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Physiologic Effect of Relaxation Therapies on Autonomic Tone Early After Acute Coronary Syndromes

Rachel Summer Claire Friedman
Yale University

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Rachel Summer Claire Friedman

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ABSTRACT

PHYSIOLOGIC EFFECT OF RELAXATION THERAPIES ON AUTONOMIC TONE EARLY AFTER ACUTE CORONARY SYNDROMES.

Rachel S.C. Friedman, Matthew Burg, Rachel J. Lampert. Section of Cardiology, Department of Internal Medicine, Yale University, School of Medicine, New Haven, CT.

Post-MI patients are at increased risk of arrhythmic sudden death. Stress and sympathetic activation are known to influence arrhythmogenesis. While relaxation therapies improve psychological well-being in multiple medical illnesses, whether these therapies can positively influence sympathovagal balance in the post-MI population is unknown. We explored the physiologic effects of Reiki, a light-touch relaxation therapy, and music on post- acute coronary syndrome (ACS) inpatients, using heart rate variability (HRV) to assess changes in cardiac autonomic function during treatment. Forty-eight patients with ACS within the last 72 hours were randomized to received a single 20-minute session of either Reiki, classical music, or a control "minimal distraction environment". All subjects underwent ambulatory ECG Holter monitoring. Emotional state was assessed by Likert scale. HRV was analyzed by spectral analysis via fast Fourier transformation during the baseline, intervention, and post-intervention periods and high-frequency power (log-normalized) compared via ANOVA with repeated measures. Adequate Holters were recorded in 12 control, 13 music, and 12 Reiki patients. High frequency (HF) component of HRV, an index of parasympathetic tone, increased significantly during Reiki (0.58±0.16) but not during music (-0.1±0.16) or control (0.06±0.16). RR interval increased significantly with Reiki and control, but not with music. Reiki significantly reduced reported anxiety and increased sense of relaxation compared to control ($p=0.04$), whereas music did not. In conclusion, post-MI recipients of light-touch from nurses trained in Reiki experienced increased vagal activity and decreased anxiety. Whether longer-term use of this therapy can improve outcomes requires further study.

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Glossary of abbreviated terminology

ACS	Acute coronary syndrome
ANS	Autonomic nervous system
BP	Blood pressure
CAD	Coronary artery disease
CAM	Complementary and alternative medicine
ECG	Electrocardiogram
HF	High frequency component of the power spectrum of the ECG
HR	Heart rate
HRV	Heart rate variability
MDE	Minimal distraction environment
MI	Myocardial infarction
NCCAM	National Center for Complementary and Alternative Medicine
SCD	Sudden cardiac death

BACKGROUND & SIGNIFICANCE

Sudden Cardiac Death Following Myocardial Infarction

Cardiovascular disease is the leading cause of death among adults in the United States. Sudden cardiac death (SCD) is estimated to account for approximately half of these deaths, most often due to coronary heart disease (CHD) and the consequences of CHD-induced myocardial infarction.[1] Each year, approximately 1.1 million Americans suffer a new or recurrent myocardial infarction (MI). Despite major advances in treatment, in particular aspirin, thrombolytic therapy and primary percutaneous coronary interventions (PCI),[2] the overall adjusted 28 day mortality for all patients who were hospitalized for definite or probable MI is still approximately 5%.[3] All patients who have had an acute MI are at increased risk for sudden cardiac death (SCD), and approximately 30% of post-MI patients will die of SCD, most often due to ventricular arrhythmias, with the majority occurring within one year post-infarct.[4]

Role of Autonomic Nervous System in Fatal Arrhythmias

The autonomic nervous system has an important role in the triggering or sustaining of malignant ventricular arrhythmias leading to SCD.[5] Fluctuations in the balance of sympathetic and parasympathetic nerve activity on the heart alter the electrophysiologic properties of the myocardium, either promoting or preventing arrhythmogenesis. Sympathetic activity, including increased nerve traffic and the elevated plasma catecholamines that result, has been shown to decrease the threshold for ventricular fibrillation[6] This mechanism underlies some of the increased risk of arrhythmias following MI; in the setting of cardiac ischemia, norepinephrine is reflexively released

from sympathetic nerve terminals to maintain blood pressure (BP) in response to decreased cardiac output, leading to increased sympathetic tone.[7] Furthermore, interruption of nerve fibers along the infarct leads to further sympathetic and parasympathetic dysfunction.[1]

Emotional Triggers of Sudden Cardiac Death

The autonomic nervous system is known to provide the physiological substrate for emotions. In patients prone to SCD due to existing structural abnormalities of the heart, such as a prior MI, increasing evidence suggests that emotional triggers of heightened sympathetic elevation, such as extreme anger, sudden fear, and significant acute/chronic stress, may trigger SCD. Epidemiological studies have measured sharp increases in the number of sudden deaths from cardiac causes during earthquakes,[8] wartime missile attacks,[9, 10] and sporting events.[11] A recent study examining incidence of cardiovascular events in the greater Munich area during the 2006 World Cup found an over twofold increase in events during days when the German soccer team played (2.66, 95% CI 2.33 to 3.04; $p<0.001$) compared to a control time period.[12] Moreover, on the day of the 1996 European football final that Holland narrowly lost to France after penalty kicks, Dutch men had an increased relative risk of MI or stroke of 1.5 (95% CI 1.1 to 2.1), whereas there was no increased risk among Dutch women, French men, or French women.[11, 13] In addition to the existing epidemiological evidence, several prospective studies have further confirmed the observed association between strong emotion and malignant arrhythmias. One prospective study asked patients with implantable cardioverter-defibrillator (ICD) to fill out diaries documenting presence of certain emotions

throughout the day. Interrogation of the ICD record at the time of ICD-treated arrhythmias and during control periods revealed that anger and anxiety were significantly associated with initiation of ventricular tachycardia and fibrillation.[14, 15]

Pathophysiology of Stress and Anxiety

Additionally, increasing evidence suggests that emotional stress, associated with increased sympathetic activity, appear to cause pathophysiologic changes that may explain these demonstrated clinical changes. Lampert and colleagues looked at how acute stress in the laboratory setting affected T-wave alternans, a measure of repolarization abnormalities associated with vulnterability to SCD.[16] Anger in subjects correlated with increased T-wave alternans and was also associated with higher serum catecholamine levels.

Furthermore, both animal and human studies have demonstrated that increases in vagal activity are associated with reduced susceptibility to ventricular arrhythmias.[17] For example, Vanoli and colleagues demonstrated in dogs that applying direct vagal stimulation in the setting of acute ischemia prevented ventricular fibrillation.[18]

Physiologic Effects of Relaxation

Although the detrimental effects of emotional stress on cardiovascular disease are now well recognized, the beneficial effects of therapeutic interventions aimed at reducing stress have not been well studied. In the 1970s, Harvard cardiologist Herbert Benson studied the physiological changes occurring in subjects practicing meditation, Benson

found that he could distill the tenets of the meditation into a simple set of instructions to elicit a so-called “relaxation response”,[19] which he described as the balancing counterpart to the “fight or flight response” associated with sympathetic nervous system activation. This “relaxation response” was found to be associated with decreased blood pressure both acutely and long-term,[20] as well as decreased frequency of premature ventricular complexes in patients with existing cardiac disease.[21]

Use of Complementary Therapies for Healing and Relaxation

Benson pioneered the study of non-Western healing practices using scientific method and medical technology. Many complementary and alternative medicine (CAM) therapies, defined by the National Institutes of Health as “a group of diverse medical and health care systems, practices, and products that are not presently considered to be part of conventional medicine,"[22] may have significant therapeutic potential.

Complementary therapies are often touted for their potential relaxing and healing effects. The goals of CAM treatment are primarily to return the system to balance, strengthen immune functioning and support continued well being. The last 20 years have witnessed a remarkable increase in the use of CAM by patients,[23] and in the last 5-10 years, increasing utilization by physicians,[24] clinics,[25, 26] and hospitals.[27] The National Institutes of Health created the National Center for Complementary and Alternative Medicine (NCCAM) specifically to study CAM, yet compared to available pharmacologic treatments, there remains a paucity of rigorous clinical research evaluating the safety, efficacy, and physiologic effects of these therapies. However, because many of these

modalities stem from alternative systems of medical theory, such as Traditional Chinese Medicine or the notion of energy medicine, the explanations or mechanisms proposed for their therapeutic effect are difficult to translate into biomedical concepts that can be understood and evaluated by the scientific community. This general lack of understanding of specific mechanisms has therefore presented a challenge to the development of appropriately rigorous studies.[28, 29] One approach to understanding the mechanisms of complementary therapies may involve analysis of their effect on the autonomic nervous system.

Heart Rate Variability

Analysis of heart rate variability (HRV) from ambulatory ECG monitoring has become a powerful non-invasive tool to assess cardiac autonomic function through measuring the effects of the ANS on the sinus node control of the heart rate.[30-32] HRV is a measure of variations in the R-R intervals of the heart rate that represent the composite effect of rhythmic fluctuations in factors that influence the heart rate. HRV is usually calculated by analyzing the time series of beat-to-beat intervals from ECG (see Figure 1).

Although there are multiple physiologic feedback mechanisms that as a whole determine HR, the autonomic nervous system is most prominent among all factors. When both sympathetic and parasympathetic cardiac inputs are blocked pharmacologically or surgically, intrinsic HR is higher than normal, demonstrating that under normal conditions, parasympathetic influence is greater than that sympathetic influence.[33]

Of particular interest is a fast, high frequency (HF) component of the HRV that affects beat-to-beat variation and is related to respiration; it is this component that leads to sinus

arrhythmia, the phenomenon wherein the heart rate increases with each inhalation and slows with each exhalation. The size of the variations at this high frequency represents the amount of "HF power" (see Figure 2). As HF power is eliminated with parasympathetic blockade,[34] an increase in HF power has been interpreted to indicate increased vagal, or parasympathetic, activity.[35] A loss of HF power would likewise indicate vagal withdrawal or suppression. Physiologically, vagal stimulation on the heart is reflected by instantaneous change in the heart rate, leading to variability at a high frequency (HF); conversely, sympathetic stimulation is slower, requiring more metabolic steps, and therefore has no substantial contribution to the HF power. HF power has been studied in a multitude of physiological states, and high HF power is associated with health, youth, and other positive physical and behavioral aspects of human functioning.[36]

Loss of High Frequency HRV

Low vagal influence on the HR control, as measured by reduced HF HRV, has been associated with a variety of pathological states. Heart rate variability naturally diminishes with aging.[37] Reduced HF HRV is also observed in diabetes[38] and a variety of cardiovascular disease states, including sudden cardiac death[39, 40] and CHF.[41] The absence of variability is a highly significant risk factor for adverse outcomes following MI, including all cause mortality,[42] arrhythmic,[43] and sudden death.[44] Initial studies of HRV in the post-MI setting were motivated by evidence linking ANS abnormalities to development of ventricular tachyarrhythmias as a primary trigger for SCD during ischemic events.[39]

Heart Rate Variability Post-Myocardial Infarction

Over the last two decades HRV has been shown to have significant prognostic value following acute myocardial infarction. Lombardi et al. studied post-MI sympathovagal interactions by analyzing spectral components of HR in 70 patients two weeks after MI and 26 age-matched controls.[32] They found a post-infarction shift in autonomic balance toward sympathetic predominance, as demonstrated by smaller HF peaks (representing vagal activity) and larger LF peaks (representing sympathetic activity) in the post-infarction patients compared to controls.

Kleiger et al. published the first large study documenting the ability of HRV measures to predict mortality following MI.[42] HRV data were collected in 808 patients an average of 11 days after AMI and then correlated with mortality outcomes an average of 31 months later. This study found a relative risk of mortality 5.3 times higher in the patient group with lowest HRV compared to the patient group with highest HRV; this correlation remained significant even after adjusting for possible confounders. In the largest recent study of the predictive value of HRV post-MI, the ATRAMI trial, 1284 patients were followed for almost 2 years after MI; patients with the lowest values of HRV within 28 days post-MI were found to carry a threefold multivariate risk of cardiac mortality (95% CI 1.42 – 7.36), and this relative risk jumped to 6.7 (95% CI 3.1 – 14.6) in the subgroup of low HRV subjects who also had LVEF below 35%.[45, 46] Thus, HRV has been shown repeatedly to be one of the strongest independent predictors of death in the post-MI period.

