Published in final edited form as:

Psychophysiology. 2011 January ; 48(1): 121–135. doi:10.1111/j.1469-8986.2010.01044.x.

Airway response to emotion- and disease-specific films in asthma, blood phobia, and health

Thomas Ritz^{1,*}, Frank H. Wilhelm², Alicia E. Meuret¹, Alexander L. Gerlach³, Walton T. Roth⁴

¹Department of Psychology, Southern Methodist University, Dallas, TX, USA ²Department of Psychology, University of Basel, Basel, Switzerland ³Department of Psychology, University of Münster, Münster, Germany ⁴Psychiatry Service, Stanford University School of Medicine and VA Palo Health Care System, Palo Alto, CA, USA

Abstract

Earlier research found autonomic and airway reactivity in asthma patients when exposed to blood-injection-injury (BII) stimuli. We studied oscillatory resistance (R_{os}) in asthma and BII-phobia during emotional and disease-relevant films and examined whether muscle tension counteracts emotion-induced airway constriction. Fifteen asthma, 12 BII phobia patients, and 14 healthy controls viewed one set of negative, positive, neutral, BII-related, and asthma-related films with leg muscle tension and a second set without. R_{os} , ventilation, cardiovascular activity, and skin conductance were measured continuously. R_{os} was higher during emotional compared to neutral films, particularly during BII material, and responses increased from healthy over asthmatic to BII phobia participants. Leg muscle tension did not abolish R_{os} increases. Thus, the airways are particularly responsive to BII-relevant stimuli, which could become risk factors for asthma patients.

Keywords

asthma; blood-injection-injury phobia; respiratory resistance; emotion; respiration; autonomic nervous system; electrodermal activity

Bronchoconstriction is one of the key clinical features of asthma (National Heart, Lung, and Blood Institute [NHLBI], 2002). Prior studies have demonstrated that experimental emotion induction is capable of constricting the airways, and that this reaches clinically relevant levels in a considerable proportion of asthma patients (for reviews, see Isenberg, Hochron & Lehrer, 1992; Ritz & Kullowatz, 2005). While the weight of the evidence suggests that states of negative affect are particularly potent in eliciting airway responses, instances of airway constriction in positive emotional states have also been reported (Liangas & Morton, 2003; Ritz, Steptoe, DeWilde, & Costa, 2000; von Leupoldt & Dahme, 2005), suggesting a nonspecific arousal modulation of airway smooth muscle tone. Both asthma patients and

* Address reprint request to: Thomas Ritz, Department of Psychology, Southern Methodist University, Hyer Hall 306C, 6424 Hilltop Lane, Dallas, TX 75205, phone: +1 214 768 3724, fax: +1 214 768 3910, tritz@smu.edu.

healthy controls show qualitatively, and in many studies quantitatively, comparable responses of the airways to such stimulation (e.g. Lehrer et al., 1996; Ritz et al., 2000; Ritz, Thöns, Fahrenkrug, & Dahme, 2003; von Leupoldt & Dahme, 2005). Although this indicates that under emotional challenge the airways do not provide strong evidence for disease-specific responding in asthma, airway responses of asthma patients to negative experimental film stimuli have been found to be correlated with lung function decline in strong states of negative affect in daily life (Ritz & Steptoe, 2000). In addition, responses to both negative and positive film stimuli correlate with patients' perceived importance of emotions as triggers of asthma in their daily lives (Ritz, Steptoe, Bobb, Harris & Edwards, 2006).

Previously we observed that asthma patients show particularly strong emotional arousal and increases in respiratory resistance to blood, injection, or injury (BII) stimuli, which consisted of a series of pictures depicting blood, injuries and mutilated bodies (Ritz et al., 2000). Similar stimuli have been associated with reductions in heart rate in healthy individuals (e.g. Carruthers & Taggart, 1973; Lang, Greenwald, Bradley, & Hamm, 1993) and vasovagal fainting in individuals who are sensitive or phobic to BII stimuli (Engel, 1978; Graham, Kabler, & Lunsford, 1961). Because vagal excitation is a powerful constrictor of the airways, it has been speculated that the airways of asthma patients may be more sensitive to blood and injury stimuli (Lehrer, Isenberg, & Hochron, 1993). Given the uniqueness of our findings, we sought to replicate them and compare the size of the response to BII stimuli to that elicited by general emotional stimuli, in particular unpleasant emotional states. In the previous study (Ritz, Steptoe, et al., 2000), a direct comparison with effects of general emotional stimuli was not possible, because these stimuli were presented as films rather than still pictures as the BII stimuli. Differences in stimulus type were thus confounded with the emotion induction method. For the current study, we used films only.

