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INVITED COMMENTARY

Personality Factors, Emotional Triggers and Acute Cardiac Events: a Comment on Compare et al.

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In this edition of the *Annals of Behavioral Medicine*, Compare, Steptoe and colleagues [1] describe the association between Type D personality and stress-induced cardiomyopathy. Scientific and clinical interest in this acute cardiac condition is increasing [2], and the syndrome is also often referred to as "takotsubo cardiomyopathy" [3] or "apical ballooning syndrome" [4]. The emotional triggering of apical ballooning has been documented in animal models of experimental stress [5] and in meta-analytical research in humans [6]. About 75 % of the patients with apical ballooning syndrome report a preceding event including physical and/or emotional distress [3]. In the study of Compare and colleagues, only those acute cardiomyopathy admissions that were precipitated by an emotional (37 cases) or physical (38 cases) trigger were included [1]. Therefore, the investigators' use of the term "stress cardiomyopathy" seems accurate, and will also be used here.

Stress cardiomyopathy is characterized by major cardiac left ventricular dysfunction with typical apical ballooning of the left ventricle and symptoms and signs mimicking acute coronary syndromes, but without any evidence of obstructive coronary artery disease [2]. The most common presentation includes chest pain and/or dyspnea with ECG changes suggesting myocardial infarction, combined with substantial ballooning of the left ventricle (see Fig. 1 for example of a previously published case report [7]). Biological markers of cardiac muscle damage show only modest changes [3], and cardiac abnormalities in stress cardiomyopathy generally resolve within days. Little is known, however, about the psychological and physiological characteristics of stress cardiomyopathy.

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By definition, the typical precipitants of emotion-triggered stress cardiomyopathy involve mental and psychological distress [2]. In humans, the threat of negative social evaluations is a main source of distress in daily experience [8]. However, the experience of social challenge, to a large extent, reflects the subjective appraisal of conflicts and pressures in daily life [9], and thus may differ substantially among individuals. The distressed (Type D) personality, characterized by high levels of social inhibition and negative affectivity, characterizes these individual differences in vulnerability to social and other environmental challenges and could account for elevated distress levels [10].

The observation of Compare et al. [1] that individuals with stress cardiomyopathy triggered by emotional events more often meet criteria for Type D personality than patients with stress cardiomyopathy that are not triggered by emotional events (76 vs 43 %) is of interest as it suggests individual trait characteristics that predispose to stress-induced events. Strengths of the study include the short-term assessments of emotional triggers (within 48 h), the careful classification of the nature of the triggers, and the assessment of the Type D construct as both a dichotomized and continuous measure, including the interaction term between social inhibition and negative affectivity [11]. The finding that this interaction term was associated with emotion-triggered stress cardiomyopathy is consistent with findings from other groups showing that the interaction of social inhibition and negative affectivity was associated with more subjective stress [12], dysfunctional cardiac output [13], and higher cortisol levels [14]. Type D was assessed 3 months after hospital discharge, which is probably a strong point because delayed assessment avoids potential biases in the assessment of Type D that could be related to biomedical and psychosocial factors associated with hospitalization, and because social inhibition and negative affectivity are stable personality traits [15].