High Frequency HRV Recovery During the Post-MI Period

During the first three months following MI, the majority of patients will experience marked recovery of HF HRV, and **a**utonomic balance tends to return to normal by approximately six months after MI.[47] The post-MI recovery of the high frequency component of HRV represents recovery of parasympathetic tone and correlates best with improved outcome.[48] Beta-adrenergic blockers, one of the best pharmacologic interventions associated with improved outcomes after acute MI, enhance the recovery of HF HRV during the post-MI state; analyzing HRV data from the Beta-blocker Heart Attack Trial (BHAT), Lampert and colleagues found that 6 weeks after acute MI, HF HRV increased more in propranalol-treated patients (4.28 ± 0.1 to 5.17 ± 0.09 ms^2) than in placebo-treated patients (4.26 ± 0.09 to 4.77 ± 0.1 ms^2).[49] Thus, pharmacologic treatments that improve recovery of parasympathetic tone appear to be associated with improved outcomes. It is not, however, currently known whether other non-pharmacologic interventions may further improve recovery of parasympathetic tone, thus providing additional therapeutic value.

Effects of Complementary Therapies on HRV

Based on the premise that HRV can be used as a measure for parasympathetic activity, several investigators have begun to look at physiologic effects of complementary therapies by measuring changes in HRV. Biofeedback training, in which subjects are trained to voluntarily modify physiologic variables such as respiratory rate or heart rate, has been shown in several small studies to successfully improve sinus arrhythmia, a marker of high HRV.[50, 51] Del Pozo and colleagues conducted a randomized controlled

trial of a six-session cardiorespiratory biofeedback training on 63 patients with established coronary heart disease (CHD); they found a significant increase in HRV compared to control groups during the treatment and three months after treatment.[51] Nolan et al. further randomized 46 patients with known CHD to receive either five 1.5-hour sessions of HRV biofeedback or an active control;[52] both groups experienced improvement in psychological adjustment scores, but only in the HRV biofeedback group were these improvements associated with increased HF HRV. Concentration meditation has also been demonstrated to elicit changes in HRV.[53] However, the majority of these previous studies of non-pharmacologic interventions to improve HRV have been done on healthy volunteers, limiting the extent to which efficacy can be extrapolated to therapeutic potential. Furthermore, both biofeedback and meditation require hours of instruction and specific equipment or instructors, limiting the ease of incorporating into conventional care settings.

Reiki: Theory, Practice, Evidence

Reiki, a complementary therapy originating in Japan, is a healing practice that can be easily learned and practiced. Although there are three degrees of training, the highest of which is a teacher, or Master, level of training, Reiki treatment is typically provided at the First degree level, in which healing is facilitated by light touch.[28] Although Reiki is historically a self-care practice, it is increasingly learned by health care providers, especially nurses, and used as a complementary therapy to support conventional medical care.[28]

Reiki treatment is given by placing hands lightly on the body in a series of non-invasive positions on the head and torso. The hands are held comfortably in place while offering Reiki; there is no manipulation of skin or muscles as in massage[28]. Practitioners believe that Reiki treatment gently influences the body toward balance, enabling the body's self-regulating mechanisms to recalibrate, thereby strengthening the body's ability to heal itself.[54] Although the subjective experience of Reiki appears to differ among recipients, a sense of profound relaxation is commonly reported.[28] A recent survey of complementary therapy use by Americans found that 1% had ever used Reiki.[55] As no extra equipment or special environment is required, Reiki is easily used in hospital settings, and can be administered simultaneously with conventional interventions such as bone marrow biopsy, chemotherapy, and surgery. In fact, Reiki is currently practiced by several members of the nursing staff at Yale New Haven Hospital, where there is also a Reiki volunteer program that provides treatments to patients who request them.

Preliminary studies suggest that Reiki may have psychological and physiological benefits in a variety of settings. Reiki can help promote relaxation and is associated with many psychological benefits, including reductions in anxiety,[56] perceived stress,[57] and pain severity.[58] Furthermore, preliminary studies suggest that Reiki may have positive effects on autonomic balance in healthy subjects. One small randomized controlled study found that Reiki lowered blood pressure compared to both a control group and treatment by a person with no knowledge of Reiki.[57, 59] However, this study used healthy volunteers as subjects and did not look at heart rate variability as an outcome measure. Thus, despite the ease of providing Reiki in a hospital setting compared to a more extensive treatment

like massage, there is no existing research investigating the potential for sympathovagal changes by Reiki on cardiac patients in any cardiac inpatients.

Summary of Background & Significance

Sympathetic activation is known to promote arrhythmogenesis, while prominent parasympathetic tone, particularly in the setting of MI, is protective against both arrythmogenesis and sudden cardiac death. Increasing evidence suggests that emotional stress can trigger sympathetic activation, measured as depression in heart rate variability, leading to the associated negative sequelae. In theory, then, reducing or preventing emotional stress would be therapeutically beneficial, particularly in an at-risk cardiac population. There are a variety of approaches to reducing emotional stress acutely and chronically, and some of these modalities have been shown to enhance vagal autonomic tone. Reiki is one such intervention that has shown promise not only as a relaxation therapy, but also as a simple, safe, and feasible intervention in the critical care setting. However, it is not yet known whether interventions like Reiki can lead to measurable improvements in parasympathetic activity, such as would be clinically relevant in a post-MI patient population in whom pathologic depression of HRV is closely linked to prognosis. Rigorous study of complementary therapies has thus far been limited, in part because of the difficulties in designing rigorous trials to measure these modalities. Exploration of physiologic effects of interventions like Reiki on measures of HRV in post-MI patients may help further understanding of their therapeutic value and bolster an evidence base that has been slow to build.

STATEMENT OF PURPOSE

In 2004, we developed and conducted the first study to investigate the feasibility and physiologic effects of providing Reiki to in-hospital cardiac patients. Specifically, we aimed to determine whether receiving a single 20-minute session of this intervention could be associated with improvement in HRV. We hypothesized that Reiki treatment would induce a relaxation response in subjects, lowering heart rate and blood pressure, and most importantly, generating an increase in high frequency (HF) HRV associated with increased parasympathetic tone and better cardiovascular outcomes. As a pilot designed to assess feasibility and potential for detecting physiologic change of the Reiki intervention, this study provided a promising platform for future inquiry. However, because the study was not controlled, we could not evaluate whether Reiki itself was providing the effects observed.

The next step in understanding exactly whether and how a treatment such as Reiki may exert its effects was to conduct well-controlled trial. This study aimed to determine with increased rigor whether Reiki treatment can demonstrate a specific, measurable improvement in HF HRV of patients recovering from acute coronary syndrome, a population known to have pathologically depressed HRV. We hypothesized that Reiki treatment would induce a measurable effect on autonomic balance and emotional state in subjects compared to both a resting control group and a complementary therapy without human interaction.

Through this randomized, controlled, prospective clinical trial, we sought to (a) rigorously measure the short-term physiologic effects of complementary therapies in an at-risk patient population, beginning to distinguish characteristics of these therapies that may provide the specific mechanism for their effects, (b) determine the feasibility of integrating these therapies into standard hospital care, and (c) identify any clinical or psychological patient characteristics that may predict response to these modalities.

METHODS

Study Design

We first conducted a nonrandomized pilot investigating the feasibility of providing Reiki treatment and measuring its potential physiologic effects in cardiac patients at Yale-New Haven Hospital. Reiki is an ongoing clinical program offered to patients at Yale-New Haven hospital by a specific group of trained hospital volunteers; additionally, a subset of nurses working on the three cardiac units – Cardiac Intensive Care Unit and the two step-down cardiac units, 5-2 and 5-3 – at Yale-New Haven Hospital have been trained up to at least First degree Reiki. Nursing managers on these units agreed to allow those nurses trained in Reiki and other volunteers to act as the primary intervention providers in this study.

Fourteen subjects underwent continuous ambulatory ECG monitoring during a 20-minute baseline period followed by a 20-minute intervention period, during which each subject received a Reiki treatment. HRV data were collected for all treatment subjects, and data from eleven of the fourteen proved of sufficient quality for analysis. HRV data were analyzed for change in vagal output at baseline vs. during Reiki treatment. A five-point Likert scale was used to obtain data on patient's perceived emotional states before and after the Reiki treatment. After analysis of pilot study data showed expected changes in physiologic parameters with few logistical difficulties in administering the intervention, we then designed and conducted the randomized, controlled trial.

For the controlled trial, the participant population included inpatients on any of the three cardiac units at YNHH with confirmed acute coronary syndrome (ACS) within the past

72 hours. ACS was defined as (1) ST elevation MI (STEMI), characterized by typical symptoms or ECG with ST segment elevation, plus elevated CK-MB or troponin, (2) non-ST elevation MI (NSTEMI), characterized by typical symptoms or ECG without ST elevation, plus elevated CK-MB or troponin, and (3) Unstable angina (UA), characterized by symptoms or ECG indicative of ischemia, with normal enzymes. Patients receiving angioplasty were only eligible for the study > 12 hours after undergoing the procedure, or on the day(s) prior to the procedure if scheduled non-urgently.

Potential participants were excluded from the study if they had a paced heart rhythm or atrial fibrillation, both of which would preclude measurement of heart rate variability by the Holter monitor. Patients were also excluded if they were on sympathomimetic drips, which alter autonomic function, or on chronic mood-altering drug therapy, which can alter HRV. Patients who did not speak English, those with other ongoing acute illnesses, and intubated patients were excluded as well.

All patients meeting inclusion criteria were asked if they would like to participate in a study of relaxation in patients with acute heart conditions. All subjects were told prior to randomization that they would be randomized to receive “one of three relaxation therapies: a resting relaxation, a hands-on relaxation called Reiki, or relaxing music”; patients were also reassured that they would be able to receive any of the three therapies following the study protocol, if they were interested in one that they did not get randomized to receive. This was done in an attempt to blind patients to the intent of the study and avoid biasing patient expectations. The 48 patients who met criteria for the

study and provided consent were randomly assigned to receive Reiki treatment, music, or the resting control. See Figure 3 for a visual description of the randomization protocol.

An ambulatory ECG monitor (Holter) was placed on each consented participant and remained in place through a 20 minute baseline phase, a 20 minute intervention phase (Reiki, music, or control), and a two hour post-intervention phase. Blood pressure was taken and emotions measured by Likert scale (see Figure 4) were measured in each subject at the end of the baseline phase and immediately following the intervention.

Human Investigational Committee (HIC) approval was obtained prior to the study, and this study adhered to all current privacy policies regarding the use of patient information. All patients signed informed consent prior to initiation of any study procedures.

Randomization

Randomization was generated by a statistician not involved with patient recruitment or enrollment. Treatment assignments were generated by random numbers. Up to three randomizations were allowed for each day of study intake. The three treatment arms of Reiki, music, and resting control were balanced every six days. Either “Control”, “Music”, or “Reiki” was written on an index card and sealed in envelopes which had been numbered to correspond to days of enrollment. Determination of group designation was revealed only after informed consent was obtained, and both study coordinator and patient were blinded to the group designation until they were enrolled.

Intervention Protocol

Baseline Phase

The intention of the baseline phase was to generate a referen

ce point for the standard of care experience in the acute post-ACS hospital setting; i.e., to get patients to their own physiologic baseline, avoiding both excessive stimulation and prematurely inducing a relaxation intervention. During the 20 minute baseline phase, all subjects wore the Holter monitor and were instructed to continue doing whatever they had been doing; the only restrictions placed were that subjects were asked not to eat, sleep, or walk around (all of which might cause wide variations in heart rate or blood pressure). Each subjects' nurse was notified in advance of the study protocol and no procedures, tests, or nursing care was provided during either the baseline or intervention periods.