Intense emotional responses to BII stimuli, as they are observed in BII phobia patients, can potentially provide an interesting model for studying the specificity of this emotional stimulus type on the airways in asthma. These patients have healthy airways but are affected by strong phobic responses to BII stimulus material. For the purpose of this study we measured respiratory resistance continuously in patients with asthma and BII phobia as well as in healthy controls during general emotional films and blood- and injury-related films. To control for the disease-relevance of the material, we also administered asthma-related film scenes portraying asthma attacks and labored breathing, because such disease-relevant material had also been suggested to be particularly potent in its effect on the airways in asthma (Levenson, 1979). A specific airway responsiveness of asthma patients to BII relevant stimuli would be demonstrated if resistance increases were (i) stronger in asthma compared to BII patients and healthy control, and (ii) were stronger in asthma during BII stimuli than any other emotional and disease-relevant stimulus.

Physical exercise and skeletal muscle tension have an immediate dilatory effect on the airways (Mansfield, McDonnell, Morgan, & Souhrada, 1979; Warren, Jennings & Clark, 1984) in both health and asthma. While exercise is a well-known trigger of airway obstruction in asthma (McFadden & Gilbert, 1994), the typical exercise response is bronchodilation in the early phase of exercise followed by a constriction in later phases and following exercise (Beck, Offord & Scanlon, 1994). Similarly, brief static contraction of

facial or arm muscles has been shown to reduce respiratory resistance below baseline levels (Ritz, Dahme & Wagner, 1998). There is also solid evidence from animal studies that skeletal muscle contractions dilate the airways (e.g. Kaufman, Rybicki, & Mitchell, 1985; Longhurst, 1984; Padrid, Haselton & Kaufman, 1990), with vagal withdrawal as the major mechanism behind these changes. Although increases in sympathetic activation may contribute to autonomic exercise effects, they most probably play a subordinate role on the airways, as no direct functional innervation of the airway smooth muscles is observed in humans (Barnes, 1986; Canning & Fischer, 2001). Circulating catecholamines can dilate the airways through β_2 -adrenergic receptors on the smooth muscles, but this pathway is less likely in shorter or mild to moderate levels of exercise (Lewis et al., 1985; Wasserman, Whipp & Casaburi, 1986).

Thus, tensing skeletal muscles could be a simple behavioral maneuver to reduce emotion-induced airway constriction and thereby help limit airway obstructions and symptoms in patients with emotion-induced asthma. However, this has never been tested. In BII phobia, voluntary contractions of the skeletal muscles have been used to counteract vasovagal fainting (Kozak & Miller, 1985; Öst & Sterner, 1987). Such contraction may also serve to dampen any stronger airway response to blood and injury stimuli in this patient group. Therefore, we studied respiratory resistance during conditions of emotional film viewing both with and without voluntary skeletal muscle contraction.

Some of the data from blood phobia patients and healthy controls in this study has been presented before (Ritz, Wilhelm, Meuret, Gerlach, & Roth, 2009; Ritz, Wilhelm, Gerlach, Kullowatz, & Roth, 2005). The focus of those analyses was to explore hyperventilation in blood phobia patients during exposure to feared stimuli. In contrast, the data reported here focus on the additional asthma group and on airway responses to both general emotional and disease-relevant film clips. In addition to respiratory resistance, we explored a number of autonomic and ventilatory parameters that are potentially linked to airway obstruction (Ritz et al., 2002). For instance, strong vagal excitations, as the most prominent autonomic pathway to airway obstruction in asthma, could be manifested in marked reduction in heart rate, which is typically observed in response to BII stimuli as part of the vasovagal response (e.g., Engel, 1978; Graham, Kabler, & Lunsford, 1961). In addition, marked increase in ventilation could constrict the airways in asthma patients either by drying of the airways (Freed, 1995) or by effect of reduced carbon dioxide partial pressure (PCO_2) (van den Elshout, van Herwaarden, & Folgering, 1991). To explore such patterns of ventilatory and autonomic change associated with airway constriction, we planned to study between- and within individual correlations of R_{os} change with these parameters.

We also sought to study changes in skin conductance relative to those observed in respiratory resistance. In previous studies of emotion induction, we found that skin conductance level (SCL) or skin conductance response closely matched the responses seen in respiratory resistance (Ritz, Steptoe, et al., 2000; Ritz, George & Dahme, 2000). Similarly, asthma patients with more airway hyperresponsiveness to methacholine have been previously found to respond with greater SCL increases during stressful tasks (Lehrer et al., 1996). A link between these parameters may seem less obvious, given that vagal excitation is the major source of airway constriction (Barnes, 1986; Canning & Fischer, 2001) and

sympathetic activation is responsible for skin conductance increases. Both systems, however, have cholinergic neurotransmission in common, and a link between allergic status and these two manifestations of cholinergic activity had been proposed earlier (Kalinin, 1976; Marshall, 1989). Thus, we compared response patterns of both parameters as well as their between- and within-individual associations.

