

Rollercoaster asthma: When positive emotional stress interferes with dyspnea perception[☆]

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Abstract

The current study assessed how negative and positive stress is related to dyspnea perception. The participants were 25 young women with a medical diagnosis of severe asthma, and 15 matched controls. Stress was induced during repeated rollercoaster rides. Results showed that negative emotional stress and blood pressure peaked just before, and positive emotional stress and heart beat peaked immediately after rollercoaster rides. Dyspnea in women with asthma was higher just before than immediately after rollercoaster rides, even in women with asthma with a rollercoaster-evoked reduction in lung function. These results suggest that stressed and highly aroused individuals with chronic asthma tend to perceive dyspnea in terms of acquired, familiar associations between dyspnea and positive versus negative feeling states, favoring either underperception or overperception of dyspnea, depending on the emotional valence of a situation.

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Introduction

Asthma is the most common of chronic diseases and is characterized by recurrent attacks of airway obstruction, which can be measured via lung-function testing. A reduction in lung function is associated with dyspnea (the generic term for breathlessness, shortness of breath, tightness of the chest, difficult breathing), concern, worry, and negative emotional stress (Lehrer, Feldman, Giardino, Song, & Schmalig, 2002). Although reduction in lung function and dyspnea are generally coinciding, physicians and researchers are now familiar with a phenomenon that caused lots of surprise in the 70s of the last century, when it was found that lung function and dyspnea often poorly relate. That is, individuals with asthma may complain of dyspnea when testing does not reveal any deviation in lung function, or, contrarily, individuals with asthma do not report dyspnea in the midst of an asthma attack (Lehrer et al., 2002).

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Many studies have shown that this relationship between lung function and dyspnea is influenced by emotional situations, particularly because anxious patients tend to overperceive dyspnea (Boulet, Cournoyer, Deschesnes, Leblanc, & Nouwen, 1994; Janson, Bjornsson, Hetta, & Boman, 1994; Rietveld, Everaerd, & Van Beest, 1999; Rietveld, Van Beest, & Everaerd, 1999; Spinhoven, Van Peski-Oosterbaan, Van der Does, Willems, & Sterk, 1997; Tiller, Pain, & Biddle, 1987). Likewise, the counter-intuitive observation that individuals with asthma were less accurate than controls in the perception of externally applied interruptions in airflow while breathing through a tube-like apparatus has also been attributed to emotional influences (Rietveld, Kolk, Prins, & Van Beest, 1997). Furthermore, recent research has shown that long-term negative emotional stress in individuals with asthma is associated with a shift in immune response, favoring airway sensitivity. For example, one study showed that a reduction in lung function in students with asthma was more likely during a high-stress examination period, as compared to a low-stress post-examination period (Liu et al., 2002).

However, the effects of acute positive emotional stress on parameters of asthma seem to have been neglected. Positive stress would be defined as a state of heightened physiological arousal in conjunction with subjective reports of positive mood or emotion, e.g. joy or happiness. From a psycho-physiological perspective, there seems to be little difference in the physiological response during emotional stress that subjects perceive negatively versus positively (Anderson, 1990; Blascovich, 1990). For example, in vivo assessment with ambulatory equipment revealed similar effects of positive and negative emotions on heart rate and blood pressure (Jacob et al., 1999). A difference would be that negative emotions may have longer lasting effects on heart beat than positive emotions, e.g. until 5 min after the emotional event (Brosschot & Thayer, 2003). Physiological differences within minutes following a negative or positive stressor would probably be negligible.

