

## Biological Challenge Manipulation of PCO<sub>2</sub> Levels: A Test of Klein's (1993) Suffocation Alarm Theory of Panic

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D. F. Klein (1993) proposed that patients with panic disorder (PD) have a hypersensitive suffocation monitor that predisposes them to experience panic attacks under certain conditions. The suffocation alarm theory predicts differential emotional responding to biological challenges that affect arterial partial pressure of carbon dioxide (PCO<sub>2</sub>). These PD patients should exhibit (a) lower fear and less likelihood of panic in response to biological challenges that lower PCO<sub>2</sub> levels (e.g., hyperventilation), and (b) increased fear and greater likelihood of panic in response to biological challenges that raise PCO<sub>2</sub> levels (e.g., inhalation of 35% CO<sub>2</sub> gas). The following indicators of the suffocation monitor were assessed: (a) severity of dyspnea symptoms, (b) frequency of dyspnea symptoms, (c) heightened respiration rate, and (d) lowered PCO<sub>2</sub> levels. Ratings of physiological and subjective responding, as well as panic, were obtained during both a hyperventilation and a 35% CO<sub>2</sub> challenge. None of the classification methods predicted differential emotional responding to hyperventilation versus 35% CO<sub>2</sub> challenge.

During the past decade, there has been a proliferation of research on the etiology of panic disorder (PD) from both biological and psychological perspectives (e.g., Ballenger, 1990; McNally, 1990). Klein's (1993) intriguing suffocation alarm theory of panic proposes that PD patients possess hypersensitive suffocation monitors that produce false suffocation alarms. The proposed suffocation detector monitors arterial partial pressure of carbon dioxide (PCO<sub>2</sub>) levels and triggers a suffocation alarm when rising PCO<sub>2</sub> levels signal that asphyxiation is imminent.

Klein (1993) integrated a variety of data to draw a linkage between suffocation and panic. Klein argued that dyspnea is common in patients with PD but rare in fear reactions among nonpatients. Nonpatients experiencing fear typically recall cardiovascular (e.g., heart palpitations) rather than respiratory (e.g., dyspnea) symptoms (McMillian & Rachman, 1988). On the other hand, respiratory distress is commonly reported by

PD patients during spontaneous panic attacks (Gross, 1989; Katerndahl, 1988; Keyl & Eaton, 1990). In addition, biological challenge agents that produce physiological correlates of asphyxiation (e.g., high levels of CO<sub>2</sub>) are reliably panicogenic in PD patients (Hollander, Liebowitz, Fyer, Gorman, & Klein, 1989; Levin et al., 1987; Woods, Charney, Goodman, & Heninger, 1988). Klein also highlighted the association between respiratory conditions and panic. For example, PD is prevalent in patients with pulmonary disease (Yellowlees, Alpers, Bowden, Bryant, & Ruffin, 1987; Yellowlees, Haynes, Potts, & Ruffin, 1988; Yellowlees & Kalucy, 1990).

Patients with PD characteristically display chronic hyperventilation (Gorman & Papp, 1990). Klein offered this as evidence that these patients are attempting to correct for the hypersensitive suffocation alarm by increasing the buffer between arterial PCO<sub>2</sub>, which is lowered during hyperventilation, and the alarm threshold. Chronic hyperventilation, which lowers PCO<sub>2</sub> levels, is posited as a coping response to the lowered suffocation threshold so as to prevent frequent firing of the alarm.

Klein's (1993) suffocation alarm theory has inspired several studies. Asmundson and Stein (1994) compared the maximal duration of voluntary breath holding in patients with PD, patients with generalized social phobia, and controls. No group differences in end-tidal CO<sub>2</sub> levels were found either before or after the breath-holding task. However, PD patients showed a significantly shorter duration on the breath-holding task compared to the other groups. Thus, PD patients tended to discontinue breath holding even though they were not significantly more hypercapnic (i.e., did not have excessive CO<sub>2</sub> in the blood). These findings were interpreted as evidence that PD patients attempt to avoid the activation of their hypersensitive suffocation alarm. However, these findings are also consistent with an alternative psychological explanation.

Cognitive models of panic (cf. Clark, 1986; Reiss & McNally, 1985) suggest that PD patients possess a cognitive disposition

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to panic in the context of aversive sensations. According to these models, panic is created when benign physical symptoms are catastrophically misinterpreted. Perceiving the dyspnea sensations that are produced during breath holding, patients with PD in the Asmundson and Stein (1994) study may interpret these sensations as indicating suffocation. The belief that one is suffocating will necessarily result in the termination of the breath-holding procedure. Thus, findings from the Asmundson and Stein study are also consistent with the hypothesis that PD patients, compared to individuals with social phobia or non-phobic controls, display greater fear of unpleasant respiratory sensations.