Intervention Phases

At the end of the baseline phase, blood pressure was taken and emotional state was assessed through a 10-point Likert scale. Then, all subjects were instructed to lie in the supine position, with eyes closed, covered in bed linen. A set of standard environmental conditions, which we termed "minimal distraction environment" (MDE), was created for all subjects during the intervention phase; resting control subjects received no further intervention, while music and Reiki subjects received 20 minutes of the modality. The MDE was created by asking all visitors to leave, turning off television and room lights, closing the door, and placing a 'Do Not Disturb' sign outside the door. The subject's

nurse was asked to support the patient in not being disturbed for the 20-minute intervention phase. In non-single rooms, the patient in the other bed was asked to lower TV and voice volume and refrain from having visitors for the 20-minute intervention phase.

Resting Control

All subjects randomized to receive resting control experienced the MDE during the intervention phase. This was chosen as a passive control in order to rule out the possibility that simply reducing noise and distractions for 20 minutes, in the otherwise often hectic environment of the CCU or cardiac floors, might have as much of an effect on HRV as any of the active interventions.

Reiki Intervention

Subjects randomized to receive Reiki were given a 20-minute Reiki treatment in addition to the MDE during the intervention phase. Reiki treatments were administered by a nurse or certified hospital volunteer trained to at least First Degree Reiki practice. During the intervention phase, the practitioner introduced herself to the patient and told the patient that she would be providing Reiki treatment; no further explanation or discussion about the history or mechanism of Reiki was discussed. Practitioners then offered Reiki to subjects in the supine position, over their hospital gown and bed linen, by placing their hands lightly on the subject's head, chest, and abdomen in a series of seven standardized positions (see Figure 5 for a visual description of the position used). These seven positions were chosen with the help of Pamela Miles, an international expert on the

integration of Reiki into clinical settings[28], and were designed to adhere to traditional Reiki hand positions without requiring subjects to move or turn over.

Prior to initiation of the protocol, Pamela Miles led a one-hour training session for all Reiki providers to ensure consistency of treatments. At this session the providers were instructed to adhere to the following standardized protocol:

Begin with the three head positions and then proceed to the four torso positions. The first 6 positions will be held for an average of 3 minutes and the seventh position for approximately 2 minutes, for a total treatment of 20 minutes. The emphasis of treatment is to treat each person relatively uniformly within a consistent time frame, and to cover all seven of the placements chosen for this protocol.

Music – An "Active Control"

Because there are many components of Reiki treatment which might exert any effects seen, we wanted to choose an active control which would potentially offer more insight into the active component, if any, of Reiki. As Reiki is a touch-based relaxation treatment, we wanted to compare the effects of Reiki to both the resting control described above a known and well-studied relaxation therapy without human contact that would control for the element of human contact and touch during the intervention. Music was therefore chosen based on the wealth of preexisting literature on music for relaxation and anxiety-reduction in the inpatient clinical setting. Meditative music, defined as music with tempos at or slower than normal resting human heart rate, decreases heart rate, blood pressure, and plasma catecholamines.[60] Tempo has been demonstrated to be the most important factor affecting physiologic response in listeners.[61] Slow music may also increase the HF component of HRV in post-MI patients.[62] Music can therefore provide an active control for human contact and touch, as a therapy that has proven relaxation effects but does not have the added human component of Reiki.

For the music group, patients listened through headphones to a standardized selection of five classical music pieces, each with tempo between 60 and 75 beats per minute, totaling 20 minutes of music: Adagio cantabile from Bach Sonata in B minor, Op. 5; Ave Maria (instrumental version); Beethoven's Cello Sonata No 3 Adagio Cantabile; Romance for Guitar and String Orchestra; and Debussy's Claire De Lune.

Some previous controlled studies on Reiki have used a "sham" form of Reiki treatment for the control group, in which people not trained in Reiki practice place their hands in the same positions as those used by Reiki-trained practitioners in the intervention group.[63] However, there is continued disagreement among researchers about the true placebo nature of supposed sham Reiki treatments using untrained personnel; some investigators have suggested that even the sham Reiki may have subtle effects that would preclude its usefulness as a true control. [64] Therefore, we felt that introducing a second touch-based intervention group would not offer enough distinction among potential active mechanisms, and we did not deem a sham Reiki control group to be ideal for this study.

Data Acquisition

See Figure 6 for a visual description of the timing of data acquisition.

Physiological data acquisition

Holter monitors recorded ECG data from each subject continuously from the beginning to the conclusion of each protocol. Each subject's blood pressure was measured at the

beginning of the baseline period upon placement of the Holter, at the end of the baseline period immediately prior to the intervention, and at the end of the intervention period.

Psychological data acquisition

Measurement of anxiety state by single item Likert scale has been demonstrated to yield equivalent results to the more extensive State Train Anxiety Index[65]. In order to assess potential state changes in anxiety and other emotions associated with cardiovascular disease or relaxation therapies, all subjects completed a Likert scale regarding their mood states (range 1-10, where 1 = "feeling this emotion not at all right now" and 10 = "feeling this emotion extremely strongly right now") at the end of the baseline phase and after the intervention period (see Figure 4).

HRV Analysis

Continuous ECG recording was collected by cassette or digital Holter from several minutes before the onset of the baseline phase through two to twenty-four hours after the intervention phase. Data from Holter monitors were read and put through a computer algorithm by technologists blinded to the subject assignment. Each tape was digitally sampled and analyzed by technologists at the Yale-New Haven Hospital Holter Laboratory to ensure accurate identification of QRS complexes. Tapes were then manually processed and edited to determine from each R-R data point whether to label each beat as normal, ectopic, or noise. The ectopic beats and noise were removed from the data set and replaced with interpolated linear splines.[66]

HRV was analyzed by spectral analysis via fast Fourier transform, measured in 5 minute segments with a rolling window, corresponding to the times of each study phase as was previously recorded by the researchers. The power spectrum was integrated over the high frequency (HF) band (0.15 to 0.40Hz)[43], the frequency band of interest for this study.

Statistical Analyses

Power Calculations

Based on preliminary data from the pilot study, described in the Results section, as well as results from complementary studies looking at negative effects of stress on HRV, we estimated that at most, Reiki treatment might lead to a 0.5 unit change in HF HRV compared to a control that had no effect, and perhaps half of that compared to an active control. The resulting power calculations estimated 91% power to detect a difference of .49 HF unit change with Reiki versus passive control, at an alpha level of 0.05, with 23 patients per group. Given this estimate, a target enrollment of 23 patients per group would also provide 80% power to detect a difference of .34 HF change with Reiki vs. music.

Data Analyses

All HRV parameters were log-normalized to normalize the distribution as is routine for studies of frequency-domain HRV. Twelve-minute blocks corresponding to the middle of the baseline and intervention phases were evaluated for the mean high-frequency power (0.15 – 0.50 Hz).

Demographic and baseline clinical data was compared across groups using contingency table analysis for categorical variables and ANOVA for continuous variables. Then, using repeated measures ANOVA with appropriate controls for multiple comparisons to compare all three interventions and t-test to directly compare each pair of interventions, intervention versus control groups were compared for changes in vagal output as measured by ln HF between the baseline and intervention periods. The same analysis was done to compare average changes in heart rate, blood pressure, and Likert scores from baseline to intervention phase across each group of subjects.

To evaluate whether the observed effects of relaxation interventions on HF HRV may have been unduly influenced by other clinical or demographic variables, we first conducted a series of bivariate analyses looking at the effect on the change in HF HRV of factors known to independently influence HRV: age,[67] sex, race,[68] ejection fraction, peak troponin, location of MI, prior MI, and diabetes mellitus.[69] Bivariate analysis was also used to assess whether patients' prior use of CAM, a factor that was nonsignificantly higher in the Reiki group, affected the change in HF HRV. We utilized t-test for dichotomous variables (sex, race, location of MI, prior MI, diabetes, prior use of CAM) and standard least squares regression for continuous variables (age, ejection fraction, peak troponin). For any variables influencing change, we also conducted a multivariable analysis controlling for those variables. Because the number of patients was small, we chose a parsimonious model to evaluate only those characteristics that might influence response. Because baseline HF HRV was found to influence the change in HF HRV and was lower in the Reiki group, we conducted a stratified analysis, dividing all patients at

the median baseline HF HRV, 4.47, into a “high baseline HF HRV” group and a “low baseline HF HRV” group.

Role of the Student

With the help of my advisor, I designed both the pilot study and this randomized controlled trial. I conducted the literature review, consulted with Reiki masters to develop the study protocol, obtained permission to use cardiac floor nurses to provide Reiki treatment, and recruited the nurses. During the summer of 2004, I recruited and enrolled 14 subjects in the pilot study, performing all aspects of the study flow and data analysis. At this time I learned how to obtain Holter data from Teresa Donovan, a study coordinator with Dr. Lampert, and I applied the Holters for all subjects in both studies. For both the pilot study and this study, HRV processing was done using programs previously created by Dr. Forrester Lee and used by Dr. Lampert in her HRV research. During the 2006-2007 academic year, I designed and conducted all aspects of this study, including screening and recruiting patients, randomization and patient enrollment, organizing the flow of the study, overseeing the nurses performing Reiki treatment, and data analysis with my advisor.

I presented a partial analysis of the results at the Yale Student Research Day poster session in May 2007, and I was invited to give a one-hour presentation of the study and results at the Harvard Osher Integrative Medicine Fellows monthly conference, which I did in January 2008. Furthermore, I submitted an abstract and was selected to be one of the student oral presentations at the American College of Physicians national conference

in May 2008. We have submitted our results as an abstract to one other national conference, the Heart Rhythm Society meeting, and plan to prepare the manuscript for publication submission within the month.

RESULTS

Pilot Study

A total of fourteen treatment subjects completed the study protocol. HRV data were collected for all treatment subjects, and data from eleven of the subjects were analyzed for change in vagal output at baseline vs. during Reiki treatment (in three patients, noise or other technical issues did not allow for HRV analysis). As shown in Table 1, preliminary analysis showed significant increase in high frequency output and HR during the Reiki session compared to baseline. This effect was moderately sustained throughout the post-treatment period. Additionally, nearly all subjects reported increased subjective feelings of relaxation or calmness post-treatment; as shown in the table, subjects' Likert scale self-report of feeling "relaxed" improved significantly post-treatment, while a significant decrease in the emotions "tense" and "scared" was also noted. Other psychological markers showed similar trends but not significant change.

Randomized Trial

Subjects

Figure 7 illustrates the flow of patients throughout this study. 229 patients were screened for the study via chart review on the cardiac units based on initial ACS diagnosis and <72 hours since admission. Of these, 110 participants met inclusion and did not have exclusion criteria, and sixty-five were available for explanation of the study and invitation to participate. Unavailability was largely due to patients being off the floor for imaging or procedures, or imminent discharge from the hospital. Fifty out of sixty-five consented and were randomly assigned to one of three intervention groups. Fifteen

patients declined to participate because of procedure scheduling, anticipated discharge, lack of interest in participating, or concomitant participation in other research protocols. Two consented patients withdrew from the study due to reasons unrelated to the study, and one subject's data were lost due to technical difficulties.

Subjects ranged in age from 35 to 86 years old. Demographic and clinical characteristics of the 47 subjects who completed the protocol are presented in Table 2. There were no significant differences between the intervention groups with respect to any of these variables. There were more subjects with prior MI in the music group, more subjects with prior CAM usage in the Reiki group, and more current smokers in the resting control group; however all of these were non-significant differences.

Baseline heart rate, blood pressure, and HF HRV of subjects are shown in Table 3. As a group, the Reiki subjects had a lower average baseline HF HRV. RR and blood pressure were not significantly different at baseline across groups. Additionally, there were no significant differences in numbers of patients from the CCU amongst the three groups.