On the subjective level, the difference between positive and negative emotional stress could be crucial for dyspnea perception. Literature has suggested that individuals with asthma have learned to associate negative situations, emotional stress, being upset or distressed, with difficult breathing, and are thus likely to overperceive dyspnea in such situations (Bass & Gardner, 1995; Boulet et al., 1994; Houtveen, Rietveld, & De Geus, 2003; Meyer, Kroner-Herwig, & Sporkel, 1990; Rietveld, Everaerd, & Creer, 2001; Ritz, Steptoe, DeWilde, & Costa, 2002; Schmaling, McKnight, & Afari, 2002). The possibility of an opposite effect, a learned association between happy, joyful situations, and breathing without dyspnea, resulting in underperception of airways obstruction, has been suggested in literature, but not experimentally tested (Kang & Fox, 2000; Miller & Wood, 1997; Rietveld & Everaerd, 2000). The few studies to the positive emotion–asthma relationship (e.g. Miller & Wood, 1997) focused on diary self-reports of individuals with asthma, or while they were watching emotional films, whereas severe dyspnea may generally relate to acute high-arousal situations (Janson et al., 1994). Indeed, substantial errors in dyspnea perception are to be expected when individuals are emotionally stressed and highly aroused (Rietveld & Houtveen, 2004). Hence, when studying the influence of positive emotions on dyspnea perception, high levels of subjective stress in conjunction with physiological arousal, e.g. enhanced heart rate and blood pressure seems warranted.

The current study tested the effect of acute positive emotional stress on dyspnea perception. Volunteer women with asthma and matched controls underwent rollercoaster rides to evoke positive emotional stress (cf. Meston & Frohlich, 2003). Dyspnea, lung function, and control variables were repeatedly measured over a 2-day period. The hypothesis was tested that positive emotional stress after riding a rollercoaster interferes with dyspnea perception, resulting in low-dyspnea scores among women with asthma. It was expected that the rollercoaster would evoke anticipatory negative emotions in conjunction with enhanced heart rate and blood pressure shortly before the task, and positive emotions in conjunction with enhanced heart rate and blood pressure immediately after the task.

The rollercoaster rides were selected for this study because they would not meet the ethical and practical problems of induction of emotional states. The participants were tested whether their anticipatory feelings met the criterion of a positive emotional stress task. In this respect, all participants had prior experience with a rollercoaster ride and claimed that they would enjoy the task. Pilot testing showed that the heart rate acceleration mentioned from novice riders in literature (Kuschyk, 2005; Pringle, Macfarlane, & Cobbe, 1989) were not reached. Because of this rather mild heart rate acceleration after a single ride, it was decided to have all participants do the ride twice, one immediately following the other.

Only women were selected for this study because of huge gender differences in both stress response and symptom perception. With respect to stress response, Taylor et al. (2000) emphasized the gender difference in their ‘Tend–Befriend’ versus ‘Fight–Flight’ stress response. They argued that these basic differences have physiological and endocrinological correlates with consequences for all aspects of the stress response (cf. Matthews, Davis, Stoney, Owens, & Caggiula, 1991; Matthews and Stoney, 1988). With respect to gender differences in symptom perception, both clinical and experimental studies have shown that women report more somatic symptoms, including dyspnea, and cough more often, than men (Gijsbers-van Wijk, Huisman, & Kolk, 1996; Rietveld & Rijssenbeek-Nouwens, 1998). Although these differences may reflect gender differences in mood states, rather than differences in perceptual capacity (Gijsbers-van Wijk & Kolk, 1997), the current study was based on a homogenous group with only young women with and without asthma taking part.

Heart rate and blood pressure were repeatedly measured as markers of physiological arousal, in order to confirm the effect of the rollercoaster, and to gain insight in the physiological response underlying dyspnea perception. Positive and negative mood states were measured as a rough indicator of feeling states throughout the study. Somatic symptoms relating to the rollercoaster were also repeatedly measured. Trait anxiety was measured as a marker of neuroticism, to be able to exclude highly anxious individuals, and to gain insight in the influence of neuroticism on dyspnea perception. Individuals with high scores on neuroticism would (a) eagerly scan their body for signs and symptoms of disease; (b) be vulnerable to falsely perceive general sensations in terms of asthma and dyspnea; and (c) react emotionally and excessively to dyspnea (Rietveld & Houtveen, 2004).

Methods

Participants

The participants were 25 female university students with a medical diagnosis of severe asthma who were matched on age, gender, and education with 15 healthy controls. Control women were, according to self-report intake forms, free of chronic disease and were symptom-free at intake.

