Respiratory Muscle Tension as Symptom Generator in Individuals With High Anxiety Sensitivity

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Objective: Anxiety and panic are associated with the experience of a range of bodily symptoms, in particular unpleasant breathing sensations (dyspnea). Respiratory theories of panic disorder have focused on disturbances in blood gas regulation, but respiratory muscle tension as a source of dyspnea has not been considered. We therefore examined the potential of intercostal muscle tension to elicit dyspnea in individuals with high anxiety sensitivity, a risk factor for developing panic disorder. Methods: Individuals high and low in anxiety sensitivity (total N = 62) completed four tasks: electromyogram biofeedback for tensing intercostal muscle, electromyogram biofeedback for tensing leg muscles, paced breathing at three different speeds, and a fine motor task. Global dyspnea, individual respiratory sensations, nonrespiratory sensations, and discomfort were assessed after each task, whereas respiratory pattern (respiratory inductance plethysmography) and end-tidal carbon dioxide (capnography) were measured continuously. Results: In individuals with high compared to low anxiety sensitivity, intercostal muscle tension elicited a particularly strong report of obstruction (M = 5.1, SD = 3.6 versus M = 2.5, SD = 3.0), air hunger (M = 1.9, SD = 2.1 versus M = 0.4, SD = 0.8), hyperventilation symptoms (M = 0.6, SD = 0.6 versus M = 0.1, SD = 0.1), and discomfort (M = 5.1, SD = 3.2 versus M = 2.2, SD = 2.1) (all p values <.05). This effect was not explained by site-unspecific muscle tension, voluntary manipulation of respiration, or sustained task-related attention. Nonrespiratory control sensations were not significantly affected by tasks (F < 1), and respiratory variables did not reflect any specific responding of high–Anxiety Sensitivity Index participants to intercostal muscle tension. Conclusions: Respiratory muscle tension may contribute to the respiratory sensations experienced by panic-prone individuals. Theories and treatments for panic disorder should consider this potential source of symptoms. Key words: respiration, respiratory muscle tension, anxiety, panic, respiratory sensation.

INTRODUCTION

Individuals with anxiety disorders or intense states of anxiety, particularly panic, often report experiencing dyspnea, which includes unpleasant respiratory symptoms such as feelings of suffocation, chest tightness, and breathlessness (1–5). Research on respiratory processes in patients with anxiety has largely focused on hyperventilation, with hypopnea being viewed as a major source of respiratory sensations (6,7). However, in addition to changes in carbon dioxide (CO2) levels, other physiological processes can also lead to dyspnea (8). Respiratory muscle tension is a source of proprioceptive input contributing to the experience of breathlessness. Feedback from respiratory muscles seems to be essential in explaining basic phenomena such as voluntary breath-hold duration (9). It has also been suggested that respiratory muscles provide an alternative pathway for symptom generation in patients with hyperventilation symptoms (10). Elevated activity of respiratory muscles has been observed during experimental anxiety provocation with CO2 inhalation in highly anxious patients with panic disorder (11). However, a paradigm that directly manipulates tension levels is needed to experimentally study the effects of respiratory muscle tension on symptoms perception.

In a pilot study with healthy individuals, we used electromyogram (EMG) biofeedback targeting the tension levels of intercostal muscles (12). Our findings showed substantial increases in dyspnea during periods of tension. However, we did not study the effects of this task on more anxiety-prone individuals. In addition, because the study lacked a comparison muscle site, it is possible that any type of muscle tension could elicit similar levels of dyspnea. Also, our previous study did not evaluate whether elevated dyspnea was simply caused by sustained attention to a demanding task independent of the actual muscle tension or caused by nonspecific voluntary manipulation of breathing. We also did not determine whether dyspnea report was indicative of respiratory processes or just an instance of a general elevation of physical symptom report.