Method

Parts of the methods have been presented in detail before (Ritz, Wilhelm et al., 2009; Ritz, Wilhelm et al., 2005). In short, 15 asthma patients (11 women, 4 men; mean age 40.1, range 20–55), 12 BII phobia patients (9 women, 3 men; mean age 37.3 years, range 21–57) and 14 non-anxious controls (10 women, 4 men; mean age 36.4 years, range 22–57) participated. Across groups, 75.6% of participants indicated being Caucasian, 7.3% Hispanic, 12.2% Asian, and 4.9% African- American. General selection criteria were age between 18 and 60 years and no history of epilepsy or seizures. All participants were screened using the Structured Clinical Interview for DSM-IV Patient Edition (First, Spitzer, Gibbon, & Williams, 1994). BII phobia patients were required to meet Axis I criteria for simple phobia, BII type. Seven phobic patients reported prior fainting in BII situations and two more had almost fainted. BII phobia patients also were required to have a normal 12-lead electrocardiogram (one patient with abnormality of the T wave was excluded for suspected inferior ischemia). Healthy participants and patients with BII phobia had to be free of current psychological disorders. They were only included if they reported no acute or chronic respiratory diseases and no current smoking. Asthma patients had intermittent to moderately persistent asthma (NHLBI, 2002) and had not received systemic corticosteroids in the previous three months. All but one asthma patient had undergone skin testing for allergies, with all of them positive. Symptoms were present more than two times per week in 53.3% of the asthma patient sample, nighttime symptoms more than two times per month in 46.7%, and restrictions in daily activities by asthma more than two times per week by 26.7%. Seasonal variations in symptoms were reported by 66.7% of the sample and a family history of asthma or allergies by 66.7%. Anti-asthmatic medication was used by all patients, with 80% using short-acting bronchodilators, 33.3% long-acting bronchodilators, 80% inhaled corticosteroids, 40% leukotriene inhibitors, 20% antihistaminics, and 13.3% mast cell stabilizers. We paid subjects \$60 for their participation in the experiment. Local ethics committees approved the study and we obtained informed consent from all participants.

Emotion Induction

Film sequences extracted from movies and medical education material were shown (lasting approximately 150 to 300s) (Gross & Levenson, 1995; Ritz, Steptoe, et al., 2000). Two films of each category were selected: negative (bullying scene, boy who cries about the death of his father), neutral (economics lecture, screen saver), positive (scenes from British and American comedy series), asthma-related films (scene of an asthma attack, scenes of intense emotion with labored breathing and wheezing), and BII phobia-relevant (scenes from educational surgery films with needle/injection images, cutting tissue, and spilling blood). For one set of films (constituted by one film from each category) the instruction was to view

all sequences for their entire length, and for the other set to view the films while tensing the leg muscles.

Participants were advised that some of the material they were to view would be of a medical nature and would contain scenes with blood and injuries. Participants were also asked to view the film sequences for their entire length and not to close their eyes. However, they were allowed to stop if they felt unable to continue viewing a film. They signaled that to the experimenter by taking out the tube for respiration measurement and raising their right hand. It was stressed, however, that the scientific value of the assessment would be greatest if they continued viewing for as long as possible. The experimenter confirmed that the patients' eyes were open by observing them through a one-way mirror during the surgery films. Two BII phobia patients refused to view the first surgery film in full (total viewing time 58 and 192s) and one patient refused to watch both surgery films in full (total viewing time 32 and 64s). For one more BII patient, the experimenter stopped the presentation of the second surgery films because of signs of extreme distress. These patients, as well as two who were close to fainting, were provided with ample additional recovery time after finishing the ratings. In addition, the experimenter did not initiate the next film presentation before physiological values were well within the initial range and participants indicated feeling sufficiently recovered.

Instructions for muscle tension

Leg muscle tension was kept at a subjective level of approximately 30–50% of the individual's maximum possible effort. In addition, the legs were to be crossed below the knees and pressed together at the same level of effort. We restricted voluntary contractions to leg muscles to avoid possible interferences of upper body muscle tension (in particular respiratory muscle tension) with breathing and respiratory resistance measurements.

Physiological Measurements

Oscillatory resistance (R_{os} , a measure of respiratory impedance, expressed in $\text{kPa} \cdot \text{l}^{-1} \cdot \text{s}$) was measured continuously with the single-frequency (10Hz) forced oscillation technique (Siemens Siregnost FD 5). Participants breathed air through an elastic tube (approximately 80cm length, 85 ml deadspace) (Ritz et al., 2002) using a mouthpiece and nose clip. In order to reduce shunt characteristics of the upper airways a padded elastic strap was attached to stabilize the cheeks and base of the mouth. Recordings of R_{os} were averaged after swallowing artifacts (rapid and brief 0.5–1.5-s increases) had been removed manually in an interactive scoring program.