Pine et al. (1994) tested the hypothesis that individuals who are unable to perceive hypercapnia would display lower anxiety compared to those with accurate CO<sub>2</sub> perception. They examined the rates of anxiety disorders and anxiety symptoms in children with congenital central hypoventilation syndrome (CCHS), children with asthma, children with other chronic medical conditions, and controls. Pine et al. predicted lower rates of anxiety in the children with CCHS because it is a disorder of the central nervous system in which children cannot increase respiration in response to hypercapnia and do not report dyspnea even when they are significantly hypercapnic. This prediction was partially supported—children with CCHS showed a nonsignificant trend indicating lower rates of anxiety disorders compared to children with asthma. Children with CCHS also reported significantly fewer anxiety symptoms compared to children with chronic illnesses and the community sample as a whole.

Pine et al.'s (1994) findings are consistent with Klein's (1993) theory as well as cognitive models of panic. A psychological model of panic would posit that children with CCHS are less likely to experience anxiety because they lack the capacity to detect sensations that could lead to anxiety. Children become frightened of benign dyspnea sensations when they overestimate the danger of such sensations. However, children with CCHS are unable to perceive dyspnea and, therefore, are unable to catastrophically misinterpret these sensations. Thus, it may be the absence of a necessary cognitive mechanism, rather than the presence of a dysregulated biological alarm, that is responsible for the lower levels of anxiety symptoms among children with CCHS.

Gorman et al. (1994) examined the anxiogenic effects of 5% CO<sub>2</sub> inhalation, 7% CO<sub>2</sub> inhalation, and hyperventilation in patients with PD and controls. None of the controls had a panic attack during the hyperventilation challenge or 5% CO<sub>2</sub> challenge, whereas 13% of the patients with PD panicked during the hyperventilation challenge and 29% panicked during the 5% CO<sub>2</sub> challenge. During the 7% CO<sub>2</sub> challenge, 12% of the controls panicked compared to 68% of the patients with PD. These findings are consistent with the hypothesis that PD patients display a hypersensitivity to CO<sub>2</sub>. However, similar to the studies reviewed above, the differential reactions displayed by PD patients may be explained by either an aberrant suffocation monitor or the fear of challenge-induced sensations. Salkovskis and Clark (1990) argued that the wide range of biochemical panic-provoking agents, such as CO<sub>2</sub>, produce panic through their ability to create physical sensations that can be misinterpreted rather than through some specific biochemical pathway.

Despite the support for Klein's (1993) suffocation model of panic, the findings reviewed above are not compelling. It is unclear whether the group differences are best accounted for by the proposed hypersensitive alarm or whether there are psychological mechanisms that may account for the differences. As noted, the findings are also congruous with cognitive models of panic in which PD patients are believed to possess a cognitive disposition that leads to panic in the context of dyspnea sensations. More rigorous tests are needed such that findings that support or disconfirm Klein's theory are less prone to alternative interpretation.

The present study was designed to provide a less ambiguous test of the purported suffocation monitor. Because the present level of technology does not allow us to directly measure the suffocation monitor, this monitor is best considered a construct that can only be indirectly assessed. Thus, we investigated several markers of the suffocation alarm construct. We derived these markers from Klein's (1993) theory, which suggests that CO<sub>2</sub> hypersensitivity can be operationalized in several ways: (a) self-reports of dyspnea symptom severity, (b) frequency of dyspnea symptoms during spontaneous panic attacks, (c) increased respiration rate, and (d) lowered PCO<sub>2</sub> levels.

The principal aim of this study was to evaluate several operationalizations of the hypersensitive suffocation alarm in the context of biological challenges that affect CO<sub>2</sub>. Patients with PD can be classified as exhibiting greater or lesser CO<sub>2</sub> hypersensitivity according to the degree to which they display the characteristic marker (e.g., dyspnea symptoms, increased respiration, decreased PCO<sub>2</sub> levels). Patients exhibiting greater hypersensitivity should also show differential responding to biological challenges that significantly change PCO<sub>2</sub> levels. Specifically, more hypersensitive patients should exhibit (a) lower fear and fewer panic attacks in response to biological challenges that lower PCO<sub>2</sub> levels (e.g., hyperventilation), and (b) increased fear and more panic attacks in response to biological challenges that raise PCO<sub>2</sub> levels (e.g., inhalation of 35% CO<sub>2</sub> gas).

## Method

### Participants

The sample consisted of 71 participants who met the following entry criteria: (a) principal *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM-III-R*; American Psychiatric Association, 1987) Axis I diagnosis of PD; (b) at least one panic attack during the past 4 weeks; (c) no change in medication type or dose during the past 8 weeks; (d) no evidence of serious suicide intent; (e) no evidence of current substance abuse; (f) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder; and (g) no medical history of respiratory disease, renal disease, heart disease, epilepsy, or stroke. Participants' mean age was 33 (range = 21–65). The majority of participants were female (68%) and Caucasian (67%). Participants were drawn from a pool of individuals presenting at an academic research center specializing in the evaluation and treatment of anxiety disorders.