Therefore, in the present study, we sought to evaluate the specificity of respiratory muscle tension in eliciting dyspnea and whether this depends on anxiety sensitivity, which is associated with a fear of bodily symptoms. Anxiety sensitivity has been established as a risk factor for panic disorder (13). We sought to establish the specificity of dyspnea elicitation with regard to a) a tendency to respond fearful to bodily sensations, as measured by the Anxiety Sensitivity Index (ASI; 14), b) the intercostal muscle site rather than any type of skeletal muscle tension, c) task-related sustained attention, or d) voluntary manipulation of breathing pattern, as well as specificity with regard to e) respiratory symptom report rather than symptoms unrelated to respiration. We expected respiratory muscle tension to induce greater dyspnea than an equivalent task increasing leg muscle tension, a fine motor task that requires sustained attention, and a task that requires voluntary changes in breathing pattern, such as paced breathing. In addition, the primary symptoms elicited by this task should be respiratory in nature, and individuals with higher levels of anxiety sensitivity were expected to show higher levels of dyspnea specifically during the respiratory muscle tension task. As an improvement on our earlier pilot research, we also added a detailed analysis of the specific quality of respiratory sensation elicited by respiratory muscle tension and the specific breathing pattern associated with it. Ultimately, our findings

ANOV A = analysis of variance; ASI = Anxiety Sensitivity Index; EMG = electromyogram; PCO2 = partial pressure of carbon dioxide.
METHODS
Participants
Sixty-two undergraduate students were preselected based on their ASI scores being high (M ≥ 20) or low (M < 20). Equal proportions of women and men were targeted for high-ASI and low-ASI groups. Participants had expressed interest in research participation in an initial screening questionnaire that included ASI. They provided informed consent and received course credit for participation. The study was approved by the local institutional review board. Data collection took place between January and December 2007.

Instruments and Measures
EMGs were measured with the Biopac System (Biopac Systems Inc., Goleta, CA), using surface electrodes (inner diameter, 18 mm) with a bipolar placement on the left external intercostal muscle of the second and third intercostal spaces (12,15). Leg muscle EMG was measured simultaneously with a bipolar placement of electrodes on the left calf (approximate left gastrocnemius placement; 16). To reduce cross-talk from electrocardiogram, we applied a 500-Hz low-pass filter and a 30-Hz high-pass filter to the signal. Raw signals were rectified, integrated into a 1-second time window, and averaged. During the biofeedback tasks, participants were provided a visual feedback of their raw EMG signals on a computer screen and asked to increase the overall amplitude of the signal (chest or leg, depending on the task). No further instructions were provided.

Respiratory pattern was recorded with an ambulatory biosignal recorder (LifeShirt; Vivometrics, Ventura, CA) that featured a lightweight vest with integrated inductance bands around the thorax and abdomen. Signals were amplified and digitized at 50 Hz, and data were stored in a portable microrecorder. Expansion of the inductance bands was calibrated for volume using a fixed-volume (800 mL) plastic bag that the participant inflated and deflated completely for seven consecutive breaths. The following respiratory parameters were extracted using Vivologic software for biosignal analysis (Vivometrics): respiration rate $R$, tidal volume $V_T$, mean inspiratory flow $V_{T/I}$, and minute ventilation $V_{E}$. Because elevated intercostal muscle tension typically leads to increases in functional residual capacity (dynamic hyperinflation; 17), we also evaluated end-expiratory volume level changes from the tracings of the $V_{E}$ curve. Because a quantitative measure of end-expiratory level changes from respiratory inductance plethysmography has not been validated in prior research, we restricted our analyses to a blind rating of observable increases versus no increases in end-expiratory levels across all sequences of the chest and leg muscle tension tasks by two independent raters. Interrater reliability was high ($\kappa = 0.86$ for task periods, $\kappa = 0.81$ for rest periods).

End-tidal partial pressure of CO$_2$ (PCO$_2$) was monitored with a portable, battery-operated capnometry device (Tidal Wave Sp, Respionics, Murrysville, PA) that samples exhaled gas through a nasal cannula.

Psychological Measures
ASI (13) measures fear of symptoms linked to anxiety and the potential undesirable effects of these symptoms. This 16-item scale has a five-point response format ranging from 0 = very little to 4 = very much. The measure has shown good internal consistency, test-retest reliability, and criterion validity. Dyspnea experienced during the task was rated on the Borg scale (18), with anchors 0 = none, 4 = slight, 9 = moderate, 15 = severe, and 19 = extreme. Participants rated their experienced breathlessness after each trial of a task.

Hyperventilation Symptoms
After each task, nine hyperventilation items from the inventory of respiratory symptoms devised by Kinsman et al. (19) were rated on 11-point scales (0 = not at all to 10 = very much).

