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Impact of Mental Stress and Anger on Indices of Diastolic Function in Patients with Heart Failure

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Abstract

Background: Under controlled conditions, mental stress can provoke decrements in ventricular function, yet little is known about the effect of mental stress on diastolic function in patients with heart failure (HF).

Methods and Results: Twenty-four HF patients with ischemic cardiomyopathy and reduced ejection fraction (HFrEF; n=23 men; mean LVEF=27±9%; n=13 with baseline elevated E/e') completed daily assessment of perceived stress, anger, and negative emotion for 7 days, followed by a laboratory mental stress protocol. 2D Doppler echocardiography was performed at rest and during sequential anger recall and mental arithmetic tasks to assess indices of diastolic function (E, e', and E/e'). Fourteen patients (63.6%) experienced stress-induced increases in E/e', with an average baseline to stress change of 6.5±9.3, driven primarily by decreases in early LV relaxation (e'). Age-adjusted linear regression revealed an association between 7-day anger and baseline E/e'; patients reporting greater anger in the week prior to mental stress exhibited higher resting LV diastolic pressure.

Conclusions: In patients with HFrEF, mental stress can provoke acute worsening of LV diastolic pressure, and recent anger is associated with worse resting LV diastolic pressure. In patients vulnerable to these effects, repeated stress exposures or experiences of anger may have implications for long-term outcomes.

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Keywords

mental stress; anger; diastolic; heart failure

Mental stress and psychological distress (anger and depression) can negatively impact ventricular function in patients with heart failure (HF) and coronary artery disease (CAD).¹⁻³ Less is known about the impact of mental stress specifically on diastolic function in HF. One small study of idiopathic cardiomyopathy patients demonstrated mental stress-induced increases in early to late peak transmitral flow velocity ratio (E/A) and decreases in e-wave deceleration time.⁴ The impact of mental stress on left ventricular (LV) diastolic filling pressure (E/e' ratio), another measure of diastolic function related to HF mortality, is unknown.⁵⁻⁶ Among HF patients with reduced ejection fraction (HFrEF), the present study therefore determined: (1) the acute effect of mental stress on E/e' and its components – peak early transmitral flow velocity (E) and mitral annular early diastolic velocity (e'); and (2) the association of recent psychological distress with resting values and mental stress-induced changes in these parameters. We hypothesized that mental stress exposure would be associated with acute alterations in diastolic function, and that effects would be greater in patients reporting higher levels of recent distress.

Methods

Participants

A subset of Biobehavioral Triggers in HF (BETRHEART) study participants^{7,8} with symptomatic HF (NYHA II-IV) 3 months due to ischemic cardiomyopathy, history of CAD, and LV ejection fraction (LVEF) 40% within the past year were recruited from the University of Maryland Medical Center. Patients with myocarditis, significant mitral valve disease, current alcohol abuse, LV assist device, active cancer treatment, significant cognitive impairments, or primary HF etiology of thyroid dysfunction were excluded. BETRHEART was approved by Institutional Review Boards at the recruitment site and Uniformed Services University.

Procedures

Consented patients completed 7 daily assessments of perceived stress,⁹ anger,¹⁰ and negative emotion (e.g., depressed, tense),¹¹ with scores averaged (ranges – stress:0-40; anger: 12-60; negative emotion:0-36), after which they underwent a laboratory mental stress protocol. Patients fasted and held morning doses of β -blockers and calcium channel blockers before testing. Baseline echocardiography (ECHO) was performed during a 30-minute rest period. Patients then completed the mental stress procedure – sequential 4-minute anger recall (describe recent anger-provoking incident) and 4-minute mental arithmetic tasks (count backwards by 7 from a 4-digit number while staff provide corrections to improve performance) – during which a repeat ECHO was performed. Patients rated their stress and anger at baseline and during stress on a scale from 1 'none' to 7 'high'.