Effect of Intervention on High Frequency HRV and Heart Rate

Adequate Holters were recorded in 12 control, 13 music, and 12 Reiki patients. Table 4 and Figure 8 provide a summary of the HRV results. The mean HF HRV increased significantly from baseline in the Reiki group (0.58±0.16) but did not appreciably change in the control group (0.06±0.16), and was decreased slightly overall in the music group (-0.10±0.16). Reiki treatment increased HF HRV to a greater extent than either of the two

control groups (p=0.02 by ANOVA with repeated measures); this was also true when comparing Reiki treatment to music (p=0.007) and Reiki treatment to control (p=0.025). The effects of the resting and control and music interventions were not significant when compared to one another. Of note, there were no significant differences in HRV change amongst different practitioners providing Reiki treatment.

As shown in Table 5 and Figure 9, mean RR interval increased significantly with Reiki treatment (46.49±11.3), i.e. a significant drop in heart rate during the intervention. This effect was significantly different compared to the two control groups (p=0.01 by ANOVA with repeated measures for differences among the three groups). However, mean RR interval also increased substantially in the resting control group (41.40±11.3), leading to a nonsignificant difference between Reiki and control. Music had no overall effect on mean RR interval (0.22±10.9); a significant difference was measured when compared to both Reiki (P=0.002) and resting control (P=0.01). No meaningful changes were observed in either systolic or diastolic blood pressure within or across groups (see Table 5).

Emotional State

Emotional state was assessed by self-reported Likert scale (on a scale of 1-10, where 1 = not feeling that emotion at all, and 10 = feeling that emotion maximally) at the end of the baseline and intervention phases. Results are summarized in Table 6. Reiki treatment led to substantial increases in magnitude of all positive emotional states (happy, relaxed, calm) with corresponding reductions in mean Likert score of all negative states (stressed,

angry, sad, frustrated, worried, scared, anxious. As illustrated in Figures 10 and 11, emotional state changes trended from most positive emotional change with Reiki treatment to least positive emotional change with resting control; with the majority of the variables, the emotional change with music was intermediate between that of resting control and Reiki. The most significant changes among all three groups together were in reported stress (P=0.01 by ANOVA with repeated measures) and feeling relaxed (P=0.03 by ANOVA with repeated measures). In addition, in several cases both Reiki and music differed significantly from control, but there was no significant difference between Reiki and music. Subjects reported feeling significantly less angry during Reiki compared to control (P=0.046) but not music (P=0.68), and the same was true for feeling calm (Reiki vs. control P=0.001, Reiki vs. music, P=0.56).

Effects of Clinical and Demographic Factors on Change in HRV

Bivariate regression analyses were conducted to identify any clinical or demographic factors that might have predicted response to intervention. As shown in Table 7, the only two clinical factors found to influence change were baseline HF HRV and peak troponin. Given that baseline HF HRV was already found to be lower in the Reiki group, a stratified analysis was done to exclude the possibility of a ceiling effect. Figure 12 shows the two resulting analyses, after dichotomizing all subjects into a “Low baseline HRV” and “High baseline HRV” at 4.47, the median baseline HRV value of all subjects. Although subjects with higher baseline HF HRV experienced less positive change with any intervention, Reiki treatment effects remained significantly greater, with a p-value of 0.05 in both high and low baseline groups. We created multivariate models to evaluate

the effects of peak troponin and baseline HF HRV; effects of the intervention on HF HRV were still significant.

DISCUSSION

This study is the first randomized controlled trial to study the effects of Reiki treatment on heart rate variability, a measure of autonomic activity, in an early post-MI inpatient population. In this study, a 20-minute session of light touch Reiki treatment given by nurses trained in Reiki practice was found to acutely increase vagal activity and decrease self-reported negative emotional state more than either classical music or a resting control in post-MI inpatients. Heart rate was reduced with Reiki treatment and resting control, but not music, and there seemed to be no measurable effect on either systolic or diastolic blood pressure with any of the interventions. Although preliminary, these findings suggest that Reiki treatment may have a role in the care of cardiac patients following myocardial infarction.

Change in vital signs with intervention

Reiki treatment had a small but significant effect on heart rate, increasing the R-R interval by 47 milliseconds, which corresponds to an average drop in heart rate of about 3 beats per minute. The resting control also exerted a similarly small but significant effect (increased R-R by 41 msec), while music resulted in no overall change in heart rate over baseline. Despite the somewhat similar-appearing results of heart rate with resting control and Reiki, HF HRV was quite significantly different between these two groups, underscoring the importance of using more complex physiologic parameters vital signs in exploring the effects of complementary therapies. Heart rate represents the combination of sympathetic and parasympathetic influence, whereas isolation of the high frequency power of the heart rate variability spectrum allows for more direct measurement of the parasympathetic component only. Therefore, what our findings imply is that

parasympathetic augmentation is much greater with Reiki treatment than with control, even though they may have similar effects on heart rate.

That blood pressure was not affected by any of the interventions may be explained patients' near ubiquitous use of anti-hypertensives, which may have exerted a ceiling effect limiting observation of appreciable changes with the interventions. As shown in Table 3, baseline blood pressure in all three groups ranged from 116/117 for systolic BP and 65-70 for diastolic BP; as these represent values on the lower side of normal, and all of the subjects had just suffered myocardial damage impairing cardiac output, any significant drop in blood pressure below these levels would not necessarily provide therapeutic advantage. A second explanation for these findings lies in the manner of data acquisition. Whereas heart rate and HRV were measured unobtrusively via Holter monitor throughout the study, measurement of blood pressure required an active, uncomfortable intervention; this may have been particularly jarring to patients immediately following the relaxation intervention, leading to a reflexive increase in blood pressure that masks actual values during the intervention.

Implications

Clinical significance

The results of this study have implications for both research and clinical practice. Interventions such as Reiki treatment may have a role in the acute post-MI period if they can be shown to enhance recovery of parasympathetic tone. The increase in HF HRV with Reiki treatment was statistically significant, but these results will ultimately be

useful only if they can prove to offer clinically significance changes. As described earlier, post-MI propranolol therapy was found to improve recovery of HF HRV by about 0.9 units in six weeks; therefore, the 0.58 increase in HF HRV found with a single Reiki treatment has major clinical implications if it can be found to be sustainable over time.

Cardiovascular Outcomes

Sudden death remains a major health problem in this country, leading to approximately 300,000 deaths annually. Any measures that could help prevent or reduce the number of patients who experience SCD would therefore be of great clinical value. And, as vagal HR regulation is known as an important protector from SCD, HRV is gaining awareness as a useful tool for furthering understanding of interventions and mechanisms around altering vagal activity. Our results therefore contribute to this growing body of evidence, suggesting that interventions that induce states of perceived psychological relaxation may also generate real improvements in vagal activity and may have use in primary or secondary prevention of arrhythmic events.

There are two major lines of inquiry around improvement of cardiovascular outcomes addressed by the results of this study: (1) the extent to which emotional stress or relaxation can be demonstrated to affect morbidity and mortality, and (2) the extent to which complementary therapies that positively affect the experience of stress or induce relaxation by various mechanisms can be demonstrated to have specific, measureable effects that affect morbidity and mortality.

Alterations in cardiac autonomic tone may provide a mechanistic explanation for the increased mortality observed in cardiac patients with depression or anxiety.[70] In these studies, the psychological metric of interest is not emotional state—the emotions a person is feeling at a particular moment, such as the extreme anxiety experienced during a heated soccer game and extreme anger after the favored team loses—but emotional trait, a person's general emotional milieu over a period of time. A recent cohort study by Shibeshi and colleagues found that high levels of anxiety maintained over an extended period of time following diagnosis of CAD represented a nearly twofold risk of nonfatal MI or total mortality compared to though patients with low anxiety scores.[71] Moser and Dracup found that patients with MI who reported higher levels of anxiety were nearly 5 times more likely to have complications in the post-MI period.[72] Furthermore, depression is an independent risk factor for poor prognosis among ischemic heart disease patients, and approximately 1 in 5 survivors of acute MI have been found to meet diagnostic criteria for major depression.[73]

Consistent with these findings, attempts to reduce stress in the post-MI period have been shown to improve outcomes. Frasure-Smith studied 461 men who took part in a trial of psychological stress monitoring and intervention following acute MI; psychological stress was assessed just prior to hospital discharge, and half of the patients were assigned to a treatment group that involved regular stress level assessment and nursing support during high stress times. Results showed that high reported stress at discharge was associated with a close to threefold increase in cardiac mortality over three years among the control patients ($p=0.0003$), with no significant increase in mortality risk among high

stress patients who underwent the intervention.[74] A 5-year followup study of Dutch patients assigned to either conventional cardiac rehabilitation or standard treatment plus a comprehensive training in relaxation skills found that patients in the relaxation group experienced significantly fewer cardiac events (OR 0.72; 95% CI 0.38-1.36) and hospitalizations were reduced by 31% compared to the control group.[75] Thus, Reiki treatment and other complementary therapies that reduce acute or chronic levels of anxiety and stress may offer significant therapeutic and preventative effects on the severity and progression of cardiovascular disease. An important avenue for future research would be to evaluate whether ongoing Reiki treatment could generate lasting effects on autonomic balance and psychological wellbeing, potentially reducing the risk of developing depression and offering cardiovascular protection.

Advancing rigorous study of complementary therapies

Further, as a second line of inquiry, this study has important implications regarding the rigorous evaluation of complementary therapies and their subsequent integration into conventional medicine. Despite the rising interest in complementary therapies, multiple barriers prevent their automatic adoption by physicians and hospitals.[76] This study was designed to address several of those barriers. The main argument physicians have used against CAM is the lack of available research proving safety and efficacy. Though the scientific community has argued that there is a lack of evidence base for these modalities, it has not, in the name of evidence-based medicine, been quick to generate a body of rigorous studies exploring potential efficacy; as Raschetti and colleagues pointed out in their review of the CAM literature in the last five years, though CAM articles have

increased in the scientific literature, over half are published in journals without an impact factor (used as a rough indication of quality in the scientific community).[77]

Unfortunately, this has led to a vicious cycle: physicians as a whole have had a reluctant attitude or limited interest in exploring CAM, placing the onus on CAM practitioners and other non-medical experts to design and conduct trials to determine efficacy. Without sufficient funding and expertise in applying scientific methodologies, the literature that arose from such studies was based on weakly designed studies on healthy subject populations of limited use in a clinical setting; this in turn only bolstered the argument by physicians about the lack of research on these therapies. However, weakly designed studies, whether positive or negative, cannot offer true conclusions in either direction about a particular intervention. It is therefore imperative that the scientific community embrace the rigorous study of these modalities in order to build a strong foundation of research upon which to make rational decisions about the utility of these therapies.

Previous Studies

Studies on the effects of Reiki

No large randomized controlled trials focused on the physiologic effects of Reiki treatment have been published at this time. An increasing number of small studies have reported interesting data, and NCCAM currently has five research projects on Reiki. A review of the scientific literature on Reiki has been published by Miles.[28, 78] Preliminary evidence suggests that Reiki treatment influences the body toward relaxation and health both psychologically and physiologically.[58] In terms of psychological benefit, Shore found that a 1-1.5 hour weekly treatment of Reiki for six weeks resulted in significant

reduction in symptoms of psychological distress compared to subjects receiving placebo Reiki, and these differences persisted one year later.[79] Reiki has been found to reduce anxiety and fatigue in patients with cancer,[80] and a randomized trial of 24 cancer pain subjects by Olson et al[81] demonstrated significant improvement in pain ($P = 0.35$) and psychological component of a Quality of Life assessment tool ($P = 0.002$) in the group receiving standard opioid therapy plus 2 Reiki sessions compared to a control arm receiving standard opioid therapy and rest. Of note, the investigators stopped enrollment after 24 patients because of overwhelming request for Reiki treatment from all study participants.

