Emotional and Physical Precipitants of Ventricular Arrhythmia

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Background—Observational studies have suggested that psychological stress increases the incidence of sudden cardiac death. Whether emotional or physical stressors can trigger spontaneous ventricular arrhythmias in patients at risk has not been systematically evaluated.

Methods and Results—Patients with implantable cardioverter-defibrillators (ICDs) were given diaries to record levels of defined mood states and physical activity, using a 5-point intensity scale, during 2 periods preceding spontaneously occurring ICD shocks (0 to 15 minutes and 15 minutes to 2 hours) and during control periods 1 week later. ICD-stored electrograms confirmed the rhythm at the time of shock. A total of 107 confirmed ventricular arrhythmias requiring shock were reported by 42 patients (33 men; mean age, 65 years; 78% had coronary artery disease) between August 1996 and September 1999. In the 15 minutes preceding shock, an anger level $\geq$3 preceded 15% of events compared with 3% of control periods ($P<0.04$; odds ratio, 1.83; 95% confidence intervals, 1.04 to 3.16) Other mood states (anxiety, worry, sadness, happiness, challenge, feeling in control, or interest) did not differ. Patients were more physically active preceding shock than in control periods. Anger and physical activity were independently associated with the preshock period.

Conclusions—Anger and physical activity can trigger ventricular arrhythmias in patients with ICDs. Future investigations of therapies aimed at blocking a response to these stressors may decrease ventricular arrhythmias and shocks in these patients. (***Circulation***. 2002;106:1800-1805.)

Sudden cardiac death has long been linked anecdotally with strong emotion. Informal case series[1,2] describe individuals experiencing cardiac arrest or sudden death in settings of acute grief, fear, or anger. Epidemiological studies have shown that sudden death increases in populations experiencing emotionally devastating disasters, such as earthquake or war.[3,4] Sudden death has also been linked in epidemiological studies to vigorous exercise, especially in less active individuals.[5] Exercise and emotion each increase sympathetic nervous system activation.[6,7] Either ischemia or arrhythmia could provide the physiological link among these stressors, catecholamines, and sudden death. Both anger[8,9] and vigorous exercise[10] can trigger nonfatal myocardial infarction, as well as transient ischemia.[11,12] Whether emotional or physical stress can directly trigger potentially fatal arrhythmias and how frequently this occurs had not been previously systematically investigated.

Indirect evidence suggests that emotional stressors do precipitate arrhythmic events. Ventricular tachycardia (VT) occurs more frequently on Mondays in working patients with implantable cardioverter-defibrillators (ICDs).[13] In addition, atrial and ventricular ectopy and nonsustained arrhythmias increase during the stress of being on-call in medical interns[14] and during exposure to a hostile environment in animals.[15] In invasive studies in animals[16] and humans,[7] psychological stress facilitates the induction of VT and makes termination more difficult. VT,[17] like sudden death,[18] occurs more frequently in the morning, the time of peak catecholamine levels[19] and lowest vagal tone,[20] further suggesting that sympathetic nervous system arousal can facilitate ventricular arrhythmia.

To investigate whether emotional or physical factors can trigger ventricular arrhythmias in patients with ICDs, we conducted a controlled, diary-based study of the events and emotions preceding VT or ventricular fibrillation (VF) that caused shock using a case-crossover design. Several factors make this population uniquely suited to investigate potential arrhythmia triggers. Many patients anecdotally report psychological or physical stressors preceding ICD shock. The generally rapid and successful termination of arrhythmic events leaves the patient with full recall of preceding events, unlike many cardiac arrest survivors. Also, shock identifies the occurrence of potentially fatal arrhythmias, and diagnostic data stored by the device confirms the presence of VT or VF.

Methods

Study Population

A total of 277 patients who had received ICDs for standard indications (clinical or inducible ventricular arrhythmias) and were followed at the Yale New Haven Hospital ICD Clinic were invited to

participate in the study between July 1996 and March 1999. Exclusion criteria were significant psychiatric illness and inability to read or be interviewed in English. Seventeen declined to participate, and 20 later withdrew. The study was approved by the Yale Human Investigation Committee, and all patients gave informed consent. The procedures followed were in accordance with institutional guidelines.