Transthoracic Doppler ECHO (Vivid Seven: GE-Vingmed Ultrasound AS) was used to determine qualitative 2-dimensional LV systolic function, Doppler transmitral valve inflow

consisting of peak early (E) and late velocity (A), and mitral annular early diastolic velocity (e') measured from the lateral wall. The E/ e' ratio, an estimate of LV filling pressure, was calculated. When only lateral e' is available, E/ e' >13 is considered abnormal and suggestive of diastolic dysfunction.^{5,6,12} Blood pressure (BP) was continually recorded every 2 minutes, with average resting and peak stress values calculated.

Statistical Analyses

Data distribution was evaluated with Kolmogorov-Smirnov statistics and non-parametric tests used for non-normally distributed variables. Patients were grouped based on the change in E/ e' from baseline to mental stress – patients displaying worsened LV diastolic pressure in response to mental stress (i.e., E/ e' increased from baseline to stress), and those with no response (i.e., no change or E/ e' decreased from baseline to stress). Paired samples t-test or Wilcoxon signed-rank tests evaluated within-group differences in baseline and stress values of ventricular function and hemodynamics. Independent samples t-test, Mann-Whitney U test, and chi-squared tests evaluated between-group differences in demographic and clinical characteristics, ventricular function and hemodynamics (baseline, stress, and change values), and 7-day perceived stress, anger, and negative emotion averages. Pearson correlation examined associations of 7-day perceived stress, anger, and negative emotion with baseline and stress-induced changes in diastolic function indices (E, e' , and E/ e'), hemodynamics, and baseline to stress changes in patient-reported stress and anger. Significant correlations with diastolic indices were evaluated further using linear regression controlling for age¹³ with normality of standardized residuals verified by probability plots. SPSS Statistics for Mac v.26 (IBM Corp., USA) was used for analyses, and $p<.05$ used for significance.

Results

Thirty-five patients were enrolled. One patient remained hypertensive at rest and was withdrawn, ECHO was not obtained on 4 patients due to body habitus, and 6 patients did not complete 1 daily psychological assessment, resulting in a sample size of 24. Excluded patients had a higher prevalence of anxiety disorders but did not differ from sample patients in other characteristics. Mean ($\pm$ SD) LVEF was $27\pm 9\%$ and $n=13$ (54.2%) patients exhibited baseline LV diastolic pressures indicative of diastolic dysfunction (lateral E/ e' >13) (see Table 1).¹²

Baseline or mental stress e' was not obtained for two patients. Of the 22 with E/ e' at both assessments, fourteen (63.6%) experienced acute baseline to stress worsening in LV diastolic pressure. Among patients demonstrating this response, average E/ e' change was 6.5 ± 9.3 and driven primarily by reductions in e' , as the E peak increase from baseline to stress was not significant (see Table 2). Baseline ventricular and hemodynamic parameters and demographic and clinical characteristics did not differ between patients with vs. without acute stress-induced worsening in LV diastolic pressure. Similarly, there was no difference in 7-day perceived stress, anger, or negative emotion between groups. Mental stress significantly increased diastolic BP in the group demonstrating an E/ e' increase and systolic BP and heart rate (HR) in both groups.

Seven-day perceived stress, anger, and negative emotion ratings were positively correlated, and perceived stress and negative emotion were each positively correlated with baseline HR (see Table 3). Age-adjusted linear regression revealed an association between 7-day anger and baseline E/e' ($\beta=.53$, $p=.01$; adjusted $R^2=.23$, $p=.03$), whereby patients reporting higher recent anger exhibited worse baseline LV diastolic pressure. Anger was not associated with baseline E or e', nor were perceived stress or negative emotion associated with baseline or stress-induced changes in E, e', or E/e'. Magnitude of baseline to stress change in E, e', and E/e' was not associated with these baseline values, nor with baseline to stress changes in patient-reported anger and stress levels (all $p>.10$).