The respiratory pattern was monitored using a thoracic and abdominal pneumatic belt system (James Long Company, Caroga Lake, NY). The system was calibrated using a fixed volume bag (800ml). Breath-by-breath respiration rate (RR) and tidal volume (V_T) was extracted from the calibrated respiration curve and minute ventilation (V'_E) was calculated by $RR \times V_T$.

End-tidal PCO_2 was measured with an infrared capnograph (Datex B, Puritan-Bennett Corporation, San Ramon, CA). Exhaled air was sampled continuously through a plastic tube (1.2mm diameter) at a flow rate of 150ml/min. Only breaths with distinct plateaus (Gardner,

1994) were scored by raters blind to the group assignment and experimental sequence. Hyperventilation is known to increase R_{OS} in asthma, although in healthy individuals a certain intensity of hyperventilation (drops of approximately 10mmHg or more) seems to be needed to affect the airways significantly (van den Elshout et al., 1991). We also counted frequency of sigh breaths/min (breaths 2 times the average V_T of the individual). Sighing is thought to be related to hyperventilation (Wilhelm, Trabert & Roth, 2001), which could increase R_{OS} . However, deep breaths have also been shown to reduce airway tone in health, a mechanism impaired in asthma (e.g., Scichilone et al., 2007).

For electrocardiogram measurements, three Ag-AgCl electrodes were attached, active ones on the sternum and laterally on the left costal arch at the level of the 10th rib and a ground electrode on the left clavicle. Data were lost for one BII patient during one set of films due to a defective lead. The time between successive R-waves of the electrocardiogram was converted to HR and mean HR was calculated across the entire film sequence. In addition, to explore whether HR slowing related to vasovagal responses (Engel, 1978; Graham et al., 1961) had occurred, HR_{minimum} was identified from successive 10-s means throughout each film. Although HR is under both sympathetic and parasympathetic influence and therefore does not allow unambiguous inferences on the underlying autonomic regulation (Berntson, Cacioppo & Quigley, 1991), HR slowing under BII stimulus exposure is thought to reflect a predominance of vagal excitation.

Beat-to-beat systolic blood pressure (SBP) and diastolic blood pressure (DBP) were monitored continuously with a Finapres model 2300 (Ohmeda, Madison, WI). The cuff was fitted to the middle phalanx of the middle finger of the left hand. Due to connector problems, BP was not recorded for one patient during the first three films. However, the experimenter estimated average values from a numerical display. A second patient felt highly uncomfortable with the cuff, so the experimenter discontinued recording. In addition, recordings were incomplete in two healthy participants due to cold fingers or equipment malfunction.

Muscle tension was monitored by electromyographic activity with two Ag-AgCl electrodes (3mm inner diameter) from an approximate soleus placement (Basmajian & Blumenstein, 1982) on the right leg. Defective leads led to data loss for one BII phobia patient and three healthy controls. Skin conductance level (SCL) was measured with two Ag-AgCl electrodes (6 mm contact area diameter, filled with 0.05-molar NaCl in Unibase) attached to the thenar and hypothenar eminences of the left hand. Data were lost for one control.

Biosignals were recorded with a Vitaport 2 digital recorder/analyzer (16-bit A/D converter, 512Hz sample rate) attached to an IBM-compatible microcomputer (Intel Pentium II processor). For storage, sampling rate was reduced to 256Hz for the electrocardiogram and plethysmographic pulse wave, and to 32Hz for respiratory signals. Raw EMG was rectified and integrated with a time constant of 62.5ms. Biosignals were edited for artifacts and analyzed using customized MATLAB software (Gerlach, et al., 2006).

Psychological Measures

Following each presentation, participants rated their emotions and symptoms during each film on a list of bipolar and unipolar rating scales. For the current analysis, we focused on dimensional bipolar ratings of pleasantness and arousal from the Self Assessment Manikin (Hodes, Cook III, & Lang, 1985). The two scales were scored with 1 assigned to the 'unpleasant' and 'calm' pole and 9 to the 'pleasant' and 'excited' pole. Analysis of unipolar scales was focused on the asthma-relevant symptoms of shortness of breath and chest tightness, both rated on 11-point scales (0='not at all', 10='extremely'). Ratings were incomplete for one asthma patient.

Before the session, participants also filled out the Medical Fears Survey (MFS; Kleinknecht, Thorndike, & Walls, 1995), which measures the intensity of fear (item scale 1–5, label range from “no fear” to “terror”) towards BII situations. Subscales with 10 items each were extracted for 'Injection & Blood Draws', 'Sharp Objects', 'Mutilations', 'Blood', and 'Examinations and Symptoms as Intimation of Illness'. In addition, to explore potential group differences in habitual affect we administered the Affect Intensity Measure (Larsen & Diener, 1987) and the Toronto Alexithymia Scale-20 (Bagby, Parker & Taylor, 1994), the latter with the three subscales for identification of emotions, communication of emotions, and externally oriented thinking. Data from two asthma patients, one BII patient, and one control were incomplete.

