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BRIEF REPORT

Impact of negative affectivity and trait forgiveness on aortic blood pressure and coronary circulation

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Abstract

Prior research suggests that negative affectivity (NA) may have a direct adverse effect on coronary circulation, whereas forgiveness may provide cardioprotection. This study examined whether NA and forgiveness were independently related to aortic hemodynamics and the subendocardial viability index (SVI), a marker of coronary perfusion. A sample of 131 adults ($M = 21.11$ years, $SD = 2.52$) were evaluated for NA (depression, anxiety, and anger symptoms) and forgiveness (Tendency to Forgive Scale; TTF). Aortic hemodynamic parameters via applanation tonometry were assessed at rest and during sympathostimulation (cold pressor test; CPT). Hierarchical multiple regression analyses of resting values showed that NA was related to higher aortic blood pressure (ABP) and lower SVI. After controlling for demographics and for NA, TTF scores were significantly associated with decreased ABP, but increased SVI. CPT changes from baseline indicated that, after controlling for demographics and NA, TTF scores were significantly associated with SVI. Results indicate that NA significantly predicts ABP and decreased SVI. Conversely, forgiveness seems to provide cardioprotection by evoking decreased ABP while improving SVI.

Descriptors: Aortic hemodynamics, Blood pressure, Coronary artery disease, Forgiveness, Negative affect, Subendocardial viability index

The association between psychological risk factors (e.g., depression, anxiety, anger) and coronary artery disease (CAD) has gained considerable medical attention (Haynes, Feinleib, & Kannel, 1980; Hoen, Denollet, de Jonge, & Whooley, 2013; Lichtman et al., 2014; Suls, 2013). In general, individuals with increased psychological risk factors display a tendency to perceive emotions as negative (negative affectivity), manifest maladaptive alterations in cardiovascular functioning, and are at increased risk for CAD (Almeida et al., 2012; Bajko et al., 2012; Chida & Steptoe, 2009; Lichtman et al., 2014; Suls & Bunde, 2005; Williams et al., 2000). Some of the mechanisms that may explain the link between negative affective symptoms and CAD could include autonomic dysfunction, inflammation, and impaired vascular function (Bajko et al., 2012; Betensky & Contrada, 2010; Epel, 2009; Sanchez-Gonzalez, May, Koutnik, Kabbaj, & Fincham, 2013). Moreover, it appears that negative affectivity may also have a direct adverse effect on central (aortic) hemodynamics and the coronary circulation (Seldenrijk et al., 2011; Vaccarino et al., 2009). However, studies examining the impact of negative affectivity, and more importantly the relative contribution of its main components (depression, anxiety, and anger), on aortic hemodynamics and the coronary circulation are lacking.

The noninvasive assessment of aortic hemodynamics is easily performed by means of radial artery applanation tonometry. A pencil-like device (tonometer) is placed on top of the radial artery (wrist) in a way that allows for the collection of blood pressure waveforms. This technique allows for evaluation of aortic blood pressure, which is considered a superior predictor of cardiovascular risk compared to peripheral (brachial) blood pressure, and the evaluation of noninvasive surrogates of coronary perfusion adequacy and coronary flow reserve, namely, the subendocardial viability index (SVI; Bunckberg, Fixler, Archie, & Hoffman, 1972; May, Sanchez-Gonzalez, Hawkins, Batchelor, & Fincham, 2014; Roman et al., 2007). The SVI comprises the ratio between the systolic pressure-time index (STI) and the diastolic pressure-time index, which are surrogates of left ventricular work and coronary perfusion, respectively (Bunckberg et al., 1972; Gobel, Norstrom, Nelson, Jorgensen, & Wang, 1978; Hoffman & Buckberg, 1978). Recently, Held et al. (2013) documented that the SVI is decreased in patients with acute depression. Seldenrijk et al. (2011) demonstrated associations between depressive and anxiety symptoms with altered aortic hemodynamic markers. We demonstrated an association between anger and markers of aortic hemodynamics including central blood pressure (BP) and systolic pressure-time index, a

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surrogate of ventricular work (May et al., 2014). In addition, individuals high in depressive symptoms displayed increased aortic pressure and lower SVI during sympathetic stimulation, by means of the cold pressor test (CPT), compared to those low in such symptoms (Sanchez-Gonzalez, May, Brown, Koutnik, & Fincham, 2013). Considered together, these studies suggest that negative affectivity may increase BP as well as ventricular work and ultimately decrease coronary blood flow. It is important to note that some cardiovascular anomalies may be detected during sympathetic stimulation using applanation tonometry, and hence we have adopted its use in the present study to screen out potential anomalies (Sanchez-Gonzalez, May, Brown et al., 2013).