Discussion

This study demonstrates that under controlled conditions, mental stress can provoke acute worsening of ventricular diastolic function in HFrEF patients, and reports an association of recent anger with baseline LV diastolic pressure. Most patients, including those with and without evidence of resting diastolic dysfunction, experienced decreased early LV relaxation and increased LV diastolic pressure when exposed to mental stress. These patients also had higher systemic vascular resistance and lower stroke volume during stress and exhibited greater stress-induced increases in diastolic BP. Thus, while the exact mechanisms for the maladaptive diastolic response are unknown, it may be due to acute pressure load or be a marker of remodeled myocardium from chronic hypertension. In vulnerable patients, stress exposures during daily living may produce acute decrement in diastolic function. The long-term consequences of these transient effects are unknown. However, this pathway warrants further investigation given that E/e' is an independent predictor of reduced survival in HFrEF,^{5,6} and since mental stress-induced reductions in e' predict major adverse cardiovascular events in CAD patients.¹⁴

Although daily perceived stress, anger, and negative emotion in the week before the study procedure did not predict mental stress-induced diastolic alterations, a novel association was found between 7-day average anger and baseline resting LV diastolic pressure. Previous research has shown a relationship between chronic anger and stiffness of the common carotid artery in healthy men.¹⁵ It is possible that repeated stiffening of conduit arteries among individuals who frequently experience anger may over time, contribute to myocardial remodeling and eventual diastolic dysfunction. Indeed, earlier findings from the BETRHEART study demonstrated that a greater frequency of anger episodes was associated with worse symptom burden and functional status in HFrEF patients.⁸ More work is needed to determine if the present finding is mediated by arterial stiffness.

Strengths of this study include acquisition of ECHO during mental stress allowing for direct comparisons with baseline resting ventricular function, and daily assessments to capture patient's emotional experience during their daily routine. Primary limitations include the small, primarily male sample, and the possibility that baseline ECHO values may not reflect usual resting diastolic function if patients were anxious about the impending mental stress task. Additionally, without a post-stress ECHO we could not determine the prolonged effects of mental stress on diastolic indices though we previously found no lasting effect on LVEF assessed by SPECT myocardial perfusion imaging.⁷

While greater attention is being devoted to understanding the impact of psychological disorders (e.g., depression) on HF symptoms and disease progression, other emotional factors can go unrecognized or be under-addressed. The present findings contribute to the growing literature indicating that mental stress and anger are two such factors with clinical implications in HF. Further research is needed with larger, more diverse samples of HF patients with both reduced and preserved LVEF using invasive hemodynamic monitoring to promote a better understanding of the effect that stress and negative emotion have on ventricular function in HF. This research should determine whether vulnerability differs among HF phenotypes, if the effect is attenuated by cardiovascular medications, and how this effect contributes to disease outcomes.

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Table 1.**Patient Demographic and Clinical Characteristics**