Additional descriptors of respiratory sensation (adapted from Simon et al. (20)) were rated on 11-point scales after each task. Because no consistent clustering of various respiratory sensations has been established across studies to date (19,21–24), we focused on three item groups that showed the greatest face validity for distinguishing qualitatively different manipulations of breathing and seemed to differentiate specific aspects of cardiorespiratory pathophysiology (25–27): obstruction items (“My chest feels constricted,” “My chest feels tight”) typical of airway obstruction seen in asthma; work or effort of breathing questions typical of respiratory muscle weakness or increased mechanical load as seen in chronic obstructive pulmonary disease (“My breathing requires more effort,” “My breathing requires more work”); and air hunger (“I feel a hunger for more air,” “I feel that I am suffocating,” “I feel that I am smothering”) typical of hypercapnia and manipulations of respiratory gas exchange.

Task Evaluation
Participants evaluated the tasks according to various criteria on additional on 11-point scales. From these ratings, we extracted the item “I felt uncomfortable throughout the task,” which came closest to capturing the possible negative affect elicited by the tasks.

Protocol and Procedure
Participants were scheduled for individual laboratory sessions. EMG electrodes were attached, and the LifeShirt equipment was fitted and calibrated. Participants then performed four tasks: biofeedback of intercostal muscles, biofeedback of leg muscles, fine motor task, and paced breathing at three different speeds (9, 13, and 18 breaths/min). A 5-minute quiet sitting baseline was also administered. Task and baseline conditions were presented randomly across participants. The following standard instructions were provided to participants:

Biofeedback tasks: “Please try to modify the biofeedback signal in its intensity. Try to increase the signal and continue to let the signal remain intense until directed to stop.”

Paced breathing: “You will hear tones with a rising and falling pitch. We would like you to breathe in with the rising pitch and breathe out with the falling pitch and pause between breaths when there is no tone. During the first 2 minutes, the tones will be rather slow, then the tones will switch to a medium speed, and, finally, to a fast speed. There will be 1-minute rest periods between slow and normal breathing tones, and between normal and fast breathing tones. Make sure to always breathe through your nose with your mouth closed.”

Fine motor task: “You will be given four small hand-held toys, where your goal is to get the small metal ball into the innermost circle by following the maze. Remain seated, and make sure that you do not move your entire hand, but rather just tap with your fingers. If you complete the task within the given time, please do it one more time. There will be a 1-minute rest period between each new toy.”

Biofeedback tasks and fine motor task were presented four times for 60 seconds, with a 60-second break in-between each presentation. Breaks of 60 seconds were also included between the three different speeds of paced breathing. After each 60-second episode, participants rated the degree of dyspnea felt during the task on the Borg scale. Variable breaks between tasks and conditions were offered at a minimum of 5 minutes, at which time participants rated the descriptors of respiratory and nonrespiratory sensations and their discomfort during the task. After all assessments, the participants were fully debriefed.

Data Reduction and Analysis
Measurements of EMG and aspects of the respiratory pattern were averaged across baseline, each of the three paced breathing sequences, and each of the 1-minute task and rest periods of the two muscle tension tasks and the fine motor task. Integrated EMG data (in μV * s) were log-transformed to improve distributional characteristics. We used three-way repeated-measures analyses of variance (ANOVA), with group (high versus low ASI), four tasks (chest tension,
leg tension, fine motor task, paced breathing), and period (task, rest) as independent variables, and with ratings of dyspnea and physiological measures (chest and leg EMGs, respiratory parameters) as dependent variables. Because symptoms of hyperventilation, obstruction, air hunger and effort, as well as nonrespiratory symptoms, were only rated after each task block, they were analyzed by two-way repeated-measures ANOVA, with group (high versus low ASI) and four tasks (chest tension, leg tension, fine motor task, paced breathing) as independent variables. We expected respiratory sensations, particularly dyspnea and obstruction, but not nonrespiratory sensations, to be more pronounced during the chest muscle tension tasks than during other tasks; compared with the leg muscle tension task, this would indicate that these respiratory sensations are specific for the chest muscle site; compared with paced breathing, this would indicate that they are not nonspecific consequences of respiratory manipulation; and compared with the fine motor task, this would show that they are not nonspecific consequences of sustained attention. We also expected significant interactions between tasks and group, in that the specificity of the chest muscle tension task would be most pronounced in high-ASI individuals.