As opposed to the cardiotoxic effects associated with psychological risk factors promoting negative affectivity, psychological strengths such as trait forgiveness (TF—a potential marker of psychological well-being) appear to be associated with improved cardiovascular functioning in CAD patients (Brown, 2003a; Friedberg, Suchday, & Srinivas, 2009). There is evidence to show that interventions promoting forgiveness decrease brachial BP and increase coronary flow reserve in CAD patients (Tibbits, Ellis, Piramelli, Luskin, & Lukman, 2006; Waltman et al., 2009). In addition, May et al. (2014) demonstrated that TF is associated with decreased aortic BP and increased SVI in healthy young females, suggesting that TF may provide cardioprotection and may even prevent the development of cardiovascular disease (CVD). In view of the fact that many patients with CAD display high negative affectivity and that forgiveness may ameliorate adverse cardiovascular functioning in this patient population, it is clinically relevant to evaluate the relative contributions of negative affect and forgiveness to cardiovascular functioning.

Accordingly, the aim of the present study was to document the impact of negative affectivity (i.e., depression, anxiety, anger) and TF on aortic hemodynamics at rest and during sympathetic stimulation by means of the CPT. It was hypothesized that negative affect would be associated with higher aortic blood pressure and decreased SVI, both at rest and during sympathetic stimulation, whereas TF would have a cardioprotective effect by ameliorating aortic hemodynamics and increasing SVI at rest and during sympathetic stimulation.

Method

Participants

A total of 131 healthy young adults ($M = 21.11$ years, $SD = 2.52$, 82 females) gave informed consent to be part of the research project as approved by the university's Institutional Review Board. To minimize potential cardiovascular risk confounds, respondents were excluded from study participation by means of an online health screening assessment if they smoked, had a history of psychiatric or mood disorders (major depressive disorder, anxiety disorders), exercised regularly (defined as >120 min per week in the previous 6 months), were hypertensive (blood pressure ≥140/90 mmHg), had major chronic diseases, or were taking beta blockers, antidepressants, or stimulants. Participants were asked to abstain from caffeine, alcohol, and strenuous physical activity for at least 24 h prior to testing and were asked to not eat for 4 h prior to testing. Female participants were tested in the early follicular phase of the menstrual cycle in order to avoid potential variations in pressure wave morphology and cardiovascular functioning (Lustyk, Douglas, Shilling, & Woods, 2012).

Study Design

Participants were first introduced to the laboratory setting and familiarized with the study procedures. Anthropometric measurements (height and weight) were taken followed by the completion of a health questionnaire that included a health history form and psychological scales. Data collection was conducted in the afternoon in a quiet, dimly lit, temperature-controlled room (23 ± 1°C) at the same time of the day (±2 h). Participants were seated and given a 10-min rest period before any measurements were performed. Within 3 min after the rest period, measurements for peripheral brachial blood pressure and applanation tonometry of the radial artery for central aortic hemodynamics were taken. Immediately following the rest measurements, participants completed the CPT by submerging their hand in cold water (4°C) for 3 min in order to evoke SNS stimulation and increased aortic hemodynamics (Casey, Braith, & Pierce, 2008; Sanchez-Gonzalez, May, Brown et al., 2013). During the CPT, a research assistant made sure the participant kept their hand in the water throughout the entire task. Blood pressure and applanation tonometry were obtained between 2 to 3 min into the CPT.

Psychological Scales

Depression. Depressive symptoms were measured using the 10-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977; Santor & Coyne, 1977). The CES-D has been widely used as a measure of depressive symptoms in nonclinical samples. It asks participants to respond to a list of ways they may have felt or behaved during the previous week. Sample items include "I was bothered by things that usually don't bother me," and "I felt hopeful about the future," (reverse coded). Responses ranged from 0 = *rarely or none of the time (less than 1 day)* to 3 = *most or all of the time (5–7 days)*. Responses were summed into one overall score, with a possible range of 0 to 30. Reliability for the sample was $\alpha = .87$.

