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Beta Blocker Administration During Emergency Department Evaluation for Acute Coronary Syndrome is Associated with Lower Posttraumatic Stress Symptoms 1 Month Later

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Abstract

We examined whether beta blocker administration in the emergency department (ED) during evaluation for suspected acute coronary syndrome (ACS) was associated with posttraumatic stress disorder (PTSD) symptoms 1 month later. Participants ($N = 350$) were enrolled in the Reactions to Acute Care and Hospitalization (REACH) study, an ongoing observational cohort study of ED predictors of medical and psychological outcomes after evaluation for suspected ACS. Beta blockade during evaluation in the ED was extracted from medical records, and PTSD symptoms in response to the experience of suspected ACS were assessed 1 month later via telephone. Beta blockade in the ED was associated with lower PTSD symptoms 1 month later ($b = -2.80$, $\beta = -.09$, $p = .045$), after adjustment for demographics, preexisting psychological and medical covariates, and participants' distress during ED evaluation. Despite small effects, findings suggest that beta blockade during ED evaluation for suspected ACS—a time period relevant to fear consolidation of the memory of this potentially life-threatening event—may have protective effects for later psychological health.

Evaluation for a potentially life-threatening cardiac event in the emergency department (ED) is a highly stressful experience that can trigger posttraumatic stress disorder (PTSD) (Edmondson, 2014). Posttraumatic stress disorder, characterized in part by intrusive reexperiencing and distressing memories of a traumatic event, has been associated with a 2-fold increase in the risk of morbidity and mortality due to subsequent cardiovascular events (Edmondson, Richardson, Falzon, Davidson, Mills, & Neria, 2012). Unless contraindicated due to comorbidities, beta blockers are often used in the acute treatment of suspected cardiac events in order to decrease heart rate and improve myocardial oxygenation (Chatterjee et al., 2013), although practice variation exists between and within ED settings. Recent research

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has proposed beta blocker administration as a pharmacological intervention for PTSD prevention, particularly when administered acutely after a trauma (Pitman & Delahanty, 2005), although findings regarding beta blockade for PTSD prevention are mixed (Sijbrandij, Kleiboer, Bisson, Barbui, & Cuijpers, 2015; Pitman et al., 2002; Vaiva et al., 2003).

Traumatic experiences have been found to overstimulate noradrenergic activity (Pitman & Delahanty, 2005). This increase in noradrenergic activity, in turn, contributes to overconsolidation of memories of the traumatic event, which is associated with the generation of PTSD symptoms (Pitman & Delahanty, 2005). Mechanistically, beta blockers interfere with this process by blocking β -adrenergic receptors and disrupting the consolidation and reconsolidation of emotional and traumatic memories in particular (Cahill, Prins, Weber, & McGaugh, 1994; Debiec & LeDoux, 2006; Lonergan, Olivera-Figueroa, Pitman, & Brunet, 2013). Lipophilic beta blockers are able to cross the blood-brain barrier and bind to postsynaptic β -adrenergic receptors in the basolateral amygdala (Pitman & Delahanty, 2005). The blockade of these receptors affects the noradrenergic system, thereby reducing noradrenergic hyperactivity and resulting in the release of fewer endogenous stress hormones. This blockade also disrupts the process of protein synthesis, inhibiting the consolidation of new memories into long-term memory storage (Lonergan et al., 2013). Indeed, research suggests that beta blocker administration before memory consolidation reduces recall of negative emotional information (e.g., Cahill et al., 1994).

Prior research has explored the effect of beta blockers on subsequent PTSD symptoms in response to cardiac-related traumas, namely intracardiac defibrillator discharge and cardiac surgery recovery, and findings suggest that beta blockers may be associated with lower PTSD symptom levels after these events (Bhuvaneswar, Ruskin, Katzman, Wood, & Pitman, 2014; Krauseneck et al., 2010). Although intriguing, these initial findings have somewhat limited generalizability as the existing studies were conducted in relatively small, all-male or predominantly (74%) male samples of cardiac patients who experienced circumscribed traumas (i.e., intracardiac defibrillator discharge and cardiac surgery). In the current study, we extended this literature by examining the effects of beta blocker administration on the development of PTSD symptoms 1 month later in patients evaluated in the ED for acute coronary syndrome (ACS). Acute coronary syndrome is common (over 1.1 million patients were discharged from hospitals in the United States with a diagnosis of ACS in 2010; Benjamin et al., 2017) and evaluation for suspected ACS is highly stressful (Edmondson, 2014). Thus, it is of interest to examine the beta blocker-PTSD relation in a diverse sample of male and female patients evaluated for this relatively common condition, as we do here.