We had no specific expectations for respiratory variables, except for stronger evidence for dynamic hyperinflation in the blind ratings of end-expiratory volume levels from the respiratory impedance plethysmography device. Differences between means were evaluated with the Newman-Keuls procedure. Greenhouse-Geisser correction of df was performed where appropriate, with the original df, correction factor ε, and corrected p levels reported. Partial η² (η²p) was used as a measure of effect size. Associations between ASI and changes in symptoms were also studied within and across ASI subgroups. Because ASI was not normally distributed, Spearman rank correlation was used. Equipment problems led to partial or full loss of chest EMG in 8 participants (4 high ASI, 3 low ASI), thus reducing original df.

**RESULTS**

**Basic Sample Characteristics**

Selection of participants resulted in two groups that differed significantly in ASI scores (Table 1). They were largely equivalent in demographic characteristics and physiological baseline values.

| TABLE 1. Demographic Characteristics of High-ASI and Low-ASI Subgroups |
|---------------------------------------------------------------|
| **High ASI (n = 31)** | **Low ASI (n = 31)** | **p** |
|-------------------|-------------------|-------|
| **Basic characteristics** | | |
| Female, n (% of total) | 15 (48.3) | 14 (45.1) | .799 |
| Age, y | 20.3 (2.7) | 21.77 (5.2) | .171 |
| Body mass index, kg/m² | 24.3 (5.3) | 24.7 (5.0) | .462 |
| Current smokers | 5 (16.1) | 1 (3.2) | .09 |
| **Psychological variables** | | |
| ASI, possible score range 0–64 | 29.7 (7.4) | 11.3 (3.9) | <.001 |
| **Physiological variables** | | |
| EMG, μV V⁻¹s | | |
| Chest muscle site | 1.21 (0.54) | 1.14 (0.75) | .705 |
| Leg muscle site | 1.84 (0.53) | 1.54 (0.61) | .329 |
| Respiration rate fR, breaths/min | 16.0 (4.2) | 15.9 (2.5) | .924 |
| End-tidal PCO₂, mm Hg | 36.3 (4.8) | 36.0 (3.6) | .788 |
| Tidal volume Vₜ, ml | 399.5 (163.8) | 343.9 (128.6) | .157 |
| Mean inspiratory flow Vᵢ/TVᵢ, ml/s | 257.2 (116.2) | 220.8 (82.1) | .175 |
| Minute ventilation Vᵢ', L | 6.4 (3.3) | 5.4 (2.0) | .172 |

ASI = Anxiety Sensitivity Index; EMG = electromyogram; PCO₂ = partial pressure of carbon dioxide. Data are presented as mean (SD) or frequency (%). P values are for t test or χ² test.
indicating that low and high groups differed for specific tasks and tension versus rest periods. Overall, high-ASI participants reported higher dyspnea levels \( \text{[group effect } F(1,60) = 14.20, p < .001, \eta^2_p = 0.191\]}. Hyperventilation Symptoms, Air Hunger, Obstruction, and Effort The two-way repeated-measures ANOVA for hyperventilation symptoms yielded a Task × ASI Group interaction \( [F(3,171) = 5.60, p = .003, \epsilon = 0.77, \eta^2_p = .0089]\). For low-ASI participants, symptoms were significantly higher for paced breathing than for chest muscle tension, whereas high-ASI participants reported equally high hyperventilation symptoms for both the chest tension task and the paced breathing task, which were higher than those for other tasks (Fig. 3A). A similar pattern of findings was observed for air hunger symptoms \( [\text{Task } \times \text{ ASI Group interaction: } F(3,174) = 6.02, p = .002, \epsilon = 0.81, \eta^2_p = 0.094]\) (Fig. 3B), but tasks were not differentiated significantly in low-ASI participants. There was also a significant Task × ASI Group interaction for obstruction symptoms \( [F(3,177) = 2.93, p = .05, \epsilon = 0.80, \eta^2_p = 0.047]\), with high-ASI participants showing significantly higher values for the chest tension task compared with all other tasks, as did high-ASI participants, but the distinction between chest tension and paced breathing was less pronounced \( (p < .05 \text{ level})\). Overall, obstruction symptoms were much more pronounced than suffocation symptoms across tasks (Fig. 3C). Effort symptoms only showed a main effect of task \( [F(3,177) = 65.9, p < .001, \epsilon = 0.80, \eta^2_p = 0.528]\), with paced breathing rated highest \( (M = 4.7, \text{SD} = 2.9)\), followed by chest muscle tension \( (M = 4.0, \text{SD} = 2.7)\), leg muscle tension \( (M = 1.6, \text{SD} = 2.0)\), and fine motor task \( (M = 0.8, \text{SD} = 1.6)\). Across tasks, high-ASI participants reported higher symptom levels for hyperventilation \( [F(1,57) = 11.44, p < .001, \eta^2_p = 0.167]\), obstruction \( [F(1,59) = 17.24, p < .001, \eta^2_p = 0.226]\), suffocation \( [F(1,58) = 14.52, p < .001, \eta^2_p = 0.200]\), and effort \( [F(1,59) = 9.53, p = .003, \eta^2_p = 0.139]\).
Nonrespiratory Symptoms

