

# Defensive Mobilization in Specific Phobia: Fear Specificity, Negative Affectivity, and Diagnostic Prominence

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**Background:** Understanding of exaggerated responsivity in specific phobia—its physiology and neural mediators—has advanced considerably. However, despite strong phenotypic evidence that prominence of specific phobia relative to co-occurring conditions (i.e., principal versus nonprincipal disorder) is associated with dramatic differences in subjective distress, there is yet no consideration of such comorbidity issues on objective defensive reactivity.

**Methods:** A community sample of specific phobia ( $n = 74$  principal;  $n = 86$  nonprincipal) and control ( $n = 76$ ) participants imagined threatening and neutral events while acoustic startle probes were presented and eyeblinks (orbicularis oculi) recorded. Changes in heart rate, skin conductance level, and facial expressivity were also measured.

**Results:** Principal specific phobia patients far exceeded control participants in startle reflex and autonomic reactivity during idiographic fear imagery. Distinguishing between single and multiple phobias within principal phobia and comparing these with nonprincipal phobia revealed a continuum of decreasing defensive mobilization: single patients were strongly reactive, multiple patients were intermediate, and nonprincipal patients were attenuated—the inverse of measures of pervasive anxiety and dysphoria (i.e., negative affectivity). Further, as more disorders supplanted specific phobia from principal disorder, overall defensive mobilization was systematically more impaired.

**Conclusions:** The exaggerated responsivity characteristic of specific phobia is limited to those patients for whom circumscribed fear is the most impairing condition and coincident with little additional affective psychopathology. As specific phobia is superseded in severity by broad and chronic negative affectivity, defensive reactivity progressively diminishes. Focal fears may still be clinically significant but not reflected in objective defensive mobilization.

**Key Words:** Anxiety, chronicity, comorbidity, corrugator, depression, EMG, emotional reactivity, facial expressivity, fear, heart rate, imagery, mental imagery, narrative imagery, psychophysiology, SCL, skin conductance, specific phobia, startle

Specific phobia is considered the prototypical anxiety disorder of defensive hyperreactivity, a view supported by extensive evidence of pronounced mobilization to fear cues—in reflex psychophysiology (1–4), electrocortical response (5,6), neural circuitry activation (7–10)—from a variety of elicitation procedures (e.g., pictures [2], movies [11], imagery [3], and conditioning [7]). Surprisingly, however, this literature does not include examination of the broader clinical constellation within which specific phobias are often embedded. Much of the research is based on nonpatient samples, and the minority of physiological investigations addressing clinically significant specific phobia have assessed presence or absence of the disorder irrespective of its severity relative to co-occurring conditions. As such, there is a dearth of research utilizing objective measures of emotional responding that considers critical features such as diagnostic primacy/prominence and comorbidity. In phenotypic studies, careful consideration of ranked severity (i.e., principal versus nonprincipal problem) across anxiety disorders has revealed important variation in liability for co-occurring disorders. For example, in a sample of over 1,000 anxiety patients, Brown *et al.* (12) observed that as a whole, 70% of patients with a specific phobia diagnosis had a comorbid anxiety or mood disorder. The comorbidity rate dropped to 33% among

the subset of patients for whom specific phobia was the principal disorder. In other words, whether specific phobia is the principal (i.e., worst) disorder or not covaries dramatically with the level of functional interference and, perhaps, the burden that affective psychopathology imposes on defensive reflex physiology.

## Narrative Imagery

The current investigation of specific phobia examines whether the presence/absence as well as number of fears and gradations in diagnostic primacy (i.e., principal, secondary, tertiary) reflect differences in defensive reflex physiology during narrative imagery. Script-driven emotional imagery is a valuable tool in studies of anxiety disorders, permitting presentation of both standard and idiographic threat challenges, akin to methods of imaginal exposure therapy (13). Physiological arousal during aversive imagery parallels anticipatory reactions to threatening events (14), similarly mobilizing the autonomic nervous system (e.g., heart rate, skin conductance), communicating threat through facial musculature (e.g., corrugator frown muscle), and prompting somatic reflexive action (e.g., startle potentiation [15,16]). Animals confronting survival threat show similar reactions, mediated by the brain's defense circuit (centered on the amygdale [17,18]), and neuroimaging studies suggest a comparable circuit (19–21) underlies human fear.

In a series of imagery investigations, Lang *et al.* (22–32) assessed differences in defensive arousal within several anxiety disorders. In general, each principal diagnostic group (e.g., social phobia, post-traumatic stress disorder [PTSD], panic disorder) showed greater defensive reactivity than control participants. However, consideration of within-diagnosis features revealed dramatic differences in defensive mobilization. That is, reactivity was robust in patients with focal affective disruptions (e.g., social phobia limited to structured performance situations), whereas reactivity was increasingly reduced as the principal disorder features were more generalized (e.g., apprehension extending to routine social interaction) and coinci-

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Received Nov 9, 2011; revised Jan 17, 2012; accepted Jan 18, 2012.

dent with increased disorder severity and duration, poorer prognosis, and higher anxiety and depressive disorder comorbidity. Blunted reflex responding was also related to symptom elevations across numerous domains including anhedonia, unspecified/trait anxiety, anger, and functional interference. The confluence of dimensional and categorical dysphoria was termed negative affectivity (26–28) to highlight the synergy of multiple pathologies as opposed to isolated disorders in modulating defensive reflex physiology. Taken together, these findings suggest that defensive engagement during imagery might be compromised by prolonged and diffuse anxious hyperarousal and accompanying negative affectivity (29–32).

### The Research Problem

In the current study, a similar distress-related reflex pattern was expected within specific phobias—varying as a function of phobia precedence and comorbid symptomatology. First, principal specific phobia patients were compared with control participants with the expectation that similar to preceding studies (2–10), principal phobia would be characterized by exaggerated defensive mobilization (i.e., potentiated startle and autonomic action) during imagery of idiographic fear narratives, whereas patients and control participants would react similarly during threatening imagery for which defensive mobilization is normal and adaptive (e.g., facing an attacking animal).

Next, principal phobia patients were distinguished according to whether they endorsed a single or multiple specific phobias. Further, another set of patients who had at least one specific phobia exclusive of their principal problem (i.e., nonprincipal/additional phobia) were identified. Concerning number of fears within principal phobia, competing hypotheses were tested: as shown in a nonpatient investigation of individuals endorsing solitary or numerous fears (24), multiple phobia patients might be putatively more fearful than individuals with an isolated phobia and hence show the most robust physiological reactivity during aversive imagery. Alternatively, in a clinical sample, greater negative affectivity could be expected with multiple fears and correspondingly, reduced reactivity. These debilitating symptom features might be yet more extreme in the nonprincipal phobia group whose foremost difficulties could include far more generalized anxiety and dysphoria (e.g., generalized anxiety disorder [GAD], panic disorder with agoraphobia [PDA]), thus prompting the most pronounced attenuation of defensive action.

### Methods and Materials

#### Participants

Participants were assessed at the University of Florida Fear and Anxiety Disorders Clinic: 160 treatment-seeking adults with a diagnosis of specific phobia ( $n = 74$  with principal specific phobia;  $n = 86$  with non-principal/additional specific phobia<sup>1</sup>) and 76 healthy community control participants. Fear focus was distributed as follows: animal 19.4%, blood-injury-injection 15.6%, situational 40%, natural environment 18.1%, and other 6.9%.

