for most of the day, almost daily, for at least two weeks. One of these symptoms must be either depressed mood or loss of interest or pleasure in usual activities, and the symptoms must cause significant distress in social, occupational, or other areas of functioning (Table 2.1).[3] However, patients can experience clinically significant depressive symptoms without the presence of major depressive disorder.[4] Depressive symptoms may include depressed mood, irritability, guilt, hopelessness, low self-esteem, fatigue, sleep disturbances, appetite change, and inability to concentrate.[5]

The adverse effects of clinical depression and depressive symptoms on mortality and hospitalizations in patients with HF have been well documented.[1, 2, 6-8] Results from a recent meta-analysis demonstrated that patients with HF who have depressive symptoms are more than twice as likely to die or experience a cardiac event compared to patients without depressive symptoms.[1] Moreover, the presence of depressive symptoms has a negative impact on every dimension of health-related quality of life in patients with HF, including physical functioning, social functioning, and mental health.[2]

The problems associated with depression and depressive symptoms in patients with HF are well described. It is time for researchers to test interventions. Importantly, there is a lack of research on non-pharmacological interventions for depressive symptoms in patients with HF.[9] Cognitive therapy (CT) has been used successfully to treat depression in multiple populations.[10, 11] Therefore, it may also be useful for treating depression in patients with cardiovascular illnesses, including HF. The purpose of this critical review was to examine the empirical support for the use of CT in treating depression and depressive symptoms in patients with cardiovascular-related illnesses.

2. Background

Beck, a psychiatrist, developed the Cognitive Model of depression in 1967 to explain the psychological processes that occur in depression. The underlying

assumption behind the Cognitive Model was that human minds are biased and cannot interpret stimuli objectively. This bias leads to cognitive errors, or dysfunctional thinking.[10] The Cognitive Model holds that dysfunctional thinking influences the emotions, behaviors, and psychosomatic symptoms associated with depression. Thus, interventions aimed at changing dysfunctional thinking should improve the emotional, behavioral, and somatic symptoms of depression.[11]

Cognitive therapy, also referred to as cognitive behavioral therapy, is the psychotherapeutic intervention based on the Cognitive Model of depression. The primary goal of CT is to alter emotions and behavior by redirecting negative cognitive processes. CT is typically a short term therapy that consists of 4 to 14 sessions, depending on the individual's progress. The role of the therapist is to develop a collaborative, therapeutic relationship with the client and to teach the client to become his or her own therapist. The therapist teaches the client to identify, analyze, and question dysfunctional thinking. For example, the therapist may help the client to identify negative thoughts such as "I'm a burden to others." Together, the therapist and the client explore the evidence behind this negative thought. After evaluating the rationale for the thought, the therapist then guides the client to challenge the thought, and eventually change the client's thinking. By creating changes in thinking, the client may modify negative emotions and behaviors.[11]

CT offers several potential advantages for the treatment of depression and depressive symptoms in patients with HF. First, CT is a non-pharmacological intervention. Non-pharmacological interventions may have several advantages over pharmacological treatments such as a lack of drug-drug interactions, immediate short-term relief of symptoms, and greater involvement of patients in their own self-care. Second, CT is an intervention that nurses can be trained to administer. CT is compatible with common nursing interventions of teaching patients to accurately appraise stressors, determining the best coping method, and increasing perceived control. Despite the potential benefits of CT, its effectiveness in patients with cardiovascular illness including HF remains unknown. The following review provides a critical analysis of the existing research on the effectiveness of CT for treating depression or depressive symptoms in patients with cardiovascular illnesses.

3. Methods

The databases searched for relevant literature were PUBMED, PsychInfo, CINAHL, and MEDLINE. Keywords included depress* and cognitive therapy (or cognitive behavioral therapy) and cardiovascular (or heart failure, chronic illness,

coronary artery disease, cardiac, stroke, or diabetes). The search was limited to English language papers published between 1980 and 2009.

Studies were included in the review if they met the following criteria: randomized, controlled trial; a cognitive therapy intervention; depression or depressive symptoms measured as an outcome; sample consisted of patients with cardiovascular disease, non-hemorrhagic stroke, or diabetes. Diabetes mellitus commonly coexists with HF[12] and is an important risk factor for the development of HF.[13] Thus samples that contained patients with diabetes were also included.

The search resulted in 335 articles of which the titles or abstracts were screened for inclusion criteria. Reference lists of relevant articles were screened for additional studies. A total of fourteen papers met the inclusion criteria and were extracted for review.

4. Results

4.1. Cardiovascular disease

Eight studies were identified in which the impact of CT on depression in patients with cardiovascular illness was evaluated (Table 2.2).

In the largest randomized controlled trial on CT in cardiovascular disease to date, the ENRICHD (Enhancing Recovery in Coronary Heart Disease) investigators[14] compared the impact of CT with usual care on depressive symptoms and event-free survival in 2,481 patients with a recent myocardial infarction (MI). Patients were eligible to participate if they were defined as depressed on a diagnostic interview or if they had low perceived social support. Patients in the intervention group attended 6 to 19 individual or group CT sessions (median of 11 sessions) over six months in conjunction with selective serotonin reuptake inhibitor when indicated. The intervention group experienced a statistically significant decrease in depressive symptoms compared to the control group at six months. However, this difference was no longer present at 30 or 42 months of follow up. There was no difference in event-free survival between the intervention and control groups.

The lack of impact on event-free survival may have been due to several factors. The ENRICHD investigators assumed that CT should begin as soon as possible (within 28 days) after an MI. Thus, the study may have included patients in the control group who had transient rather than clinical depression and recovered without intervention. The investigators also assumed that the usual care group would not receive treatment for depression, but this was not the case. Antidepressant use was comparable in the

control group (21%) and intervention group (28%).[14-16] It is not known why a decrease in depressive symptoms in the intervention group in this study did not translate into improved outcomes. It may be that the decrease in depressive symptoms was insufficient to affect clinical outcomes. This conclusion is supported by the observation that while depressive symptoms in the intervention group decreased by 49%, depressive symptoms in the control group also decreased by 33%.[14]

More recently, Freedland et al. [17] compared the effects of CT, supportive stress management, and usual care for the treatment of depression in 123 patients who were post coronary artery bypass graft surgery (CABG). Patients were included if they scored 10 or higher on the Beck Depression Inventory (BDI) and met the DSM-IV criteria for depression based on a diagnostic interview. The investigators tested two separate interventions: 12 weeks of CT and 12 weeks of supportive stress management (progressive relaxation, controlled breathing, and imagery); both interventions were delivered in individual sessions by a trained clinical social worker or psychologist. Follow-up information on depression was collected at 3, 6, and 9 months using the BDI and the Hamilton Rating Scale for Depression.

The investigators found that patients in both intervention groups were more likely to experience remission from depression at all time points compared to patients in the usual care group. At nine months, 73% of the CT group and 57% of the stress management group experienced remission from depression, compared to 23% of patients in the usual care group ($p = .003$). This study was underpowered to detect differences between the CT and stress management arms of the study. Therefore, it is difficult to draw conclusions as to whether CT or stress management was superior for the treatment of depression in patients who are post-CABG. However, patients who received CT experienced greater improvement in secondary outcomes such as lower anxiety, hopelessness, and perceived stress; these results suggest that CT may offer more benefits to patients in comparison to stress management.

Twenty years ago, Burgess et al.[18] reported that an intervention combining CT, social support, and job return counseling failed to reduce depressive symptoms in patients who had recently experienced an acute MI. In this randomized, controlled trial, 180 patients were randomized to the intervention group or usual care. There were no differences in depression scores between groups at baseline, three months, or thirteen months of follow-up. The authors did not offer any explanations for the null effects on

depression. Females were underrepresented in the study; thus, it is not known whether the intervention would have been effective for females.

CT may be effective in reducing depressive symptoms in survivors of sudden cardiac death. Cowan et al[19, 20] tested the impact of CT, biofeedback therapy, and health education on depressive symptoms and mortality in 133 survivors of sudden cardiac death. Only 11% of the sample had depressive symptoms at baseline and all of these were male. Approximately half of the sample had chronic heart failure (NYHA Functional Class II-IV). The treatment group experienced a significant decrease in depressive symptoms compared to the control group. Although the study was not originally powered to detect differences in mortality, the treatment group experienced an 86% reduction in the risk of cardiac death compared to the control group. Therefore, the results of this study suggest that CT and biofeedback may be beneficial for survivors of sudden cardiac death, whether or not they are experiencing depressive symptoms.

Limited evidence suggests that CT may be more effective than exercise at reducing depressive symptoms in patients with coronary artery disease. Black et al[21] randomized 60 patients who had recently been hospitalized for a coronary event and were psychologically distressed to one of two groups: a special intervention that consisted of stress management, relaxation training, and CT administered by a psychiatrist, or a cardiac rehabilitation group that included exercise and risk reduction counseling. At six months, the CT group experienced a significant reduction in depressive symptoms compared to the cardiac rehabilitation group. There was no difference between groups with regard to rehospitalizations. There were several limitations of the study such as low representation of women (12%), exclusion of elderly (>80 years old), and lack of a true control group. Most importantly, adherence to the intervention was low. Less than 50% of participants attended more than one intervention session. Crossover between groups was also a problem, as six participants in the usual care group were treated with antidepressants or psychological counseling. Combined, these limitations severely weakened the internal and external validity of the study.

Results from three studies suggest that CT may be helpful for reducing depressive symptoms in patients with implanted cardioverter defibrillators (ICDs). Frizelle et al.[22] compared the impact of a cardiac rehabilitation and CT to a wait list control group on depressive symptoms in patients with ICDs. Despite the small sample size (N = 22), the intervention group experienced a significant reduction in depressive symptoms compared to the control group at three months. The intervention included

exercise, which makes it impossible to ascertain whether the CT alone had an impact on depressive symptoms.

In another study, Kohn et al.[23] evaluated the effects of CT on depressive symptoms in 49 patients with ICDs. This study was limited by the lack of a comprehensive measure of depressive symptoms at baseline. Only three biological indicators of depression were measured at baseline: sexual functioning difficulties, changes in appetite, and sleep disturbance. At nine months follow-up, the CT group reported fewer sexual functioning difficulties than the control group; however, sexual difficulties increased in both groups over time. The BDI version II (BDI-II) was administered only at follow-up. Although the CT group had a lower BDI-II score than the control group at follow-up, the lack of depression score data at baseline limits the conclusions that can be drawn from findings.

Lewin et al.[24] tested the effects of a brief, self-help CT booklet for the treatment of depressive symptoms in patients who were undergoing ICD implantation. In this RCT, eight ICD implantation centers were randomized to intervention or control. The intervention consisted of several booklets that were given to the patient and family by healthcare providers who had received a half day of training in administering the intervention. One of the booklets consisted of a self-help cognitive behavioral rehabilitation program, but the intervention in this booklet was not described in further detail. Patients in the intervention arm also received three phone contacts after ICD implantation to discuss their progress and set goals. The authors defined depression as a HADS score of 8 or greater, and patients were followed for six months.

In a logistic regression, the investigators found that patients in the intervention group were less likely to experience depression at six months compared to patients from control centers, after adjusting for baseline depression score (OR -0.46, Confidence interval -1.93 to 1.00). Furthermore, the intervention group had a greater reduction in the proportion of patients with depression at six months compared to the control group (-13.2% vs. -2.1%, *p* value not specified). However, this study was limited by the lack of a thorough description of the CT intervention, which limits researchers' ability to replicate the results and translate the study findings to the clinical setting.

Finally, results from a small study suggest that an intense regimen of CT combined with exercise may reduce depressive symptoms in patients with heart failure (HF).[25] In this study, 20 patients with HF (NYHA Class II/III) were randomized to three groups: group-based CT plus exercise, Digoxin titrated to achieve drug levels between

0.8 – 2.0 ng/mL, or placebo. Despite the small sample size, the intervention group experienced a 52% reduction in depressive symptoms while the other groups experienced a 15% and 25% increase in depressive symptoms, respectively. As a result of the combination of exercise with CT, it is not known whether CT alone would have been effective in decreasing depressive symptoms. In addition, the follow up period was short, only 12 weeks, and therefore no conclusions can be drawn regarding the long-term effects of the intervention.

4.2. Stroke

There is less evidence regarding the effectiveness of CT on depression in patients with stroke. Lincoln and Flannaghan[26] conducted a randomized, controlled trial in which 123 patients who had recently experienced a stroke and depressive symptoms (scored > 10 on the BDI) were assigned to one of three groups: CT, an attention placebo group, or a control group. Surprisingly, depressive symptoms improved over time in all of the groups. There were no significant differences between the groups at baseline, three months, and six months of follow up.

The authors suggested that the intervention, which consisted of ten one-hour sessions, may not have been intense enough to improve depressive symptoms more than would occur naturally. However, other investigators have tested CT in chronic illness and found positive results using a similar number of CT sessions.[14, 27] It is possible that the improvement in depressive symptoms in all three groups over time may have reflected the natural improvement of depression that occurs over time after an acute stroke.[26]

4.3. Diabetes

The effectiveness of CT for the management of depression in patients with diabetes has been tested in four randomized, controlled trials. Lustman et al.[27] conducted a RCT in which 51 patients with type 2 diabetes and clinical depression were assigned to either ten weeks of CT and diabetes education or an education-only group. As a result of the intervention, the treatment group had a higher rate of remission from depression compared to the control group (58.3% vs. 25.9%).

In contrast, investigators from three studies have found that CT was not effective for treating depressive symptoms in adults with diabetes. Snoek et al.[28] compared the effects of two interventions on depressive symptoms in adults with type 2 diabetes. The two interventions that were compared were a six week group CT intervention and a blood glucose awareness training. The results showed no differences between groups at

6 and 12 months follow up. There was no true usual care group; by comparing two active interventions, the investigators may have been less likely to detect a difference in the improvement of depressive symptoms between groups.

Similarly, Henry et al.[29] also found that CT did not improve depressive symptoms in patients with type 2 diabetes. The investigators in this small RCT used a wait list control to evaluate the effects of CT plus progressive muscle relaxation on depressive symptoms in nineteen patients with type 2 diabetes and elevated levels of glycosylated hemoglobin. Although there was an overall decrease in depressive symptoms from pre-treatment to post-treatment in both the intervention and wait-list groups, there was no difference between groups. Because of the small sample size, the study likely was under-powered to detect changes in depressive symptoms between groups.

In a much larger study of 344 adults with type 1 diabetes, Ismail et al.[30] also found that CT did not improve depressive symptoms. In this RCT, the investigators compared CT plus motivational enhancement therapy and motivational enhancement therapy alone to usual care in the treatment of depressive symptoms. Despite the lengthy intervention (up to 12 sessions over 6 months), a large sample size, and a rigorous design, neither of the intervention groups experienced a greater reduction in depressive symptoms over time than the usual care group.

5. Discussion

5.1. Discussion of findings

Fourteen randomized, controlled trials were identified in which investigators tested the impact of CT on depression or depressive symptoms in patients with cardiovascular-related illnesses. Positive effects of CT on depression or depressive symptoms were reported in eight of the fourteen studies. This discussion section will report possible reasons for the mixed results, as well as limitations that prevent wide generalization of study findings.

The major factors that contributed to the mixed results were that: 1) more than one intervention was tested in most studies, 2) there was a lack of a true, no-intervention control group in most studies, 3) most had small sample sizes, and 4) follow-up periods were short. The consequences of each of these factors are described below.

The presence of more than one treatment intervention in several studies restricted the ability to determine the effect of CT on outcomes. In two studies which found positive outcomes, CT and antidepressant therapy were combined,[14, 21] making it impossible to determine the effect of CT alone on depressive symptoms. Similarly, in two

other studies finding positive outcomes, the intervention consisted of CT plus exercise.[22, 25] Although combined interventions may be a valuable addition to the treatment options for depressive symptoms in patients with cardiovascular disease, it is difficult to assess the value of CT by itself when it is tested in combination with other interventions. For example, a growing body of evidence has demonstrated that exercise is an effective treatment for persons with major depressive disorders or with depressive symptoms.[31, 32] Thus exercise in combination with CT may have yielded a larger effect than CT alone or may obscure the effect of CT.

Six of the fourteen studies lacked a true, no-intervention group. These studies included a variety of comparison groups. For example, one group of investigators compared patients who received a CT intervention to patients who received a cardiac rehabilitation exercise intervention.[21] Although these investigators found positive results with the CT intervention, the comparison of CT to exercise may have yielded a smaller effect than would have been seen if the investigators compared CT to a third, true control group.

Seven of the fourteen studies had small sample sizes (total N ranging from 19 to 86), and eight studies had a follow-up period of six months or less. Interestingly, despite the ENRICHD trial's large sample size and long follow up period (30 months), the investigators found only modest clinically significant reductions in depressive symptoms. Moreover, this benefit was no longer present by 30 months of follow-up, as all groups improved over time.[14] In contrast, researchers of smaller studies found that CT reduced depressive symptoms at a relatively short follow-up time. It is possible that the improvements in depressive symptoms found in these studies did not persist. Short follow-up times limit the ability of researchers to determine whether CT is a potential long-term treatment for depression.

Researchers' ability to generalize the findings of several of the reviewed studies is compromised by several factors: 1) vaguely described interventions, 2) underrepresentation of women, 3) use of a wide variety of instruments to measure depressive symptoms, and 4) failure to adhere to the CONSORT guidelines for the reporting of clinical trials.

First, researchers often included vague descriptions of the intervention in articles; this limits the ability of future researchers to replicate results from these studies as well as translate the findings to clinical practice. It is also possible that the investigators tested different forms of CT, which could contribute to the mixed findings.

Second, only nine of the fourteen studies had samples that consisted of at least 30% women. Of these nine studies, investigators in four studies found that CT reduced depressive symptoms. Previous researchers demonstrated that women may react differently to psychological interventions than men. In a post-hoc analysis, the ENRICHD investigators reported that white men who received the CT intervention had a reduced risk of experiencing cardiac mortality or recurrent MI. In contrast, there was no similar beneficial effect for women and minorities.[33] Frasure-Smith et al.[34] reported that women who received an intense psychosocial nursing intervention after a myocardial infarction experienced an increase in all-cause mortality when compared to the control group (10.3% vs. 5.4%). This adverse effect was not found in the male participants. The results of these two studies suggest that it is important to evaluate the impact of psychological interventions on both men and women. Overall, the studies in this review provide insufficient evidence to determine the impact of CT on depressive symptoms in women with cardiovascular disease.

Third, researchers used a variety of methods to measure clinical depression and depressive symptoms. Many of these measures, such as the Beck Depression Inventory, have established reliability and validity. In contrast, Kohn et al.[23] used an instrument that measured three biological indicators of depression. The reliability and validity of this instrument was not provided. Overall, there was a lack of consistency on the measurement of depression in the reviewed studies. This limitation may restrict researchers from conducting future meta-analyses to examine the overall effect of CT for the treatment of depression in patients with cardiovascular disease. For this reason, in 2006 a working group of the National Heart, Lung, and Blood Institute made recommendations as to which instruments researchers should use to measure depressive symptoms in clinical trials that include patients with cardiovascular disease.[35]

Finally, of the twelve studies that were published after 1996, only five followed all of the CONSORT guidelines.[14, 26] The CONSORT (Consolidated Standards of Reporting Trials) guidelines were originally published in 1996 and have since been revised.[36, 37] These guidelines provide a standardized framework for the reporting of clinical trials. The CONSORT guidelines allow the reader to understand the design, conduct, analysis, and interpretation of a randomized controlled trial, and to judge whether a trial has internal or external validity.[37] A lack of adherence to the CONSORT guidelines in several of the studies reduces the transparency of the reported clinical trials and could contribute to a bias in over-estimating the effects of interventions.

5.2. Implications for nursing research

The problems associated with depression and depressive symptoms in patients with HF have been adequately described. Interventions such as CT for the treatment of depression in patients with HF are now needed to move us forward. As only one small trial has studied the effects of CT in the treatment of depression in patients with HF, this article reviewed the empirical evidence for CT in the treatment of depression in patients with cardiovascular-related illnesses. Overall, the current evidence to support CT as a treatment for depression or depressive symptoms in patients with cardiovascular-related illnesses is inconclusive due to limitations of existing studies.

Based on this review, future clinical trials should include the following recommendations. Researchers should test the effect of a CT intervention alone as well as in combination with other treatments. The CT intervention should be replicable in a clinical setting. Careful consideration should be paid to the inclusion of an appropriate comparison group. Given that depression in patients with HF is associated with a high risk for morbidity and mortality, it may be unethical to withhold treatment for depression from patients who are severely depressed. Furthermore, CT alone may not be appropriate for severely depressed individuals. It is suggested that researchers should exclude patients with severe depression and instead refer them to their primary care providers for treatment.

Studies should be designed with sufficient sample sizes to be adequately powered to detect changes in depressive symptoms and related outcomes. Researchers should also make special efforts to include a representative sample of women. In addition, trials should include an adequate follow-up time of at least one year in order to provide information on the long-term effects of CT on both depression and other health outcomes, such as morbidity and mortality. It is also important that researchers use consistent methods for measuring depressive symptoms, such as issued in the National Heart, Lung, and Blood Institute's recommendations.[35] Finally, to improve transparency in the reporting of randomized, controlled trials, researchers should follow the CONSORT guidelines.[37].

Several gaps in understanding of CT as a treatment for depression in cardiovascular conditions, including HF, remain. It is not known if there is a dose-response relationship between CT and depression in patients with cardiovascular disease. For example, we do not know how many CT sessions are necessary to improve depression outcomes, or whether the effective dose varies among different

cardiovascular populations. The best time to intervene for depressive symptoms in patients with cardiovascular disease is also unknown. The results of the ENRICHD trial showed that patients who had recently experienced a cardiac event and received a CT intervention only experienced a minimal improvement in depressive symptoms. This led researchers to question whether CT should be offered to patients who have recently experienced a cardiac event or if treatment should be delayed.[15] Likewise, it is not known whether CT should be offered to patients with HF who have been recently hospitalized or whether their depression may remit on its own. Next, it is not known whether CT interventions should be offered only to patients with cardiovascular disease or HF who are clinically depressed or to all patients, regardless of depression status. Finally, it is unknown whether CT can affect outcomes related to depression such as mortality, morbidity, or health-related quality of life in patients with cardiovascular disease or HF.

5.3. Implications for nursing practice

Cognitive therapy is compatible with nursing practice and has been used successfully to treat depression in medically-healthy populations. Because there is a scarcity of evidence for patients with HF, this review was broadened to include all patients with cardiovascular-related illnesses. Based on the findings of this review, the current evidence is insufficient to recommend CT as a treatment for depressive symptoms in patients with cardiovascular illness. Although the majority of the studies reviewed demonstrated that CT may be effective, the limitations in study design prevent wide generalization of the results. More evidence is needed before it can be recommended that nurses routinely refer patients with cardiovascular disease to CT for the treatment of depression or depressive symptoms. It is important to also note that CT may not be appropriate for all patients with HF or cardiovascular disease, particularly for patients with cognitive impairment or those who may have difficulty adhering to the CT treatment protocol. These patients may benefit from alternative treatments for depression.

6. Conclusion

Depression is a significant clinical problem in patients with HF. The time has come for researchers to focus their efforts on designing and testing non-pharmacological interventions for depression in patients with HF. Cognitive therapy holds promise as an intervention that may decrease depressive symptoms in patients with cardiovascular illness including HF. Nurses should continue to monitor the literature for new evidence

regarding the effectiveness of CT for treating depression in patients with HF and other cardiovascular disease.

