Differentiation in the Momentary Rating of Somatic Symptoms Covaries With Trait Emotional Awareness in Patients at Risk for Sudden Cardiac Death

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Objectives: Somatic symptom ratings covary with neuroticism. Yet, people vary from one another in their ability to report their own emotions and differentiate them from bodily sensations. We hypothesized that stressed individuals with greater emotional awareness would experience somatic symptoms in a more differentiated way independent of neuroticism. **Methods:** Over 3 days, ecological momentary assessments were completed in 161 patients (72.6% female; mean age, 35 years) with Long QT Syndrome, a genetic disorder associated with increased risk for sudden cardiac death. Patients were paged randomly ten times per day to report their momentary experience of nine somatic symptoms (e.g., headache, sore throat, tiredness) as well as other variables. We examined the intercorrelation between somatic symptom ratings, reasoning that greater intercorrelation among ratings indicated less differentiation. Subjects completed measures of neuroticism, depression, and the Levels of Emotional Awareness Scale, a trait measure of the tendency to experience emotions in a complex and differentiated way. **Results:** Higher Levels of Emotional Awareness Scale-Self scores were associated with greater differentiation in the momentary rating of somatic symptoms ($p < .001$) in men and women independently. This association did not change after removing variance due to neuroticism, depression, or symptom intensity. **Conclusions:** Among individuals stressed by having a life-threatening condition, those who are more emotionally aware report somatic symptoms in a more differentiated way. These findings regarding symptoms largely unrelated to the disorder are consistent with other evidence that medically unexplained physical symptoms, which tend to be nonspecific, may be accompanied by relatively undifferentiated negative affect. **Key words:** somatic symptoms, negative affect, differentiation, emotional awareness.

BDI = Beck Depression Inventory; **EMA** = ecological momentary assessments; **LEAS** = Levels of Emotional Awareness Scale; **LQTS** = Long QT Syndrome; **PANAS** = Positive and Negative Affect Scale; **PDA** = personal digital assistant; **TAS-20** = 20-item Toronto Alexithymia Scale.

INTRODUCTION

Presentation of physical symptoms for which no organic basis can be found, a phenomenon known as somatization (1), is extremely common and is estimated to account for 10% to 30% of all outpatient medical visits (2). Somatic symptoms unrelated to physical disease are thought to arise from amplification of the normal physiological concomitants of emotional arousal (3). Studies (4) in healthy volunteers have demonstrated that both state and trait negative affect are correlated positively with the number of self-reported somatic symptoms. In addition, the combination of negative affect and self-focus, not either one alone, predicts higher levels of somatic symptom reporting (5). Consistent with these preclinical studies, the most common causes of physical complaints for which no organic basis can be found are depressive and anxiety disorders (2). It is well known, for example, that pain and depression each increase the intensity of the other and that treatment of depression tends to ameliorate the suffering component of pain (6).

When the reporting of physical symptoms is both persistent and dissociated from objective findings, however, the rela-

tionship to self-reported emotion is more complicated. Many patients with functional somatic syndromes, such as fibromyalgia or irritable bowel syndrome, do not link their somatic symptoms with emotions (7). For example, they do not acknowledge and may even resist the notion that negative emotions are contributory or that cognitive or behavioral interventions can influence their condition. They firmly believe that their problem is in the body and often resist considering that psychological factors may play a role (8). Symptoms in functional somatic syndromes tend to be diffuse, nonspecific, and ambiguous (3), giving them a nonspecific or undifferentiated quality.

A phenomenon known as "alexithymia" has been used to explain such a stance (9). Alexithymia, meaning "lacking words for emotion," consists of a deficit in the cognitive processing of emotion that includes difficulty distinguishing between feelings and bodily sensations (10). An association between alexithymia and somatization has been reported in several empirical studies (11,12), but not all (13,14). According to this perspective, persistent medically unexplained symptoms reflect the somatic presentation of emotional distress. However, the leading measure of alexithymia, the 20-item Toronto Alexithymia Scale (TAS-20) (15,16), tends to show strong positive correlations with self-reported negative affect (17). Moreover, contrary to expectations, recent evidence (18) suggested that higher scores on the TAS-20 are associated with greater range and greater differentiation of emotional experiences. Given that the concept of alexithymia originated with the goal of capturing impairment in experiencing and describing emotions (9), difficulties arise when attempting to use the TAS-20, a self-report measure, to disentangle reported emotional distress from the type of dysfunctional processing of distress that leads to it not being reported. This difficulty is consistent with the general fallibility of self-assessments (19).