Procedure

Laboratory assessments were scheduled in the afternoon. Reliever medication (bronchodilators) were to be withheld for at least 8 hours before the laboratory session. Participants viewed films in a sound-attenuated chamber sitting in a comfortable armchair with a TV screen (approximately 40cm diagonal) at a distance of approximately 1.5m. The experimenter observed the participants through a one-way mirror from an adjacent room and communicated by intercom. Following sensor attachment and calibration, the first set of five film sequences was shown with the scheduled instruction, viewing only or viewing with muscle tension. The film set order was counterbalanced between groups. Both the order of the film sequences within sets as well as the assignment of sequences to sets were randomized. Each film presentation was followed by a 1-min recovery, during which recording of physiological parameters continued. Flexible amounts of time were allowed after each film for ratings of emotions and symptoms after which the experimenter inquired whether the participant was relaxed and ready to continue with the protocol. After completion of the first set of films, the second set followed with the other of the two instructions, viewing only or viewing with muscle tension.

After each surgery film, the experimenter inquired about symptoms suggestive of vasovagal responses (such as dizziness or faintness) and participants' general emotional state. Flexible recovery time was granted in cases of apparent distress or faintness. In one patient who showed extreme distress and signs of faintness following recovery measurements, the laboratory chair was tilted into the horizontal position and legs were elevated above the level of the head by cushions.

Data Analysis

Three-way repeated-measures Analyses of Variance (ANOVAs) with 3 groups (asthma, blood phobia, control) as the between-individual variable and 2 instructions (viewing only vs. film and tension), and 5 film categories (unpleasant, neutral, pleasant, surgery, and asthma) as within-individual variables were calculated with absolute values of physiological measures as dependent variables. Greenhouse-Geisser corrected degrees of freedom were used when appropriate. For post-hoc comparison of means, the Tukey HSD test was used in all ANOVAs (significance level $p < .05$). For comparing individual film effects within groups, these tests were performed for the average instruction condition effect, whenever no Instruction by Film or Instruction by Film by Group effects were found.

The analytic strategy largely followed steps related to the major questions outlined in the introduction:

1. Initially, we explored differences in physiological parameters between the three groups under neutral film viewing only conditions using one-way ANOVAs followed by post-hoc tests. We expected no group differences for most parameters, except for higher R_{OS} values and potentially elevated ventilation in asthma.
2. We then tested for arousal effects by quadratic trend across negative (average of unpleasant, surgery, and asthma films) vs. neutral vs. positive films. We expected higher R_{OS} , arousal, and SCL for negative and positive vs. neutral films.
3. Next, we tested for specific responding of asthma patients to BII-relevant material, expecting a Film by Group interaction with evidence for stronger R_{OS} responses for surgery films in asthma patients compared to the other groups and compared to other films within asthma patients. For the latter, we calculated a priori contrasts comparing the surgery film with the average of all other films (across conditions) within each group. Under the specificity assumption, this contrast would be significant only in asthma patients. Additional post-hoc comparisons were made between the surgery and negative films, which controlled for simple effects of elevated negative affect (which is well-known to increase R_{OS} , Ritz, 2004), as well as the asthma-relevant film to control for disease-relevance of the material.

Supplementary analysis was also planned to include calculation of effect sizes (g) for surgery film R_{OS} changes (relative to neutral films and an initial 3-min R_{OS} baseline), dependency of R_{OS} change on initial values, and comparison of R_{OS} changes with absolute thresholds for just noticeable obstruction in added resistive load studies (asthma: $0.076 \text{ kPa} \cdot \text{l}^{-1} \cdot \text{s}$, BII phobia and healthy controls: $0.061 \text{ kPa} \cdot \text{l}^{-1} \cdot \text{s}$). These criteria were derived from a review of added load studies and were medians of the thresholds found across these studies (Dahme et al., 1996). Such analysis was informative for learning more about airway responses to BII stimuli and their potential clinical relevance.

4. To explore similarities in response patterns with R_{OS} , additional analyses of other autonomic, respiratory, and self-report parameters (beyond those analyzed in 2)

followed within the three-way ANOVA design. No specific hypotheses were held except for HR_{minimum} , which was expected to be particularly low in BII phobia patients during surgery films. All other means comparisons were post-hoc. To reduce the number of comparisons for rating scales of emotion and symptoms, we restricted post-hoc tests to differences between the BII-relevant, negative, and asthma-relevant films.

