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The goal of the proposed research is to identify targets for new interventions to reduce the doubled cardiac event recurrence and mortality risk faced by the 1 in 8 survivors of non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA) who develop PTSD secondary to their life-threatening cardiac event. Research on the mechanisms likely to carry that risk is converging on autonomic dysregulation as the culprit.

PTSD is associated with high heart rate (HR) and low heart rate variability (HRV), both established secondary risk markers in NSTEMI/UA patients. In our recently offered Enduring Somatic Threat (EST) model, we propose a vicious cycle in which PTSD intrusion symptoms cause acute autonomic imbalance, leading to heart rhythm alterations that are perceived as threatening by the patient, causing further autonomic imbalance. Further, the model proposes that this vicious cycle is exacerbated by nonadherence to beta-blockers (medications that blunt sympathetic influences on HR), a common avoidance behavior in patients with PTSD. Surprisingly, although autonomic imbalance is the leading candidate for PTSD's influence on cardiovascular morbidity and mortality in populations from young veterans to older adults with cardiovascular disease, its candidacy is based almost solely on research conducted in the clinic or the laboratory. No study has ever tested the association of PTSD symptoms and cardiovascular parameters in the real world.

We propose to test the EST model in our Reactions to Acute Care and Hospitalization observational cohort study of 1,741 NSTEMI/UA patients. We will enroll 100 participants with NSTEMI/UA-induced PTSD, and 100 without, at 1-month after hospital discharge. For 1 week, participants will (1) report on PTSD intrusion symptoms in the 30 minutes prior to each of 10 daily electronic momentary assessments (EMA); (2) wear an ambulatory smart shirt embedded with ECG and an accelerometer, from which we will derive heart rate and heart rate variability (HRV); and (3) have their adherence to beta blockers electronically monitored.

We will test whether NSTEMI/UA patients with PTSD have higher 24-hr HR and lower 24-hr HRV than those without PTSD and, if so, whether the frequency and intensity of intrusive thought(s) explains the difference. Further, we will test whether HR is higher, and HRV lower, for epochs in which patients report intrusions relative to those in which they do not—and whether the difference is more pronounced in patients with PTSD. Finally, we will test whether patients with PTSD are less adherent to beta-blockers, and explore whether associations of intrusions with HR/HRV are more pronounced on days that patients miss their dose.

More than 150,000 Americans develop PTSD secondary to NSTEMI/UA each year, and they are at high risk for adverse outcomes. This research will determine whether autonomic dysregulation in the real world is truly a candidate mechanism, describe the dynamics by which PTSD causes it, identify the factors most important to target—and the point in the vicious cycle to intervene, and suggest new interventions to offset risk.