No significant effects of task or Task × Group interaction \( (Fs < 1, p > .409) \) were observed for nonrespiratory symptoms (Fig. 3D). There was a trend for highASI participants in that they reported more nonrespiratory symptoms across tasks \( \text{F}(1,54) = 3.34, p = .07, \eta_p^2 = 0.058 \). When controlling for baseline levels of nonrespiratory symptoms in analyses of covariance for ratings of respiratory sensations, results remained essentially the same for hyperventilation and air hunger, with the critical Task × ASI Group interactions remaining significant \( (p = .02) \). For obstruction, the Task × ASI Group interactions were reduced to a trend \( (p = .07) \). For the dyspnea ratings, the three-way interaction became nonsignificant \( (p = .146) \), whereas this interaction was only marginally significant for \( \text{R} \) \( (F(3,168) = 3.34, p = .051, \eta_p^2 = 0.056) \). Post hoc

Association of ASI With Symptom Report During Tasks

ASI was significantly and positively associated with changes in at least one of the respiratory symptoms, but not with changes in nonrespiratory symptoms, during the four tasks (Table 2). No significant associations were observed within subgroups of lowASI and highASI participants.

Respiratory Parameters

Three-way repeated-measures ANOVAs showed significant Task × Period interaction effects for \( V_T \) \( \text{[F(3,162) = 59.4, p < .001,} \), \( \eta_p^2 = 0.56, \eta_p^2 = 0.524], \( V_T/T_I \) \( \text{[F(3,168) = 14.48, p < .001,} \), \( \eta_p^2 = 0.61, \eta_p^2 = 0.205], \( V_E \) \( \text{[F(3,162) = 17.38, p < .001,} \), \( \eta_p^2 = 0.51, \eta_p^2 = 0.244], \text{and PCO}_2 \) \( \text{[F(3,135) = 7.71, p = .003,} \), \( \eta_p^2 = 0.48, \eta_p^2 = 0.146], \text{whereas this interaction was only marginally significant for } \text{R} \) \( \text{[F(3,168) = 3.34, p = .051,} \), \( \eta_p^2 = 0.52, \eta_p^2 = 0.056] \). Post hoc

| TABLE 2. Spearman Rank Correlations Between ASI Scores and Change in Symptoms From Baseline During Tasks for the Total Sample \( (N=62) \) |
|-------------------------------------------|-------|-------|-------|-------|
|                                          | Chest Muscle Tension | Leg Muscle Tension | Paced Breathing | Fine Motor Task |
| Dyspnea \( \Delta \)                      | 0.25\( ^{\dagger} \) | 0.40*** | 0.14 | 0.30* |
| Hyperventilation symptoms                 | 0.29* | 0.16 | 0.20 | -0.28* |
| Air hunger                                | 0.24\( ^{\dagger} \) | 0.22\( ^{\dagger} \) | 0.37** | -0.10 |
| Obstruction                               | 0.22\( ^{\dagger} \) | 0.25\( ^{\dagger} \) | 0.30* | -0.12 |
| Effort                                    | 0.02 | 0.25* | 0.09 | -0.12 |
| Nonrespiratory symptoms                  | -0.02 | 0.09 | 0.04 | 0.11 |

