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ECG signatures of psychological stress☆,☆☆

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Abstract Psychological stress can lead to atrial and ventricular arrhythmias, but the physiological pathways have not been fully elucidated. Signal processing techniques can provide insight into electrophysiological mechanisms of stress-induced arrhythmia. T-wave alternans, as well as other ECG measures of heterogeneity of repolarization, increases with emotional and cognitive stress in the laboratory setting, and may also in "real life" settings. In the atrium, stress impacts components of the signal-averaged ECG. These changes suggest mechanisms by which everyday stressors can lead to arrhythmia.

Keywords: Psychological stress; Repolarization; Atrial fibrillation

Introduction

Acute psychological stress can lead to atrial and ventricular arrhythmias, but the physiological pathways have not been fully elucidated. Signal processing techniques can provide insight into electrophysiological mechanisms of stress-induced arrhythmia. T-wave alternans, as well as other ECG measures of heterogeneity of repolarization, increases with emotional and cognitive stress in the laboratory setting, and may also increase with stress in "real life" settings. In the atrium, stress impacts components of the signal-averaged ECG. These changes suggest mechanisms by which everyday stressors can lead to arrhythmia.

ECG signatures of psychological stress in the ventricle

Epidemiological and clinical studies demonstrate that psychological distress, defined as a consciously experienced mismatch between expectations and the perceived environment associated with aversiveness [1], can trigger both ventricular and atrial arrhythmias. The first epidemiological evidence linking acute stress and ventricular arrhythmias comes from data showing increases in sudden cardiac death (SCD) during stress-inducing population disasters such as earthquake or war. For example, increases in cardiovascular and sudden death were reported during the Iraqi missile war in Israel in 1981 [2], as well during air raids in Zagreb [3]. On the day of the earthquake in Northridge, CA, in 1994, there was a 6-fold increase in SCD compared to days prior to and following the disaster [4]. These reported sudden deaths during each of these population disasters were not related to physical injury or other direct physical involvement, implying a role of psychological rather than physical stress. SCD can be precipitated by ischemic or arrhythmic events, and the effects of stress on ischemia have been long understood. However, data from ICD patients showing an increase in ventricular arrhythmia after the World Trade Center attacks of 9/11/2001, suggest that autonomic changes related to stress may directly modulate arrhythmogenesis [5].

Clinical studies also show a link between stress and arrhythmia. We looked more directly at the question of whether anger or other emotions can trigger ventricular arrhythmias using a case–control study of ICD patients. In this study, patients were asked, whenever they received a shock from the ICD, to record in a diary their activities and emotions in the 15 minutes and 2 hours prior to ICD shock. They were then asked to fill out a similar diary one week later at the same time of day to serve as controls. Anger levels were greater prior to appropriate shock for ventricular arrhythmia than during control periods, demonstrating that anger can trigger ventricular arrhythmias [6]. Overall, anger-triggered arrhythmias were more likely to be polymorphic, PVC-initiated, and pause-dependent, characteristics associated with lethality (see Fig. 1) [7]. Because this diary study relied on self-report of anger, and because many

