The psychophysiology of anxiety disorder: Fear memory imagery

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Abstract
Psychophysiological response to fear memory imagery was assessed in specific phobia, social anxiety disorder, panic disorder with agoraphobia, post-traumatic stress disorder (PTSD), and healthy controls. Heart rate, skin conductance, and corrugator muscle were recorded as participants responded to tone cues signaling previously memorized descriptor sentences. Image contents included personal fears, social fears, fears of physical danger, and neutral (low arousal) scenes. Reactions to acoustic startle probes (eyeblink) were assessed during recall imagery and nonsignal periods. Participants were significantly more reactive (in physiology and report of affect) to fear than neutral cues. Panic and PTSD patients were, however, less physiologically responsive than specific phobics and the socially anxious. Panic and PTSD patients also reported the most anxiety and mood symptoms, and were most frequently comorbidly depressed. Overall, physiological reactivity to sentence memory cues was greatest in patients with focal fear of specific objects or events, and reduced in patients characterized by generalized, high negative affect.

Descriptors: Anxiety, Imagery, Startle, Depression, Panic, PTSD

Introduction

It has been suggested (Lang, 1979, 1985, 1994) that vivid recollection of an emotional event depends on central processing of an associative information network. Emotional networks include three classes of information (or types of representations): information about the physical stimulus context in which the event occurred, interpretive associations that elaborate the event’s meaning, and most importantly for the current investigation, procedural representations of the efferent reflexes—visceral and somatic—that define expressed emotion. Given the presence of procedural representations (and assuming high strength of association among network elements), retrieval of an emotional memory is expected to prompt actual activation of the represented muscles and glands, albeit generally below the threshold of overt action.

Network processing (memory retrieval) is instigated by external stimuli that match some network representations. Furthermore, because network representations are associatively connected, only a few matches may be needed to activate an entire memory structure. In this way, pictures, movies, poetry, and narrative text cue emotional memories, and the reflex physiology of emotion can be primed by no more than words on a page or the flickering light of film and television screens.

This memory model has been extensively studied in normal participants instructed to process verbal descriptions of a fear context (e.g., Lang, Levin, Miller, & Kozak, 1983; Miller et al., 1987). In this research, perceptual-motor memories were consistently activated by text cues, with an accompanying efferent reaction that varied in strength with reports of emotional arousal. The process was modulated by imagery instructions, training, and variations in text content. Optimal physiological responses resembled preparation to avoid/escape, as it occurs in an actual fearful encounter. Curiously, however, in pilot studies with anxiety patients, the efferent effects were greatly diminished in some disorders—even with recall of patients’ primary clinical fears. These seemingly counterintuitive data prompted Lang (1985) to speculate that anxiety disorders might differ in memory organization, according to principal diagnosis. He proposed a continuum in the coherence of fear memory networks, varying over anxiety diagnoses from high to low associative network strength. In this view, phobia is characterized by fear networks of high associative strength, with specific, reliable cues to active avoidance, assuring strong physiological activation at memory recall; the fear networks of socially anxious patients are somewhat less coherent, with many more stimulus and meaning representations, but with a lower overall associative strength; coherence is further reduced in panic, and lowest in generalized
anxiety disorder. These variations in network coherence determine a parallel variation in strength of physiological reaction at memory recall.

A hypothesized practical consequence of low network coherence is that emotional language is less likely to activate emotional expression in the context of psychological treatments. For example, imagery methods would be less effective in cognitive and exposure therapy. Findings consistent with this view have been reported, showing that participants who respond physiologically in emotional imagery have a better therapeutic outcome (Bryant, Sullivan, Strauss, Cuthbert, & Lang, 1997; Lang, 1970).

**Studying Emotional Memory in Anxious Patients**

The present research is a broader reconsideration of an experimental problem first addressed by Cook, Melamed, Cuthbert, McNeil, and Lang (1988). Consistent with the network hypothesis, they found that panic disorder patients with agoraphobia did not show robust autonomic response to emotional imagery of their fears, as was consistently found for specific phobics and, to a lesser extent, for social phobics. This difference was not apparent in verbal report of emotional arousal during imagery.

In research with normal participants, language descriptive of a fear context (with imagery instructions) has been shown to activate perceptual-motor memories that include metabolic mobilization, presumably preparatory to active avoidance as it would occur in an actual fearful encounter (e.g., Lang, 1979, 1994; Lang et al., 1983; Miller et al., 1987). The observed absence of cardiovascular arousal and skin conductance activation in panic patient—who, nevertheless, report intense fear—suggests that for this diagnosis, language cues are not strongly associated with a procedural physiology. This appears to involve both a reduced effectiveness of instructional and descriptive verbal input and an output discordance between the reactive physiology and evaluative language.

In examining their data, Cook et al. (1988) noted other differences between phobia and panic. On questionnaire measures of anxiety and depression, panic patients consistently had significantly higher scores than phobics. That is, diagnoses characterized by high “negative affect” (as defined by Tellegen, 1985; see also Clark, Watson, & Mineka, 1994), nevertheless showed the smallest physiological response. Curiously, this apparent association between high negative affect and low autonomic reactivity was not seen if diagnosis was ignored. That is, when the entire sample of anxiety patients was considered as a single group, correlations between physiological reactivity and questionnaire scores were low and nonsignificant. Furthermore, there was no significant covariation of these variables within the panic sample.

McNeil, Vrana, Melamed, Cuthbert, and Lang (1993) followed up this experiment using a similar methodology, studying fearful normal and anxious participants. The research results suggested that this population could be divided into two different groups with characteristics similar to those distinguishing specific phobics from panic patients. One group was characterized predominantly by fear, that is, an active avoidance of a specific fear object or group of objects. These subjects showed clear physiological arousal, concordant with verbal report of affective intensity, during fear image processing. The second group, like the panic patients, reported strong fear but also had higher scores on scales evaluating more generalized symptoms of anxiety and social distress (e.g., passive avoidance, restlessness, negative self-talk). The physiological response to imagery text of this “anxious” group was small, discordant with verbal report, and significantly less than that of the “fear” group.

**The Experimental Protocol**

The present psychophysiological protocol was originally developed in studies of normal participants (e.g., Vrana, Cuthbert, & Lang, 1986; Vrana & Lang, 1990), and differs in important ways from the one employed in previous studies of anxiety patients. Instead of using a paragraph of text to prompt fear imagery (e.g., Cook et al., 1988; McNeil et al., 1993; see also Orr et al., 1998), the image cues were simplified by reducing them to single, descriptive sentences. To further assure that the textual cues were understood, and to eliminate the possibility that the physical properties of auditory presentation (which can vary with speaker and sentence content) might compromise imagery measurement, participants memorized the sentence material in advance. Subsequently, during psychophysiological assessment, imagery was cued by brief auditory tones high or low in frequency, distinguishing two previously learned sentences of either fearful or neutral content.

The array of physiological measures studied here was expanded to include somatic as well as the autonomic responses (heart rate and skin conductance) previously reported. Thus, potentials from the corrugator (“frown”) muscle were measured as a covert index of facial expression. Furthermore, the eyeblink component of the startle reflex was recorded, as evoked by brief acoustic stimuli presented both during imagery processing and in the intertrial intervals (ITI). The potentiation of the startle reflex during fearful states, based upon extensive animal models documenting the involvement of the amygdala and other limbic structures (e.g., Davis, 1992), has been extensively documented in recent years both for perceptually presented stimuli (Lang, Bradley, & Cuthbert, 1997; Vrana, Spence, & Lang, 1988) and in imagery (Bradley, Lang, & Cuthbert, 1991; Cook, Hawk, Davis, & Stevenson, 1991; Vrana & Lang, 1990).

**Research Aims and Hypotheses**

The primary aim of the present experiment is to further explore emotional memory differences among anxiety disorders, first determining if the previously observed phenomena are reliable. That is, do phobic disorders differ from other anxiety diagnoses (e.g., panic disorder) in psychophysiological response to fear memory imagery? Are higher questionnaire scores in anxiety and depression found for diagnoses that also show reduced physiological reactivity? A further aim was to better understand the determinants of, and the relationship between, these phenomena. In advancing this aim, another anxiety disorder, post-traumatic stress disorder (PTSD), was included in the study sample and the several diagnostic groups were compared with a normal control group. Finally, the relationship between depression comorbidity and physiology was assessed for both base level activity and imagery response.

It was anticipated that autonomic reactivity to personal fear imagery would again be diminished in panic patients relative to specific phobics and socially anxious patients. This analysis was performed for all physiological measures of image processing. To evaluate the generality of effect, participants also imagined two standard fear contents, involving situations that are dangerous (an automobile accident, threat of physical violence) and situations that are socially difficult and distressing (giving a
speech, suffering verbal abuse). It was anticipated that socially anxious patients would show greater reactivity to the standard social scripts than the other diagnoses. All diagnoses and controls may be expected to react strongly to the danger sentences.