ASI = Anxiety Sensitivity Index. All tests are two-tailed.
\( ^{\dagger} \) \( p < .10. \)
\( * p < .05. \)
\( ** p < .01. \)
\( *** p < .001. \)
\( \Delta \) Difference score relative to average rest periods between task episodes.
TABLE 3. Respiratory Parameters Across Tasks and Associated Rest Periods

| Chest Muscle Tension | Leg Muscle Tension | Paced Breathing | Fine Motor Task | Post Hoc Means Differences (p < .05) |
|----------------------|-------------------|-----------------|-----------------|-------------------------------------|
| Task                 | Rest              | Task            | Rest            | Task Versus Task                     |
| Tidal volume V₉, mL  | 629.7 (368.7)     | 508.4 (302.9)   | 408.9 (183.5)   | 436.0 (181.5)                       |
| Minute ventilation  Vₑ, mL | 423.4 (193.0)     | 382.6 (171.9)   | 285.4 (149.9)   | 297.2 (134.3)                       |
| Mean inspiratory flow V₉/I, mL/s | 19.3 (8.3)         | 16.4 (5.1)      | 16.8 (6.7)      | 15.3 (3.4)                          |
| Respiratory rate fᵣ, breaths/min | 16.5 (9.9)        | 16.4 (3.4)      | 16.8 (7.7)      | 15.3 (3.4)                          |
| End-tidal PCO₂, mm Hg | 35.3 (5.4)        | 36.5 (7.7)      | 36.9 (9.7)      | 36.6 (4.5)                          |

C = chest muscle tension; P = paced breathing; F = fine motor task; L = leg muscle tension; PCO₂ = partial pressure of carbon dioxide.

Data are presented as mean (SD). Post hoc differences indicate changes in the respective respiratory parameter attributable to the task challenge. All differences are independent of ASI status (high versus low).

Visual inspection of the recordings for dynamic hyperinflation was only performed for both muscle tension tasks. An increase in functional residual capacity was seen in 74.1% of participants in at least three of four trials of the chest muscle tension task, and 89.7% of participants showed increases in at least two of the four trials. Changes in end-expiratory levels during the leg muscle tasks were observed only in four participants. There were no significant differences between high-ASI and low-ASI groups in the occurrence of hyperinflation during the chest muscle tension task (χ² = 1.45, df = 1, p = .227). Figure 4 illustrates the changes in end-expiratory volume level (functional residual capacity) in one participant during chest muscle tension (top) and leg muscle biofeedback (bottom).

### Task Evaluation

Ratings for feeling uncomfortable for the four tasks yielded a significant effect of task [F(3,180) = 12.34, p < .001, ηp² = 0.171] and a trend for Task × Group interaction [F(3,180) = 2.41, p = .08, ηp² = 0.039] (Fig. 5). High-ASI participants reported feeling most uncomfortable during the chest tension task, which was rated significantly more uncomfortable than the leg tension and fine motor tasks and, in tendency (p < .080), more uncomfortable than paced breathing. For low-ASI participants, tasks were not differentiated. There was a significant group effect for feeling uncomfortable [F(1,60) = 11.91, p < .001, ηp² = 0.039] such that high-ASI participants rated the tasks, on average, more uncomfortable than low-ASI participants. However, this was mostly attributable to high-ASI participants rating chest tension substantially higher than low-ASI participants (M = 5.1, SD = 3.2 versus M = 2.2, SD = 2.1, respectively) on the 0 to 10 scale of feeling uncomfortable. Other tasks were not significantly different between groups; only paced breathing showed a trend toward higher values for high-ASI participants (p < .10).

### DISCUSSION

In this study, we found that individuals who are characterized by high anxiety sensitivity, a risk factor for panic disorder, are specifically sensitive to respiratory muscle tension, which is one pathway that contributes to sensations of dyspnea (8). By manipulating the levels of the intercostal muscle EMG, we were able to experimentally test the effects of respiratory muscle tension as a pathway of symptom production. Confirming our earlier research (12), we found that dyspnea substantially increased during this task. In this study, we were also able to rule out a number of alternative explanations for elevated dyspnea and showed that this effect was specific for the chest.