Structured Diary

Patients were given a structured diary to record their activities and mood states, as described and validated by Hedges et al[21] and used in previous studies of triggers of cardiac events.[11,12] The diary incorporated a 5-point Likert rating scale of intensity to evaluate levels of anger, anxiety, worry, sadness, happiness, and feelings of challenge, interest, and being in control (explained during diary orientation as "the ability to effectively respond to circumstances"). Exposure to an emotion was defined as a recording ≥3, which represented moderate or higher levels. The diary also included a checklist of specific activities. These were classified into 5 levels of physical intensity, as validated with activity monitoring as described by Patterson et al.[22] Level 1 corresponds to sleep or rest; level 2, activities of daily living, talking, or clerical work; level 3, driving; level 4, slow walking, recreational activity, housework, shopping, sex, or other physical activity; and level 5, effortful walking, stairs, or more vigorous activity.[11] Exposure to activity was defined as a recording ≥4 (mild-to-moderate). Patients were instructed to complete a diary page for 2 predefined hazard periods when they experienced a shock: the 15-minute period preceding shock and the 15-minute to 2-hour period preceding shock

Study Design

Patients were enrolled at the time of first ICD follow-up (2 months after implantation) and were given diaries. Those with previous implants were enrolled at ICD clinic or by phone. All received detailed orientation to the diary at enrollment. Patients were asked to call on any day of shock received, and they were then contacted by the study coordinator within 48 hours to review the diary questions with them and give guidance as needed. Patients were then asked to fill out a second diary page to serve as a control, for a period corresponding to the same day of the week and time of day that they had received the shock, but one week later. They received another call on that day as a reminder.

Data analysis was performed using the case-crossover method.[23] In this approach, which has been used previously in studies of triggers of cardiac events,[5,8,10,12] "case" information gathered at a time of interest is compared with "control" information from the same patient gathered at a comparable time (eg, 1 week later). For the current study, the 2 predefined hazard periods (0 to 15 minutes before shock and 15 minutes to 2 hours before shock) were compared for each patient with the same periods at the same time 1 week later.

Rhythm Analysis

Data stored by the ICD, including electrograms and event details, were reviewed to determine the rhythm at the time of shock. Only events showing VT or VF for which shock was received were included in the analysis. An arrhythmia requiring >1 shock for termination was counted as a single shock event.

Statistical Analysis

Comparison of the hazard versus control period for mood states and physical activity, dichotomized as above, was done by the McNemar test for matched case-control data.[23,24] Crude odds ratios (OR) were calculated in the standard manner for a matched case-control study.[24] Conditional logistic regression with person as the stratifying variable was used to adjust for multiple events occurring within a single individual, and adjusted ORs and 95% confidence intervals (CI) were calculated from the conditional logistic regression models. Multivariable analysis to determine independence of association for variables with significant univariate associations was performed with
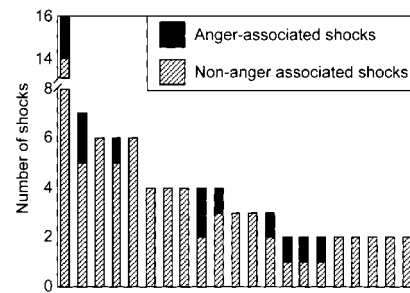

Figure 1. Distribution of all shocks and of shocks associated with anger in the 15-minute hazard period preceding shocks in patients with >1 shock. Each column represents one patient. Twenty-one additional patients reported one shock each, of which 5 were anger-associated.

nominal logistic regression, with interaction terms included where necessary (eg, emotion level by physical activity level).

Results

Shock Events and Patient Characteristics

Sixty-one patients recorded diaries for 187 shock events, each occurring on different days, between August 1996 and September 1999. Eighty events were excluded for the following reasons: in 14, no therapy was received ("phantom shocks"); 15 were antitachycardia pacing events that were interpreted by the patient as shock; 11 were due to atrial fibrillation; 5 were caused by sinus tachycardia; 2 were due to supraventricular tachycardia; 8 were caused by lead malfunction (in the same patient); and in 8, no electrograms were available. Seventeen were VT but did not have matching control period data completed. The remaining 107 shocked ventricular arrhythmia events from 42 patients and their matched control data comprise the analysis. Distribution of shock frequencies among the patients is shown in Figure 1. Patient characteristics are shown in Table 1.