The reactions of PTSD patients in the present context were not easily predicted. Researchers have observed significant autonomic reactivity to fear imagery in these patients, relative to controls (e.g., Pitman, Orr, Gorgue, de Jong, & Claiborn, 1987) and more marked startle probe potentiation in the context of a shock threat (e.g., Morgan & Grillon, 1998). However, there are few data comparing the fear reactions of PTSD patients with other anxiety diagnoses. Thus, for example, previous data indicate that PTSD patients show greater heart rate increases when imaging combat scenes than veteran controls without PTSD (Pitman et al., 1987). However, it is not clear that the responses of PTSD patients are greater than, for example, specific phobics responding to their own fear content. Similarly, it has not been shown that PTSD patients are more reactive than normal controls when both groups respond to image cues of their own worst fears.

Considering the hypothesis that high cue specificity is the key feature determining a strong psychophysiological response, PTSD patients might be expected to show a marked reaction similar to that of specific phobics. On the other hand, PTSD is characterized by high comorbidity with other anxiety disorders and depression. From this perspective, previous results suggest that they would show a diminished reaction similar to panic disorder.

Method

Participants

Participants were seen at the University of Florida Fear and Anxiety Disorders Clinic (N = 130). The sample included 106 patients, grouped by principal diagnoses: 28 specific phobia (5 men), 30 social anxiety disorder (17 men), 26 panic disorder with agoraphobia (10 men), and 22 PTSD (8 men). These patients were predominantly Caucasian, and between 30 and 40 years of age. They presented for treatment following referral by a physician or other health care worker or in response to clinic advertisements. In addition to the patients, 24 controls (9 men) without behavior disorders were recruited through a newspaper advertisement and paid for their participation. Potential participants who showed active psychotic symptoms or had health problems that would compromise the physiological recording were excluded. However, 3 patients were included in the sample that had a past history of bipolar disorder (1 in the social and 2 in the panic groups).

Interviews were conducted using the Anxiety Disorders Interview Schedule–Revised (ADIS-R; DiNardo & Barlow, 1988) in order to assign diagnoses according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; American Psychiatric Association, 1987). The ADIS has been used extensively by senior members of the research team (see Cook et al., 1988; Lang et al., 2001; McNeil et al., 1993). They monitored uniformity in its use and interpretation, and trained and supervised new assessors. Diagnostic categories for all participants were independently rated by two of three clinical psychologists on the evaluation team, who either conducted the evaluation interview or observed it (live or on videotape). Using the ADIS-R data, each anxiety and mood disorder was assigned a severity score ranging from 0 to 5; the numbers 1 or 2 represented levels of subsyndromal features, and codes of 3 to 5 denoted presence of a diagnosable disorder with one of three increasing levels of severity. Axis II disorders were assigned according to presenting symptomatology, although time constraints precluded structured Axis II interview measures. A “principal diagnosis” was assigned according to the patient’s chief presenting complaint—“the condition established after study to be chiefly responsible for occasioning the admission of the individual.” (American Psychiatric Association, 2000, p. 3). Additional diagnoses were assigned as appropriate in decreasing order of severity or impairment. Any assessment differences were resolved at a meeting of the full evaluation team; the interrater agreement on principal diagnoses was high (Kappa = .86). Control participants were free of any Axis I or II disorders.

Anxiety disorder comorbidity was similar in pattern to that reported by Brown, Campbell, Lehman, Grisham, and Mancell (2001), that is, over half of all patients had another anxiety diagnosis in addition to their principal diagnosis. Patients in the panic group had the highest anxiety comorbidity (76%); specific phobics were the least likely to have an additional anxiety diagnosis (32%). Social anxiety disorder and specific phobia were the additional anxiety diagnoses showing the greatest overlap among experimental groups: 26% of the patients had an additional diagnosis of social anxiety disorder (highest incidence for the panic group, n = 10); 17% had specific phobia as an additional diagnosis (highest incidence in the social phobic group, n = 6). Additional diagnoses of panic with agoraphobia or PTSD were rare—5% and 4%, respectively. Other than the four diagnoses that defined the patient groups, generalized anxiety disorder was the additional diagnosis most frequently assigned (27% of the patient sample, highest incidence in the panic group, n = 11). The incidence of comorbid mood disorder (major depressive episode or dysthymia) was lowest for specific phobia (11%) and highest for panic disorder (42%) and PTSD (55%). Axis II additional diagnoses were not significant in number.

Procedure

Upon arrival at the Fear and Anxiety Disorders Clinic and after providing informed consent, participants were interviewed (ADIS-R) and administered a questionnaire battery. The questionnaires included the Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961); the Fenz–Epstein manifest anxiety questionnaire, assessing autonomie, somatic, and psychosocial symptom reports (FEQ; Fenz & Epstein, 1965); the Marks–Mathews self-rating scale of phobic symptoms (Marks & Mathews, 1979); and the Fear Survey Schedule of reactions to a catalogue of phobic objects (FSS; Wolpe & Lang, 1964). A temperament questionnaire assessing negative emotionality (EASI; Buss & Plomin, 1975) was added to the battery after data collection had begun, and was administered to the last 97 participants.2

1At the inception of this research, DSM-IV was not yet in general use. After the revision’s publication, considering that the changes would not significantly affect patient groupings, it was elected not to alter criteria in midstream.

2The EASI was introduced after the research was already underway. Therefore there are only data for 74 patient participants who were administered the measure, at least half of each diagnostic group.
Stimulus materials consisted of 12 one- or two-sentence imagery scripts, 6 each describing fearful and neutral scenes. Two of the fear scripts were personalized for each participant, based on interview and on Scene Construction forms filled out during the initial session just after the ADIS-R interview. For the latter, both patients and controls were first asked to describe their “worst fear experience.” For patients, the content of the fear scripts was their central, clinical fear (e.g., having a panic attack or confronting the phobic object); for control participants, these sentences described what they reported to be their “worst fear experience.” For all participants, guided by the bio-informational view of affective imagery (Foa & Kozak, 1986; Lang, 1979, 1994), the fear report was used to construct sentences that specifically included stimulus events (who, what, where), meaning (reported feelings and interpretations), and response information (expressive physiology and actions) coded in fear memory. The second personalized script comprised another scene related to the central clinical fear for patients, and another severe fear experience for controls. Examples are provided in Table 1.

In addition to these personal fear sentences, four standard fear imagery sentences and six standard neutral sentences were identical for all participants (Table 1). Standard fear scenes included two common social fear situations, that is, giving a speech and being humiliated in front of others, and two danger-oriented situations, that is, hearing an intruder while home alone and seeing a friend struck by a moving car. As for personal fear scripts, standard scenes included details of stimulus events, meaning, and response information. The six low-arousal neutral sentences described clearly nonfearful, normal, mildly pleasant situations, for example, relaxing in a chair at home or waking up on a weekend morning. Each of the 12 sentences was typed on an index card.

### Table 1. Standard Fear and Neutral Scenes and Sample Personal Fear Scenes

<table>
<thead>
<tr>
<th>Standard fear scenes</th>
<th>Personal fear scenes (clinically relevant for patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. My heart pounds in the suddenly silent room; everyone is watching me, waiting impatiently to hear what I will say. (social)</td>
<td>1. I feel tense all over and I fight back the tears as I walk past all the birds in the park; my eyes are closed and I feel so frightened I want to scream.</td>
</tr>
<tr>
<td>2. I’m wrong. They say, “Stupid! You never get anything right!”: my face is hot, and I have to stand there and take it. (social)</td>
<td>2. I tense my arms when I wait to receive the shot from the nurse to treat the spider bite; my heart races and my palms become sweaty.</td>
</tr>
<tr>
<td>3. I flinch at the screech of brakes; my companion is struck by a speeding car: Her leg is crushed, bone protruding, and blood pumps onto the road. (danger)</td>
<td>3. I feel closed in and like I am losing control as I sit in my car at the traffic light with cars all around me.</td>
</tr>
<tr>
<td>4. Taking a shower, alone in the house, I hear the sound of someone forcing the door, and I panic. (danger)</td>
<td>4. My heart quickens and my veins constrict as the lab tech wraps the rubber band around my arm; my face is flushed and when I see the needle I begin to feel sick.</td>
</tr>
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</table>

**Social anxiety**

1. I’m jittery, with heart pounding and stomach knotted, as I scan the room full of people for the disapproving looks that I know will come when I do something wrong.
2. As I wait to give my report on the conference call, I progressively feel nervous and unable to breathe; I put them on hold twice but still am unable to catch my breath.
3. My voice quivers and I begin to hyperventilate as I give a speech in front of a large group of people who are watching me uneasily.
4. After rehearsal, I gather together with the other members of our church choir to talk; my face feels as if it is swelling, my heart races and I am unable to articulate ideas properly.

