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The effects of a forgiveness intervention on patients with coronary artery disease

Martina A. Waltmanab, Douglas C. Russellec, Catherine T. Coylea,
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This research assesses the effects of a psychology of forgiveness pilot study on anger-recall stress induced changes in myocardial perfusion, forgiveness and related variables. Thirty-two patients were administered baseline rest and anger-recall stress imaging studies, and 17 of these participants who demonstrated anger-recall stress induced myocardial perfusion defects (forgiveness group, n = 9; control group, n = 8) were randomly assigned to a series of 10 weekly interpersonal forgiveness or control therapy sessions with a trained psychologist, and underwent additional anger-recall stress myocardial perfusion nuclear imaging studies post-test and at 10-week follow-up. Patients assigned to the forgiveness group showed significantly fewer anger-recall induced myocardial perfusion defects from pre-test to the 10-week follow-up as well as significantly greater gains in forgiveness from pre-test to post-test and from pre-test to follow-up compared to the control group. Forgiveness intervention may be an effective means of reducing anger-induced myocardial ischemia in patients with coronary artery disease.

Keywords: forgiveness; anger-recall; mental stress; coronary artery disease; myocardial ischemia; myocardial perfusion imaging

Introduction

The psychology of forgiveness has emerged as a popular area of study within the past decade. For example, edited volumes exploring the multi-faceted aspects of the construct have begun to appear (see, e.g. Enright & North, 1998; McCullough, Pargament, & Thoresen, 2000; and Worthington, 2005). Interventions with randomised experimental and control groups have shown that forgiveness can have positive effects on emotional health (see, e.g. Colye & Enright, 1997, Freedman & Enright, 1996; Harris et al., 2006; Reed & Enright, 2006; Rye et al., 2005). In most of these cases, those who are in forgiveness therapy reduce anger, anxiety and/or psychological depression, compared to those in control groups, and maintain these effects through follow-up.

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There is now consensus in the field of psychology that forgiveness is not the same as making excuses for unfair treatment, condoning, ignoring justice or reconciling. Those who forgive, in other words, acknowledge the other person’s wrong without excusing or simply letting the offense go. Forgiveness is a person’s individual act of offering mercy, compassion, and empathy towards an offender. Reconciliation is the negotiated resolution of conflict between two or more people. Thus, one might, under certain circumstances, forgive an offender but then not reconcile if that offender continues in his or her hurtful behavioral patterns. Worthington and Wade (1999) make a distinction between forgiveness and reducing unforgiveness (i.e. eliminating resentment without the added compassion towards an offender). The study here focuses on the more complete concept of forgiveness.

Over the past decade, several studies have begun to explore not only the relationship between forgiving an offender and psychological health but also between forgiveness and physical well-being. For example, Huang and Enright (2000) found a relationship between participants’ level of forgiveness and blood pressure when recalling an incident of deep unfairness from another person. The relation between forgiveness and blood pressure, hemodynamic responses and other indicators of physical health have also been reported by Carson, Keefe, and Goli (2005), Lawler et al. (2003) and Witvliet, Ludwig, and Vander Laan (2001). Harris and Thoresen (2005) make a distinction between the study of forgiveness in the context of physiological effects and in the context of actual health benefits. As an example of the latter, Lawler et al. (2003) report correlations between degree of forgiveness and a host of physical variables such as the number of medications used in the past month, quality of sleep and somatic complaints.