5. We then explored effects of muscle tension on R_{OS} and other parameters, expecting significant Instruction effects as evidence of general bronchodilation, as well as Instruction by Film effects, which would have indicated particular effectiveness in reducing R_{OS} responses to the surgery film.
6. An additional set of three-way repeated-measures ANOVAs was calculated to explore whether film effects on R_{OS} and other parameters lasted into the recovery period. This would potentially inform about more tonic effects of airway constriction that may have greater disease relevance than short-lived changes in activation (Levenson, 1979).
7. We then explored associations of airway responses with other physiological and psychological parameters, between-individual correlations (Spearman's *Rho*, two-tailed) were calculated between R_{OS} changes on the one hand, and ventilatory (RR , V_T , V'_E , PCO_2 , sighs), autonomic (HR , HR_{minimum} , SBP , DBP , SCL), emotion and symptom changes on the other hand (difference scores calculated for negative, positive, surgery, and asthma films, each minus neutral film). To reduce the possibility of Type 1 error, patterns of correlation were interpreted only when one physiological or psychological parameter yielded 2 or more significant coefficients (out of a possible total of 24 coefficients: 3 groups x 2 instructions x 4 film change scores). In addition, for individual participants who showed marked distress and autonomic changes suggestive of vasovagal fainting in response to surgery films, we planned to conduct exploratory within-individual correlation analyses for concurrent and prospective (lag 1) associations. The focus was on associations of R_{OS} with ventilatory and autonomic parameters across successive 10-s averages during surgery film presentation and recovery. We also calculated within-individual multiple regression analyses controlling for the R_{OS} lag 1 autocorrelation, which provide a conservative estimate of such within-individual associations.

Results

Physiological and psychological characteristics before the experiment and during the neutral condition

Analysis of the MFS yielded a significant overall group effect, Wilks lambda = .234, $F(10,58)=6.19$, $p<.001$, $p\eta^2=.588$, with univariate tests showing significance for four of the five subscales (excluding Examinations and Symptoms), $F(2,33)=6.70-36.32$, $ps=.004-.001$, $p\eta^2s=.289-.688$. Post-hoc comparisons showed higher values in BII phobia patients than in asthmatic and healthy participants, the latter two not being significantly different from each other.

Analysis of neutral film viewing only as baseline showed significantly higher R_{OS} in asthma patients than the other groups, $F(2,37)=6.12$, $p=.005$ (Table 1), which is compatible with the airway disease activity in this group. In addition, they showed a significantly lower PCO_2 level than the BII patients, $F(2,37)=4.24$, $p=.002$, with healthy controls taking an intermediary position. None of the other physiological or psychological parameters distinguished the three groups during the neutral viewing condition.

A three-minute baseline measurements preceding the film viewing protocol was also available for R_{OS} . Two-way repeated measures ANOVA showed increases from baseline to neutral film R_{OS} , $F(1,38)=4.76$, $p=.035$, $\eta^2=.111$. Values of R_{OS} increased during neutral film for asthma by 6.9% (range -25–38%) and for BII phobia by 4.9 % (range -14–21%), whereas for controls they decreased by 1.2% (range -12–11%).

Overall arousal effects

As expected, nonspecific arousal effects were demonstrated by significant quadratic trend tests with higher values during the average of negative and positive films compared to the neutral films for R_{OS} ($F(1,38)=11.74$, $p<.001$) (Figure 1, upper panel), SCL ($F(1,37)=6.17$, $p=.018$), and arousal ratings ($F(1,38)=53.37$, $p<.001$).

For arousal ratings, an additional interaction of Film by Group was found, $F(8,148)=6.03$, $p<.001$, $\epsilon=.84$, $\eta^2=.246$; post-hoc tests indicated higher values for surgery films compared to asthma-relevant and negative films (as well as all other films) in BII phobia patients (Figure 2, lower panel). For other groups, no difference between disease-relevant and negative films was found.

Specificity of airway response to BII stimuli in asthma

Surgery films had the strongest effect on R_{OS} ; however, this effect was not specific to participants with asthma as the overall Film by Group interaction was not significant (Table 1). Within all groups, surgery film R_{OS} was significantly higher than the average of all other films, $F(1,38)=13.00$, 26.41, and 9.54, $p=.004$, .001, and .015, for asthma, BII phobia, and controls, respectively. In addition, post-hoc tests showed higher values for the surgery film compared to asthma-relevant and negative films in all groups.

Supplementary analysis are shown in Table 2, with difference scores and percentage change, effect sizes relative to neutral films or initial baseline, as well as the number of participants exceeding typical thresholds for just noticeable differences. On average, R_{OS} increased for surgery films compared to neutral films in asthma by 12.5%, in BII phobia by 21.5%, and in controls by 9.5%. Effect sizes generally were large for BII phobia, small to medium for asthma, and small for healthy controls. Relative to baseline, differences in average R_{OS} increase to both surgery films was significantly stronger in asthma and BII phobia patients compared to healthy controls, but relative to the neutral film R_{OS} increases were only significantly different between BII phobia patients and healthy controls, with asthmatics taking an intermediate position (one-way ANOVAs, $F(2,38)=4.29$ and 3.62., $p=.021$ and .037, followed by post-hoc tests). Correlations of R_{OS} change scores with R_{OS} baseline between and within groups were generally low for all sequences viewing only and viewing with tension, the number of significant associations not exceeding chance level.