**Post-traumatic stress disorder**

1. My heart beats faster as I hear the robbers in the front room; one of them shoots me and I want to scream.
2. I panic, as the man starts toward me; my hands tremble and I feel dizzy; I want to get away.
3. My eyes water and my stomach is in a knot as I share a last cigarette with my best friend; I know he won’t survive his stomach wound, but I can’t leave him. My heart pounds as I lift the gun to shoot him in the head.
4. While reading a book, I freeze as I hear someone forcing the door, my heart pounds, my body shakes and I feel nauseous.

**Panic disorder with agoraphobia**

1. I feel jittery when my brother tells me a tornado is coming. My heart races as the tornado passes over my house.
2. I panic as my boyfriend punches my best friend in the nose. He hits him again and again. I want to scream.
3. At the baseball park, I am walking and suddenly I feel an intense rush of fear; my heart is pounding, my breath is labored, and my stomach is in a knot.
4. 1 feel tense all over and I fight back the tears as I walk past all the birds in the park; my eyes are closed and I feel so frightened I want to scream.

**Control participants**

1. I feel tense all over and I fight back the tears as I walk past all the birds in the park; my eyes are closed and I feel so frightened I want to scream.
2. I tense my arms when I wait to receive the shot from the nurse to treat the spider bite; my heart races and my palms become sweaty.
3. I feel closed in and like I am losing control as I sit in my car at the traffic light with cars all around me.
4. My heart quickens and my veins constrict as the lab tech wraps the rubber band around my arm; my face is flushed and when I see the needle I begin to feel sick.

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<table>
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<tr>
<th>Specific phobia</th>
<th>Personal fear scenes (clinically relevant for patients)</th>
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<td>1. I feel tense all over and I fight back the tears as I walk past all the birds in the park; my eyes are closed and I feel so frightened I want to scream.</td>
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Imagery assessment began 1–2 hr after the diagnostic session and lasted approximately 2 hr (several patients, due to scheduling conflicts, were seen at a later time). Participants were seated in a reclining chair in a dimly lit room and given a brief introduction to the procedure, followed by placement of the recording electrodes. Overall, the session consisted of six trial blocks. For each trial block, the participant’s task was to memorize two sentences (one fearful and one neutral in content), and when subsequently cued, separately recall each of the sentences and imagine its content “vividly, as a personal experience.” The selection of fear and neutral sentences for the six trial blocks was determined randomly for each subject, as was the order of trials within a block.

The tone-cued recall task. Before each trial block, two sentences were memorized to a criterion of one perfect repetition. Participants were then instructed to close their eyes and listen to a series of brief, soft nonsignal tones presented at regular intervals, one every 6 s (0.5 s at 1000 Hz, 70 dB, 50 ms rise time, using Coulbourn Instruments S24-05 Tone Generator, S84-04 Rise Time Gate, and S82-24 Gated Amplifier). Participants were told to relax during these tones, breathe out slowly, and say the word “one” to themselves as in a secular meditation exercise (Benson, 1975). This had the general effect of reducing and stabilizing background physiological activity.

From time to time in the series of nonsignal stimuli, tones occurred that were discriminably higher or lower in pitch (700 or 1500 Hz, respectively). Participants were instructed that these special cue tones signaled that they were to recall and imagine the event described by one of the memorized sentences. Assignment of high or low tones to fear or neutral sentences was balanced across participants. Signal tones were always presented in a sequence of two 6-s intervals: Participants were instructed to silently articulate the sentence in the 6-s processing period following the first cue tone and to actively imagine the scene described at the second tone. A series of nonsignal tones then ensued.

Recall of each sentence was cued four times across a trial block, with a perceptually random fear–neutral order of presentation. A rapid double-tone signaled the end of a trial block. Participants were instructed to then open their eyes and to provide self-assessments of their emotional experience during imagery. Participants judged their feelings of pleasure and arousal, separately for fear and neutral sentences. They also rated imagery vividness, “interest” in the sentence, and chose one of seven specific emotions best representing their affective state. All six fear–neutral sentence pairs were presented in this manner, each with its own trial block. After the final block of trials, electrodes were removed and participants were debriefed.

The startle probe. Acoustic startle probes were 50-ms, 95-dB white noise bursts with instantaneous rise time, presented over Telephonic TDH-49 earphones using a Coulbourn Instruments S81-02 White Noise Generator and S82-24 Gated Amplifier. Over the course of the imagery assessment session, 48 probes were presented: Across the four trials for each sentence, probes were presented once during the silent articulation period, once during the imagery period, once during both periods, and once for neither period. Twelve probes were presented during the intertrial interval between sentence processing periods, two in each block of trials. Participants were told that the extra noises would sometimes occur, but that they were irrelevant to their experimental tasks.

Experimental Control and Data Collection
A PC-compatible computer and VPM software (Cook, Atkinson, & Lang, 1987) were used for timing, stimulus presentation, and physiological and rating data acquisition. Analog physiological measures were digitized at 20 Hz throughout each trial. Coulbourn Instruments S75-01 bioamplifiers and S76-01 contour-following integrators were used to record electromyographic (EMG) potentials from over the left orbicularis oculi and the left corrugator regions (bandpass of 90–1000 Hz, time constants of 125 and 500 ms, respectively). For more accurate recording of the blink response, the orbicularis channel was digitized at 1000 Hz from 50 ms before until 250 ms after-startle probe; eyeblink responses were scored with an interactive computer program that scored each blink for latency and peak. Skin conductance was recorded from electrodes placed adjacent on the hypothenar eminence of the nondominant hand, using Sensorsmedics standard electrodes filled with K–Y Jelly; the signal was transduced by a Coulbourn S71-22 skin conductance amplifier calibrated for a range of 0–40 µS. The ECG was recorded using electrodes placed on each forearm, amplified through a Coulbourn S75-01 bioamplifier; heart rate was recorded with a Schmitt trigger that interrupted the computer to record R–R intervals to the nearest 1 ms. The data were edited with an off-line program, and then transformed to beats/minute averages for each half-second. Ratings were acquired using the Self-Assessment Manikin (Bradley & Lang, 1994; Hodes, Cook, & Lang, 1985; Lang, 1980), an interactive computer graphics display that records judgments along the affective dimensions of valence and arousal on 21-point scales. In addition, participants used a computerized line rating display to report their judgments of “imagery vividness.”

Data Reduction and Analysis
Statistical analyses for all measures were accomplished with the SYSTAT package; all repeated measures with more than two levels employed the Wilks’ Lambda multivariate calculation. For demographic data and questionnaires, diagnostic group differences (specific, social, PTSD, panic, and control) were assessed with univariate analyses of variance (ANOVA) and follow-up Tukey Honestly Significant Difference (HSD) tests. The VPM software was used for reduction and initial processing of all physiological data.

Direct measures: Physiology and ratings. Physiological responses evoked directly by sentence imagery—i.e., heart rate, skin conductance, and corrugator EMG—were scored for each trial as the average activity during the combined silent articulation and imagery periods, deviated from the 6-s period immediately preceding silent articulation. For all three measures, a single value was derived for each category (personal, danger, social, and neutral) by averaging scores for the two sentences within each fear category, and for all six neutral trials. Skin conductance and corrugator data were lost for 3 and 4 participants, respectively, due to equipment failures.

Given the specific hypotheses, separate analyses of the three fear categories comprised the primary analytic focus. However, initial omnibus tests were conducted to verify the expectation that across all contents, fear categories would prompt palpably larger physiological effects, as well as higher arousal and lower pleasantness/dominance ratings. Accordingly, physiological responses directly evoked by sentence imagery were all analyzed initially with mixed-model ANOVA, followed by planned com-
The startle probe. Eyeblink magnitude was measured as the maximum excursion in microvolts of the orbicularis oculi signal from the level immediately prior to the response. Trials with clear artifacts were rejected, and trials with no response were scored as zero-magnitude blinks; 14 subjects were excluded from startle analyses for failing to meet the criterion of measurable blinks on at least 40% of all trials. Differences in base reflex reactivity between groups were examined using the absolute startle response magnitudes obtained during the intertrial intervals. For the analyses of startle potentiation during imagery trials, standardized scores were computed (i.e., \( T \) scores with a mean of 50 and standard deviation of 10) using the mean and standard deviation of each subject's ITI startle responses as the reference distribution. This normalization procedure (Bonnet, Bradley, Lang, & Requin, 1995; Requin & Bonnet, 1993) is designed to reduce the influence of arbitrary, between-subjects variance in reflex size while preserving probe response differences that occurred in the context of sentence imagery.