Table 2.1: DSM-IV-TR criteria for a major depressive episode

At least 5 of the following symptoms have been present most of the day, nearly every day, during the same 2 week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure:
1. Depressed mood 2. Loss of interest or pleasure 3. Weight loss or changes in appetite 4. Insomnia or hypersomnia 5. Psychomotor agitation or retardation 6. Fatigue 7. Feelings of worthlessness or guilt 8. Decreased ability to concentrate 9. Thoughts of death, suicidal ideation, or suicide attempt.
• The symptoms must cause significant impairment in functioning (i.e., work, social) • The symptoms must not be directly due to a medical condition (i.e., hypothyroidism) or a medication • The episode is not better accounted for by a different diagnosis such as bereavement, bipolar disorder, or schizoaffective disorder

DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition

Table 2.2: Study characteristics and findings

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Cardiovascular Disease						
ENRICHD[14] (2003)	RCT 2 arms 30 months	N = 2481 28 days post-MI Eligible if classified as depressed or low perceived social support Females 44% Minorities 34%	DISH, BDI, HRSD	11 tailored CT sessions over 6 months, group therapy as needed, referral to psychiatry for antidepressants as needed	Usual care; physicians were notified if patients were depressed or had low perceived social support	The CT group had a lower BDI score compared to control (9.1 vs. 12.2, p <.001), and a lower HRSD score (7.6 vs. 9.4, p <.001) at 6 months. This difference was not present at 30 or 42 months

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Freedland (2009)[17]	RCT 3 arms 9 months	N = 123 CABG surgery within the past year Eligible if scored ≥ 10 on the BDI and met DSM-IV criteria for major or minor depression based on the DISH Females 50% Minorities 19%	HRSD score derived from the DISH; BDI	1. CT: 12 weekly, individual, 50-60 minute sessions with a therapist 2. Supportive stress management: 12 weekly, individual, 50-60 minute session. Training included progressive relaxation, imagery, and controlled breathing	Usual care	Patients in the CT and stress management group were more likely to experience remission from depression at 3, 6, and 9 months compared to patients in the usual care group
Burgess[18] (1987)	RCT 2 arms 13 months	N = 180 Post acute MI Females 14% Minorities not reported	ZDS	A mean of 6.32 CT visits per patient, social support, facilitation of job return	Usual care	There were no differences between groups on depression scores at baseline or follow-up

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Cowan[19, 20] (2001)	RCT 2 arms 3 months	N = 133 Sudden cardiac death survivors Females 27% Minorities 10%	SCR-90: Depression subscale	11 sessions of combined CT, biofeedback, and health education, administered biweekly for six weeks	90 minute health education class	Depressive symptoms decreased in the treatment group when compared to the control group
Black[21] (1998)	RCT 2 arms 6 months	N = 60 Recently hospitalized for angina, MI, angioplasty, or CABG Eligible if scored as distressed Females 12% Minorities not reported	SCR-90: Depression subscale	1 to 7 weekly sessions with a psychologist including: relaxation training, stress management, reduction of risk factors, efforts to improve adherence, and CT intervention; antidepressants if necessary	Cardiac rehabilitation with monitored exercise 1-3 times per week, for 8 weeks, daily home exercise. Education on stress management, support group meeting with spouses, individual nutrition counseling	The CT group had significant reductions in depressive symptoms compared to the control group (-5.2 vs. -0.2, $p < .034$)

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Frizelle[22] (2004)	RCT 2 arms 3 months	N = 22 Patients with ICD's Females not reported Minorities not reported	HADS	Group-based therapy, six sessions, 1 hour each: home-based exercise, education, relaxation, behavioral goal setting, education on identifying and challenging negative thoughts	Wait list	The treatment group experienced decreases in depressive symptoms compared to the control group (-4.25 vs. -0.2, p = .001)
Kohn[23] (2000)	RCT 2 arms 9 months	N = 49 Post-ICD implantation Females 35% African American 8%	BDI version II; 4 biological measures of depression: sexual functioning, appetite, weight change, and sleep patterns	9 sessions ranging from 15-90 minutes, sessions included psycho-education on: anxieties about ICD, avoidance behavior, fear of shocks, stress management, work and social activities, distorted cognitions	Usual care	The CT group had lower levels of depressive symptoms at follow-up compared to the control group (6.9 vs. 15, p = .037), but depressive symptoms were not measured at baseline

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Lewin[24] (2007)	Clustered RCT 2 arms 6 months	N = 192 Patients undergoing ICD implantation 8 implantation centers in the UK were randomized to intervention or control Females 20% Minorities 3%	HADS-Depression subscale	Intervention consisted of 2 booklets for patients, 1 booklet for family, a goal-setting diary and a relaxation tape. The first booklet targeted fears prior to ICD implantation. The second booklet consisted of a self-help CT program. The intervention was delivered by healthcare staff that underwent a half day of training.	Usual care and contact by a study facilitator to discuss postoperative progress	The intervention group experienced a greater reduction in the proportion of patients with depression at six months compared to the control group (-13.2% vs. -2.1%, *p* value not reported)

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Kostis[25] (1994)	RCT 3 arms 3 months	N = 20 Patients with congestive heart failure Female 30% Minorities not reported	BDI	12 weeks of exercise training at a cardiac rehab facility for 1 hr 3 times per week; weekly meetings with a dietician; group-based CT intervention: twice weekly for 60-90 minutes (relaxation, positive imagery, appraisal of negative cognitions)	1. Lanoxin titrated to achieve levels between 0.8-2.0 ng/mL 2. Placebo	There was a 52% decrease in BDI scores in the intervention group compared to a 15% and 25% increase in the control groups at follow up (p = .04)

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Stroke						
Lincoln[26] (2003)	RCT 3 arms 6 months	N = 123 1 to 6 months post-stroke Eligible if scored as depressed Female 49% Minorities not reported	BDI WDI	Ten 1-hour sessions over 3 months, tailored CT intervention: education, task assignment, activity scheduling, identification and modification of inaccurate thoughts	1. No intervention 2. Attention placebo: Ten 1 hr visits over 3 months	No significant differences between groups in depression scores
Diabetes						
Lustman[27] (1998)	RCT 2 arms 6 months	N = 51 Type II diabetes and major depression Females 60% Minorities 19%	DIS BDI	1 hour per week of individual CT for 10 weeks, strategies included: behavioral strategies, problem solving, and cognitive techniques to change cognitive errors	Attention placebo: 1 hour, biweekly, individual sessions with a diabetes educator	The CT group had a higher rate of remission from depression compared to the control group (58.3% vs. 25.9%, p = .03)

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
Snoek[28] (2008)	RCT 2 arms 12 months	N = 86 Adults with poorly controlled type I diabetes Females 58% Minorities: not reported	CES-D	6 weekly group sessions of CT delivered by a diabetes nurse educator and a psychologist. Sessions focused on cognitive restructuring, behavior change and stress management	Blood glucose awareness training focused on symptom management and diabetes education, delivered by a diabetes nurse educator and a psychologist	There were no differences between groups with regard to change in depressive symptoms over time
Henry[29] (1997)	RCT 2 arms 7 weeks	N = 19 Type II Diabetes Females 53% Minorities not reported	BDI	Six sessions of 1.5 hours: progressive muscle relaxation, cognitive coping training (such as identifying and modifying negative thoughts), problem-solving skills, homework assignments	Wait-list	Depressive symptoms decreased across time in both groups; there was no difference between groups
Ismail[30] (2008)	RCT 3 arms 12 months	N = 344 Adults with poorly controlled type I diabetes	PHQ-9	1. CT plus motivational enhancement: 4 individual sessions of motivational enhancement	Usual care	Neither group experienced an improvement in depressive symptoms compared to usual care

Table 2.2: (Continued)

First author (year)	Design, follow-up time	Sample	Measurement of depression	Treatment	Control	Results
		Females 60% Minorities 20%		therapy (see below) and 8 sessions of CT delivered over 6 months by trained diabetes nurses 2. Motivational enhancement: 4 individual sessions delivered over 2 months by a diabetes nurse. Sessions focused on assessment of readiness to change, diabetes behavior modification, and problem solving		

BDI: Beck Depression Inventory version I; CABG: Coronary bypass grasft surgery; CT: Cognitive Therapy; DIS: Diagnostic Interview Schedule; DISH: Depression Interview and Structured Hamilton; HADS: Hospital Anxiety & Depression Scale; HRSD: Hamilton Rating Scale for Depression; PHQ-9: Patient Health Questionnaire; SCR-90: Symptom Checklist 90 Revised; WDI: Wakefield Depression Inventory; ZDS: Zung Depression Scale

CHAPTER THREE:

Living with Depressive Symptoms: Patients with Heart Failure

1. Introduction

Heart failure (HF) is a chronic syndrome that affects 5.8 million people in the United States[1] and is the most common cause of hospitalization in elderly adults.[2] Major depressive disorder is common in hospitalized patients with HF. One-third of hospitalized patients with HF suffer from major depression, and 40% of these are still depressed one year later.[3] According to the American Psychiatric Association, a major depressive episode consists of five or more symptoms which are present most of the day for at least two weeks. One of these symptoms must be depressed mood or loss of interest in usual activities, and the symptoms must cause significant distress in social, occupational, or other areas of functioning.[4]

Individuals can experience significant depressive symptoms without a diagnosis of major depressive disorder.[5] Sometimes called subthreshold depression, depressive symptoms include depressed mood, guilt, hopelessness, low self-esteem, fatigue, sleep disturbances, appetite change, and inability to concentrate.[6] Researchers have proposed that depressive symptoms lie within a continuum of depressive illness severity, which ranges from mild depressive symptoms to a diagnosis of major depressive disorder.[7] It is estimated that up to 48% of outpatients with HF experience clinically significant levels of depressive symptoms.[8] The presence of depressive symptoms has important clinical consequences for patients with HF. Patients with HF who have depressive symptoms experience reduced health-related quality of life and an increased risk of rehospitalization and mortality.[8,9]

Although researchers have examined the predictors and outcomes of depressive symptoms in patients with HF, none have described the experience of living with depressive symptoms. Qualitative research can help researchers and clinicians understand a phenomenon from the patient's point of view and guide the development of future interventions. The purpose of this qualitative study was to describe the experience of living with depressive symptoms in persons with HF. The primary research question was: How do persons with HF describe their depressive symptoms? Secondary research questions included: What factors influence depressive symptoms? What are strategies that persons with HF use to manage depressive symptoms?

2. Methods

2.1. Qualitative description

This investigation was conducted using a qualitative descriptive design. Qualitative description involves low-inference interpretation and is often the first step in exploration of a phenomenon. The result is a descriptive summary of the experience in everyday language.[10] This topic is particularly suited for qualitative description because little is known about how patients with heart failure describe their depressive symptoms.

2.2. Participants

The study was nested in a clinical trial of biofeedback and cognitive therapy in patients with HF. Institutional Review Board approval was obtained prior to recruitment. Patients were eligible to participate if they were diagnosed with chronic HF, stable on cardiac medications, had no coexisting terminal illness, and had not had a stroke or myocardial infarction within three months.

Purposive sampling was used to select ten outpatients who had a self-reported history of past or current depression, or were experiencing a clinically significant level of depressive symptoms, defined as a score of 14 or greater on the Beck Depression Inventory-II[11] upon enrollment in the trial (Table 3.1). Purposive sampling ensured equal numbers of men and women and equal representation from the intervention and control groups. The intervention group received six weeks of biofeedback training and cognitive therapy from a psychiatric nurse practitioner, while the control group received six weeks of attention placebo, which consisted of self-selected relaxation exercises delivered by a psychiatric nurse practitioner.

2.3. Data collection

One-time, semi-structured interviews were conducted in the participants' homes after the intervention or placebo portion of the trial was completed. Results from two initial interviews found that patients had difficulty answering direct questions about symptoms of depression. Therefore, interviews began with questions regarding recent stressful events to elucidate depressive symptoms. Questions explored the participant's symptoms that occurred during the stressful event. Each participant was asked to share techniques that they used to prevent or cope with depressive symptoms. Interviews lasted between 30 and 60 minutes and were audio taped. Participants were reimbursed $30 for their time.

2.4. Data analysis

An inductive approach to content analysis was used in this study. Interviews were transcribed verbatim and transcriptions compared to the taped interviews to assure accuracy. A codebook was developed to assist in the analysis. The codebook contained *a priori* codes based on a literature review, as well as codes that emerged from the data. We used Atlas ti (version 5.0) to code the data. Each interview was read, re-read, and coded by the primary author. The transcripts were compared to ensure that coding was consistent throughout, and codes were first sorted into categories and then into larger themes. Data displays that depicted the frequencies of the codes were developed to assist in the reduction of the coded data into categories.[12] The abbreviated data display for factors influencing depressive symptoms is provided in Table 3.2 as an example of the data displays used for data reduction.

2.5. Trustworthiness and credibility

To establish trustworthiness and credibility, we used three procedures recommended by Creswell.[13] First, we clarified bias prior to data collection by bracketing assumptions. Bracketing assumptions consists of writing down personal assumptions about the phenomenon; this process allows the researcher to collect data with as unbiased a viewpoint as possible. Second, the coauthors met frequently throughout data analysis to verify the coding accurately reflected the content of the interviews. Third, the research results were returned to three participants to verify accuracy of the findings.

3. Results

3.1. Depressive symptoms

The participants' descriptions of depressive symptoms were similar to symptoms of depression as defined by the Diagnostic and Statistical Manual-IV-TR criteria.[4] The participants described emotional symptoms of sadness, irritability, tearfulness, and anxiety. The participants also experienced somatic symptoms that they attributed to depression, including lack of energy, changes in appetite, sleep disturbances, and difficulty concentrating. Finally, the participants described experiencing negative thinking and cognitive distortions.

3.1.1. Emotional symptoms

Eight of the ten participants described sadness as part of their experience with depression. The participants used the terms "terrible sadness," "depressed," "moody," "bad blue mood," and "upset" to describe their mood. Six of the participants stated that tearfulness was a frequent symptom. One participant cried during the interview, stating:

"It's just I'm sad. And I think it's just because there's a lot of sad stuff. It's just been really hard."

Half of the participants complained of feeling irritable. They expressed feelings of aggravation, frustration, and anger. In many cases irritability resulted in strained relationships with children or spouses. As one participant said, "Sometimes I could just scream and one day I did, several times I have, about them not cleaning."

In addition to sadness and irritability, feelings of anxiety were described by nine participants. The participants used "tense," "tight," "shaky," "nervousness," "worry," and "fear" to describe their anxiety. One participant shared:

"At night I start thinking about it, the consequences of what could happen and what couldn't and what would come out good, and I just, just get in a knot again."

3.1.2. Somatic symptoms

Seven participants described somatic symptoms attributed to depression that included changes in appetite, sleep disturbances, and lack of energy. Only one patient described an increase in appetite while three others described a loss of appetite. Three of the participants described sleep disturbances. One participant in particular spent much of her time sleeping. At her request, the interview took place in her bedroom, while she sat in bed. She said, "Sometimes I just come in the room, shut the door and go to, and just go to sleep, just lay here." Only three participants complained of a lack of energy.

3.1.3. Negative thinking

All ten participants described negative thinking, or self-critical thoughts.[14] Participants described thoughts such as, "I can't justify my existence," "I have nothing to offer anymore," and "I'm a failure." The participants stated that the negative thoughts reinforced their depressed mood. As one participant shared:

"Sometimes it gets me down because I start thinking about other things... You know, it just kind of moves up to keep going up the ladder."

Participants described cognitive distortions that accompanied negative thinking. Magnification is a cognitive distortion in which a person inflates the magnitude of the problem.[15] For example, when one participant underwent surgery, she could only think about the possibility of death:

"Am I going to come out of this, am I going to die, you know is this my last time, or I'm going to die in there on the operating table."

Another frequently mentioned distortion was dwelling. Dwelling is defined as long, uninterrupted series of depressive thoughts that worsen depressive symptoms.[15, 16] When asked what advice she would give to someone in her situation, one participant answered:

"I think I would tell them you know from my experience, the more you dwell on it, the worse off you get."

3.2. Stressors

Participants described stressors that worsened their depressive symptoms. The stressful experiences that the participants lived with included financial difficulties, family problems, health issues, and loss.

3.2.1. Financial difficulties

Seven participants described financial difficulties, including bankruptcy, living on a fixed income, and being disabled. One participant said that finances were the primary contributor to her depressed mood:

"I think the main thing well that, that upsets me is finances. If my finances get to the point where I get to a week before payday and I don't have any money, then I get very stressed and I think that stresses everybody. But I think it especially stresses me."

3.2.2. Family problems

Nine participants talked about stressful situations in their families. Problems that the participants dealt with ranged from children who were incarcerated, to grandchildren who were having problems, to alcoholism and arguments among family members. The participants described feeling worried about family situations and saddened or irritated by conflict. One participant felt depressed because he was estranged from his only child: "Well I felt sad there for a long time because my daughter, I wanted to be in contact with her and I felt hurt... I want to be somebody my daughter can depend on me being there when she needs me."

3.2.3. Health issues

Interestingly**,** only five of the participants mentioned HF as influencing depressive symptoms. Three participants stated that depressive symptoms were present prior to the development of HF. One participant said that having HF did not make her depressed. Instead, "it's just something else for me to be depressed about." The majority of participants described other health conditions as being stressful and contributing to depressive symptoms. Participants described dealing with cancer, diabetes, arthritis, and chronic pain.

3.2.4. Loss

The participants described numerous losses that worsened their depressive symptoms. Four of the participants described the death of a son or daughter. One participant had experienced the death of two children. Although some of these deaths had occurred years ago, it was clear that the participants still felt the effects of the loss. Complicating the bereavement process for these participants was the fact that all of the deaths were sudden. One participant said:

"Well of course you know it's been in 2003 when my daughter died suddenly and I still haven't gotten over that and of course at night sometimes I cry over that."

Other participants did not discuss the loss of a loved one, but they experienced other losses. One participant described the loss associated with not being able to work: "I used to get really depressed. I can't work anymore; what am I going to? I mean, it really bothered me at first."

3.3. Strategies for managing depressive symptoms

The overarching strategy described by the participants was "taking my mind off of it." Four types of strategies were used by participants to take their minds off of depressive symptoms: activities, distraction, social support, and medical intervention.

3.3.1. Activities

Exercise was mentioned by nine participants as being an important method of reducing depressive symptoms. The most commonly mentioned exercise was walking. The participants repeatedly said that walking made them feel better, and that any amount of exercise was better than just "sitting around." One participant stated:

"I've started walking. It's fun. I put my, I take my radio and listen to my music and it makes me feel good because I kind of walk and do a little dance while I'm walking and so, so it has made me feel good when I was walking."

In response to the question, "*What are some of the things that help you feel better when you feel down,*" nine participants mentioned reading. The participants who had difficulty reading would listen to books on tape instead. As one participant said, "Rather than sit here and think about the bad things you know, I can get me a book to listen to." For most participants, reading allowed them to "take their mind off things" and focus on something other than the depressive symptoms.

Being active, or "staying busy," was mentioned by six participants as a behavioral tactic that kept them from thinking about depressive symptoms. Participants used a variety of activities to stay busy, such as bird watching, crocheting, and watching

television. The simple act of getting involved in an activity helped relieve the participant's depressive symptoms.

"Sometimes I like to do crafts. I like to work crosswords and do Sudoku. Other times that just gets my mind off things and just concentrating on that one thing."

3.3.2. Distraction

For these participants, distraction is defined as the ability to redirect one's internal focus away from negative thoughts or emotions. For seven participants, spirituality or religiosity was an important method of distraction. Participants received relief by praying, attending church, and reading scripture. One participant quoted Biblical scriptures several times. He said, "When we fill our mind and our heart with the scriptures, that's something that we can depend on."

A central component of spirituality was a relationship with God. One participant described God as "my other best friend." Another participant said:

"I feel I have a great relationship to God because I talk to him about a lot of things and when I do, I feel better. I feel that a burden has been lifted from me; not all the time right away but it's been, you know it's been lifted."

When asked: "If you were talking to someone like you who felt depressed or down, what advice would you give them?" nine of the ten participants discussed positive thinking. Positive thinking is a central component of the cognitive therapy intervention which five of the participants had received. However, four of the five control group participants also discussed positive thinking. As one control group participant said:

"The main thing I think that I've found is trying to keep my mind off of things that make me sad. If I find myself drifting that way, well then I know right away I've got to think of two positives. Because that's a negative. And like I said, when you've got a negative, you've got to come up with two positive things to replace that."

Instead of dwelling on negative thoughts, the participants redirected their thoughts towards the positive. One participant focused on the future:

"Think about the future. Think about the future; don't dwell on the past, there's nothing you can do. It's happened; it is depressing. You know that your life has changed; it's changed your life completely. But think about the positive you know, life goes on."

Others focused their thoughts on affirmations. One participant read his affirmations out loud:

"My heart may be sick, but my mind and soul are strong and healthy. My problems are an opportunity for me to grow as a person. I'm a person of value even if I have heart failure, back trouble, knees and shoulder, and I'm old."

Additional examples of affirmations included: "I'm alive, I'm thankful for that, I'm alive," "Things are going to get better, going to get better," and "I might not be able to do the things I used to do but there's new things I can do."

3.3.3. Social support

All ten participants stated that social support was an important factor in reducing depressive symptoms. Family and friends provided emotional, physical, and financial support. The married participants all provided positive descriptions of their marriages. As one participant said:

"I do appreciate that lady [indicating wife] over there. The Lord drawed us together and we were married after getting out of the service…Yes we celebrated our 50th about 3 years ago. And pretty soon we'll come up with number 54."

3.3.4. Medical intervention

Seven of the participants (five females and two males) were taking antidepressants at the time of the interview, yet only four participants—all women—mentioned antidepressants as a strategy for managing depressive symptoms. One participant described how antidepressants helped her enjoy sunlight again:

"I used to hate days like today where it was sunny. Like I couldn't stand to be in the sun and ever since I've been on antidepressants, I enjoy sunshine."

4. Discussion

4.1. Discussion of findings

The participants in this study described emotional and somatic symptoms of depression, as well as negative thoughts that worsened their mood. Stressors such as financial difficulties, family problems, and health conditions exacerbated the depressive symptoms. Individuals described "taking their mind off" depressive symptoms through activities, distraction, social support, and medical intervention.

The depressive symptoms experienced by the participants were similar to those experienced by the general population with depressive symptoms. The majority of the sample experienced a depressed mood. This is particularly interesting since seven participants were currently receiving antidepressant therapy. The finding that our participants experienced significant emotional symptoms contrasts with results from a prior study in which a sample of outpatients with HF and major depression living in

Germany had fewer emotional symptoms of depression, as measured by the Patient Health Questionnaire-9 (PHQ), when compared to patients with major depression who did not have HF.[17] It is possible that the PHQ did not effectively capture the emotional symptoms that are experienced by patients with chronic HF. The participants in our study may have also been more likely to divulge emotional symptoms in their home environment, as opposed to a clinic or research setting.

Few somatic symptoms of depression were mentioned by participants. It is possible that the participants attributed somatic symptoms to HF rather than depression. Several common symptoms of HF, such as fatigue, anorexia, and sleep disturbances, overlap with symptoms of depression. This symptom overlap may contribute to the participants' difficulty in recognizing somatic symptoms of depression.[17] In addition, clinicians may also attribute somatic symptoms to HF instead of depression. This lack of recognition of the somatic symptoms of depression could contribute to the low treatment rates of depression in persons with HF,[3] as well as under-treatment which results in individuals not receiving therapeutic doses of antidepressants.

An important finding of this study was that negative thinking was common and had a detrimental impact on the participants' mood. The presence of negative thoughts has been described as a characteristic of major depression.[15] However, this was the first to describe negative thinking as important in patients with HF who have depressive symptoms. The participants experienced cognitive distortions that maintained their negative thought processes. Researchers have found that magnification is an independent predictor of depressive symptoms in the elderly.[16] In addition, depressed patients who "dwell" on negative thoughts experience a worsened mood and are less able to problem solve.[18]

The participants described a variety of stressors that worsened their depressive symptoms. This finding is consistent with results from other qualitative studies on living with HF. Investigators have found that patients with HF experience physical, emotional, and social turmoil in their everyday lives[19], as well as multiple personal struggles and losses.[20,21] In combination, these studies demonstrate that having a diagnosis of HF is only one stressor in a life full of difficulties.

Many clinicians assume that patients with HF are depressed because they have been diagnosed with HF. However, only half of the participants mentioned HF as influencing depressive symptoms. Future qualitative research should explore whether patients with HF attribute depressive symptoms to HF. For three participants, depression

was something that they struggled with for years prior to the onset of HF. Pre-existing depression has been demonstrated to be an independent risk factor for development of HF.[22,23] Thus, the long history of depression may have increased the risk of developing HF in these participants.

Of particular interest are the multiple strategies described to counteract depressive symptoms. The overarching strategy was "taking my mind off of it." Participants used activities, distraction, social support, and medical intervention to alleviate their symptoms. Activity in the form of exercise appears to be as effective as antidepressants for the treatment of depression in the elderly.[24] Results from several small trials have shown that exercise improves depressive symptoms in patients with HF.[25,26]

The participants' use of social support to alleviate depressive symptoms is supported by the literature. Results from one study demonstrated that an increase in perceived social support was the only factor that predicted faster remission from depression in hospitalized patients with HF.[3] In contrast, poor social support, defined as living alone, independently predicts the development of depressive symptoms in HF patients[27], while single marital status also predicts depressive symptoms in patients with HF.[28]

For most of the participants, spirituality was an important method of managing depressive symptoms. Researchers have demonstrated an inverse relationship between spiritual well-being and depressive symptoms in patients with chronic HF.[29] In addition, researchers have demonstrated that older patients with HF who are involved in religious activities remit faster from depression than non-religious patients.[30]

The participants used positive thinking more than any other strategy to manage their depressive symptoms. They also expressed that affirmations were an important method of reducing negative thoughts. The evidence to support positive thinking and affirmations as effective strategies is found in cognitive therapy, which is based on the Cognitive Model of depression. The Cognitive Model proposes that redirecting negative cognitive processes into positive ones can improve the symptoms of depression.[31] The use of cognitive strategies was not surprising for the five participants who received cognitive therapy in the parent study. However, four of the five control group participants also discussed positive thinking as a strategy for managing depressive symptoms.