Anxiety. Anxiety was measured using the 20-item State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970). Participants were asked to respond to anxiety items such as "upset," "calm," "secure," "at ease," and "nervous." Responses were scored on a 4-point Likert scale (1 = *not at all* to 4 = *very much so*). Half of the items were reverse coded so that higher scores indicated greater anxiety. Items were then summed to create a composite anxiety score with a possible range of 20 to 80. Reliability for the sample was $\alpha = .85$.

Anger. The trait subscale of the State-Trait Anger Expression Inventory-2 (STAXI-2) was used to measure trait anger (Spielberger et al., 1999). The trait anger subscale of the STAXI-2 comprises 10 items asking participants to rate how they generally feel and behave on a scale of 1 (*almost never*) to 4 (*almost always*). Reliability for the sample was $\alpha = .81$.

Trait forgiveness. Trait forgiveness was measured using the 4-item Tendency to Forgive Scale (TTF; Brown, 2003b). The TTF asks participants to report how they usually respond when someone offends them. Sample items include "I tend to get over it quickly when someone hurts my feelings," and "I have a tendency to harbor grudges," (reverse coded). Responses ranged from 1 = *completely disagree* to 5 = *completely agree*. Responses were

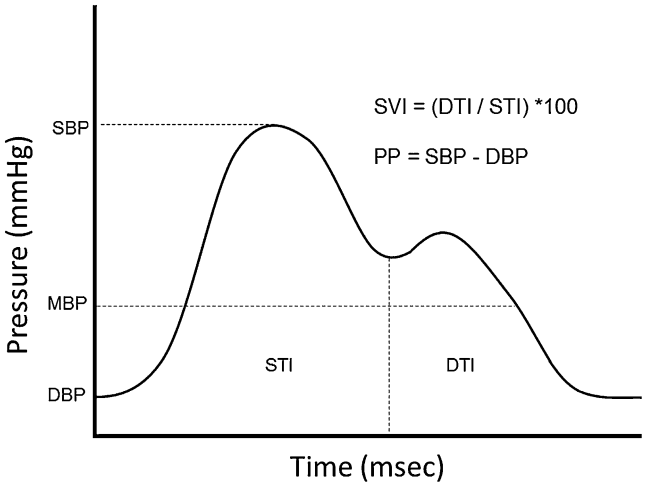

Figure 1. Representative aortic pressure wave including the systolic and diastolic pressure-time intervals.

summed into one overall score, with a possible range of 4 to 20. Reliability for the sample was $\alpha = .91$.

Cardiovascular Measurements

Pulse wave analysis. Indices of vascular function and aortic hemodynamics were obtained using brachial blood pressure and applanation tonometry via pulse wave analysis (PWA), which is defined as the examination of the functioning of the arterial (central) pulse wave, allowing for accurate assessment of central hemodynamic functioning (Nichols & Singh, 2002). Brachial blood pressure was recorded using an automated oscillometric device (HEM-705CP; Omron Healthcare, Vernon Hill, IL). Brachial systolic blood pressure and diastolic blood pressure were used to calibrate radial waveforms obtained from a 10-s epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX). PWA provides more sensitive markers of cardiovascular function than brachial blood pressure (Hashimoto, Imai, & O'Rourke, 2007; Roman et al., 2007). We therefore measured heart rate; brachial systolic, diastolic, and mean arterial blood pressure; brachial pulse pressure (surrogate of arterial stiffness); aortic systolic blood pressure; aortic diastolic blood pressure; aortic mean blood pressure; aortic pulse pressure; systolic pressure-time index (STI; left ventricular work); diastolic pressure-time index (DTI; coronary perfusion); and the ratio of DTI to STI expressed as a percentage (SVI; surrogate of subendocardial blood flow and coronary flow reserve) (Bunckberg et al., 1972; Gobel et al., 1978; Hoffman & Buckberg, 1978; May et al., 2014; Figure 1). All measurements were obtained in duplicate and averaged. Aortic pressure waveforms were derived using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). Only high-quality measurements (>85% operator index) were considered for analysis.

Statistical Analysis

Pearson correlations evaluated measurement scale and health demographic associations (see Table 1). Hierarchical multiple regression (HMR) analyses were conducted to test the relationship between, depression, anxiety, anger, and TF scores with aortic homodynamic parameters at rest (Model 2) while controlling for the health demographics of age and body mass index (BMI; Model 1). Change scores were created (CPT values minus baseline values) and analyzed via HMR to evaluate the associations between negative affect and forgiveness on aortic hemodynamics during CPT (Model 2) while controlling for health demographics (age and BMI) (Model 1). Protection against the inflation of experimentwise Type I errors for the HMR analyses was addressed by requiring the multivariate model test be significant as a precondition for performing tests on individual variables (Cohen, Cohen, West, & Aiken, 2003). SPSS version 20.0 (SPSS Inc., Chicago, IL) was used for all analyses.