Diagnostic assessment was based on a structured diagnostic interview using the Structured Clinical Interview for *DSM-III-R*—Nonpatient Edition (SCID-NP; Spitzer, Williams, Gibbon, & First, 1990). Interviews were conducted by advanced graduate students in clinical psychology who had received extensive training in SCID administration

and scoring. Each interview was reviewed by both Norman B. Schmidt and Michael J. Telch during weekly staff meetings. Twenty-one videotaped SCID-NP interviews were randomly selected for reliability analysis that indicated 100% interrater agreement for PD diagnosis ( $\kappa = 1.0$ ). Medication status and medical history were assessed by the project physician based on a semistructured clinical interview. Of the original pool of participants, 78 completed the diagnostic assessment and the biological challenges. Seven of these participants had missing data and were excluded from analyses.

### *Operationalization of the Hypersensitive Suffocation Monitor*

Four indices were used to operationalize the hypersensitive suffocation monitor. Two respiration indices (i.e., resting respiration rate, resting end-tidal  $\text{PCO}_2$  levels) and two symptom indices (i.e., dyspnea symptom frequency, dyspnea symptom severity) served as physiological and subjective markers of a hypersensitive suffocation monitor. The dyspnea severity index was based on reported severity of dyspnea symptoms during the past week (i.e., difficulty in getting one's breath or overbreathing, smothering or choking sensations). The dyspnea frequency index was based on the percentage of spontaneous panic attacks with dyspnea symptoms, relative to all spontaneous panic attacks, during a 1-week prospective self-monitoring period.

### *Assessments*

#### *Physiological Measures*

**Vital capacity (VC).** The Respirodyne II Plus respirometer and Respirodyne disposable flow sensors (Sherwood Medical, St. Louis, MO) were used to measure each participant's VC. VC is measured in liters and is the maximum volume of air that can be moved in and out of the lungs. VC was assessed three times and averaged to yield a VC index.

**$\text{CO}_2$  intake volume.**  $\text{CO}_2$  intake volume indexes the amount of  $\text{CO}_2$  inhaled by the participant during the provocation, relative to the participant's VC. The amount of  $\text{CO}_2$  remaining in a 4.8 liter venticomp bag following the inhalation procedure was measured and subtracted from the participant's VC.

#### *Psychophysiological Measures*

Psychophysiological monitoring provided an index of sympathetic responding to the challenges. Data were continuously monitored by a J & J I-330 Physiological Monitoring System (J. & J. Engineering Inc., Poulsbo, WA). The skin was prepared by cleansing with rubbing alcohol followed by an abrasive and a conductive electrode medium (Parker Signa Creme; Parker Laboratories, Inc., Orange, NJ). Heart rate (beats per minute) was measured by a J & J heart rate module P-401 with three silver/silver chloride electrodes attached to a cardiometer electrode adapter and placed on three digits, the most distal pad of the first and third digit of the nondominant hand and the most distal pad of the third digit of the dominant hand. Skin conductance (in microsiemens) was measured by J & J Electrodermograph Module T-601 with two silver/silver chloride electrodes that were attached to the middle pad of the fourth and fifth digits on the nondominant hand. A pneumograph was fitted to the participant's chest on top of the clothing to measure respiration rate (breaths per minute), and end-tidal  $\text{PCO}_2$  levels (mmHg) were monitored by a TMM Capnometer Model 2200 (Traverse Medical Monitors, Saline, MI) that was interfaced with the J & J I-330 System using a small rubber tube attached to a sample line leading to the capnometer taped below the participant's left nostril.

### *Self-Report Measures*

**Hyperventilation Checklist (HVC).** The HVC (Schmidt & Telch, 1994) is a 16-item scale assessing physical symptoms (e.g., shortness of breath, palpitations) and fears (e.g., fear of dying, fear of going crazy) associated with panic attacks that has been used in hyperventilation challenge tasks. The HVC closely matches criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) for panic as well as other acute measures of panic symptoms such as the Acute Panic Inventory (API; Liebowitz, Gorman, Fyer, Dillon, & Klein, 1984). However, the HVC includes ratings of two physical sensations that are rarely associated with hyperventilation to assess for response bias (i.e., sweet taste in mouth, itchiness on bottom of feet). Symptoms are rated on a 5-point Likert scale (0 = *absent*; 4 = *very severe*). The HVC total score is the sum of the 14 symptoms. The two response bias items were analyzed separately. Subjective level of anxiety was measured using a 100-point Subjective Units of Distress (SUDS) scale. Ratings for SUDS were anchored along a panic-relevant continuum (0 = *not at all anxious*; 50 = *moderately anxious*; 100 = *full-blown panic*). The presence of a panic attack was determined by a composite index of self-reported distress including (a) endorsing the presence of a panic attack; (b) reporting a 30-point increase in SUDS from baseline to challenge indicating a significant increase in anxiety; and (c) reporting four or more *DSM-IV* panic symptoms as moderate to severe during the challenge.