#### Diagnostic Classification

Diagnostic groups were established using the Anxiety Disorder Interview Schedule for DSM-IV (33), a structured interview for assessing

<sup>1</sup>Among the nonprincipal specific phobia group, principal disorders were limited to anxiety, mood, and adjustment disorders: GAD 24.4%; PDA 19.8%; PTSD secondary to repeated trauma 14.0%; generalized social phobia 12.8%; obsessive-compulsive disorder 9.3%; panic disorder without agoraphobia 8.1%; PTSD secondary to single trauma 4.7%; anxiety disorder not otherwise specified 4.0%; adjustment disorder with mixed anxiety and depression 1.2%; recurrent major depressive disorder 1.2%; depressive disorder not otherwise specified 1.2%; and circumscribed performance phobia 1.2%.

current anxiety, mood, substance use, and somatoform disorders and for screening psychosis and major physical disease. For multiple Axis I disorders, diagnostic primacy was determined by clinician-rated severity (ranging from 0 = no features present to 5 = diagnosis present; severe) reflecting both distress and interference. Control participants denied current or lifetime diagnoses of psychiatric illness. Interrater reliability (via videotape) was calculated for 20% of patients, yielding agreement at 100% for principal and 82.35% for nonprincipal phobia diagnosis among three masters- or doctoral-level clinicians.

Patients whose foremost clinical complaint was specific phobia (i.e., principal phobia) were further classified according to whether the patient indicated a single phobia ( $n = 50$ ) or multiple phobias ( $n = 24$ ).<sup>2</sup>

#### Procedure

The University of Florida Institutional Review Board approved the study. Participants provided informed consent and completed questionnaires and interviews in the morning; psychophysiological assessment and clinical debriefing followed in the afternoon.

**Experimental Stimuli.** Twenty-four narrative imagery texts were used (34). Analyses focused on two idiographic, personal threat narratives representing each participant's primary clinical fear or for control participants their worst fear experiences. Standard scenes included two panic attack (crowded checkout line, driving alone), four survival threat (physical attack by animal/human), and two neutral (watching documentary, reading magazine) events. Filler scripts were low arousal or engaging pleasant scenes to impede an overall unpleasant arousal context. Scripts were ~20 words designed to quickly reveal affect and reflect active participation. A woman recorded the scenes using minimal prosody for presentation over earphones (Telephonics TDH-49; Telephonics Corporation, Huntington, New York).

**Imagery Assessment.** Seated in a quiet, dimly lit room, with electrodes placed, participants were instructed to listen to the auditory scripts with eyes closed, vividly imagining the events described as if actively involved. Throughout the recording session, soft tones cued participants to relax, breathe slowly, and silently repeat the word "one" to stabilize between-trial physiological activity (35). Imagery scripts were interspersed every 36 seconds in the tone series, with content pseudorandomized so that no more than two stimuli of the same hedonic valence (pleasant, neutral, unpleasant) or content category (e.g., panic attack) were presented consecutively. The script series was repeated in a counterbalanced order.

Trials consisted of a 1-second baseline, a 6-second auditory script, and 12 seconds of imagery. Startle probes (50-msec 95 dB[A] white noise, instantaneous rise time) were presented at 4 to 5.5 seconds or 10 to 11.5 seconds postscript onset, or both, and on 25% of intertrial intervals (ITIs), at 22 to 23.5 seconds postimagery offset.

Following imagery assessment (approximately 45 minutes) participants rated each scene for experienced pleasure and emotional arousal (36).

#### Experimental Control and Data Collection

A computer running VPM software (37) controlled stimulus presentation and data acquisition. Bioamplifiers recorded electro-

<sup>2</sup>The vast majority (78%) of principal specific phobia patients were diagnosed only with specific phobia(s). More specifically, 82% of the single, principal phobia patients endorsed no other disorders, while the remaining 18% were diagnosed with a range of disorders, typically anxiety, adjustment, and/or mood syndromes, of lesser intensity than the specific phobia. Among the principal phobia patients with multiple disorder-level phobias, 62.5% met criteria for two phobias and no other disorders and 8% endorsed three phobias and no other disorders. The remaining patients endorsed two (16.7%) or three (12.5%) specific phobias in addition to a range of less severe Axis I disorders.

**Table 1.** Mean Responses and Standard Deviations to Imagery Scenes by Control and Principal Specific Phobia Groups

Response Modality/Imagery Scene	Control	Principal Specific Phobia	Group Effect
<b>Emotional Valence (1–9)</b>			
Neutral	6.93 (1.53) <sup>a</sup>	6.91 (1.29) <sup>a</sup>	$F(1,150) = .15, ns$
Panic attack	4.00 (1.08) <sup>a</sup>	3.96 (1.30) <sup>a</sup>	$F(1,149) = .12, ns$
Survival threat	2.68 (.99)	2.95 (1.24) <sup>a</sup>	$F(1,150) = 3.20, ns$
Personal threat	2.65 (1.41)	2.18 (1.67)	$F(1,148) = 3.35, p < .05^b$
<b>Emotional Arousal (1–9)</b>			
Neutral	2.31 (1.54) <sup>a</sup>	2.73 (1.74) <sup>a</sup>	$F(1,147) = 2.40, ns$
Panic attack	5.37 (1.90) <sup>a</sup>	5.86 (1.61) <sup>a</sup>	$F(1,147) = 2.82, ns$
Survival threat	6.69 (1.53) <sup>a</sup>	6.64 (1.65) <sup>a</sup>	$F(1,147) = .05, ns$
Personal threat	7.69 (1.67)	8.17 (1.36)	$F(1,145) = 3.63, p < .05^b$
<b>Startle Reflex (t Score)</b>			
Neutral	49.76 (5.01) <sup>a</sup>	51.15 (7.02) <sup>a</sup>	$F(1,133) = 2.75, ns$
Panic attack	51.51 (7.20) <sup>a</sup>	55.25 (14.08) <sup>a</sup>	$F(1,133) = 3.80, ns$
Survival threat	54.57 (8.38)	57.34 (16.09) <sup>a</sup>	$F(1,133) = 1.62, ns$
Personal threat	53.58 (7.33)	61.55 (20.87)	$F(1,133) = 8.81, p < .01$
<b>Heart Rate Δ (bpm)</b>			
Neutral	-.25 (2.07) <sup>a</sup>	-1.17 (1.98) <sup>a</sup>	$F(1,145) = 7.67, p < .01$
Panic attack	.10 (2.25) <sup>a</sup>	-.07 (1.91) <sup>a</sup>	$F(1,145) = .26, ns$
Survival threat	.23 (1.16) <sup>a</sup>	.26 (1.29) <sup>a</sup>	$F(1,145) = .27, ns$
Personal threat	1.21 (2.26)	2.61 (3.01)	$F(1,144) = 10.21, p < .01$
<b>SCL Δ [log (μS + 1)]</b>			
Neutral	-.006 (.036) <sup>a</sup>	-.011 (.058) <sup>a</sup>	$F(1,143) = .35, ns$
Panic attack	.004 (.046) <sup>a</sup>	-.006 (.057) <sup>a</sup>	$F(1,142) = 1.26, ns$
Survival threat	.004 (.022) <sup>a</sup>	.009 (.051) <sup>a</sup>	$F(1,142) = .69, ns$
Personal threat	.045 (.086)	.139 (.189)	$F(1,142) = 15.05, p < .001$
<b>Corrugator EMG Δ (μV)</b>			
Neutral	-.07 (.79) <sup>a</sup>	-.32 (1.10) <sup>a</sup>	$F(1,146) = 2.43, ns$
Panic attack	.49 (1.40) <sup>a</sup>	.54 (2.53) <sup>a</sup>	$F(1,146) = .02, ns$
Survival threat	.96 (1.98)	.93 (1.62) <sup>a</sup>	$F(1,146) = .01, ns$
Personal threat	1.17 (2.60)	1.45 (3.29)	$F(1,146) = .33, ns$

Valence rated on Self-Assessment Manikin (36) 1 = completely unhappy, 9 = completely happy; arousal rated on Self-Assessment Manikin 1 = completely relaxed, 9 = completely aroused.

bpm, residual beats per minute after removal of baseline effects; EMG, electromyographic; SCL, skin conductance level.