Results

Correlations among measurement scales indicated a statistically significant inverse association between forgiveness and both depression and anxiety scores (Table 1). No statistically significant correlations were found between forgiveness scores and health demographics. As expected, all the hemodynamic parameters significantly increased ($p < .01$) from rest values in response to CPT, but the SVI significantly ($p < .01$) decreased (-31.76 ± 15.63) in response to sympathetic stimulation (Table 2).

Aortic Hemodynamics at Rest

Model 1 of the HMR indicated that age ($\beta = .17$) and BMI ($\beta = .18$) were significant predictors for only one cardiovascular parameter (increased systolic blood pressures) at rest ($p < .05$). No other cardiovascular parameters were significantly associated with Model 1 predictors. In regards to negative affectivity, Model 2 of the HMR analyses of resting values showed that, after accounting for demographics (BMI and age) and controlling for forgiveness scores, depression scores were significantly associated with higher brachial systolic blood pressure, brachial diastolic blood pressure, brachial mean blood pressure, aortic systolic blood pressure, aortic

Table 1. *Correlation Matrices of Demographics and Psychological Status Scales*

Variable	*M* ± *SD*	1	2	3	4	5	6	7	8
1. CES-D	5.94 ± 3.07	1	0.39	0.22*	−0.43***	−0.18*	0.00	0.07	−0.15
2. STAI	31.59 ± 6.87	–	1	0.61***	−0.26**	0.16	0.54***	0.41***	0.02
3. STAXI-2	15.94 ± 1.77	–	–	1	−0.08	−0.01	0.16	0.16	0.01
4. TTF	12.77 ± 1.55	–	–	–	1	0.01	−0.02	−0.07	0.01
5. Height (cm)	179.18 ± 7.43	–	–	–	–	1	0.20*	−0.32***	0.11
6. Weight (kg)	79.59 ± 12.31	–	–	–	–	–	1	0.86***	0.61***
7. BMI (kg/m^2)	24.85 ± 3.95	–	–	–	–	–	–	1	0.54***
8. Age (years)	24.00 ± 5.59	–	–	–	–	–	–	–	1

Note. $n = 131$. CES-D = Center for Epidemiologic Studies Depression Scale; STAI = State-Trait Anxiety Inventory; STAXI-2 = State-Trait Anger Expression Inventory-2; TFF = Tendency to Forgive Scale; BMI = body mass index.
*$p < .05$; **$p < .01$; ***$p < .001$.

Table 2. *Aortic Hemodynamic Indices at Rest and During Sympathetic Stimulation*

Variable	Rest	CPT	Δ	95% CI
Heart rate (bpm)	61.92 ± 9.83	66.94 ± 8.53*	5.02 ± 2.83	[4.16–5.88]
Brachial systolic blood pressure (mmHg)	112.76 ± 6.15	139.00 ± 13.32*	26.23 ± 12.15	[24.17–28.30]
Brachial diastolic blood pressure (mmHg)	69.35 ± 8.03	89.76 ± 12.69*	20.41 ± 9.21	[18.85–21.98]
Brachial pulse pressure (mmHg)	43.41 ± 6.45	49.23 ± 9.09*	6.81 ± 2.88	[21.67–25.15]
Brachial mean blood pressure (mmHg)	82.65 ± 7.02	106.06 ± 13.19*	23.01 ± 10.07	[25.39–30.02]
Aortic systolic blood pressure (mmHg)	96.86 ± 6.05	124.59 ± 14.94*	27.80 ± 13.67	[19.09–22.23]
Aortic diastolic blood pressure (mmHg)	70.34 ± 8.11	91.00 ± 12.89*	20.65 ± 9.27	[21.67–25.15]
Aortic pulse pressure (mmHg)	26.54 ± 4.81	33.59 ± 7.93*	7.22 ± 4.81	[19.09–22.23]
Aortic mean blood pressure (mmHg)	82.65 ± 7.017	106.06 ± 13.19*	23.41 ± 10.27	[21.66–25.15]
STI (mmHg/s.min^{-1})	1548.88 ± 232.01	2197.12 ± 402.39*	648.22 ± 289.40	[599.24–697.73]
DTI (mmHg/s.min^{-1})	3410.72 ± 298.08	4162.71 ± 482.19*	752.21 ± 382.33	[686.24–817.73]
SVI (%)	224.76 ± 35.63	193.53 ± 29.38*	−31.76 ± 15.63	[−35.31–−27.13]