The studies to date indicate a general relationship between degree of forgiveness and physical health. These are important findings primarily because they are the first to establish such a relationship between the psychology of forgiveness and both physiological variables and physical health. Perhaps it is now time to extend this line of work to explore the possibility of promoting physical health through forgiveness. If so, which variable may be appropriate for an initial investigation? We chose cardiac health as the central dependent variable (along with forgiveness) within this intervention pilot study primarily because of the research associating both excessive anger with forgiveness (Huang & Enright, 2000; Reed & Enright, 2006; Lawler et al., 2003) and excessive anger with coronary disease. For example, an association between intense emotions, such as anger, and risk for cardiac events is well-established (Farmer & Gotto, 1992; Fava, Abraham, Pava, & Rosenbaum, 1996; Fredrickson et al., 2000; Mendes de Leon & Meesters, 1991; Moser & Dracup, 1996; Siegman, Townsend, Blumenthal, Sorkin, & Civilek, 1998). Numerous studies have demonstrated a variety of cardiovascular responses to mental and emotional stress in healthy normal subjects (Fang & Meyers, 2001; Fichera & Andreassi, 2000; Gallo, Smith, & Kircher, 2000; Harrison et al., 2000; Palomba, Sarlo, Angrilli, & Stegagno, 2000; Pifer & Lawler, 2000; Waldstein, Bachen, & Manuck, 1997) and patients with coronary artery disease (CAD) (Burg, Jain, Soufer, Kerns, & Zaret, 1993; Ironson et al., 1992; Jain, Burg, Soufer, & Zaret, 1995; Jain, et al., 1998, 2001; Kaufman et al., 1998; Kop et al., 2001; Schöder et al., 2000; Stone et al., 1999). Anger especially can trigger or increase vulnerability to serious clinical events, including myocardial ischemia, coronary thrombosis, myocardial infarction and sudden arrhythmic cardiac death in CAD patients (Mittleman, Maclure, & Sherwood, 1995; Reich, SeSilva, Lown, & Murawski, 1981; Verrier & Mittleman, 1996).

Physiological mechanisms mediating these effects include increased cardiac sympathetic activation, transient cardiac sympatho-vagal imbalance leading to cardiac
electrophysiological instability, increased circulating catecholamines, corticosteroids and other endocrine factors (Kop et al., 2001; Mittleman et al. 1995; Reich et al., 1981; Schöder et al., 2000; Stone et al., 1999; Verrier & Mittleman, 1996). Associated increases in heart rate and blood pressure, and increases in systemic and coronary vascular tone, lead to myocardial ischemia through a mismatch of increased myocardial metabolic demand and decreased myocardial perfusion (Kop et al., 2001; Schöder et al., 2000).

Mental stressors such as anger recall, public speaking, Stroop colour-word testing and mental arithmetic can induce myocardial ischemia in patients with CAD (Schöder et al., 2000; Stone et al., 1999; Sheps et al., 2002). Furthermore, patients with mental stress induced myocardial ischemia are at increased risk of daily life myocardial ischemia and of subsequent cardiac events over a three year period (Stone et al., 1999; Sheps et al., 2002). Anger-recall can result in myocardial perfusion defects on nuclear imaging and myocardial ischemic induced reductions in left ventricular ejection fraction (LVEF) (Burg et al., 1993; Ironson et al., 1992; Jain, et al., 1998, 2001). It is not known, however, whether such anger induced effects can be modified by a psychological forgiveness intervention. The objective of the present study was to evaluate the effectiveness of a forgiveness program on anger-recall stress induced myocardial perfusion defects, using $^{99m}$Tc-Tetrofosmin psychological stress SPECT imaging, on associated anger-recall induced haemodynamic responses contributing to myocardial ischemia, and on modification of psychometric profiles in patients with CAD.

Method

Participants

The study was conducted with enrollment of US Veteran patients at a veteran’s hospital in a Midwestern city. The study protocol was approved by the Institutional Review Board and the study was conducted in accordance with the university and hospital ethics standards. Written informed consent was obtained from all patients.

Participants for the study were males between 21 and 79 years of age with documented reversible myocardial ischemia by previous exercise tolerance test, exercise or pharmacological stress imaging study (by echocardiography, gated blood pool or myocardial perfusion nuclear imaging), or by angiographic findings of at least one major coronary vessel with >50% reduction in vessel diameter, or left ventricular ejection fraction ≤40%. Patients were required to identify a specific, deep and unresolved psychological injury at a structured screening interview. Exclusion criteria included coronary artery bypass graft surgery, percutaneous coronary intervention or prior myocardial ischemic event within six months, diabetes mellitus with known or suspected autonomic neuropathy, contraindications to discontinuation of anti-anginal medications prior to psychological stress testing, and underlying medical and psychiatric conditions that might interfere with giving informed consent or co-operating with the study.

Of 218 patients who met the initial inclusion criteria, 75 male patients agreed to participate and gave written informed consent. Sixty-seven patients entered the protocol; 28 of these did not qualify by psychometric measures and three withdrew after completion of the initial questionnaires. Of the remaining 36 patients, 32 completed the baseline rest and anger-recall stress imaging studies. Eighteen of these patients demonstrated anger-induced myocardial perfusion defects on stress nuclear imaging and one patient withdrew after completing these tests. Therefore, the remaining 17 participants who demonstrated anger-induced myocardial perfusion defects were randomised to
a psychological intervention program: nine were assigned to the forgiveness program and eight to the control program. One patient in the forgiveness program was not able to undergo the 10-week follow-up tests.