**Texas Panic Attack Record Form.** Participants were provided with panic diary forms modeled after those used in the Upjohn Multi-Center Panic Study (Ballenger et al., 1988). For each panic episode, participants recorded the (a) date; (b) time; (c) duration; (d) severity; (e) symptoms experienced including dyspnea; (f) setting parameters (e.g., place, activity, accompanied); and (g) type of attack (e.g., spontaneous, situational).

**API.** The API is a 17-item inventory for assessing symptoms of arousal associated with panic attacks (Liebowitz et al., 1984). It has been used extensively in panic provocation studies (Gorman, Papp, & Klein, 1990; Harrison et al., 1989). Participants rate the severity of each symptom from 0 (*absent*) to 3 (*severe*). Questions on the inventory include: "Did you feel faint?" "Were you afraid of dying?" The API includes a SUDS rating of self-reported anxiety and breathlessness (0 = *not disturbed at all*, 100 = *the worst imaginable experience*). The API also includes a "Yes" or "No" response question used to assess subjective report of panic in response to the challenge. The presence of a panic attack was determined by a composite index of self-reported distress including (a) endorsing "yes" on the API panic attack question; (b) reporting a 30-point increase in SUDS from baseline to challenge indicating a significant increase in anxiety; and (c) reporting four or more symptoms as moderate to severe during the challenge.

**Sheehan Patient-Rated Anxiety Scale (SPRAS).** The SPRAS (Sheehan, 1983) is a widely used and well-validated self-report scale for assessing the intensity of anxiety symptoms. Each of the 35 symptoms (e.g., shaking or trembling) is rated on a five-point scale ranging from 0 (*not at all distressing*) to 4 (*extremely distressing*). The instructions were modified so that symptom ratings were based on a 1-week time frame.

### *Procedure*

After completing the screening interview, SCID, and SPRAS, participants underwent a 2-day biological challenge assessment, which involved a hyperventilation challenge on the first day and a 35%  $\text{CO}_2$  challenge on the second day. Following the biological challenges, participants prospectively self-monitored their panic attacks using Texas Panic Attack Record Forms. Participants were provided with specific instructions for completing the record forms to increase reliable reporting of panic episodes. Instructions stressed the importance of immediately re-

cording panic episodes. To reduce the likelihood that anxiety would be mislabeled as panic, participants were provided with a clear definition of panic that emphasized the sudden onset of symptoms. Panic attacks were classified as spontaneous when the attack occurred in the absence of any identifiable fear-provoking cue. Reported panics with fewer than four symptoms (i.e., limited symptom attacks) were excluded in the panic attack count. Completed forms were returned following a 7-day prospective self-monitoring period.

### *Hyperventilation Challenge*

During the morning of the hyperventilation challenge, participants taking benzodiazepines were asked to delay their dose until the assessment was completed. Following informed consent, participants were administered the HVC. The physiological apparatus was explained and attached. Following a 5-min resting baseline, participants completed a voluntary hyperventilation procedure in which they were instructed through cassette tape to breathe at a rate of 30 breaths/min for 2 min. The hyperventilation procedure was explained using the following instructions:

I will be taking you through a rapid breathing procedure. This procedure will involve having you breathe for a period of 2 minutes at a significantly accelerated pace, approximately three times the rate you normally breath. During this procedure, you may experience a number of physical sensations similar to those experienced during an anxiety attack.

The HVC was completed after the hyperventilation. The experimenter left the room during each of the phases when assessments were not being taken to minimize the effects of safety cues (Carter, Hollon, Carson, & Shelton, 1995). The participant was then disconnected from the equipment and debriefed. Debriefing consisted of informing the participants that the effects of the hyperventilation procedure were transient and harmless. Participants were also told to contact the research project should they experience any distress attributable to the procedure.

### *CO<sub>2</sub> Challenge*

During the morning of the CO<sub>2</sub> challenge, participants taking benzodiazepines were asked to delay their dose until the assessment was completed. After informed consent, participant's VC was measured. The experimenter provided instructions and a demonstration of the VC procedure. The participant inhaled as much air as possible, placed his or her mouth around the flow sensor to make a tight seal, and exhaled through the flow sensor. Following the VC measurements, the participant was fitted with the psychophysiological apparatus. The psychophysiological monitoring was started, and the experimenter left the room for a 5-min baseline. Participants were provided with the following instructions regarding the CO<sub>2</sub> procedure:

We are going to do a sensation induction exercise. You will be taking a single vital capacity breath of a mixture containing 35% carbon dioxide and 65% oxygen. You will need to exhale completely, and then take a full and complete inhalation using the mouth piece attached to this bag. Please hold the inhalation for five seconds. I will count to five for you and then you can exhale.

Next, the experimenter demonstrated taking a vital capacity breath from the venticomp bag and had the participant practice the same. Following the practice trial, the experimenter assisted the participant in taking a vital capacity breath of 35% CO<sub>2</sub> and 65% oxygen. The mixture was delivered to participants through 4.8 liter venticomp bags filled to capacity. With nostrils closed, the participant exhaled all of the air in his or her lungs and then inhaled from the venticomp bag through a

one-way flow valve with the goal of inhaling as much of the mixture as possible. The challenge phase consisted of the inhalation period plus 30 s following CO<sub>2</sub> inhalation. The CO<sub>2</sub> intake volume was assessed by measuring the amount of CO<sub>2</sub> remaining in the venticomp bag. The API was completed after each phase. The participant was then disconnected from the apparatus and debriefed.