Note. $n = 131$. Model 2 parameter estimates reported. Values are mean ± *SD*. CPT = cold pressor task; HR = heart rate; BSBP = brachial systolic blood pressure; BDBP = brachial diastolic blood pressure; BMAP = brachial mean blood pressure; BPP = brachial pulse pressure; ASBP = aortic systolic blood pressure; ADBP = aortic diastolic blood pressure; AMAP = aortic mean arterial pressure; APP = aortic pulse pressure; STI = systolic pressure-time index; DTI = diastolic pressure-time index; SVI = subendocardial viability index; *sr* = semi-partial correlation.
*$p < .01$ vs. rest.

diastolic blood pressure, aortic mean blood pressure, aortic pulse pressure, and DTI; anxiety scores were also associated with higher heart rate, systolic blood pressure, brachial pulse pressure, aortic pulse pressure, STI, DTI, and lower SVI; anger scores were also associated with higher values of all cardiovascular parameters, but lower SVI. Regarding forgiveness, after accounting for health demographics and controlling for negative affective symptomatology (i.e., depression, anxiety, anger), HMR analyses indicated that forgiveness scores were significantly associated with lower heart rate, brachial systolic blood pressure, aortic systolic blood pressure, STI, but higher SVI (Table 3).

Aortic Hemodynamics in Response to Sympathetic Stimulation

Model 1 of HMR analysis of CPT changes from baseline values indicated that no change scores were significantly associated with Model 1 predictors. Model 2 of the HMR analysis indicated that, after accounting for health demographics and controlling for forgiveness scores, depression scores were significantly associated with a greater increase in all cardiovascular parameters except for SVI where there was significant decrease; anxiety scores with a greater increase in heart rate and brachial pulse pressure, but a decrease in brachial diastolic blood pressure, brachial mean blood pressure, aortic systolic blood pressure, aortic diastolic blood pressure, aortic mean blood pressure, DTI, and SVI; anger scores with a greater increase in all the cardiovascular parameters, except for SVI where there was a significant decrease (see Table 4). Conversely, HMR analysis of CPT changes from baseline values indicated that, after accounting for health demographics and controlling for negative affective symptomatology, forgiveness scores were significantly associated with decreased cardiovascular parameters, but increased DTI and SVI (Table 4).

Discussion

One goal of the present study was to document the association between negative affectivity (depression, anxiety, anger) and poor cardiovascular outcomes via aortic hemodynamics and markers of coronary blood flow. Additionally, TF was examined as a potential cardioprotective factor for improving aortic BP and noninvasive markers of coronary flow. The novelty of the present study is twofold. First, even though negative affectivity was found to be associated with higher aortic BP and lower SVI, TF evoked counteracting effects by predicting lower aortic systolic blood pressure and higher SVI. Second, the effect of TF on SVI is also apparent in response to sympathetic stimulation as demonstrated by the positive association between TF and the change in SVI from rest values to CPT. These results suggest that negative affectivity may trigger and prompt deteriorated coronary circulation, whereas TF may point towards a potential cardioprotective psychological intervention.

As expected, the results of the present study demonstrate that negative affectivity is predictive of higher peripheral and, more importantly, central blood pressures. Although the association between elevated peripheral BP and negative affectivity seems to be apparent, the impact of depression, anxiety, and anger on central blood pressures is less clear (Bajko et al., 2012; Hamang, Eide, Rokne, Nordin, & Oyen, 2011). Recently, we demonstrated that anger is associated with higher aortic systolic blood pressure and lower SVI (May et al., 2014). Interestingly, in the present study, depressive and anger symptoms positively correlated with aortic systolic blood pressure. In addition, anxiety and anger symptoms were significant predictors of lower SVI. Conversely, TF was associated with lower aortic systolic blood pressure and ventricular work (STI), which seems to be in line with a prior study reporting antihypertensive effects of an intervention aimed at increasing TF (Tibbits et al., 2006). It is worth noting that TF predicts lower central blood pressures and STI, which are considered stronger predictors of cardiovascular outcomes when compared to peripheral blood pressures (Hashimoto et al., 2007; Roman et al., 2007). Together, these results suggest that, as opposed to negative affect, forgiveness may positively impact cardiovascular functioning by ameliorating central BP and decreasing ventricular work while promoting adequate coronary blood flow. Hence, it appears that mechanistically forgiveness promotes adequate coronary blood flow by decreasing ventricular work (STI) and central blood pressure, an effect that may be mediated via decreased sympathetic activity (May et al., 2014).