Although the majority of the participants were taking antidepressants, few mentioned medications as a method to manage depressive symptoms. It is possible that

participants did not view their depressive symptoms as a medical condition. Thus they may not have considered antidepressants as a strategy for managing depressive symptoms. Selective serotonin reuptake inhibitors (SSRIs) are often prescribed for depression in patients with HF because of the lack of cardiac side effects.[32] Currently, there is little evidence regarding the efficacy of SSRIs for treating depression in patients who have HF. However, a large randomized, controlled trial is currently underway.[33]

4.2. Limitations

Because of the heterogeneity of depressive symptoms experienced by participants it may be necessary to obtain more viewpoints to fully understand the experience of living with depressive symptoms in persons with HF. Similarly, only one interview was conducted with each participant. Additional interviews may have revealed other depressive symptoms and coping strategies or provided additional insight into the connection between HF and depressive symptoms. This study included participants who experienced a cognitive therapy intervention which may have influenced participant experiences. This strategy can also be considered a strength because it included patients with a broad range of perspectives.

5. Conclusion

The results of this study have important implications for clinicians and researchers who work with patients with HF. Given the negative outcomes associated with depressive symptoms, clinicians must assess hospitalized patients with HF for the presence of depressive symptoms as well as stressors that might exacerbate depressive symptoms. It also is important for clinicians and researchers to note that depression in patients with HF may benefit from the same interventions that are effective for treating depression in the general population. Participants in this study managed depressive symptoms through the use of activities, distraction, and social support. These strategies are worthy of development and testing among patients with HF in the acute care arena.

This study is the first to identify that negative thinking may be an important component of depressive symptoms in patients with HF. By targeting negative thinking, clinicians may be able to reduce depressive symptoms in patient with HF. Clinicians can teach hospitalized patients with HF to recognize negative thoughts and replace them with positive thoughts and affirmations. For example, an acute care nurse could ask a hospitalized patient with HF, “What kinds of thoughts are you experiencing right now?” After assessing for the presence of negative thoughts, the nurse can teach the patient how to replace negative thoughts with positive ones. Clinicians also may help alleviate

hospitalized patients' depressive symptoms by assessing their spiritual well-being and referring patients with needs to appropriate spiritual care. Encouraging the patient to engage in activities such as ambulating or reading while they are hospitalized may also reduce depressive symptoms. Finally, helping patients' significant others provide enhanced social support may be an important method for reducing depressive symptoms in patients with HF.

Clinicians can no longer ignore the fact that patients with HF experience levels of depressive symptoms that reduce quality of life and increase the risk for mortality. This study is the first description of the experience of living with depressive symptoms in patients with HF. Clinicians can use the results from this study to gain understanding of the patients' perspective of living with depressive symptoms. Researchers can use the results to develop and test interventions to reduce depressive symptoms.

Table 3.1: Participant characteristics

Characteristic	Percentage or Mean ± standard deviation
Female	50%
Age	62.5 ± 13.2 Range: 37-81 years
Minority	20%
Married	70%
NYHA Class I Class II Class III Class IV	10% 20% 60% 10%
Some college education or greater	70%
Employed	20%
Beck Depression Inventory –II score	18.9 ± 11.8 Range: 7 - 40
Antidepressant use	70%

NYHA = New York Heart Association functional classification

Table 3.2: Data display for factors influencing depressive symptoms

Code	Frequency	Example Quote
Family problems	9/10	"Kids and grandkids…something is always going on in their lives. And at least one of them always has a problem of some kind to deal with."
Loss	9/10	"Even though it's been 11 years since my daughter died, I have days where I think about her a lot. I mean it never gets easy."
Finances	7/10	"We just went through a bankruptcy. And that was not easy. I don't like things like that but there was nothing we could do."
Health problems	8/10	"I've been having a lot of problems with the back, a lot of pain and so on. I've had all kinds of treatments and nothing works."
Heart failure	5/10	"The only thing that stresses me really is I want to be able to breathe better so that I can do more around the house."
History of depression	3/10	"See, I'm a person who's fought depression all of my life, I mean even when I was a child."

CHAPTER FOUR:
Measurement of Negative Thinking in Patients with Heart Failure:
A Critical Review and Analysis
Synopsis

1. Introduction

The purpose of this paper is to present a critical review of the measurement of negative thinking to provide direction for the measurement of negative thinking in patients with heart failure (HF). Negative thinking can be defined as depressive cognitions about the self, world, future, and withdrawal from others.[1] Negative thinking is a cognitive process that is automatic and persistent;[2] it originates in childhood and dominates the perceptions of the depressed individual.[3] Examples of negative thoughts include, "I'm worthless," "Everything I do is a failure," and "Nothing's ever going to work out for me."[1] Although the content of negative thoughts is irrational, these thoughts are believable to the person experiencing them.[2]

Negative thinking has several adverse consequences in the general population. First, negative thinking is a risk factor for depressive symptoms,[4, 5] as well as an independent predictor of the later development of depression among women.[6, 7] Second, negative thinking exacerbates and maintains depressive symptoms, which contributes to a downward spiral of depressive symptoms that may end in major depressive disorder.[2, 8] Third, negative thinking independently predicts a worsened response to treatment for depression, even after controlling for history of depression and depressive symptoms at baseline.[9]

Depression is a significant clinical problem in patients with HF. Twenty percent of outpatients with HF suffer from major depressive disorder,[10] and up to 48% of outpatients experience clinically significant depressive symptoms.[11] Patients with HF who are depressed have a two-fold greater risk of death or hospitalization compared to patients who are not depressed.[12]

In a recent qualitative study, patients with HF who had depressive symptoms described experiencing negative thinking which exacerbated their depressed mood.[13] This finding is consistent with Beck's Cognitive Model of depression, which proposes that negative thinking influences the emotional, behavioral, and somatic symptoms of depression.[14] The patients in the qualitative study who experienced depressive symptoms stated that positive thinking was a strategy they used to manage depressive symptoms.[13] Cognitive Therapy, a treatment for depression which re-directs negative

thoughts into positive ones, may be an effective treatment for depression in patients with HF.[15] In order to test the effectiveness of Cognitive Therapy as a treatment for depressive symptoms in patients with HF, researchers need a valid and reliable measure of negative thinking in this population.

Therefore, the objectives of this paper were to provide an overview of the state of measurement of negative thinking, analyze three existing measures of negative thinking, and make recommendations for new directions in the measurement of negative thinking in patients with HF.

2. Overview of the state of measurement of negative thinking

Negative thinking was first defined in 1967 by Beck, a psychiatrist, as the *cognitive triad*: negative thoughts about the self, world, and future.[2] Since then, researchers have suggested a fourth type: negative thoughts about interpersonal relationships or withdrawal from others.[1]

Endorsement questionnaires are the most common method of measuring negative thinking.[16] These questionnaires contain a list of self-statements or thoughts. Examples of thoughts on an endorsement questionnaire include, "I wish I were a better person," "My life is a mess," and "It's just not worth it."[17] Patients are instructed to rate how often they experience each thought on a Likert scale. Patients usually fill out endorsement questionnaires retrospectively, although a few instruments call for the questionnaire to be administered at the same time as a stressful stimulus.

Endorsement questionnaires are cost-effective, easy to administer, and quick to complete.[16] There are only a few disadvantages to using endorsement questionnaires. A retrospective questionnaire could lead to selective memory bias and socially desirable responses. It is also possible that endorsement questionnaires do not measure the frequency of an individual's actual thought content, but rather the importance of the thought, the similarity of the thought to other ones they may have experienced, or the emotional salience of the thought.[16, 18] However, researchers have provided evidence supporting the construct validity and concurrent validity of endorsement questionnaires for measuring negative thinking.[18] As a result, endorsement questionnaires remain the most widely used method of measuring negative thinking in clinical trials. At least 28 endorsement instruments have been developed to measure negative self-statements. Only seven of these instruments are used to measure negative thinking specific to depression. The other instruments measure negative thinking found in conditions such as anxiety, pain, eating disorders, and panic disorder.[16, 18]

For this paper, three of the most widely used questionnaires that measure negative thinking in depression were selected for review. These instruments include the Crandell Cognitions Inventory, the Automatic Thoughts Questionnaire, and the Cognition Checklist- Depression subscale. The instruments are compared in Table 4.1, and studies which have examined their psychometric properties are described in Table 4.2.

3. Description of three existing measures of negative thinking

3.1. Crandell Cognitions Inventory

The Crandell Cognitions Inventory (CCI) was developed by Crandell and LaPointe in 1979 to measure self-statements of depression. The authors initially defined depressive thoughts as the *cognitive triad*—negative thoughts about the self, world, and future. A preliminary version of the CCI was developed with a sample of psychiatric inpatients, psychiatric outpatients, and persons without a psychiatric illness, and it contained 81 negative items and 19 non-scored, positive buffer items. Individuals were asked to rate how frequently they experienced each thought on a scale from 1 (almost never) to 5 (almost always); the instructions do not give a specific time frame for thought recall.

The preliminary version of the CCI demonstrated strong evidence of reliability (Cronbach's alpha = 0.98) but possible redundancy among the items. The preliminary version also performed well in discriminating between the three groups; however, the authors felt that the scale was too lengthy to be clinically useful.[1]

In 1986, Crandell and Chambless[1] revised the CCI with a sample of depressed psychiatric patients, non-depressed psychiatric patients, and normal controls. Patients were grouped on the basis of the following: Hamilton Rating Scale scores for depression, Maudsley Personality Inventory scores, whether or not the patient was receiving psychological treatment for depression, and whether or not patients had a clinical diagnosis of depression based on the Research Diagnostic Criteria.

The final version of the CCI consisted of 45 items—34 negative self-statements and 11 non-scored positive buffer statements. The authors intended the CCI to have three dimensions: negative views of the self, world, and future, but factor loadings revealed four dimensions: self-rated inferiority (negative view of the self), helplessness (negative view of the world), hopelessness (negative view of the future), and detachment (negative view about withdrawal from others).

The authors stated that the item-total correlations provided support for good internal consistency (exact correlations not reported). The Cronbach's alpha was 0.95,

demonstrating evidence of strong internal consistency reliability, but possible redundancy among items. The sample in which the CCI was initially tested consisted of mostly male participants. Researchers have since used the CCI to measure negative thinking in college women and low-income single mothers, providing evidence of internal consistency of the CCI among women (Cronbach's alpha = 0.90 and 0.97; respectively).[4, 19]

Support for construct validity of the CCI was demonstrated by a strong correlation with depressive symptoms measured by the Beck Depression Inventory (r = 0.79, $p < .001$).[1] Divergent validity was tested by comparing the CCI scores with a measurement of IQ. As expected, the authors found a moderately low correlation between the CCI and the IQ score (r = -0.23, $p < .05$), supporting divergent validity.

In order to test the construct validity of the CCI among women, Crandell and Chambless[1] investigated the CCI in a sample of 65 outpatients with panic disorder (82% female), since clinically significant levels of depression are common in this population. The authors found a strong correlation between the CCI and the Beck Depression Inventory (r = 0.77, $p < .001$), lending support for convergent validity of the CCI among women. Other researchers have also found evidence for convergent validity of the CCI among women (see Table 4.2).

Finally, Crandell and Chambless[1] demonstrated that the final version of the CCI had strong evidence of concurrent validity. The total score of the CCI correctly predicted the classification of 92% of the participants among the three groups: depressed psychiatric patients, non-depressed psychiatric patients, and normal controls.

3.2. Automatic Thoughts Questionnaire

Hollon and Kendall[17] developed the ATQ in 1980 to measure the frequency of negative automatic thoughts in individuals with depression. The authors defined automatic thoughts as involuntary, stream-of-consciousness cognitions. The items were developed by asking undergraduate students to recall a depressing event. The students were asked to record thoughts or self-statements that "popped into their head." A total of 100 items were generated for further testing.

After developing the initial items, Hollon and Kendall revised and validated the ATQ with a sample of 348 undergraduate students. Participants were asked to indicate how frequently, the thought occurred over the last week, on a scale from 1 (not at all) to 5 (all the time). Twenty-six students were classified as having clinically significant depressive symptoms according to the Beck Depression Inventory. The authors found

that 30 items discriminated between students with and without depressive symptoms. These 30 items were included in the final version of the ATQ. Principal components analysis revealed that the scale contained four dimensions: personal maladjustment and desire for change, negative self-concept and negative expectations, low self-esteem, and giving up/helplessness. The authors state that these four dimensions reflect only two components of Beck's *cognitive triad*: negative views of the self and future.

Hollon and Kendall reported good evidence for internal consistency of the ATQ using split-half (r = 0.97) and Cronbach's alpha (r = 0.96). Although the strong Cronbach's alphas suggest there could be redundancy among the 30 items, item-total correlations were all significant and ranged from moderate to strong (r = 0.47 - 0.78), suggesting that the strong Cronbach's alpha may reflect strong internal consistency rather than redundancy. Harrell and Ryon[20] provided evidence for internal consistency reliability, but possible redundancy of the ATQ in a sample that included depressed outpatients and non depressed medical outpatients. Cronbach's alpha was 0.98 for both the depressed and the non depressed groups, while the item-total correlations ranged from 0.56 to 0.91 Wong et al. also provided support for internal consistency of the ATQ (Cronbach's alpha = 0.96) among a sample of Chinese patients with chronic physical illness.[21]

Hollon and Kendall[17] provided support for construct validity of the ATQ. In a sample of undergraduate students, participants with depressive symptoms had higher scores on the ATQ when compared to participants without depressive symptoms (79.6 vs. 48.6, $p < .001$). Further support for the construct validity of the ATQ was found in moderate to strong correlations (exact correlations not reported) with the Beck Depression Inventory and the Minnesota Multiphasic Personality Inventory Depression Scale.

In evaluating divergent validity, Hollon and Kendall[17] hypothesized that there would be a weak correlation between the ATQ and the State-Trait Anxiety Inventory. Surprisingly, there was a strong correlation (r = 0.79) between the two scales. The authors suggest that the lack of divergent validity of the ATQ in this sample may have been due to the small number of participants with depressive symptoms (8%) and the frequent comorbidity of anxiety and depressive symptoms.

In contrast, Lamberton and Oei[22] reported evidence for the divergent validity of the ATQ in a sample of patients with major depressive disorder. In this study, there was a strong correlation between the ATQ and the Beck Depression Inventory (r = 0.63), and

a weak correlation between the ATQ and the Beck Anxiety Inventory (r = 0.17). Lamberton and Oei suggest that they were able to find support for divergent validity of the ATQ because their sample contained large numbers of both depressed and anxious patients.

Although the ATQ was originally developed with undergraduate students, Harrell and Ryon[20] provided evidence for concurrent validity of the ATQ with psychiatric patients. Participants were divided into three groups: a depressed psychiatric group, a non-depressed psychiatric group, and a non-depressed medical patient group. Group assignments were made on the basis of a combination of Beck Depression Inventory scores, the Minnesota Multiphasic Personality Inventory Depression Scale, and clinician depression rating scores. The ATQ scores correctly classified 93% of the patients as depressed psychiatric patients, non-depressed psychiatric patients, or non-depressed medical patients.

3.3. Cognition Checklist- Depression

Beck, Brown, Steer, Eidelson, and Riskind[23] developed the Cognitive Checklist (CCL) in 1987 to measure the frequency of negative thinking found in depression and anxiety. The authors defined negative thinking of depression as automatic thoughts, perceptions, and images that center on negative attitudes toward the self, past and future. In contrast, negative thinking of anxiety was defined as automatic cognitions focused on a theme of danger. The authors stated that previous scales, such as the CCI and ATQ, did not adequately differentiate between cognitions found in depression and anxiety. Therefore, Beck et al.[23] designed a depression subscale (CCL-D) and an anxiety subscale (CCL-A) that specifically measure cognitions found in depression and anxiety.

Items for the CCL were derived from collections of automatic thoughts that were recorded by patients in daily thought diaries during Cognitive Therapy treatment. The preliminary version of the CCL contained a total of 43 items. In order to revise the scale and provide evidence of validity, the authors recruited a sample of 618 psychiatric outpatients. The sample was divided into two subsamples: an index sample and a cross-validation sample. All of the patients were diagnosed according to a Structured Clinical Interview. Major depressive disorder was a primary diagnosis in 51% of the index sample and 46% of the cross-validation sample. Generalized anxiety disorder was a primary diagnosis in 24% of the index sample and 35.7% of the cross-validation sample.

Participants were asked to indicate how often each thought occurred to them on a 5-point scale ranging from 0 (never) to 4 (always) when recalling specific situations (attending a social occasion, with a friend, working on a project, and experiencing pain or physical discomfort) and regardless of the situation. No specific time frame was given for the situation recall. During the revision process, the first 212 participants were also asked to label the predominant affect (depression or anxiety) they experienced while thinking each thought. A SPSS Discriminant program was used to determine whether each item was able to discriminate between patients with a primary diagnosis of depression versus anxiety. Items which discriminated between depressed and anxious patients were included in the CCL-D subscale only if patients labeled the thought as depressing. The final CCL-D subscale contained 14 items, and the CCL-A subscale contained 12 items. The authors conducted factor analysis with the entire CCL scale and found evidence for two dimensions: negative thinking of depression and negative thinking of anxiety. The items on the CCL-D subscale fit into only three of the five recalled situations: attending a social occasion, with a friend, and regardless of the situation.

Results from the cross-validation sample showed good evidence for internal consistency. Cronbach's alpha for the CCL-D was 0.92, and the average item-total correlation was strong (r = 0.65).[23] Steer et al.[24] also found good evidence for the internal consistency of the CCL- D in a large study that tested the psychometric properties of the CCL-D among undergraduate students and psychiatric outpatients (Cronbach's alpha = 0.90 – 0.93).

Beck et al.[23] provided evidence of construct validity for CCL-D. Although the CCL-D was correlated with both depressive symptoms and anxiety symptoms as measured by the Hamilton Psychiatric Rating Scales, the CCL-D was more strongly correlated with the depression scale (r = .62) than with the anxiety scale (r = .37). This lends support for both convergent and divergent validity.

Results from a study by Steer et al.[24] provide additional evidence for convergent and divergent validity of the CCL-D. The sample consisted of psychiatric outpatients diagnosed with a primary mood disorder (51.6%), a primary anxiety disorder (35.3%), or other psychiatric disorders (13.1%) The authors also tested the CCL-D with a sample of undergraduate students. The results demonstrated that the CCL-D was strongly correlated with the Beck Depression Inventory among both psychiatric outpatients (r = .66, p < .001) and undergraduate students (r = .58, p < .001). In contrast, the CCL-D had

a weaker correlation with the Beck Anxiety Inventory among both psychiatric outpatients (r = .32, p < .001) and undergraduate students (r = .37, p < .001).

The CCL-D has less evidence for divergent validity among patients who are hospitalized for a chronic physical illness. Clark, Cook, and Snow[25] reported that the CCL-D demonstrated divergent validity among psychiatric inpatients; however, when the CCL-D was administered to patients who were hospitalized with a chronic physical illness, the CCL-D was strongly correlated with both depression and anxiety subscales of the Hospital Anxiety and Depression Scale (r = .60 and .69, p < .001). This indicates that the CCL-D may be less likely to discriminate between negative thinking of anxiety and depression in patients with a chronic physical illness.[25]

In testing concurrent validity of the CCL, Beck et al.[23] divided patients into a depressed group and an anxious group based on a Structured Clinical Interview and Hamilton Anxiety and Depression Scale scores. Patients were included in the depressed group if they had both a primary diagnosis of depression and a Hamilton Depression Scale score at least 0.5 standard deviation higher than the Hamilton Anxiety Scale score. Similarly, patients were included in the anxious group if their primary diagnosis was anxiety and their Hamilton Anxiety Scale score was at least 0.5 standard deviation higher than the Hamilton Depression Scale score. Using a discriminant classification analysis, the CCL was able to correctly classify 83% of the depressed patients and 79% of the anxious patients.

Researchers have used the CCL-D to compare levels of negative thinking between depressed psychiatric patients and depressed physically ill patients. Clark, Cooke, and Snow[25] classified patients as depressed according to the Structured Clinical Interview for the DSM-III. The mean score of the CCL-D was substantially lower in the depressed physically ill group compared to the depressed psychiatric group (12.22 vs. 24.24, p < 0.05). Martens et al.[26] also found a low level of negative thinking among patients who had recently experienced a myocardial infarction and were classified as depressed according to a Structured Clinical Interview. The depressed post-myocardial infarction patients had significantly lower scores on the CCL-D compared to psychiatric outpatients (28 vs. 34.9, p = 0.01), after adjusting for age, sex, educational level, and marital status. The results from these studies raise the possibility that depressed patients who have recently experienced a myocardial infarction have lower levels of negative thinking than depressed, physically healthy patients; however, these results

could also indicate that the CCL-D does not effectively measure negative thinking in patients who are post-myocardial infarction.

4. Comparison of the strengths and weaknesses of the measures

The three instruments reviewed in this paper each have strengths and weaknesses (see Table 4.3). All three measures have evidence for internal consistency reliability and four types of construct validity: factorial, convergent, divergent, and concurrent validity. With regard to internal consistency, all three measures demonstrated strong Cronbach's alphas (greater than 0.90) among various populations, providing good evidence of internal consistency reliability. The strong Cronbach's alphas could reflect possible redundancy, but it is also possible that these Cronbach's alphas could be due to strong internal consistency rather than redundancy, as reflected in the item-total correlations reported by some authors.[23,24]

With regard to factorial validity, a component of construct validity, each instrument displayed a different number of dimensions of negative thinking. The CCI contained four dimensions: the classic *cognitive triad* of negative thoughts of the self, world, future, plus an additional dimension—negative thoughts of withdrawal from others. The ATQ also displayed four dimensions; however, these dimensions reflected only two components of the *cognitive triad*: negative views of the self and future. The CCL as a whole showed evidence of two dimensions: negative thinking of depression and anxiety. The CCL-D subscale was not subjected to its own factor analysis, so it is not known how many dimensions of depressive negative thinking it contains.

The CCI, ATQ and CCL-D all have evidence supporting convergent validity with measures of depressive symptoms. The instruments also have strong support for concurrent validity as evidenced by the ability to discriminate between patients with and without depression; however, the instruments have varying levels of support for divergent validity. The CCI exhibited divergent validity with a measure of IQ, but the authors did not test its divergent validity with anxiety. The ATQ has conflicting evidence regarding its divergent validity with anxiety; therefore, it is not clear whether the ATQ can distinguish negative thoughts of depression from those of anxiety. In contrast, Beck e al.[23] specifically designed the CCL to discriminate between negative thoughts found in depression and anxiety. Since then, researchers have demonstrated strong evidence of divergent validity of the CCL-D with anxiety among clinical and non-clinical populations.

The length of the CCI (45 items) and the ATQ (30 items) make them less practical for use in research and particularly in clinical settings. In addition, researchers

have not described whether the 11 positive buffer items on the CCI are necessary. In contrast, the CCL-D is brief (14 items), making it easier to administer in a clinic setting or research trial.

Researchers have yet to provide strong support for the reliability and validity of these three measures among populations with a chronic physical condition, such as in patients with HF. The reliability and validity of the CCI has not been tested in patients with chronic physical conditions. The ATQ has been shown to have good internal consistency reliability among Chinese patients with a chronic physical condition, but its construct validity has not been tested among patients with a chronic physical condition. The reliability and validity of the CCL-D have been tested among patients who are physically ill; however, results from two studies have demonstrated that depressed patients who are physically ill have significantly lower mean scores on the CCL-D when compared to depressed psychiatric patients.[25, 26] These results may indicate that depressed patients with a physical illness have lower levels of negative thinking. On the other hand, these results could raise concerns regarding the ability of the CCL-D to effectively measure negative thinking in patients with a chronic physical condition such as HF.

The CCI and CCL-D were developed with a psychiatric population, while the ATQ was developed with undergraduate students. Heart failure is a chronic physical condition that increases in prevalence with age and is the most common reason for hospitalization in elderly adults.[27, 28] It is possible that the ATQ's items, generated by college students, may not capture negative thinking found in older patients with a chronic physical condition. For example, patients with HF have described negative thoughts such as, "I'm a burden to my family," "I have nothing to offer anymore," and "Useless, I'm just no good."[13] These thoughts may not be reflected by questionnaires that were developed in physically healthy populations; however, all three instruments have been tested in a variety of populations, including patients with mental illness. Therefore, it may only be necessary to collect more psychometric data to support the reliability and validity of these instruments among patients who have chronic HF.

5. Recommendations for the measurement of negative thinking in heart failure

In summary, the CCI, ATQ, and CCL-D have good evidence of reliability and validity among clinical and non-clinical populations. The CCI has the greatest potential for measuring negative thinking in patients with HF. This instrument has the advantages of being developed with a clinical psychiatric population and captures a wide range of

negative thinking content: negative thoughts about the self, world, future, and withdrawal from others. In contrast, the ATQ was developed with undergraduate students, and factor analysis has revealed that it only measures two components of Beck's *cognitive triad*. Although the psychometric properties of the CCI have not been tested among patients with chronic physical illness, the CCI is preferred over the CCL-D because results from several studies raise questions regarding the construct validity of the CCL-D for measuring negative thinking in patients with a chronic physical condition.