<sup>a</sup>Within-group comparison to personal threat significant at  $p < .05$ .

<sup>b</sup>One-tailed planned between-group comparison.

myography (EMG) potentials at left orbicularis oculi and corrugator supercilii, skin conductance level (SCL), and electrocardiogram as reported previously (25).

### Data Reduction and Analysis

Univariate analyses of variance and Tukey Honestly Significant Difference tests for planned comparisons determined group differences in demographic and questionnaire data.

Using VPM software, EMG, SCL [ $\log(\text{SCL} + 1)$ ], and electrocardiogram R-R intervals (converted to beats per minute) were reduced into half-second bins. Responses were determined by subtracting amplitude during the 1 second before script presentation from averages during the 12-second imagery period.

Startle blinks from orbicularis oculi EMG represented the magnitude difference between onset and peak muscle potential (38), standardized within subject in relation to the mean and standard deviation of intertrial probe responses (25).

Using SPSS (SPSS, Inc., Chicago, Illinois), omnibus repeated measures analyses of variance were conducted separately for each physiological measure, with diagnostic status as a between-subjects factor and imagery content as a within-subjects factor. Analyses were initially performed with control versus principal specific phobia as a between-subjects factor. Startle and autonomic reactivity during imagery have been shown to strongly covary with rated

emotional arousal (29–31); thus, contents were entered according to the mean linear increase in arousal reported by the patients (i.e., neutral, panic attack, survival threat, idiographic/personal threat). Significant overall group effects were followed up with between-group tests by content to specify which imagery scenarios evoked different sensitivities in patients and control participants, facilitating comparisons to preceding studies that utilized different prompts (22–25). Within-group comparisons explicated interactions.

Finally, the data for 86 nonprincipal phobia patients were included and analyses were repeated for all patients, this time also accounting for presence of single or multiple phobias in the principal phobia group (i.e., single phobia, multiple phobia, nonprincipal phobia). Wilks' lambda addressed sphericity issues (39).

## Results

### Principal Specific Phobia and Control Groups

**Affective Judgments.** Across groups, rated displeasure reliably increased from neutral to panic attack, survival threat, and personal threat at the extreme,  $F(3,143) = 258.88, p < .001$  (Table 1). Control participants rated personal and survival threat scenes equally aversive, all comparisons *ns*, whereas patients rated personal threat more aversive than all other contents, all  $ps < .001$ ;

content  $\times$  diagnosis interaction  $F(3,143) = 3.82, p < .05$ ; diagnosis  $F(1,145) = .90, ns$ . Patients rated personal threat scenes more unpleasant than control participants,  $p < .05$ .<sup>3</sup>

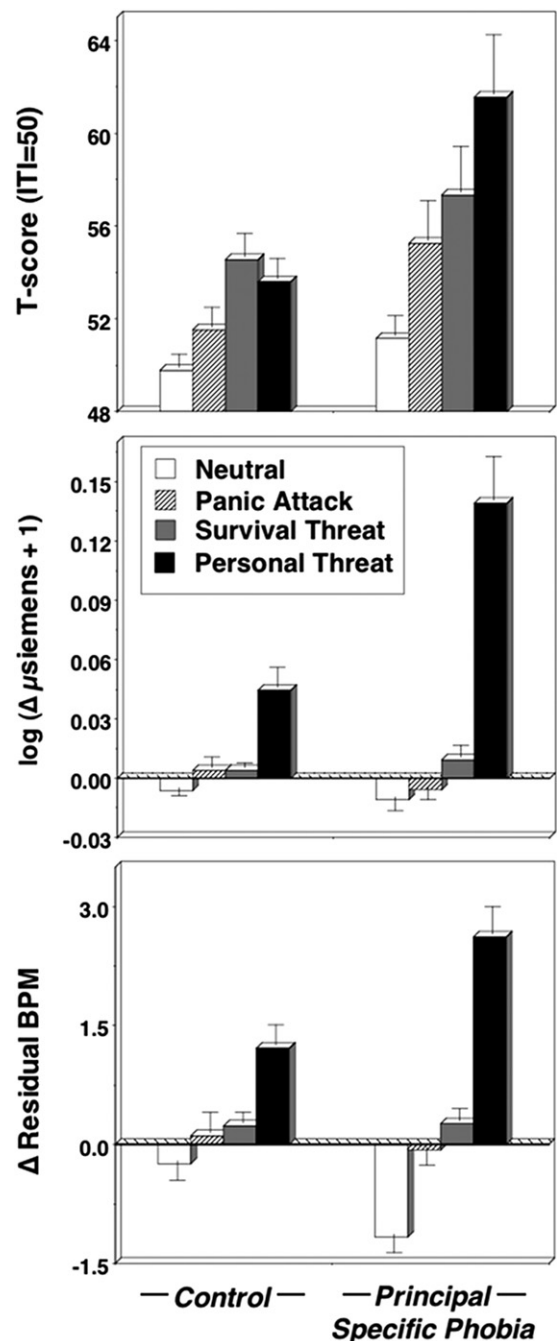
Both control participants and patients rated personal threat scenes most arousing followed by survival threat, panic attack, and neutral scenes, content  $F(3,143) = 259.67, p < .001$ ; content  $\times$  diagnosis  $F(3,143) = 2.64, p = .05$ ; diagnosis  $F(1,145) = 3.81, p = .05$ . Paralleling hedonic valence ratings, patients endorsed higher arousal than control participants during personal threat imagery,  $p < .05$ .<sup>3</sup>

**Startle Reflex Potentiation.** Blink magnitude during imagery relative to ITI acoustic startle probes (Figure 1, top panel) was consistently larger during unpleasant compared with neutral imagery, content  $F(3,131) = 10.17, p < .001$  (all unpleasant versus neutral comparisons,  $ps < .001$ ). Extreme responses differed by group, diagnosis  $F(1,133) = 6.26, p < .05$ ; content  $\times$  diagnosis  $F(3,131) = 3.35, p < .05$ ; content  $\times$  diagnosis (cubic contrast)  $F(1,133) = 4.58, p < .05$ . Whereas control participants responded with similar robustness to both survival and personal threat imagery,  $F(1,67) = 1.0, ns$ , principal phobia patients showed the greatest reflex responding during personal threat imagery, reliably exceeding responding elicited by survival threat imagery,  $F(1,66) = 8.05, p < .01$ , and surpassing control responses to personal threat imagery,  $F(1,133) = 8.81, p < .01$ .<sup>4</sup>

**Autonomic and Facial Responses.** Heart rate (Figure 1, bottom panel) increased above neutral during unpleasant imagery, content  $F(3,142) = 21.94, p < .001$ . Whereas both groups showed the most extreme accelerations to personal threat followed by survival threat and then panic attack imagery, patients also showed a significant decrease during neutral imagery, yielding more pronounced affective discrimination in heart rate, content  $\times$  diagnosis  $F(3,142) = 4.20, p < .01$ ; content  $\times$  diagnosis (linear contrast)  $F(1,144) = 95.35, p < .001$ ; diagnosis  $F(1,144) = .48, ns$ . Conspicuous increases to personal threat, diagnosis  $F(1,144) = 10.21, p < .01$ , as well as decreases to neutral, diagnosis  $F(1,145) = 7.67, p < .01$ , demonstrated by patients both differed reliably from control responses.