Lane and Schwartz (20) developed an alternative theoretical approach, called "levels of emotional awareness," which

explains how putting emotions into words transforms emotional experience from a bodily, sensorimotor state to an emotional feeling state. Structurally parallel to Piaget's stages of cognitive development, this model conceptualizes awareness of one's own and others' emotions in a hierarchically ascending order: 1) physical sensations; 2) action tendencies; 3) single emotions; 4) blends of emotion; and 5) blends of blends of emotion. A key aspect of this model is that the levels are related to one another in a nested hierarchy (21). Thus, as each level is reached, emotional experience becomes more differentiated and integrated and modulates the preceding levels so that they too become more differentiated and integrated.

This model holds that somatic sensations (Level 1) and action tendencies (Level 2) do not disappear when emotional experience reaches Level 3 (a discrete feeling state, such as anger) but rather continue as components of emotional experience. Moreover, higher levels of emotional awareness are hypothesized to be associated with more differentiated somatomotor (e.g., action tendencies, Level 2) and more differentiated visceromotor (somatic sensations, Level 1) manifestations of emotion. The present study was undertaken to explore the latter hypothesis.

One can measure an individual's level of emotional awareness using the Levels of Emotional Awareness Scale (LEAS), a paper-and pencil performance measure that asks subjects to describe in an open-ended manner how they and another person would feel in scenarios described in two to four sentences (22). Scoring of the LEAS consists of the degree of differentiation of words used to describe the experiences of self and other following the five-level hierarchy just described. Unlike the TAS-20, the LEAS does not rely on the respondents' own ratings of their ability to put emotions into words or their ability to differentiate between emotions and somatic sensations.

The LEAS correlates moderately positively with two cognitive-developmental measures (22): the Sentence Completion Test of Ego Development by Loevinger et al. (23,24); and the cognitive complexity of descriptions of parents by Blatt and colleagues (25). These results support the claim that the LEAS is measuring a cognitive-developmental continuum and that the LEAS is not identical to these other measures. Two independent studies of undergraduates, involving 63 subjects and 55 subjects, respectively, revealed that greater emotional awareness is associated with greater self-reported impulse control ($r = .35$, $p < .01$; and $r = .30$, $p < .05$, respectively) (Dr. Lisa Feldman-Barrett, unpublished observations). Given that the five levels are hierarchically related and that action tendencies represent Level 2 function, this finding is consistent with the theory that functioning at higher levels of emotional awareness (Levels 3–5) modulates function at lower levels (actions and action tendencies at Level 2) (21).

Several additional findings indicated that higher scores on the LEAS are associated with more differentiated (i.e., more accurate) emotion information processing. Scores on the LEAS are correlated positively with the understanding emo-

tions section of the Mayer Salovey Caruso Emotional Intelligence Test (26), the perception of emotions in stories of the Multifactor Emotional Intelligence Scale (27), the Range and Differentiation of Emotional Experience Scale (28), the accuracy of facial emotion recognition (29,30), and self-reported empathy (27). A key next step is to determine whether higher levels of emotional awareness are associated with more differentiated somatic symptoms. This hypothesis follows directly from both theory and empirical evidence that higher levels of emotional awareness are associated with greater differentiation of function at lower levels; e.g., Level 3 experiences should be associated with greater differentiation in somatic symptoms (Level 1) than Level 2 experiences. We also hypothesize that this association will hold for the "Self"-score on the LEAS in contrast to the "Other" score on the LEAS. This latter hypothesis follows from the literature linking somatic symptoms with self-focus (5).