Although the CCI demonstrated the best potential for measuring negative thinking in patients with HF, the CCI in its current form is too long to be clinically useful. Future research is needed to develop a shortened version of the CCI for use in clinical research and practice. It is suggested that researchers use a combination of strategies to determine which items to retain, including: exploratory factor analysis, corrected item-total correlations, Cronbach's alpha if the item were deleted, and examination of means and standard deviations of items to examine variability or lack thereof.

Following the development of a shortened version of the CCI, studies are needed to: 1) provide evidence for internal consistency reliability of the CCI as a measure of negative thinking in patients with HF, 2) examine the dimensionality of the CCI via confirmatory factor analysis to determine if the four original dimensions are retained in a sample of patients with HF who have depressive symptoms, 3) provide evidence for convergent validity with depressive symptoms and divergent validity with anxiety among patients with HF, and 4) determine whether the CCI is sensitive to changes in negative thinking in patients with HF as a result of intervention.

6. Conclusion

Negative thinking is a modifiable target for the treatment of depressive symptoms in patients with HF. In order to determine the effectiveness of interventions designed to reduce negative thinking in patients with HF, researchers need a reliable and valid instrument to measure negative thinking. The CCI has excellent potential for measuring negative thinking in patients with HF. However, research is needed to shorten the CCI and examine its psychometric properties of the CCI among patients with HF before it can be recommended for routine use to measure negative thinking in this population.

Table 4.1: Description of three instruments of negative thinking

Instrument (Year)	No. Items	Response Options	Scoring and Range	Sample Item	Normative Values*
Crandell Cognitions Inventory[1] (1986)	34 negative items; 11 positive buffer items	Indicate the frequency of each thought on a scale from 1 (almost never) to 5 (almost always)	Total scores are scored by summing the item responses to the 34 negative items (11 buffer items are not scored) Range = 34 - 170	I've made such a mess of my life.	*Crandell and Chambless[1]* • Depressed psychiatric patients: 116 ± 20.1 • Non-depressed psychiatric patients: 74.2 ± 21.4 • Normal controls: 44.6 ± 8.3
Automatic Thoughts Questionnaire[17] (1980)	30 negative items	Over the past week, rate the frequency of each thought on a scale from 1 (not at all) to 5 (all the time)	Total scores are calculated by summing the 30 item responses Range = 30-150	It's just not worth it.	*Hollon and Kendall[17]* • Depressed college students: 79.6 ± 22.3 • Non-depressed college students: 48.6 ± 10.9 *Dozois et al.[29]* • Elderly: 41.6 ± 13.7 • Women: 53.5 ± 18.6 • Men: 48.4 ± 16 *Harrell and Ryon[20]* • Depressed psychiatric outpatients: 88.9 ± 21.2 • Non-depressed psychiatric outpatients: 42.4 ± 10 • Non-depressed medical outpatients: 38.4 ± 8.2
Cognition Checklist-Depression[23] (1987)	14 items	Frequency of each thought on a scale from 1 ("never") to 5 ("always")Indicate how often each	Total scores are calculated by summing the 14 responses Range = 14-70	I don't deserve to be loved.	*Beck et al.[23]* • Depressed psychiatric outpatients: 54.8 ± 9 • Anxious psychiatric outpatients: 45 ± 7.7

Table 4.1 (Continued)

Instrument (Year)	No. Items	Response Options	Scoring and Range	Sample Item	Normative Values*
		thought occurs on a 5-point scale ranging from 0 (never) to 4 (always) during specific situations (being with a friend or attending a social occasion) or regardless of the situation. No specific time frame is given for the situation recall.			*Martens et al.*[26] • Depressed psychiatric outpatients: 34.9 ± 11.9 • Depressed post-MI patients: 28 ± 9.6 • Non-depressed post-MI patients: 17.8 ± 8.5 *Clark et al.*[25] • Depressed hospitalized medical patients: 12.2 ± 9.1 • Non-depressed hospitalized medical patients: 5.1 ± 7.2 • Healthy controls: 4.5 ± 6.2

*Normative values are given as mean ± standard deviation

Table 4.2 (Continued)

4.2: Psychometric properties of three measures of negative thinking

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
Crandell Cognitions Index (CCI)						
Crandell[1] (1980)	To develop a measure of depressive self-statements	Cross-sectional	*N* = 212 Psychiatric depressed group, psychiatric non-depressed group, normal control group Females 16%	Cronbach's alpha = 0.95	Concurrent validity, convergent and divergent validity, and factor validity were supported	The authors did not describe support for content validity; however, items were developed based on Beck's Cognitive Model of Depression
Crandell[1] (1980)	To test the validity of the CCI among women	Cross-sectional	*N* = 65 Outpatients with agoraphobia with panic disorder Female 82%	Not reported	Convergent validity was supported: CCI was strongly correlated with the BDI	This study was reported in the same journal article as above
Peden[7] (2000)	To test the effectiveness of a cognitive behavioral group intervention on depressive symptoms	Experimental, repeated-measures	*N* = 92 College women	Cronbach's alpha = 0.95-0.97	Not reported	The intervention group experienced a significant decrease in CCI scores at one month and six months
Peden[4] (2004)	To determine predictors of depressive symptoms in	Cross-sectional	*N* = 205 Low-income single mothers	Cronbach's alpha = 0.97	Convergent validity: CCI strongly correlated with the BDI, CES-D,	Divergent validity was not evaluated in this study.

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
	low-income single mothers				Everyday Stressors Index, and Rosenberg Self-Esteem Scale	
Peden[30] (2005)	To test a cognitive behavioral group intervention on depressive symptoms	Experimental, repeated measures	N = 136 Low income, single mothers	Cronbach's alpha = 0.94-0.97	Not reported	The authors found a decrease in negative thinking in the intervention group at one month and six months
Automatic Thoughts Questionnaire (ATQ)						
Hollon[17] (1980)	To develop an instrument that measures automatic thoughts of depression	Cross-sectional	*N* = 348 College students Female 42%	Split half coefficient = 0.97; coefficient alpha = 0.96; item-total correlations ranged from 0.47 to 0.78	Convergent validity was supported but divergent validity (with anxiety) was not supported. Factor validity was supported.	There was a low percentage of students with depressive symptoms (7.5%); depression was not diagnosed with a diagnostic interview
Dobson[31] (1983)	To test the psychometric properties of	Cross-sectional	*N* = 456 College students Female 49%	Cronbach's alpha = 0.96	Convergent validity was supported: the ATQ was strongly	The ATQ had stronger support for construct

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
	three cognitive assessment scales			(males) and 0.95 (females)	correlated with the BDI (r = 0.62, p < .001)	validity and reliability than two other cognitive measures: the Dysfunctional Attitude Scale and the Interpretation Inventory
Harrell[20] (1983)	To test the psychometric properties of the ATQ in a clinical sample	Cross-sectional	N = 61 Adult outpatients divided into three groups: depressed, non-depressed psychiatric, and non-depressed patients who were seeking medical care Female 56%	Split-half coefficient = 0.96 Cronbach's alpha = 0.98 Item-total correlations ranged from 0.56 to 0.91 (p = .001)	Convergent validity was supported: depressed patients had higher ATQ scores than non-depressed patients. Concurrent validity was supported: ATQ correctly classified 93.1% of patients.	Patients were separated into groups based on BDI scores and a clinical depression rating given by a therapist
Lamberton[22] (2008)	To determine whether anxiety and depression can be differentiated by thought content	Cross-sectional	N = 135 Outpatients with depressive disorder, anxiety disorder, or both Female 55%	Not reported	Convergent and divergent validity were supported: ATQ was strongly correlated with BDI (r = 0.63, p < .05) and weakly	

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
					correlated with the Beck Anxiety Inventory (r = 0.17, p < .05)	
Wong[21](2007)	To test the efficacy of cognitive behavioral group therapy on depressive symptoms in patients with chronic physical illness	Randomized, controlled trial	*N* = 73 Chinese patients with chronic physical illness Female 75%	Cronbach's alpha = 0.96	Not reported	The authors used a Chinese version of the ATQ but did not specify how it was translated and validated
Cognition Checklist- Depression subscale (CCL-D)						
Beck[23] (1987)	To develop and test the psychometric properties of the CCL	Cross-sectional	*N* = 618 Outpatient psychiatric patients Female 55%	Cronbach's alpha = 0.92; Average corrected item-total correlation = 0.65; Test-reliability was tested by administerin	Convergent and divergent validity were supported: the CCL-D was strongly correlated with the HAM-D (r = 0.62, p < .001) and weakly correlated with the HAM-A (r = 0.37, p < .001). Concurrent validity was supported: the CCL-D correctly	The CCL also contains an anxiety subscale

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
				g the CCL-D to 66 patients after six weeks: the correlation between the two scores was 0.76 ($p < .001$)	classified 83% of depressed patients	
Steer[24] (1994)	To test the psychometric properties of the CCL in psychiatric outpatients and university students	Cross-sectional	*N* = 2,197 Psychiatric outpatients (*n* = 1,907) and university students (*n* = 290) Female 58%	Cronbach's alpha = 0.91-0.93; Item-total correlations ranged from 0.56 to 0.85 in the psychiatric sample and 0.41 to 0.72 in the student sample	Factor analysis supported the original two dimensions (anxiety and depression) only in the outpatient group. Construct validity was supported: the CCL-D was strongly correlated with the BDI and HAM-D, but only weakly correlated with the Beck Anxiety Inventory and HAM-A.	The results from this study provide additional support for the strong convergent and divergent validity of the CCL-D.
Clark[25] (1998)	To compare depressive and	Cross-sectional	*N* = 152 Depressed	Not reported	Among medical patients, convergent	The CCL-D has stronger support

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
	anxious symptoms among physically ill patients and depressed psychiatric inpatients		psychiatric inpatients (*n* = 75), hospitalized medical patients with chronic physical illness (*n* = 52), healthy controls (*n* = 25) Female 59%		validity was supported but not divergent validity; the CCL-D was strongly correlated with the BDI and the HADS-D (r = 0.77, 0.60, $p < .001$), but it was strongly correlated with measures of anxiety such as the HADS-A (r = 0.69, $p < .001$). In contrast, the CCL-D showed good evidence of convergent and divergent validity in psychiatric inpatients; the CCL-D was weakly correlated with HADS-A (r = 0.29, $p < .05$).	for divergent validity among psychiatric patients compared to medical patients.
Martens[26] (2006)	To describe level of negative thinking in patients who	Cross-sectional	*N* = 120 Non-depressed post MI patients (n = 40), depressed post MI patients (n	Not reported	Not reported	The depressed post-MI patients had lower levels of negative thinking than

Table 4.2 (Continued)

First Author (date)	Purpose	Design	Sample	Reliability	Evidence of Validity	Comments
	are post MI		= 40), and depressed psychiatric outpatients (n = 40) Females = 38%			psychiatric outpatients (28 vs. 34.9, *p* = .013) after controlling for age, sex, education, and marital status

BDI = Beck Depression Inventory; CES-D = Center for Epidemiologic Studies Depression scale; HAM-A = Hamilton Rating Scale for Anxiety; HAM-D = Hamilton Rating Scale for Depression; HADS-A = Hospital Anxiety and Depression Scale- Anxiety subscale; HADS-D = Hospital Anxiety and Depression Scale- Depression subscale, MI = myocardial infarction

Table 4.3: Strengths and weaknesses of the three measures

	Strengths	Weaknesses
Crandell Cognitions Inventory	• Captures four dimensions of negative thinking • Evidence for internal consistency • Evidence for convergent and concurrent validity • Developed with a clinical psychiatric population	• Psychometric properties have not been tested among patients with chronic physical illness • Divergent validity with anxiety has not been tested • Possible redundancy • Length (45 items)
Automatic Thoughts Questionnaire	• Evidence for internal consistency • Evidence for convergent, divergent, and concurrent validity	• Validity has not been tested in patients with chronic physical illness • Developed with healthy undergraduate students • Captures only two dimensions of the cognitive triad • Possible redundancy • Length (30 items)
Cognitive Checklist-Depression	• Evidence for internal consistency • Evidence for convergent, divergent, and concurrent validity • Brevity (14 items) • Developed with a clinical psychiatric population	• Possible redundancy • Evidence for lack of divergent validity among patients with chronic physical illness • Depressed patients with a chronic physical illness have lower scores on the CCL-D than depressed psychiatric patients, raising questions regarding its validity among patients with chronic physical conditions

CHAPTER FIVE:

Evaluating the Psychometric Properties of a Measure of Negative Thinking in Patients with Heart Failure

1. Introduction

Heart failure (HF) is a chronic condition which is increasing in prevalence and incidence worldwide.[1] Depressive symptoms are common in patients with HF; up to 48% of patients with HF experience symptoms of depression,[2] while 20% have major depressive disorder.[3] Both depressive symptoms and depression have a profound effect on morbidity, mortality, and health-related quality of life among patients with HF.[2] Patients with HF who experience depressive symptoms have a two-fold increase in the risk of death compared to patients who do not have depressive symptoms.[3]

Negative thinking is a type of cognition in which the individual experiences automatic, repetitive thoughts regarding the self, world, future, and withdrawal from others.[4, 5] Negative thinking is a risk factor for depressive symptoms,[6, 7] and the presence of negative thinking in major depressive disorder lessens the response to treatment for depression.[8] In a qualitative study, we found that patients with HF who have depressive symptoms described experiencing negative thinking, which worsened the depressed mood. Examples of negative thoughts described by patients with HF who were experiencing depressive symptoms include “I have nothing to offer anymore,” “I’m a failure,” and “I can’t justify my existence.”[9]

Cognitive therapy, which focuses on the reduction of negative thinking, was developed by Beck in 1967 and has been shown to be an effective treatment for depression in the general population.[4, 10] Cognitive therapy is based on Beck’s *cognitive model* of psychopathology.[11] The main tenet of the *cognitive model* is that each psychological disorder has a distinct cognitive content. For example, negative thinking of depression focuses on negative thoughts about the self, world and future, while negative thinking of anxiety focuses on a theme of danger. Researchers have proposed that cognitive therapy may be an effective treatment for depressive symptoms in patients with HF.[12] However, in order to test the effects of cognitive therapy on depressive symptoms in patients with HF, researchers need a reliable and valid instrument for measuring negative thinking in this population.

The Crandell Cognitions Inventory (CCI) has been used to measure negative thinking in psychiatric outpatients, healthy adults,[5] low income single mothers,[6] and

college women.[13] There is no evidence for the reliability and validity of this instrument as a measure of negative thinking in patients with chronic HF. Furthermore, researchers have not tested the divergent validity of the CCI with anxiety. Therefore, the purpose of this study was to evaluate psychometric properties of the Crandell Cognitions Inventory among patients with HF. The specific aims of this study were to 1) determine the reliability and internal consistency of the CCI, and 2) evaluate the construct validity of the CCI by examining the dimensionality of the CCI and testing the following hypotheses in patients with HF: (a) patients with depressive symptoms will have higher levels of negative thinking than patients without depressive symptoms, (b) patients will demonstrate a dose-response relationship between depressive symptoms and negative thinking, and (c) the correlation between the CCI and depressive symptoms will be stronger than the correlation between the CCI and anxiety symptoms.

2. Methods

2.1. Design

This study was nested in a clinical trial testing biofeedback and cognitive therapy in patients with HF (Biobehavioral Intervention in Heart Failure, NIH, NINR R01 NR008567). Institutional Review Board approval was obtained both for the original study and this data analysis. Only baseline data were used for data analysis.

2.2. Sample and setting

The sample consisted of 179 outpatients with a confirmed diagnosis of impaired left ventricular systolic HF or preserved systolic function HF. Patients were recruited from June 2004 to May 2009 from the cardiology clinic at an academic medical center as well as a Veteran's Administration cardiology clinic. Patients were eligible for enrollment if they were on stable doses of HF medications for at least three months and were not referred for cardiac transplantation. Patients were excluded for valvular or peripartum HF etiology, myocardial infarction or stroke within the past three months, major stroke sequelae, or coexisting terminal illness. Informed consent was obtained and patients completed questionnaires at a General Clinical Research Center. Research associates were present at all times to answer questions and confirm that no items were inadvertently skipped.

2.3. Measures

2.3.1. Negative thinking

Negative thinking was defined as cognitions about the self, world, future, and withdrawal from others, and was measured using the Crandell Cognitions Inventory (CCI).[5] The CCI is a 45-item scale that contains of 34 negative items and 11 positive, non-scored, "buffer" items. Patients are asked to endorse how frequently they experience a thought on a scale from 1 (almost never) to 5 (almost always). Total scores are calculated by summing the item responses to the 34 negative items. Higher scores indicate a greater frequency of negative thinking.

The CCI was developed by Crandell and LaPointe in 1979 with a sample of depressed psychiatric patients, non-depressed psychiatric patients, and normal controls.[5] Researchers have demonstrated strong evidence for the reliability of the CCI among psychiatric patients, healthy adults, college women, and single mothers.[5, 6, 13] Crandell and Chambless[5] stated that the CCI displayed good internal consistency; however, item-total correlations for the CCI have not been reported.

Researchers have examined the validity of the CCI using factor analysis, criterion-related validity and construct validity.[5] Crandell and Chambless theorized that the CCI would have three dimensions with factor analysis: negative thoughts about the self, world, and future. However, factor loadings revealed four dimensions: self-rated inferiority, helplessness, hopelessness, and detachment from others. These results led Crandell and Chambless to add negative thoughts about withdrawal from others to their conceptual definition of negative thinking.[5]

Crandell and Chambless have found good evidence for criterion-related and construct validity of the CCI.[5] The CCI was able to correctly predict the classification (depressed psychiatric patients, non-depressed psychiatric patients, and normal controls) of 92.5% of the sample. Construct validity was tested with convergent and divergent validity; researchers demonstrated that the CCI strongly correlates with depressive symptoms as measured by the Beck Depression Inventory.[5, 6] Crandell and Chambless[5] also found evidence that the CCI has divergent validity with measures of IQ.[5] However, researchers have not explored the divergent validity of the CCI with regards to anxiety symptoms.

2.3.2. Depressive symptoms

Depressive symptoms were defined as clinically significant symptoms of depression that can exist with or without the presence of major depressive disorder.[14] The Beck Depression Inventory version II (BDI)[15] was used to measure depressive symptoms. The BDI is a 21-item questionnaire that assesses the presence and severity of depressive symptoms. Patients who score greater than 13 are considered to have clinically significant depressive symptoms. BDI scores can also be used to categorize people into 4 groups; those with no symptoms (0-13), mild (14-19), moderate (20-28), and severe depressive symptoms (29-63). The original version of the BDI has been shown to predict mortality and hospitalizations in patients with HF.[16] The Cronbach's alpha for our sample was 0.88, providing evidence of adequate reliability.

2.3.3. Anxiety symptoms

Anxiety symptoms were defined as the negative emotional response to a perceived threat.[17] Anxiety symptoms were measured with the state anxiety subscale of the Brief Symptom Inventory (BSI).[18] The BSI anxiety subscale consists of six items. Patients rate each symptom on a 5-point scale (0-4) of distress ranging from 'not at all' to 'extremely'. Item scores are summed and the mean obtained. The possible range of mean scores for the subscale is 0 to 4, with higher scores indicative of higher levels of anxiety. Researchers have demonstrated evidence for internal consistency, reliability, factor validity, and construct validity of the BSI anxiety subscale among medically ill patients.[18, 19]

2.4. Data analysis

Data analysis was conducted using SPSS version 16. The baseline characteristics of the sample were described using percentages, means, and standard deviations. The homogeneity or internal consistency of the CCI was assessed using Cronbach's alpha. Alpha coefficients greater than 0.70 were considered to indicate adequate internal consistency, while coefficients greater than 0.90 were considered to indicate item redundancy. Corrected item-total correlations were also used to determine the internal consistency of the CCI. An acceptable coefficient for item-total correlations was greater than 0.20.[20]

Dimensionality was assessed using principal components analysis with direct oblimin rotation because correlations between factors were assumed. Factors would be

retained for rotation if they had an eigenvalue of greater than one, and if the factors were located above the elbow on the scree plot.

To further assess construct validity of the CCI, we tested three hypotheses. First, independent t-test was used to determine whether patients with depressive symptoms had higher levels of negative thinking than patients without depressive symptoms. Second, one-way analysis of variance and post-hoc comparisons using the Tukey HSD test were used to evaluate whether patients demonstrate a dose-response relationship between depressive symptoms and negative thinking. Third, Pearson correlation coefficients were used to test the relationships among CCI scores, depressive symptoms, and anxiety symptoms.

3. Results

3.1. Sample characteristics

Sample characteristics are described in Table 5.1. The mean level of negative thinking in our sample was 58.6 ± 24.7. The potential range for the CCI is 34 to 170; the range for our sample was 34 to 158. Approximately 24% of the sample (n = 42) experienced clinically significant depressive symptoms based on BDI scores.

3.2. Reliability

Cronbach's alpha for the CCI was 0.96, indicating adequate internal consistency, but possible redundancy of items. The corrected item-total correlations for each of the items were all greater than 0.4, indicating that each of the items contributed to the scale (Table 5.2).

3.3. Construct validity

3.3.1. Principal components analysis

The 34 items were subjected to principal components analysis. Based on the scree plot, one primary component emerged that explained 46% of the variance (see Figure 5.1). Three other components had eigenvalues greater than 1. These components explained an additional 5%, 4%, and 3% of the variance. When the component matrix was examined, all of the items loaded strongly on the first component. A few items loaded on components two through four but these loadings were weaker than the loadings for the first component. Additionally, only one factor was located above the elbow on the scree plot. These results indicate that the CCI exhibited only one component; therefore, direct oblimin rotation was not performed.

3.3.2. Hypothesis testing

The level of negative thinking among patients with depressive symptoms was higher than that of patients without depressive symptoms (86.7 ± 25 vs. 50 ± 26, $p <$ .001). There was a dose-response relationship between depressive symptoms and negative thinking. Patients with mild moderate, and severe depressive symptoms had progressively higher levels of negative thinking when compared to patients with no depressive symptoms, providing evidence for convergent validity ($F = 61.8$, $p < .001$). Post-hoc tests revealed significant differences in levels of negative thinking between each of the groups (see Figure 5.2). The correlation between negative thinking and depressive symptoms ($r = .732$, $p = .01$) was slightly higher than that of negative thinking and anxiety symptoms ($r = .595$, $p = .01$), providing evidence for divergent validity.

4. Discussion

This was the first study to examine the psychometric properties of an instrument that measures negative thinking in patients with HF. The results of our study demonstrate that the CCI has adequate internal consistency and reliability in patients with HF. However, the high Cronbach's alpha suggests that there may be redundancy in the items. The CCI contains 45 items and is longer than other measures of negative thinking, such as the Cognitions Checklist- Depression scale (14 items).[11] To improve the clinical and research utility of the CCI, future researchers should develop and test the reliability and validity of a shortened version. Researchers also should identify whether the non-scored, positive buffer items are necessary.

Only one strong component emerged in our principal components analysis. Our findings contrast with those of Crandell and Chambless,[5] who found that the CCI contained four components—negative thoughts about the self, world, future, and withdrawal from others.[5] It is possible that we were unable to adequately assess the dimensionality of the CCI due to a relatively small number of patients in our study with clinically significant depressive symptoms ($n = 42$). Additional factor analyses of the CCI that include samples of patients with HF who have a high level of depressive symptoms are needed.

We hypothesized that patients with depressive symptoms would have higher levels of negative thinking compared to patients without depressive symptoms. Furthermore, we hypothesized that patients with mild, moderate, and severe depressive symptoms would have progressively higher levels of negative thinking compared to

patients with no depressive symptoms. Our findings supported both hypotheses. Most compelling was the dose-response relationship between negative thinking and depressive symptoms. The observation that as levels of negative thinking increase severity of depressive symptoms increased accordingly provides strong support for the convergent validity of the CCI among patients with HF.

Finally, we provided evidence for divergent validity of the CCI with regards to anxiety symptoms. Evidence of divergent validity with anxiety is important because in the *cognitive model*, Beck proposes that depression and anxiety have separate, distinct content.[21] Thus a valid measure of depressive negative thinking in patients with HF should be more strongly correlated with depressive symptoms than anxiety symptoms. In our sample of patients with HF, the CCI was strongly correlated with both depressive symptoms and anxiety symptoms. It is possible that the strong correlation between the CCI and both measures reflects frequent comorbidities of depressive symptoms and anxiety in patients with HF.[22] However, the correlation between the CCI and depressive symptoms was slightly stronger than that of the CCI and anxiety, providing some support for divergent validity of the CCI with anxiety symptoms.

Finally, one limitation should be noted. We did not use a diagnostic interview to categorize patients as depressed or non-depressed. Therefore, we could not evaluate the concurrent validity of the CCI by testing its ability to distinguish levels of negative thinking between depressed and non-depressed patients with HF. The inclusion of a diagnostic interview for major depressive disorder would be beneficial in future psychometric analyses of a shortened version of the CCI.