All women with asthma used inhalation corticosteroids in excess of 800 mg daily. They continued using this kind of medication during the test period, which serves to control airway inflammation and decreases the sensitivity of the airways to agents evoking airway obstruction. However, they were instructed not to use B-antagonist bronchodilators on the morning of the 2 days of testing, because this kind of medication, serving to relief airway obstruction, would possibly result in a feeling of insensibility or invulnerability regarding the airways, influencing dyspnea perception. The ethical protocol prohibited participation of women with asthma with a pretest lung function < 70% of the value predicted for a woman of similar size and weight.

Participants were recruited via posters in the university building, asking for women with and without asthma who would enjoy riding rollercoaster. They were fully informed about the experimental procedures in advance and asked to sign an informed consent. All participants responded with ‘yes’ on an intake inquiry whether they (a) had prior experience with riding a rollercoaster, and (b) expected that they would enjoy the rollercoaster rides. According to intake responses, the participants were not pregnant, had no acute or chronic disease or disability, and no history of psychological dysfunction (no current or past psychotherapy or specific medication), and all of them scored within the normal range on trait anxiety (neuroticism); see Sections *Assessment of Trait Anxiety* and *Results*. The mean age of the women with asthma was 20.5 (SD = 1.3) years, and of the controls 20.6 (SD = 1.3) years. They received 12 euro for their participation.

All participants were carefully debriefed about the background of the study, including recommendations in case of post-experimental symptoms of asthma. The study was approved by the ethics committee of the Department of Psychology. A physician supervised rollercoaster rides for patients’ safety. Patients with a reduced lung function were monitored until their lung function was back to normal. If needed (but this was actually never the case), patients were given bronchodilator medication.

Measures

Assessment of dyspnea

Dyspnea was assessed with a single-item self-report measure. Responses ranged from ‘not at all’ (0) to ‘most severely’ (8). The same measure has extensively been used in former research (cf. Rietveld, Everaerd et al., 1999; Rietveld, Van Beest et al., 1999).

Assessment of lung function

Lung function was measured with a portable spirometer (Spirosense, Lode BV, Groningen, The Netherlands), connected with a laptop computer (Compac type 1130), and expressed in the percentage of predicted forced expiratory volume in 1 s (FEV₁). The best of three trials was used for analysis. Note that a reduction in lung function of 20% is the gold standard for lung physicians and considered to be clinically relevant, although 10% reduction may cause considerable dyspnea.

Assessment of heart rate

Heart rate was measured with a band that was strapped around the chest of the participants (Tamarac Systems, Tampere, Finland). The values were expressed in mean number of beats per minute. Note that post-rollercoaster assessment was conducted within approximately 45 s after the actual rides.

Assessment of blood pressure

Blood pressure was measured by a physician with an Imron HEM-711 sphygmomanometer (Fuzzy Logic, USA) and expressed in systolic and diastolic values.

Assessment of somatic rollercoaster symptoms

Three somatic symptoms that are associated with riding a rollercoaster were measured: dizziness, nausea, and heart pounding. The items were based on a pilot test where participants scored physical symptoms just before and immediately after a rollercoaster ride on the 28 items of the symptom scale used previously (Rietveld & Houtveen, 2004). The selected items distinguished best between the self-report just before and immediately after the rollercoaster rides. These symptoms were presented in a 9-point Likert-type scale format. Responses ranged from ‘not at all’ (0) to ‘most severely’ (8). Lowest Cronbach’s α was .71 (just after the rollercoaster ride) (cf. Rietveld & Houtveen, 2004).

Assessment of positive and negative emotional stress

Subjective emotional stress was measured with four items for positive and four items for negative emotional stress: elation, joy, happiness, cheerfulness, fear, anger, sadness, and frustration. The items were based on a pilot test where participants scored mood states just before and immediately after a rollercoaster ride on the items of the POMS-BI (McNair, Lorr, & Droppleman, 1971, 1992). The selected items distinguished best between the mood states just before and immediately after the rollercoaster rides. The item frustration was added to make the current stress results comparable with our previous studies with experimental stress induction (cf. Houtveen et al., 2003). These items were presented in a 9-point Likert-type scale format. Responses ranged from ‘not at all’ (0) to ‘most severely’ (Rietveld & Houtveen, 2004). The total score was 0–32 points for positive emotional stress, and 0–32 points for negative emotional stress. Lowest Cronbach’s α for the positive scale was .57 (just before the ride). Lowest Cronbach’s α for the negative scale was .47 (just before the ride).