Enhanced sympathetic activation was evident in increased SCL during unpleasant relative to neutral imagery (Figure 1, middle panel), content  $F(3,140) = 18.53, p < .001$ . Modulation of SCL differed by group, diagnosis  $F(1,142) = 7.79, p < .01$ ; content  $\times$  diagnosis  $F(3,140) = 5.47, p < .01$ ; content  $\times$  diagnosis (quadratic contrast)  $F(1,142) = 7.79, p < .001$ ; patients showed progressive SCL increases from neutral to panic, survival, and finally to personal threat, while control participants showed similar magnitude increases to both panic and survival threat imagery. Similar to startle responses, patients' heightened SCL—specifically during personal threat imagery—exceeded that of control participants,  $F(1,142) = 15.05, p < .001$ .

Principal phobia patients and control participants showed the same pattern of facial frowning, covarying strongly with their shared pattern of rated displeasure, content  $F(3,144) = 14.05, p < .001$ ; content  $\times$  diagnosis  $F(3,144) = .32, ns$ ; diagnosis  $F(1,146) = .004, ns$ .



**Figure 1.** Mean startle reflex responses (standardized to the distribution of responses during intertrial intervals [ITI]; top panel), skin conductance level change (middle panel), and heart rate change (residuals; bottom panel) during neutral, panic attack, survival threat, and personal threat imagery for control and principal specific phobia groups. Error bars refer to standard error of the mean. BPM, beats per minute.

<sup>3</sup>One-tailed test based on prediction that principal phobia patients would exceed control participants during personal threat imagery.

<sup>4</sup>No group differences emerged for blink magnitude to intertrial startle probes or for baseline SCL or corrugator activity,  $F_s = .12$ – $2.53$ . Consistent with preceding studies (29–31), heart rate was higher for patients (mean = 73.38, SD = 9.90) than control participants (mean = 65.12, SD = 10.02), diagnosis  $F(1,144) = 25.08, p < .001$ , and as such analyses for heart rate change were calculated on residuals secondary to removing the trial-specific baseline (1-second average before script onset) effects via linear regression.

### Fear Generalization, Phobia Primacy, and Defensive Reactivity

**Affective Judgments.** All three phobia groups endorsed a similar pattern of rated displeasure, content  $F(3,147) = 204.37, p < .001$ ; content  $\times$  diagnosis  $F(6,294) = 1.37, ns$ ; diagnosis  $F(2,149) = 5.27, p < .001$ , with the exception that panic attack imagery was characterized as more aversive by nonprincipal than single phobia patients (Table 2).



**Table 2.** Mean Responses and Standard Deviations to Imagery Scenes by Control and Principal and Nonprincipal Specific Phobia Groups

Response Modality/Imagery Scene	Principal Specific Phobia: Single Fear	Principal Specific Phobia: Multiple Fears	Nonprincipal/Additional Specific Phobia	Group Effect
<b>Emotional Valence (1–9)</b>				
Neutral	7.09 (1.27) <sup>a</sup>	6.52 (1.29)	6.84 (1.46) <sup>b</sup>	$F(2,154) = 1.43, ns$
Panic attack	3.96 (1.30)	3.96 (1.34)	3.27 (1.56)	$F(2,152) = 4.32, p < .05$
Survival threat	2.92 (1.22)	3.02 (1.30)	2.53 (1.10)	$F(2,155) = 2.66, ns$
Personal threat	2.03 (1.51)	2.50 (1.97)	1.83 (1.18)	$F(2,150) = 1.97, ns$
<b>Emotional Arousal (1–9)</b>				
Neutral	2.70 (1.80)	2.79 (1.65)	2.68 (1.63)	$F(2,154) = .04, ns$
Panic attack	5.82 (1.77) <sup>a</sup>	5.94 (1.24)	6.69 (1.55) <sup>b</sup>	$F(2,154) = 5.41, p < .05$
Survival threat	6.62 (1.73)	6.66 (1.51)	7.11 (1.51)	$F(2,155) = 1.79, ns$
Personal threat	8.17 (1.41)	8.15 (1.27)	7.87 (1.82)	$F(2,151) = .63, ns$
<b>Startle Reflex (<i>t</i> score)</b>				
Neutral	52.01 (7.68)	49.52 (5.32)	49.86 (6.34)	$F(2,143) = 2.82, ns$
Panic attack	56.20 (16.38)	53.45 (8.04)	51.81 (7.34)	$F(2,143) = 2.26, ns$
Survival threat	59.02 (18.44)	54.12 (9.78)	54.31 (11.95)	$F(2,143) = 1.79, ns$
Personal threat	63.83 (24.33) <sup>a</sup>	57.19 (10.94)	53.97 (10.97) <sup>b</sup>	$F(2,143) = 5.25, p < .01$
<b>Heart Rate Δ (bpm)</b>				
Neutral	−1.07 (2.00)	−1.39(1.96)	−.64 (1.84)	$F(2,156) = 1.55, ns$
Panic attack	−.11 (2.02)	.00 (1.71)	.37 (1.49)	$F(2,156) = 1.47, ns$
Survival threat	.12 (1.28)	.55 (1.28)	.05 (1.12)	$F(2,156) = 1.61, ns$
Personal threat	2.84 (3.26) <sup>a</sup>	2.17 (2.42)	1.29 (2.56) <sup>b</sup>	$F(2,155) = 4.21, p < .05$
<b>SCL Δ [log (μS + 1)]</b>				
Neutral	−.015 (.059)	−0.001 (.055)	.005 (.049)	$F(2,153) = 2.41, ns$
Panic attack	−.001 (.058)	−0.015 (.055)	.005 (.046)	$F(2,152) = 1.46, ns$
Survival threat	.013 (.046)	.002 (.061)	.002 (.050)	$F(2,152) = .52, ns$
Personal threat	.158 (.186) <sup>a</sup>	.099 (.192)	.052 (.093) <sup>b</sup>	$F(2,152) = 7.98, p < .01$
<b>Corrugator EMG Δ (μV)</b>				
Neutral	−.27 (1.08)	−.43 (1.16)	.14 (1.26)	$F(2,155) = 3.01, ns$
Panic attack	.43 (.88)	.76 (4.28)	.40 (1.31)	$F(2,155) = .31, ns$
Survival threat	.99 (1.80)	.81 (1.18)	.86 (1.86)	$F(2,155) = .09, ns$
Personal threat	1.44 (2.55)	1.47 (4.50)	.86 (1.73)	$F(2,155) = 1.03, ns$

Valence rated on Self-Assessment Manikin (36) 1 = completely unhappy, 9 = completely happy; arousal rated on Self-Assessment Manikin 1 = completely relaxed, 9 = completely aroused.

bpm, residual beats per minute after removal of baseline effects; EMG, electromyographic; SCL, skin conductance level.

<sup>a</sup>Post hoc between-group comparison to nonprincipal phobia group significant at  $p < .05$ .