Because of randomisation, we expected no differences between the groups, and thus a two-tailed t-test was performed, showing no significant between-group differences for mean age, mean myocardial perfusion defects during anger recall stress testing and the psychological measures prior to entering the psychological intervention program (means and standard deviations are in Table 1).

**Measures**

Three measures are central to this investigation: forgiveness, myocardial perfusion imaging and level of anger.

**Enright Forgiveness Inventory**

The Enright Forgiveness Inventory (EFI) is a self-report measure of interpersonal forgiveness. Participants are instructed to respond in terms of a specific interpersonal injury which they have identified as painful and still unresolved. The EFI consists of 60 items divided equally among six subscales: positive affect, negative affect, positive behaviour, negative behaviour, positive cognition and negative cognition. Respondents indicate on a six point Likert scale the degree to which they agree or disagree with a specific statement. The range of scores is 60–360, with a high score representing a high level of forgiveness. The Cronbach’s alpha of internal consistency for the full scale was

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**Table 1. Means and standard deviations for dependent variables.**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Pre-test $(n=9)$</th>
<th>Post-test $(n=9)$</th>
<th>10-week follow-up $(n=8)$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Forgiveness program participants</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger-recall stress responses</td>
<td>5.5 (2.1)</td>
<td>3.7 (2.7)</td>
<td>2.4 (4.2)</td>
</tr>
<tr>
<td>Myocardial perfusion defect increase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(% left ventricular myocardium)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychosocial scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree of forgiveness (range 60–360)</td>
<td>189.2 (53.1)</td>
<td>262.1 (72.9)</td>
<td>248.5 (69.1)</td>
</tr>
<tr>
<td>State of anger (range 10–40)</td>
<td>24.9 (8.7)</td>
<td>14.9 (7.8)</td>
<td>15.0 (8.5)</td>
</tr>
<tr>
<td>Trait anger (range 10–40)</td>
<td>27.3 (5.4)</td>
<td>23.0 (6.0)</td>
<td>25.6 (7.4)</td>
</tr>
<tr>
<td><strong>Aiding coping strategies program participants</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger-recall stress responses</td>
<td>1.6 (4.8)</td>
<td>2.9 (6.2)</td>
<td>3.3 (5.1)</td>
</tr>
<tr>
<td>Myocardial perfusion defect increase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(% left ventricular myocardium)</td>
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<td></td>
</tr>
<tr>
<td>Psychosocial scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree of forgiveness (range 60–360)</td>
<td>169.6 (62.3)</td>
<td>184.9 (86.0)</td>
<td>178.8 (80.9)</td>
</tr>
<tr>
<td>State anger (range 10–40)</td>
<td>20.5 (7.1)</td>
<td>18.6 (7.1)</td>
<td>16.9 (7.1)</td>
</tr>
<tr>
<td>Trait anger (range 10–40)</td>
<td>23.1 (6.5)</td>
<td>23.0 (4.5)</td>
<td>22.1 (4.0)</td>
</tr>
</tbody>
</table>
calculated for all participants at pre-test with a value of 0.98, consistent with previously published intervention research (Lin, Mack, Enright, Krahn, & Baskin, 2004; Subkoviak, Enright, Wu, & Gassin, 1995; Reed & Enright, 2006).

Myocardial perfusion imaging

Single photon emission computer tomographic (SPECT) myocardial perfusion radionuclide imaging was used to assess myocardial perfusion at rest and following anger-recall mental stress testing. The imaging procedure was similar to that employed for routine diagnostic exercise or pharmacological stress imaging for patients with suspected coronary artery disease and demonstrable stress inducible myocardial perfusion defects, with substitution of the mental stress task for the more commonly employed physiological stress. All studies were performed in a dedicated Nuclear Cardiology Laboratory. Medications, including beta-adrenoceptor blocking agents, nitrates and calcium channel blocking drugs, which might influence study findings, were withheld for up to five plasma clearance half-lives prior to imaging, conforming to standard clinical practice for stress testing. An initial resting pre-psychological stress imaging was performed after i.v. injection of 22 mCi (if <70 kg weight) or 33 mCi (if weight 70–127 kg) of the radionuclide $^{99m}$Tc-Tetrofosmin, which has the property of being taken up by myocardium in proportion to regional myocardial blood flow at the time of injection. The pre-test mental stress imaging procedure was performed one week later, and was repeated post-test and at 10-week follow-up (with changes from pre-test to follow-up as our primary outcome), using an identical protocol, but with addition of the anger-recall task prior to radionuclide injection. All stress imaging data were compared with pre-test rest imaging data. Associated changes in heart rate and systemic arterial blood pressure were recorded.