Emotional Triggers

Tables 2 and 3 show the associations between each mood state in the preshock periods compared with the paired control periods, for the 0- to 15-minute and 15-minute to 2-hour hazard periods, respectively. Moderate anger (level 3 or above) occurred during 15% of the 0- to 15-minute preshock hazard periods, 3% of control periods, and 1% of both ($P<0.03$ in unadjusted analysis). The 17 anger-associated shocks occurred in 14 patients, with 3 patients

TABLE 1.   Patient Characteristics

	n=42
Male sex, n	33
Age, mean±SD, y	65±7
Coronary artery disease, %	78
Indication for ICD, %	
Sustained VT	50
VF or cardiac arrest	24
NSVT or syncope with inducible VT	26

VT indicates ventricular tachycardia; VF, ventricular fibrillation; and NS, nonsustained.

TABLE 2. Frequency of Emotions in the 15 Minutes Preceding Shock Compared With Control Period

	Shock Only, %	Control Only, %	Both, %	OR (Unadjusted)	OR (Adjusted)	95% CI (Adjusted)	P
Anger	15	3	1	5.33	1.83	1.04–3.16	<0.04
Anxiety	19	7	2	2.86	1.51	0.93–2.42	0.09
Worry	13	8	4	1.55	1.16	0.72–1.84	0.54
Sadness	7	4	2	2.0	1.22	0.67–2.25	0.52
Happiness	11	19	52	0.6	0.87	0.60–1.25	0.44
Challenge	20	9	7	2.1	1.24	0.85–1.83	0.27
Interest	13	12	44	1.07	1.02	0.70–1.50	0.92
In control	12	11	62	1.08	1.02	0.67–1.56	0.92

OR indicates odds ratio; CI, 95% confidence intervals. Mood states are dichotomized at level 3 or above. Unadjusted (crude) ORs calculated from McNemar χ^2. Adjusted ORs, CIs, and P values calculated with conditional logistic regression.

reporting 2 anger-associated shocks and 11 patients one each. The distribution of anger-associated shocks is shown in Figure 1. Anger was significantly associated with shock after adjustment for multiple events occurring within single individual (OR, 1.8; 95% CI, 1.04 to 3.16; $P<0.04$). Intense anger (level 5), was reported less frequently, occurring in 8% of 0- to 15-minute preshock hazard periods compared with 1% of control periods (Figure 2); this difference was significant in unadjusted analysis ($P<0.03$), although it was not significant after adjustment (OR, 2.4; 95% CI, 0.94 to 6.0; $P=0.06$).

Among the 17 shocks for which control data were not available, 2 were associated with anger ≥3 and 2 with anxiety ≥3 (12% for each). This did not differ from the proportion of shocks with control data that were anger- or anxiety-associated ($P>0.5$).

In unadjusted analysis, anxiety was significantly associated with shock in both the 15-minute and 2-hour hazard periods. The association of anxiety with shock did not remain significant after adjustment (for the 15-minute period: OR, 1.5; 95% CI, 0.93 to 2.42; for the 2-hour period: OR, 1.40; 95% CI, 0.87 to 2.2).

There was no difference in levels of other mood states (worry, sadness, happiness, or feelings of challenge, interest, or being in control) between the hazard period and the control period. Specific activities reported preceding shock that were potentially associated with emotional arousal included driving (n=6), arguing (n=2), and gambling and receipt of bad news (n=1 each).

Physical Activity

Vigorous activity (level 5) was rarely reported during either preshock periods (n=2) or control periods (n=1), and it was not significantly associated with shock. Patients were engaged in mild-to-moderate physical activity (level 4 or greater) within the 15 minutes preceding 53% of shocks and during 32% of control periods (OR, 3.87; 95% CI, 1.97 to 14.8; $P<0.001$.) After adjustment for multiple events, this level of activity remained associated with shock (OR, 1.46; 95% CI; 1.02 to 2.08; $P<0.04$). Activity was associated with the preshock period independent of emotion, and there was no activity-emotion interaction ($P=NS$ for interaction). Specific physical activities reported more commonly in the 15 minutes preceding shock than during control periods included walking slowly (40 versus 19), physical activity at work (9 versus 4), vigorous walking (2 versus 1), and recreational activity (7 versus 5). Activity in the 2-hour period did not differ between preshock and control periods.