Very few published studies have examined physiologic effects of Reiki treatment beyond heart rate and blood pressure. Mackay et al.[59] published the only study to date examining autonomic response to Reiki treatment, using cardiac baroreflex sensitivity as a marker of vagal tone. Although this study demonstrated significant changes in HR ($P < 0.005$) and diastolic BP ($P = 0.005$) in subjects receiving Reiki treatment compared to those in a placebo group, no other significant changes were observed. Because this study used healthy volunteers, who may have all possessed healthy functioning autonomic nervous systems, this study has limited value in assessing the potential use of Reiki treatment in a patient population with autonomic imbalance. Thus, while previous studies have evaluated psychological effects and some limited physiologic effects of Reiki treatment, we are the first to evaluate vagal activity of Reiki treatment on cardiac patients, a group in which increased vagal activity is protective.

Previous studies of Music

While sedative music has been consistently shown to enhance perceived relaxation in the listener, physiological responses to music have been inconsistent. A small meta-analysis of studies evaluating the impact of music on anxiety in hospital patients found strong evidence that music effectively reduces anxiety by the State Trait Anxiety Inventory (SMD -0.71; 95% CI -0.97, -0.46).[82] However, this effect has not been shown consistently in hospital patients undergoing invasive procedures[83]. While previous studies have demonstrated reductions in HR and BP with sedative music, more recent controlled trials of music in hospital inpatients have demonstrated no overall effects of music on either HR or BP over control. White and colleagues conducted a 3-arm randomized controlled trial of post-MI patients, comparing the effects of relaxing music, resting control, and treatment as usual on psychological and physiological markers.[62] Results showed no significant changes in heart rate or blood pressure, but the intervention groups were found to have increases in high-frequency HRV immediately after the intervention.

In our study, music had a significant effect on several of the psychological markers of emotional state, but it had no measurable effects on any of the physiological parameters used in this study. These results suggest that while music may reduce people's experience of certain emotional states, it does not have the physiological effect that Reiki or other touch-based healing modalities may offer. There are several additional potential explanations for these findings. The music used for this study was chosen based on a combination of availability, instrumental quality, and having a slow tempo in the range

that has been suggested to slow the heart rate. Nevertheless, it may be that a different type of music, such as those used in other studies of music,[84] may have offered a more potent relaxation effect. Additionally, there is some evidence that music preference plays a role in its effect on the listener;[85] because we did not give subjects a choice and used a standardized selection of music, we may have limited the overall effect that the music would exert on the group randomized to receive it. Finally, it may be that music has a complex effect on physiology, including both stimulating and relaxing features. These factors may explain the inconsistent evidence on the physiologic effects of music; the studies themselves may have had inconsistent rigor, people may respond inconsistently to the same musical stimulus, or the effects may be more complex and may therefore appear to change depending on the variables studied.

Mechanisms

Emotions, Autonomic Balance, and Cardiovascular Disease

Walter Cannon was the first to elucidate the "fight-or-flight" stress response in 1929.[86] Cannon described increases in sympathetic nervous system activity, with corresponding cardiovascular responses, in response to real or perceived threat or anxiety. As discussed previously, sudden strong emotions have been linked to the precipitation of severe or even fatal arrhythmic events. On a less dramatic scale, acute mental stress has been demonstrated to cause silent myocardial ischemia in 30-60% of patients with CAD subjected to a laboratory stressor.[87] Through research describing the effects of stress and emotions on the cardiovascular system, a multi-factorial model of the pathophysiology of stress and its role in SCD and CAD has emerged. Coumel describes the foundation for

arrhythmogenesis as a mixture of structural substrate within an autonomic milieu, with a tipping point occurring with a transient initiating event.[88] In this model, the transient initiating event generally refers to an emotional or psychosocial stress that induces a burst of sympathetic activity with a corresponding withdrawal of vagal activity.[89] This model also explains why patients who have suffered myocardial infarction are particularly vulnerable to arrhythmia, as they have damaged heart muscle acting as a structural substrate, as well as parasympathetic withdrawal represented by the suppressed HF HRV. Our study suggests that Reiki treatment can affect the autonomic milieu in post-MI patients through increased HF HRV, and it may also offer prevention against the transient initiating event through increased relaxation and stress-mediating effects.

The Importance of Variability

Systems-oriented models propose that HRV is an important indicator of both physiological resiliency and behavioral flexibility, reflecting the individual's capacity to adapt effectively to stress and environmental demands.[90] Although the *homeostasis* model of physiological change generally extols the virtues of stability around a set normative point, study of markers such as HRV has led to the realization that while too much instability to a system can be pathological, so too can too little variation. A more recent model used in cardiovascular regulation, called *homeodynamics,* purports that a certain amount of variability within an organism's key regulatory systems is critical to the inherent flexibility and adaptability that epitomize healthy function.[30] This principle is aptly illustrated by a simple analogy: just as a young tree with a flexible trunk and branches can way with, and survive, heavy winds, while an old or dead tree with rigid

limbs will crack under the pressure of a sudden gust – the healthy heart with high variability remains similarly responsive and resilient against potential arrhythmic assault. As a diagnostic or prognostic tool, HRV may therefore be helpful in identifying breeches in the normal healthy functioning of this homeodynamic system. Ziegler and colleagues found that HRV dysfunction in patients with diabetes mellitus preceded onset of any symptoms of autonomic neuropathy.[91]

It is important to point out the limitations assessing potential therapeutic value from change in HRV. Though increase in vagal tone has been associated with improved health outcomes in certain situations, vagal activity has been shown to have selective, independent effects at the sinus node and ventricles, suggesting that vagal activity alone may not be enough to presume clear benefit.[92] As an example of this, scopolamine, a muscarinic parasympathetic agonist, has been demonstrated to increase vagal tone in both animals and humans. In spite of this, it has not shown any significant benefit in preventing ventricular arrhythmias. Thus, future studies on the long-term outcome benefits of Reiki treatment should consider evaluating other physiologic parameters in addition to HRV.

Mechanisms Involved in Reiki

How can we explain potential mechanisms of Reiki treatment in light of these results? As was mentioned above, this study set out to measure physiologic effects of the whole modality Reiki against a distinct other modality used for relaxation, music, as well as a control environment without intervention. This study did not, therefore, attempt to

distinguish what, if any, specific components of Reiki might account for the effects measured. From the psychological standpoint of the patient, this study suggests that Reiki treatment offers significant benefit for patients' perceived levels of stress and anxiety. Furthermore, there appears to be a physiological effect greater than that elicited by relaxing music. Reiki treatment differs from music in several key ways, the most prominent of which is the presence of another person offering the intervention. Whether the performance of Reiki treatment over music stems from the mere presence of a human, the presence of a person with healing intention, the light touch technique, something in the realm of energy or subtle biofields, or a combination of these or other factors, is unknown.

It is possible that the human touch component of Reiki treatment is the most important aspect of the modality. Touch-based healing has been practiced throughout the world for thousands of years. Kerr, Wasserman, and Moore have proposed a neuroscientific explanation for the effectiveness of Reiki and other touch healing treatments; they hypothesize that the commonalities among all of these modalities – light tactile contact, somatosensory attention directed toward the body, and the relaxed or healing context – lead to sensory reorganization in somatosensory cortical maps, leading to the healing effects observed.[93] Indeed, this may explain the challenges in designing trials using "sham" control groups for touch-based healing modalities – several such studies have observed similar effects in both the real and sham groups, with no effects observed in control groups.[94, 95] This supports the theory that even light touch by a person without training in one of these modalities may have an important therapeutic effect.

However, Reiki is thought by its practitioners to involve more than just light touch; according to the National Institutes of Health's model of CAM modalities, Reiki falls into the category of techniques thought to affect the human "biofield". Asian medical models include the concept of biofields, subtle vibrational layers that are said to surround and penetrate the physical body.[96] NCCAM also refers to biofields as "putative energy fields", because their existence has defied measurement to date by reproducible methods using conventional biomedical technology.[55] Traditional Asian medicine views extended, chronic dissonance in the biofield as unhealthful, and as a precursor of pathology. According to the Asian medical paradigm, the human system requires balance and consonance throughout the subtle vibrational body to maintain optimal health and wellness; dissonance leads to disregulation of the body's self-healing mechanisms, creating a milieu conducive to disease.[97] Reiki treatment is therefore thought to merely help balance and restore these natural self-healing mechanisms, in contrast to modalities like *qi gong*, which purport to actually manipulate In explaining the nature of Reiki, Pamela Miles writes:

> *In Japanese, the pictogram Reiki refers to aspects of reality that are too subtle to be detected by conventional scientific measurement… In its most expanded usage, the term Reiki connotes the extremely subtle vibrational reality refered to in physics as the unified field, accessing which is the goal of meditation and other spiritual practices of indigenous cultures around the world… The practice of Reiki is understood to connect the recipient directly to this omnipresent but often inaccessible level of reality, rather like an applied meditation, thereby facilitating a profound release of vibrational dissonance within the system and increasing coherence.*[96]

However, one major barrier to the undertaking of rigorous research around modalities such as Reiki is that biomedicine has not yet found a language or set of tools with which to adequately study and understand the mechanisms through which these proposed effects may occur on the physical level. Although the ability of mind-body interventions to influence the functioning of the physical body has been increasingly validated, and the although indigenous medical systems have viewed "harmonizing the biofield" as an essential component of healing and cure for thousands of years, there is as yet no agreed upon scientific evidence for the existence of biofields. [55] Though models in quantum physics and super string theory may offer scientific models that might be relevant to the understanding of biofields,[98] these models are not currently mainstream medical knowledge. Without any way to understand what might be occurring in the language of physiology, doctors have therefore been deterred from embracing Reiki; it is not surprising, then, that most of the prior studies on Reiki treatment have been conducted by nurses or psychologists, and not physicians.[78] A related value of this study is therefore in its use of experts from both the conventional cardiology world and the CAM/Reiki world to design and execute the protocol.

Bridging the gap between medical doctors and CAM practitioners, collaborative studies like this one can bring new insight into understanding the mechanism of a modality such as Reiki from a biomedical viewpoint. If the efficacy of Reiki treatment to measurably affect vagal activity can be convincingly demonstrated through this and future studies, then perhaps this association can offer a mechanistic understanding of the effects of Reiki treatment. Parasympathetic activity occurs through the vagus nerve and its branches, and

spectral analysis of HRV demonstrates that there are rhythmic fluctuations in this activity. Furthermore, as described numerous times above, high vagal tone is indicative of health and wellbeing on both physical and psychological levels, and improvements in vagal activity from a pathologically depressed state can offer significant protection against negative sequelae. Perhaps, then, the rhythmic activity of the parasympathetic nervous system could act as a biomedical translation of this healthful vibrational dynamic described by so many Asian therapeutic traditions. Our scientific study is therefore in concordance with the traditional view of Reiki, adding support to the idea that Reiki treatment can influence the system toward balance (i.e., healthy sympathovagal balance) facilitating the resetting of the body's self-regulating mechanisms (e.g., through enhancing post-MI parasympathetic recovery). We hope that further collaborations between CAM experts and biomedical experts will continue along this same path, utilizing rigorous evidence-based inquiry as well as a commitment to translation rather than automatic dismissal of unknowns.

Secondary Aims: Feasibility and Cost Effectiveness

Along with the pilot study done in 2004, this study demonstrates that Reiki treatment is a safe and feasible intervention for use in the acute care hospital setting. There were no adverse events recorded in any patient during any part of the study protocol, and no patient procedures, tests, or discharge was meaningfully delayed through participation in the study.

Moreover, the large majority of the Reiki treatments were provided by Reiki-trained nurses working on the same cardiac unit as the subject randomized to receive treatment on that day. Despite their busy schedules, the nurses were nearly always able to break from their work for the 20-minute session, aided by the support of their colleagues and the blessing of the nurse manager on the floor who had given approval. The nurses' cooperation was motivated in part by their expressed enjoyment of the experience of giving Reiki treatment.