Myocardial imaging data were reconstructed into vertical long, horizontal long and short-axis myocardial slices, using an AutoQUANT program (ADAC Laboratories, 1999). Data were generated for extent of myocardial perfusion defects expressed as percent total myocardial mass. Perfusion defects were defined as areas of percent reduction in myocardial tracer uptake lower than two standard deviations below mean values in a normal subject data set in each of 20 myocardial segments. Imaging data were assessed by an investigating physician blinded to the patients’ intervention-group assignments.

Spielberger state-trait anger expression inventory

The Spielberger state-trait anger expression inventory (STAXI) is a self-report scale measuring the intensity state anger, trait anger and anger expression. For the anger state and trait scales, respondents indicate the intensity of their feelings on a four point Likert scale. The range of possible scores on each of these two subscales is 10–40. High scores on the scale reflect a high degree of anger experience. Internal consistency reliability in this study was as follows: State Anger = 0.93, Trait Anger = 0.83.

Procedure

Anger-recall mental stress testing

Anger-recall mental stress testing was administered by a trained psychologist, and required the active recall by the study participants of a prior specific event causing deep emotional hurt (Burg et al., 1993; Ironson et al., 1992; Jain et al. 1995; Jain et al., 2001; Waldstein et al., 2000). Participants were asked to discuss in detail an incident in which they felt
deeply hurt by another person, which made them feel angry, frustrated, agitated or irritated, and were encouraged to recreate the event from beginning to end. This mental stress testing was performed in the nuclear cardiology laboratory, with monitoring of heart rate and blood pressure responses before and after completion of the mental stress task, and with provision for radionuclide injection by a nuclear cardiology technician on completion of the mental task.

Inclusion criteria
Study participants were interviewed initially to ensure they could identify a specific hurt triggering anger on recall, and were administered the STAXI (Spielberger, 1988; Spielberger, Jacobs, Russell, & Crane, 1983; Spielberger et al., 1985; Spielberger, Reheiser, and Sydeman, 1995) and the EFI (Subkoviak et al., 1995). Participants proceeded in the study only if they had elevated STAXI scores (>20% above the norms of two subscales or >30% above the norm on one of three subscales) and <75% of the maximum EFI score.

Baseline 99mTc-Tetrofosmin myocardial perfusion single-photon emission computed tomography (SPECT) imaging then was performed. One week later, the stress imaging procedure was performed with the addition of the anger-recall task prior to tracer injection.

Only participants who demonstrated anger/resentment-induced myocardial perfusion defects on stress imaging proceeded to further study. These participants were randomised to either a 10-week forgiveness program or control intervention (aiding coping strategies) using a blocking procedure to ensure similar distributions of the anger variable between the intervention and control groups. Psychometric testing and anger-recall stress imaging were repeated after completion of the program and at a 10-week follow-up visit.

Interventions
The forgiveness program was based on a forgiveness process model integrating cognitive, affective and behavioural components (Enright, 1996, 2001; Enright & Fitzgibbons, 2000). The program comprises four phases in which 20 psychological variables are identified (Enright, 2001), such as confrontation and release of anger, willingness to consider forgiveness as a possibility for change, viewing the offender in his context and finding meaning in the painful event. This form of intervention has been shown to significantly increase hope and self-esteem and to decrease anger, anxiety and grief (Al-Mabuk, Enright, & Cardis, 1995; Coyle & Enright, 1997; Freedman & Enright, 1996; Hebl & Enright, 1993; Lin et al., 2004; Baskin & Enright, 2004; Huang & Enright, 2000).