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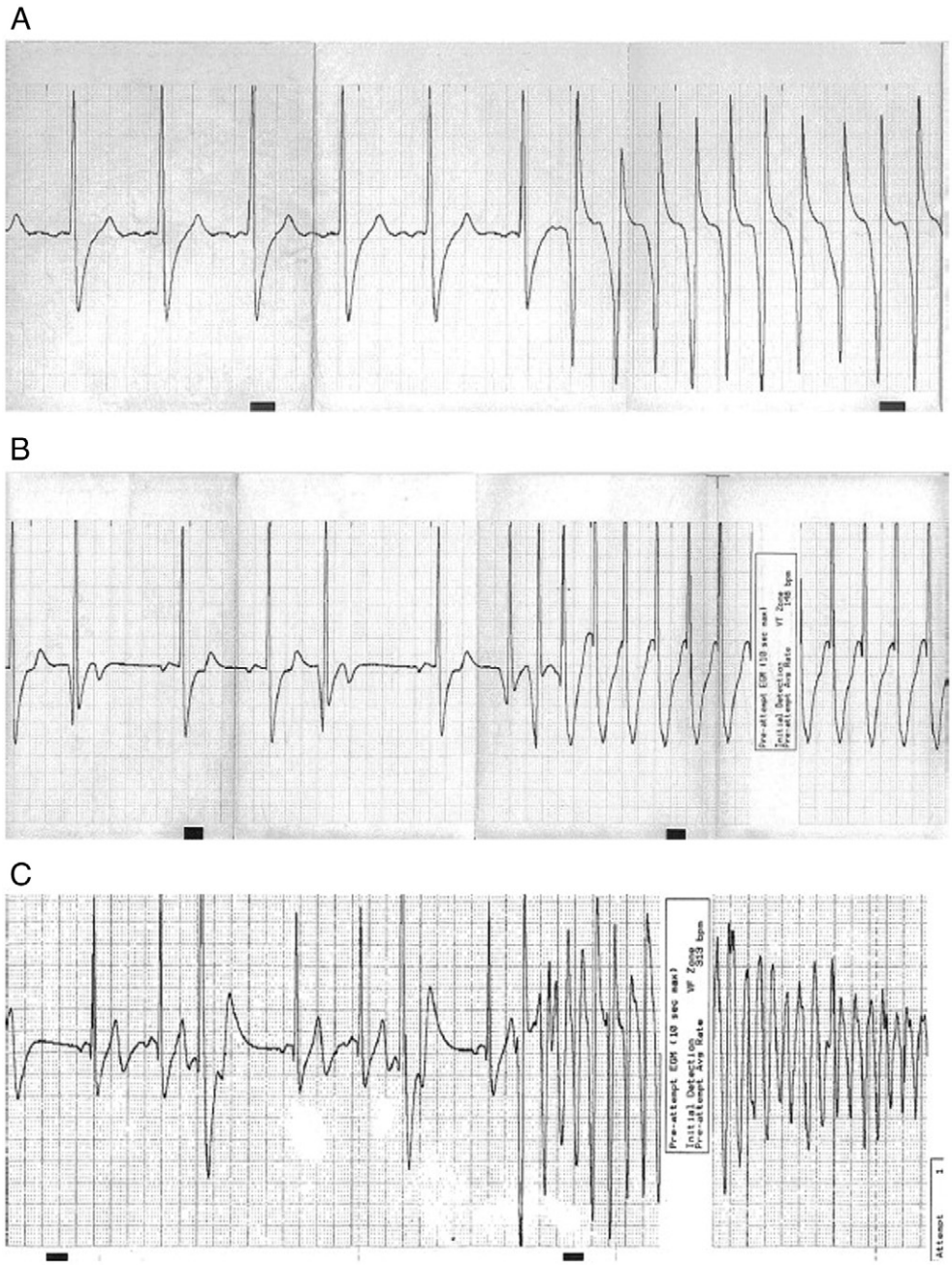

Fig. 1. Electrograms of anger-triggered and non-anger-triggered ventricular arrhythmias. (A) Monomorphic sudden-onset arrhythmia (non-anger-triggered); (B) monomorphic PVC-initiated pause-dependent arrhythmia (anger-triggered); (C) polymorphic PVC-initiated pause-dependent arrhythmia (anger-triggered). From Stopper, et al., Heart Rhythm 2007 (Ref. [6]) with permission.

individuals (one-third in some studies) [8] suppress the expression of anger, which is associated with physiological changes [8], it is possible that the impact of anger on arrhythmia is even greater than seen here.

Signal processing techniques can provide insight into electrophysiological mechanisms of stress-induced arrhythmia. Toivonen et al. [9] used a human model of stress – the on-call medical house officer – to look at QT changes during stress. QT intervals during periods of arousal due to a page were compared to periods of rest with identical heart rates. They found hysteresis of the QT interval during periods of stress, with longer QT during stress than rest, similar to the QT hysteresis found during exercise [10]. Exaggerations of QT hysteresis with exercise are thought to be one mechanism underlying sudden death in exercise, and could similarly be a mechanism of stress-related SCD.

We looked directly at effects of stress on heterogeneity of repolarization, long-recognized to be an important factor in arrhythmogenesis, by measuring T-wave alternans (TWA) during a laboratory mental stress protocol. Similar to a creating physical stress on a treadmill exercise test, we can create mental stress in the laboratory through a variety of methods. These include asking the patient to do arithmetic in his head, such as serial subtraction of 7 from a 3-digit number, or can involve a speaking task with emotional content. In our laboratory, we do a stressor called “anger recall”. We ask subjects to tell us about a recent incident in