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Anger and ventricular arrhythmias

Rachel Lampert

Section of Cardiology, Yale University School of Medicine, New Haven, Connecticut, USA

Abstract

Purpose of review—Although anecdotal evidence has long suggested links between emotion and ventricular arrhythmia, more recent studies have prospectively demonstrated the arrhythmogenic effects of anger, as well as mechanisms underlying these effects.

Recent findings—Epidemiological studies reveal that psychological stress increases sudden death, as well as arrhythmias, in patients with implantable cardioverter-defibrillators, in populations during emotionally devastating disasters such as earthquake or war. Diary-based studies confirm that anger and other negative emotions can trigger potentially lethal ventricular arrhythmias. Anger alters electrophysiological properties of the myocardium, including T-wave alternans, a measure of heterogeneity of repolarization, suggesting one mechanistic link between emotion and arrhythmia. Pilot studies of behavioral interventions have shown promise in decreasing arrhythmias in patients with implantable cardioverter-defibrillators.

Summary—Anger and other strong emotions can trigger polymorphic, potentially life-threatening ventricular arrhythmias in vulnerable patients. Through autonomic changes including increased sympathetic activity and vagal withdrawal, anger leads to increases in heterogeneity of repolarization as measured by T-wave alternans, known to be associated with arrhythmogenesis, as well as increasing inducibility of arrhythmia. Further delineation of mechanisms linking anger and arrhythmia, and of approaches to decrease the detrimental effects of anger and other negative emotions on arrhythmogenesis, are important areas of future investigation.

Keywords

anger; triggering; ventricular arrhythmias

Introduction

Evidence linking anger and ventricular arrhythmias continues to mount. Epidemiological studies reveal that psychological stress increases sudden death in populations during emotionally devastating disasters such as earthquake or war [1,2]. Although sudden cardiac death (SCD) may be due to either ischemic or primary arrhythmic causes, more recent data from individuals with implantable cardioverter-defibrillators (ICDs) have demonstrated that both population stressors, such as the World Trade Center attacks of 11/9/2001 [3], and individual emotional experiences, such as anger and anxiety [4], can trigger ventricular arrhythmias in patients with known propensity for these arrhythmias. Although pathways linking emotion to ventricular arrhythmias remain incompletely understood, laboratory studies in both animals and humans suggest that anger can render ventricular arrhythmias more easy to induce and more difficult to terminate. Further, anger and other strong emotions can alter repolarization, increasing heterogeneity, known to be associated with

arrhythmogenesis. An understanding of mechanisms of emotion-induced arrhythmias will have important implications for future therapies. Pilot studies of behavioral interventions, known to improve prognosis in coronary artery disease, have shown promise in decreasing arrhythmias in patients with ICDs, and ongoing randomized trials are investigating this further.

Review

Case 1

This patient is a 66-year-old man with a history of myocardial infarction (MI) in 1984. While playing a slot machine at a Connecticut casino, he had an episode of witnessed loss of consciousness and received bystander cardiopulmonary resuscitation until the arrival of Emergency Medical Services providers, who found him in ventricular fibrillation and delivered a 360-J shock that converted the rhythm to sinus. On awakening, he had no complaints, laboratory evaluation and ECG did not suggest MI, and cardiac catheterization revealed an occluded left anterior descending artery and ejection fraction of 35%. He received an ICD and did well.

Case 2

This patient is a 69-year-old man with a history of MI and an ejection fraction of 25%, who received an ICD for primary prevention. Following a simmering dispute over a bordering fence with a neighbor for several weeks, Mr W.B. confronted the neighbor in the yard. Shortly into the argument, he became dizzy and received a shock from the ICD. On device interrogation, stored electrogram revealed ventricular tachycardia (Fig. 1c, [5]).