In most existing therapies, including cognitive behavioural therapy (CBT), the therapeutic emphasis is on the client’s cognitive states and behavioural relationships that have led the person to therapy. In contrast, forgiveness therapy (FT) includes in its focus the offender as the client engages in both cognitive and affective work to enlarge his perception of the injury and of the wrongdoer. This wider cognitive perspective in FT is accomplished as the injured expands his view of the wrongdoer by incorporating the historical background as well as the psychological and environmental factors that may have contributed to the wrongdoing. Following this cognitive reframing, FT diverges from CBT with its facilitation of new affective states toward the offender, such as empathy and compassion, as they begin to emerge in therapy (Greenberg, 2002).
Another distinguishing feature of FT is its clarification of the differences among such concepts as forgiveness, excusing and reconciliation. Thus, one who genuinely forgives: (a) understands that he or she was treated unfairly, (b) chooses to offer mercy, compassion, and empathy to the wrongdoer and (c) realises that reconciliation ultimately depends on the trustworthiness of the offender. Only when the injured person clearly understands such issues can he freely choose forgiveness and avoid the risks inherent in pseudo-reconciliation with an abusive wrongdoer.

The control program was based on aiding coping strategies without deliberate intent to reduce anger and aimed at helping to make life, in spite of being diagnosed with CAD, fulfilling. Both programs consisted of 10 weekly individual meetings of 60 min. For each program a manual was used describing the topics to be discussed for each meeting.

Results
Because we had hypotheses expecting differences between the treatment groups, we performed one-tailed, gain-score t-tests on each dependent measure between the experimental and control groups. The analysis was run as several between–group t-tests while considering the inflated type I error rate with multiple comparisons given full alpha. Each conceptually independent set of comparisons shared an alpha of 0.05 using the Holm method. The type I error rate is controlled for each outcome.

The use of gain scores analysis is considered ‘sufficiently reliable for research purposes’ when certain conditions – such as high pre-test reliability – are met (Williams & Zimmerman, 1996; Zimmerman & Williams, 1998, p. 350). Furthermore, the t-test is a sufficient test statistic to use in this instance since we are examining simple gain scores between two independent samples (Field, 2005; Keppel, 1991). The statistical procedure employed here followed precedent in previously published forgiveness interventions (Freedman & Enright, 1996; Lin et al., 2004; Reed & Enright, 2006).

Between-group comparisons
First, as described in the ‘Participants’ section, two-tailed t-tests were conducted between the experimental and control groups at pre-test-only for each variable in Table 1. Two-tailed tests were indicated, rather than one-tailed, because we could not hypothesise which of the two groups, randomly assigned, would be higher on any outcome variable. None of the comparisons were statistically significant.

Table 2 describes the findings between experimental and control groups from both pre-test to post-test and from pre-test to the 10-week follow-up for the dependent measures. One-tailed, t-test gain score analyses indicated that mental stress-induced myocardial perfusion defect showed a statistically significant decrease from pre-test to the 10-week follow-up for the forgiveness condition relative to the control condition, with a strong effect size ($d = -1.01$, 95% C.I. of $-1.98$ to $-0.08$). We did not hypothesise, and therefore did not test pre-test to post-test differences for this cardiac measure, because the effects were hypothesised to be gradual. Because of the small sample size, we wanted to minimise the number of analyses and it seemed most reasonable to do a pre-test to follow-up only analysis of the cardiac variable because long-term gains are far more important than short-term gains that fade with time.

The participants in the forgiveness intervention group demonstrated a statistically significant increase in degree of forgiveness compared to the control group participants at
both testing times, with strong effect sizes from pre-test to post-test \((d = 1.09, 95\% \text{ C.I. of } 0.0 - 2.05)\) and from pre-test to follow-up \((d = 1.12, 95\% \text{ C.I. of } 0.02 - 2.11)\).

Participants in the forgiveness group also demonstrated statistically significant reduction in state anger from pre-test to post-test when compared with the control group, again with a strong effect size \((d = -1.21, 95\% \text{ C.I. of } -2.18 \text{ to } -0.12)\). This reduction appeared to diminish by follow-up. The same pattern was held for the forgiveness condition relative to the control condition in trait anger from pre-test to post-test; however, the pre-test to post-test difference was not statistically significant while controlling type I error rate at 0.05 across multiple comparisons on the same measure. For the between-group comparison, in each case of statistical significance, the effect sizes are strong by Cohen’s (1988) criteria.