<sup>b</sup>Post hoc between-group comparison to single phobia group is significant at  $p < .05$ .

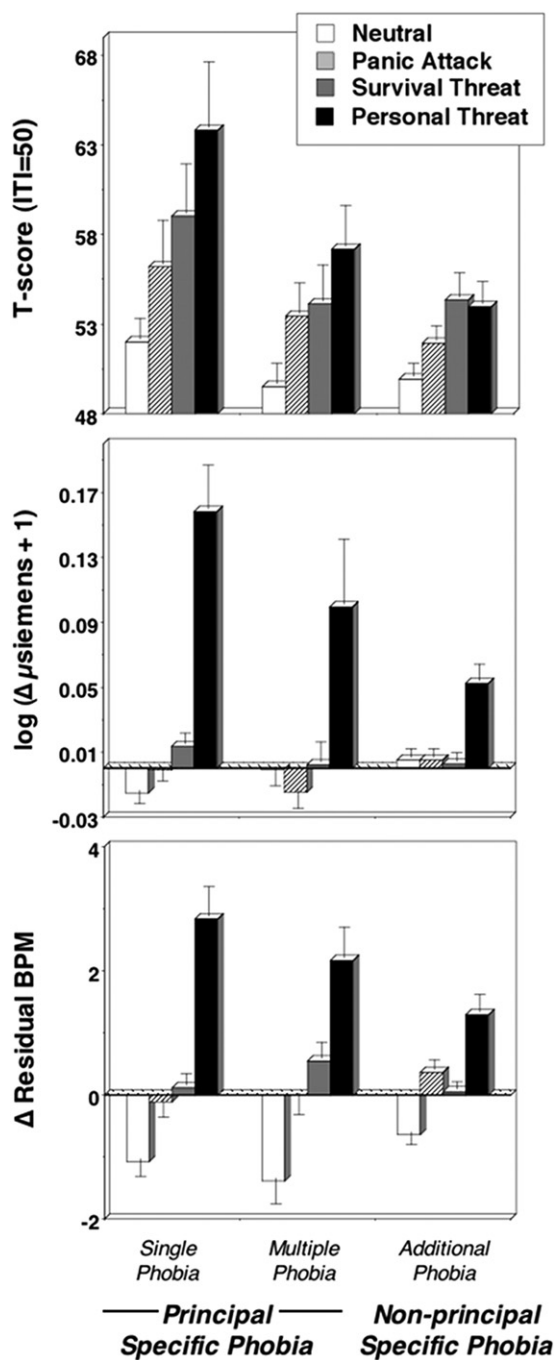
The same sensitivity to panic attack imagery emerged in more extreme ratings of emotional arousal by the nonprincipal relative to the single phobia group, content  $F(3,148) = 163.67, p < .001$ ; content  $\times$  diagnosis  $F(6,296) = 2.79, p < .05$ ; content  $\times$  diagnosis (quadratic trend),  $F(2,150) = 5.18, p < .01$ ; diagnosis  $F(2,143) = 1.42, ns$ . Importantly, across patient groups, personal threat scenes were rated as equally extreme in both unpleasantness and arousal.

**Startle Reflex Potentiation.** Whereas affective modulation was similar between groups, content  $F(3,141) = 9.32, p < .001$ ; content  $\times$  diagnosis  $F(6,282) = 1.59, ns$ , marked differences were evident in magnitude of responding during imagery relative to ITI startle probes, diagnosis  $F(2,143) = 4.30, p < .05$ , with the single phobia group exceeding the nonprincipal phobia group,  $p < .05$ —the multiple phobia group intermediate (Figure 2, top panel). Follow-up pairwise tests (Table 2) clarified that the overall group difference was foremost attributable to exaggerated responses in the single phobia group, specifically during personal threat imagery.<sup>5</sup>

<sup>5</sup>No differences emerged between patient groups in resting physiology and intertrial interval startle probe responses (Table S1 in Supplement 1).

**Autonomic and Facial Responses.** Single and multiple phobia patients showed heart rate deceleration to neutral and incremental acceleration starting with panic, survival, and finally the most pronounced acceleration during personal threat imagery. Consistent with their subjective aversion and arousal ratings, nonprincipal phobia patients showed the second largest heart rate increase during panic attack imagery, content  $F(3,153) = 24.44, p < .001$ ; content  $\times$  diagnosis  $F(6,306) = 2.31, p < .05$ ; diagnosis  $F(2,155) = .49, ns$ . Concordant with the startle findings, the only between-group difference in response magnitude was observed for personal threat imagery due to the single group showing larger accelerations than the nonprincipal group—the multiple phobia group, again, intermediate (Table 2; Figure 2, bottom panel).

Reliable sympathetic reactivity was indexed in SCL change during imagery, content  $F(3,150) = 20.19, p < .001$ . Single phobia patients showed a strong linear skin conductance increase from neutral to personal threat, whereas the multiple,  $F(1,22) = 8.19, p < .01$ , and nonprincipal phobia,  $F(1,83) = 26.14, p < .001$ , groups demonstrated less differentiation among the lower arousing contents, diagnosis  $F(2,152) = 2.90, p = .06$ ; content  $\times$  diagnosis  $F(6,300) = 3.43, p < .01$ . Finally, the single phobia group demonstrated more pronounced reactivity than the nonprincipal group,



**Figure 2.** Mean startle reflex responses (standardized to the distribution of responses during intertrial intervals [ITI]; top panel), skin conductance level change (middle panel), and heart rate change (residuals; bottom panel) during neutral, panic attack, survival threat, and personal threat imagery for control and principal (single and multiple phobia) and nonprincipal specific phobia groups. Error bars refer to standard error of the mean. BPM, beats per minute.

specifically in response to personal threat imagery—the multiple phobia group, again, evinced intermediate reactivity (Table 2; Figure 2, middle panel).

In contrast to the concordant hyperreactivity in single phobia patients across startle, SCL, and heart rate indices—specifically during personal threat imagery—corrugator changes, content  $F(3,153) = 16.70, p < .001$ ; diagnosis,  $F(2,155) = .07, ns$ ; content  $\times$

diagnosis  $F(6,306) = 1.12, ns$ , suggested consonant, reliable facial expressivity across patient and control groups.

### Diagnostic Primacy and Comorbid Negative Affectivity

Questionnaire measures of nonspecific trait anxiety (State Trait Anxiety Inventory [STAI]), cognitive/somatic symptoms of depression (Beck Depression Inventory [BDI]) as well as anhedonia (Mood and Anxiety Symptom Questionnaire [MASQ] anhedonia subscale), interoceptive sensitivity (Anxiety Sensitivity Index [ASI]), trait anger (State Trait Anger Expression Inventory), broad fearfulness (Fear Survey Schedule), and interference across functional domains (Illness Intrusiveness Rating Scale [IIRS])<sup>6</sup> reliably increased from control participants at the minimum to single phobia, multiple phobia, and finally nonprincipal/additional phobia at the extreme (Table 3; Figure 3).<sup>7</sup> The latter patients also surpassed principal phobia patients in total number of Axis I disorders and frequency of comorbid anxiety, as well as mood disorders. To further specify the pattern of comorbidity across the phobia spectrum, subgroups were compared in terms of presence or absence of single episode versus recurrent major depression, which reflected marginal differences across subgroups for transient depression in comparison with stark differences in refractory depression, with much greater prevalence of the latter in nonprincipal phobia. By nature of the analyses undertaken here, all patients were positive for a significant fear disorder. To further characterize the anxiety/mood comorbidity, disorders were defined as predominantly broad distress/anxious-misery (i.e., GAD, PTSD, obsessive-compulsive disorder, PDA, recurrent major depression) or not, drawing on the findings of epidemiological (50–55) as well as psychophysiological (31,32) studies. Similar to the presence of more intractable depression, the frequency of broad distress disorders was far greater in nonprincipal (85%) than principal phobia (6% to 8%). In fact, for 84% of nonprincipal patients, at least one anxious-misery disorder superseded specific phobia in severity. Consistent with protracted functional interference, the nonprincipal group reported the lowest educational attainment (Table 3). This overall pattern of broad dysphoria and impairment (indexed both dimensionally and categorically) in the nonprincipal phobia group was consistent with clinician estimates of poorer treatment prognosis.