As was found here, the basic startle circuit is characterized by marked habituation (Davis & File, 1984); however, despite the overall reduction in reflex amplitude, emotional startle modulation (presumably due to amygdala projections) appears to be preserved over trials (see Bradley, Lang, & Cuthbert, 1993). These findings were considered in analyzing imagery content effects, so fear sentences were always compared with the neutral sentence from the same trial. As for the other measures, analyses began with omnibus ANOVAs including diagnosis and content. Then, based on the a priori hypotheses, fear/neutral differences were separately tested for social, danger, and personal fear contents. Initial analyses were conducted to examine differences in response between the silent articulation and imagery periods. No systematic pattern of effects could be found (perhaps due to a relatively limited number of startle probes in each period), and so these two periods were combined in estimating participants’ reactions to the different contents.

### Results

**Questionnaire Measures**

As expected, the intercorrelations among questionnaires used to measure various aspects of anxiety and depressive pathologies were all positive and uniformly high (ranging from .79 to .46). Mean questionnaire scores for each scale, by diagnosis, are presented in Table 2. The overall difference among groups was significant, \( F(4,124) = 18.0, p < .0001 \), based on analysis of \( Z \) scores for the four measures for which there are complete data (FSS, BDI, FEQ, Marks–Mathews scale). Overall, specific phobics were not different from controls and both these groups were significantly lower in Tukey pairwise comparisons than the social anxiety, PTSD, and panic groups; panic patients had the most elevated scores, differing from all other groups except PTSD.

This pattern was remarkably consistent when questionnaires were considered individually. Control and specific phobics had the lowest scores on each measure and were not themselves significantly different. In contrast, the PTSD and panic patient groups had the highest mean scores on all questionnaires, and also did not differ from each other. Furthermore, both these high scoring diagnoses differed significantly from both specific phobics and controls on manifest anxiety and the Beck Depression Inventory. In addition, the panic group differed from the low scorers on both phobia measures, whereas the PTSD group differed significantly in negative emotionality. Social phobics were intermediate, and not discriminable from

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Control (( n = 24 ))</th>
<th>Specific (( n = 28 ))</th>
<th>Social (( n = 30 ))</th>
<th>PTSD (( n = 22 ))</th>
<th>Panic-A (( n = 26 ))</th>
<th>Overall ( F, p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.2 (9.7)</td>
<td>33.2 (13.2)</td>
<td>32.0 (11.1)</td>
<td>36.5 (15.2)</td>
<td>34.2 (11.2)</td>
<td>n.s.</td>
</tr>
<tr>
<td>F-E Manifest Anxiety Scale</td>
<td>94.7 a (21.9)</td>
<td>109.4 a (22.3)</td>
<td>131.5 b (32.4)</td>
<td>135.0 b (27.9)</td>
<td>142.0 b (38.7)</td>
<td>11.0, &lt;.0001</td>
</tr>
<tr>
<td>Marks–Mathews Phobia Quest.</td>
<td>15.5 a (12.3)</td>
<td>23.9 ab (13.3)</td>
<td>35.4 bc (14.7)</td>
<td>38.4 cd (23.0)</td>
<td>40.2 d (26.5)</td>
<td>12.9, &lt;.0001</td>
</tr>
<tr>
<td>Fear Survey Schedule</td>
<td>167.7 a (39.3)</td>
<td>202.8 ab (44.1)</td>
<td>225.8 bc (57.7)</td>
<td>229.9 bc (57.7)</td>
<td>262.9 c (75.5)</td>
<td>9.6, &lt;.0001</td>
</tr>
<tr>
<td>Negative Emotionality (Scale from the EASI)</td>
<td>22.2 a (5.5)</td>
<td>26.6 ab (7.2)</td>
<td>28.6 bc (5.7)</td>
<td>33.8 c (7.0)</td>
<td>30.9 bc (6.3)</td>
<td>9.3, &lt;.0001</td>
</tr>
<tr>
<td>Beck Depression Inventory</td>
<td>3.9 a (3.5)</td>
<td>9.2 ab (7.7)</td>
<td>15.2 bc (9.7)</td>
<td>19.5 c (7.5)</td>
<td>21.0 c (12.0)</td>
<td>16.5, &lt;.0001</td>
</tr>
<tr>
<td>% comorbidity (additional anxiety and/or mood disorder)</td>
<td>39.3</td>
<td>60.0</td>
<td>81.8</td>
<td>84.6</td>
<td>15.62, &lt;.002</td>
<td></td>
</tr>
<tr>
<td>% comorbidity (additional mood disorder)</td>
<td>10.7</td>
<td>26.7</td>
<td>54.5</td>
<td>42.3</td>
<td>12.61, &lt;.006</td>
<td></td>
</tr>
</tbody>
</table>

*Notes.* Values sharing a common letter do not differ at \( p < .05 \), Tukey HSD post hoc test. \( SD \) in parentheses. A Chi-square tests were used to assess differences in the percent of patients with an additional anxiety disorder or comorbid mood disorder (either current major depressive episode or dysthymia, including patients with partial remission). The EASI (Buss & Plomin, 1975) consists of four Scales, Emotionality, Activity, Sociability, and Impulsivity. The Emotionality Scale is an estimate of negative affect, and includes both fear and anger related items.
either the high or low groups on almost all the assessment instruments. Only on the Manifest Anxiety Scale was the social phobic group’s mean score significantly higher than the mean of the specific phobics.

The co-occurrence of additional diagnoses of anxiety or depression was similar to what has been observed in other clinical samples of the anxiety disorders. That is, the specific phobias and social anxiety groups showed a comorbidity incidence (at least one additional diagnosis) of approximately 40 and 60%, respectively, whereas the incidences for PTSD and panic were 82 and 85%. Similarly, the specific incidence of current major depression was only 11% for specific phobics. In contrast, panic and PTSD patients both showed an incidence of major depression that was over 40%. Social phobics were again intermediate, at 27%.

Physiological Activity Levels

Base startle magnitude. ITI responses provided an estimate of participants’ base reflex reactivity (Table 3, ITI probes). The test of differences between diagnoses was not quite statistically reliable, $F(4,116) = 2.31, p < .07$. Interestingly, however, the pattern of mean values was similar to that seen for the anxiety and depression questionnaires: Control subjects and simple phobic patients had the smallest probe responses, panic and PTSD patients were the largest, and social phobics were intermediate.

Prestimulus activity in autonomic and facial EMG measures. Heart rate base levels were recorded during the interval that immediately preceded the tones that cued sentence recall. As can be seen in Table 3, these initial levels differed significantly over the experimental groups, $F(4,125) = 7.7, p < .0001$. Panic patients had the highest base values, significantly higher than both social phobics and controls; control subjects showed the slowest heart rates, significantly slower than both specific phobics and panic patients. The PTSD patients were not statistically different from the other groups. Base skin conductance levels were also statistically significant across groups, $F(4,122) = 2.75, p < .04$ (see Table 3). However, only the extreme groups, controls and panic patients, differed significantly in the Tukey pairwise analyses.

Pre-onset levels of corrugator activity did not vary greatly across groups, and no test of diagnostic group difference approached significance.

Sentence Imagery: Evaluative Judgments

Affective reports. Sentence image ratings are presented in Table 4. For most patient groups, the personally relevant fear images were rated maximally unpleasant, with danger images next in aversiveness, followed by social fear imagery, fear sentence type $F(2,124) = 38.97, p < .0001$. Social phobics were an exception, Fear Sentence Type $\times$ Diagnosis $F(8,248) = 5.58, p < .0001$: Not unexpectedly, they rated social fear images more unpleasant than did the other groups, $F(4,125) = 5.98, p < .0005$, and equivalent in aversiveness to the other fear images. Groups did not differ significantly in valence ratings of clinical or danger imagery.

Arousal ratings were distributed in a similar pattern by diagnosis and fear imagery content, fear sentence type $F(2,124) = 24.68, p < .0001$; Fear Sentence Type $\times$ Diagnosis $F(8,248) = 3.96, p < .0005$. All patients rated the personal fear images most arousing, and there was no significant difference among groups. Danger images generally received high ratings, though less for panic disorders, diagnosis, $F(4,125) = 3.29, p < .02$. Paralleling their strong judgments of unpleasantness, social phobics rated social fear images more arousing than did the other groups, $F(4,125) = 4.52, p < .002$.

Vividness reports. Groups did not show a significant overall difference in reports of imagery vividness, whereas clinical fear imagery was rated as more vivid than the other two fear contents, $F(1,124) = 16.93, p < .0001$. As can be seen in Table 3, all groups gave nearly equivalent ratings to social and danger images. For clinical fear imagery, however, social phobics failed to show the greater image vividness reported by the other diagnostic groups, Diagnosis $\times$ Fear Sentence Type $F(8,248) = 2.31, p < .03$. Uniquely for this group—perhaps because of redundancy with the standard social fear cues—there was no significant difference among their ratings of the three fear contents. Overall, fear images were rated as less vivid than neutral images, $F(1,125) = 7.02, p < .01$.