**Case example**

Figure 1 shows a typical experimental group patient’s three-dimensional myocardial perfusion images at the three testing times. The images give a powerful image of the physiological changes induced by mental stress (shown as changes between the baseline and pre-test images during mental stress testing), and that amelioration of responses can be seen, at least qualitatively, following intervention (shown as changes between pre-test, post-test and follow-up images during mental stress testing). The three-dimensional surface rendered images (lower row) show the distribution of myocardial blood flow within the heart, as viewed from the interventricular septum, the large black area representing an area of greatest reduction of blood flow. In the baseline image, this is probably caused by a previous heart attack, with loss of heart muscle tissue in this area. The increased extent of these changes following mental stress represents further reduced

![Figure 1](image_url)

Figure 1. Representative 99mTc-Tetrofosmin polar plots and three-dimensional perfusion images at baseline and during anger-recall before, immediately after and 10 weeks after intervention in a patient in the forgiveness program.
blood flow in the areas surrounding this injured region. The post-test images show less marked extension of the black colour-coded region of severe blood flow reduction compared with the pre-test rest and stress images, implying reduction of the stress induced effect. We did not repeat the rest images each time, assuming this would be unchanged in the short period of the study. The bull’s eye pictures (upper row) give the same information, although the anatomic information is displayed (like a polar map of the world in two dimensions), with the apex of the heart represented in the middle, the anterior wall above, the septum to the left, lateral wall to the right and inferior wall below. Haemodynamic responses of heart rate, blood pressure and double product changes to mental stress testing, which increase myocardial metabolic demand, were also ameliorated following intervention.

Discussion

Based on the previous correlational studies (such as Carson et al., 2005; Huang & Enright, 2000; Lawler et al., 2005; Witvliet et al., 2001), we expected patients within the experimental group to show significant reductions in myocardial perfusion defect. This turned out to be the case. The experimental and control groups began the study within the forgiveness range expected for a clinical sample (Lin et al., 2004; Reed & Enright, 2006). At post-test and follow-up, only the experimental group showed a mean forgiveness score that is comparable to other successful forgiveness treatments (again, see Lin et al., 2004; Reed & Enright, 2006).

As patients within the experimental group learned to forgive someone who hurt them deeply, they showed a significant change in the percent of left ventricular myocardial perfusion defect during anger recall compared to those in the control group. This effect was significant over the course of 10 weeks. We did not test how quickly this difference appeared, but we suspect from the descriptive data in Table 1 that it was a gradual process. With the present design, we cannot determine whether the forgiveness intervention alone or the forgiveness intervention plus time accounted for these significant results. By way of analogy, physical exercise does not necessarily have an immediate impact on a person and the delayed effects can last longer than the exercise program itself. In the same way, forgiveness may have an impact beyond the time of the intervention. This would be another area to be addressed in a larger study.

The idea of a refresher course seems reasonable given the results of the study. Both state and trait anger did not show differences in the pre-test to follow-up comparison, though state anger showed differences from pre-test to post-test. In the case of state anger, it is not that the experimental group went back to baseline levels of anger. As the means in Table 1 show, the control group continued to decrease (non-significantly) until they were within the range of the experimental group means at follow-up. It appears that the control group participants may have begun to change in their anger levels, even though this was not to a statistically significant degree, to cancel the between-group effects at follow-up.