To test these hypotheses, we studied emotional awareness and somatic symptoms in patients with a physical disease of known etiology, Long QT Syndrome (LQTS). LQTS is a genetic disorder that affects repolarization dynamics in the heart and puts affected individuals at risk for sudden cardiac death (31,32). LQTS is not thought to affect the brain (33), and there is no known evidence of psychological abnormality. Moreover, LQTS patients typically do not have physical symptoms related to their vulnerability to serious cardiac arrhythmias, which occur rarely. The fact that they are at risk for sudden cardiac death, however, is an ongoing source of stress. Because somatic symptoms unrelated to physical disease arise from amplification of the normal physiological concomitants of emotional arousal (3), we hypothesized that, among these stressed individuals, somatic symptoms largely unrelated to their physical disease would be rated in a more differentiated manner the higher their level of emotional awareness.

A potential disadvantage of studying patients with LQTS is that many take β blockers for its cardioprotective effects (34). Because β blockers interrupt sympathetically-driven arousal processes, their use could potentially influence physical symptoms. We, therefore, compared somatic sensations and their relationship to LEAS scores in patients who were taking β blockers with those who were not. Based on the assumption that β blockers reduce somatic symptoms (35), we predicted that the association between LEAS scores and somatic symptom differentiation would be reduced in patients taking β blockers.

To quantify somatic symptom differentiation, we examined the intercorrelation (36) of somatic symptoms in an Ecological Momentary Assessment (EMA) study in which subjects were asked to rate their physical symptoms on 30 separate occasions. We reasoned that if subjects rated their somatic sensations in a differentiated manner, the intercorrelation between somatic symptom ratings across occasions would be low.

We, therefore, tested the following hypotheses: 1) higher LEAS scores, particularly on the "Self" subscore, will be associated with greater differentiation in the rating of somatic

symptoms; 2) this association will be independent of self-reported negative affect; 3) patients taking β blockers will have less intense physical symptoms than those not taking β blockers; 4) the association between LEAS score and somatic symptom differentiation will be stronger in patients not taking β blockers.

METHODS
Participants

This study was undertaken to empirically evaluate mechanisms underlying clinical observations that emotions can trigger cardiac events and sudden cardiac death (37). This larger study examined the association between momentary experiences and ambulatory electrocardiographic (ECG) changes in patients with LQTS.

The study received approval from the appropriate Institutional Review Board/ethics committees. All patients provided their informed consent. Data were collected between January 2003 and July 2006.

Participants were selected from the International Long QT Syndrome Registry located in Rochester, New York. In all, 161 participants (72.6% female) located throughout the United States completed the protocol, with a mean age of 34.9 years (range, 16–50 years of age). Half ($n = 80$; 49.7%) of the patients had had a previous arrhythmogenic cardiac event; the majority ($n = 101$; 63%) were taking β blockers; and a minority ($n = 18$; 11%) had implanted cardioverter defibrillators. There was no clinical or ECG evidence that serious or life-threatening arrhythmias occurred during this study. Only 1.7% of pages occurred during exercise.

Psychometric Measures
LEAS

The ten-item version of the LEAS (38) was used consisting of ten vignettes that describe emotion-provoking interactions between two persons. Participants are asked to describe how they would feel as the protagonist of each scene and how "the other person" would feel. Answers are quantified using scoring rules derived from a Piagetian cognitive-developmental theory of emotional awareness (20,22). Scores are assigned for the categories "Self," "Other," and "Total," with lower scores reflecting lower levels of development.

All scoring was performed by one expert LEAS scorer trained by the creator of the scale with 12 years of LEAS-scoring experience. This rater recently achieved interrater reliability of $r = .98$ with a second rater trained independently at another institution who rated the same 44 20-item protocols. To assess intrarater reliability, 15 protocols from this study were scored by this rater a second time 2 years after the first rating and blinded to the first rating. The correlations between pairs of Self, Other, and Total ratings were .995, .995, and .993 respectively.

Neuroticism

We used the Neuroticism subscale of the Revised NEO Personality, a well-validated instrument for measuring the "Big 5" dimensions of personality (39). In the currrent sample, Cronbach's α was 0.94, indicating high internal consistency reliability.