While this study did not formally evaluate cost effectiveness, this intervention was essentially free, as it was done within the staffing capabilities on the floors. This suggests that Reiki treatment could be incorporated into standard hospital care to provide additional therapeutic benefit without additional cost, and without significantly disrupting the flow of the workday for the nurses. Not only would Reiki training enhance the clinical skills of staff with minimal cost; given that a distinguishing characteristic of Reiki practice is said to be that it benefits both recipient and practitioner, Reiki training would also provide nurses with an effective self-care practice which may have the added benefit of reducing staff burnout.[99] At the Fifth World Congress on Cardiac Rehabilitation, results were presented demonstrating the cost-effectiveness of relaxation therapies in post-MI patients, highlighting that any extra costs of individual training sessions were outweighed by savings from reduced medical consumption.[75]

Thus, this study is significant not only in the matter of potential physiologic effects of Reiki on this population, but in the feasibility and potential cost effectiveness of

providing this therapy in a hospital setting. In her review of Reiki, Miles cites two dozen of the hospitals and clinics providing Reiki treatment or training for patients and staff by 2003; the number appears to have risen considerably since then. She further describes three ways in which Reiki can support conventional medical care: (1) medical and nursing personnel can learn Reiki and use it for both their own self-care and patient care, (2) Reiki practitioners can be brought in to offer treatments to patients and staff, and (3) because of the ease of training, the potential benefits of ongoing treatment, and lay interest, hospital-based programs offer classes to train patients, family members, and caregivers in Reiki.[28]

LIMITATIONS

The actual sample size proved smaller than initially projected, in part because nearly half of potentially eligible subjects were unable to be approached for invitation into the study. The reasons for this include imminent discharge, being indisposed undergoing procedures or nursing care, or other reasons why patients were unavailable or off the floor. While this is illustrative of the general challenges of inpatient clinical research, it does not and should not be necessarily extended to mean that providing any of the interventions used in this study is impractical in the inpatient setting; the entire study protocol required patients to be available in their rooms for several hours, while a complete Reiki treatment can be provided in under 30 minutes, even much abbreviated treatment can ease a distressed patient. Among subjects who did enroll in the study, technical difficulties of Holter placement or body habitus led to unusable Holter ECG data in several subjects. However, despite the smaller sample size, analysis yielded significant results. Future

studies using larger population numbers will hopefully confirm these results with increased capacity to attribute small treatment effects to the intervention rather than chance.

Despite standardization of environmental conditions and attempted minimization of environmental distractions with the Minimal Distraction Environment, there were unavoidable minor variations in environment and study protocol across subjects; for example, patients in the CCU were all in private rooms, whereas many of the step-down unit patients had roommates, a factor which may have influenced response to the interventions. However, the randomized nature of the protocol resulted in similar numbers of CCU patients assigned to each of the three treatment groups. Furthermore, a stated goal of this study was to assess feasibility of incorporating complementary therapies into real clinical inpatient settings; therefore, although minor unavoidable environmental variations may have occurred, the study protocol was successfully executed in a very chaotic hospital environment with overall consistency and reliability.

Nevertheless, the use of the nurses for the Reiki sessions may have presented a limitation to the study. The nurses had all received equivalent outside training in Reiki, however they were not all trained by the same teacher, and there was variation in the elapsed time between their trainings and the study. In order to maximize standardization, all practitioners attended training by Reiki Master Pamela Miles in the study protocol, so for the purposes of the study, we can assume that the nurses were providing the same treatment.

The study nurses all admitted to not practicing Reiki regularly on themselves or others; given that a foundation of Reiki is the regular practice of self-Reiki, this group of nurses may not have allowed us to assess maximum efficacy of the modality. Nevertheless, the fact that even this group of Reiki practitioners elicited a specific, measurable response in subjects compared to both control groups is very encouraging. It is possible that a more practicing group of nurses or other Reiki practitioners would elicit an even greater response. Furthermore, it is notable that no difference was found among practitioners in mean response elicited.

The Reiki group started out with a lower average baseline HF HRV, despite the randomized trial-design, underscoring the need for a larger sample size. Incidentally, a larger, though non-significant percentage of the Reiki subjects had diabetes mellitus compared to the control groups, and diabetes is known to cause a blunted HRV, likely due to the effects of autonomic neuropathy. However, since our measure of interest was change in HRV, not absolute measure of HRV, this nonsignificant difference did not affect our ability to analyze the data for change scores. Stratification of all patients into a high baseline HRV group and low baseline HRV group revealed that the high baseline HRV group did experience less change across all three interventions, suggesting the presence of a ceiling effect in HRV change. Nevertheless, HF HRV change was still significantly positive with Reiki treatment compared to both controls in both the high and low baseline HRV group, suggesting that lower baseline HF HRV was not acting to confound results. Of note, the observation that patients with lower initial HF HRV (a

measure of worse prognosis) greater increase with worse initial HF HRV experienced greater positive change with Reiki than those with higher, healthier baseline HF HRV is consistent with the perspective of Reiki practitioners that Reiki treatment is balancing, assisting the body to do what is needed to optimize health and well-being rather than imposing a particular result.

HRV is known to have a substantial circadian variation, with a peak in the middle of the night and a nadir during early morning hours;[100] however, all of the patients in this study completed the study protocol between the hours of 9am and 5pm, minimizing any potential circadian interference. Studies of normal healthy subjects have demonstrated that power spectral measures of HRV appear to be highly consistent from day to day.[101] This has, in fact, also been shown to be true for patients with heart failure.[102] Bigger and colleagues demonstrated the same for patients with cardiac arrhythmias and previous myocardial infarction, concluding that this default stability would facilitate measurements of any actual changes in variability due to disease progress or medical intervention.[103] This prior evidence would indicate that the changes in HF HRV observed in this study may be attributed to the interventions and not a normal variation. Further, each patient acted as his/her own control to minimize the effects of circadian variation and other interpersonal variations in baseline HRV.

Of note, this study did not look for change in respiratory rate with the intervention. This is a limitation of the study because the HF component of HRV is known to be influenced by respiration, and it has been suggested that studies looking at HF changes should

control the respiratory rate either directly or indirectly. In studying Kundalini Yoga meditation techniques in healthy young adults, Peng and colleagues reported prominent low frequency heart rate oscillations during the meditation that correlated with breathing.[104] The authors concluded that perhaps there is a mechanism involved in slow breathing maneuvers that affects the ANS via stimulation, instead of relaxation. It is certainly possible that Reiki treatment exerted its effects on HRV through slowing of respiratory rate; this would be a useful subject for further investigation.

FUTURE DIRECTIONS

Based on the results presented here, we can identify three important directions for future research. Initially, it would be interesting to conduct further similar trials exploring ways to maximize measurable physiologic and psychological effects of this therapy – altering variables like frequency of treatment, duration of Reiki session, and practitioner expertise. Future studies might also look at other HRV parameters in addition to the high frequency component.

A second line of inquiry could investigate a potential mechanism of action through a series of differently controlled trials; each set of control groups would answer a slightly different research question. First, since this study suggests that human interaction may have a greater impact than non-human relaxation, in order to explore whether Reiki treatment has a therapeutic effect beyond that of just human touch, it would be interesting to compare a session of light touch Reiki with an equivalent session of light touch by a person not trained to practice Reiki, or a light massage. To further explore the specific

effect of the human interaction, it would be useful to utilize a control group involving a relaxation therapy like hypnosis or guided imagery that involved a person in the room with, but not touching, the patient. Because respiratory rate is known to have an effect on HRV, and relaxation may reduce respiratory rate, it would be wise to conduct a study exploring relative respiratory rates and corresponding physiologic effects of the intervention compared to a control group instructed to breathe slowly and deeply.

Finally, a third line of future inquiry might explore whether longer-term use of this therapy can improve outcomes. For example, one protocol might involve a daily 20-60 minute treatment (depending on the availability of practitioners) from admission and diagnosis of MI through discharge, with followup to assess improvement in HF HRV as well as pyschological measures of anxiety and depression. A further study might look at inpatient treatment with or without ongoing treatment in the outpatient setting during the cardiac rehabilitation phase. Given that the foundation of Reiki treatment is thought to be self-treatment, it would also be very interesting to train a group of post-MI patients in First Degree Reiki and study the effects of their daily self-practice on clinical outcomes.

In summary, this initial study could be thought of as administering a single dose of a beta-blocker; if Reiki treatment could in fact provide a non-pharmacologic way to increase recovery of heart rate variability recovery in a cardiac population, future work will require study of long-term, repeated “dosing” of Reiki, perhaps in the outpatient cardiac rehabilitation setting, to see if this benefit stabilizes with repeated treatment, and what, if any, other benefits accompany long term improvement in HRV.

CONCLUSIONS

In conclusion, this study suggests that a 20-minute session of light touch by a Reiki-trained practitioner increases vagal activity and decreases anxiety in cardiac inpatients shortly after ACS. Both the physiological and psychological effects of this treatment appear to be greater than those of either baseline standard of care, a resting control setting, or a relaxing music intervention. Based on growing understanding of the associations between emotion and cardiovascular outcomes, Reiki treatment may represent a non-pharmacologic approach to improving the high frequency component of heart rate variability, an indicator of parasympathetic activity that has important prognostic implications in the setting of myocardial infarction. Furthermore, Reiki treatment appears to be a feasible, safe, potentially cost-effective support to conventional medical care. Further study is warranted to determine whether longer-term use of this therapy can improve outcomes, and through what mechanism it might do so.

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TABLES

Table 1: Pilot Study: Autonomic and Psychological Changes During Reiki (N = 11)

Variable	*Baseline*	*Reiki*	*p-value*
Heart Rate Variability			
Max RR interval (ms)	853	932	0.004
ln HF	5.02	5.51	0.003
Psychological Measures (avg score)*			
Tense	2.00	1.27	0.04
Scared	2.09	1.36	0.02
Relaxed	2.82	4.59	0.007
Happy	2.45	3.23	0.06

ln HF = natural log of high frequency component of heart rate variability
* For the pilot study, emotional state was assessed using a 5-point Likert scale

Table 2. Baseline Demographic and Clinical Characteristics (N = 47)

Variable	*Resting Control (n = 15)*	*Music (n = 15)*	*Reiki (n = 17)*	*p-value*
Age (avg)	62.13± 3.54	60.47±3.54	58.35±3.33	0.74
Sex (% male)	80.00	80.00	64.71	0.52
Race (% non-white)	6.67	13.34	17.64	0.37
MI Area (% AWMI)	26.67	14.29	35.29	0.73
EF (%)	52.69±3.78	49.64±3.64	51.33±3.51	0.84
Peak Troponin	69.11±50.43	53.13±50.43	118.47±47.37	0.61
Beta blocker use	86.67	80.00	82.35	0.88
Statin use (%)	93.33	86.67	94.12	0.73
ACE-I use (%)	33.33	46.67	35.29	0.72
HTN (%)	46.67	66.67	64.71	0.47
Prior MI (%)	13.33	33.33	17.65	0.20
Current Smoker (%)	53.33	46.67	29.41	0.36
Hyperlipidemia (%)	40.00	53.33	58.82	0.55
Diabetes (%)	26.67	20.00	35.29	0.62
CAM use (%)	15.38	33.33	41.18	0.29

Values are Mean ± SE unless otherwise indicated
AWMI = Anterior wall myocardial infarction
EF = Ejection fraction
ACE-I = Angiotensin converting enzyme inhibitor
CAM = Complementary and alternative medicine

Table 3. Baseline Physiological Measures (N = 47)

Variable	*Resting Control (n = 15)*	*Music (n = 15)*	*Reiki (n = 17)*
R-R interval (ms)	874	904	857
SBP (mm Hg)	117.00	116.45	116.00
DBP (mm Hg)	65.83	70.27	61.78
Ln HF	4.55±0.36	4.80±0.33	3.98±0.43

SBP = systolic blood pressure DBP = diastolic blood pressure

Table 4. Change in High Frequency HRV with Intervention (N = 37)

	Control (n = 12)	*Music (n = 13)*	*Reiki (n = 13)*	*p-value*
Baseline	4.549	4.795	3.983	
Intervention	4.613	4.695	4.560	
Avg Chang	0.06±.0.16	-0.1±0.16	0.58±0.16	0.02*