Anecdotes describing the triggering of ventricular arrhythmias by anger and other strong emotions, such as grief or fear, are abundant [6,7]. More recently, data directly linking the experience of strong emotion to arrhythmia have begun to mount. Anger and other psychological stressors alter autonomic balance, increasing catecholamine levels [8] and decreasing vagal output [9]. Indirect evidence has long suggested that sympathetic arousal can trigger arrhythmic events. For example, ventricular tachycardia occurs more frequently in the morning, the time of peak catecholamine levels [10] and lowest vagal tone [11], as demonstrated in patients with ICDs [12]. Studies using heart rate variability (HRV) to measure autonomic balance have shown that sympathetic activity increases and vagal activity decreases prior to episodes of ventricular tachycardia caught fortuitously on ambulatory ECG monitoring [13].

Further, epidemiological evidence has shown increases in SCD during times of stressors on entire populations, such as earthquake or war [1,2]. For example, on the day of the Northridge earthquake in 1994, there was a six-fold increase in SCDs as compared with days prior to and following the disaster [1]. Although sudden death may be due to either ischemia or primary arrhythmia, analogous data describing an increase in ICD-treated ventricular arrhythmias at one center in New York City in the month following the World Trade Center attacks of 9/11/2001 [3] (Fig. 2) suggest that autonomic changes due to stress may directly modulate arrhythmogenesis.

In order to determine, in a prospective, controlled fashion, whether the individual experience of emotions can trigger ventricular arrhythmias, we asked ICD patients [4] to record, in a diary, their activities and emotions in the 15 min and 2 h prior to any ICD shocks they received. Patients ranked their levels of anger and other emotions on a five-point likert scale and reported what they were doing. They then filled out a similar diary 1 week later at the same time of day. One hundred and seven shocks were reported by 42 patients. Higher levels of anger, dichotomized at level 3 or above, were more common before shock,

occurring 15% of the time, than during control periods, reported in just 3% of diaries. Anger was significantly associated with the 15 min preceding shock [4]. Anger-triggered arrhythmias, defined as those preceded by an anger level of 3 or above, were more likely to have characteristics associated with lethality, that is, more likely to be polymorphic, premature ventricular contraction (PVC)-initiated, and pause-dependent than those not preceded by anger [5] (Fig. 1). Preliminary data from the Triggers Of Ventricular Arrhythmias (TOVA) trial, a multicenter study evaluating potential triggering factors, which is currently in the analysis phase, have also demonstrated that anger can precipitate ventricular arrhythmias [14].

Stable psychological traits associated with emotion, such as hostility, have long been associated with cardiac events [15]. More recent data suggest that psychological makeup may also predispose to arrhythmia. In the TOVA trial [16], depression was associated with an increased risk of appropriate ICD shock for ventricular arrhythmia, even after controlling for clinical variables. In our diary study [17], individuals with psychological profile characterized by anger or anxiety were more likely to experience triggering of arrhythmias by these emotions. Although hostility has been described as a predisposing factor for atrial fibrillation [18], whether psychological traits influence overall risk of ventricular arrhythmia has not yet been reported.

Electrophysiological pathways linking anger and arrhythmias

The underlying pathophysiological mechanisms linking anger and other negative emotions to arrhythmia remain incompletely understood. The autonomic changes associated with anger and other psychological stressors are well described: strong emotion alters autonomic balance, increasing catecholamine levels [8] and decreasing vagal output [9]. Multiple experimental studies have demonstrated that these autonomic changes – sympathetic activation and vagal withdrawal – are arrhythmogenic. In animal models of infarction, direct sympathetic stimulation of the ventricles via stellate ganglion stimulation induces fibrillation [19,20], demonstrated as early as 1964 by Han *et al.* [19], and higher levels of vagal activity are protective against ischemic arrhythmias [21]. Further, MI results in nerve injury, which causes sympathetic nerve sprouting and regional sympathetic hyperinnervation [22]. This heterogeneous sympathetic nerve sprouting has in turn been linked with ventricular arrhythmias in a postinfarct dog model [23], as well as in explanted hearts [24], further emphasizing the role of sympathetic activity in post-MI sudden death.

Other investigators have looked directly at the electro-physiologic effects of emotion. In a landmark study published in 1973, Lown *et al.* [25] investigated the possibility that mental stress could facilitate induction of ventricular arrhythmias. Using a protocol conceptually similar to electrophysiologic programmed stimulation in a dog model, they found that, in rested animals, only one PVC could be induced, and this required a high output of 35 mA. In a dog stressed by being lifted in a sling conditioned to be a noxious experience, two PVCs were induced, using just 5 mA.