Beck Depression Inventory (BDI)

We used the BDI version 1A, which is a 21-item self-report scale that assesses cognitive, affective, and somatic symptoms of depression "over the past week including today," using a multiple choice format (40). Four statements per item are rank ordered to reflect the range of symptom severity. Numerical values of 0, 1, 2, and 3 are assigned to each statement to indicate severity. The items are summed to yield a single depression score. Cronbach's α was 0.86.

Positive and Negative Affect Scale (PANAS)

We used the positive and negative affect scales from the widely used PANAS measure (41). Each scale has ten items, answered on 5-point interval scales according to how often the participant "generally feels this way." Cronbach's α was 0.86 for each scale.

Procedure

Due to the rarity of LQTS and lack of a sufficient number of patients in any one location, we made home visits to LQTS patients throughout the United States. Data collection took place over 3 days. Data collection began on day 1 with completion of questionnaires. The LEAS was always presented first and other questionnaires followed. Each day, participants engaged in their typical daily activities and were paged (on vibration mode) ten times per day at random times, for a total of 30 signals per participant. Patients responded to the page by answering a series of questions administered by a personal digital assistant (PDA) (Zire 21 running Palm OS 5.2.1, Palm Inc., Sunnyvale, California). PDAs were programmed with the Experience Sampling Program, which is open-source software used widely (available at http://www.experience-sampling.org/).

Somatic symptoms were assessed, using a measure developed by Emmons (42), which asks the extent to which the subjects had experienced each of nine physical symptoms since the last signal: headache; stomach ache/pain; chest/heart pain; sore throat/cough; runny/congested nose; faintness/dizziness; shortness of breath; stiff/sore muscles; and tiredness. A 7-point (0 = not at all; 6 = extreme) intensity scale was used. These terms were presented consecutively in the EMA protocol.

Pages were delivered according to a modified random schedule. All signals were scheduled to be delivered during a 12-hour window in the 14-hour period between 8 AM and 10 PM (or comparable hours for those participants with a different waking schedule; only one subject received pages after midnight). Signals were constrained so that no two signals could occur within 60 minutes of each other. Participants were instructed to turn on their Palm PDA as soon as possible after the page, to begin responding immediately, and to complete the 60-item protocol without interruption.

Compliance statistics for EMA ratings were computed by comparing the scheduled time of the page to the record of when recording began on the Palm PDA. Of the pages sent, 93.0% were responded to. Of these, 62.5%, 84.0%, 92.2%, 95.5%, and 96.9% were begun within 1, 5, 10, 15, and 20 minutes of the page, respectively. When we computed the percentage of reports that were begun within 10 minutes of the page for each participant, the median participant had a 96.7% compliance rate; using 15 minutes as a cutoff, the median compliance rate was 98.3%. More than half the sample began all or all but one of their reports within 10 minutes, and only 19 participants began 4 or more reports more than 15 minutes after the page. A repeated-measures analysis of variance examining the number of missed signals for each of the 3 days of the EMA period showed that compliance did not differ significantly across the 3 days, $F(2,165) = 0.20$, $p < .85$.

These compliance statistics are very high for EMA research, based on comparable studies reported in the literature. To include as much data as possible, we used all reports begun within 15 minutes of the page. This cutoff is well within the range typically recommended for EMA and similar protocols (43–45).

Statistical Analysis

To assess differentiation, we used Barrett's (36) method for assessing the magnitude of intercorrelations among reported experiences, which she termed "granularity." This measure indexes the extent to which people describe their experience at each report with similar or different ratings on all symptoms. We first computed, separately for each person, the correlation between each pair of somatic symptoms (36 correlations, based on nine symptoms) across the approximately 30 EMA reports. We then averaged these values for each person, after transforming them with Fisher's r-to-z procedure (46). This provides an index of symptom differentiation. High values indicate that participants tended to rate all nine somatic symptoms in much the same way at each report, whereas low values indicate that participants rated the nine symptoms differentially. For reporting purposes, the Fisher's z values have been converted back to raw correlation coefficients.