## **Results**

### *Clinical Characteristics*

On average, participants reported a 10-year history ( $SD = 8.2$ ) of PD with 23% meeting *DSM-III-R* (American Psychiatric Association, 1987) criteria for at least one other anxiety disorder diagnosis and 31% meeting criteria for a mood disorder diagnosis. Total panic attack frequency was 13.5 ( $SD = 15.0$ ) for the past month and 5.2 ( $SD = 4.5$ ) for the week of prospective self-monitoring. The frequency of unexpected panic attacks was 5.8 ( $SD = 6.2$ ) for the past month and 2.0 ( $SD = 2.3$ ) for the week of prospective self-monitoring. Fifty-nine percent of the participants were taking medications for their anxiety condition. Breakdown by medication type indicated that 38% were taking only benzodiazepines, 10% were taking only antidepressants, and 11% were taking both benzodiazepines and antidepressants.

### *Subjective and Physiological Measures of the Suffocation Alarm*

The dyspnea symptom severity index was derived from two items on the SPRAS that assessed the degree to which the participant was bothered by dyspnea symptoms during the past week (i.e., difficulty in getting breath or overbreathing, smothering or choking sensation or lump in throat). Only participants reporting at least one spontaneous panic attack during the self-monitoring period were used in assessing dyspnea symptom frequency ( $n = 50$ ). The dyspnea symptom frequency measure (i.e., the percentage of spontaneous panic attacks containing dyspnea symptoms) yielded a bimodal distribution such that 25 participants (50%) reported dyspnea symptoms during every panic attack and 18 participants (36%) reported no dyspnea symptoms. One participant reported dyspnea symptoms during 92% of the spontaneous panics. The remaining 6 participants (12%) reported dyspnea during 30% to 66% of their panic attacks. Participants were classified into two extreme groups representing high or low dyspnea symptom frequency, with the 6 midrange participants being excluded to maximize group differences. End-tidal PCO<sub>2</sub> levels and resting respiration rate during the baseline period of each challenge served as the physiological indices for a heightened suffocation alarm.

### *Correlations Between Suffocation Alarm Indices and Subjective and Physiological Dependent Variables at Baseline*

Means, standard deviations, and correlations between the suffocation alarm indices and the subjective and physiological measures at baseline are presented in Table 1. None of the suffocation alarm measures were significantly correlated with

Table 1  
*Intercorrelations, Means, and Standard Deviations Among All Measures at Baseline for the Hyperventilation and the 35% CO<sub>2</sub> Challenges*

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Dyspnea symptoms														
1. Severity	—													
2. Frequency <sup>a</sup>	.46*	—												
Hyperventilation														
3. PCO <sub>2</sub>	.22	.22	—											
4. RR	.21	.14	.11	—										
5. SXS	.05	-.13	.08	.18	—									
6. SUDS	.06	.05	.13	.01	.31*	—								
7. SC	-.19	-.18	-.01	.13	.20	.14	—							
8. HR	.19	.20	.01	-.07	-.18	-.11	.12	—						
CO <sub>2</sub>														
9. PCO <sub>2</sub>	.22	-.11	.61*	.28	.17	.17	.21	-.15	—					
10. RR	.19	.07	.11	.50*	.18	.01	.13	-.07	.02	—				
11. SXS	.08	.18	-.20	-.01	.05	.20	.21	.11	.08	-.11	—			
12. SUDS	.06	.07	-.28	-.15	-.06	.14	-.23	.17	-.11	-.11	.60*	—		
13. SC	-.10	-.09	.06	.24	.05	.18	.15	-.09	.11	-.14	.22	.16	—	
14. HR	-.06	.19	-.03	.05	-.02	.06	.16	.14	.14	-.02	.02	.10	.16	—
M	36.1	18.3	2.5	55.8	3.1	12.5	4.9	74.5	35.5	15.8	3.6	16.0	5.4	77.6
SD	12.5	4.7	2.1	47.3	4.0	10.2	4.5	14.6	12.0	4.0	4.3	16.5	4.3	15.3

Note. Decimal points omitted. PCO<sub>2</sub> = average end-tidal carbon dioxide (CO<sub>2</sub>; mmHg); RR = respiration rate (breaths per minute); SXS = symptoms, range = 0–56 (hyperventilation) and 0–51 (CO<sub>2</sub>); SUDS = subjective units of distress (range = 0–100); SC = skin conductance (microsiemens); HR = heart rate (beats per minute).

<sup>a</sup>  $n = 44$  for dyspnea frequency during spontaneous panic analyses;  $n = 71$  for all other analyses.