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**Notes:**
- Post hoc differences indicate differences in task levels for the respective respiratory parameter. All differences are independent of ASI status (high versus low).
- Post hoc differences indicate changes in the respective respiratory parameter attributable to the task challenge. All differences are independent of ASI status (high versus low).
muscle site. Leg muscle tension did not elicit the same level of uncomfortable breathing sensation, nor did a fine motor task that required sustained attention. Paced breathing—a voluntary manipulation of breathing pattern by following pacing tones that induces a dynamic activation of respiratory muscles in phase with the respiratory cycle, rather than in static tension throughout it—did elicit respiratory symptoms; however, for high-ASI individuals, the level of symptoms was not as high in the paced breathing task as in the chest muscle tension task. In addition, the quality of the sensations elicited was clearly associated with respiration, with sensations of obstruction (airway constriction, chest tightness) particularly standing out. The specificity of symptom generation in high-ASI participants during chest muscle tension was not explained by changes in key respiratory parameters that have been linked to dyspnea, including CO₂ levels (27–30). Interestingly, high-ASI participants also reported elevated symptoms classically associated with hyperventilation and air hunger during chest muscle tension, but only during paced breathing did PCO₂ levels fall into the hypocapnic range. Low-ASI participants reported symptoms selectively after this task, whereas for high-ASI participants, chest muscle tension served almost like an attractor of respiratory symptoms, including additional hypocapnia-related sensations. Although ASI showed a number of positive correlations with respiratory symptoms across all four tasks, the levels of respiratory symptoms were particularly high for high-ASI participants during chest muscle tension. This may suggest that at least some of the dyspnea observed in panic-prone individuals might also be a consequence of respiratory muscle tension.

Shortness of breath, chest tightness, and suffocation are among the standard symptoms that are part of the diagnostic criteria for panic disorder, with shortness of breath being one of the most intense symptoms. Research on the mechanisms of respiratory symptom generation in panic disorder has exclusively focused on dysfunctional blood gas regulation (29,30), as has research questioning the key role of respiration in panic (31,32). Our findings suggest that PCO₂ changes are not necessary to produce intense feelings of dyspnea in individuals at risk for panic disorder—it is by no means the only pathway through which respiratory sensations are generated, as has been known in respiratory physiology for some time (8,33).1 Particularly intriguing in this context is that even symptoms classically associated with hypocapnia were generated by respiratory muscle tension in high-ASI individuals, but not in low-ASI individuals. One possible link between anxiety and elevated levels of respiratory muscle tension could exist through sympathetic

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1Interestingly, the plurality of pathways for dyspnea generation has already been reflected upon in the early 1960s. As Campbell and Howell (34) put it in a caricatural note: “A respiratory physiologist offering a unitary explanation for breathlessness should arouse the same suspicion as a tattooed archbishop offering a free ticket to Heaven” (see also Fried (35)). This contrasts with the more exclusive focus of later panic disorder literature on gas exchange abnormalities as causes of respiratory symptoms (29–32).
effects on muscle spindles, which dampen stretch reflex activity and thereby provide a potential mechanism to adjust motor activity in situations of high physiological and/or psychological demand (36).

Although we were able to produce substantial dyspnea and discomfort in high-ASI participants during respiratory muscle tension, the task was not panicogenic in that we did not observe any full-blown panic attacks from it. In that respect, the task is different from other challenges, such as voluntary hyperventilation or CO₂ inhalation (37–41), which have been shown to elicit panic attacks in high-ASI (and thus panic-prone) individuals (13,42). It could therefore be questioned whether this type of respiratory task has any clinical significance for panic disorder. One factor that may have reduced the potency of the task to produce extreme states of fear or panic is perceived control (43). By voluntary tensing their skeletal muscles with the help of the biofeedback signal and by experiencing immediate relief after the offset of the self-produced tension, participants may have felt very much in control of this pathway of dyspnea production and thus had a convincing and rational explanation for their symptoms, thereby reducing the necessity to attribute the symptoms to an emotional state (44). Alterations in blood gas tension, as in hypercapnia or hypocapnia, produce symptoms with much slower time constants, thus leaving the individual potentially with an experience of a diminished level of personal control. In that respect, the EMG biofeedback paradigm probably does not fully capture the experiential quality of respiratory muscle tension in real life, in which individuals may not be aware of contractions in their respiratory muscle apparatus. Alternative paradigms, such as chest wall vibration to stimulate muscle spindles of the intercostal muscles (45), may eventually provide a more convincing stimulation paradigm. Even if the respiratory muscle tension pathway proves to be only a modulating pathway of dyspnea (8), the extent of its potential contribution to symptoms may greatly attenuate the associations between CO₂ and symptoms that are classically studied in the respiratory psychophysiology of panic disorder (6,46). It could also be speculated whether the combination of respiratory muscle tension with hypocapnia induction could produce a particularly potent stimulus in panic-prone individuals. In any case, further elucidation of the respiratory muscle tension effects on dyspnea in panic disorder could contribute to a more complete understanding of respiratory involvement in panic attacks and potential subtypes of panic disorder linked to respiratory dysregulation (47,48).