P value for Reiki vs. Control = 0.025
P value for Reiki vs. Music = 0.007
P value for Music vs. Control = 0.5

Table 5. Change in Physiological Parameters with Intervention (N = 47)

Variable	*Resting Control (n = 15)*	*Music (n = 15)*	*Reiki (n = 17)*	*P*	*p-value C vs. M*	*p-value M vs. R*	*p-value R vs. C*
R-R (msec)	41.40±11.3	0.22±10.9	46.49±11.3	0.01*	0.01*	0.002*	0.79
SBP (mm Hg)	0.11±3.0	-4.0±2.4	0.53±2.3	0.35	0.32	0.2	0.9
DBP (mm Hg)	-2.78±2.5	0.57±2.0	1.27±1.9	0.42	0.34	0.8	0.19

R-R = R-R interval (inverse of heart rate)
SBP = systolic blood pressure
DBP = diastolic blood pressure
C = resting control
M = music control
R = Reiki

Table 6. Change in Emotional State by Likert Scale with Intervention (N = 47)

Emotion	*Resting Control (n = 15)*	*Music (n = 15)*	*Reiki (n = 17)*	*P*	*P C vs. M*	*P M vs. R*	*P R vs. C*
Stressed	0.5 ±0.6	-1.07 ±0.6	-2.09 ±0.5	0.01*	0.11	0.17	0.005*
Angry	0.35 ±0.6	-0.96 ±0.5	-1.29 ±0.5	0.08	0.04*	0.68	0.046*
Sad	-0.38 ±0.7	-0.64 ±0.7	-2.00 ±0.6	0.18	0.79	0.17	0.08
Frustrated	-1.15 ±0.6	-1.50 ±0.5	-2.82 ±0.5	0.06	0.49	0.11	0.04*
Worried	-1.46 ±0.6	-2.14 ±0.6	-2.91 ±0.6	0.25	0.43	0.37	0.11
Scared	-0.88 ±0.7	-0.54 ±0.6	-2.47 ±0.6	0.07	0.67	0.06	0.07
Anxious	0.54 ±0.8	-0.39 ±0.8	-1.91 ±0.7	0.07	0.41	0.13	0.04*
Relaxed	0.5 ±0.7	1.71 ±0.7	3.03 ±0.6	0.03*	0.18	0.21	0.005*
Calm	-0.19 ±0.5	1.36 ±0.4	1.76 ±0.4	0.008	0.02*	0.56	0.001*
Happy	-0.31 ±0.6	1.11 ±0.6	1.79 ±0.6	0.055	0.09	0.47	0.01*

Value = Mean Change Score (from 10-point Likert scale) ± SE
C = resting control, M = music control, R = Reiki

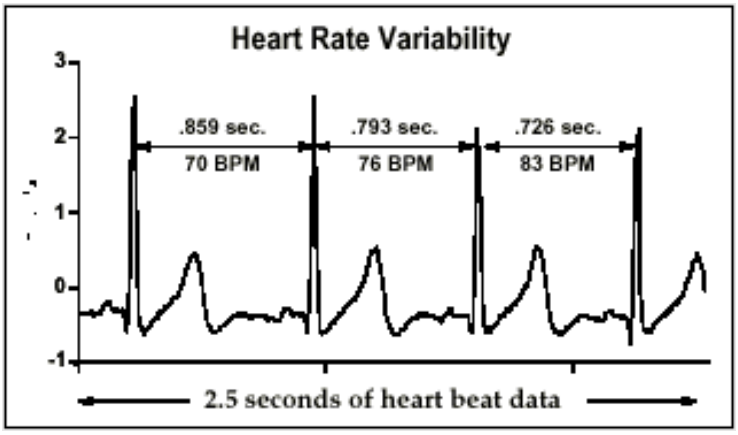
Table 7. Impact of Clinical and Demographic Factors on HRV

Factor	*Change*	*P value*
Gender		0.7
Men	0.15±0.12	
Women	0.22±0.19	
Diabetes		0.7
Yes	0.09	
No	0.20	

Factor	R^2	*P value*
Prior CAM Use		0.28
Yes	0.01	
No	0.25	
Age	-0.004	0.32
Peak Troponin	0.1	0.03
Baseline HF HRV	0.31	0.0086

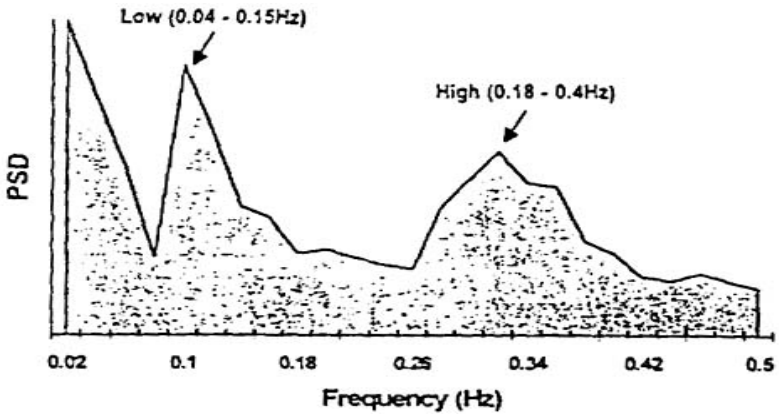
FIGURES

Figure 1. Heart rate variability is analyzed as a time series of beat-to-beat (RR) intervals.



Resting heart rate (HR) varies widely in different individuals. Both the basic HR and its modulation are primarily due to alterations in autonomic tone, parasympathetic or vagal tone slowing the HR, and sympathetic stimulation increasing HR.

Figure 2. Power spectral density (PSD) analysis of heart rate variability.



Power in the high frequency range (HF) (0.15 to 0.4 Hz), representing quicker changes in heart rate, is primarily due to parasympathetic activity.

Figure 3. Study Randomization Protocol

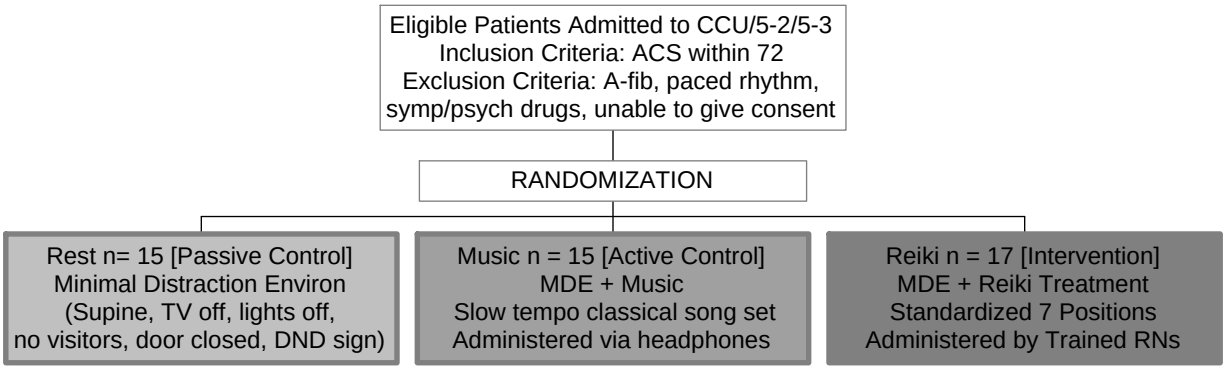


Figure 4. Likert scale for emotional state administered to subjects at the end of the baseline and intervention phases.

Instructions: Please name the number from 1-10 that describes the extent to which you are feeling each of the following emotions right now, where 1 = "I am not feeling that emotion at all right now" and 10 = "I am feeling that emotion extremely strongly right now". Note that some of the emotions are positive and some are negative – please consider each emotion separately, as the numbers are not intended to describe a spectrum of opposite emotions, but rather the intensity of each individual state.

Stressed ___

Calm ___

Angry ___

Sad ___

Frustrated ___

Anxious ___

Worried ___

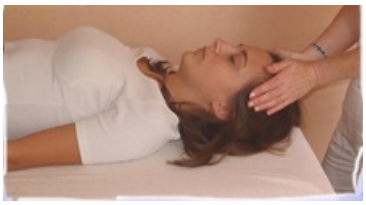
Relaxed ___

Scared ___

Happy ___

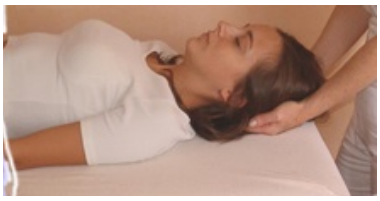
Figure 5. Reiki Study Protocol Hand Positions

1. Crown of the Head



2. Eyes

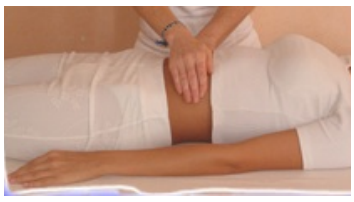
3. Back of Head



4. Upper Chest

5. Solar Plexus

6. Navel



7. Lower Abdomen at Iliac Crests (hands placed laterally for patient comfort)

Practitioners applied light touch with their hands at the indicated positions using Reiki techniques acquired during training. None of the torso hand positions in this study involved direct skin contact, as all patients wore hospital gowns covered by a sheet or blanket.

Figure 6. Data Acquisition.