Assessment of trait anxiety

Trait anxiety was measured for a possible exclusion of highly neurotic participants. Trait anxiety was also measured as an additional variable, because neuroticism is often mentioned as a substantial factor in the overperception of dyspnea (Boulet et al., 1994; Houtveen et al., 2003). Trait anxiety was measured during intake with a Dutch version of the corresponding subscale of the Spielberger State–Trait Anxiety Inventory (Spielberger, 1983). The instrument comprises 20 items with statements about positive and negative feeling states. The participants responded on a 4-point Likert-type scale how these statements applied to them,

ranging from 1 (not at all) to 4 (very much). The total score was 20–80 points. The validity and reliability of the instrument have extensively been supported (Rietveld & Houtveen, 2004). Cronbach's α in the current study was .71.

General procedure

The study comprised four phases. On the morning of rollercoaster rides, intake procedures were carried out, including assessment of dyspnea, lung function, heart rate, blood pressure, somatic rollercoaster symptoms, positive and negative emotional stress—all measured in this order. After mid-day, the participants were transported to the nearby public rollercoaster setting, where dyspnea, lung function, heart rate, blood pressure, somatic rollercoaster symptoms, and positive and negative emotional stress were measured. Next, the participants underwent two consecutive rollercoaster rides to evoke emotional stress, which lasted 12 min, with a pause of 1–2 min between the rides. Each participant underwent the rides in a group of strangers, not together with other participants. Immediately after, dyspnea, lung function, heart rate, blood pressure, somatic rollercoaster symptoms, and positive and negative emotional stress were measured. Exactly 24 h after rollercoaster rides, all participants returned to the institute for assessment of dyspnea, lung function, heart rate, blood pressure, somatic rollercoaster symptoms, and positive and negative emotional stress. All assessments were conducted without other people watching, and the results were written in a booklet with each assessment on a separate page. There were two experimenters and a lung-function assistant present, and during the rollercoaster rides a physician as well—all being women.

Results

All analyses were conducted using a 2 (group: asthma, control) \times 4 (measurement: intake, just before rollercoaster rides, immediately after rollercoaster rides, 24 h after rollercoaster rides) analysis of variance with measurement as within variable. In addition, Tukey's HSD comparisons were performed to determine the nature of the effects. The means and standard deviations of all dependent measures are presented in Table 1.

Heart rate

The analysis of heart rate yielded only a main effect of measurement, $F(3, 114) = 315.56, p < .001$, with an effect size of $\eta^2 = .89$. The main effect of group, $F(1, 38) = 1.06, ns$, and the interaction effect of group and measurement, $F(3, 114) = .71, ns$, were nonsignificant. Follow-up Tukey HSD comparisons showed that heart rate peaked immediately after rollercoaster rides.

Table 1
Means and standard deviations (within brackets) of main dependent variables as a function of asthma and measurement

	Asthma				Control			
	Intake	Before	After	Next	Intake	Before	After	Next
Hearth	82.72 _a (5.18)	96.60 _b (6.23)	120.92 _c (10.19)	80.72 _a (5.82)	81.80 _a (5.81)	96.60 _b (5.46)	116.27 _c (10.36)	80.13 _a (2.92)
Systolic	125.56 _b (6.04)	143.80 _d (9.03)	137.04 _c (8.07)	125.76 _b (4.23)	122.33 _a (4.99)	138.00 _c (4.38)	136.80 _c (4.86)	123.87 _{a,b} (3.52)
Diastolic	77.72 _{a,b} (4.88)	94.72 _d (6.33)	88.96 _c (5.58)	79.00 _b (3.61)	76.73 _{a,b} (4.76)	89.87 _c (4.17)	87.33 _c (3.22)	75.93 _a (4.11)
Stress P	3.44 _b (1.53)	10.48 _c (3.77)	17.76 _c (4.80)	2.72 _{a,b} (1.54)	3.87 _b (1.41)	11.00 _c (3.36)	14.93 _d (5.80)	1.93 _a (1.28)
Stress N	2.92 _a (1.53)	7.84 _c (2.91)	2.12 _a (2.40)	2.48 _a (1.53)	4.07 _b (1.98)	9.00 _d (3.30)	2.13 _a (1.19)	2.27 _a (1.22)
FEV ₁	87.42 _b (10.27)	87.13 _b (9.77)	83.13 _a (14.54)	87.46 _b (8.89)	98.67 _c (5.34)	99.13 _c (5.41)	98.13 _c (3.76)	98.00 _c (4.42)
Dyspn	.64 _c (.81)	1.48 _d (.87)	.72 _c (.61)	.44 _{b,c} (.51)	.20 _{a,b} (.41)	.33 _{a,b} (.48)	.73 _c (.79)	.07 _a (.26)
Roller	1.12 _c (.83)	2.88 _d (.88)	10.40 _c (1.73)	.40 _b (.50)	.20 _a (.56)	2.60 _d (.74)	10.07 _c (1.43)	.33 _{a,b} (.49)