\*  $p < .05$ .

any of the dependent variables during the baseline period of either challenge experiment ( $ps > .05$ ). The subjective suffocation alarm measures were not significantly associated with their physiological counterparts but were moderately associated with each other ( $r = .46, p < .001$ ). The physiological measures were not significantly associated with each other during the baseline periods of either the hyperventilation ( $r = .05, p > .05$ ) or the CO<sub>2</sub> ( $r = .04, p > .05$ ) challenges. Each physiological measure was moderately associated with itself during the baseline phases of the two challenges (PCO<sub>2</sub>:  $r = .61$ ; respiration rate:  $r = .50$ ).

#### *Predicting Emotional Responding to the Hyperventilation Challenge*

Regression analyses were used to examine the relationship between the suffocation alarm indices and changes in emotional responding to the hyperventilation challenge. Residualized change scores were computed for the postchallenge assessment period while controlling for baseline levels on each of the subjective (i.e., SUDS, symptoms) and physiological variables (i.e., PCO<sub>2</sub>, heart rate, skin conductance).<sup>1</sup> Dependent variables were predicted separately by each suffocation alarm index. Logistic regression was used to predict panic attacks.

As indicated in Table 2, the suffocation alarm indices largely did not predict changes in subjective or physiological responding during the challenge ( $ps > .05$ ). None of the indices significantly predicted panic to the hyperventilation challenge ( $ps >$

.05). The only significant analysis indicated that dyspnea frequency was significantly associated with changes in anxiety,  $r = -.33, t(42) = -2.38, p < .05$ . Consistent with Klein's (1993) theory, participants scoring high on dyspnea frequency exhibited lower levels of anxiety symptoms during the challenge.

#### *Predicting Emotional Responding to the 35% CO<sub>2</sub> Challenge*

The analytic strategy described for the hyperventilation challenge was also used to evaluate the relationship between the suffocation alarm indices and changes in emotional responding to the CO<sub>2</sub> challenge.

As indicated in Table 2, the pattern of findings for the CO<sub>2</sub> challenge was nearly identical to that for the hyperventilation challenge. The suffocation alarm indices largely did not predict changes in subjective or physiological responding during the challenge ( $ps > .05$ ). None of the indices significantly predicted panic ( $ps > .05$ ). Similar to the hyperventilation challenge, the only significant finding indicated that dyspnea frequency was significantly associated with changes in anxiety,  $r = -.32, t(42) = -2.23, p < .05$ . Dyspnea frequency was also marginally asso-

<sup>1</sup> Respiration rate was not predicted for the hyperventilation challenge task because respiration was a controlled part of the experimental challenge.

Table 2  
*Predicting Emotional Responding to the Hyperventilation (Hyp) and 35% CO<sub>2</sub> Challenges*

Dependent variable	PCO <sub>2</sub>		RR		Dyspnea			
	Hyp R <sup>2</sup>	CO <sub>2</sub> R <sup>2</sup>	Hyp R <sup>2</sup>	CO <sub>2</sub> R <sup>2</sup>	Severity		Frequency <sup>a</sup>	
					Hyp R <sup>2</sup>	CO <sub>2</sub> R <sup>2</sup>	Hyp R <sup>2</sup>	CO <sub>2</sub> R <sup>2</sup>
PCO <sub>2</sub>	—	—	.01	.02	.00	.00	.00	.03
RR	—	.04	—	—	—	.00	—	.04
SC	.00	.02	.03	.00	.05	.00	.00	.00
HR	.04	.00	.01	.02	.04	.00	.05	.03
Symptoms	.01	.01	.00	.02	.03	.00	.04	.06
SUDS	.01	.02	.03	.01	.01	.00	.11*	.10*
Panic	.01	.00	.03	.00	.01	.01	.00	.00

Note. Dependent variables are residual changes from baseline to postchallenge. PCO<sub>2</sub> (predictor) = average end-tidal CO<sub>2</sub> at baseline; RR = respiration rate (breaths per minute at baseline); dyspnea severity = Dyspnea Symptom Severity Index; dyspnea frequency = Dyspnea Symptom Frequency Index; SC = skin conductance; HR = heart rate; SUDS = subjective units of distress; Panic = meets criteria for panic during challenge.

<sup>a</sup> *n* = 44 for dyspnea frequency analyses; *n* = 71 for all other analyses.

\* *p* < .05.

ciated with changes in symptoms,  $r = -.24$ ,  $t(42) = -1.72$ ,  $p = .09$ . Contrary to Klein's (1993) theory, participants scoring high on dyspnea frequency showed lower levels of anxiety symptoms during the challenge.

#### *Relationship Between Medication Status and Emotional Responding to the Challenges*

Benzodiazepine use may affect emotional responding to challenge (Sanderson, Wetzler, & Anis, 1994). To determine whether medication status confounded responding on either challenge task, we examined the effects of medication status (benzodiazepines, antidepressants, both benzodiazepines and antidepressants, and no medication) on the dependent variables using separate multivariate analyses of variance (MANOVAs) for subjective (i.e., SUDS, symptoms) and physiological (i.e., respiration rate, PCO<sub>2</sub>, skin conductance, heart rate) indices and chi-square analyses for panic frequency. Medication status was not associated with subjective response, physiological response, or panic to either challenge ( $ps > .05$ ).