A total somatic symptom intensity score was also computed. For this measure, Cronbach's α was 0.65.

RESULTS

Preliminary Analyses

Regarding the LEAS, scores on the ten-item LEAS for Self (mean = 30.64; standard deviation [SD] = 5.27), Other (mean = 26.22; SD = 4.97), and Total (mean = 33.90; SD = 5.21) were typical of men and women in this age range. The two subscales of the LEAS were significantly correlated, r (142) = .61, $p < .001$. Independent sample t tests indicated that women scored higher than men on the Self subscale of the LEAS (mean values = 31.2 and 28.3, respectively), t (138) = 2.94, $p < .005$. The difference between men and women on the Other subscale was not significant, $p > .20$.

Regarding somatic symptoms, there were no significant sex differences on the overall level of symptoms or daily average per symptom (male mean = 0.26; female mean = 0.32; t (164) = 1.05; NS). Faintness/dizziness was one symptom that would likely be elevated if cardiac arrhythmic events occurred. Somatic symptoms, including dizziness, were at a low level.

Regarding depression, BDI scores were within normal limits: mean = 6.2; SD = 5.9; median = 5. Scores were distributed as follows: 126 (78.3%) scored 0 to 9 (normal); 21 (12.8%) scored 10 to 15 (minimal depression); 10 (6.2%) scored 16 to 19 (mild-to-moderate depression); 3 (2.0%) scored 20 to 29 (moderate-to-severe depression); and 1 (0.6%) scored ≥30 (severe depression). These data indicate that relatively few subjects in this sample had elevated levels of state depression.

Before the analysis, the data were checked for outliers, restriction of range, and nonlinear associations, using standard techniques (47). No evidence was found. Table 1 reports correlations among the key variables of this research, separately by sex.

Primary Analyses

As hypothesized, the intercorrelation of somatic symptom ratings (the inverse of differentiation) was negatively correlated with the Self-LEAS, r (142) = −.38, $p < .001$. Also, as expected, the correlation of somatic symptom differentiation with the LEAS Other subscale was not significant, r (142) =

−.13, $p > .10$. These correlations differed significantly from each other by a test for dependent correlations in a single sample, t (139) = 3.31, $p < .001$. Thus, higher levels of emotional awareness with regard to self, but not with regard to others, were associated with more differentiated somatic symptom reports.

We repeated these analyses to explore possible differences between men and women. The same general pattern was found for both sexes. Among men, symptom differentiation was significantly correlated with the Self-LEAS score, r (38) = −.52, $p < .001$, but not the Other-LEAS score, r (38) = −.21, $p > .20$. Similarly, among women, symptom differentiation was significantly correlated with the Self-LEAS score, r (104) = −.26, $p < .01$, but not the Other-LEAS score, r (104) = −.07, $p > .50$.

Moderating Role of β Blockers

Contrary to our hypothesis, preliminary analyses showed that the nine symptoms used in this research did not differ significantly between individuals who were ($n = 101$) and were not ($n = 60$) using β blockers. To test for an interaction between LEAS and β blocker status, we used hierarchical multiple regression (46), in which the product of centered LEAS scores and a dummy variable for β blocker status was added to an equation predicting symptom differentiation, after main effects for these variables had been included. This analysis revealed a significant interaction between β blocker status and LEAS-Self scores, R^2 change = .026, $\beta = -0.022$, t (137) = −2.09, $p < .04$. A similar result was obtained for the interaction between β blocker status and LEAS-Other scores, R^2 change = .050, $\beta = -0.024$, t (137) = −2.71, $p < .01$. The results for the 43 patients on centrally and peripherally acting β blockers (propranolol and metoprolol) and the 58 patients on peripheral β blockers (e.g., atenolol) did not differ (t (99) = 0.68 and 1.26, NS, for Self and Other, respectively).