Discussion

In this study of patients with ICDs, moderate levels of anger were more likely during the period preceding shock for

TABLE 3. Frequency of Emotions in the 15 Minutes to 2 Hours Preceding Shock Compared With Control Periods

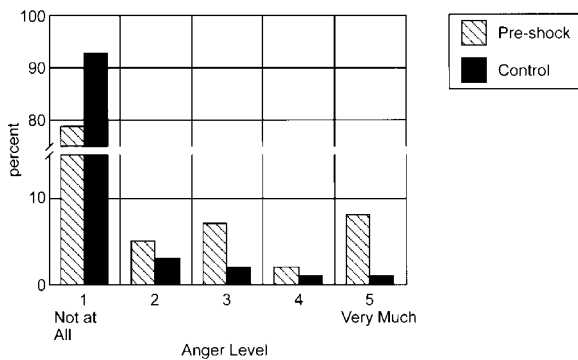|	Shock Only, %	Control Only, %	Both, %	OR (Unadjusted)	OR (Adjusted)	95% CI (Adjusted)	P
Anger	10	4	3	2.75	1.35	0.77–2.35	0.29
Anxiety	15	5	8	3.2	1.39	0.87–2.23	0.17
Worry	8	8	3	1	1.00	0.60–1.68	1.00
Sadness	12	5	0	2.6	1.54	0.83–2.87	0.17
Happiness	10	18	52	0.57	0.86	0.59–1.25	0.43
Challenge	23	12	10	1.92	1.24	0.86–1.78	0.25
Interest	13	10	42	1.27	1.05	0.73–1.52	0.78
In control	15	6	58	2.28	1.20	0.81–1.80	0.36

See footnote for Table 2.

Figure 2. Description of anger levels during the period 15 minutes preceding shock and during the control period. In analysis of anger level dichotomized at ≥3, anger was significantly associated with the preshock period.

spontaneous VT or VF than during a paired control period 1 week later, suggesting a triggering of arrhythmia by anger in a group of patients with a history of arrhythmia or known arrhythmia risk. Ventricular arrhythmia was also associated with mild-to-moderate activity such as that occurring in daily life. Vigorous activity was reported only rarely in this study.

Previous studies have indirectly suggested a relationship between emotion and arrhythmia. Descriptive and epidemiological studies have shown a link between sudden cardiac death and stressful stimuli such as population catastrophes[3,4] and personal grief.[1,2] However, in these studies, actual mode of death was unknown and whether ischemia or arrhythmia provided the physiological link could not be determined. Although ischemia and infarction have previously been shown to be triggered by psychological factors,[8,9,11,12] whether arrhythmia can be similarly triggered had remained unknown. In one case series,[1] 19% of patients interviewed after cardiac arrest or symptomatic VT described episodes of emotional distress in the 24 hours preceding the event, mostly anger. However, that study was not controlled and relied on memories of arrest patients. In the current study, each subject acted as his or her own control, using the case-crossover approach, and the rapid therapy delivered by the ICD eliminated the retrograde amnesia often experienced by cardiac arrest survivors. Although benign arrhythmias have been shown to increase in times of stress in patients with normal hearts,[14,15] the current study describes life-threatening ventricular arrhythmia spontaneously triggered by emotions of anger in patients with known susceptibility.

Experimental studies show electrophysiological properties can be altered by stress, suggesting potential mechanisms through which triggering of arrhythmia may occur. Toivonen et al[25] showed changes in repolarization in healthy medical interns exposed to the sudden stress of an on-call alarm. T-wave alternans, a marker of repolarization heterogeneity that correlates with risk of arrhythmia, has been shown to increase with induction of an anger-like state in dogs.[26] Alterations in the signal-averaged ECG have also been seen during mental stress, implying stress-induced changes in conduction.[27] Our group has used an anger recall task to examine changes in induced arrhythmia during ICD testing in the electrophysiological labo-

ratory. Anger altered the VT circuit, accelerating VT and/or rendering it more difficult to terminate; these changes correlated with increases in catecholamine levels.[7] These electrophysiological changes in repolarization and conduction brought on by anger or stress may lead to the triggering of spontaneous arrhythmia seen in the current study.