5. Conclusion

The findings from our study provide strong support for the reliability and validity of the CCI as a measure of negative thinking in patients with HF. Depressive symptoms remain a significant clinical problem in patients with HF, and negative thinking is a potential target for the treatment of depressive symptoms in this population. We recommend the CCI for use in research studies testing interventions that target the reduction of negative thinking in patients with HF. Ongoing research in our group is focused on determining whether CCI scores change over time as a result of interventions that target negative thinking in both outpatients and inpatients with HF. We also plan to develop and test a shortened version the CCI by eliminating redundant

items. A shortened version will improve the CCI's clinical practicality as a measure of negative thinking among patients with acute and chronic HF.

Table 5.1: Sample characteristics (*N* = 179)

Characteristic	Frequency (%) or mean ± standard deviation
Female	59 (33%)
Age	60 ± 13
Married	94 (52.5%)
Minority	35 (19.6%)
Education level	
Less than high school	34 (19%)
High school graduate	44 (24.6%)
Some college or greater	103 (56.4%)
Employment status	
Employed	47 (26.3%)
Disability	36 (20.1%)
Retired	84 (47%)
Body Mass Index	32.5 ± 8.3
NYHA functional class	
Class I	15 (8.4%)
Class II	75 (41.9%)
Class III	71 (39.7%)
Class IV	13 (7.3%)
Left ventricular ejection fraction	36.6 ± 14.8
Ischemic HF etiology	79 (44.1%)
Current smoker	31 (17.3%)
Months since diagnosed with HF	77 ± 84
Comorbidities	
Prior myocardial infarction	88 (49.2%)
Atrial fibrillation	78 (43.6%)
Implanted cardiac defibrillator	63 (35.2%)
Stroke	38 (21.2%)
Chronic obstructive pulmonary disease	32 (17.9%)
Diabetes	79 (44.1%)
Medications	
ACE inhibitor	127 (70.9%)
Beta blocker	157 (87.7%)
Antidepressant	42 (23.5%)
Crandell Cognitions Inventory	58.6 ± 24.7
Beck Depression Inventory-II	9.9 ± 8.2
Brief Symptom Inventory- anxiety subscale	0.61 ± 0.69

NYHA = New York Heart Association, HF = Heart failure, ACE = Angiotensin-converting enzyme

Table 5.2: Item-total correlations for the Crandell Cognitions Inventory (N = 152)

Item	Corrected item-total correlation	Cronbach's alpha if item deleted
1. I'm just a nobody	.555	.963
3. I'll never feel good again	.574	.963
4. I sure have wasted the opportunities in my life	.722	.962
5. I don't know what I should do	.662	.963
6. I'm always letting myself down	.772	.962
8. I've made such a mess of my life	.703	.962
10. Nothing ever works out for me anymore	.700	.962
11. Things really look hopeless	.675	.963
12. Why can't I be happy?	.778	.962
13. It all seems so useless	.765	.962
15. I just don't cut it	.739	.962
16. I sure am bored	.475	.964
17. My life is so confused, I'll never straighten it out	.786	.962
18. I'm a burden to m family	.605	.963
20. I'll never be happy with myself	.702	.962
22. There's no way out of this mess	.625	.963
23. I don't seem to have the energy to get through the day	.639	.963
24. I really can't do what's expected of me	.680	.963
26. No one can know how alone I feel	.661	.963
27. I'll never do as well as others	.624	.963
28. Everything I do is a failure	.698	.963
29. I don't even feel like going out of the house	.533	.964
30. I'm a real disappointment to my family	.687	.963
32. I feel so detached; I just can't communicate	.658	.963
33. I mess everything up	.727	.963
35. I know what I should do but I just can't do it	.554	.964
36. Nothing's ever going to work out for me	.717	.962
37. I feel trapped	.739	.962
38. Daytimes are bad but nighttimes are terrible	.622	.963
39. I just wish it would be all over	.600	.963
41. Nothing seems exciting anymore	.599	.963
43. I wish people would just leave me alone	.540	.963
44. Nobody cares about me	.603	.963
45. I feel so helpless	.654	.963

Figure 5.1: Scree plot of factors of the Crandell Cognitions Inventory in patients with heart failure

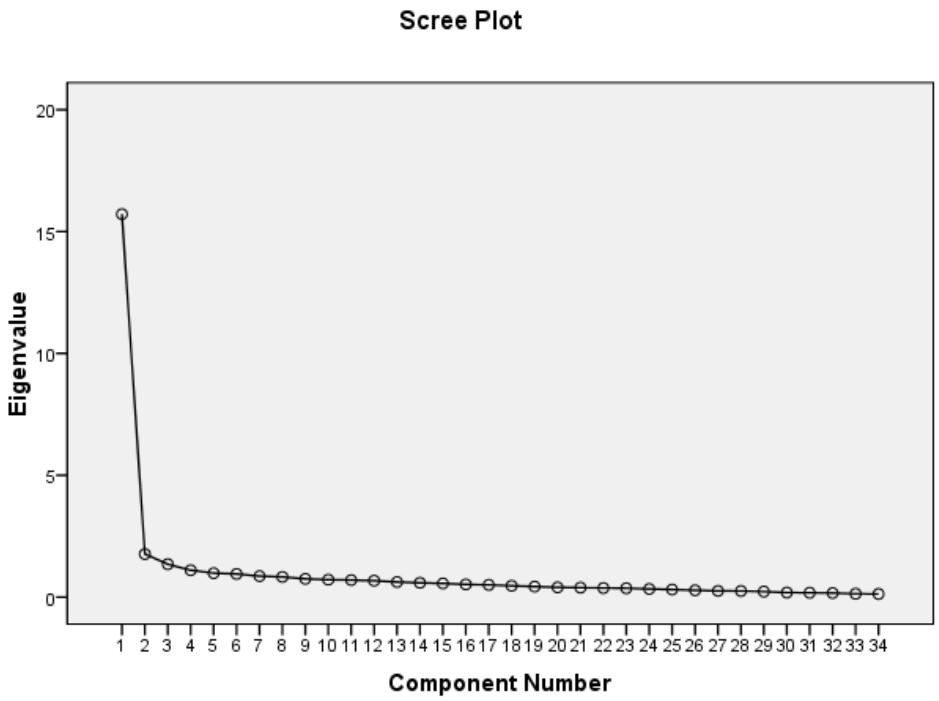
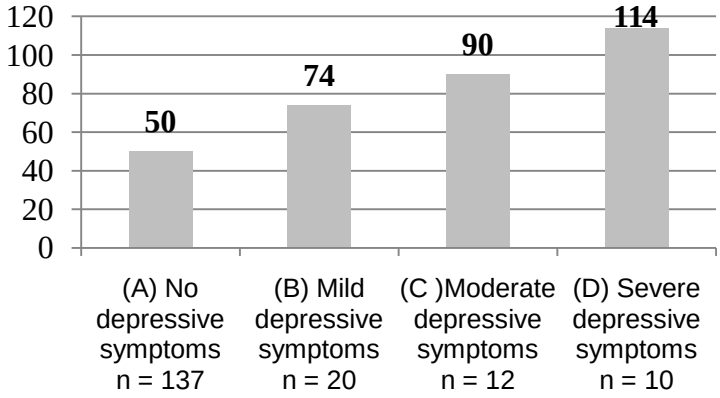


Figure 5.2: Dose-response relationship between depressive symptoms and negative thinking in outpatients with heart failure*



Tukey HSD post-hoc tests	
A < B	$p < .001$
A < C	$p < .001$
A < D	$p < .05$
B < C	$p < .001$
B < D	$p < .001$
C = D	$p = .003$

*One-way analysis of variance, F = 61.8, $p < .001$

Chapter Six:

A Brief Cognitive Therapy Intervention Improves Outcomes in Hospitalized Patients with Heart Failure: A Randomized, Controlled Pilot Study

1. Introduction

Hospitalized patients with HF are at high risk for experiencing depression and depressive symptoms. One-third of patients hospitalized with HF suffer from major depressive disorder.[1] The adverse effects of major depressive disorder and depressive symptoms on mortality and morbidity in patients with HF have been well documented.[2-6] In a meta-analysis, Rutledge et al. demonstrated that patients with HF and depressive symptoms were twice as likely to die or experience a cardiac event when compared to patients without depressive symptoms.[3] Other researchers have found patients with HF who report depressive symptoms during hospitalization are at higher risk for both short-term and long-term mortality.[2, 7] Depressive symptoms also have a negative impact on health-related quality of life (HRQOL) among patients with HF.[4] Interventions to treat depressive symptoms in patients with HF are needed to improve survival and HRQOL.

Negative thinking, a modifiable risk factor for depressive symptoms, is a potential target for intervention in patients with HF. Negative thinking, first described by Beck,[8] can be defined as automatic and persistent negative cognitions about the self, world, future, and interpersonal relationships.[8, 9] In Beck's *cognitive model* of depression, it is postulated that negative thinking influences the emotional, behavioral, and somatic symptoms of depression.[10]

Negative thinking has several adverse consequences in the general population. First, negative thinking has been described as a risk factor for depressive symptoms.[11, 12] Second, negative thinking exacerbates and maintains depressive symptoms, which contributes to a downward spiral of worsening depressive symptoms that may end in major depressive disorder.[8, 13] Third, researchers have found that the presence of negative thinking independently predicts a diminished response to treatment for depression after controlling for history of depression and depressive symptoms at baseline.[14]

Cognitive therapy (CT), the psychotherapeutic intervention based on the *cognitive model,* alters depressive symptoms by redirecting negative thinking. CT has been used successfully to treat depression in multiple populations.[10, 15] Results from previous studies indicate that brief CT interventions that use of thought-stopping and

affirmations to decrease the level of negative thinking reduce depressive symptoms in women at risk for depression.[16, 17]

The availability of a clinically feasible intervention that could be offered at the bedside may increase clinicians' ability to treat depressive symptoms in hospitalized patients with HF. One potential, clinically feasible treatment of depressive symptoms in this population is the use of a single, brief CT intervention focused on reducing negative thinking using thought-stopping and affirmations. Therefore, the purpose of this study was to test the short -term efficacy of a brief CT intervention aimed at decreasing negative thinking for the treatment of depressive symptoms in hospitalized patients with HF. We hypothesized that patients who received the brief CT intervention focused on reducing negative thinking would report lower levels of depressive symptoms and negative thinking, and better HRQOL and cardiac event-free survival at one week and three months compared to a usual care group.

2. Methods

2.1 Design

A randomized, controlled, repeated measures design was used to determine the short-term efficacy of a brief CT intervention. We followed the CONSORT (Consolidated Standards of Reporting Trials) guidelines.[18] The University of Kentucky Institutional Review Board and the Institutional Review Boards at participating hospitals approved the trial. All patients provided informed consent. Enrollment began February 2009 and ended December 2009; follow-up was completed in March 2010.

A sample size of 21 patients per group was determined by an a priori power analysis estimate for a two-group univariate repeated measures ANOVA algorithm with Greenhouse-Geisser correction.[19] A large effect size was chosen based on data from a prior study in which a large group difference in the short-term effect of a similar brief intervention was reported.[20] With 21 subjects per group and an alpha level of .05, the power of the ANOVA F tests would be at least 95% to detect a main effect for group, a main effect for time, and the group by time interaction

2.2 Sample

Patients age 21 and older who were hospitalized with a primary or secondary diagnosis of HF at Central Baptist Hospital or Saint Joseph Hospital in Lexington, KY, were screened for study eligibility. Patients were candidates for inclusion if they had a confirmed diagnosis of preserved or non-preserved systolic function HF. We excluded

patients with co-existing terminal illness, end-stage HF (defined as mechanical pump support, continuous at home inotropic infusions, referral for heart transplant, or hospice care), cognitive impairment, and self-reported severe depression or suicidal ideation.

Eligible patients were approached by the principal investigator (PI) on the second day of hospitalization or later. Recruitment was delayed if the patient was experiencing hypotension (blood pressure <80 mm Hg), a pain level of ≥ 5 on the 0-10 pain scale, or admission to an intensive care unit until medically stable. Patients who agreed to be in the study and provided written consent were screened for depressive symptoms with the Beck Depression Inventory version II (BDI). Those patients with mild to moderate symptoms of depression (BDI score 10-28) were enrolled in the trial. Participation for patients with no depressive symptoms or severe depressive symptoms ended after the baseline assessment; patients with severe depressive symptoms were referred to the attending physician for treatment. Two items on the BDI related to low energy and fatigue were not included in the upper cut-off score because patients admitted to the hospital with HF often report or experience HF-related fatigue.

2.3 Randomization

A random allocation sequence was compiled using a true random number generator.[21, 22] Group assignments were placed in sealed, numbered envelopes. The investigators were blinded to the random allocation sequence. The PI enrolled the patient, completed the baseline assessment, and calculated the baseline BDI score. Immediately after completion of the baseline assessment, the PI opened the envelope to determine group assignment. Patients and investigators were not blinded to group assignment.

2.4 Procedure

Patients were approached by the PI, who introduced herself and explained the purpose of the visit. After written informed consent was obtained, the baseline data collection took place at the patient's bedside. A chart review was conducted to obtain clinical and sociodemographic variables. Privacy was maintained throughout data collection by allowing patients to determine which family members or significant others they would like to have in the hospital room during study procedures.

Both groups completed baseline questionnaires to measure depressive symptoms, negative thinking, and HRQOL. The PI read the questionnaires out loud for patients who needed assistance due to vision impairment, fatigue, or difficulty reading.

The PI calculated the BDI score and patients with mild to moderate depressive symptoms were randomized. Patients with severe depressive symptoms were encouraged to talk to their healthcare provider about their depressive symptoms, and the staff nurse, charge nurse, attending physician, and primary care physician were informed of the patient's high level of depressive symptoms. Patients in the intervention group received the individual intervention prior to discharge, at a time convenient to the patient. Patients in the control group received usual care.

One week after discharge, patients received a phone call reminder to complete and mail the one-week questionnaires. Patients who needed assistance were given the option of having the questionnaires read out loud over the phone. At three months, patients completed the questionnaires again by phone or mail, and patients were asked about current antidepressant medication use, hospitalizations, and emergency department visits that occurred since the baseline assessment. Patients were reimbursed $20 for the baseline assessment and $10 for each follow-up assessment.

2.5 Intervention

The intervention consisted of a single, brief, one-on-one CT session that took place in the hospital and a short telephone booster that took place one week post discharge. The brief CT session and booster were delivered by the PI, a Master's-prepared cardiovascular nurse, in collaboration with a PhD-prepared psychiatric clinical nurse specialist, who had tested a similar intervention with women at risk for depression in several randomized, controlled trials.[16, 17] Previous qualitative work guided the design of the intervention.[23] A colorful, 13 page flip chart with photos was used to guide the written script of the intervention. Patients also received a color booklet to take home.

The script of the intervention followed six simple steps focused on reducing negative thinking using thought-stopping and affirmations (see Table 1). Patients were allowed to choose whether or not they would like a family member or significant other to be present during the intervention, which lasted approximately 30 minutes.

The booster CT session took place during the one week post discharge phone call (after the one week questionnaires were filled out) and lasted 5-10 minutes. During the booster, the PI talked with patients about negative thoughts they have been experiencing and asked about their experiences with the technique of thought-stopping followed by affirmation. The PI reinforced the technique and reminded patients of the importance of using thought-stopping and affirmations.

2.6 Usual care

Patients in the control group received usual care, which consisted of standard evidence-based care for HF according to the American Heart Association/American College of Cardiology 2005 guidelines[24] and the Joint Commission of Healthcare Organizations.[25] All patients with HF received individual discharge teaching on HF care, which included brief written instructions on the emotional consequences of living with HF. All patients were also offered the assistance of a chaplain if desired. In addition, patients in the control group received approximately 40-60 minutes of attention from the PI during recruitment, enrollment, and the baseline assessment.

2.7 Outcome measures

2.7.1 Depressive symptoms

The primary outcome was depressive symptoms, which was defined as self-reported symptoms that may exist with or without the presence of major depression.[26] The Beck Depression Inventory version II (BDI was used to measure depressive symptoms at baseline, one week post-discharge, and three months. The BDI contains 21 items. Scores range from 0-63, with a score of 0-13 indicating none to minimal depressive symptoms, 14 to 28 indicating mild to moderate symptoms, and 29-63 indicating severe symptoms.[27] The BDI has well-established validity, reliability, and internal consistency for measurement of depressive symptoms in medical and non-medical populations.[28, 29]

2.7.2 Health-related quality of life

Health-related quality of life (HRQOL) was defined as a patient's perception of how HF has impacted the following domains of daily life: physical functioning, emotional status, health perception, and symptom burden. The Minnesota Living with HF Questionnaire (MLHFQ) was used to measure HRQOL. This instrument has been demonstrated to be reliable and valid in patients with HF.[30] Patients respond to 21-items with the stem question: "Did your HF prevent you from living as you wanted during the past month by." The statements are rated from 0 (no impact) to 5 (most negative impact). Total scores range from 0 to 105. Higher scores indicate worse HRQOL.

2.7.3. Negative thinking

Negative thinking was defined as negative cognitions about the self, world, future, and withdrawal from others, and was measured using the Crandell Cognitions Inventory (CCI).[9] The CCI is a 45-item scale that consists of 34 negative items and 11

positive, non-scored, buffer items. Patients are asked to endorse how frequently they experience a thought. Total scores are calculated by summing the item responses to the 34 negative items; scores range from 34 to 170. Higher scores indicate a greater frequency of negative thinking. Researchers have demonstrated evidence for the reliability and validity of the CCI in psychiatric patients and healthy controls.[9]

2.7.4. Event-free survival

A secondary endpoint was three-month cardiac event-free survival, defined as time to a combined end point of cardiovascular mortality, cardiovascular hospitalization, or cardiovascular emergency department (ED) visit. Dates and reasons for death, cardiovascular hospitalizations and ED visits were determined by medical record review and patient interview. A cardiovascular rehospitalization or ED visit was defined as an unanticipated admission for the following reasons: HF exacerbation, shortness of breath, cardiovascular-related chest pain, myocardial infarction, syncope, dysrhythmias (including internal cardiac defibrillator [ICD] firing), ICD or biventricular pacemaker placement due to worsening HF, coronary artery disease, hypertension, and palpitations.

2.7.5. Additional variables of interest

To completely describe the sample and obtain data on potential confounding variables, the following demographic information was collected at baseline: age, sex, race/ethnicity, marital status, education level, and months since diagnosis. The following clinical characteristics were determined by chart review: height, weight, smoking status, most recent left ventricular ejection fraction, comorbidities, and medications on discharge (i.e. drugs, doses, frequencies)..

New York Heart Association (NYHA) functional class and comorbidities were also collected to further characterize the sample. NYHA functional class is a subjective indicator of functional status and was determined by patient interview at baseline and three months. Patients were assigned a classification of I (ordinary physical activity causes no symptoms of fatigue, dyspnea, angina or palpitations), II (symptoms with ordinary physical activity), III (symptoms occur with less than ordinary physical activity) or IV (symptoms occur even at rest).[31]

2.8 Data analysis

Demographic and clinical differences between the groups were assessed using t-tests or chi-square tests of association. Data analysis for each of the specific aims was conducted using intent-to-treat. To determine the effects of the intervention, repeated

measures ANOVA was used to compare levels of depressive symptoms, HRQOL, and negative thinking at baseline, one week post-discharge and three months between the two groups. The main effects for group, time, and group by time interaction were tested. Cardiac event-free survival was calculated using Kaplan-Meier survival curves with the log-rank test. Differences in the mean number of cardiac events between groups were determined with an independent t-test. An alpha of .05 was considered statistically significant for all analyses.

3. Results

3.1 Sample characteristics

A participant flow diagram is provided in Figure 6.1. Of the 407 patients who were screened for eligibility, 327 were excluded. The most common reasons for exclusion were cognitive impairment (n = 95), co-existing terminal illness (n = 34), and end-stage heart failure (n = 17).

Eighty patients provided informed consent and were screened for depressive symptoms. Of these 80 patients, 42 (53%) had mild-moderate depressive symptoms and were retained in the study, while 33 patients (41%) with no depressive symptoms and 5 patients (6%) with severe depressive symptoms were excluded. The characteristics of the final sample are provided in Table 6.2. The sample consisted of 42 patients with HF (45% female, 66 ± 11 years, 75% NYHA Class III). The mean depressive symptom score was 16.7 ± 6.7, which indicates mild depressive symptoms. One-third of the patients had a history of depression noted in the chart, and 41% were taking antidepressants at discharge.

There were no differences between intervention and control groups with regard to sex, age, NYHA Class, ejection fraction, marital status, ejection fraction, medications, and baseline depressive symptoms, negative thinking, or HRQOL. Patients in the control group had a higher mean body mass index, and patients in the intervention group were more likely to have prior coronary bypass graft surgery and co morbid chronic pulmonary obstructive disease. There were no baseline differences between groups with regard to depressive symptoms, HRQOL, and negative thinking.

Of the 41 patients for whom cardiac event-free survival data were collected, 19 patients (54%) experienced a cardiovascular event during the three month follow-up period. Two patients died from worsening HF, 13 patients had at least one cardiovascular hospitalization, and 4 patients visited the ED for cardiovascular reasons.

3.2 Primary outcome

As shown in Table 6.3, BDI scores improved over time in both groups ($p < .001$); however, there were no significant difference between groups. Figure 6.2 suggests a trend for patients in the intervention group to have a faster improvement in depressive symptoms compared to patients in the control group, but this trend was not significant.

3.3 Secondary outcomes

Table 6.3 also presents the data on HRQOL and negative thinking. Both groups experienced significant improvements in HRQOL and negative thinking over time. There were no significant group by time interactions for HRQOL or negative thinking. Although Figure 6.3 and 6.4 appear to show trends toward faster improvement in HRQOL and negative thinking in the intervention group compared to the control group, these trends were not significant.

Patients in the intervention group had longer cardiac event-free survival compared to the control group (Figure 6.5). At three months, 80% of patients in the intervention group were alive without a cardiac event, compared to 40% of patients in the control group ($p = .048$). Furthermore, the mean number of cardiovascular events over the three month time period was higher in the control group compared to the intervention group (0.8 vs 0.2, $p < .008$).

4. Discussion

Researchers have established that depressive symptoms contribute to poor health outcomes in patients with HF.[3] In this study, I tested the effects of a brief, clinically feasible CT intervention that can be administered by nurses for the treatment of depressive symptoms in hospitalized patients with HF. Patients in both groups experienced a significant improvement in depressive symptoms after discharge, but there were no differences between groups over time. It is likely that this was due to the large natural improvement that occurs with depressive symptoms after hospitalization for HF and attrition. Although the study was powered to detect differences in depressive symptoms between groups, I did not expect patients in both groups to experience such a large reduction in depressive symptoms during the follow-up period. In addition, data on depressive symptoms were missing from five patients in the control group (2 patients died, 2 patients declined further participation, and 1 was lost to follow-up) and 2 patients in the intervention group (1 was lost to follow-up, 1 was too busy to complete the 3

month questionnaires). Overall, the promising results of this pilot study support replicating the study with a larger sample size.

We found that, in general, depressive symptoms improve soon after discharge in patients who are hospitalized with HF. Few researchers have described the trajectory of depressive symptoms in patients with HF. Koenig et al.[32] found that although many patients with HF and depression experience spontaneous remission within several months after hospital discharge, 36% and 52% of patients continue to have symptoms of mild and major depression, respectively. The main predictor of shorter time to remission was lower levels of depressive symptoms. In our study, we enrolled patients with mild to moderate symptoms and excluded those with high levels of depressive symptoms. The mean level of depressive symptoms in our study was 16.. This relatively low level of depressive symptoms could explain why both groups improved rapidly after discharge. Future research is needed to determine whether brief CT interventions are beneficial in patients with higher levels depressive symptoms who are less likely to improve on their own.

Our finding that patients experience improvement in HRQOL after discharge is consistent with findings from Riegel et al.,[33] who combined data sets from nine experimental and quasi-experimental trials to determine the trajectory of HRQOL after hospitalization in patients who receive usual care. The investigators found that HRQOL, as measured by the Minnesota Living with Heart Failure Questionnaire, naturally improves by a mean of 13 points in the three months after hospital discharge. ,

We also found that the intervention group had the same improvement in HRQOL as the usual care group. This result may be explained by findings from Riegel et al.,[33] who found that only high-intensity interventions had an effect on HRQOL after hospitalization. Similarly, a more intense dose of the CT intervention offered in this study may be needed to improve HRQOL after discharge.

This is the first study to examine changes in negative thinking as a result of a CT intervention in patients with HF. Although there was a trend toward faster improvement in the intervention group and an increase in negative thinking in the control group after discharge, there were no significant differences between groups. Previous researchers have found that in women, negative thinking can be modified as a result of a similar CT intervention that uses thought-stopping and affirmations.[16, 17] It is probable that we were unable to detect a difference between groups because of a small sample size and

missing data on the CCI. During the follow-up time points, several patients were too ill to complete all of the instruments, and in those cases the CCI was dropped from the questionnaire administration because of its length. A reliable and valid shortened version of the CCI would make it more practical for use in patients with an acute or chronic physical health condition.