Note: Hearth = hearth rate, Systolic = systolic blood pressure, Diastolic = diastolic blood pressure, Stress P = positive emotional stress, Stress N = negative emotional stress, FEV₁ = forced expiratory volume in 1 s, Dyspn = dyspnea, Roller = rollercoaster symptoms. Means in the same row that do not share subscripts differ at $p < .05$ in the Tukey's HSD comparison.

Blood pressure

The analysis on systolic blood pressure yielded a main effect of measurement, $F(3, 114) = 125.90$, $p < .001$, $\eta^2 = .77$, and a marginal main effect of group, $F(1, 38) = 3.17$, $p = .08$, $\eta^2 = .08$. The interaction effect of group and measurement was nonsignificant, $F(3, 114) = 1.72$, *ns*. Follow-up Tukey HSD comparisons showed that systolic blood pressure just before and immediately after rollercoaster rides was significantly higher than during intake and 24 h after the rollercoaster rides.

The analysis of diastolic blood pressure yielded a main effect of group, $F(1, 38) = 4.98$, $p < .05$, $\eta^2 = .12$. Women with asthma had higher diastolic blood pressure than controls. It also yielded a main effect of measurement, $F(3, 114) = 159.31$, $p < .001$, $\eta^2 = .81$. The interaction effect of group and measurement was nonsignificant, $F(3, 114) = 2.00$, *ns*. Follow-up Tukey HSD comparisons showed that diastolic blood pressure just before and immediately after the rollercoaster rides was significantly higher than during intake and 24 h after the rollercoaster rides. Moreover, diastolic blood pressure just before the rides was also higher than immediately after the rides.

Somatic rollercoaster symptoms

The analysis of rollercoaster-related symptoms yielded a main effect of measurement, $F(3, 114) = 1016.40$, $p < .001$, $\eta^2 = .96$, and a marginal main effect of group, $F(1, 38) = 3.35$, $p = .09$, $\eta^2 = .08$. The interaction effect of group and measurement was not statistically significant, $F(3, 114) = 1.59$, *ns*. Follow-up Tukey HSD comparisons indicated that both groups reported more rollercoaster-related symptoms immediately after the rollercoaster rides.

Positive and negative emotional stress

The analysis of positive emotional stress yielded a main effect of measurement, $F(3, 114) = 200.64$, $p < .001$, $\eta^2 = .84$, and an interaction effect of measurement and group, $F(3, 114) = 2.90$, $p < .05$, $\eta^2 = .07$. The main effect of group was nonsignificant, $F(1, 38) = .81$, *ns*. Follow-up Tukey HSD comparisons showed that positive stress peaked right after the rollercoaster rides for both groups, but that this effect was stronger for women with asthma.

An analysis of negative emotional stress yielded only a main effect of measurement, $F(3, 114) = 323.82$, $p < .001$, $\eta^2 = .72$. The main effect of group, $F(1, 38) = 1.25$, *ns*, and the interaction effect of group and measurement, $F(3, 114) = 1.47$, *ns*, were nonsignificant. Follow-up Tukey HSD comparisons showed that both groups experienced more negative emotional stress just before the rollercoaster rides as compared to all other assessments.