Studies in humans

We and others have used a laboratory mental stress protocol to directly probe the electrophysiological consequences of anger and other stressors in humans. Similarly to creating a physical stress on a treadmill exercise test, mental stress can be created in the laboratory through a variety of methods. These can include anger recall, a speaking task in which patients are asked to describe a recent event in which they felt angry or irritated, with an interlocutor asking questions designed to recreate the moment, or being asked to perform mental arithmetic, such as subtracting seven serially from a three-digit number.

Cardiovascular responses to mental stress induced in the laboratory, termed cardiovascular reactivity, have been shown to accurately reflect cardiovascular responses to stresses during daily life [26-28].

We first evaluated the effects of mental stress on induced arrhythmias in patients with a history of known ventricular arrhythmias and ICDs [29]. All patients had had ventricular tachycardia, which was terminated with anti-tachycardia pacing at previous electrophysiologic studies. Some patients had ventricular tachycardia induced earlier in the protocol, using fewer extrastimuli, although this was not significant. However, arrhythmias induced during mental stress were faster than those induced at rest and were harder to terminate. In some patients, an identical ventricular tachycardia, which had been pacer-terminated in the baseline state, required shock for termination during anger recall (Fig. 3), suggesting that autonomic changes due to the anger had altered properties of conduction and refractoriness of the ventricular tachycardia circuit, eliminating the excitable gap.

In order to look further at the mechanisms underlying the arrhythmogenic effects of stress, we next looked at effects of stress on T-wave alternans (TWA), a measure of heterogeneity of repolarization, long described as important in arrhythmogenesis [30]. This variable was measured during a mental stress protocol, including mental arithmetic and anger recall, in 33 patients with ICDs and a history of ventricular arrhythmias [31]. TWA increased from 22 μ V at baseline to 29 μ V during mental stress ($P<0.001$). All other measures of heterogeneity also increased with stress. Kop *et al.* [32], in a similar study, found further that these effects were independent of ischemia.

In our study, the increase in TWA correlated with increases in catecholamines, but there was minimal increase in heart rate (HR) with mental stress, suggesting a direct effect of sympathetic stimulation on TWA. Although an increase in HR itself will increase TWA, prior studies have also suggested that sympathetic activation may increase TWA beyond the effects of HR. For example, TWA induced with exercise is greater than that with atrial pacing at the same HR [33].

In a follow-up of our study of anger and TWA [34**], we looked to determine whether TWA induced by anger in a laboratory setting predicted future ventricular arrhythmias (ventricular tachycardia/ventricular fibrillation) requiring termination by the ICD. In that study, 62 patients who had undergone TWA measurement during anger recall were followed long-term and ICD stored data were reviewed to determine incidence of ICD-terminated ventricular tachycardia/ventricular fibrillation. Patients with ICD-terminated arrhythmias during follow-up ($n = 10$) had higher TWA induced by anger, 13.2 μ V (interquartile range (IQR) 9.3–16), compared with 9.3 μ V (IQR 7.5–11.5) ($P<0.01$). Patients in the highest quartile of anger-induced TWA ($>11.9\mu$ V, $n = 15$) were more likely to experience arrhythmias by 1 year than those in the lower quartiles (33 versus 4%), and during extended follow-up (40 versus 9%, $P<0.01$ for both.) In multivariable regression controlling for ejection fraction, prior clinical arrhythmia, and wide QRS, anger-induced TWA remained a significant predictor of arrhythmia, with likelihood in the top quartile 10.8 times that of other patients (95% confidence interval 1.6–113, $P<0.05$, Fig. 4). In summary, anger-induced TWA predicted future ventricular arrhythmias in patients with ICDs, supporting the hypothesis that emotion-induced repolarization instability may be one mechanism linking stress and sudden death.