Strong, acute negative emotions are well known to evoke adverse cardiovascular events (Vlastelica, 2008). Although prior research has attempted to elucidate the mechanisms that account for the negative emotion-triggered cardiovascular events, establishing a definitive pathophysiological pathway has remained elusive

Table 3. *Hierarchical Multiple Regression Analyses of Depression, Anxiety, Anger, Forgiveness, and Aortic Hemodynamic Indices at Rest*

Variable	Predictors	β	*sr*	*p* value	Model R^2	Model $F(4,126)$
Heart rate (bpm)	CES-D	0.037	0.340	.641	0.318	$F = 15.29$
	STAI	0.590	0.430	< .001		$p < .001$
	STAXI-2	0.514	0.407	< .001		
	TF	−0.198	−0.190	.009		
Brachial systolic blood pressure (mmHg)	CES-D	0.355	0.327	< .001	0.408	$F = 22.59$
	STAI	0.366	0.267	< .001		$p < .001$
	STAXI-2	0.361	0.285	< .001		
	TF	−0.217	−0.210	.002		
Brachial diastolic blood pressure (mmHg)	CES-D	0.182	0.168	.005	0.544	$F = 39.00$
	STAI	0.104	0.076	.203		$p < .001$
	STAXI-2	0.803	0.635	< .001		
	TF	−0.088	−0.08	.155		
Brachial mean blood pressure (mmHg)	CES-D	0.243	0.224	< .001	0.554	$F = 40.68$
	STAI	0.015	0.011	.850		$p < .001$
	STAXI-2	0.761	0.602	< .001		
	TF	−0.114	−0.110	.062		
Brachial pulse pressure (mmHg)	CES-D	0.110	0.100	.167	0.280	$F = 12.69$
	STAI	0.480	0.350	< .001		$p < .001$
	STAXI-2	0.660	0.520	< .001		
	TF	−0.100	−0.090	.206		
Aortic systolic blood pressure (mmHg)	CES-D	0.413	0.380	< .001	0.538	$F = 38.18$
	STAI	−0.101	−0.100	.105		$p < .001$
	STAXI-2	0.533	0.421	< .001		
	TF	−0.294	0.214	< .001		
Aortic diastolic blood pressure (mmHg)	CES-D	0.187	0.173	.004	0.547	$F = 39.58$
	STAI	0.114	0.083	.158		$p < .001$
	STAXI-2	0.810	0.641	< .001		
	TF	−0.076	−0.07	.214		
Aortic mean blood pressure (mmHg)	CES-D	0.243	0.224	< .001	0.554	$F = 40.68$
	STAI	0.015	0.011	.850		$p < .001$
	STAXI-2	0.761	0.602	< .001		
	TF	−0.144	0.110	.062		
Aortic pulse pressure (mmHg)	CES-D	0.203	0.190	.009	0.340	$F = 16.99$
	STAI	0.563	0.410	< .001		$p < .001$
	STAXI-2	0.700	0.550	< .001		
	TF	−0.002	0.090	.978		
STI (mmHg/s.min^{-1})	CES-D	0.054	0.049	.420	0.514	$F = 34.64$
	STAI	0.404	0.295	< .001		$p < .001$
	STAXI-2	0.793	0.627	< .001		
	TF	−0.288	−0.280	< .001		
DTI (mmHg/s.min^{-1})	CES-D	0.296	0.273	< .001	0.430	$F = 24.75$
	STAI	0.303	0.221	.001		$p < .001$
	STAXI-2	0.452	0.357	< .0001		
	TF	−0.043	−0.040	.534		
SVI (%)	CES-D	−0.093	−0.090	.225	0.356	$F = 18.10$
	STAI	−0.617	−0.450	< .001		$p < .001$
	STAXI-2	−0.529	−0.420	< .001		
	TF	0.238	0.229	.001		