Dyspnea

The analysis of dyspnea yielded a main effect of group, $F(1, 38) = 9.22$, $p < .01$, $\eta^2 = .20$, a main effect of measurement, $F(3, 114) = 13.16$, $p < .001$, $\eta^2 = .26$, and an interaction effect of group and measurement, $F(3, 114) = 8.85$, $p < .001$, $\eta^2 = .18$. The main effect of group indicated that women with asthma reported overall more dyspnea than controls. The main effect of measurement indicated that participants reported most dyspnea just before the rollercoaster ride, but this was qualified by an interaction with group (see Fig. 1). Further Tukey HSD comparisons showed that dyspnea peaked before the rollercoaster rides among women with asthma, but peaked immediately after the rollercoaster rides among controls.

Lung function

The analysis of lung function yielded a main effect of group, $F(1, 37) = 18.62$, $p < .001$, $\eta^2 = .35$, a main effect of measurement, $F(3, 111) = 3.45$, $p < .05$, $\eta^2 = .09$, and a marginally significant interaction effect of measurement and group, $F(3, 111) = 2.38$, $p = .07$, $\eta^2 = .06$. The main effect of group indicated that women with asthma had overall a lower lung function than controls. The main effect of measurement indicated that

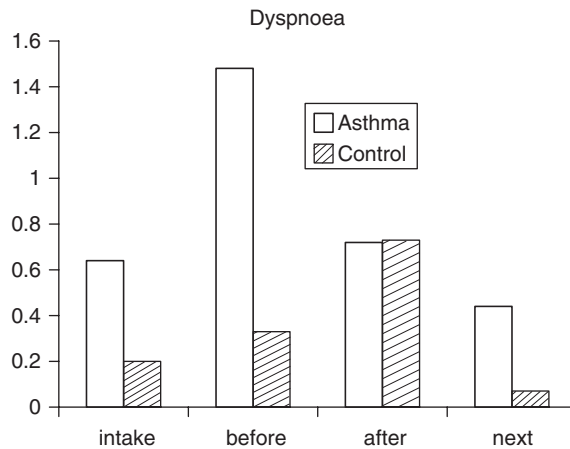


Fig. 1. Dyspnoea as a function of group and measurement. Hatched bars = control. Solid bars = asthma.

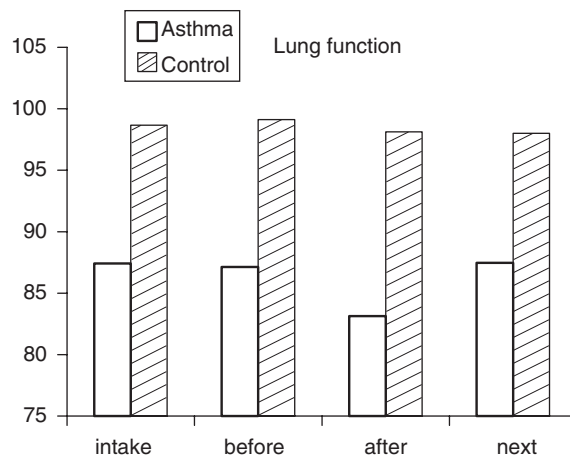


Fig. 2. Forced expiratory volume in 1 s (lung function) as a function of group and measurement. Hatched bars = control. Solid bars = asthma.

lung function was reduced immediately after the rollercoaster rides, but this was qualified by an interaction with group (see Fig. 2). Further Tukey HSD comparisons showed that the lung function of women with asthma was reduced immediately after the rollercoaster rides, but was not affected in controls. In fact, the rollercoaster rides evoked in 39% of the women with asthma a reduction in lung function (FEV_1) of >10%.

Trait anxiety

The analysis of trait anxiety yielded a marginal main effect of group, $F(1, 38) = 3.32, p = .07, \eta^2 = .08$. This marginal effect of group suggests that women with asthma scored (not significantly) higher on trait anxiety than controls. To check whether this could explain the above results, all analyses were repeated with trait anxiety as an extra independent variable (via a median split). These analyses did not yield any two-way interactions of group and trait anxiety, or three-way interactions of group, measurement, trait anxiety on any of the variables of the study. In addition, we also repeated all analyses with trait anxiety as a covariate. Again analyses did not alter the results. This shows that the above-mentioned results cannot be explained by differences in trait anxiety.