Characteristic	Total Sample (n=24)	Patient groups based on response to mental stress*	
		Increase in E/e' (n=14)	No change or decrease in E/e' (n=8)
Age, years, mean $\pm$ SD	61.2 $\pm$ 9.4	59.9 $\pm$ 9.9	62.3 $\pm$ 9.7
Sex, n (%)			
Male	23 (95.8)	13 (92.9)	8 (100.0)
Female	1 (4.2)	1 (7.1)	0 (0.0)
Race, n (%)			
African American	13 (54.2)	9 (64.3)	3 (37.5)
White	11 (45.8)	5 (35.7)	5 (62.5)
Body mass index, kg/m ² , mean $\pm$ SD	28.6 $\pm$ 6.6	27.5 $\pm$ 6.1	31.6 $\pm$ 7.3
NYHA Class, n (%)			
II	13 (54.2)	7 (50.0)	4 (50.0)
III	11 (45.8)	7 (50.0)	4 (50.0)
Baseline LV diastolic pressure, n (%) *			
Normal, E/e' $\leq$ 13	10 (41.7)	6 (42.9)	4 (50.0)
Abnormal, E/e' > 13	13 (54.2)	8 (57.1)	4 (50.0)
Extent of coronary artery disease, n (%)			
2-vessel	9 (37.5)	5 (35.7)	4 (50.0)
3-vessel	10 (29.2)	4 (28.6)	2 (25.0)
Unknown	8 (33.3)	5 (35.7)	2 (25.0)
Medical and Psychiatric History, n (%)			
Percutaneous coronary intervention	13 (54.2)	7 (50.0)	4 (50.0)
Coronary artery bypass graft surgery	11 (45.8)	7 (50.0)	3 (37.5)
Current or former smoker	20 (83.3)	12 (85.7)	6 (75.0)
Diabetes mellitus	15 (62.5)	9 (64.3)	6 (75.0)
Hypertension	21 (87.5)	13 (92.9)	7 (87.5)
Atrial fibrillation	5 (20.8)	3 (21.4)	2 (25.0)
Chronic obstructive pulmonary disease	3 (12.5)	1 (7.1)	2 (25.0)
Hypercholesterolemia	22 (91.7)	12 (85.7)	8 (100.0)
Valvular disease	2 (8.3)	1 (7.1)	0 (0.0)
Inducible ventricular tachycardia	2 (8.3)	1 (7.1)	0 (0.0)
Anxiety disorder	2 (8.3)	1 (7.1)	1 (12.5)
Mood disorder	4 (16.7)	1 (7.1)	3 (37.5)
Medications, n (%)			
ACE inhibitor	18 (75.0)	11 (78.6)	6 (75.0)
β -blocker	22 (91.7)	13 (92.9)	8 (100.0)
Calcium channel blocker	1 (4.2)	0 (0.0)	1 (12.5)
Vasodilator	5 (20.8)	4 (28.6)	1 (12.5)
Angiotensin II receptor blocker	4 (16.7)	1 (7.1)	2 (25.0)

* Baseline or mental stress e' not obtained for two patients.

Table 2.**Baseline and Mental Stress Echocardiographic and Hemodynamic Parameters**

Parameter	Patient groups based on response to mental stress								
	Increase in E/e' (n=14)			No change or decrease in E/e' (n=8)			P-values		
	Baseline	Stress		Baseline	Stress		Baseline	Stress	
Left Ventricular									
End diastolic volume, mL	182 ± 44	182 ± 45	-0.2 ± 14	212 ± 59	217 ± 64	4 ± 18	.19	.15	.51
End systolic volume, mL	132 ± 42	136 ± 47	4 ± 14	155 ± 60	155 ± 61	-0.6 ± 15	.30	.44	.48
Stroke volume, mL	50 ± 11	46 ± 8	-4 ± 13	56 ± 14	62 ± 11	6 ± 12	.30	.001	.07
Ejection fraction, %	28 ± 8	27 ± 9	-2 ± 7	28 ± 11	31 ± 9	2 ± 6	.98	.30	.17
Cardiac output, L/min	3.2 ± 0.8	3.3 ± 0.8	0.1 ± 1.2	3.7 ± 1.0	4.4 ± 1.4	0.6 ± 0.9	.21	.13	.28
Doppler Indices									
E peak velocity, cm/sec	73 ± 18	76 ± 19	3 ± 12	71 ± 26	63 ± 25 *	-8 ± 9	.87	.20	.04
A peak velocity, cm/sec	52 ± 20	52 ± 26	4 ± 18	56 ± 27	60 ± 23	-1 ± 8	.64	.53	.25
E/A ratio	1.8 ± 1.3	1.8 ± 1.1	-0.1 ± 1.0	2.0 ± 2.0	1.4 ± 1.4	-0.2 ± 0.2	.52	.18	.86
E-wave deceleration time, msec	164 ± 49	168 ± 66	4 ± 60	178 ± 60	204 ± 71 *	26 ± 26	.58	.25	.34
LV outflow tract velocity time interval, cm	14 ± 4	13 ± 3	-0.8 ± 2	15 ± 4	14 ± 4	-0.7 ± 2	.77	.66	.83
Lateral wall e', cm/sec	6 ± 2	5 ± 2 *	-1 ± 1	6 ± 2	6 ± 1	0.4 ± 1	.90	.10	.005
E/e' ratio (E peak velocity/lateral wall e')	14.9 ± 7.4	21.4 ± 12.3 *	6.5 ± 9.3	13.4 ± 6.2	11.2 ± 5.8 *	-2.2 ± 1.4	.62	.02	<.001
Hemodynamics [†]									
Systolic blood pressure, mmHg	126 ± 25	152 ± 24 *	20 ± 23	121 ± 10	138 ± 10 *	17 ± 9	.60	.15	.73
Diastolic blood pressure, mmHg	78 ± 10	94 ± 11 *	16 ± 5	70 ± 8	77 ± 4	7 ± 8	.08	.001	.01
Heart rate, bpm	65 ± 15	76 ± 16 *	12 ± 7	69 ± 14	77 ± 14 *	8 ± 2	.56	.91	.09
Systemic vascular resistance, mmHg·min·mL ⁻¹	32 ± 9	36 ± 11	2 ± 8	26 ± 9	24 ± 7	-2 ± 6	.19	.009	.28
7-day Average Ratings									
Perceived stress	11.7 ± 5.7			9.1 ± 5.2			.31		
Anger	16.0 ± 1.7			15.3 ± 0.8			.44		
Negative emotion	5.7 ± 7.6			1.9 ± 3.2			.19		