To identify the nature of the obtained interaction effects, we conducted tests of the simple slopes (48,49). Among persons not receiving β blockers, slopes for the association between Self-LEAS and Other scores and symptom differentiation were not significant (for the Self-LEAS, ß = 0.01,

TABLE 1. Correlations Between LEAS, Symptom, and Affect Measures for Men and Women

	Self-LEAS	Other-LEAS	Symptom Differentiation	Mean Level of Symptoms	Neuroticism	BDI	Negative Affect	Positive Affect
Self-LEAS		0.62**	−0.52**	0.25	0.00	0.12	0.04	−0.07
Other-LEAS	0.63**		−0.21	0.06	0.00	0.12	0.02	0.15
Symptom differentiation	−0.26**	−0.07		−0.18	−0.22	−0.14	−0.10	0.21
Mean level of symptoms	0.11	0.12	0.18		0.20	0.32*	0.12	−0.16
Neuroticism	0.11	0.03	−0.05	0.32**		0.73**	0.77**	−0.65**
BDI	0.18	0.15	0.03	0.50**	0.70**		0.70**	−0.51**
Negative Affect	0.17	0.10	0.14	0.47**	0.63**	0.64**		−0.43**
Positive Affect	0.04	0.03	0.14	−0.23*	−0.56**	−0.44**	−0.35**	

Values above the diagonal report the correlations for men; values below the diagonal report the correlations for women.
* $p < .05$; ** $p < .01$.
LEAS = Levels of Emotional Awareness Scale; BDI = Beck Depression Inventory.

$t(137) = 0.06$, NS; for Other-LEAS, $\beta = 0.28$, $t(137) = 1.61$, $p > .10$). Among persons who were receiving β blockers, both of these slopes were significant: for the Self-LEAS, $\beta = -0.45$, $t(137) = -5.53$, $p < .001$; for Other-LEAS, $\beta = -0.26$, $t(137) = -2.69$, $p > .01$). Thus, for persons taking β blockers, the higher their LEAS scores, the lower their symptom intercorrelation scores (more differentiated symptom reports).

Examination of Potential Artifacts

We conducted several additional analyses to rule out potential artifacts. First, often a correlation could not be computed involving one or another symptom because participants reported no instances of that symptom across their 30 EMA reports (resulting in zero variance). We, therefore, computed the number of correlations out of the maximum of 36 that went into each participant's symptom differentiation score. This value was significantly related to Self-LEAS, $r(142) = .17$, $p < .05$, indicating that more emotionally aware persons recorded a greater number of nonzero values for symptoms. Relevant to our hypothesis, when we recomputed correlations between the Self-LEAS and symptom differentiation partialling out the number of available correlations, the value remained significant and virtually identical in magnitude, $r(139) = -.36$, $p < .001$. The same was true in separate analyses of men, $r(35) = -.46$, $p < .002$, and women, $r(101) = -.26$, $p < .01$. Other-LEAS correlations with symptom differentiation remained nonsignificant.

Second, it is possible that symptom differentiation was confounded with mean level of symptoms reported. Higher Self-LEAS scores were significantly correlated with higher symptom reports, $r(142) = .16$, $p < .05$. Partialling out the mean symptom level again left the main hypothesized correlation essentially unchanged, $r(139) = -.36$, $p < .001$. The same was again true for men, $r(35) = -.46$, $p < .002$, and for women, $r(101) = -.26$, $p < .01$. Other-LEAS correlations with symptom differentiation remained nonsignificant.

Third, it is well established that symptom reports are correlated with the personality trait of Neuroticism (50), and we wanted to determine whether our results were attributable to the tendency of persons high in Neuroticism to report higher symptom levels. Neuroticism (mean $= 128.90$; SD $= 25.4$) was uncorrelated with either the Self- or Other-LEAS ($r(156) = .09$ and $.02$, respectively; both NS). When we repeated the main analyses partialling out Neuroticism, the key correlations remained essentially the same between the Self-LEAS and symptom differentiation, $r(134) = -.36$, $p < .001$. The same was true for men, $r(32) = -.49$, $p < .001$, and for women, $r(99) = -.26$, $p < .01$. Other-LEAS correlations with symptom differentiation remained nonsignificant. Partial correlations that removed variance due to BDI scores similarly did not alter the results. The partial correlation between the Self-LEAS and symptom differentiation was $r(134) = -.38$, $p < .001$. Among men, the correlation was $r(32) = -.54$, $p < .001$; among women, it was, $r(99) = -.27$, $p < .01$. Other-

LEAS correlations with symptom differentiation remained nonsignificant.