Additional film effects on ventilation, autonomic parameters, and self report of emotion and symptoms

Ventilation—RR yielded a significant film effect (Table 1), with the post-hoc testing indicating higher values for positive films compared to all other films. For V_T , V'_E , and PCO_2 , the Film by Group interactions were significant, while for sighs it just missed significance. These interactions mostly reflected strong increases in ventilation and sighing and a reduction in PCO_2 during surgery films in BII phobia patients, as demonstrated by post-hoc tests that showed significantly higher V_T and V'_E , more sighing, and lower PCO_2 for BII patients during surgery films than during any other films or during surgery films in other groups.

Cardiovascular activity—Mean HR yielded a significant Film by Group interaction, with post-hoc tests showing higher HR in the BII phobia group during surgery as compared to all other films (Table 1). Similarly, HR_{minimum} showed a significant Film by Group interaction., the a priori contrast test for the surgery film against all other films was significant in both BII phobia patients and healthy controls, $F(1,37)=7.35$ and 4.15 , $p=.010$ and $.049$, in the former, due to higher values and in the latter due to lower values for the surgery film. For SBP, the Film by Group effect was only borderline significant.

Additional effects on self-report of emotion and symptoms—Pleasantness yielded a strong effect for Films, $F(4,144)=46.13$, $p<.001$, $e=.66$, $p\eta^2=.562$, with a linear increase in pleasantness from negative to neutral to positive films, linear trend $F(1,35)=164.0$, $p<.001$. (Figure 2, upper panel). In addition, the interaction of Film by Group was significant, $F(8,144)=3.98$, $p<.001$, $e=.86$, $p\eta^2=.181$. Post-hoc tests did not show differences between negative, surgery and asthma-related films, but blood phobia patients showed lower values in pleasantness during surgery films compared to other films.

Both shortness of breath and chest tightness showed significant Film effects, $F(4,148)=19.40$ and 9.19 , $ps<.001$, $e=.71$ and $.64$, $p\eta^2=.344$ and $.199$, respectively, as well as Film by Group interactions, $F(8,148)=9.92$ and 3.80 , $p<.001$ and $p=.003$, $e=.71$ and $.64$, $p\eta^2=.349$ and $.170$, respectively. This was due to higher ratings of these symptoms for surgery films than any other film, whereas no differences between disease-relevant and negative films were found for the other groups.

Effects of leg muscle tension

Leg EMG was significantly elevated during films with instructions to tense leg muscles (Table 3). Leg muscle tension did not change R_{os} substantially. RR, V_T and V'_{min} were higher during viewing with leg muscle tension than during viewing only. Also, HR and HR_{minimum} , SBP, DBP, and SCL were higher during leg muscle tension. For arousal ratings, a significant Instruction by Group effect, $F(1,37)=3.29$, $p=.048$, $p\eta^2=.151$, was due to higher values during leg muscle tension in blood phobia patients and controls but lower values in asthma patients. None of the parameters yielded a three-way interaction of Instruction by Film by Group.

Replication of film and instruction effects during recovery

During 1-min recovery, R_{OS} was still higher for the surgery film than any other film for all three groups (Figure 1, lower panel), Film effect $F(4,152)=8.19$, $p<.001$, $\epsilon=.81$, $p\eta^2=.177$, all post-hoc tests were significant. V_T , V'_E , and PCO_2 still yielded significant Group by Film interactions, $F(8,152)=6.96$, 2.34 , and 7.49 , $p<.001$, $.049$ and $.001$, $\epsilon=.58$, $.61$, and $.57$, $p\eta^2=.146$, $.110$, and $.294$, which were mainly due to continued hyperventilation in BII phobia patients, with post-hoc tests showing differences between surgery and most other films in BII phobia or surgery films in other groups. Similarly, post-hoc tests showed that mean HR was still elevated in BII phobia patients during recovery from surgery films, Film by Group effect $F(8,148)=2.63$, $p=.010$, $\epsilon=.84$, $p\eta^2=.124$, as was SBP, Film by Group effect $F(8,140)=2.79$, $p=.016$, $\epsilon=.72$, $p\eta^2=.137$.

Instruction effects were still seen in recovery for HR and SCL, $F(1,37)=10.45$ and 5.95 , $p=.003$ and $.020$, $p\eta^2=.220$ and $.203$, as well as V_T ($F(1,38)=6.37$, $p=.016$, $p\eta^2=.144$), V'_E ($F(1,38)=12.24$, $p<.001$, $p\eta^2=.203$), and DBP ($F(1,35)=8.91$, $p=.005$, $p\eta^2=.203$), with higher values for the viewing with leg muscle tension condition.