#### Discussion

The present study differs from previous investigations of the suffocation theory of panic in that we attempted to identify differences in PCO<sub>2</sub> hypersensitivity in patients with PD. Both subjective and physiological indices were used to operationalize the hypersensitive suffocation alarm. Dyspnea symptoms are the key subjective marker of the purported suffocation monitor (Klein, 1993). Findings from the two dyspnea symptom markers (i.e., severity and frequency) did not support Klein's theory. Neither subjective index proved to be a potent predictor of emotional responding to the challenges. Dyspnea frequency was found to significantly predict anxiety to the challenges, but the pattern of findings was inconsistent with the differential effects hypothesis as high frequency of dyspnea symptoms was associated with lower anxiety during both hyperventilation and CO<sub>2</sub> inhalation. Thus, the infrequent experience of dyspnea appears to be a general risk factor for anxious responding to respiratory

challenges. Perhaps the respiratory symptoms created by the challenges were more unique and hence less expected and more fear provoking for patients who rarely experience them.

We also operationalized the suffocation monitor using two respiration indices that are linked with Klein's (1993) theory. According to theory, PD patients with more hypersensitive alarms attempt to readjust their PCO<sub>2</sub> levels by chronically hyperventilating. Contrary to Klein's theory, respiration rate and baseline PCO<sub>2</sub> level were not associated with emotional responding to the challenges. Similar to dyspnea symptoms, PCO<sub>2</sub> level and respiration rate do not appear to adequately classify patients with respect to CO<sub>2</sub> hypersensitivity.

A number of studies have been described as being supportive of Klein's (1993) theory (Asmundson & Stein, 1994; Gorman et al., 1994; Pine et al., 1994). One problem with interpreting the findings from these studies is that factors other than a hypersensitive suffocation alarm could account for differences between PD patients and other psychiatric patients or nonclinical controls. For example, several recent studies have found that a particular type of anxiety sensitivity, fear of suffocation symptoms, may account for differential responding to challenges that create dyspnea symptoms. Taylor and Rachman (1994) developed a measure of suffocation fears and found that nonclinical participants with high suffocation fears showed greater panic (compared to low suffocation fear participants) in response to a suffocation provocation test (i.e., breathing through a narrow straw). Similarly, McNally and Eke (1996) found that both the API and a measure of suffocation fears predicted fearful responding to a CO<sub>2</sub> challenge.

Taylor and Rachman (1994) noted that a psychological factor, suffocation fears, may tap into Klein's (1993) purported biological suffocation alarm. However, there are several advantages to using measures of the suffocation monitor that do not directly assess fear of fear. First, it is not clear that Klein would agree that dyspnea fears correspond with the hypersensitive suffocation monitor. Klein stated that a faulty suffocation monitor is indicated by the presence of dyspnea, but he made no comment about the necessity of the fear of dyspnea symptoms.

Second, only by using psychological indices that are relatively orthogonal to fear of fear can we tease apart the anxiety sensitivity contribution to the findings. The confounding influence of anxiety sensitivity or fears of specific sensations created by biological challenges led us to create indices that assessed the presence of dyspnea symptoms rather than the fear of such symptoms.

A critical assumption of this study is that the faulty suffocation monitor can be operationalized according to the presence of dyspnea symptoms. The decision to use dyspnea symptoms as an index of the suffocation alarm is based on Klein's (1993) argument that dyspnea is relatively unique to spontaneous panic because of its connection to the faulty suffocation monitor. A recent study by McNally, Hornig, and Donnell (1995) indicated that cognitive symptoms, such as fear of dying or loss of control, were the most highly associated with a clinical panic attack. However, dyspnea was the physiological symptom that best discriminated clinical from nonclinical panic attacks. Regardless of whether dyspnea symptoms are unique to spontaneous panic attacks in patients with PD, our data suggest that classification on the basis of reports of dyspnea symptoms during panic does not provide an adequate measure of CO<sub>2</sub> hypersensitivity.

Our operationalization of the hypersensitive suffocation monitor assumes there are differences in CO<sub>2</sub> sensitivity among patients with PD. This assumption is supported both theoretically and empirically. In terms of theory, Klein (1993) clearly stated that hypersensitivity to CO<sub>2</sub> varies over time: "Panic disorder patients have good and bad periods. Presumably during bad spells the suffocation alarm is pathologically depressed" (p. 309). In addition, some patients with PD, in particular those who do not report dyspnea during panic, are not believed to possess a pathologically lowered CO<sub>2</sub> threshold (D. F. Klein, personal communication, November 16, 1994). Klein (1993) suggested that the panic attacks experienced by these patients are due to hypothalamic-pituitary-adrenal axis-autonomic system activation. The biological challenge literature attests to differences in CO<sub>2</sub> sensitivity, as some PD patients panic in response to low CO<sub>2</sub> doses whereas others panic only when given higher doses (Gorman et al., 1994; Hollander et al., 1989; Levin et al., 1987; Woods et al., 1988).