Despite the observed statistical convergence of state anger between the two groups at follow-up, two important patterns emerge (trait anger has the same pattern, but the pre-test–post-test difference is not statistically significant): the degree of forgiveness continued to be statistically significant between the two groups from pre-test to follow-up and the myocardial perfusion defect improvements favoured the experimental group. This may be the case because forgiveness not only reduces negative affect but also increases positive affect towards that person. The combination of decreased negative and increased positive
Table 2. Mean, standard deviation, *p*-value, and effect size for between-group gain score analysis.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Change scores from pre- to post-test</th>
<th>Change scores from pre-test to follow-up</th>
<th>Cohen’s <em>d</em></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Forgiveness <em>(n = 9)</em></td>
<td>Aiding coping <em>(n = 8)</em></td>
<td><em>p</em>-value&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Anger-recall stress responses</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Myocardial perfusion defect increase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(% left ventricular myocardium)</td>
<td>−1.9 (2.7)</td>
<td>1.5 (7.9)</td>
<td>Not tested</td>
</tr>
<tr>
<td></td>
<td>−3.4 (4.5)</td>
<td>1.6 (5.4)</td>
<td>0.03&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Psychosocial scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree of forgiveness (range 60–360)</td>
<td>72.9 (46.2)</td>
<td>15.3 (59.1)</td>
<td>0.02&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>61.5 (37.4)</td>
<td>9.1 (54.3)</td>
<td>0.02&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>State anger (range 10–40)</td>
<td>−10.0 (6.6)</td>
<td>−1.9 (6.8)</td>
<td>0.01&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>−9.3 (9.1)</td>
<td>−3.6 (7.4)</td>
<td>0.09</td>
</tr>
<tr>
<td>Trait anger (range 10–40)</td>
<td>−4.3 (5.1)</td>
<td>−0.1 (3.4)</td>
<td>0.03</td>
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<tr>
<td></td>
<td>−1.1 (8.2)</td>
<td>−1.0 (4.5)</td>
<td>0.49</td>
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<sup>a</sup>*Significant at family-wise type I error controlled at 0.05 using the Holm method for multiple tests on the same measure.*
affect may be a key to the physiological improvements (Seligman & Csikszentmihalyi, 2000).

The myocardial perfusion defect parameter was of change in, or the development of, a new myocardial perfusion defect following the anger-recall test, representing a mental stress induced reduction in myocardial blood flow, presumably by coronary vasoconstriction. Myocardial ischemia results from an imbalance in myocardial blood flow supply to the heart (reduced by coronary vasoconstriction, or by reduced diastolic filling time with rapid heart rates, but increased by elevated arterial blood pressure), and myocardial metabolic demand (increased with increased heart rate and blood pressure, and modified by associated stress induced adrenergic stimulation and increased free fatty acid utilisation which influence mitochondrial high-energy phosphate production), thus both factors are important clinically. Although the percent in the volume of heart muscle showing stress induced reversible perfusion defects was small (mean approximately 5% in the forgiveness group and 1.6% in the control group), and not enough to cause clinical symptoms at the time of test, this was, however, superimposed on a background of a wide spectrum of other more fixed perfusion defects (in many participants from previous heart attacks).

Taken together (stress induced myocardial blood flow supply/metabolic demand imbalance) the mental stress induced changes could be enough to cause clinical symptoms of ischemia, particularly in patients with underlying severe coronary artery disease. As one example of a small change having important consequences, even an increase of heart rate of five beats per minute can cause angina symptoms in a patient with a 95% or greater coronary blockage and refractory angina symptoms. Similar changes might not cause symptoms in patients with less severe coronary disease. It seems that patients most likely to benefit from the intervention are those with the most severe coronary disease, in whom seemingly small stressful events might provoke myocardial ischemia. Further, many patients have so-called ‘silent ischemia,’ without overt symptoms, but still are at increased risk of death and coronary events due to asymptomatic induced ischemia. In summary, although the changes we found with intervention were small (in absolute terms), it can be argued that changes of this magnitude could be of clinical importance in patients with severe coronary disease, who are the ones at most risk of sudden cardiac death, arrhythmias and heart attack, or other coronary events.

Several physiological mechanisms have been proposed to explain the association between psychological responses such as anger or resentment and myocardial ischemic events. These include stress mediated sympathetic activation with elevations in heart rate, blood pressure, and systemic and coronary artery vasoconstriction, increase in circulating catecholamines, catecholamine induced increases in platelet aggregability, and increased risk of atherosclerotic plaque rupture and thrombosis (Hevey, McGee, Fitzgerald, & Horgan, 2000; Stoney & Engerbretson, 2000; McEwen, 1998). Our strategy of stress-reduction by reducing resentment and enhancing positive feelings, thoughts and behaviours through forgiveness may exert an anti-ischemic or an anti-anginal effect by both decreasing myocardial metabolic demand by virtue of reducing anger-induced increases in double-product (the product of heart rate and systolic blood pressure, which reflects myocardial oxygen consumption), and improving myocardial blood supply, by reducing anger-stress related coronary vasoconstriction. The persistence of haemodynamic benefit at 10 weeks following completion of the program indicates that this may exert not only a short-term effect on acute stress responses, but also a longer term effect, which potentially could ameliorate effects of ongoing anger-stress on progression of atherosclerotic heart disease with consequent effects on patient morbidity and mortality.
The persistence of the physiological effects of the forgiveness program may relate to its added psychosocial effects. The experimental program focused on reducing resentment and fostering more positive attitudes through forgiveness therapy. Central in this process is a decision made by the injured individual to change his current problem-solving strategy and to choose a path that involves transformation towards positive attitudes rather than simply venting feelings of anger. Several studies have indicated that catharsis tends to increase emotions rather than reducing them (Bushman, 2002; Goldstein, 1999; Kelly, Klusas, von Weiss, & Kenny, 2001; Kennedy-Moore & Watson, 2001; and Siegman & Snow, 1997). The expression of emotions can be helpful if connected with gaining insights and finding meaning, motivation to change and learn, and a means to move towards productive coping strategies (Siegman & Snow, 1997; and Bohart, 2001). As the injured individual becomes more aware of his emotional and cognitive responses to the injury, he may come to see the offender in a broader way and, as he does so, his negative thoughts, feelings and behaviours are replaced with more positive ones (Enright & Fitzgibbons, 2000).