Interestingly, features more pertinent to the specific phobia diagnosis did not distinguish subgroups (Table 3). Irrespective of phobia type, when queried about their particular phobic object/context during interview, patients reported experiencing equivalently severe fear and avoidance and consequent distress and functional interference. Furthermore, patients recalled experiencing disorder-level dysfunction of similar duration, on average 16 years.<sup>8</sup>

Regarding other factors that might mitigate defensive engagement, self-reported ability to generate vivid mental imagery was

<sup>6</sup>Similar to other studies that endeavored to utilize the IIRS for assessing functional interference in lifestyle domains associated with mental as opposed to physical health conditions (48,49), the IIRS was worded: “How much do your mental health problems (e.g., anxiety, depression) and/or their treatment interfere with your...?”

<sup>7</sup>For ease of comparison with the extant literature, the BDI and STAI scores were presented in conjunction with the MASQ anhedonia subscale in Table 3. As evident in Figure 3, the other four MASQ subscales (mixed anxiety/depression symptoms, general anxiety, general depression, anxious arousal) showed the same pattern across groups as the BDI and STAI and, not surprisingly, all six measures were highly intercorrelated,  $r_s = .69-.89, ps < .001$ .

<sup>8</sup>As some patients had more than one diagnosed specific phobia, chronicity refers to the earliest onset phobia. Patient groups also did not differ in duration of worst phobia ( $M = 15.46$  years,  $SD = 13.04$ ).

**Table 3.** Questionnaire and Interview Responses (Means and Standard Deviations) for Control and Principal and Nonprincipal Specific Phobia Groups

Measure	Control	Principal Specific Phobia: Single Fear	Principal Specific Phobia: Multiple Fears	Nonprincipal/Additional Specific Phobia	Group Effect
Questionnaire Measures of Broad Distress					
STAI-Trait (20–80)	30.80 (8.57) <sup>abc</sup>	37.17 (10.42) <sup>cd</sup>	42.29 (11.13) <sup>d</sup>	56.55 (11.00) <sup>ad</sup>	$F(3,226) = 90.09, p < .001$
BDI total (0–63)	3.51 (4.55) <sup>abc</sup>	8.08 (7.46) <sup>cd</sup>	12.38 (8.66) <sup>cd</sup>	21.80 (11.71) <sup>abd</sup>	$F(3,230) = 63.21, p < .001$
MASQ anhedonia (22–110)	46.26 (13.32) <sup>bc</sup>	52.92 (15.12) <sup>c</sup>	59.86 (17.27) <sup>cd</sup>	77.09 (15.23) <sup>abd</sup>	$F(3,227) = 62.80, p < .001$
ASI total (0–64)	9.06 (6.83) <sup>abc</sup>	21.55 (10.91) <sup>bcd</sup>	29.57 (12.34) <sup>acd</sup>	38.41 (13.64) <sup>abd</sup>	$F(3,216) = 93.33, p < .001$
FSS total (103–515)	156.47 (37.42) <sup>abc</sup>	195.00 (50.88) <sup>cd</sup>	217.69 (54.76) <sup>cd</sup>	265.89 (75.92) <sup>abd</sup>	$F(3,229) = 48.41, p < .001$
STAXI-Trait (10–40)	14.42 (3.94) <sup>c</sup>	15.73 (5.46) <sup>c</sup>	17.22 (4.60)	19.65 (5.83) <sup>ad</sup>	$F(3,227) = 15.04, p < .001$
IIRS total (13–91)	18.24 (11.64) <sup>abc</sup>	26.13 (14.68) <sup>cd</sup>	29.68 (14.69) <sup>cd</sup>	51.66 (19.44) <sup>abd</sup>	$F(3,213) = 60.20, p < .001$
QMI total (35–245)	82.96 (29.70)	88.15 (23.80)	97.09 (39.15)	89.36 (32.70)	$F(3,224) = 1.37, ns$
Interview Measures of Specific Phobia					
Fear of worst phobia (0–8)		6.72 (2.20)	6.43 (2.15)	6.75 (1.87)	$F(2,156) = .23, ns$
Avoidance of worst phobia (0–8)		6.77 (1.91)	6.61 (2.69)	6.73 (2.07)	$F(2,156) = .05, ns$
Interference secondary to worst phobia (0–8)		5.01 (2.40)	5.68 (2.35)	5.21 (1.99)	$F(2,157) = .77, ns$
Distress secondary to worst phobia (0–8)		6.02 (2.03)	6.30 (2.05)	5.53 (2.05)	$F(2,157) = 1.73, ns$
Specific phobia chronicity (years)		15.16 (14.80)	13.88 (11.70)	17.48 (13.14)	$F(2,151) = .86, ns$
Prognosis (1–4)		1.44 (.50) <sup>c</sup>	1.75 (.61) <sup>c</sup>	2.50 (.75) <sup>ab</sup>	$F(2,157) = 43.54, p < .001$
Interview Measures of Comorbidity					
Axis I disorders (Count)		1.28 (.67) <sup>bc</sup>	2.58 (.83) <sup>ac</sup>	3.64 (1.30) <sup>ab</sup>	$F(2,157) = 76.35, p < .001$
Comorbid anxiety (excluding specific phobia) disorder (%)		10.0 <sup>bc</sup>	20.83 <sup>ac</sup>	98.83 <sup>ab</sup>	$\chi^2(2) = 120.84, p < .001$
Comorbid mood disorder (%)		10.0 <sup>c</sup>	12.50 <sup>c</sup>	66.28 <sup>ab</sup>	$\chi^2(2) = 50.78, p < .001$
Comorbid single major depressive disorder (%)		2.0 <sup>c</sup>	4.12	12.79 <sup>a</sup>	$\chi^2(2) = 5.52, ns$
Comorbid recurrent major depressive disorder (%)		2.0 <sup>c</sup>	8.33 <sup>c</sup>	36.0 <sup>ab</sup>	$\chi^2(2) = 24.72, p < .001$
Comorbid anxious misery disorder (%)		6.0 <sup>c</sup>	8.30 <sup>c</sup>	84.89 <sup>ab</sup>	$\chi^2(2) = 97.21, p < .001$
Demographics					
Age at assessment (years)	31.79 (11.61)	34.98 (13.97)	36.83 (11.90)	36.20 (12.75)	$F(3,232) = 1.99, ns$
Gender (% Female)	65.79	72.0	79.17	72.09	$\chi^2(3) = 1.84, ns$
Race (% Caucasian)	84.21	86.0	75.0	77.91	$\chi^2(3) = 2.43, ns$
College graduate (%)	60.53 <sup>c</sup>	56.0 <sup>c</sup>	54.17 <sup>c</sup>	32.56 <sup>abd</sup>	$\chi^2(3) = 14.58, p < .001$

Fear/avoidance of worst phobia = severity ratings from Anxiety Disorder Interview Schedule for DSM-IV (9-point scale ranging from 0, no fear/never avoid, to 8, very severe fear/always avoids) (33). Interference/distress secondary to worst phobia = severity ratings from Anxiety Disorder Interview Schedule for DSM-IV (9-point scale ranging from 0, none, to 8, very severe). Specific phobia chronicity = years from patient-reported onset of earliest phobia to assessment. Prognosis = clinician-rated estimate of treatment outcome (4-point scale ranging from 1, excellent, to 4, poor). Superscripts = results of Tukey Honestly Significant Difference pairwise comparisons.