Our study was limited in that anxiety sensitivity served as a surrogate for studying individuals with a diagnosis of panic disorder. Having established the importance of this stimulus for dyspnea production in individuals with a heightened fear of bodily symptoms, we believe that the logical next step would be the study of a clearly defined patient group, such as individuals with panic disorder. Our sample was also limited in that we only recruited from an undergraduate student population. Smoking showed a trend toward higher prevalence in the high-ASI group, reflecting prior literature on smoking, anxiety, and panic (49,50), but the overall number of smokers was too small and precluded a meaningful analysis of potential effects on dyspnea perception. With regard to our measures of respiratory muscle tension, as with any surface electrode placement, we did not measure the activity of individual muscles but more nonspecific activity at a particular muscle site (51). Thus, we cannot exclude cross-talk from other muscles in the chest area. Although this reduces the specificity of a mechanistic explanation for dyspnea, it may actually be a more ecologically valid scenario for capturing the influences of the accessory muscles of respiration on symptoms. The intercostal muscle biofeedback procedure also resulted in a broader integrated change in breathing pattern, which included elevations in end-expiratory volume levels (dynamic hyperinflation) that are known to induce dyspnea and fatigue in chronic respiratory disease (52). Participants’ qualitative reports of our pilot study also suggested that more than one respiratory muscle site was activated by the biofeedback task, with some participants describing in the debriefing session maneuvers such as tension of chest muscles and/or the shoulders or abdomen to manipulate the biofeedback signal (12). However, our major findings demonstrate that the dyspnea elicited by this task is of a distinct quality, differs from sensations elicited by leg muscle tension, and cannot be explained by hyperventilation. Finally, it should also be noted that, within the context of the present experiment, some of the effect sizes (r²) of the interactions indicating an experiential specificity of chest muscle tension fell in the small to medium range. It remains to be seen how this translates into a daily life relevance of this mechanism when dyspnea is sampled across a wide range of ecologically valid situations. As had been noted by others, individuals high in negative affect typically report a greater amount of physical sensations across a wide range of situations (53). Controlling for this tendency in covariance analysis somewhat attenuated effects on some, but not all, respiratory symptom measures. In addition, we also observed a tendency for elevated chest muscle tension across the full range of tasks used in this experiment, suggesting a nonspecific tendency for tensing respiratory muscle tension during experimental challenge. Thus, replication and extension of our findings are necessary before we can draw stronger conclusions on respiratory dysregulation in anxiety and panic.

In conclusion, this study suggests that respiratory muscle tension is an alternative pathway for symptom generation in individuals with high anxiety sensitivity. These findings could have important implications for theories of panic disorder and for treatment approaches that focus on breathing retraining for these patients. The biofeedback technique tested here could also

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2We are grateful to an anonymous reviewer for alerting us to this pathway.

3The paced breathing task in our study was devised as a control for the possible nonspecific experiential effects of the voluntary manipulation of breathing pattern, rather than as a hyperventilation challenge. Although substantial drops in PCO₂ were observed, they were not comparable with classical hyperventilation tasks, which tend to target levels around 25 mm Hg. This may have contributed to the lack of exaggerated responding of high-ASI participants to this task specifically. Drops in PCO₂ were not different between groups and most likely were due to increases in V̇ₐ (as seen in Table 3), which may reflect a nonspecific reactivity of participants to this task, who may have exaggerated their breathing excursions in an effort to do well.
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be used for directing respiratory muscle relaxation in a treatment context. Future studies should evaluate the role of respiratory muscle tension in patients with diagnosed panic disorder.

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