Although this is the first experimental study to show the effect of forgiveness on cardiac functioning, we do not wish to leave the impression that psychological variables are the essential cause of cardiac health. On the contrary, there are myriad variables, physical, psychological and environmental, that interplay in complex ways for each individual. As a pilot study, the findings here are worthy of a more large-scale investigation.

Given that the control treatment is focused on alternative coping strategies, it is not altogether unexpected that the two groups were similar in anger from pre-test to follow-up. Hebl and Enright (1993) provided evidence that two different group interventions can both be effective, one of which was a forgiveness group and the other a support group. A Harvard Heart Letter (‘Support groups with heart’, 2006) noted the value of such support groups particularly for heart patients. Consonance of findings between experimental and alternative treatment groups is also more likely when the forgiveness intervention is done in a group rather than an individual format (Baskin & Enright, 2004). Nonetheless, on the important variables of myocardial perfusion defect and forgiveness, the forgiveness group out-performed its counterpart.

**Limitations of the study**

The strength of our study lies in the highly selected group of subjects with CAD, high anger scores and documented anger-induced myocardial perfusion defects on nuclear imaging. The possibility of intervention effects being unrelated to the forgiveness program was minimised by the use of a randomised control comparison strategy. The prospective nature of the study and blinding of data interpretation mitigated potential observer bias.

The limitations of this study included the small number of patients finally studied, and the risk of generalising findings from a select group of veteran patients to all patients with CAD. We do not think that it is warranted at this time to consider generalising the findings to other samples of patients with cardiac disease. Some patient self-selection may have played a role. It is possible that individuals with the highest anger declined while more cooperative, less angry, patients agreed to participate. In addition, the laboratory setting of the study may have influenced the efficacy of the anger-recall task. Participants were asked to recall the hurtful event, identified during the initial screening interview in the psychologist’s office, again in the laboratory while connected to monitoring equipment.
and in the presence of laboratory technicians. This condition, even when technicians were out of earshot, could have inhibited the expression of feelings about a painful memory and hence made it difficult to obtain optimal anger responses. Conversely, the lab experience could have intensified participants’ negative emotions which would suggest that forgiveness therapy is an effective means to improve such emotions.

**Future directions**

The next step is to move the research to a full-scale study with a larger sample size, to allow for greater statistical power and more analyses. Of course, cost is a central issue because of the expense involved in assessing cardiac health as was done here. A key to a future study is the number and timing of assessments, which we suggest be pre-test, 10-week, 6-month and 1-year follow ups. This would allow for a more careful examination of the interplay of the forgiveness intervention and time regarding the myocardial perfusion imaging variable.

**Conclusion**

In conclusion, this is the first study to demonstrate the beneficial effects of a psychological program enhancing a forgiving attitude on stress-induced haemodynamic responses which can cause myocardial ischemia in patients with CAD. As mental stress, and especially resentment or anger, can trigger or increase vulnerability to myocardial ischemia and myocardial infarction in patients with CAD, evaluation of the more widespread use of forgiveness programs should warrant further investigation. We consider that encouraging a short-term psychological pilot intervention helped patients to increase forgiveness, which affected cardiovascular responses to stress in a positive way. These positive changes were compellingly shown 10 weeks after completing the program. Our findings support the implementation of forgiveness therapy in the field of cardiac rehabilitation as an alternate or even a supplemental method to existing therapeutic approaches.

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**References**