Discussion

The results showed that women with asthma experienced more dyspnea during negative emotional stress in conjunction with a normal lung function, than during positive emotional stress and a reduction in lung function, even though 39% of these women had a reduction in lung function of >10%. For women without asthma, dyspnea followed the same pattern as dizziness, nausea, and heart pounding: that is, peaking after and not before the rollercoaster rides. The overall results (a) supported the hypothesis that positive emotional stress interferes with dyspnea perception in women with asthma, and (b) corroborated previous research, showing that negative emotional stress facilitates dyspnea perception in individuals with asthma (Lehrer et al., 2002).

There were no differences between women with asthma and controls in rollercoaster symptoms and hence no support for the possibility that women with asthma falsely perceived neurological signals from airway obstruction as, e.g. dizziness or nausea, instead of dyspnea. Such false labeling of somatic symptoms by individuals with asthma has been proposed as a mechanism of overperception of dyspnea (Lehrer et al., 2002; Rietveld, Everaerd et al., 1999).

The results also argue against the proposition that individuals with asthma falsely perceive physiological arousal as habitual, anticipated dyspnea (Rietveld, Everaerd et al., 1999; Tiller et al., 1987). In the current study, if physiological arousal was perceived as dyspnea, it happened only before and not after the rollercoaster rides, that is, only during negative emotional stress and not during positive emotional stress. Moreover, enhanced scores on nausea, dizziness, and heart pounding after the rollercoaster rides argue against an endocrinological rebound effect accounting for low dyspnea after the rides, e.g. endorphin release after high physiological stress interfering with dyspnea perception (Akiyama et al., 1993). Finally, dyspnea perception was not related to levels of trait anxiety c.q. neuroticism scores. Firstly, only participants with a score in the normal range of trait anxiety took part. Secondly, the inclusion of trait anxiety in the statistical analyses did not yield any significant result. Hence, the conclusions of this study are not confounded by the extreme scores of highly anxious or neurotic participants (cf. Janson et al., 1994; Tiller et al., 1987).

Excluding all these factors used in literature to explain underperception or overperception of habitual symptoms by chronic patients, the current results suggest that the women with asthma were indeed influenced in their dyspnea perception by emotional stress, facilitating dyspnea during negative emotional stress, and interfering with dyspnea during positive emotional stress. Many of these women with asthma have over and over again been exposed to often highly distressing asthma attacks and may have categorized dyspnea among negative feelings and not among positive feelings. The tentative conclusion would thus be that individuals with asthma have acquired an association between negative feeling states and dyspnea on the one hand, and between positive feeling states and absence of dyspnea on the other, which influences their symptom perception. In high-arousal situations, individuals with asthma seem to be overwhelmed by acquired associations between feeling states and dyspnea toward overperception or underperception of dyspnea, depending on the emotional valence of the situation (cf. Meston, & Frohlich, 2003; Schachter & Singer, 1966; Sinclair, Hoffman, Mark, Martin, & Pickering, 1994).

There were several limitations in this study. Only young women participated, which diminishes the generalizability of the findings. However, the study attempted to elucidate the mechanism of differences in dyspnea perception during emotional stress in asthma. Including males in the analysis would have made the interpretation of the current results problematic. Previous research by Taylor et al. (2000) postulated that men would respond to a stressor with enhanced sympathetic nervous system activation, and women with greater activation of vagal mechanisms, and greater release of oxytocin and endorphins within the brain. These differences could differently influence, e.g. airway spasm, and dyspnea intensity (Rietveld, Everaerd et al., 2001). The current limited age range makes it unclear how the results would fit an older sample of asthma patients, but an age effect on dyspnea perception in a younger age group (7–18 years) is unlikely on the basis of previous research (Lehrer et al., 2002).