Sentence Imagery: Potentiation of the Startle Reflex

As anticipated, the omnibus analysis of the startle probe reflex indicated larger blinks overall for fear sentences than for neutral sentences, $F(1,112) = 42.30, p < .0001$ (see Table 5). Furthermore, reflex potentiation during fear imagery (all contents together) varied with diagnosis, Fear versus Neutral $\times$ Diagnosis $F(4,112) = 3.44, p < .02$: The finding of greater startle magnitude during fear contents (relative to neutral) was highly reliable for control subjects and for specific and social phobics, all $p < .002$. The same test for PTSD patients also indicated reliable fear potentiation, $p < .03$; however, the overall difference in probe response between fear and neutral sentence contents was borderline for panic patients, $p < .06$.

Table 3. Startle Magnitude during Intertrial Intervals and Preimagery Base Values for Physiological Measures by Diagnostic Group

<table>
<thead>
<tr>
<th></th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>ITI startle</td>
<td>2.9 (4.1)</td>
</tr>
<tr>
<td>magnitude (µV)</td>
<td></td>
</tr>
<tr>
<td>Heart rate (B/M)</td>
<td>63.8 a (8.9)</td>
</tr>
<tr>
<td>Skin conductance</td>
<td>2.4 a (2.1)</td>
</tr>
<tr>
<td>(micromhos)</td>
<td></td>
</tr>
<tr>
<td>Corrugator muscle</td>
<td>6.2 (1.0)</td>
</tr>
<tr>
<td>(µV)</td>
<td></td>
</tr>
</tbody>
</table>

Notes. Values sharing a common letter do not differ at $p < .05$, Tukey HSD post hoc test. SD in parentheses.
Fear imagery potentiation varied significantly with the specific fear content [Fear vs. Neutral × Fear Sentence Type $F(2,111) = 3.75, p < .03$], and was largest overall for danger scenes and smallest for social fear scenes. The three-way interaction was not significant. Based on a priori hypotheses, however, separate analyses of diagnostic differences were conducted for each fear content. To illustrate these effects, fear/neutral difference scores are presented in Table 5 along with statistical results.

For personal fear imagery, a significant difference in potentiation was confirmed among diagnostic groups [Fear vs. Neutral × Diagnosis $F(4,112) = 2.28, p < .006$; see Figure 1]. When tested separately, control and specific and social phobics all showed significant fear potentiation, but this was not observed for panic or for PTSD patients. For social fear scenes, the interaction with group did not reach significance, $p < .13$. Nevertheless, the potentiation differences across diagnoses were similar to the findings for personal scene content, that is, social fear scene potentiation was significant for control participants and specific and social phobics, but did not approach significance for panic and PTSD patients. The most consistent potentiation among fear scenes was found for the danger content: Reliable potentiation was seen for control participants, specific phobics, social anxiety disorder, and panic patients, although it was less strong for PTSD, $p < .09$.

As previously noted, the groups did not differ significantly in baseline reflex magnitude. However, the pattern of means (largest for PTSD and panic patients) suggested that baseline magnitude might contribute to the overall pattern (base/change score $r = -.19$). However, this hypothesis was not supported by a subsequent covariance analysis of these same data, which produced the same results reported above. We also speculated that finding no potentiation during personal fear imagery for some of the patients might be attributed to the averaging of probe responses to the two personal scenes (on the assumption that one of the scenes was, by chance, less pertinent or less engaging). Thus, a post hoc analysis was conducted using only each participant’s largest personal fear startle reflex (which could have occurred during either of the two personal fear sentences). Startle potentiation was again not significant for panic patients, $F < 1$, and borderline for PTSD patients, $p < .09$.

### Table 4. Evaluative Judgments of Memory Imagery by Content and Diagnosis: Mean and Standard Deviations

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Control (n = 24)</th>
<th>Specific (n = 28)</th>
<th>Social (n = 30)</th>
<th>PTSD (n = 22)</th>
<th>Panic-A (n = 26)</th>
<th>$F$, $p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affective valence ratings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All fear</td>
<td>4.46 (2.29)</td>
<td>5.13 (2.51)</td>
<td>4.38 (2.09)</td>
<td>5.26 (2.95)</td>
<td>4.34 (2.82)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Personal</td>
<td>3.90 ab (3.24)</td>
<td>3.43 ab (3.08)</td>
<td>5.12 a (3.33)</td>
<td>3.59 ab (3.83)</td>
<td>2.60 b (2.99)</td>
<td>2.19 &lt; .05</td>
</tr>
<tr>
<td>Danger</td>
<td>3.44 (2.61)</td>
<td>3.98 (3.57)</td>
<td>3.95 (3.03)</td>
<td>4.68 (3.18)</td>
<td>4.42 (3.78)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Social</td>
<td>5.98 ab (3.08)</td>
<td>7.96 a (3.08)</td>
<td>4.15 b (2.95)</td>
<td>7.50 a (3.93)</td>
<td>5.87 ab (3.34)</td>
<td>5.98 &lt; .0002</td>
</tr>
<tr>
<td>Neutral</td>
<td>17.72 a (2.30)</td>
<td>16.93 ab (2.16)</td>
<td>15.26 b (2.18)</td>
<td>16.05 ab (2.56)</td>
<td>15.28 b (4.12)</td>
<td>3.94 &lt; .005</td>
</tr>
<tr>
<td>Arousal ratings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All fear</td>
<td>15.92 ab (2.98)</td>
<td>15.59 ab (3.76)</td>
<td>16.57 a (3.03)</td>
<td>14.02 ab (3.99)</td>
<td>13.99 b (3.80)</td>
<td>2.84 &lt; .03</td>
</tr>
<tr>
<td>Personal</td>
<td>15.96 (4.56)</td>
<td>17.95 (3.66)</td>
<td>17.74 (3.20)</td>
<td>16.18 (4.77)</td>
<td>17.00 (2.81)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Danger</td>
<td>16.75 b (4.03)</td>
<td>16.04 ab (4.20)</td>
<td>15.75 ab (4.27)</td>
<td>13.77 ab (4.99)</td>
<td>12.62 a (5.93)</td>
<td>3.39 &lt; .02</td>
</tr>
<tr>
<td>Social</td>
<td>14.94 ab (3.65)</td>
<td>12.80 a (5.40)</td>
<td>16.67 b (3.89)</td>
<td>12.11 a (4.59)</td>
<td>12.62 a (5.67)</td>
<td>4.52 &lt; .002</td>
</tr>
<tr>
<td>Neutral</td>
<td>2.56 (2.11)</td>
<td>2.88 (2.66)</td>
<td>4.01 (2.76)</td>
<td>4.09 (3.19)</td>
<td>3.34 (3.28)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Vividness ratings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All fear</td>
<td>14.27 (2.66)</td>
<td>13.32 (2.59)</td>
<td>12.28 (3.64)</td>
<td>12.48 (3.63)</td>
<td>12.86 (3.56)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Personal</td>
<td>15.98 b (3.29)</td>
<td>14.64 ab (3.74)</td>
<td>12.18 a (4.31)</td>
<td>13.98 ab (5.39)</td>
<td>14.23 ab (4.12)</td>
<td>2.90 &lt; .03</td>
</tr>
<tr>
<td>Danger</td>
<td>13.73 (3.46)</td>
<td>13.70 (3.22)</td>
<td>12.02 (3.97)</td>
<td>12.25 (3.09)</td>
<td>11.92 (4.12)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Social</td>
<td>13.06 (3.08)</td>
<td>11.63 (3.65)</td>
<td>12.47 (4.82)</td>
<td>11.20 (3.96)</td>
<td>12.54 (4.11)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Neutral</td>
<td>15.22 b (2.78)</td>
<td>14.37 ab (2.56)</td>
<td>12.62 a (3.32)</td>
<td>12.75 ab (3.95)</td>
<td>13.41 ab (3.44)</td>
<td>2.99 &lt; .03</td>
</tr>
</tbody>
</table>

Notes. All ratings were administered by computer and are based on a 21-point scale. Valence and arousal ratings are from the Self-Assessment Manikin (SAM; Lang, 1980). Values sharing a common letter do not differ at $p < .05$. Tukey HSD post hoc test. SD in parentheses. Lower scores on valence ratings indicate greater unpleasantness.