Method

Participants and Procedure

English- and Spanish-speaking participants were enrolled during evaluation for suspected ACS in the Columbia University Medical Center ED as part of the Reactions to Acute Care and Hospitalizations (REACH) study, an observational cohort study of ED predictors of medical and psychological outcomes after suspected ACS (Sumner, Kronish, Pietrzak, Shimbo, Shaffer, Parsons, & Edmondson, 2015). Potential participants were identified by a

provisional diagnosis of “probable ACS” by the treating ED physician, including non-ST-elevation myocardial infarction (NSTEMI) on an electrocardiogram and unstable angina (UA). Exclusion criteria included: ST-elevation myocardial infarction (STEMI) on an electrocardiogram, as this triggers a rapid emergency protocol and transfer to the cardiac catheterization laboratory such that enrollment in the ED was not possible; inability to follow the protocol (due to dementia or substance abuse); need for immediate psychiatric intervention; and lack of availability for follow-up (e.g., due to terminal noncardiovascular illness). This study was approved by the Columbia University Institutional Review Board, New York, NY, United States. All participants provided written informed consent. Of those eligible, 61% enrolled. The analyzed sample comprised 350 individuals who completed the ED and 1-month follow-up assessments; retention at 1-month was 93%. Participant characteristics are presented in Table 1.

Measures

In the ED, participants completed measures of their ED experience, including current perceived life threat (assessed with 12 items based on Ozer, Best, Lipsey, & Weiss, 2003; see Sumner et al., 2015, for details). In an interview after transfer to an inpatient bed (or by telephone if inpatient interview was not possible), participants completed measures of lifetime trauma history (the Life Events Checklist; Gray, Litz, Hsu, & Lombardo, 2004), past month PTSD symptoms due to a previous trauma (the PTSD Checklist [PCL]-Civilian version; Weathers, Litz, Herman, Huska, & Keane, 1993), and past 2-week depressive symptoms (the Patient Health Questionnaire depression scale; Kroenke et al., 2009). A research nurse extracted ED beta blocker administration from medical records. Global Registry of Acute Cardiac Events (GRACE) score (Cakar et al., 2014; Eagle et al., 2004) and the Charlson Comorbidity Index (Charlson, Pompei, Ales, & MacKenzie, 1987) were calculated from medical records as measures of event severity and mortality risk, and medical comorbidity, respectively. A board-certified cardiologist determined discharge diagnosis by record review. One month after enrollment, participants completed the PCL-Stressor Specific version (Weathers, Litz, Herman, Huska & Keane, 1993) via telephone to assess PTSD symptoms in response to the evaluation for suspected ACS. PTSD symptom severity was calculated by summing responses to the 17 items (Cronbach’s $\alpha = .92$).

Data Analysis

Analyses were conducted using SPSS Version 23. First, we compared participants who did and did not receive beta blockade on demographics and psychological and medical covariates using chi-squared tests for categorical variables and independent samples *t*-tests for continuous variables. Next, we used stepwise logistic regression to examine medical predictors of beta blockade to determine any additional covariates that might be relevant. For our main analyses, we assessed the relation of beta blockade with 1-month PTSD symptom severity using multiple linear regression. The final model adjusted for demographics, pre-existing psychological and medical covariates, and participants’ distress during ED evaluation (Table 2).

Results

In the ED, 14.3% of patients received beta blockers. Participants who were and were not administered beta blockers did not differ significantly in terms of demographics, pre-existing psychological factors, or medical comorbidities (Table 1). However, a higher percentage of patients with confirmed ACS at discharge received beta blockade compared to those with non-ACS diagnoses, $\chi^2(1) = 4.790, p = .029$. To examine predictors of beta blocker administration, we conducted a stepwise logistic regression predicting beta blockade from ED presentation and medical history variables from medical records (GRACE score, Charlson Comorbidity Index, confirmed ACS status, initial heart rate, initial systolic blood pressure, initial troponin, initial creatinine, electrocardiogram ST-segment depression = 0.05mV, history of: myocardial infarction; stroke; hemiplegia; peripheral arterial disease; congestive heart failure; chronic obstructive pulmonary disease). Participants with higher systolic blood pressure ($OR = 1.020, p < .001$) and a history of myocardial infarction ($OR = 2.418, p = .013$) were more likely to receive beta blockers. Neither variable significantly predicted 1-month PTSD symptoms ($p = .198$ to $.250$). Thus, they were not included in subsequent models.