This study is the first to demonstrate that a brief, nurse-delivered CT intervention has a measurable impact on cardiac event-free survival in patients with HF. It is not clear why the intervention group experienced longer cardiac event-free survival when there were no differences between groups on depressive symptoms, negative thinking, or health-related quality of life. It is possible that patients in the intervention group experienced improvements in an unmeasured construct such as positive thinking or sympathetic nervous system activation.

Furthermore, we do not know why a survival benefit was demonstrated in our small study when other investigators did not find a survival difference when testing CT in a much larger sample. The ENRICHD[35] investigators tested the effects of CT on survival in 2,481 patients with cardiovascular disease who had recently experienced a myocardial infarction and were diagnosed with clinical depression or self-reported poor social support. Although patients in the intervention group experienced a significant reduction in depressive symptoms as compared to the control group, there were no differences in cardiac event-free survival between groups. Our study differs from the ENRICHD study with regard to both patient population and timing of the cardiac outcome. Whereas the ENRICHD investigators recruited patients who had recently experienced a myocardial infarction, we enrolled patients who were experiencing an acute exacerbation of HF and were highly vulnerable to cardiac events during the three months post-hospitalization. In our study, we had an overall cardiac event rate of 54%, which may have contributed to our ability to detect a difference in outcomes. Furthermore, the ENRICHD investigators followed patients for an average of 29 months, while we measured cardiac event-free survival for three months. Future research is needed to test the effects of the brief CT intervention on long-term cardiac event-free survival in patients with HF.

4.1 Limitations

Several limitations should be noted. First, this study needs to be replicated using a larger sample before definitive conclusions can be drawn. Second, due to resource

limitations, the follow-up questionnaires were administered by the same researcher (PI) who also administered the intervention. However, several steps were taken to reduce the potential for social desirability or researcher bias. The baseline assessment was completed prior to randomization so that the PI was blinded to group assignment during the first assessment. In addition, follow-up questionnaires were administered by mail whenever possible, and the PI did not discuss the intervention with the patient during the one-week and three-month assessment.

5. Conclusions

Our findings indicate that although depressive symptoms improve rapidly after discharge, patients would benefit from a nurse-administered, brief CT intervention while hospitalized for HF. This intervention is replicable, practical for the acute care setting, and may improve short-term cardiac event-free survival in patients with HF. Future research is needed to replicate this study using a randomized controlled trial that is powered to detect differences in primary and secondary outcomes, includes patients with higher levels of depressive symptoms, and incorporates a longer follow-up period.

Table 6.1 Summary of the six steps of the brief cognitive therapy intervention

Step 1: Depression and heart failure
Description of symptoms of depression Depression is common in people with heart failure Bad news: Depression is bad for the heart Good news: There are treatments for depression *Discussion question:* "Do any of these symptoms of depression sound familiar to you?"
Step 2: Thoughts, feelings, and behaviors
The connection between thoughts, feelings and behaviors Thoughts can be false and negative, and these thoughts contribute to painful feelings and negative behaviors of depression Two real-life patient stories are told that demonstrate the link between a stressful situation and negative thinking, feelings, and behaviors *Discussion question:* "Do these stories sound familiar to you? Do you see how the person's negative thinking led to painful feelings and negative behaviors?"
Step 3: Your story
Interactive component: Patient is asked to describe a recent stressful situation *Discussion questions:* "What kind of thoughts were going through your head? How did these thoughts make you feel? Did you notice any behaviors?"
Step 4: Thought-stopping
Notice you have a negative thought Snap your fingers or clap your hands and say, "Stop!" By stopping the negative thought you help take away the painful feelings that go with the thought *Interactive component:* Patient practices the stop technique with the interventionist
Step 5: Affirmations
An affirmation is a short, simple phrase that states how life is at its very best Use first person, present tense, and positive words; if you have a religious faith you may use it in your affirmation *Interactive component:* The patient comes up with 3-4 affirmations with the help of the interventionist The interventionist writes the affirmations on 2 brightly colored sticky notes: one for the hospital room and one for the patient to take home
Step 6: Homework
Practice the stop technique every time you hear a negative thought Hang the list of affirmations somewhere you will see it every day For every negative thought, think at least 2 positive thoughts

Table 6.2: Baseline characteristics of entire sample and patients randomly assigned to the brief cognitive therapy intervention versus usual care*

Characteristic	Overall sample (n = 42)	Intervention group (n = 21)	Usual care group (n = 21)	*p* value
Female	19 (45%)	10 (48%)	9 (43%)	0.8
Age	66 ± 11	68 ± 10	64 ± 12	0.2
Married	24 (57%)	14 (67%)	10 (48%)	0.2
Minority	4 (10%)	2 (10%)	2 (10%)	1.0
Education level				
Less than high school *High school graduate* *Some college or greater*	15 (36%) 12 (29%) 14 (33%)	10 (48%) 6 (30%) 5 (24%)	5 (25%) 6 (30% 9 (45%)	0.3
Employment status				
Employed *Sick leave or disability* *Retired*	3 (7%) 15 (36%) 21 (50%)	2 (10%) 9 (43%) 10 (48%)	1 (5%) 6 (30%) 13 (65%)	0.5
Body Mass Index	31± 8.6	28.2 ± 6.4	33.8 ± 9.7	0.03
NYHA functional class				
Class II *Class III* *Class IV*	8 (19%) 31 (74%) 3 (7%)	6 (29%) 15 (71%) 0 (0%)	2 (10%) 16 (76%) 3 (14%)	0.08
Left ventricular ejection fraction	39.5± 16.4	40.8 ± 17	38.1 ± 16.1	0.6
Etiology of HF				
Ischemic *Hypertensive* *Idiopathic* *Other*	26 (62%) 8 (19%) 6 (14%) 2 (10%)	13 (62%) 4 (19%) 2 (10%) 2 (10%)	13 (62%) 4 (19%) 4 (19%) 0 (0%)	0.5
Smoking status				
Never smoked *Former smoker* *Current smoker*	17 (41%) 14 (33%) 7 (17%)	7 (33%) 9 (43%) 5 (24%)	10 (50%) 8 (40%) 2 (10%)	0.4
Second hand smoke exposure	12 (29%)	6 (29%)	6 (29%)	0.6
Months since diagnosed with HF	39 ± 56	45 ± 68	32 ± 37	
Newly diagnosed with HF in the past month	13 (31%)	8 (38%)	5 (24%)	0.3
History of depression	14 (33%)	9 (43%)	5 (24%)	0.2
History of anxiety	13 (31%)	8 (38%)	5 (24%)	0.3
Comorbidities				
Prior myocardial infarction	17 (41%)	8 (38%)	9 (43%)	0.8
Atrial fibrillation	19 (45%)	8 (38%)	11 (52%)	0.4
CABG surgery	12 (29%)	10 (48%)	2 (9.5%)	.006
Biventricular	17 (41%)	8 (38%)	9 (43%)	0.8

pacemaker				
Implanted cardiac defibrillator	19 (45%)	8 (38%)	11 (52%)	0.4
Prior stroke	8 (19%)	4 (19%)	4 (19%)	1.0
Chronic obstructive pulmonary disease	17 (41%)	12 (57%)	5 (25%)	0.04
Diabetes	19 (45%)	9 (43%)	10 (48%)	0.8
Renal dysfunction	16 (38%)	9 (43%)	7 (33%)	0.5
Medications on discharge				
ACE inhibitor	19 (45%)	10 (48%)	13 (62%)	0.4
Antiogensin receptor blocker	6 (14%)	3 (14%)	3 (14%)	1.0
Beta blocker	36 (86%)	17 (81%)	19 (91%)	0.4
Digoxin	17 (41%)	9 (43%)	8 (38%)	0.8
Diuretic	26 (62%)	16 (76%)	10 (48%)	.06
Antidepressant	17 (41%)	8 (38%)	9 (43%)	0.8
Laboratory values on admission				
B-type natriuretic peptide	746 ± 914	963 ± 1171	529 ± 496	0.2
BUN	26.2 ± 18.9	31.7 ± 23.6	20.5 ± 9.8	0.06
Creatinine	1.3 ± 1.2	1.5 ± 1.6	1.1 ± 0.43	0.3
Sodium	138.5 ± 6.2	138 ± 5.3	139 ± 7.2	0.7
Beck Depression Inventory-II	16.7 ± 6.7	15.8 ± 6.5	17.6 ± 6.7	0.4
Minnesota Living with Heart Failure Questionnaire	54 ± 25	50 ± 25	58± 25	0.3
Crandell Cognitions Inventory	61 ± 19	59 ± 19	64 ± 18	0.5

*Data are given as n (%) or mean ± standard deviation unless otherwise indicated. Independent t-test used to test difference between 2 groups. Chi-square analysis was used for categorical variables.

Table 6.3 Primary and secondary outcomes in patients with heart failure

	Least-Squares Mean (SE)		Time			Group x Time		
Measure	**Brief CT Intervention**	**Usual care**	***F* test**	***p* value**	***Partial eta squared***	***F* test**	***p* value**	***Partial eta squared***
BDI-II (n = 34)								
Baseline	14.8 (5.5)	17.5 (7.3)						
1 week	9.9 (6.8)	14 (9.6)	16.7	< .001	0.52	.1.0	.38	0.06
3 months	9.3 (5.4)	10.7 (8.8)						
CCI (n = 32)								
Baseline	61 (20)	64 (20)						
1 week	56 (23)	68 (25)	3.3	.05	0.19	0.8	.47	0.05
3 months	54 (23)	59 (22)						
MLHFQ (n = 32)								
Baseline	51 (23)	67 (18)						
1 week	42 (25)	64 (19)	13.7	<.001	0.49	0.5	.59	0.04
3 months	29 (25)	42 (25)						

CT = Cognitive Therapy, BDI-II = Beck Depression Inventory version II, CCI = Crandell Cognitions Inventory, MLHFQ = Minnesota Living with Heart Failure Questionnaire

Figure 6.1 Study flow diagram

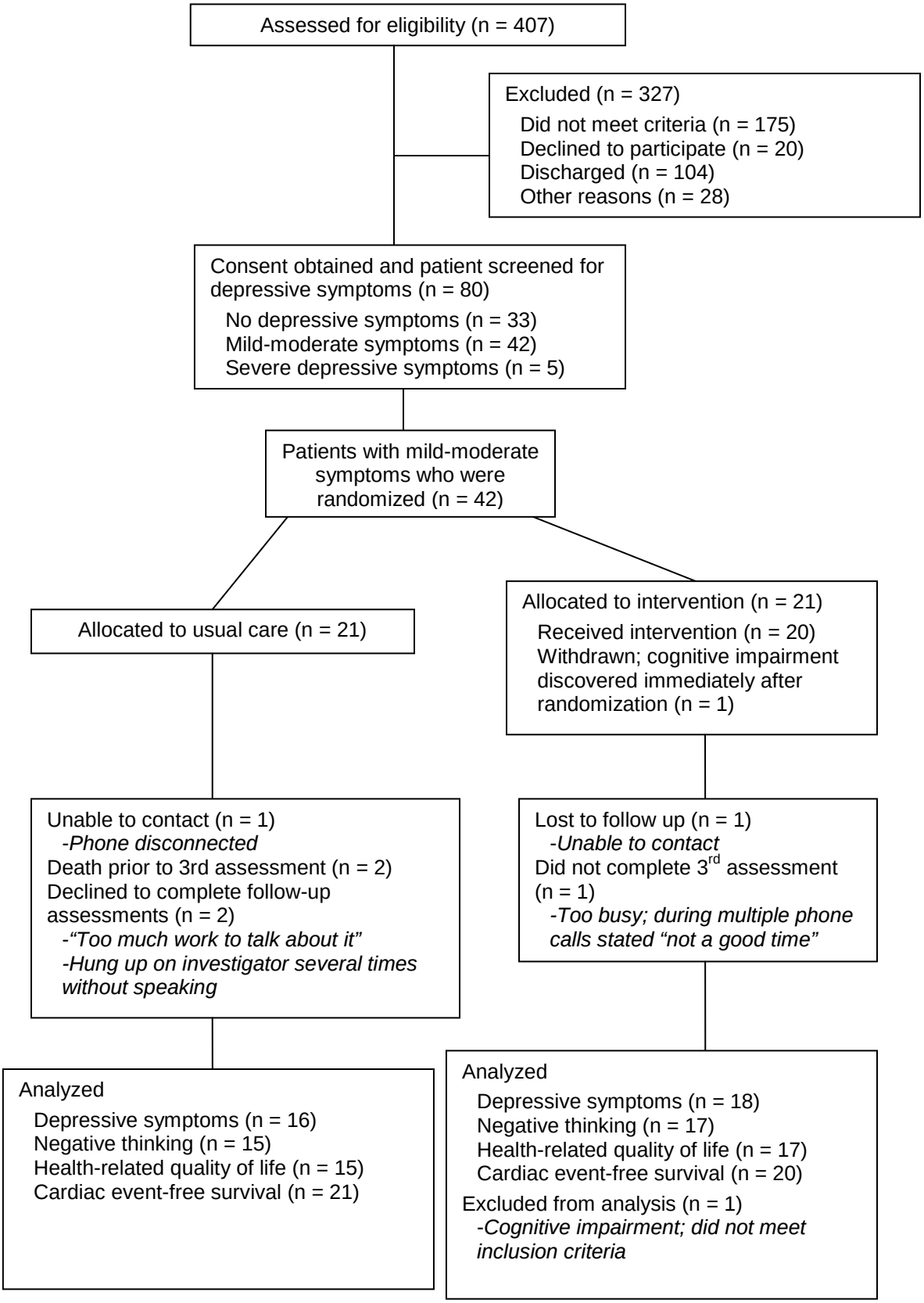


Figure 6.2 Change in depressive symptoms over time (n = 34)

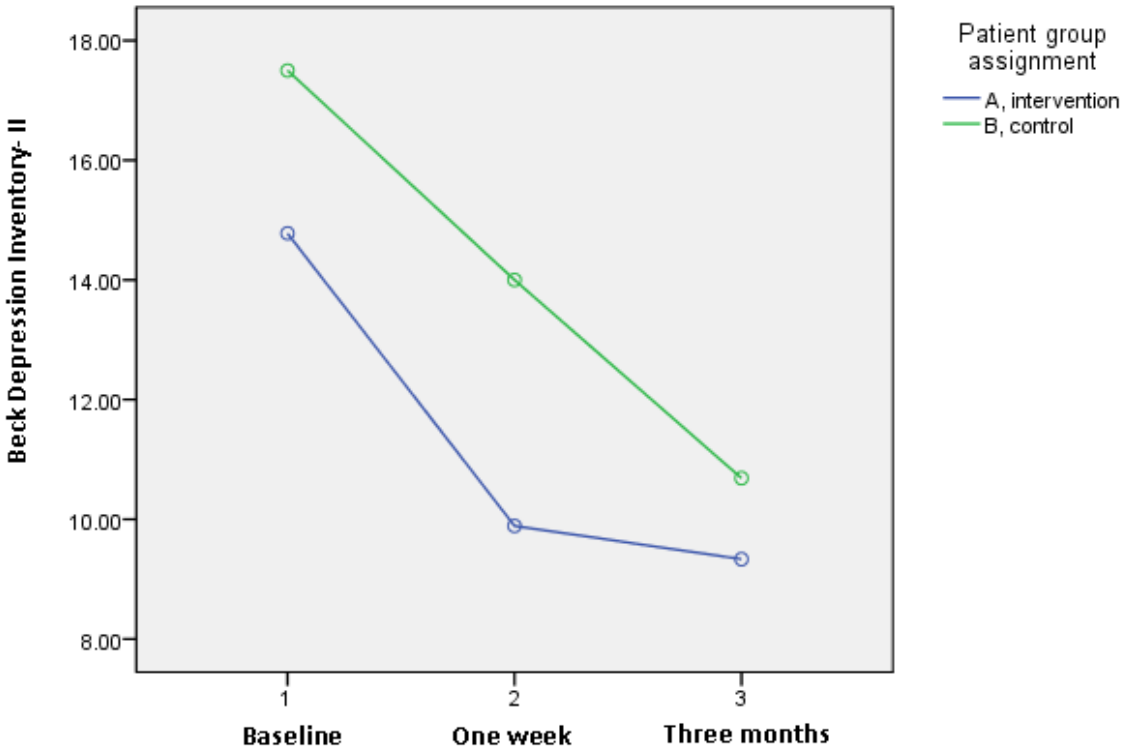


Figure 6.3 Changes in health-related quality of life over time (n = 32)

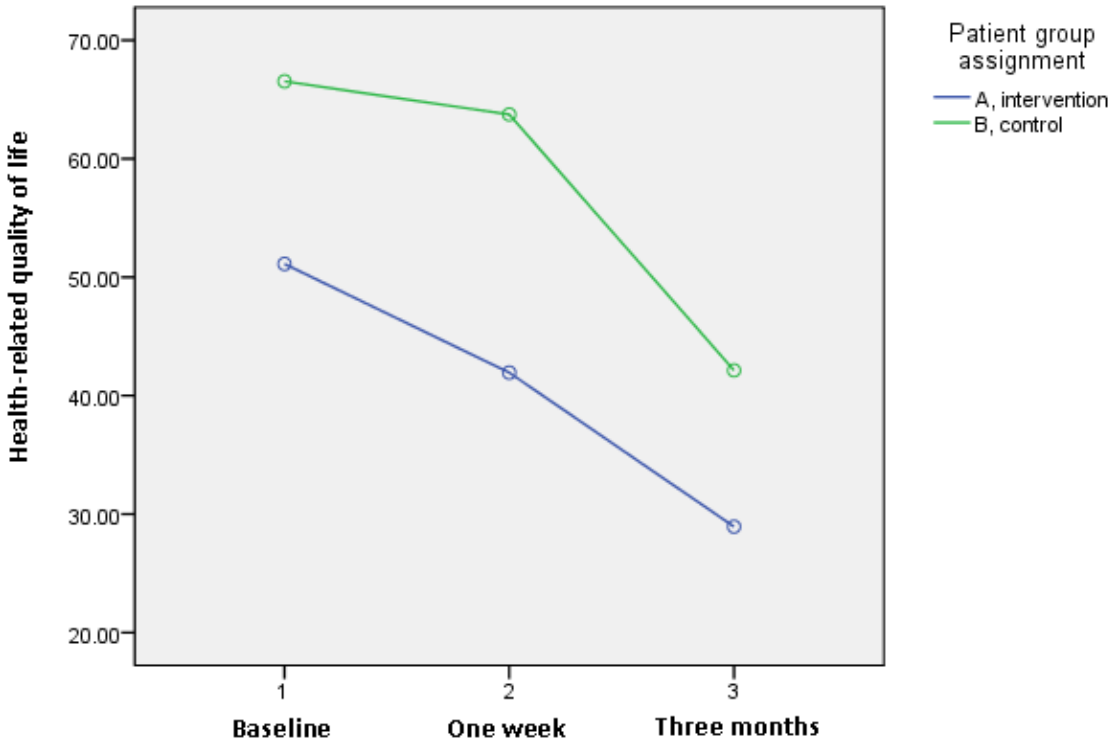


Figure 6.4 Changes in negative thinking over time (n = 32)

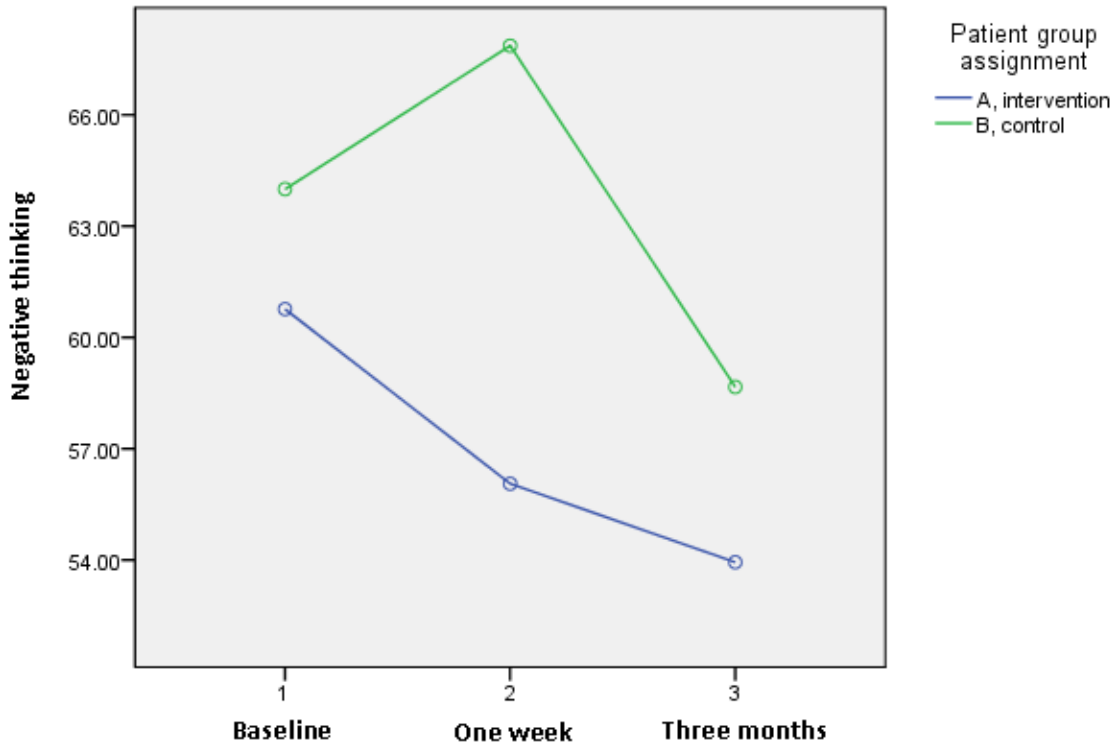
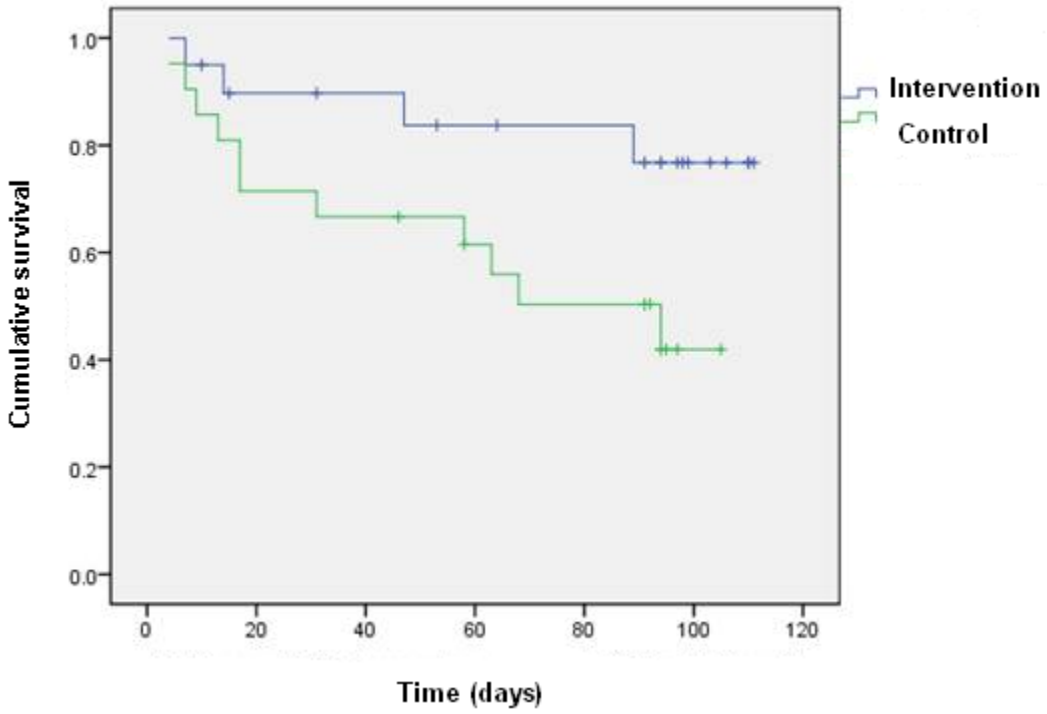


Figure 6.3 Kaplan-meier cardiac event-free survival curve (n = 41; log rank test p < .05).



CHAPTER SEVEN:

Conclusions

1. Background and purpose

The overall purpose of this dissertation was to develop and test a brief CT intervention for the treatment of depressive symptoms in patients with HF. The following manuscripts were completed prior to testing the CT intervention: 1) a review of the evidence for CT in treating depressive symptoms in patients with cardiovascular-related conditions,[1] 2) a qualitative study to describe the experience of living with depressive symptoms in patients with HF and identify strategies that could be used to manage these symptoms,[2] 3) a critical review of the literature to identify a measure of negative thinking that can be used in patients with HF[3], and 4) an evaluation of the psychometric properties of this measure of negative thinking. Based on findings from these manuscripts, a randomized, controlled pilot study was conducted to test the effects of a brief CT intervention on outcomes of hospitalized patients with HF who report depressive symptoms.