### Table 5. Startle Reflex Potentiation during Fear Memory Imagery: Means, Standard Deviations, and Significance Tests

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Personal</th>
<th>Danger</th>
<th>Social</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n = 22)</td>
<td>5.82 (7.68)</td>
<td>$p &lt; .002$</td>
<td>6.10 (9.15)</td>
</tr>
<tr>
<td>Specific (n = 27)</td>
<td>11.72 (17.00)</td>
<td>$p &lt; .002$</td>
<td>9.78 (15.72)</td>
</tr>
<tr>
<td>Social (n = 28)</td>
<td>3.57 (8.07)</td>
<td>$p &lt; .03$</td>
<td>7.88 (10.67)</td>
</tr>
<tr>
<td>PTSD (n = 19)</td>
<td>2.03 (9.27)</td>
<td>n.s.</td>
<td>3.26 (7.68)</td>
</tr>
<tr>
<td>Panic-A (n = 21)</td>
<td>0.81 (8.57)</td>
<td>n.s.</td>
<td>4.94 (7.44)</td>
</tr>
<tr>
<td>n.a (n = 21)</td>
<td>5.13 ab (11.51)</td>
<td>6.71 b (11.0)</td>
<td>3.89 a (10.17)</td>
</tr>
</tbody>
</table>

Notes. Reflex potentiation is defined as the $T$ value difference (fear minus neutral imagery). The significance of these differences is given for each diagnostic group for each image content. Tukey tests of the pairwise differences between groups in potentiation magnitude over all contents are given with the marginal values in the right column. Potentiation differences between contents over all participants are based on pairwise within-subjects tests (bottom row). For all these marginal values, a common lowercase letter indicates the absence of a significant difference.
An analysis was conducted for the Social Anxiety Disorder group to determine if there might be subtype differences within this diagnosis ($N = 30$). Unfortunately, only 4 members of the group could be classified as of the Specific Social Phobic type, precluding a meaningful statistical analysis of potentiation; the remainder were Generalized. Nonetheless, reflex potentiation during imagery of personal scenes for these 4 patients was indeed high, $T$ score = 12.61, and similar to the Specific Phobia group (see Table 5). Furthermore, when these 4 subjects were removed from the sample and the Social Anxiety Disorder group was reanalyzed ($N = 26$), personal scene potentiation was no longer significant, $p < .16$. Finally, startle potentiation to personal fear scenes was significant for the Social Anxiety group participants with only a single Social Anxiety Disorder diagnosis ($N = 12$; $p < .01$), but not significant for patients with additional diagnoses ($N = 18$; $F < 1$).

**Sentence Imagery: Physiological Responses to Image Processing**

**Heart rate.** The amount of heart rate change prompted by the sentence imagery task differed significantly over diagnoses, $F(4,125) = 3.43, p < .02$, and the four different image contents, $F(3,123) = 33.43, p < .0001$ (see Table 6). As expected, there was a general increase in heart rate during fear imagery, contrasted, for most participants, with a slight heart rate decrease for neutral imagery, which was confirmed by the planned contrast, fear versus neutral, $F(1,125) = 92.62, p < .0001$. When neutral image processing was analyzed alone, heart rate change did not vary significantly over diagnostic groups. However, heart rate increase during fear imagery differed significantly over diagnoses, $F(4,125) = 3.25, p < .02$, and also differed among the three fear categories, fear sentence type $F(2,124) = 14.18, p < .0001$. Specific and social phobics responded consistently with the greatest increases to all fear images, controls were intermediate, and PTSD and panic patients were the least responsive during fear imagery (see Figure 2). A retest of this analysis, covarying for base heart rate, produced the same result.\(^3\)

The planned contrasts of panic versus specific and social phobic patients were significant for both personal fear and danger imagery. Consistent with the startle data, the heart rate imagery response of panic patients to both these contents was markedly less acceleratory than for the two phobic groups, $p < .02$. The social fear content test (social anxiety disorder vs. all groups) only approached significance, $F(1,125) = 3.89, p < .06$.

**Corrugator.** The initial overall analysis again showed a strong difference among imagery contents, $F(3,119) = 10.5, p < .0001$, with corrugator muscle activity significantly greater for fear than neutral content, $F(1,121) = 31.14, p < .0001$. Furthermore, this effect interacted with diagnosis, $F(4,121) = 3.14, p < .02$. In follow-up tests, the effects of diagnosis were only marginal for neutral and for fear contents, both $p < .11$. Following this effect further with separate analyses of each fear type, only the diagnostic group test for personal fear scenes approached significance, $F(4,121) = 2.25, p < .07$. Specific phobia and PTSD patients showed a markedly greater mean corrugator response when processing clinically relevant fear imagery than did the other groups, contrast $F(1,121) = 8.46, p < .005$.

**Skin conductance.** This measure was consistent with heart rate and corrugator EMG activity in revealing palpable differences among contents, $F(3,120) = 8.49, p < .0001$, with fear imagery prompting significantly larger responses than neutral imagery, $F(1,122) = 23.32, p < .0001$. However, the overall analysis and the separate analyses for fear and neutral content were not significant for diagnosis, nor were the analyses of individual fear contents. The only significant planned contrast was for social content: Social phobics responded with greater skin conductance increase to social fear imagery than did the other groups, $F(1,122) = 4.7, p < .04$.

**Depression Comorbidity and Physiological Response**

Participants were divided into two groups, those having a current mood disorder diagnosis and those without (see Table 2 for percentage by diagnosis). For heart rate change during imagery, depressed and noncomorbid participants did not differ significantly for heart rate, skin conductance, or corrugator muscle. Similarly, these groups did not differ in startle potentiation during fear imagery, relative to neutral imagery. However, the magnitude of base startle reflexes (ITI) was significantly larger for anxiety patients with mood disorder (5.3 $\mu$V) than for patients without depression (3.1 $\mu$V), $F(1, 93) = 5.11, p < .03$. Furthermore, prestimulus corrugator muscle activity was greater than controls, $F(1, 93) = 9.17, p < .01$.

\(^3\)The overall correlation between base heart rate and imagery response was $- .13$. A covariance analysis (using base heart rate as the covariate) did not change the significance of the results reported here for ANOVA. This is consistent with an inspection of the means, in that higher base level differences among groups did not prompt reduced reactivity: Specific phobics recorded the second-highest base levels and the largest fear-related increases, whereas panic patients were average among the groups in baseline and the lowest reactivity. For skin conductance also, baseline levels failed to show a significant relationship to reactivity for any of the fear contents, and again covariance did not change the results of any analyses.
in amplitude for patients with comorbid depression than for nondepressed participants, $F(1,100) = 4.5, p < .04$.

**Moderator Variables**

There were no interactions between diagnostic differences and sex of the participant in analyses of either physiological change or base scores. The only significant gender effect found was that men had higher base skin conductance levels than women, $p < .03$.

All subjects were instructed to avoid prescription medications taken on an as-needed basis and recreational drug use 24 hr prior to psychophysiological evaluation. (Patients were not instructed to discontinue antidepressant medications.) Current use of antidepressant medications (tricyclics or SSRIs; 3 each in the specific phobia and social anxiety groups, 8 panic patients, and 1 PTSD). Although these numbers were too small to permit reliable comparisons between subgroups (drug/no-drug), reanalyses of startle potentiation by diagnostic group—including only patients not on medications—showed no change in the results.

Specific phobic, social phobic, and PTSD patients showed significant potentiation for all fear versus neutral content, $p s < .03$, whereas the result for panic patients no longer even approached significance, $p < .19$. For personal fear scenes, startle was again strongly potentiated for the two phobic groups, but not at all for PTSD and panic patients, $F s < 1$. Similarly, the pattern of results for heart rate was unchanged in the reanalysis of diagnostic groups, with patients on antidepressants excluded.

Eleven patients reported use of antianxiety medications (primarily benzodiazepines) in the 24 hr preceding the session (2, 1, 7, and 1 for specific phobia, social anxiety, panic, and PTSD, respectively). With these patients excluded, the overall pattern of results was again highly similar to the results obtained with the full sample. The same general startle potentiation effects were apparent, although the response of social phobics to personal fear scenes was now just below the significance level, $p < .07$. Heart rate outcomes were unchanged. In summary, the psychophysiological results of the study were not affected to any appreciable extent by medication use.

**Discussion**

The overall results are consistent with the view that reactions to fear memory cues differ significantly among the anxiety disorders. As hypothesized by the network coherence model, phobic participants were overall the most physiologically responsive at memory retrieval and panic patients tended to be the least reactive across all measures. However, the data are open to a variety of interpretations, as to the specific factors that may determine diagnostic differences in emotional expression.

The present findings replicate and extend the Cook et al. (1988) results: Panic patients consistently scored higher than phobics or the socially anxious on depression and anxiety questionnaires (see also Turner, McCann, Beidel, & Mezzich,
Fear imagery and anxiety

1986), consistent with high trait negative affect (Zinbarg & Barlow, 1996). In the present research, furthermore, patients diagnosed with panic had a higher incidence of comorbid depression (42%) and additional anxiety disorder (77%). PTSD patients also differed from phobia and socially anxious patients, reporting more anxiety symptoms on questionnaires and at interview, and they also showed a high incidence of mood disorder (55%). Finally, these groups with higher questionnaire scores and greater comorbidity, panic and PTSD patients, also differed from the other groups in showing smaller heart rate changes and no startle potentiation to personal fear memories.