Because of practical reasons, breathing patterns during rollercoaster rides were not assessed. This lack of assessing breathing patterns could have been informative about the mediating role of respiration in the lung function–dyspnea relationship. With 39% of the current women with asthma having a reduction in lung function of >10%, and a clinical significant degree of airway obstruction in some, differences in respiratory

patterns may have been substantial, e.g. some women with airway obstruction breathing shallowly, others breathing deeply, sighing, coughing, etc. (Bass & Gardner, 1995; Chadha, Schneider, Birch, Jenouri, & Sackner, 1984). Nevertheless, our former research provided no substantial difference in breathing patterns during emotional stress after a stressful public ‘intelligence test’ between (a) women with asthma and controls, (b) women with asthma with a relatively high versus a low lung function, and (c) with a high versus a low score on dyspnea (Rietveld & Houtveen, 2004). Moreover, there is abundant literature that dyspnea perception in general does not—or only poorly—relate to objective physical measures, e.g. baseline respiratory rate, airway sensitivity, lung function, used medication, and asthma severity (Lehrer et al., 2002; Rietveld, Prins, & Colland, 2001).

There were also no physiological data providing insight in the mechanism underlying changes in lung function, e.g. differentiating between women with and without a reduction of >10% in lung function. However, this study focused on dyspnea and not on mechanisms of stress-evoked airway obstruction, which would require a battery of additional physiological and immunological measures. In fact, endocrinological measures (cortisol, eosinophils) were conducted in this study, but samples turned out to be unreliable and results were too diverse to legitimate publication. Nonetheless, semi in vivo induction of airway obstruction is rare in research and all factors involved should have been assessed with relevant measures.

The current results yielded a difference in physiological arousal during positive and negative stress, which may appear contrary to the introductory statements about a lack of such a difference. These differences in physiological arousal during positive versus negative stress probably result from worrying about negative stressors, i.e. self-generated arousal accounting for the physiological difference, which would thus merely be a difference in duration of arousal (Brosschot & Thayer, 2003). Furthermore, it may seem strange that joy and happiness coincided with heart rate acceleration, and rollercoaster-related symptoms such as enhanced dizziness and nausea. However, the experience of stress during high levels of physiological arousal as something positive is also well documented in studies on sensation-seeking behavior, sport, and sexuality (Ekkekakis, 2003; Sinclair et al., 1994). Indeed, it is exactly the reason why most people enjoy riding a rollercoaster.

In sum, previous research focused on the relation between negative stress and overperception of dyspnea. The factors underlying underperception, i.e. no dyspnea during a reduction in lung function, were not well understood (Lehrer et al., 2002). The current study extended knowledge about underperception of dyspnea. After riding a rollercoaster, which induced a positive feeling state, women with asthma seemed to underperceive dyspnea, even those with a reduction in lung function. This poor perception of dyspnea seems to be independent of anxiety or neuroticism, in the sense that relatively normal individuals with asthma may, sometimes, or in certain situations, either not perceive airway obstruction, or experience dyspnea in the absence of airway obstruction (Lehrer et al., 2002; Spinhoven et al., 1997). The clinical implication is that individuals with asthma may sometimes not use their bronchodilator medication when they should, or use their bronchodilator medication when they should not (Rietveld & Koomen, 2002). More specifically, the current finding suggests that individuals with asthma are vulnerable to underperceive dyspnea when they are in a state of positive emotional stress, and are thus likely to omit using required medication in such situations.

In this respect, the current study may have contributed to a better understanding of the complex relationship between feeling states and dyspnea in asthma. It seems clear that individuals with asthma are influenced by feeling states in the critically important self-management of asthma, perhaps sometimes for better, but clearly more often for worse. The factors underlying poor symptom perception in asthma are—except for isolated cases—not physical, but psychological, relating to expectations on the basis of previous asthma attacks (Meyer et al., 1990; Rietveld, 2003); distraction (Rietveld et al., 1996); and emotional stress (Lehrer et al., 2002). Hence, the role of feeling states and anticipations in symptom perception may be greater than suggested in literature and may particularly overwhelm chronic patients when they are highly aroused. Physicians should inform patients of these influences of feeling states when coping with and managing asthma.

To conclude, the current study subscribes to the need for new and specific research on the physiological and emotional factors underlying symptom perception in asthma. Such research should be multi-disciplinary, combining sound physiological (respiratory, immunological) factors and a manipulation of feeling states or extensive in vivo assessments.

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