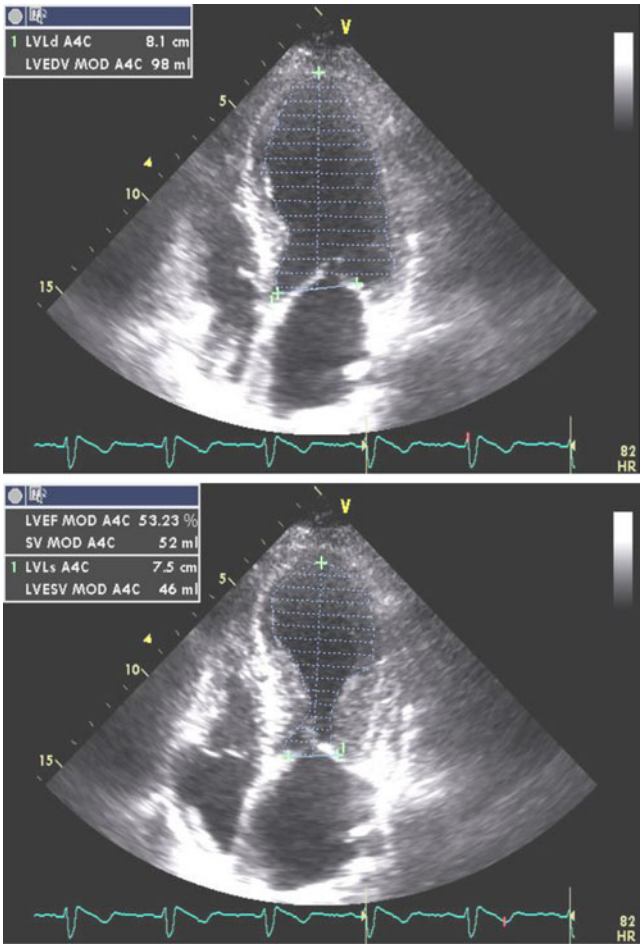

Fig. 1 A case of apical ballooning in a patient with stress cardiomyopathy. Adapted from Kop and Gottdiener [7]. This Fig. 1 displays echocardiographic images of a patient with stress cardiomyopathy. The patient was a 59-year-old woman who was very distressed when visiting one of her family members admitted to an academic shock trauma clinic following a lethal motor vehicle accident. She experienced typical anginal chest pain radiating to the jaw and was admitted to the emergency department with ventricular fibrillation. Coronary angiography revealed an ejection fraction of 10–20 % (anterolateral, apical, inferior, and posterobasal akinesia) but only moderate non-obstructive disease in the left anterior descending artery. Echocardiography was consistent with moderately severe LV dysfunction with elevated left atrial pressure and mild dilation. The increase in troponin levels were modest (peak 29.9) relative to the degree of LV impairment. The patient had a prior history of a myocardial infarction, atrial fibrillation, and mitral valve repair, and echocardiography 3 months earlier documented a normal ejection fraction of approximately 65 %. The non-cardiac medical history included restless leg syndrome and joint disease (unspecified). The patient recovered well and was discharged after three days in stable condition. This case illustrates the substantial cardiac responses to acute emotional distress, including severely compromised left ventricular function, life threatening arrhythmias, in the absence of severe coronary disease and with a fast and uncomplicated recovery

However, it is not entirely clear to what extent the association of Type D personality with emotionally triggered stress cardiomyopathy is specific to this condition or whether this association would also be observed in other potentially triggered events, such as acute coronary syndromes or stroke. The control group of patients with an acute myocardial infarction triggered by an emotional trigger displayed a lower prevalence of Type D (32 %). A limitation of this study is that no information was obtained about the prevalence of Type D in patients with acute coronary syndromes that were not triggered by emotions. Assuming that the prevalence of Type D would have been lower than 32 % in that group, the authors' conclusion that emotionally triggered stress cardiomyopathy is associated with Type D seems reasonable.

The proposed underlying hypothesis of this study is that Type D is a risk factor for emotionally triggered stress cardiomyopathy, but the risk ratios were only reported for *z* score transformed continuous measures, not for the Type D typology. From the tables, it can be inferred that the risk associated with Type D personality for an emotionally triggered stress cardiomyopathy is 5.3 ([25×28]/[11×12]); the estimated risk of a triggered stress cardiomyopathy for the dichotomized social inhibition component is even higher (OR=16.6). If confirmed in larger studies, then these risks are remarkably high.

The biological mechanisms accounting for the observed associations are not known. Mental and psychological distresses are not only implicated in the onset of stress cardiomyopathy [5] but can also trigger myocardial ischemia [16] and acute coronary syndromes such as myocardial infarction or sudden cardiac death [16–18]. Emotionally triggered stress cardiomyopathy is purportedly associated with excess catecholamine release [2]. Structured mental challenge tasks elicit higher catecholamine responses in stress cardiomyopathy patients [4], and Type D has also been related to mental stress-induced catecholamine responses [19]. In patients with chronic heart failure, Type D has been associated with decreased heart rate recovery after exercise [20]. Endothelial dysfunction and impaired vascular response to mental stress have also been implicated in the pathogenesis of stress cardiomyopathy [4], and there is some evidence to suggest that Type D is related to endothelial dysfunction in patients with chronic heart failure [21].

It is possible that the pathophysiological processes involved in stress cardiomyopathy are associated with exaggerated neurohormonal and cardiovascular response patterns, particularly sustained elevated levels and lack of restoration (i.e., delayed recovery), whereas acute coronary syndromes such as myocardial infarction and sudden cardiac death are preceded by elevated bouts of short-term elevations of stress hormones and hemodynamic demand combined with reduced coronary supply [16]. Additional research is needed to investigate these pathways in controlled laboratory settings, to further identify specific psychological factors (personality traits and episodes of elevated distress) to increase the accuracy of patient risk stratification and to develop biobehavioral

interventions that are based on these biobehavioral and epidemiological observations.

Importantly, the study of Compare et al. [1] also suggests a potential key role for social inhibition [22] in increased cardiovascular reactivity to acute emotional distress. Socially inhibited individuals have a specific brain response to social threat [23] and a cognitive interpretation bias toward social threat [24]. This may explain why they frequently expect negative reactions from others, and may have an inadequate cardiovascular response to acute social stress [19, 25]. Multiple issues remain to be resolved in this new area of biobehavioral research, and the findings reported by Compare et al. [1] support the need to further explore the role of individual differences in the triggering of acute cardiac events.

Conflict of Interest No conflict of interest exist for any of the authors.

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