The presence of depressive symptoms is an important clinical problem in patients with HF. One out of five patients with HF suffers from major depressive disorder, and an even higher percentage of patients experience self-reported depressive symptoms.[4] Experiencing even mild symptoms of depression place a patient with HF at higher risk for mortality, morbidity, and worse health-related quality of life.[4, 5]

Depressive symptoms are under-diagnosed and under-treated in patients with HF. Up to 40% of patients with HF who have depression are not recognized as depressed by health care providers.[6] The treatment of depression in this population is complicated by a lack of specific guideline recommendations for depressive symptoms in patients with HF.[7] Although researchers have described the problem of depression in patients with HF, little is known about which treatments may be effective. Recent evidence suggests that nursing interventions may be more effective than antidepressants for the treatment of clinical depression in patients with HF.[8] Cognitive therapy (CT) is an intervention that can be administered by nurses[9] and may be an effective treatment for depressive symptoms in patients with HF.

The purpose of this chapter is to summarize and synthesize the findings of this dissertation. This chapter will also advance the state of the science of the treatment of depressive symptoms in patients with heart failure (HF) by making recommendations for

practice and future research.

2. Summary of findings

Chapter Two is a review of the literature[1] on the current evidence for CT interventions in patients with cardiovascular-related illnesses. Based on limitations of the studies and mixed results, it was concluded that there is insufficient evidence to recommend the use of CT for the treatment of depressive symptoms in patients with cardiovascular conditions. It was recommended that future researchers test CT interventions in patients with HF via large, randomized, controlled trials that incorporate the following design elements: the CONSORT guidelines for reporting of clinical trials, adequate representation of women, long-term follow up (at least one year), and replicable descriptions of the CT intervention. It was also concluded that research is needed to determine whether CT is effective for patients with HF who have an acute exacerbation, the appropriate dose of a CT intervention, and the impact of the intervention on other outcomes, such as health-related quality of life and cardiovascular events.

Chapter Three is a qualitative study[2] in which patients with HF described the experience of living with depressive symptoms. This study was nested in a randomized, controlled trial testing biofeedback and CT in outpatients with HF. The patients described experiencing negative thoughts such as “I can’t justify my existence,” “I have nothing to offer anymore,” and “I’m a failure.” The patients stated that “dwelling” on these negative thoughts worsened their depressed mood. The patients also described using positive thinking to manage their depressive symptoms, thus providing support for a CT intervention in this population. The use of a cognitive strategy was an expected finding in the five participants who received biofeedback and CT in the larger study. However, the control group participants, who never received a CT intervention, also discussed positive thinking as a strategy for managing depressive symptoms. This finding suggests that interventions focused on reducing negative thinking using thought-stopping and affirmations may be particularly welcomed by patients with HF. However, negative thinking has never been quantitatively measured in patients with HF.

Chapter Four is a critical review of the literature[3] to identify a measure of negative thinking that has the best potential for use in patients with HF. Three of the most widely used instruments for measuring negative thinking were reviewed: the Crandell Cognitions Inventory, Automatic Thoughts Questionnaire, and Cognitive

Checklist- Depression. The Crandell Cognitions Inventory (CCI) was recommended as having the highest potential for measuring negative thinking in patients with HF. The CCI has the advantage of being developed with a clinical population and captures a wide range of negative thinking content. However, the CCI required psychometric testing among patients with HF before it can be recommended for use.

Chapter Five is an evaluation of the reliability and validity of the CCI in patients with HF. Cross-sectional questionnaire data were collected from 179 outpatients with HF. Cronbach's alpha was 0.96, indicating adequate internal consistency but probable redundancy. Corrected item-total correlations were greater than 0.4, indicating adequate homogeneity. Negative thinking displayed a dose-response relationship with depressive symptoms. The CCI had a stronger correlation with depressive symptoms than anxiety. Overall, the results support the reliability and validity of the CCI as a measure of negative thinking in patients with HF, although a shortened version would be more practical for use in the clinical setting.

Chapter Six is the RCT testing the brief CT intervention in hospitalized patients with HF. Forty-two patients with HF who reported mild-moderate depressive symptoms were randomized to a single, brief CT intervention focused on reducing negative thoughts with thought-stopping and affirmations, or to usual care control. Although there were trends toward faster improvement in depressive symptoms, HRQOL and negative thinking in the intervention group, these trends were not significant. While both groups improved in all three variables over time, the intervention group experienced longer cardiac event-free survival and fewer cardiovascular events compared to the control group. Combined, the results suggest that it is beneficial to administer a brief CT intervention in the acute care setting to patients with HF who experience mild-moderate symptoms of depression.

3. Impact of dissertation on the state of the science

There is little evidence supporting non-pharmacological interventions for the treatment of depressive symptoms in patients with HF. Although researchers and clinicians had established that depressive symptoms are a significant clinical problem in patients with HF, no interventions had been shown to improve the negative sequelae associated with depressive symptoms. The state of the science in treating depressive symptoms is such that the latest American College of Cardiology and American Heart

Association guidelines[7] did not include specific recommendations for screening or treatment of depressive symptoms in patients with HF.

I have advanced the state of the science in the treatment of depressive symptoms in patients with HF by: 1) identifying components that are necessary for the design of clinical trials that test CT interventions for depressive symptoms in patients with HF, 2) describing negative thinking as an important target for the treatment of depressive symptoms in patients with HF, 3) developing a brief CT intervention based on evidence from the qualitative study, 4) identifying a measure of negative thinking and providing evidence for its reliability and validity in patients with HF, 5) showing that it is both practical and beneficial to provide a CT intervention to patients with HF who are hospitalized, 6) and demonstrating that a nurse-delivered, brief CT intervention improves cardiac event-free survival in patients with HF.

First, Chapter Two[1] identified important design components that are needed in future clinical trials testing CT treatments for depressive symptoms in patients with HF. Subsequently, the RCT presented in Chapter Six incorporated these recommendations. The RCT included recommended design components such as: intentional sampling of 50% females, a scripted, replicable intervention, measurement of HRQOL and cardiac event-free survival, and following the CONSORT guidelines for reporting the results of the trial.

Next, in the qualitative study in Chapter Three,[2] I identified negative thinking as a key modifiable target for depressive symptoms in patients with HF. This finding is consistent with Beck's *cognitive model* of depression which posits that negative thinking influences the emotional, behavioral, and somatic symptoms of depression.[10] For patients in the qualitative study, the most commonly used strategy for managing depressive symptoms was the use of positive thinking. Positive thinking for these patients was defined as the redirection of thoughts away from the negative (i.e., thought-stopping) and focusing instead on affirmations, such as "I'm alive" and "My mind and soul are strong and healthy."

Based on the findings from Chapter Three, a brief CT intervention was developed for use in hospitalized patients with HF who have depressive symptoms. Strategies discussed by patients comprised four of the six steps of the brief, 30 minute intervention (Table 1) presented in Chapter Six. These strategies included: understanding the relationship between stressful situations, negative thinking, and symptoms of

depression; personal stories told by the patients about how negative thoughts influenced their feelings and behaviors; thought-stopping or refusing to "dwell" on negative thoughts; and the use of written and verbal affirmations. A colorful, 13 page flip chart with photos was developed to guide the script of the intervention, and color booklet versions were printed for patients to take home. Although a similar CT intervention using thought-stopping and affirmations has previously been tested in women at risk for depression,[11, 12] this intervention has never been tested in patients with HF. The intervention tested in this dissertation was particularly innovative because of its brevity (30 minutes), practicality, and potential to be implemented by nurses in the acute care setting.

To determine the effectiveness of CT to reduce negative thinking as a treatment for depressive symptoms in patients with HF, researchers need a valid and reliable measure of negative thinking in this population. In Chapters Four[3] and Five, I advanced the state of the science in depression and heart failure by identifying an instrument with the best potential for measuring negative thinking and providing strong evidence for its reliability and validity in patients with HF. It is recommended that the CCI be used to measure changes in negative thinking in studies testing CT interventions in patients with HF. However, the CCI may need to be shortened and undergo further psychometric testing to make it more practical for the clinical setting.

In the review of the literature presented in Chapter Two,[1] I determined that we do not know whether it is practical or beneficial to offer a CT intervention to patients with HF who are hospitalized. For example, the ENRICHD investigators[13] found that a CT intervention did not improve survival outcomes in patients with HF who had recently experienced an acute myocardial infarction. These investigators questioned whether it was appropriate to administer an intervention after an acute cardiac event. The findings from our study in Chapter Six suggest otherwise—we found that it is both practical and beneficial to deliver a brief CT intervention to hospitalized patients with HF.

Finally, this dissertation represents an important contribution to the literature because it was the first study to show that a nurse-delivered, non-pharmacological intervention for depressive symptoms in patients with HF is associated with longer cardiac event-free survival and fewer cardiovascular events compared to usual care.

4. Recommendations for nursing practice and research

Depressive symptoms are an important predictor of mortality and morbidity in patients with HF that require intervention. Results from the recent SADHART-HF study[8] suggest that nursing interventions may be more effective than antidepressant therapy for the treatment of depressive symptoms in patients with HF. If future clinical trials provide additional evidence that antidepressant therapy has limited benefit for patients with HF, non-pharmacological interventions could emerge as a preferred method of treatment for depressive symptoms in patients with HF. This however, will also require additional evidence to demonstrate the effectiveness of non-pharmacological interventions for depressive symptoms in patients with HF.

The CT intervention tested in this dissertation was designed to be brief (30 minutes) because of the time constraints of nurses working in the acute care setting. It is recommended that acute care nurses use this brief intervention to improve outcomes of patients with HF who report depressive symptoms.

Additional studies are planned to expand upon this pilot study. My next step will be evaluate the psychometric properties of a shortened version of the CCI using data collected from outpatients (N = 179) and inpatients with HF (N = 80) who enrolled in the studies described in Chapters Five and Six. After shortening the CCI, I plan to conduct a large, randomized controlled trial to test the long-term effects of the brief CT intervention provided to patients who are hospitalized with HF. This study will be adequately powered to detect differences in all primary and secondary outcomes. Furthermore, the study will include patients with higher levels of depressive symptoms and incorporate a follow-up period of at least one year.

Depressive symptoms have important clinical consequences in patients with HF, and nursing interventions may be an important treatment strategy of the future. This dissertation has fulfilled an important gap in the evidence base for depression treatment in patients with HF by demonstrating that a nurse-delivered, brief CT intervention may improve cardiac event-free survival in patients with HF. This brief CT intervention is replicable, practical, and can be delivered by acute care nurses to improve clinical outcomes in patients with HF.

Table 7.1: Summary of the six steps of the brief cognitive therapy intervention

Step 1: Depression and heart failure
Description of symptoms of depression Depression is common in people with heart failure Bad news: Depression is bad for the heart Good news: There are treatments for depression *Discussion question:* "Do any of these symptoms of depression sound familiar to you?"
Step 2: Thoughts, feelings, and behaviors
The connection between thoughts, feelings and behaviors Thoughts can be false and negative, and these thoughts contribute to painful feelings and negative behaviors of depression Two real-life patient stories are told that demonstrate the link between a stressful situation and negative thinking, feelings, and behaviors *Discussion question:* "Do these stories sound familiar to you? Do you see how the person's negative thinking led to painful feelings and negative behaviors?"
Step 3: Your story
Interactive component: Patient is asked to describe a recent stressful situation *Discussion questions:* "What kind of thoughts were going through your head? How did these thoughts make you feel? Did you notice any behaviors?"
Step 4: Thought-stopping
Notice you have a negative thought Snap your fingers or clap your hands and say, "Stop!" By stopping the negative thought you help take away the painful feelings that go with the thought *Interactive component:* Patient practices the stop technique with the interventionist
Step 5: Affirmations
An affirmation is a short, simple phrase that states how life is at its very best Use first person, present tense, and positive words; if you have a religious faith you may use it in your affirmation *Interactive component:* The patient comes up with 3-4 affirmations with the help of the interventionist The interventionist writes the affirmations on 2 brightly colored sticky notes: one for the hospital room and one for the patient to take home
Step 6: Homework
Practice the stop technique every time you hear a negative thought Hang the list of affirmations somewhere you will see it every day For every negative thought, think at least 2 positive thoughts

REFERENCES

1. Chapter One

1. Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, De Simone G, et al. Heart disease and stroke statistics--2010 update. A report from the American Heart Association. *Circulation*. 2009.
2. Roger VL, Weston SA, Redfield MM, Hellermann-Homan JP, Killian J, Yawn BP, Jacobsen SJ. Trends in heart failure incidence and survival in a community-based population. *JAMA*. 2004;292(3):344-350.
3. Merrill CT, Elixhauser A. Hospitalization in the United States, 2002. HCUP Fact Book No. 6; AHRQ Publication No. 05-0056. Rockville, MD: Agency for Healthcare Research and Quality 2005.
4. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol*. 2006;48(8):1527-1537.
5. Jiang W, Kuchibhatla M, Clary GL, Cuffe MS, Christopher EJ, Alexander JD, et al. Relationship between depressive symptoms and long-term mortality in patients with heart failure. *Am Heart J*. 2007;154(1):102-108.
6. Joynt KE, Whellan DJ, O'Connor C M. Why is depression bad for the failing heart? A review of the mechanistic relationship between depression and heart failure. *J Card Fail*. 2004;10(3):258-271.
7. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, Marshall J, Minshall S, Robinson S, Fisher ML, Potenza M, Sigler B, Baldwin C, Thomas SA. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol*. 2004;43(9):1542-1549.
8. Faller H, Stork S, Schuler M, Schowalter M, Steinbuchel T, Ertl G, Angermann CE. Depression and disease severity as predictors of health-related quality of life in patients with chronic heart failure--a structural equation modeling approach. *J Card Fail*. 2009;15(4):286-292 e282.
9. Stanek EJ, Oates MB, McGhan WF, Denofrio D, Loh E. Preferences for treatment outcomes in patients with heart failure: symptoms versus survival. J *Card Fail*. 2000;6(3):225-232.
10. Lane DA, Chong AY, Lip GY. Psychological interventions for depression in heart failure. *Cochrane Database Syst Rev*. 2005(1):CD003329.

11. Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, et al. Focused update incorporated into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration With the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol*. 2009;53(15):e1-e90.
12. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, Johnson BT. Initial severity and antidepressant benefits: a meta-analysis of data submitted to the Food and Drug Administration. *PLoS Med*. 2008;5(2):e45.
13. O'Connor C M. Safety and efficacy of Sertraline for depression in patients with congestive heart failure (SADHART-CHF). Paper presented at: Heart Failure Society of American Scientific Meeting; September 22, 2008; Toronto, ON.
14. Beck AT. *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row; 1967.
15. Beck AT. The current state of cognitive therapy: a 40-year retrospective. *Arch Gen Psychiatry*. 2005;62(9):953-959.
16. Freeman SM, Roy SC. Cognitive behavior therapy and the Roy Adaptation Model: Integrating CBT into nursing practice. In: Freeman SM, Freeman A, eds. *Cognitive behavior therapy in nursing practice*. New York: Springer Publishing Company; 2005:3-28.
17. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
18. Crandell CJ, Chambless DL. The validation of an inventory for measuring depressive thoughts: the Crandell Cognitions Inventory. *Behav Res Ther*. 1986;24(4):403-411.
19. Peden AR. Negative thoughts of women with depression. J*ournal of the American Psychiatric Nurses Association*. 2000;6(2):41-48.
20. Dekker RL. Cognitive behavioral therapy for depression in patients with heart failure: a critical review. *Nursing Clin North America*. 2008;43(1):155-170; viii.
21. Dekker RL, Peden AR, Lennie TA, Schooler MP, Moser DK. Living with depressive symptoms: patients with heart failure. *Am J Crit Care.* 2009;18(4):310-318.
22. Dekker, RL. Measurement of negative thinking in patients with heart failure: A critical review and analysis. *J Cardiovasc Nurs*. 2010. (In press).

2. Chapter Two

1. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* 2006;48(8):1527-1537.
2. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol.* 2004;43(9):1542-1549.
3. *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. 4th ed. Washington, D. C: American Psychiatric Association; 2000.
4. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, et al. Psychosocial disability during the long-term course of unipolar major depressive disorder. *Arch Gen Psychiatry.* 2000;57(4):375-380.
5. Stuart GW. Emotional responses and mood disorders. In: Stuart GW, Laraia MT, eds. *Principles and practice of psychiatric nursing.* 7th ed. St. Louis: Mosby; 2001:345-380.
6. Jiang W, Kuchibhatla M, Clary GL, Cuffe MS, Christopher EJ, Alexander JD, et al. Relationship between depressive symptoms and long-term mortality in patients with heart failure. *Am Heart J.* 2007;154(1):102-108.
7. Faris R, Purcell H, Henein MY, Coats AJ. Clinical depression is common and significantly associated with reduced survival in patients with non-ischaemic heart failure. *Eur J Heart Fail.* 2002;4(4):541-551.
8. Jiang W, Alexander J, Christopher E, Kuchibhatla M, Gaulden LH, Cuffe MS, et al. Relationship of depression to increased risk of mortality and rehospitalization in patients with congestive heart failure. *Arch Intern Med.* 2001;161(15):1849-1856.
9. Lane DA, Chong AY, Lip GY. Psychological interventions for depression in heart failure. *Cochrane Database Syst Rev.* 2005(1):CD003329.
10. Beck AT. The current state of cognitive therapy: A 40-year retrospective. *Arch Gen Psychiatry.* 2005;62(9):953-959.
11. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
12. Masoudi FA, Inzucchi SE. Diabetes mellitus and heart failure: epidemiology, mechanisms, and pharmacotherapy. *Am J Cardiol.* 2007;99(4A):113B-132B.
13. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from

hypertension to congestive heart failure. *JAMA.* 1996;275(20):1557-1562.

14. Berkman LF, Blumenthal J, Burg M, Carney RM, Catellier D, Cowan MJ, et al. Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *JAMA.* 2003;289(23):3106-3116.
15. Joynt KE, O'Connor CM. Lessons from SADHART, ENRICHD, and other trials. *Psychosom Med.* 2005;67 Suppl 1:S63-66.
16. Frasure-Smith N, Lesperance F. Depression--a cardiac risk factor in search of a treatment. *JAMA.* 2003;289(23):3171-3173.
17. Freedland KE, Skala JA, Carney RM, Rubin EH, Lustman PJ, Davila-Roman VG, et al. Treatment of depression after coronary artery bypass surgery: a randomized controlled trial. *Arch Gen Psychiatry.* 2009;66(4):387-396.
18. Burgess AW, Lerner DJ, D'Agostino RB, Vokonas PS, Hartman CR, Gaccione P. A randomized control trial of cardiac rehabilitation. *Soc Sci Med.* 1987;24(4):359-370.
19. Cowan MJ, Pike KC, Budzynski HK. Psychosocial nursing therapy following sudden cardiac arrest: Impact on two-year survival. *Nurs Res.* 2001;50(2):68-76.
20. Cowan MJ. Innovative approaches: a psychosocial therapy for sudden cardiac arrest survivors. In: Dunbar SB, Ellenbogen KA, Epstein AE, eds. *Sudden cardiac death: Past, present, and future*. Armonk, NY: Futura Publishing Company, Inc; 1997:371-386.
21. Black JL, Allison TG, Williams DE, Rummans TA, Gau GT. Effect of intervention for psychological distress on rehospitalization rates in cardiac rehabilitation patients. *Psychosomatics.* 1998;39(2):134-143.
22. Frizelle DJ, Lewin RJ, Kaye G, Hargreaves C, Hasney K, Beaumont N, et al. Cognitive-behavioural rehabilitation programme for patients with an implanted cardioverter defibrillator: a pilot study. *Br J Health Psychol.* 2004;9(Pt 3):381-392.
23. Kohn CS, Petrucci RJ, Baessler C, Soto DM, Movsowitz C. The effect of psychological intervention on patients' long-term adjustment to the ICD: a prospective study. *Pacing Clin Electrophysiol.* 2000;23(4 Pt 1):450-456.
24. Lewin RJ, Coulton S, Frizelle DJ, Kaye G, Cox H. A brief cognitive behavioural preimplantation and rehabilitation programme for patients receiving an implantable cardioverter-defibrillator improves physical health and reduces

psychological morbidity and unplanned readmissions. *Heart.* 2009;95(1):63-69.

25. Kostis JB, Rosen RC, Cosgrove NM, Shindler DM, Wilson AC. Nonpharmacologic therapy improves functional and emotional status in congestive heart failure. *Chest.* 1994;106(4):996-1001.
26. Lincoln NB, Flannaghan T. Cognitive behavioral psychotherapy for depression following stroke: A randomized controlled trial. *Stroke.* 2003;34(1):111-115.
27. Lustman PJ, Griffith LS, Freedland KE, Kissel SS, Clouse RE. Cognitive behavior therapy for depression in type 2 diabetes mellitus. A randomized, controlled trial. *Ann Intern Med.* 1998;129(8):613-621.
28. Snoek FJ, van der Ven NC, Twisk JW, Hogenelst MH, Tromp-Wever AM, van der Ploeg HM, et al. Cognitive behavioural therapy (CBT) compared with blood glucose awareness training (BGAT) in poorly controlled Type 1 diabetic patients: long-term effects on HbA moderated by depression. A randomized controlled trial. *Diabet Med.* 2008;25(11):1337-1342.
29. Henry JL, Wilson PH, Bruce DG, Chisholm DJ, Rawling PJ. Cognitive-behavioural stress management for patients with non-insulin dependent diabetes mellitus. *Psychology, Health, & Medicine.* 1997;2(2):109-118.
30. Ismail K, Thomas SM, Maissi E, Chalder T, Schmidt U, Bartlett J, et al. Motivational enhancement therapy with and without cognitive behavior therapy to treat type 1 diabetes: A randomized trial. *Ann Intern Med.* 2008;149(10):708-719.
31. Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, Khatri P, et al. Effects of exercise training on older patients with major depression. *Arch Intern Med.* 1999;159(19):2349-2356.
32. Dunn AL, Trivedi MH, Kampert JB, Clark CG, Chambliss HO. Exercise treatment for depression: Efficacy and dose response. *Am J Prev Med.* 2005;28(1):1-8.
33. Schneiderman N, Saab PG, Catellier DJ, Powell LH, DeBusk RF, Williams RB, et al. Psychosocial treatment within sex by ethnicity subgroups in the Enhancing Recovery in Coronary Heart Disease clinical trial. *Psychosom Med.* 2004;66(4):475-483.
34. Frasure-Smith N, Lesperance F, Prince RH, Verrier P, Garber RA, Juneau M, et al. Randomised trial of home-based psychosocial nursing intervention for patients recovering from myocardial infarction. *Lancet.* 1997;350(9076):473-479.
35. Davidson KW, Kupfer DJ, Bigger JT, Califf RM, Carney RM, Coyne JC, et al.

Assessment and treatment of depression in patients with cardiovascular disease: National Heart, Lung, and Blood Institute working group report. *Ann Behav Med.* 2006;32(2):121-126.

36. Begg C, Cho M, Eastwood S, Horton R, Moher D, Olkin I, Pitkin R, R et al. Improving the quality of reporting of randomized controlled trials. The CONSORT statement. *JAMA.* 1996;276(8):637-639.
37. Moher D, Schulz KF, Altman D. The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomized trials. *JAMA.* 2001;285(15):1987-1991.

3. Chapter Three

1. Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, De Simone G, et al. Heart disease and stroke statistics--2010 update. A report from the American Heart Association. *Circulation*. 2009.
2. Elixhauser A, Yu K, Steiner C, Bierman AS. Hospitalization in the United States, 1997. *HCUP Fact Book No. 1; AHRQ Publication No. 00-0031*. Rockville, MD: Agency for Healthcare Research and Quality; 2000.
3. Koenig HG. Depression in hospitalized older patients with congestive heart failure. *Gen Hosp Psychiatry.* 1998;20:29-43.
4. *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. 4th ed. Washington, D. C.: American Psychiatric Association; 2000.
5. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, et al. Psychosocial disability during the long-term course of unipolar major depressive disorder. *Arch Gen Psychiatry.* 2000;57:375-380.
6. Stuart GW. Emotional responses and mood disorders. In: Stuart GW, Laraia MT, eds. *Principles and practice of psychiatric nursing.* 7th ed. St. Louis: Mosby; 2001:345-380.
7. Lyness JM. Treatment of depressive conditions in later life: Real-world light for dark (or dim) tunnels. *JAMA.* 2004;291:1626-1628.
8. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol.* 2004;43:1542-1549.
9. Jiang W, Alexander J, Christopher E, Kuchibhatla M, Gaulden LH, Cuffe MS, et

al. Relationship of depression to increased risk of mortality and rehospitalization in patients with congestive heart failure. *Arch Intern Med.* 2001;161:1849-1856.