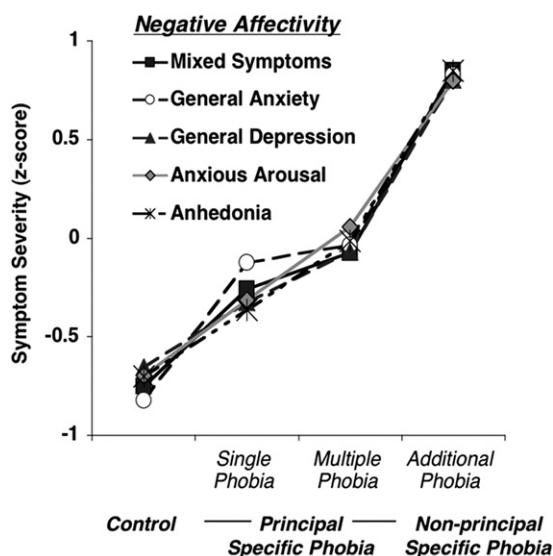
ASI, Anxiety Sensitivity Index (43); BDI, Beck Depression Inventory (41); FSS, Fear Survey Schedule (44); IIRS, Illness Intrusiveness Rating Scale (46); MASQ anhedonia, anhedonia subscale of the Mood and Anxiety Symptom Questionnaire (42); QMI total, Questionnaire on Mental Imagery (47); STAI-Trait, Trait scale of State Trait Anxiety Inventory (40); STAXI-Trait, Trait scale of State Trait Anger Expression Inventory (45).

<sup>a</sup>Post hoc between-group comparison to single phobia is significant at  $p < .05$ .

<sup>b</sup>Post hoc between-group comparison to multiple phobia is significant at  $p < .05$ .

<sup>c</sup>Post hoc between-group comparison to nonprincipal phobia is significant at  $p < .05$ .

<sup>d</sup>Post hoc between-group comparison to control is significant at  $p < .05$ .



**Figure 3.** Mean subscale scores on the Mood and Anxiety Symptom Questionnaire (42) for control and principal (single and multiple phobia) and nonprincipal specific phobia groups. Given the different ranges of the subscales, total scores were standardized across participants.

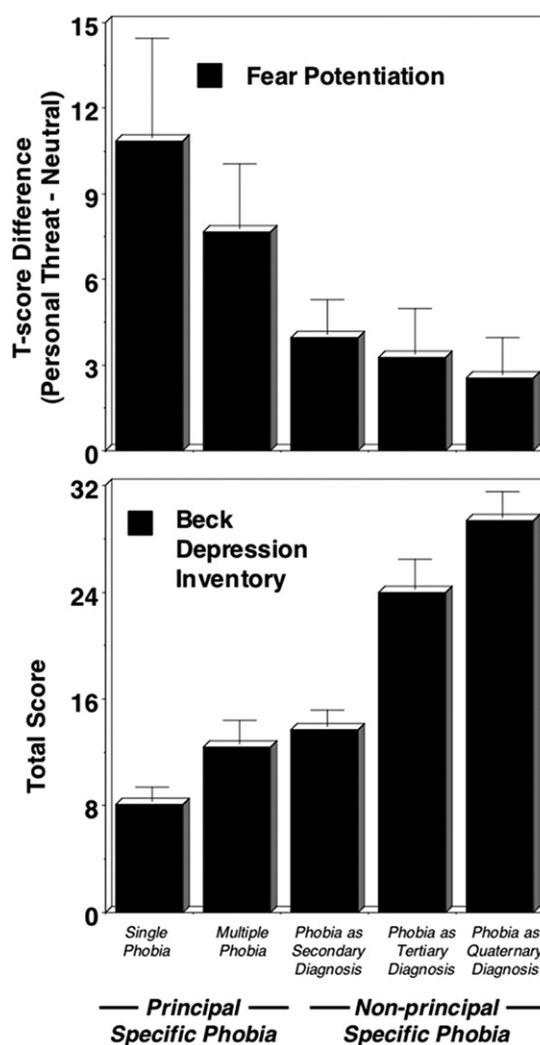
equivalent across participants (Table 3). Furthermore, medication and substance use patterns exerted no discernible influence on physiological patterns.<sup>9</sup>

#### Gradations in Specific Phobia Prominence

In light of the observed pattern of decreasing physiological reactivity during personal threat imagery across single and multiple principal phobia and finally nonprincipal phobia, post hoc analyses were conducted to assess whether a more refined spectrum of reactivity might be revealed if the latter group were further considered according to the relative primacy of specific phobia within the diagnostic profile. Toward this aim, nonprincipal phobia patients were distinguished as those whose specific phobia was the second-most ( $n = 32$ ), third-most ( $n = 29$ ), or fourth (or more)-most severe disorder ( $n = 25$ ) and repeated measures were performed on neutral and personal threat conditions. While subjective, autonomic,

<sup>9</sup>Psychotropic usage mirrored broad symptom severity with single (32%) and multiple phobia (29%) reporting appreciably lower rates than nonprincipal phobia (55%) patients [ $\chi^2(2) = 9.04, p < .05$ ]. Most commonly, these medications were selective serotonin reuptake inhibitors [single 14%; multiple 13%; non-principal 36%,  $\chi^2(2) = 10.05, p < .05$ ] and/or benzodiazepines [single 19%; multiple 14%; nonprincipal 32%,  $\chi^2(2) = 4.05, ns$ ]. The effects of these and less frequently endorsed compounds (e.g., serotonin norepinephrine reuptake inhibitors, 5.5%; beta blockers 3.7%; tricyclics 1.9%) were assessed by comparing resting and imagery reactivity among the medicated and nonmedicated patients. Considering either general psychotropic usage or more specific classes of drugs, no reliable effects emerged, consistent with prior psychophysiological studies of anxiety and depression (56–58). Reported usage of both prescription and over the counter medications for promoting physical health, as well as recreational substance use, were also collected but low frequencies of endorsement precluded statistical analysis. As previously demonstrated in samples characterized by highly comorbid anxiety and depression (59), nonprincipal patients (26%) were significantly more likely, pairwise comparisons,  $ps < .05$ , than control participants (6.6%) and single phobia patients (6.0%) to be current smokers, with multiple phobia patients intermediate (16.7%),  $\chi^2(3) = 16.26, p < .001$ . Importantly, no physiological effects were observed comparing resting and imagery reactivity of smokers and nonsmokers.