Note. $n = 131$. Model 2 parameter estimates reported. CES-D = Center for Epidemiologic Studies Depression Scale; STAI = State-Trait Anxiety Inventory; STAXI-2 = State-Trait Anger Expression Inventory-2; TF = Tendency to Forgive Scale; BMI = body mass index; HR = heart rate; BSBP = brachial systolic blood pressure; BDBP = brachial diastolic blood pressure; BMAP = brachial mean blood pressure; BPP = brachial pulse pressure; ASBP = aortic systolic blood pressure; ADBP = aortic diastolic blood pressure; AMAP = aortic mean arterial pressure; APP = aortic pulse pressure; STI = systolic pressure-time index; DTI = diastolic pressure-time index; SVI = subendocardial viability index; *sr* = semi-partial correlation.

(Strike et al., 2006; Vlastelica, 2008). However, it appears that activation of the sympathetic nervous system plays a critical role in view of the fact that the majority of cardiac deaths occur during times of augmented sympathetic activity (Fontes et al., 2014; Muller, Tofler, & Verrier, 1995). In the present study, we induced sympathetic stimulation via the CPT as it alters aortic hemodynamics and coronary blood flow offering a window to evaluate cardiovascular functioning (Casey et al., 2008; Momen et al., 2009). Previously, we demonstrated that individuals high in depressive symptoms, compared to those low in such symptoms, display higher ventricular work (STI) in response to the CPT (Sanchez-Gonzalez, May, Brown et al., 2013). In the present study, negative affectivity was associated with lower SVI in response to sympathetic stimulation. Conversely, TF positively correlated with SVI in response to the CPT. These findings seem to suggest that negative affectivity may trigger acute coronary events by decreasing coronary blood flow during periods of sympathetic hyperactivity, but TF may provide a counteracting effect on the coronary circulation. It is worth noting that, among negative affective factors, depression and anger were associated with higher responses to the CPT, whereas anxiety was associated with attenuated responses. It appears that anxiety may attenuate the cardiovascular responses;

Table 4. *Hierarchical Multiple Regression Analyses of Depression, Anxiety, Anger, Forgiveness, and Aortic Hemodynamic Indices in Response to Sympathetic Stimulation*

Variable	Predictors	β	*sr*	*p* value	Model R^2	Model $F(4,126)$
Δ Heart rate (bpm)	CES-D	0.359	0.331	< .001	0.238	$F = 11.52$
	STAI	0.296	0.216	.005		$p < .001$
	STAXI-2	−0.053	−0.042	.577		
	TF	−0.177	−0.113	.135		
Δ Brachial systolic blood pressure (mmHg)	CES-D	0.132	0.122	.099	0.301	$F = 14.10$
	STAI	−0.042	−0.030	.678		$p < .001$
	STAXI-2	0.214	0.168	.023		
	TF	0.466	0.448	< .001		
Δ Brachial diastolic blood pressure (mmHg)	CES-D	0.525	0.484	< .001	0.448	$F = 26.60$
	STAI	−0.396	−0.289	< .001		$p < .001$
	STAXI-2	0.374	0.296	< .001		
	TF	0.363	0.349	< .001		
Δ Brachial mean blood pressure (mmHg)	CES-D	0.324	0.298	< .001	0.315	$F = 15.03$
	STAI	−0.251	−0.183	.013		$p < .001$
	STAXI-2	0.356	0.282	< .001		
	TF	0.368	0.354	< .001		
Δ Brachial pulse pressure (mmHg)	CES-D	0.390	0.360	< .001	0.190	$F = 11.87$
	STAI	0.470	0.280	< .001		$p < .001$
	STAXI-2	0.140	0.080	.270		
	TF	−0.280	−0.270	< .001		
Δ Aortic systolic blood pressure (mmHg)	CES-D	0.235	0.217	.004	0.274	$F = 12.35$
	STAI	−0.229	−0.167	.026		$p < .001$
	STAXI-2	0.311	0.246	.001		
	TF	0.409	0.393	< .001		
Δ Aortic diastolic blood pressure (mmHg)	CES-D	0.508	0.468	< .001	0.404	$F = 22.19$
	STAI	−0.361	−0.264	< .001		$p < .001$
	STAXI-2	0.352	0.278	< .001		
	TF	0.328	0.316	< .001		
Δ Aortic mean blood pressure (mmHg)	CES-D	0.324	0.298	< .001	0.315	$F = 15.03$
	STAI	−0.251	−0.183	.013		$p < .001$
	STAXI-2	0.356	0.282	< .001		
	TF	0.368	0.354	< .001		
Δ Aortic pulse pressure (mmHg)	CES-D	0.210	0.190	.017	0.17	$F = 6.77$
	STAI	0.030	0.020	.780		$p < .001$
	STAXI-2	0.140	0.110	.171		
	TF	−0.36	−0.340	< .001		
Δ STI (mmHg/s.min^{-1})	CES-D	0.397	0.366	< .001	0.244	$F = 11.88$
	STAI	−0.037	−0.027	.719		$p < .001$
	STAXI-2	0.246	0.194	.011		
	TF	0.112	0.107	.153		
Δ DTI (mmHg/s.min^{-1})	CES-D	0.22	0.203	.004	0.368	$F = 19.08$
	STAI	−0.389	−0.284	< .001		$p < .001$
	STAXI-2	0.369	0.292	< .001		
	TF	0.53	0.509	< .001		
ΔSVI (%)	CES-D	−0.144	−0.114	.066	0.325	$F = 15.76$
	STAI	−0.662	−0.483	< .001		$p < .001$
	STAXI-2	−0.44	−0.349	< .001		
	TF	0.207	0.199	.006		