10. Sandelowski M. Whatever happened to qualitative description? *Res Nurs Health.* 2000;23:334-340.
11. Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *J Pers Assess.* 1996;67:588-597.
12. Miles MB, Huberman AM. *Qualitative data analysis: An expanded sourcebook*. 2nd ed. Thousand Oaks, CA: Sage Publishing, Inc.; 1994.
13. Creswell JW. *Qualitative inquiry and research design: Choosing among five traditions*. Thousand Oaks, CA: SAGE Publications, Inc; 1998.
14. Teasdale JD, Rezin V. The effects of reducing frequency of negative thoughts on the mood of depressed patients-tests of a cognitive model of depression. *Br J Soc Psychol.* 1978;17:65-74.
15. Beck AT. *Depression: Clinical, Experimental, and Theoretical Aspects*. New York: Harper & Row; 1967.
16. Kraaij V, Pruymboom E, Garnefski N. Cognitive coping and depressive symptoms in the elderly: a longitudinal study. *Aging Ment Health.* 2002;6:275-281.
17. Holzapfel N, Muller-Tasch T, Wild B, Junger J, Zugck C, Remppis A, et al. Depression profile in patients with and without chronic heart failure. *J Affect Disord.* 2008;105:53-62.
18. Donaldson C, Lam D. Rumination, mood and social problem-solving in major depression. *Psychol Med.* 2004;34:1309-1318.
19. Zambroski CH. Qualitative analysis of living with heart failure. *Heart Lung.* 2003;32:32-40.
20. Rhodes DL, Bowles CL. Heart failure and its impact on older women's lives. *J Adv Nurs.* 2002;39:441-449.
21. Riegel B, Carlson B. Facilitators and barriers to heart failure self-care. *Patient Educ Couns.* 2002;46:287-295.
22. Abramson J, Berger A, Krumholz HM, Vaccarino V. Depression and risk of heart failure among older persons with isolated systolic hypertension. *Arch Intern Med.* 2001;161:1725-1730.

23. Williams SA, Kasl SV, Heiat A, Abramson JL, Krumholz HM, Vaccarino V. Depression and risk of heart failure among the elderly: a prospective community-based study. *Psychosom Med.* 2002;64:6-12.
24. Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, Khatri P, et al. Effects of exercise training on older patients with major depression. *Arch Intern Med.* 1999;159:2349-2356.
25. Koukouvou G, Kouidi E, Iacovides A, Konstantinidou E, Kaprinis G, Deligiannis A. Quality of life, psychological and physiological changes following exercise training in patients with chronic heart failure. *J Rehabil Med.* 2004;36:36-41.
26. Gary RA, Sueta CA, Dougherty M, Rosenberg B, Cheek D, Preisser J, et al. Home-based exercise improves functional performance and quality of life in women with diastolic heart failure. *Heart Lung.* 2004;33:210-218.
27. Havranek EP, Spertus JA, Masoudi FA, Jones PG, Rumsfeld JS. Predictors of the onset of depressive symptoms in patients with heart failure. *J Am Coll Cardiol.* 2004;44:2333-2338.
28. Vollman MW, Lamontagne LL, Hepworth JT. Coping and depressive symptoms in adults living with heart failure. *J Cardiovasc Nurs.* 2007;22:125-130.
29. Bekelman DB, Dy SM, Becker DM, Wittstein IS, Hendricks DE, Yamashita TE, et al. Spiritual well-being and depression in patients with heart failure. *J Gen Intern Med.* 2007;22:470-477.
30. Koenig HG. Religion and remission of depression in medical inpatients with heart failure/pulmonary disease. *J Nerv Ment Dis.* 2007;195:389-395.
31. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
32. Roose SP, Miyazaki M. Pharmacologic treatment of depression in patients with heart disease. *Psychosom Med.* 2005;67:S54-57.
33. Angermann CE, Gelbrich G, Stork S, Fallgatter A, Deckert J, Faller, H, et al. Rationale and design of a randomised, controlled, multicenter trial investigating the effects of selective serotonin re-uptake inhibition on morbidity, mortality and mood in depressed heart failure patients (MOOD-HF). *Eur J Heart Fail.* 2007;9:1212-1222.

4. Chapter Four

1. Crandell CJ, Chambless DL. The validation of an inventory for measuring

depressive thoughts: The Crandell Cognitions Inventory. *Behav Res Ther.* 1986;24(4):403-411.

2. Beck AT. *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row; 1967.
3. Peden AR. Negative thoughts of women with depression. *J Am Psychiatr Nurses Assoc.* 2000;6(2):41-48.
4. Peden AR, Rayens MK, Hall LA, Grant E. Negative thinking and the mental health of low-income single mothers. *J Nurs Scholarsh.* 2004;36(4):337-344.
5. Peden AR, Hall LA, Rayens MK, Beebe L. Negative thinking mediates the effect of self-esteem on depressive symptoms in college women. *Nurs Res.* 2000;49(4):201-207.
6. Evans J, Heron J, Lewis G, Araya R, Wolke D. Negative self-schemas and the onset of depression in women: longitudinal study. *Br J Psychiatry.* 2005;186:302-307.
7. Peden AR, Hall LA, Rayens MK, Beebe LL. Reducing negative thinking and depressive symptoms in college women. *J Nurs Scholarsh.* 2000;32(2):145-151.
8. Beck AT. The Evolution of the cognitive model of depression and its neurobiological correlates. *Am J Psychiatry.* 2008;165(8):89-98.
9. Beevers CG, Wells TT, Miller IW. Predicting response to depression treatment: the role of negative cognition. *J Consult Clin Psychol.* 2007;75(3):422-431.
10. Rumsfeld JS, Havranek E, Masoudi FA, Peterson ED, Jones P, Tooley JF, et al. Depressive symptoms are the strongest predictors of short-term declines in health status in patients with heart failure. *J Am Coll Cardiol.* 2003;42(10):1811-1817.
11. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol.* 2004;43(9):1542-1549.
12. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure: A meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* 2006;48(8):1527-1537.
13. Dekker RL, Peden AR, Lennie TA, Schooler MP, Moser DK. Living with depressive symptoms in patients with heart failure. *Am J Crit Care.* 2009;18(4):310-318.

14. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
15. Guck TP, Elsasser GN, Kavan MG, Barone EJ. Depression and congestive heart failure. *Congest Heart Fail.* 2003;9(3):163-169.
16. Glass CR, Arnkoff DB. Questionnaire methods of cognitive self-statement assessment. *J Consult Clin Psychol.* 1997;65(6):911-927.
17. Hollon SD, Kendall P. Cognitive self-statements in depression: Development of an Automatic Thoughts Questionnaire. *Cognitive Therapy and Research.* 1980;4(4):383-395.
18. Clark DA. The validity of measures of cognition: A review of the literature. *Cognitive Therapy and Research.* 1988;12(1):1-20.
19. Peden AR, Rayens MK, Hall LA, Beebe LH. Preventing depression in high-risk college women: A report of an 18-month follow-up. *J Am Coll Health.* 2001;49(6):299-306.
20. Harrell TH, Ryon NB. Cognitive-behavioral assessment of depression: Clinical validation of the automatic thoughts questionnaire. *J Consult Clin Psychol.* 1983;51(5):721-725.
21. Wong DF, Chau P, Kwok A, Kwan J. Cognitive-behavioral treatment groups for people with chronic physical illness in Hong Kong: Reflections on a culturally attuned model. *Int J Group Psychother.* 2007;57(3):367-385.
22. Lamberton A, Oei TP. A test of the cognitive content specificity hypothesis in depression and anxiety. *J Behav Ther Exp Psychiatry.* 2008;39(1):23-31.
23. Beck AT, Brown G, Steer RA, Eidelson JI, Riskind JH. Differentiating anxiety and depression: A test of the cognitive content-specificity hypothesis. *J Abnorm Psychol.* 1987;96(3):179-183.
24. Steer RA, Beck AT, Clark DA, Beck JS. Psychometric properties of the Cognition Checklist with psychiatric outpatients and university students. *Psychol Assess.* 1994;6(1):67-70.
25. Clark DA, Cook A, Snow D. Depressive symptom differences in hospitalized, medically ill, depressed psychiatric inpatients and nonmedical controls. *J Abnorm Psychol.* 1998;107(1):38-48.
26. Martens EJ, Denollet J, Pedersen SS, Scherders M, Griez E, Widdershoven J, et al. Relative lack of depressive cognitions in post-myocardial infarction depression. *J Affect Disord.* 2006;94(1-3):231-237.

27. Lloyd-Jones, D, Adams, RJ, Brown, TM, Carnethon M, Dai S, De Simone G, et al. Heart disease and stroke statistics--2010 update: A report from the American Heart Association. *Circulation.* 2009; in press.
28. Merrill CT, Elixhauser A. Hospitalization in the United States, 2002. *HCUP Fact Book No. 6; AHRQ Publication No. 05-0056*. Rockville, MD: Agency for Healthcare Research and Quality; 2005.
29. Dozois DJ, Covin R, Brinker JK. Normative data on cognitive measures of depression. *J Consult Clin Psychol.* 2003;71(1):71-80.
30. Peden AR, Rayens MK, Hall LA, Grant E. Testing an intervention to reduce negative thinking, depressive symptoms, and chronic stressors in low-income single mothers. *J Nurs Scholarsh.* 2005;37(3):268-274.
31. Dobson KS, Breiter HJ. Cognitive assessment of depression: reliability and validity of three measures. *J Abnorm Psychol.* 1983;92(1):107-109

5. Chapter Five

1. Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, De Simone G, et al. Heart disease and stroke statistics--2010 update. A report from the American Heart Association. *Circulation*. 2009.
2. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol.* 2004;43(9):1542-1549.
3. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* 2006;48(8):1527-1537.
4. Beck AT. *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row; 1967.
5. Crandell CJ, Chambless DL. The validation of an inventory for measuring depressive thoughts: the Crandell Cognitions Inventory. *Behav Res Ther.* 1986;24(4):403-411.
6. Peden AR, Rayens MK, Hall LA, Grant E. Negative thinking and the mental health of low-income single mothers. *J Nurs Scholarsh.* 2004;36(4):337-344.
7. Peden AR, Hall LA, Rayens MK, Beebe L. Negative thinking mediates the effect of self-esteem on depressive symptoms in college women. *Nurs Res.*

2000;49(4):201-207.

8. Beevers CG, Wells TT, Miller IW. Predicting response to depression treatment: the role of negative cognition. *J Consult Clin Psychol.* 2007;75(3):422-431.
9. Dekker RL, Peden AR, Lennie TA, Schooler MP, Moser DK. Living with depressive symptoms: patients with heart failure. *Am J Crit Care.* 2009;18(4):310-318.
10. Beck AT. The current state of cognitive therapy: A 40-year retrospective. *Arch Gen Psychiatry.* 2005;62(9):953-959.
11. Beck AT, Brown G, Steer RA, Eidelson JI, Riskind JH. Differentiating anxiety and depression: A test of the cognitive content-specificity hypothesis. *Journal of abnormal psychology.* 1987;96(3):179-183.
12. Guck TP, Elsasser GN, Kavan MG, Barone EJ. Depression and congestive heart failure. *Congest Heart Fail.* 2003;9(3):163-169.
13. Peden AR, Rayens MK, Hall LA, Beebe LH. Preventing depression in high-risk college women: a report of an 18-month follow-up. *J Am Coll Health.* 2001;49(6):299-306.
14. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, Endicott J, Coryell W, Kunovac JL, Mueller TI, Rice JP, Keller MB. Psychosocial disability during the long-term course of unipolar major depressive disorder. *Arch Gen Psychiatry.* 2000;57(4):375-380.
15. Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *J Pers Assess.* 1996;67(3):588-597.
16. Sherwood A, Blumenthal JA, Trivedi R, Johnson KS, O'Connor CM, Adams KF, Jr., et al. Relationship of depression to death or hospitalization in patients with heart failure. *Arch Intern Med.* 2007;167(4):367-373.
17. De Jong MJ, Hall LA. Measurement of anxiety for patients with cardiac disease: A critical review and analysis. *J Cardiovasc Nurs.* 2006;21(5):412-419.
18. Derogatis LP. *Brief Symptom Inventory: Administration, scoring, and procedure manual*. Minneapolis: National Compute Systems, Inc. ; 1993.
19. Derogatis LR, Melisaratos N. The Brief Symptom Inventory: An introductory report. *Psychol Med.* 1983;13(3):595-605.
20. Streiner DL, Norman GR. *Health measurement scales: A practical guide to their*

development and use. 3rd ed. Oxford: Oxford University Press; 2003.

21. Beck AT. Cognitive models of depression. *J of Cog Psychotherapy.* 1987;1:5-37.
22. Jiang W, Kuchibhatla M, Cuffe MS, Christopher EJ, Alexander JD, Clary GL, et al. Prognostic value of anxiety and depression in patients with chronic heart failure. *Circulation.* 2004;110(22):3452-3456.

6. Chapter Six

1. Koenig HG. Depression in hospitalized older patients with congestive heart failure. *Gen Hosp Psychiatry.* Jan 1998;20(1):29-43.
2. Jiang W, Kuchibhatla M, Clary GL, Cuffe MS, Christopher EJ, Alexander JD, et al. Relationship between depressive symptoms and long-term mortality in patients with heart failure. *Am Heart J.* Jul 2007;154(1):102-108.
3. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* Oct 17 2006;48(8):1527-1537.
4. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. *J Am Coll Cardiol.* May 5 2004;43(9):1542-1549.
5. Faris R, Purcell H, Henein MY, Coats AJ. Clinical depression is common and significantly associated with reduced survival in patients with non-ischaemic heart failure. *Eur J Heart Fail.* Aug 2002;4(4):541-551.
6. Jiang W, Alexander J, Christopher E, Kuchibhatla M, Gaulden LH, Cuffe MS, et al. Relationship of depression to increased risk of mortality and rehospitalization in patients with congestive heart failure. *Arch Intern Med.* Aug 13-27 2001;161(15):1849-1856.
7. O'Connor CM, Abraham WT, Albert NM, Clare R, Gattis Stough W, Gheorghiade M, et al. Predictors of mortality after discharge in patients hospitalized with heart failure: An analysis from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF). *Am Heart J.* Oct 2008;156(4):662-673.
8. Beck AT. *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row; 1967.
9. Crandell CJ, Chambless DL. The validation of an inventory for measuring

depressive thoughts: the Crandell Cognitions Inventory. *Behav Res Ther.* 1986;24(4):403-411.

10. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
11. Peden AR, Rayens MK, Hall LA, Grant E. Negative thinking and the mental health of low-income single mothers. *J Nurs Scholarsh.* 2004;36(4):337-344.
12. Peden AR, Hall LA, Rayens MK, Beebe L. Negative thinking mediates the effect of self-esteem on depressive symptoms in college women. *Nurs Res.* Jul-Aug 2000;49(4):201-207.
13. Beck AT. The evolution of the Cognitive Model of Depression and its neurobiological correlates. *Am J Psychiatry.* Jul 15 2008.
14. Beevers CG, Wells TT, Miller IW. Predicting response to depression treatment: the role of negative cognition. *J Consult Clin Psychol.* Jun 2007;75(3):422-431.
15. Beck AT. The current state of cognitive therapy: A 40-year retrospective. *Arch Gen Psychiatry.* Sep 2005;62(9):953-959.
16. Peden AR, Hall LA, Rayens MK, Beebe LL. Reducing negative thinking and depressive symptoms in college women. *J Nurs Scholarsh.* 2000;32(2):145-151.
17. Peden AR, Rayens MK, Hall LA, Grant E. Testing an intervention to reduce negative thinking, depressive symptoms, and chronic stressors in low-income single mothers. *J Nurs Scholarsh.* 2005;37(3):268-274.
18. Moher D, Schulz KF, Altman D. The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomized trials. *Jama.* Apr 18 2001;285(15):1987-1991.
19. *nQuery Advisor: Statistical Solutions* [computer program]. Version 6.02. Boston: Sangus; 2005.
20. Thompson DR. A randomized controlled trial of in-hospital nursing support for first time myocardial infarction patients and their partners: Effects on anxiety and depression. *J Adv Nurs.* Apr 1989;14(4):291-297.
21. Haahr M. Random numbers. In: Salkind NJ, ed. *Encyclopedia of Measurement and Statistics.* Vol 3. Thousand Oaks, CA: Sage; 2007:815-816.
22. Haahr M. Random.org: True random number service. Available at: www.random.org. Accessed January 3, 2009, 2009.
23. Dekker RL, Peden AR, Lennie TA, Schooler MP, Moser DK. Living with depressive symptoms: patients with heart failure. *Am J Crit Care.* Jul

2009;18(4):310-318.

24. Hunt SA. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). *J Am Coll Cardiol.* Sep 20 2005;46(6):e1-82.
25. Specifications Manual for National Implementation of Hospital Quality Measures. *Joint Commission on Accreditation of Healthcare Organizations*. Available at: http://www.jointcommission.org/performancemeasurement/performancemeasurement/current+nhqm+manual.htm. Accessed January 21, 2009.
26. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, et al. Psychosocial disability during the long-term course of unipolar major depressive disorder. *Arch Gen Psychiatry.* Apr 2000;57(4):375-380.
27. Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *J Pers Assess.* Dec 1996;67(3):588-597.
28. Grothe KB, Dutton GR, Jones GN, Bodenlos J, Ancona M, Brantley PJ. Validation of the Beck Depression Inventory-II in a low-income African American sample of medical outpatients. *Psychol Assess.* Mar 2005;17(1):110-114.
29. Whisman MA, Perez JE, Ramel W. Factor structure of the Beck Depression Inventory-Second Edition (BDI-II) in a student sample. *J Clin Psychol.* Apr 2000;56(4):545-551.
30. Heo S, Moser DK, Riegel B, Hall LA, Christman N. Testing the psychometric properties of the Minnesota Living with Heart Failure questionnaire. *Nurs Res.* Jul-Aug 2005;54(4):265-272.
31. Mills RM, Jr., Haught WH. Evaluation of heart failure patients: objective parameters to assess functional capacity. *Clin Cardiol.* Jun 1996;19(6):455-460.
32. Koenig HG. Depression outcome in inpatients with congestive heart failure. *Arch Intern Med.* May 8 2006;166(9):991-996.
33. Riegel B, Moser DK, Glaser D, Carlson B, Deaton C, Armola R, et al. The Minnesota Living With Heart Failure Questionnaire: sensitivity to differences and responsiveness to intervention intensity in a clinical population. *Nurs Res.* Jul-Aug 2002;51(4):209-218.

34. Berkman LF, Blumenthal J, Burg M, Carney RM, Catellier D, Cowan MJ, et al. Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *JAMA.* Jun 18 2003;289(23):3106-3116.

7. Chapter Seven

1. Dekker RL. Cognitive behavioral therapy for depression in patients with heart failure: A critical review. *Nurs Clin North America.* 2008;43(1):155-170.
2. Dekker RL, Peden AR, Lennie TA, Schooler MP, Moser DK. Living with depressive symptoms: Patients with heart failure. *Am J Crit Care.* 2009;18(4):310-318.
3. Dekker, RL. Measurement of negative thinking in patients with heart failure: A critical review and analysis. *J Cardiovasc Nurs*. 2010. (In press).
4. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure: A meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol.* 2006;48(8):1527-1537.
5. Faller H, Stork S, Schuler M, Schowalter M, Steinbuchel T, Ertl G, et al. Depression and disease severity as predictors of health-related quality of life in patients with chronic heart failure--a structural equation modeling approach. *J Card Fail.* 2009;15(4):286-292 e282.
6. Koenig HG. Recognition of depression in medical patients with heart failure. *Psychosomatics.* 2007;48(4):338-347.
7. Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, et al. 2009 Focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines developed in collaboration with the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol.* 2009;53(15):e1-e90.
8. O'Connor C M, Jiang W, Silva SG, Kuchibhatla M, Cuffe MS, Callwood DD, Zakhary B, et al. Safety and efficacy of sertraline plus nurse facilitated supportive intervention versus placebo plus nurse facilitated supportive intervention for depression in patients with CHF. *J of Card Fail.* 2008;14(9):797.
9. Freeman SM, Roy SC. Cognitive behavior therapy and the Roy Adaptation

Model: Integrating CBT into nursing practice. In: Freeman SM, Freeman A, eds. *Cognitive behavior therapy in nursing practice*. New York: Springer Publishing Company; 2005:3-28.

10. Beck JS. *Cognitive therapy: Basics and beyond*. New York: Guilford Press; 1995.
11. Peden AR, Rayens MK, Hall LA, Grant E. Testing an intervention to reduce negative thinking, depressive symptoms, and chronic stressors in low-income single mothers. *J Nurs Scholarsh.* 2005;37(3):268-274.
12. Peden AR, Hall LA, Rayens MK, Beebe LL. Reducing negative thinking and depressive symptoms in college women. *J Nurs Scholarsh.* 2000;32(2):145-151.
13. Berkman LF, Blumenthal J, Burg M, Carney RM, Catellier D, Cowan MJ, et al. Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *Jama.* 2003;289(23):3106-3116.

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Education

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Professional Experience

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2006 - 2006	Staff Nurse, St. Joseph Hospital, Oncology, Lexington, KY
2005 - 2005	Staff Nurse, Cardinal Hill Rehabilitation Hospital, Stroke, Lexington, KY
2002 - 2004	Staff Nurse, Spectrum Health Blodgett Hospital, Telemetry, Grand Rapids, MI

Honors and Awards

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2009 Academic Year Fellowship
University of Kentucky Graduate School

2008 International Scholar Award
Philanthropic Educational Organization

2008 Finalist in Research Presentation Competition (2nd place)
American Association of Heart Failure Nurses

2008 Best Doctoral Poster
Student Scholarship Showcase, University of Kentucky, College of Nursing

2007 Finalist in Research Presentation Competition (3rd place)
American Association of Heart Failure Nurses

2006 Pre-doctoral Fellowship
RICH Heart Program, University of Kentucky, College of Nursing

2006 Academic Year Fellowship
University of Kentucky Graduate School

2006 Presidential Award for Outstanding Master's Student
University of Kentucky, College of Nursing

2006 Academic Scholarship
Sigma Theta Tau International

2002 Award for Excellence in Student Nurse Performance
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Publications

Journal Articles

1. **Dekker, R. L.** (In press). Measurement of negative thinking in patients with heart failure: A critical review and analysis. *Journal of Cardiovascular Nursing*.
2. **Dekker, R. L.**, Peden, A. R., Lennie, T. A., Schooler, M. P., & Moser, D. K. (2009). Living with depressive symptoms: Patients with heart failure. *American Journal of Critical Care*, *18,* 310-318.
3. Chung, M. L., Lennie, T. A., Riegel, B., Wu, J. R., **Dekker, R. L.**, & Moser, D. K. (2009). Marital status is an independent predictor of event-free survival of patients with heart failure. *American Journal of Critical Care, 18*, 562-570.
4. **Dekker, R. L.** (2008). Cognitive behavioral therapy for depression in patients with heart failure: A critical review. *Nursing Clinics of North America, 43*,155-70.
5. **Dekker, R. L.** (2008). Human papillomavirus vaccine legislation in Kentucky: A policy analysis. *Policy, Politics, and Nursing Practice, 9,* 40-49.

Published Abstracts

1. Wu, J., Lennie, T. A., Chung, M. L., Frazier, S. K., **Dekker, R. L.**, Biddle, M. J., & Moser, D. K. (2009). Medication adherence mediates the relationship between marital status and event-free survival in patients with heart failure. *Circulation*, *120* (18 Suppl.), S516.
2. Moser, D. K., Wu, J., Chung, M. L., Biddle, M. J., **Dekker, R. L.**, & Lennie, T. A. (2009). What happens when heart failure patients don't know what they don't know. *Circulation*, *120* (18 Suppl.), S449.
3. **Dekker, R. L.**, Lennie, T. A., Peden, A. R., Chung, M. L., Wu, J, & Moser, D. K. (2008). Negative thinking: A modifiable target for the treatment of depressive symptoms in patients with heart failure. *Circulation*, *118*: S976.
4. **Dekker, R. L.,** Peden, A. R., Lennie, T. A., Schooler, M. P., & Moser, D. K. (2008). Living with depressive symptoms in patients with heart failure. *Progress in Cardiovascular Nursing, 23*, 103.
5. **Dekker, R. L.**, Moser, D. K., Chung, M. L., Heo, S., & Lennie, T. A. (2007). The impact of depressive symptoms on survival in patients with heart failure is altered by body mass index. *Circulation, 116*, II_533.
6. **Dekker, R. L.**, Lennie, T. A., Chung, M. L., & Moser, D. K. (2007). Depressive symptoms do not predict physical activity levels in patients with heart failure. *Progress in Cardiovascular Nursing, 22*, 113.
7. Chung, M. L., Lennie, T. A., Riegel, B., **Dekker, R. L.**, Wu, J., Heo, S., & Moser, D. K. (2007). Presence of spouses impacts positively survival in patients with heart failure regardless of presence of depressive symptoms. *Circulation, 116*, II_633.
8. Lennie, T. A., Chung, M. L., Heo, S., **Dekker, R. L.**, & Moser, D. K. (2007).Three gram sodium intake is associated with better event-free survival only in patients with advanced heart failure. *Circulation, 116*, II_486 - II_487.