and facial expressivity measures were similarly representative of the group mean across the nonprincipal phobia subtypes, startle potentiation during personal threat imagery systematically decreased as the specific phobia diagnosis grew less prominent in the diagnostic profile (Figure 4, top panel), diagnostic subtype  $F(4,142) = 3.88, p < .01$ ; content  $\times$  diagnostic subtype  $F(4,142) = 3.88, p < .05$ . Whereas responses elicited during neutral imagery did not vary across groups,  $F = 1.90, ns$ , reliable differences were observed during personal threat imagery,  $F(4,142) = 3.92, p < .01$ : probe reflex responses in the single group exceeded both groups for whom specific phobia was superseded by at least two other disorders ( $ps < .05$ ). Coincident with progressive startle response diminution, measures of broad negative affectivity (ASI, MASQ, BDI) systematically and reliably increased ( $F_s = 13.52\text{--}39.51, ps < .001$ ; Figure 4, bottom panel). Furthermore, the precedence of an anxious-misery disorder over specific phobia showed a parallel increase among nonprincipal patients,  $\chi^2(2) = 17.34, p < .001$ : phobia as second-most (56%), third-most (93%), or fourth (or more)-most severe disorder (100%). Finally, duration of anxious-misery showed the same pattern,  $F(2,70) = 3.90, p < .05$ , with patients



**Figure 4.** Mean fear potentiation of startle reflexes (startle response magnitude during personal threat minus neutral imagery; top panel) and Beck Depression Inventory total score (41) for principal and nonprincipal specific phobia patients according to diagnostic prominence. Error bars refer to standard error of the mean.



whose phobia was most distant from the principal disorder (mean years = 19.8; SD = 13.4) recalling those symptoms longer than the other two groups (phobia as second-most [mean years = 10.7; SD = 11], third-most [mean years = 11.6; SD = 13.3]).

## Discussion

Consistent with the extant conceptual and empirical literature, principal specific phobia relative to control participants demonstrated amplified defensive mobilization to their worst fears, showing greater startle potentiation, skin conductance increases, and heart rate acceleration during personal threat imagery, concordant with more extreme ratings of aversion and emotional arousal. Responses to standard panic attack and survival threat imagery were similar in patients and control participants consistent with functional neuroimaging data (8,19,60) and suggesting that specific phobia does not broadly sensitize fear circuitry.

Considering the number and relative primacy of specific phobia revealed that single principal phobia patients demonstrated the most robust personal threat reactivity, greatly exceeding the non-principal phobia patients in overall responsivity. Multiple phobia patients were intermediate—not exceeding the single phobia group as in the McNeil *et al.* (24) analogue study but consistent with intermediate negative affectivity—showed somewhat less reactivity. For all three patient groups, their personal, worst threatening scenes were highest in facial expressivity (corrugator) and similarly rated most arousing and unpleasant. The observed spectrum of decreasing physiological reactivity (i.e., startle reflex, heart rate, SCL) from single to multiple and finally nonprincipal phobia is not surprising considering the inverse increase in dimensional and categorical negative affectivity, particularly comorbidity of refractory depression and anxious-misery disorders—a pattern previously observed within social phobia (29), PTSD (30), and PDA (31).

In contrast to the influence of broad distress, features essential to meeting threshold for specific phobia diagnosis (e.g., fear-related avoidance, impairment) did not distinguish subgroups, suggesting that relative hyperreactivity and hyporeactivity did not reflect differences in phobia intensity. Furthermore, principal (single and multiple) and nonprincipal phobia patients recalled phobia-related dysfunction of similarly long duration (average 16 years). This differs markedly from our results for other anxiety disorders: generalized social phobia (29), PTSD (30), and PDA (31) all showed hyporeactivity amidst more enduring and broader disorder-related distress. This difference is not wholly unforeseen: specific phobia is characterized by phasic defensive mobilization in the face of fear-relevant cues (61), which can typically be readily avoided, leading to rapid dissipation of distress. In contrast, generalized social phobia, PTSD, and PDA, as well as other disorders of anxious misery, are marked by persistent, uncontrollable symptom exacerbation often only moderately ameliorated by escape/avoidance. Such sustained distress may ultimately take a toll on the defensive systems subserving chronic hyperarousal, whereby protective homeostasis follows quickly—in the event of fear resolution. In concert with this, in the current research, the strongest, most punctate responding was observed in those with the most isolated or encapsulated fear-specific psychopathology (i.e., principal single phobia). Meanwhile, the least physiologically reactive patients, also most disparate from principal phobia, endorsed symptom levels on the BDI and STAI beyond the 99th percentile relative to nonclinical samples. Furthermore, among those with nonprincipal phobia, patients with the most impaired responding (right-most group in Figure 4) recalled the most chronic and pervasive anxious misery.

Animal research varying stressor intensity and duration (62–66)

has provided some support for the hypothesis that the stress of chronic anxiety and depression may relate to dampening of defensive reflexes. For example, rats exposed to brief and/or less threatening stress demonstrate hypervigilance and hyperarousal, whereas rats exposed to longer duration stress develop more generalized anxiety and depressive-like symptoms, including passivity and reduced movement and communication behaviors (63–66). Taken together, the cumulative, chronic stress inherent in pervasive anxiety and dysphoria and the associated functional impairments may have a debilitating effect on the integrity of the underlying fear/defense system—a consequence not produced by intermittent focal fear episodes.

Alternatively, however, these defensive and subjective response profiles may be stable, time-invariant dispositions throughout the trajectories of affective dysfunction, potentially reflecting genetic underpinnings (67,68). In a recent latent class analysis of two epidemiological samples, Vaidyanathan *et al.* (69) found that specific phobia diagnoses typically occur in one of two diagnostic profiles, one marked by multiple phobic disorders (and few other disorders) and the other by a wide array of Axis I disorders concurrent with specific phobia. The former group is more akin to the highly reactive principal phobia patients in the current study, while the latter group resembles the nonreactive, nonprincipal phobia patients. Vaidyanathan *et al.* (69) speculated that rather than reflecting gross severity of psychopathology (70), these classes distinguished individuals fundamentally disposed to different disorder combinations and, as our findings strongly suggest, different defensive propensities as well.

## Conclusion

Simply considering the presence or absence of specific phobia within the clinical presentation provides an incomplete picture of the associated defensive mobilization. The most robust defensive responding to fear-relevant imagery is evident in those patients with the most focal fearfulness and the least overall distress—a finding paradoxical in relation to the majority of conceptual and empirical work in anxiety disorders. Conversely, as specific phobia is superseded in severity by more pervasive and chronic anxious apprehension and dysphoria, defensive reactivity progressively diminishes. Essentially, the exaggerated responsivity considered characteristic of specific phobia is limited to those patients for whom their circumscribed fear is the most impairing condition and coincident with little additional affective psychopathology. Taken together, the primacy of focal fears and the extent of comorbid negative affectivity must be considered simultaneously to capture the associated phenotypic and endophenotypic constellation.

*This work was supported, in part, by National Institute of Mental Health Grants P50 MH 72850 to the Center for the Study of Emotion and Attention, University of Florida, Gainesville, Florida (P.J.L. Director and Principal Investigator) and R21 MH082702 to P.J.L. and National Research Service Award Research Fellowship (F31 MH069048) to L.M.M.*

*We thank Bruce N. Cuthbert for assistance with study design and interpretation of findings and the following individuals for their assistance in data collection: Cyd C. Strauss, Denise M. Sloan, Eleni Dimoulas, Jose M. Soler-Baillo, Reid Scott, Greg Perlman, Joshua R. Shumen, Esther Jean-Baptiste, and Hailey W. Bulls.*

*All authors report no biomedical financial interests or potential conflicts of interest.*

*Supplementary material cited in this article is available online.*

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