**Base Levels**

Panic patients tended to have somewhat higher base physiological levels than the other diagnoses. Their mean values were the highest of all groups for heart rate and skin conductance (only base heart rate differed significantly over diagnosis, with values for panic patients significantly greater than normal controls and the social anxiety group). Panic patients were also highest in base startle response (with PTSD second), although again the overall analysis of the group means fell short of significance.

**Startle Potentiation**

In numerous studies with animals and humans (Davis, 1992; Davis & Lang, 2003), probes presented during exposure to fear-conditioned stimuli potentiate the startle reflex. In the present clinical experiment, the normal control, specific phobic, and social anxiety disorder groups showed a similar, significant probe startle potentiation during personal fear imagery. Potentiation was not generally found for panic or PTSD patients. Furthermore, additional analyses of the social anxiety patients showed patients diagnosed specific/social phobic subtype were critical to the positive findings for social anxiety group. That is, when the few specifics were dropped from the analysis, potentiation during personal fear scenes could not be confirmed for the social anxiety group. Thus, it is increasingly clear that fear text imagery instigates a stronger, more reliable fear physiology in focal phobics, relative to other anxiety diagnoses—consistent with Cook et al. (1988) and McNeil et al. (1993).

**Negative Affect**

Overall, the data suggest that the high negative affect seen in panic and PTSD patients, defined here by a higher incidence of comorbidity and higher scores on the questionnaires (see Watson & Clark, 1984), might directly determine their reduced reaction to fear cues. Furthermore, the internal analysis of the social anxiety groups lends support to this hypothesis. Patients from this group with additional anxiety diagnoses (greater anxiety comorbidity) were similar to panic patients—i.e., they did not show the significant startle potentiation during fear scenes that was found in patients with the single, primary diagnosis of social anxiety.

Despite the above, several factors suggest that the relationship between negative affect and a reduced fear imagery response, if truly causal, is complex and indirect. First, when the correlations between questionnaires and physiology were computed, independent of diagnosis, the resulting values were low and nonsignificant. Second, although comorbid mood disorder was significantly associated with base corrugator activity and noncued (ITI) startle magnitude, it was not independently related to any measure of reactivity. Furthermore, although the PTSD and panic groups had similar questionnaire scores and incidence of comorbidity, patterns of physiological response over fear imagery contents were different for the two diagnoses. For example, PTSD patients showed the highest corrugator (“frown”) muscle reactivity to personal fear memories (along with specific phobics, significantly greater than all other groups). On the other hand, significant startle potentiation to danger scenes was found for panic, but not for PTSD. These findings suggest that different factors orchestrate physiological responding in the two diagnoses.

**PTSD, Panic, and Nonanxious Controls**

The mean baseline activation of the normal control participants was the lowest of all experimental groups, and for heart rate and skin conductance, significantly less than panic patients. The other anxiety diagnoses did not differ consistently from controls in base values. Furthermore, controls, social anxiety patients, and specific phobics showed similar reactivity to fear memories. For these three groups, the defense motive system was generally functioning normally: They responded with appropriate arousal to fear cues—however inconvenient or inappropriate the fears might be. Interestingly, these groups had the lowest scores on all questionnaire measures of psychopathology and the lowest incidence of comorbid depression. In contrast, panic and PTSD patients showed a broader, more severe affective pathology, but a hyporeactive physiology in fear imagery. The findings suggest that defense responding in these disorders—characterized by a more general pathology—is somehow compromised.

Considering the fact that an exaggerated sensitivity to startle cues is a pathognomonic sign of PTSD in *DSM-IV*, the startle results appear to merit particular comment. In fact, laboratory studies of traumatized patients have not produced results wholly consistent with the broad DSM view. For example, Grillon, Morgan, Davis, and Southwick (1998) reported that base blink reflex magnitude did not differ between Vietnam combat veterans with PTSD and either veteran or civilian non-PTSD controls. Furthermore, although the veterans showed a relatively greater increase in startle when under “threat of shock,” the actual increase, compared to a safe condition, did not differ among the three groups. Using a different measure, parallel response inconsistencies in conditioning were noted by Machulda (1999). In brief, the role of startle in PTSD continues to be a work in progress, particularly in terms of comparative startle sensitivity among anxiety disorders.

**Physiological Hyporeactivity in Anxiety**

The finding of a reduced physiological fear reaction in the most distressed patients seems at first counterintuitive, and one naturally first looks for explanation in possible differences among the personal texts. These texts were developed in collaboration with each participant. It was emphasized that the fear text should describe their “worst fear” experience. The consistency in participant’s emotional ratings across groups suggests that they generally followed this instruction (panic patients actually had the most extreme negative valence ratings, and their arousal ratings were within the same high range as the social anxiety and phobic groups). As can be seen from the examples in Table 1, although scenes varied in specific content, the texts for all participants contain cues to physiological reactions, as well as to the sensory context and cognitive elaborations reported to be part of the fear experience. In all cases, the patient is confronting the feared event or it is imminent and physiological and behavioral distress is ongoing. Never-
theless, despite imaging evocative texts that were similar in rated affect to those of the other groups, panic and PTSD showed reduced startle and heart rate responses. Moreover, it appears to be a characteristic image/text processing reaction for these patients, as their mean physiological response to standard fear scenes was also generally lower than the other experimental groups.

**Autonomic flexibility.** Discordance between affective or physiological symptom reports in anxious patients, and measured behavioral or physiological reactivity, has frequently been observed (e.g., Lang, 1968, 1978; Mandler, Mandler, Kremen, & Sholiton, 1961; Pennebaker, 1982). Furthermore, there are data suggesting that it is associated with a more generalized pathology. Relative to normal controls, patients with Generalized Anxiety Disorder (GAD) or panic have shown reduced reactivity on a variety of tasks in Hoehn-Saric, McLeod, and Zimmerli (1989, 1991). Roemer and Borkovec (1993) also reported reduced autonomic reactivity in GAD. Kirsch and Geer (1988) found a similar reduced response in women with Post-Menstrual Syndrome.

Hoehn-Saric and McLeod (2000) have suggested that anxiety patients have a chronically less flexible autonomic nervous system (ANS). A related view is held by Thayer and colleagues (e.g., Friedman & Thayer, 1998), who found that patients with Generalized Anxiety Disorder show less heart rate variability at rest than normals. They suggest that this reflects a deficit in the parasympathetic branch of the ANS: Autonomic balance is chronically weighted in a sympathetic direction, associated with a sustained arousal but impaired reactance. This physiological pattern is seen to be a consequence of the patient's cognitive style (e.g., Borkovec & Inz, 1990). That is, GAD patients are characterized by persistent worry—language processing that has the effect of elevating and flattening the heart rate sinus arrhythmia. Friedman, Thayer, Borkovec, and Tyrell (1993) have reported a resting cardiac pattern in panic patients that is similar to that found for GAD, whereas this reduced heart rate variability was not found in specific (blood) phobics. An affinity between panic (with agoraphobia) and generalized anxiety disorder, compared to more focused phobic states, is suggested by studies of personality questionnaire patterns (e.g., Turner et al., 1986). Thus, different habitual patterns of worry and an associated reduced cardiac variability, along with high negative affect, could be a determinant of the response reactivity differences seen here between panic and phobia.

**Brain function and the anxiety response.** Gray (1987) has proposed an elaborate theory of brain function in anxiety that might also accommodate the hyporesponding observed in the present experiment. In Gray's conceptual brain, a “behavioral activation system” (BAS) mediates reactivity to motivationally relevant stimuli, presumably including the abrupt metabolic mobilization that precedes and accompanies “fight or flight.” This is contrasted with the “behavioral inhibition system” (BIS), mediating passive avoidance, and approach–avoidance conflict.

In a recent revision of the theory (McNaughton & Gray, 2000), the amygdala circuit described by Davis (1992; see also LeDoux, 1992; Fanselau, 1994)—which also determines potentiated startle—is seen as the activation system mediating the “fear” response. In contrast, the BIS is a septal-hippocampal circuit mediating suppression of approach and an excessive avoidance of threat. Davis (1992; see also, Lang, Davis, & Öhman, 2000; Davis & Lang, 2003) has also proposed an “anxiety” path that is separate from “fear,” but one different from the BIS, and involving the bed nucleus of the stria terminals.