There are a variety of alternative subjective and physiological indices for the assessment or operationalization of the hypersensitive alarm. In terms of subjective indices, a more detailed assessment of self-reported respiratory symptoms (e.g., breathlessness, frequency of sighing-yawning, feeling that breathing will stop, smothering) may reveal a particular subjective symptom or constellation of symptoms that are associated with CO<sub>2</sub> hypersensitivity. In regard to physiological indices, minute ventilation, arterial PCO<sub>2</sub>, and blood pH offer additional respiratory estimates not assessed in the present study. Finally, studies of CCHS may advance our knowledge sufficiently such that a central nervous system locus could be identified and evaluated further.

Approximately 40-45% of the participants experienced a panic attack during the 35% CO<sub>2</sub> challenge task. Panic was assessed using fairly conservative criteria that required the subjective report of panic, a 30-point rise in SUDS ratings of anxiety from baseline, and the presence of four or more *DSM-III-R*

(American Psychiatric Association, 1987) panic attack symptoms. These criteria were used to capture the *DSM-IV* (American Psychiatric Association, 1994) requirements of intense fear plus the sudden onset of symptoms. This conservative index is likely to account for the lower panic frequency in the present report compared with other reports assessing the inhalation of 35% CO<sub>2</sub> (Fyer et al., 1987) or even lower concentrations of CO<sub>2</sub> (Gorman et al., 1994).

The present study indicates that CO<sub>2</sub> is significantly panicogenic in a substantial proportion of patients with PD. Yet, the fact that a majority of participants did not panic during the challenge raises a question about the suffocation alarm threshold. Consider that the 35% CO<sub>2</sub> concentration is 875 times greater than inspired dry room air (Mines, 1992). Even alternative CO<sub>2</sub> challenge concentrations (5-7% CO<sub>2</sub>) are 125-175 times greater than dry room air. It would be expected that these seemingly potent doses will cross even the least sensitive threshold, thus triggering panic in 100% of the PD patients. Taking this a step further, these potent doses should cross even the normal threshold of controls, that is, an adaptive suffocation monitor should be triggered during conditions where PCO<sub>2</sub> levels are significantly elevated. However, controls rarely panic when given 5%, 7%, or even 35% CO<sub>2</sub> gas (Gorman et al., 1994; van den Hout, van der Molen, Griez, & Lousberg, 1987).

Data from the present study do not support a PCO<sub>2</sub> threshold model of suffocation. Perhaps the suffocation alarm is defined by a more complex biological monitoring system rather than Klein's (1993) proposed unidimensional system defined solely by PCO<sub>2</sub> levels. After all, the respiratory drive is regulated by medullary and peripheral chemoreceptors as well as mechanoreceptors such as the pulmonary stretch receptors, pulmonary irritant receptors, and type J receptors (Mines, 1992). The suffocation alarm may require an appropriate pattern of signals based on various receptors rather than a single necessary and sufficient signal from the CO<sub>2</sub> receptors. For example, a single vital capacity breath of 35% CO<sub>2</sub>/65% O<sub>2</sub> signals "suffocation" in terms of CO<sub>2</sub> but "no suffocation" in terms of O<sub>2</sub> receptors. Rather than suffering from a hypersensitivity in one receptor, patients with PD may experience faulty integration of data from various receptors that spuriously signals a suffocation alarm. It is recommended that multidimensional biological models be considered in order to better describe a faulty suffocation alarm system in PD.

Dyspnea symptoms are a salient feature of clinical panic attacks (McNally et al., 1995). Suffocation symptoms are also prominent in challenge-induced panic (Zandbergen, Pols, Fernandez, & Griez, 1991). Although this evidence is consistent with the suffocation theory, it can also be interpreted in light of cognitive theories of panic (Clark, 1986). Clark's theory asserts that dyspnea symptoms trigger panic when they are catastrophically misinterpreted. Findings from recent studies inspired by the suffocation alarm theory (Asmundson & Stein, 1994; Gorman et al., 1994; Pine et al., 1994; Zandbergen et al., 1991) do not take into account cognitive explanations of panic. These studies do not address the possibility that cognitions mediate the relationship between symptoms and panic. In fact, there is a growing literature to suggest that cognitive factors, such as predictability, perceived control, and perceived safety, influence anxious responding to CO<sub>2</sub> in both clinical (Carter et al., 1995;



Rapee, Mattick, & Murrell, 1986; Sanderson, Rapee, & Barlow, 1989) and nonclinical (Telch & Harrington, 1993; Van den Bergh, Van den Driessche, de Broeck, & Van de Woestijne, 1993) populations. Future work addressing the suffocation alarm theory should account for the potential influences of these psychological parameters.

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