At 1-month follow-up, 15.1% ($n = 53$) of participants screened positive for PTSD in response to evaluation for suspected ACS (using a PCL score cutoff 35); the mean PTSD symptom severity was 24.36 ($SD = 10.54$, range = 17–76). Mean 1-month PTSD symptom severity did not differ significantly for participants who did ($M = 23.32, SD = 9.12$) and did not receive beta blockade in the ED ($M = 24.53, SD = 10.77$), $t(348) = 0.753, p = .452$. Beta blockade in the ED was associated with lower PTSD symptoms 1 month later, after adjustment for demographics, pre-existing psychological and medical covariates, and distress during ED evaluation (Table 2). Whether participants received beta blockade in the ED accounted for a small percentage of the variance in PTSD symptoms, $R^2 = .008$, although the model with all predictors (including beta blockade) accounted for 32.4% of the variance in PTSD symptoms. The beta blockade-PTSD symptom association was nearly identical in effect size, although not significant, when excluding the 92 patients with systolic blood pressure < 90mmHg, peripheral arterial disease, and acute heart failure exacerbations (i.e., patients for whom beta blockers may not be ideal; $b = -2.546, SE = 1.815, \beta = -.076, p = .162$). Although prior research in the REACH sample has shown that risk factors for, and prevalence of, PTSD after evaluation for ACS are similar in those with and without confirmed ACS at discharge (Velazquez et al., 2017), we conducted a sensitivity analysis of the association of beta blockade with PTSD symptoms separately in those with and without confirmed ACS. Among those without confirmed ACS at discharge ($n = 236$), there was a trend for association between beta blockers and lower PTSD symptoms, $b = -3.244, SE = 1.775, \beta = -.099, p = .069$. The association was not as strong for individuals with confirmed ACS, $n = 114$; $b = -0.778, SE = 2.224, \beta = -.029, p = .727$.

Discussion

This is the first known demonstration that beta blockade during ED evaluation for a potentially life-threatening cardiac event is associated with lower subsequent PTSD symptoms. Prior research suggests that beta blockers may attenuate PTSD symptoms after

other traumatic medical events, such as cardiac surgery and intracardiac defibrillator discharge (Bhuvaneswar et al., 2014; Krauseneck et al., 2010). Although the effect was small, these findings suggest that beta blockade during ED evaluation for suspected ACS—a time relevant to consolidation of the memory of this potentially life-threatening event—may have protective effects for psychological health.

This study has limitations. Beta blocker administration was extracted from medical records, but beta blocker type was not recorded. Research has proposed that certain types of beta blockers may differentially affect the development of subsequent PTSD symptoms. These effects are based on a pathophysiological model of PTSD, which suggests that an excessive stress response leads to the overconsolidation of memories after a traumatic event and, ultimately, an increased risk of PTSD. Bhunvaeswar et al. (2014) examined the difference between lipophilic and hydrophilic beta blockers on PTSD symptoms after ICD discharge and reported a 35% lower rate of PTSD in those who were prescribed lipophilic beta blockers, a type of beta blocker capable of penetrating the blood-brain barrier and interfering with memory consolidation triggered specifically by stress. Given that moderately lipophilic beta blockers (e.g., metoprolol) are used commonly in the ED setting, future research may explore the impact of lipophilic beta blockers, specifically, on PTSD symptoms after evaluation for ACS.

In addition, our study did not examine the method of beta blocker administration. Research suggests that there may be varying effects on cardiovascular outcomes based on intravenous versus oral beta blocker administration. In a review by Chatterjee et al. (2014) researchers identified an 8 to 15% short-term mortality risk reduction associated with intravenous administration, whereas prior research had been inconclusive, highlighting a potential benefit of intravenous administration. Future research may take into account beta blocker administration methods.

Also, our study was small, and beta blocker administration was not randomly assigned. Additionally, although all patients were evaluated for suspected ACS in the ED, our sample comprised individuals with and without confirmed ACS diagnoses at discharge. However, prior research suggests that factors that influence the subjective experience of evaluation for ACS (e.g., perceived life threat, neuroticism)—and not objective clinical severity or diagnosis—are most associated with risk of PTSD after evaluation for ACS (see Edmondson & Cohen, 2013, for a review). Research from the REACH study has shown that levels of threat perceptions during evaluation for ACS are similar in those with and without confirmed ACS at discharge (Velazquez et al., 2017). Given that patients perceive evaluation for ACS as threatening regardless of objective diagnosis at discharge, we hypothesized that the effects of beta blockade could have beneficial effects with respect to fear memory consolidation in both those with and without confirmed ACS. Despite these limitations, our study is one of the largest of beta blockers and PTSD in male and female patients evaluated for cardiac events, and we accounted for various psychological and medical covariates relevant to PTSD in our racially and ethnically diverse sample.