As Fowles (2000) points out, the BAS/BIS systems seem to map on to Barlow's (1988) distinction between fear and anxiety. From this perspective, phobia might be determined by the amygdala fear circuit, whereas GAD is the prototypic “anxiety” disorder. However, more than phobia, Barlow views panic as specifically driven by the fear system. The panic attack is defined by “a subjective sense of extreme fear or impending doom accompanied by a massive autonomic surge and strong flight-or-fight behavioral action tendencies” (Bouton, Mineka, & Barlow, 2001, p. 7). Furthermore, as Craske (1999, p. 26) states, “In contrast to worry, ...anticipatory anxiety, elicited by an approaching feared stimulus, is associated with definite autonomic arousal relative to baseline conditions.” Thus, the memory of an imminent or ongoing panic attack should prompt an associated somato-visceral response. The present data set suggests that, on the contrary, the reactivity of panic patients is more like GAD—a blunted autonomic and somatic response. It

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4There is now considerable psychophysiological evidence that patterns of response vary with the imminence of threat (temporal, spatial distance from danger or pain) in animals (Blanchard & Blanchard, 1989; Campbell, Wood, & McBride, 1997), and to some extent in humans (Lang et al., 1997). In the context of patient’s fears, however, the time/distance parameter is often reduced to a symbolic analogue that is difficult to quantify. What seems clear, nevertheless, is that for all participants, their “worst fear” involved a reported active visceral and somatic physiology, which may be the best index of symbolic, close “imminence.” None of the scenes included what might be considered blunting, cognitive symptoms of anxiety. Scenes for all groups were similar. They were immediately anticipatory or at full engagement with the fear event, that is, in the Fanselau (1994) circa-strike zone (see Table 1). An important related feature is the degree of event predictability/controllability possessed by the victim. In the real world, things are generally less predictable for the panic patient. In this laboratory context, however, all the panic texts involved an already developed fear physiology. Phasic unpredictability can heighten activation—at least at first. However, persistent, chronic uncontrollability of aversive events will, over time, produce a state of “learned helplessness” (Seligman, 1975), characterized by a general blunted sympathetic reaction to uncertain fear cues. The view would not be inconsistent with the finding that panic and PTSD patients are less physiologically reactive to their fear memories.

5Based on research using the electroencephalogram to analyze brain asymmetry, Heller, Nitschke, and colleagues have defined two anxiety trait characteristics in the normal population that appears to parallel Gray's two systems (e.g., Nitschke, Heller, Palmeri, & Miller, 1999; see also Bruder, Fong, Tenke, & Leite, 1997). They propose that a fundamental distinction can be made between a trait pattern of “anxious arousal (somatic anxiety)” and “anxious apprehension (worry).” Participants with apprehension anxiety (defined by the Penn State Worry Questionnaire: Meyer, Miller, Metzger, & Borkovec, 1990) are held to show greater posterior hemisphere brain activity than right, whereas it is predicted that participants characterized by anxious arousal will show greater right posterior activity. The supporting data are suggestive, but mixed (e.g., Nitschke et al., 1999). A possible mechanism for the brain effects hypothesized for anxious apprehension might be: Worry involves persistent language processing. Language processing centers are generally left lateralized. Thus, greater activity would be seen in the left hemisphere. Furthermore, if this cognitive pattern characterizes panic and GAD, and not phobics, these diagnoses might show differences in brain asymmetry that parallel those expected for anxious apprehension and anxious arousal.
is not obvious, however, that there is a common mechanism. Panic patients may either fail to accurately encode panic attacks (e.g., input to the distance receptors that cues a phobic could be more readily encoded verbally than the physiological response information that may cue panic) or succumb to some inhibitory interference or disattention at the retrieval stage that compromises the verbally mediated, reevocation of a fear experience.

**Attention, cognition, and language processing.** Distraction is a possible fear–cue countermechanism. If the brain is continuously occupied processing worries, cognitive resources are not available to attend to external cues or to retrieve the relevant fear memories. It is also known that the “spotlight of attention” can be directed to one part of the perceptual field, to the neglect of other features that might otherwise be salient (Posner & Petersen, 1990). For PTSD patients, particularly, such an altered attentional state is described as dissociation—related to a dulling of responsivity, emotional “numbing.” Feeny, Zoellner, Fitzgibbons, and Foa (2000) reported that severity of initial and chronic PTSD-assaulted women was related to patterns of depression, numbing, and dissociation (Dissociative Experiences Scale: Bernstein & Putnam, 1986). The dissociative mechanism is sometimes considered to be an effortful suppression of fear cue information, or alternatively, an automatic process by which fear information is rendered unavailable (see Wenzlaff & Wegner, 2000).

Differences in the prevalence of suppression/dissociation might explain the apparently greater heart rate responsivity seen in PTSD veterans (e.g., Pitman et al., 1987), than was observed here in predominantly female, nonveteran patients. These variables clearly need further study. Ehlers, Mayou, and Bryant (1998), for example, found evidence that patients injured in auto accidents who tried to suppress thoughts about the trauma were more likely to show PTSD symptoms 1 year later. Unfortunately, the present sample of PTSD subjects (N = 22) lacks the power for an analysis of subtypes (dissociative vs. nondissociative; suppressors vs. nonsuppressors) that might help to clarify these issues.

At present, it is not known how an inhibitory reaction to emotional cues can be generally achieved. Autonomic arousal is a frequently observed response in thought suppression experiments (e.g., Gross & Levenson, 1993). It is not clear, however, if the observed arousal is an emotional reaction, expressed despite suppression, or alternatively, an arousal physiology that is the product of the effort of suppression. Vrana, Cuthbert, and Lang (1989) studied college student subjects, using a paradigm similar to the one employed here, in which participants were asked to delay their imagery response (not respond immediately to the tone cue). These healthy subjects were indeed able to considerably reduce their heart rate response during this delay period, but not so much that the fear–neutral difference was completely eliminated.

**Encoding fear memories.** It has been suggested (Lang, 1979, 1985, 1994; see also Foa & Kozak, 1986) that fear-event memories have an associative structure that includes three broad categories of information: These are information about the evoking stimulus, as a sensory event; information about the reacting individual’s responses—behavioral acts and somatic and autonomic patterns of affective arousal; and associated semantic elaboration (about where, when, what, how) that broaden the context and define its meaning as a fearful event. It is clear that many of these data are not encoded linguistically—particularly stimulus and response information. Human beings can report, for example, only a fraction of the enormous data set that is transmitted from retina to occipital cortex, when a visual stimulus impinges on the eye. The computation that occurs during perceptual processing, resolving image into object, is also unavailable for comment. Similarly, the complex changes in the cardiovascular system and the assembly of muscle actions involved in an escape response are, in the main, completely unknown to the actor. Furthermore, it is probable that most associative connections that link the units of a fear representation are formed independently of language mediation. That is, the learning of patterns of autonomic and somatic arousal, escape and avoidance, the creation of event memories that determine future behavior, appear to follow conditioning processes that are essentially the same in humans and in organisms that wholly lack any capacity for language.

The semantic elaborations (“All snakes are dangerous”; “I’m having a heart attack”; “The audience probably thinks I’m ridiculous”), that is, meaning information, are mainly coded linguistically. We often view such language as a kind of carryall that can transfer an affective reaction (behavioral and physiological) to contexts that are remote from the stimulus environment in which the primary association was formed (e.g., the cognitive theories of Beck, Emery, & Greenberg, 1985; Clark, 1996). On the other hand, the relationship of verbal responses to the other information (behavioral and physiological) can be tenuous, absent, or unidirectional in associative strength (e.g., physiological activation might prompt descriptive language, but language might not evoke the physiology). In the present experiment an effort was made to assure that the patient had, at least, coded the verbal description of the fear event in memory. That is, patients memorized their personal fear sentences beforehand to a common criterion. Furthermore, the sentences were based on information from a structured interview and contained descriptive language for all three elements in the fear structure—stimulus, response, and meaning information. Finally, all patients, regardless of diagnoses, reported their fear imagery experiences to be affectively negative and highly arousing. Nevertheless, as noted, diagnoses characterized by a broad range of neurotic complaints (panic and PTSD) failed to respond to these prompts with a robust physiology of fear.

To summarize, assuming that the patient’s conditioning history is a major determinant of anxiety disorder (e.g., Bouton et al., 2001), we may conjecture that some patients have not acquired language representations that are strongly associated with the efferent and sensory (perhaps, interoceptive) memories of fear. Thus, fear language input (“Tell me about your fear.”) gets fear language output (a report of a fear experience).
It is important to note in conclusion that, although there has been much speculation concerning physiology’s role in determining the different anxiety pathologies, there are as yet very few data comparing physiological reactivity across the spectrum of anxiety disorders, considering the wide range of relevant tasks and challenges. To decide among the various explanatory models, we will need to repair this deficit. It is only with a view from a larger data platform that we will be able to determine if diagnostic differences like those observed here reflect variations in attentional patterns, language processing, associative learning, efferent inhibition, or some interaction of all these factors with the dynamics of memory retrieval.

REFERENCES


Deffenbacher, K. A. (1983). The influence of arousal on reliability of data comparing physiological reactivity across the spectrum of anxiety disorders, considering the wide range of relevant tasks and challenges. To decide among the various explanatory models, we will need to repair this deficit. It is only with a view from a larger data platform that we will be able to determine if diagnostic differences like those observed here reflect variations in attentional patterns, language processing, associative learning, efferent inhibition, or some interaction of all these factors with the dynamics of memory retrieval.


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