Genetic factors may also play a role in emotionally induced arrhythmias. Primary ventricular fibrillation accompanying MI is increased in those with a history of familial sudden death, suggesting a genetic predisposition to vulnerability to ventricular fibrillation in general [35]. A recent intriguing case report [36] describes two sisters who developed ventricular

arrhythmias on the anniversary of their father's death (one ICD shock, one SCD), suggesting that vulnerability to emotionally triggered arrhythmias may also have a genetic component.

Clinical implications

Emotion, including negative emotion, is part of life and avoidance of stress not possible for most of us in a life fully lived. However, determining approaches to decrease the detrimental effects of anger and other negative emotions on cardiovascular physiology is an active area of investigation. Beta-blockers provide one simple intervention. In a pig model using lifting stress, similar to the protocol used by Lown *et al.* [25], Kirby *et al.* [37] compared induction of ventricular tachycardia in pigs with a baseline infarct at rest and with lifting stress. Induction of monomorphic ventricular tachycardia occurred earlier in the pacing protocol in the stressed state, in 50% of the pigs, and ventricular tachycardia was faster in the pigs during stress. However, both of these effects were decreased by beta-blockade. Beta-blockers have also been shown to diminish the usual withdrawal of vagal activity seen with laboratory stressors [38].

In patients with coronary artery disease, behavioral interventions aimed at stress reduction have shown excellent results, decreasing stress-induced ischemia, improving quality of life, and improving event-free survival [39,40]. Preliminary studies have similarly investigated the impact of behavioral interventions in decreasing the arrhythmogenic effects of negative emotion. Two successful studies [41,42] utilized group-based cognitive behavior therapy that incorporated elements of relaxation training and cognitive reframing, with significant benefits seen on measures of quality of life, anxiety and depression, activity level, and sexual functioning, particularly among those patients who received ICD shock(s) during the course of the study. Further, Chevalier *et al.* [43] have demonstrated in a small pilot study of 70 patients that this type of therapy, administered in small groups for 3 months after ICD implant, could reduce shock incidence. HRV was also higher, and HR lower, in the therapy-treated patients. A current National Institutes of Health (NIH)-funded randomized trial, the 'Reducing ICD Shock Treated Arrhythmias' study, will shed further light on the efficacy of stress reduction therapy in reducing ventricular arrhythmias as well as improving quality of life.

Conclusion

Anger and other strong emotions can and do trigger polymorphic, potentially life-threatening ventricular arrhythmias in vulnerable patients. Through autonomic changes, including increased sympathetic activity and vagal withdrawal, anger leads to increases in heterogeneity of repolarization as measured by TWA, known to be associated with arrhythmogenesis, as well as increasing inducibility of arrhythmia. Further delineation of mechanisms linking anger and arrhythmia, as well as determination of approaches to decrease the detrimental effects of anger and other negative emotions on arrhythmogenesis, are important areas of future investigation.

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 69).

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Figure 1. Electrograms of anger-triggered and non-anger-triggered ventricular arrhythmias (a) Monomorphic sudden-onset ventricular arrhythmia (non-anger-triggered), (b) monomorphic PVC-initiated pause-dependent ventricular arrhythmia (anger-triggered) and (c) polymorphic PVC-initiated pause-dependent ventricular arrhythmia (anger-triggered). PVC, premature ventricular contraction. Data from Stopper *et al.* [5].

Figure 2. Incidence of ventricular arrhythmias terminated by an implantable cardioverter-defibrillator in the months prior to and following the World Trade Center attacks of 9/11/2001
Data from Steinberg *et al.* [3].

Figure 3. Alteration of ventricular tachycardia termination by anger

(a) In the resting-awake state, ventricular tachycardia of CL 360ms is induced and terminated by ATP. (b) During anger recall, an identical ventricular tachycardia is induced but is accelerated by ATP into a zone requiring shock. CL, cycle length. Data from Lampert *et al.* [29].

Figure 4. Kaplan–Meier curves depicting survival from implantable cardioverter-defibrillator-treated arrhythmias in patients with anger-induced T-wave alternans in the top quartile as compared with the other quartiles
TWA, T-wave alternans. Data from Lampert *et al.* [34**].