Similar analyses that controlled for PANAS Positive Affect (mean $= 2.15$; SD $= 0.77$) and Negative Affect (mean $= 0.40$; SD $= 0.32$) scores also had negligible effects on the primary analyses reported above. When controlling for negative affect, the correlation between the Self-LEAS and symptom differentiation was $r(135) = -.38$, $p < .001$. Among men, the correlation was $r(34) = -.52$, $p < .001$; among women, it was $r(98) = -.27$, $p < .01$. Other-LEAS correlations with symptom differentiation remained clearly nonsignificant. After controlling for positive affect, the correlation between the Self-LEAS and symptom differentiation was $r(135) = -.39$, $p < .001$. Among men, the correlation was $r(34) = -.52$, $p < .001$; among women, it was $r(98) = -.27$, $p < .01$. Other-LEAS correlations with symptom differentiation again remained clearly nonsignificant.

DISCUSSION

We studied patients with LQTS, a rare genetic syndrome that puts affected individuals at risk for sudden cardiac death. The disease is unusual in that it is life threatening; yet, patients do not experience manifestations of their condition under routine circumstances. There was no clinical or ECG evidence of serious arrhythmias during this study. However, because LQTS is life-threatening, its inherent ongoing stress made the present sample especially suitable for examining whether individual differences in processing emotional arousal influence differentiation in somatic symptom reports. Across the entire sample, in both men and women, the predicted relationship between greater trait emotional awareness and greater differentiation in the momentary rating of somatic symptoms was observed.

In this sample, 63% of LQTS patients were taking β blockers. Beta blockers reduce mortality in LQTS by blocking the effects of sympathetic discharge during physiological or mental challenge (34). As this study was conducted during routine daily activities, this protective effect was probably relatively infrequent (e.g., subjects were exercising during only 1.7% of pages). Contrary to expectations, we found that mean levels of somatic symptoms did not differ between those on β blockers and those not on β blockers for any of the nine somatic symptoms rated in the EMA protocol. Moreover, whether patients were on peripheral-only β blockers or mixed central and peripheral effects did not influence these results. These findings are consistent with the conditions of the study in which high levels of arousal were infrequent.

The absence of differences in somatic symptom intensity as a function of β blocker status contradicted the fundamental assumption underlying our prediction that the relationship between emotional awareness and somatic symptom differentiation would only be observed in patients not taking β blockers. We observed the opposite, i.e., the relationship between greater emotional awareness and greater somatic symptom differentiation was present only in patients who received β blockers.

This interaction, in light of the absence of an association between β blocker status and intensity of somatic symptoms, is consistent with the hypothesis that the relationship between LEAS and somatic symptoms would be most evident under conditions of stress. LQTS patients who are on β blockers are at greater risk for mortality than those not on β blockers (34). Indications for β blocker use in LQTS patients include: a) being more symptomatic (e.g., palpitations or dizziness); b) having more severe clinical manifestations (more frequent arrhythmogenic events); c) having higher familial risk (family history of sudden cardiac death in close relatives); or d) other clinical factor(s) that increases risk, such as longer QT interval. By contrast, those not on β blockers have a genetic mutation for LQTS but are not considered to be at high risk for mortality. As such, compared with those not on β blockers, patients on β blockers are under greater stress from LQTS. Moreover, although β blockers protect against mortality, they provide only partial protection in that patients with LQTS on β blockers remain at higher mortality risk than those not on β blockers (34). Due to their higher level of stress, one would expect that the relationship between emotional awareness and the degree of differentiation in somatic symptom reporting would be more evident in the β blocker group, which is what we found. Therefore, the study findings as a whole are reinforced by the comparison between the subgroup on β blockers versus those not on β blockers.