The present study showed a higher incidence of anger-associated triggering than that reported for myocardial infarction in the Myocardial Infarction Onset Study, which showed 2.4% of events to be associated with anger.[8] It may be that the threshold for triggering recurrent ventricular arrhythmia by emotion is lower than that for infarction. Also, ICD shocks occur in the outpatient setting, and although patients were asked to call in with all shocks received, not all enrolled patients remembered or chose to participate. Whether individuals who chose to participate had a different propensity to anger or to triggering of arrhythmia by anger cannot be determined. Although also dependent on outpatient participation, the ongoing Triggers of Ventricular Arrhythmia trial may help further quantify the specific risk of a given episode of anger in triggering VT or VF.

Patients in this study experienced varying numbers of arrhythmic events, ranging from 1 (half of the patients) to 16. The phenomenon of anger-triggered events was seen both in patients reporting multiple events and those reporting just one. Of patients with multiple events, most reported a mixture of emotion-associated and unassociated events. Whether some individual patients with multiple events have a greater propensity to triggering of arrhythmia by emotion requires further study.

Understanding arrhythmia triggering by emotional factors may help lead to therapeutic strategies that will decrease arrhythmia and shock frequency in patients with ICDs and potentially decrease the incidence of sudden death. Long-term, both healthy individuals and patients after myocardial infarction who have a greater propensity to anger or anxiety have a higher incidence of sudden death[28,29] and ischemic events.[30] In one study, higher scores on measures of anxiety shortly after implantation of an ICD predicted subsequent arrhythmia.[31] Avoidance of anger or anxiety is not possible for individuals leading a full life. However, psychosocial and behavioral interventions, such as stress management, and anger-reducing interventions have been shown in some,[32] although not all,[33] studies to decrease recurrent ischemic events. The application of these approaches in patients with ICDs warrants further investigation.

The present study showed an association between mild-to-moderate activity, such as slow walking or shopping, and incidence of arrhythmia. Together with previous studies showing that these activities can be associated with ambulatory ischemia,[11,12] these data suggest that even modest activities such as those occurring during daily life can provide sufficient sympathetic nervous system activation to trigger cardiovascular events.

Limitations

Although patients were instructed to focus on the periods preceding shock, the possibility of recall bias, with patients attributing postshock feelings of anger or anxiety to the

preshock period as well, cannot be excluded. However, because other emotions such as worry, which might have been susceptible to similar bias, did not differ, recall bias seems unlikely as an explanation of the findings.

Importantly, we cannot exclude the possibility that patients may have altered their behavior during the control period 1 week after the shock in response to the shock itself or in anticipation of filling out the diary. Although patients were instructed to make no change in their daily life to minimize this possibility, the magnitude of this potential bias cannot be assessed. Literature evaluating patients' changes in behavior in response to shock is sparse. Although fear of exertion after an ICD storm has been described in one case report,[34] a larger series showed ICD patients decreased their activity after inappropriate but not appropriate shocks[35] (those described in the present study were appropriate shocks). Previous studies of triggers of cardiovascular events have used a variety of control periods, each with its own limitations. These have included filling out multiple diaries,[11,12] which may alter patients' behavior to an even greater degree; the use of the preceding day as a control period,[8,10] which may be subject to significant recall bias, particularly for mood; and the use of "usual frequency" of activities and emotions to calculate a risk ratio,[5,8,10] which fails to take into account the effects of circadian variation, day, and season. A control period at the same time 1 week later was chosen for the current study to replicate the circadian and daily timing of the event, which are factors known to influence occurrence of ventricular arrhythmias,[13,17] while minimizing recall bias.

Anxiety, which preceded 19% of shocks, was significantly associated with shock in the unadjusted analysis but not after adjustment for multiple events. Whether this is because anxiety as a trigger of arrhythmia is a more individual-specific phenomenon or whether anxiety would prove to be a significant trigger in a larger study population requires further evaluation.

For 17 shocks, no control diary had been filled out, which could have created a selection bias if those with emotionally-triggered shocks had a different propensity to completing the control diary. However, because the proportion of these shocks that were anger- or anxiety-associated was similar to those shocks with control data available, it is unlikely that exclusion of these events skewed the results.

In addition, only episodes of arrhythmia that resulted in shock were included, because most patients are unaware of episodes of anti-tachycardia pacing. Whether these more stable arrhythmias can also be triggered by emotion remains unknown.

Conclusion

Anger can trigger ventricular arrhythmias in patients with ICDs, as can physical exertion. Future investigations of therapies aimed at blocking response to these stressors may decrease ventricular arrhythmias and shocks in these patients.

Acknowledgments

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