Note. $n = 131$. CES-D = Center for Epidemiologic Studies Depression Scale; STAI = State-Trait Anxiety Inventory; STAXI-2 = State-Trait Anger Expression Inventory-2; TF = Tendency to Forgive Scale; BMI = body mass index; HR = heart rate; BSBP = brachial systolic blood pressure; BDBP = brachial diastolic blood pressure; BMAP = brachial mean blood pressure; BPP = brachial pulse pressure; ASBP = aortic systolic blood pressure; ADBP = aortic diastolic blood pressure; AMAP = aortic mean arterial pressure; APP = aortic pulse pressure; STI = systolic pressure-time index; DTI = diastolic pressure-time index; SVI = subendocardial viability index; RPP = rate pressure product; *sr* = semi-partial correlation.

however, SVI was decreased in responses to sympathetic stimulation, suggesting increased cardiovascular risk. Hence, anxiety may be related to blunted cardiovascular responses to stress, which are equally indicative of deleterious cardiovascular functioning. To the best of our knowledge, the present study is the first to document the relationship between the psychological constructs of negative affectivity and TF and aortic hemodynamics in response to sympathetic stimulation. We speculate that negative affectivity evokes increased ventricular work without concurrent increases in coronary blood flow (DTI). On the other hand, TF seems to attenuate the hemodynamic responses to stress while allowing for adequate blood flow through the coronary circulation. It remains to be determined whether forgiveness improves endothelial cell function.

Notwithstanding its strengths, several issues need to be considered regarding the findings of this study. First, we did not use direct measures of aortic blood pressure and coronary blood flow. However, we evaluated aortic hemodynamics noninvasively via applanation tonometry, which provides reliable validated markers of aortic BP and coronary flow reserve (Roman et al., 2007; Tsiachris et al., 2012). Second, participants were healthy young adults and nonregular exercisers, and thus our results may not generalize to other populations. Third, we did not use emotional stress as the

experimental manipulation but instead evaluated a more neutral stressor (CPT). Finally, there is no data on biochemical markers of endothelial function or coagulation factors that would clarify some of the underlying mechanisms. Nevertheless, there is existing evidence to suggest that negative affectivity is associated with increased coagulation and deteriorated endothelial function (Nabi et al., 2010; Osika et al., 2011; Rozanski, Blumenthal, & Kaplan, 1999).

In sum, the results of this study indicate that negative affectivity (depression, anxiety, and anger) is a strong predictor of aortic systolic blood pressure and decreased SVI. On the other hand, forgiveness seems to provide cardioprotection by evoking decreased aortic systolic blood pressure and STI while improving SVI at rest. It is important to note that the negative affectivity and forgiveness effects each operated independently of the other. In addition, the cardioprotective effects of forgiveness may also be apparent during sympathetic stimulations as TF was positively correlated with SVI in response to the CPT. Prospective studies evaluating the effects of interventions aimed at improving TF for improving cardiovascular functioning and coronary blood flow are warranted.

Conclusion

The findings of the present study point towards the conclusion that promoting forgiveness may be a feasible clinical intervention for patients with CAD. Prospective studies are warranted to determine whether TF may serve as a marker of mental wellness and cardiovascular health in patients with CAD.

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