In psychiatry and psychology, it is typical to examine the association between emotional intensity and the intensity of physical symptoms. It is well established that greater self-reported negative affect, whether it be trait neuroticism (4,50) or state depression or anxiety (51), is associated with greater intensity of somatic symptoms. Table 1 shows that same finding replicated in this study, especially in women. Although important and clinically useful, this approach fails to explain why some (depressed or anxious) individuals with high levels of negative affect have many somatic symptoms and seek medical evaluation for them, whereas others have few somatic symptoms or none. In this study we demonstrated that even when controlling for neuroticism the positive correlation between LEAS score and somatic symptom differentiation was still significant. This observation provides clear support for the claim that intensity of emotions and symptoms alone is insufficient, and that differentiation and complexity in awareness of one's own emotions advances understanding of how emotional experience and somatic symptoms covary.

The somatic symptoms investigated in this study were largely unrelated to the medical condition of the patients. The applicability of these findings to the phenomenon of somatization is supported by two studies (38,52) from a German psychosomatic inpatient unit. Patients with somatoform disorders had lower LEAS scores at onset of treatment than other inpatients with symptomatic psychiatric disorders, such as depression (38) and lower LEAS scores compared with healthy age-, sex-, and education-matched volunteers (52). The former follow-up study revealed that, in patients with somatoform disorders, LEAS scores showed significant in-

creases over 3-month multimodal treatment. Importantly, this increase remained significant when controlling for changes in self-reported negative affect. By contrast, although TAS-20 scores and self-reported negative affect both declined over the course of treatment, TAS-20 scores did not change when controlled for change in self-reported negative affect (38). Similarly, in studies of psoriasis (53), eating disorders (54), and essential hypertension (55), the LEAS successfully disentangled processing of distress from reported distress, whereas the TAS-20 did not.

Clinical observations (8) indicate that prior to treatment the somatic symptoms of patients with somatoform disorders are amplified by emotional distress in other areas of their lives that cannot be acknowledged or expressed. Improvements in emotional awareness during inpatient treatment can be attributed to the fact that the coordinated multimodal treatment program is designed to facilitate the transition from implicit to explicit emotion processing by targeting somatic (e.g., massage, dance therapy) and psychological (intensive individual and group psychotherapy) levels of function as well as their integration (52). A practical implication of such changes is that, when distress occurs in problematic life contexts, it is less likely to be expressed in the form of somatic symptoms (52,56). Just as greater emotional awareness, as measured on the LEAS by greater differentiation in the use of words describing emotion, is associated with more accurate emotion information processing (26–30), the same could be true of somatoform patients and the "accuracy" of their somatic symptom reports as they become more emotionally aware—as their somatic symptom reports become more differentiated, these symptoms may become more specific and more congruent with objective findings. A question for future research is whether somatic symptoms in somatoform patients are in fact reported in a more specific and differentiated manner as their emotional awareness improves, and whether physical symptom complaints match objective findings to a greater degree than they did before treatment.

This study had several limitations. We studied individuals with LQTS, which is a life-threatening physical condition. Although characterized by a physiological abnormality that is typically asymptomatic, having LQTS could conceivably heighten bodily concerns and affect somatic symptom ratings. Thus, it would be important to replicate this study in samples of nonstressed individuals, patients with common medical conditions and patients with somatoform disorders. Second, we did not obtain a measure of typical somatic symptoms to determine their frequency or severity independent of the 3 days of testing, and we cannot comment on the degree to which the reported somatic symptoms had an organic basis. Third, we did not attempt to examine the relationship between the degree of differentiation in the momentary emotional response and the degree of differentiation in the momentary somatic symptom rating. Fourth, we did not examine within-symptom variation in intensity over time as another index of symptom differentiation. Fifth, we did not conduct a structured interview for the presence of mental disorders, such as

depression. Thus, replication in a sample without either psychiatric or medical conditions would be desirable.

In conclusion, we demonstrated in a large, intensively studied group of patients at risk for sudden cardiac death that greater trait emotional awareness is associated with greater differentiation in the momentary rating of somatic symptoms. This finding was predicted and extends current understanding of the relationship between emotion and somatic sensations by highlighting the importance of differentiation in both domains. This perspective may also help to explain the puzzling clinical phenomenon of somatization.

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