Our results suggest that beta blockade during evaluation for suspected ACS—an often threatening event—may reduce the severity of subsequent posttraumatic stress, although

findings should be weighed against the possible negative impact of beta blockade for certain patients (Bangalore et al., 2014). Future studies should consider a prospective, randomized controlled trial to build on findings from this observational cohort study. In addition, it is of interest to investigate if certain groups of individuals at risk for PTSD after suspected ACS are particularly likely to benefit from beta blockade in order to inform more targeted delivery of prevention efforts. Future studies should also pay particular attention to beta blocker class (lipophilic versus hydrophilic), beta blocker dosage, and time period of administration (e.g., during the peritraumatic window, after discharge). Further research on this topic may elucidate ways to diminish the burden of potentially life-threatening cardiac events, and should factor into decisions about beta blockade during ED evaluation for ACS.

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Participant Characteristics as a Function of Beta blocker Administration in the Emergency Department during Evaluation for Suspected Acute Coronary Syndrome (N = 350)

Table 1

Characteristic	Administered beta blockers (n = 50)				Not administered beta blockers (n = 300)			
	M	SD	%	n	M	SD	%	n
Demographics								
Age, years	59.5	11.8			60.7	12.7		
Female sex			38.0	19			47.7	143
Race								
White			22.0	11			39.0	117
Black			34.0	17			25.0	75
Other			44.0	22			36.0	108
Hispanic ethnicity			40.0	20			52.0	156
Medical record covariates								
GRACE score	91.6	28.3			92.4	29.5		
Charlson Comorbidity Index	2.1	2.0			1.9	2.2		
Hospital assessment								
Perceived threat in the ED	11.6	4.1			10.5	3.9		
Depressive symptoms at baseline ^a	6.5	5.9			6.4	5.7		
Lifetime trauma burden ^b	3.3	3.3			3.0	2.9		
PTSD total severity score at baseline for prior trauma ^c	27.9	16.5			26.3	12.3		
Confirmed ACS upon discharge [*]			46.0	23			30.3	91

Note. GRACE=Global Registry of Acute Coronary Events, ED=emergency department, PTSD=posttraumatic stress disorder, ACS=acute coronary syndrome.

^aPast 2-week depressive symptoms assessed with the Patient Health Questionnaire depression scale (PHQ-8).

^bLifetime trauma burden assessed with the Life Events Checklist (LEC).

^cPTSD total severity score at baseline calculated by summing responses on the PTSD Checklist-Civilian version (PCL-C) with respect to the most stressful event identified on the LEC.

^{*} $p < .05$.

Table 2

Results of Regression Analysis Examining Associations Between Beta Blocker Administration in the Emergency Department During Evaluation for Suspected Acute Coronary Syndrome and Posttraumatic Stress Symptoms at 1-Month Follow-Up

Variable	<i>b</i>	95% CI	SE	β	<i>t</i>
Age	-0.04	[-0.17, 0.10]	0.07	-0.04	-0.54
Female gender	1.35	[-0.63, 3.33]	1.01	0.06	1.34
White race	-1.92	[-4.37, 0.54]	1.25	-0.09	-1.54
Black race	-1.31	[-4.34, 1.73]	1.54	-0.06	-0.85
Hispanic ethnicity	-0.24	[-2.65, 2.17]	1.22	-0.01	-0.20
GRACE score	-0.003	[-0.07, 0.06]	0.03	-0.01	-0.09
Charlson Comorbidity Index	0.28	[-0.24, 0.80]	0.26	0.06	1.06
Confirmed ACS upon discharge	1.41	[-0.67, 3.49]	1.06	0.06	1.33
Perceived threat in the ED ^{**}	0.64	[0.37, 0.91]	0.14	0.24	4.73
Baseline depressive symptoms ^{**}	0.36	[0.17, 0.55]	0.10	0.20	3.79
Lifetime trauma burden	0.26	[-0.12, 0.63]	0.19	0.07	1.35
Baseline PTSD total severity score for prior trauma ^{**}	0.20	[0.10, 0.29]	0.05	0.24	4.22
Beta blocker administration in the ED [*]	-2.80	[-5.54, -0.06]	1.39	-0.09	-2.01

Note. GRACE=Global Registry of Acute Coronary Events, ACS=acute coronary syndrome, ED=emergency department, PTSD=posttraumatic stress disorder.

^{*} $p < .05$.

^{**} $p < .001$