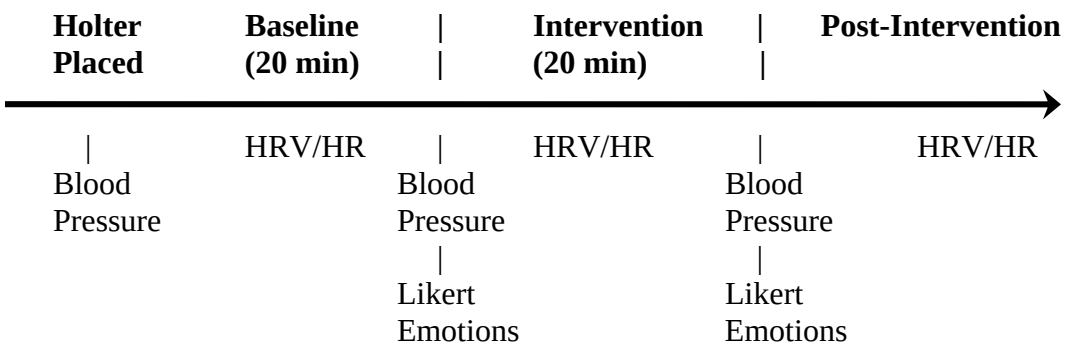


Figure 7: Patient Flow Through Study

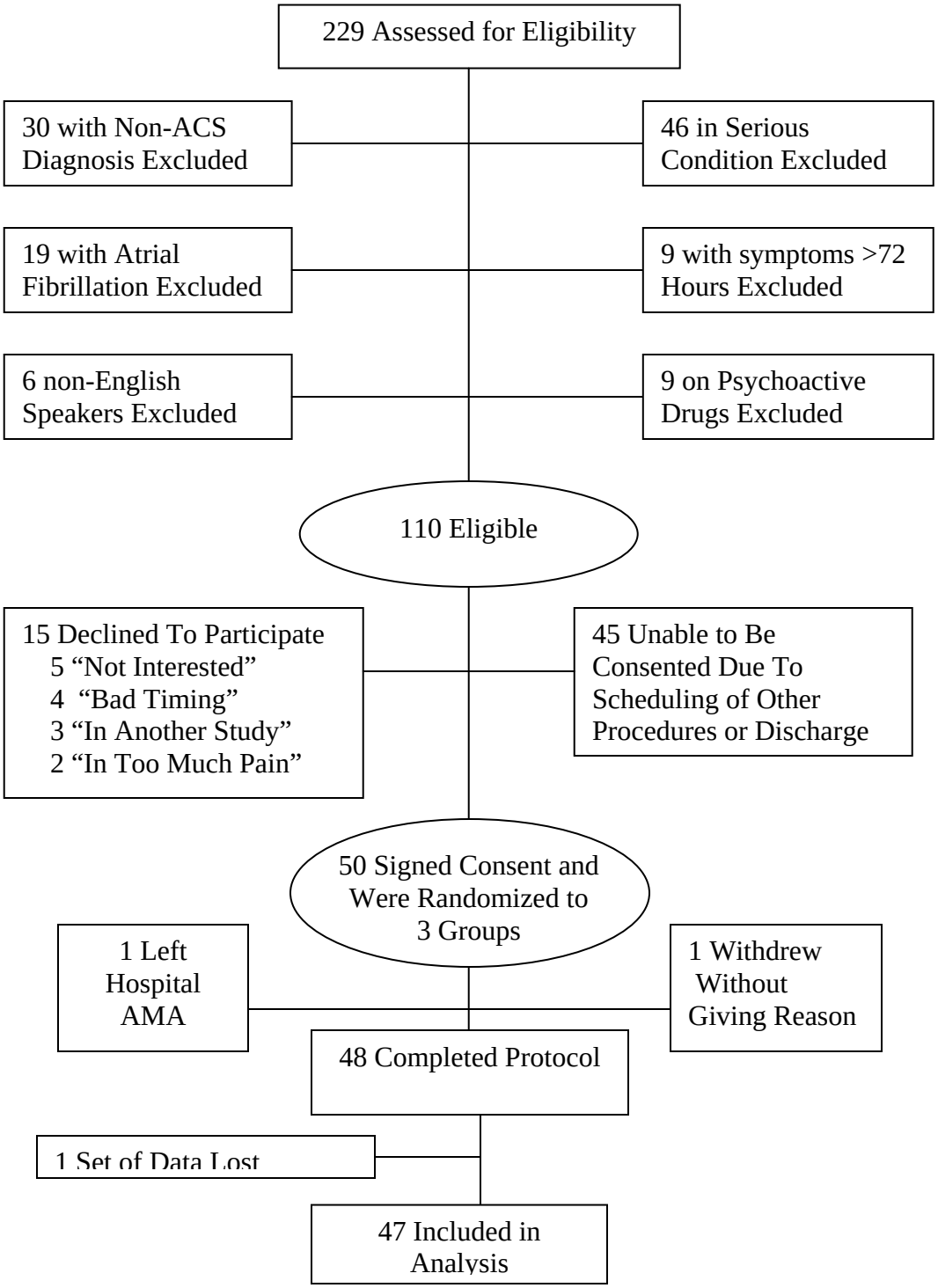


Figure 8. Effect of Intervention on HF HRV

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p = 0.01

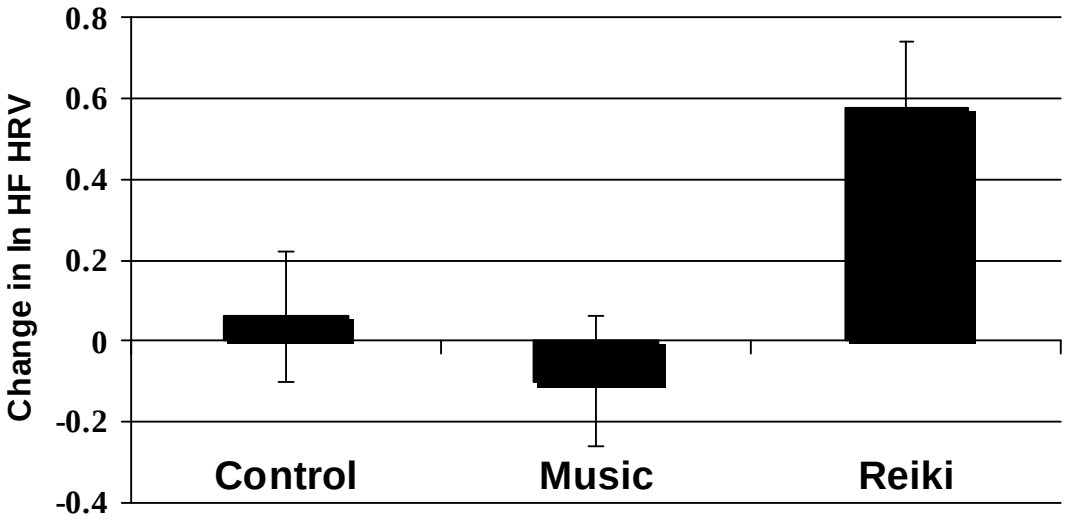

Figure 9. Effect of Intervention on Heart Rate

P= 0.01

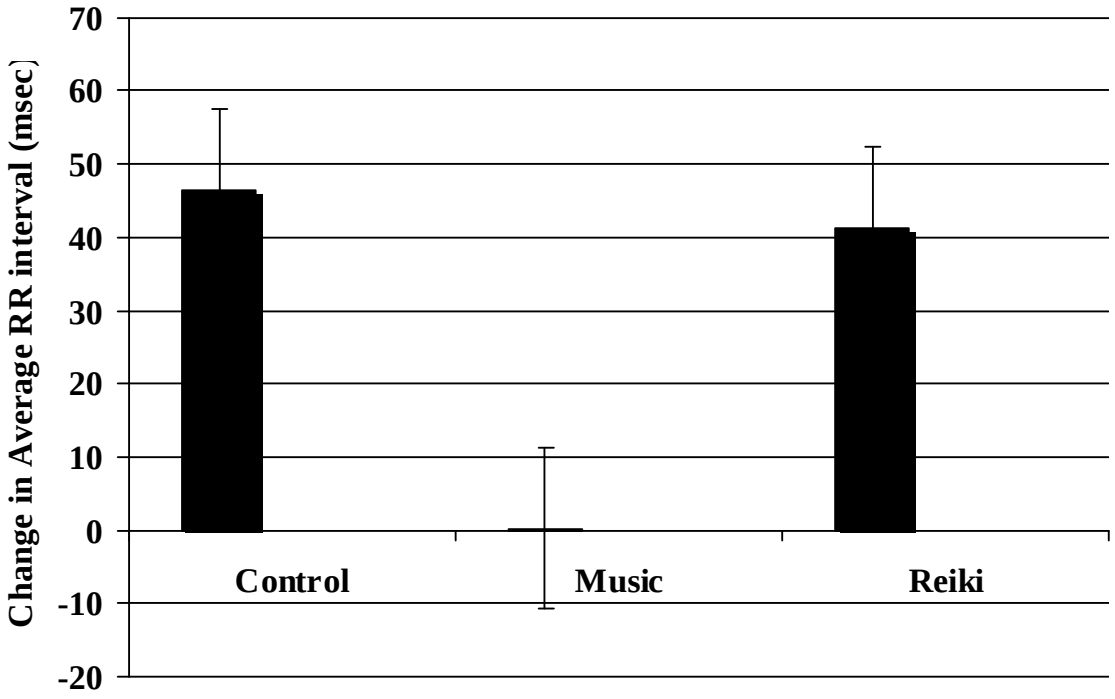

Figure 10. Negative Emotional State Change With Intervention

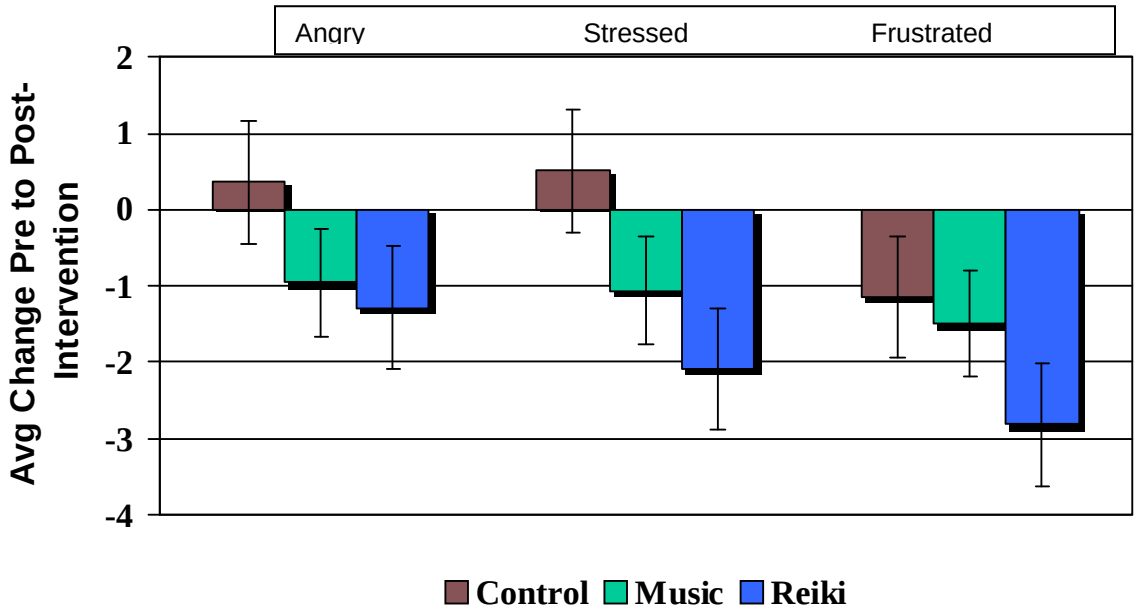

Figure 11. Positive Emotional State Change with Intervention

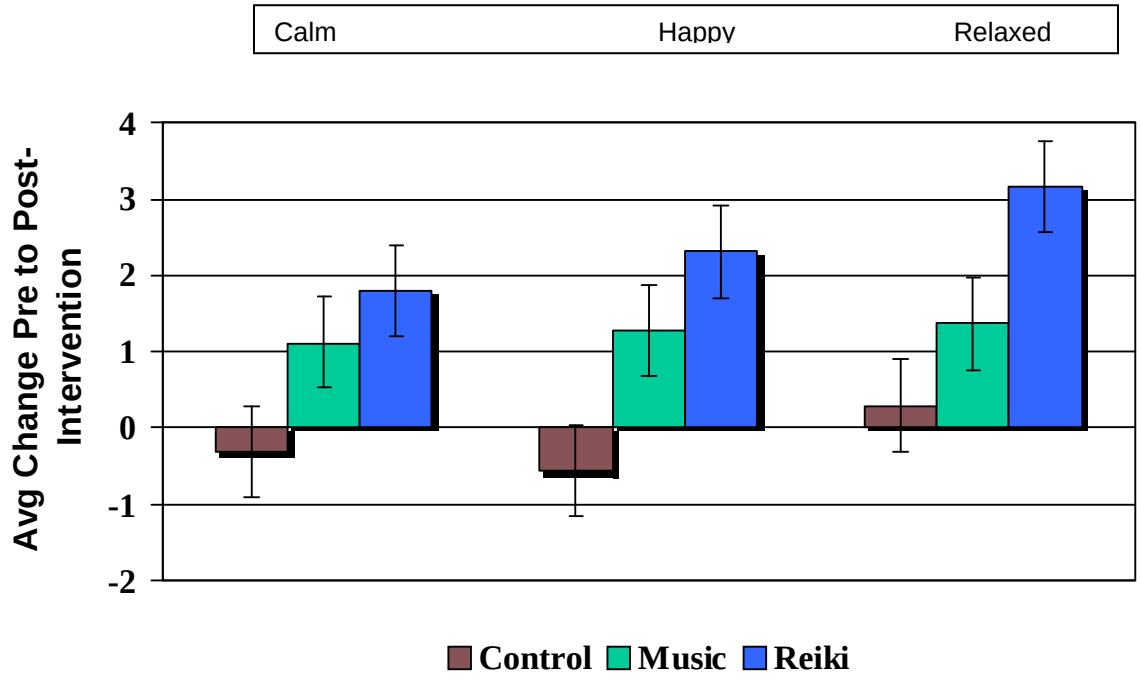

Figure 12. Stratified Analysis of HF HRV Changes with Intervention