All values represent *mean* ± SD; = Stress-baseline change;

* $p < .05$ for within-group paired samples t-tests of baseline and stress values;

[†] Stress values represent the peak value obtained during the anger recall or mental arithmetic stress tasks;

P-values represent independent samples t-test comparisons of baseline, stress, and stress-baseline change between groups.

Table 3.

Pearson Correlations of 7-Day Daily Ratings and Baseline and Stress-Induced Changes in Indices of Diastolic Function

Variables	M ± SD	7-Day Average Ratings				Baseline				Stress-Induced Chanaes			
		Stress	Anger	NE	SBP	DBP	HR	SVR	E	e'	E/e'	E	e'
7-Day Average Ratings													
1. Perceived Stress	11.0 ± 5.6	----											
2. Anger	16.6 ± 4.6	.46 *	----										
3. Negative Emotion (NE)	4.9 ± 7.1	.44 *	.54 **	----									
Baseline													
4. SBP, mmHg	123 ± 21	.10	.07	.25	----								
5. DBP, mmHg	74 ± 10	.06	.07	.24	.58 **	----							
6. HR, bpm	66 ± 14	.53 *	.22	.50 *	.26	.25	----						
7. SVR, mmHg·min·mL ¹	29 ± 9	-.19	.08	.002	.09	.30	-.42	----					
8. E peak velocity, cm/sec	71 ± 21	.02	.04	.12	.20	.003	.07	.18	----				
9. Lateral wall e', cm/sec	5 ± 2	-.06	-.30	-.14	.22	-.06	.11	.05	.22	----			
10. E/e' ratio	15.5 ±8.5	.09	.54 **	.26	.06	.15	-.11	.27	.34	-.73 ***	----		
Stress-Induced Changes [†]													
11. Change in E, cm/sec	-0.3 ± 11.4	.19	.10	.29	-.10	.10	.12	-.22	-.20	-.47 *	.26	----	
12. Change in e', cm/sec	-0.6 ± 1.3	-.19	-.16	-.02	-.19	-.27	.08	-.25	-.15	-.38	.41	.05	----
13. Change in E/e'	3.3 ±8.5	.14	.14	.15	-.03	.26	-.03	.13	-.12	-.22	.08	.58 **	-.58 **

Values represent mean ± standard deviation and Pearson correlation coefficients;

* $p < .05$;

** $p < .01$;

*** $p < .001$;

SBP=systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate, SVR = systemic vascular resistance;

[†]Stress-baseline values measured as continuous variable