Autonomic, ventilatory, and self-report correlates of airway responses

Between-individual correlations for R_{OS} with other parameters—Airway responses (R_{OS} change for emotional or disease-relevant minus neutral film) were positively associated with V'_E for blood phobia patients during the surgery film with tension, $r(12)=.74$, $p=.006$, and during the asthma film viewing only, $r(12)=.68$, $p=.015$. R_{OS} change was also positively associated with change in SCL for asthma patients during negative films both during viewing only and viewing with tension, $r(14)=.64$ and $.56$, $p=.010$ and $.030$, respectively, as well as for the positive film viewing only, $r(14)=.65$, $p=.009$. For BII phobia patients, SCL change was positively associated with R_{OS} change for the surgery film viewing only, $r(12)=.64$, $p=.026$, but negatively associated during the asthma film with tension, $r(12)=-.62$, $p=.003$. Overall, the number of significant and almost significant ($p<.10$) coefficients for physiological parameters was 6% and 8% of the total number of calculated coefficients.

For correlations of changes in dimensional mood and symptom ratings with R_{OS} changes, only one coefficient was significant across all three groups and all films, which may be due to chance.

Oscillatory resistance during vasovagal episodes: within-individual associations for two cases

—One female BII phobia participant showed massive signs of distress and nausea during the surgery film with tension. Her HR increased and fluctuated markedly from initially 96 to 115b/min after 80s into the film, while her SBP showed an increase from 128 to 150mmHg after 60s into the film, fluctuated until 110s, and then gradually fell again (Figure 3). The experimenter terminated the surgery film presentation after 170s. Following recovery, she reported feeling faint and was brought into the horizontal position. R_{OS} increased markedly during the film from its initial lowest level at $0.345\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ to $0.573\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ (66% increase), with both V_T and RR varying between very high and low values.

One healthy male participant showed a steady drop in SBP and fluctuations in HR during the surgery films (viewing only), which continued into the recovery (HR from 61 to 48bpm, SBP from 123 to 94mmHg) (Figure 4). He subsequently reported having been close to fainting. R_{OS} increased gradually from the lowest initial level of $0.241\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ to $0.382\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ (59% increase) in the first half of the film and then dropped back to initial levels. At the same time, V_T increased massively and RR dropped. At the end of the recovery, R_{OS} was again markedly increased to $0.473\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ (96% increase).

Table 4 shows within-individual Pearson correlations across successive 10-s intervals for both participants. Negative concurrent associations of V_T and SCL with R_{OS} and a negative prospective association of HR lag 1 with R_{OS} were found for the healthy participant. In within-individual multiple regression analyses controlling for the R_{OS} lag 1 autocorrelation these concurrent and prospective associations were still significant. For the BII phobia patient, R_{OS} was concurrently associated with HR and SBP and prospectively with DBP. These associations did not remain significant when accounting for the R_{OS} lag 1 autocorrelation.

Discussion

Bronchoconstriction in response to blood and injury stimuli

In this study, we found that the airways responded particularly strongly to film presentation of blood and injury stimuli. Surgery films also elicited stronger responses than asthma-related film sequences in asthma patients and were the only film category that elicited resistance increases, which lasted well into the 1-min recovery period. However, the exaggerated airway responses were not limited to asthma patients. BII patients, who showed strong emotional arousal to the surgery films, showed similarly strong airway responses. This observation extends our earlier findings, which had only suggested a strong response of asthma patients to BII-related stimuli (Ritz, Steptoe, et al., 2000). In fact, expressed in overall percent from neutral film presentation levels, BII phobia patients exhibited the strongest increase ($>20\%$) in R_{OS} . We also found particularly strong increases in two individual participants (one of which was a healthy control) who were close to fainting during one of the surgery films. In both cases, the time course of R_{OS} showed a gradual increase from initial levels that reached a high plateau (approximately 60% increase from initial values) 70 to 120s into the film. These findings add to earlier speculations about potential similarities in autonomic regulation between asthma and blood phobia (Graham et al., 1961; Knapp & Nemetz, 1960; Lehrer et al., 1993). Our third group, healthy controls, also showed stronger R_{OS} increases to the surgery films than to any other film category, suggesting a nonspecific responding of the airways to this type of stimulus.

Self-report only partly reflected the findings in R_{OS} : although BII patients rated unpleasantness and arousal significantly higher for the surgery film than for other films, this was not the case for the asthma patients and healthy controls. Thus, BII-related material is a particularly potent bronchoconstrictor, which is not necessarily reflected in elevated individual distress levels. However, when self-reported or behavioral distress (fainting behavior) is specifically high with this type of stimulus, as in our BII phobia group, airway constriction is potentiated.

In this study, we were not able to determine the contribution of a hyperresponsiveness of the airways to cholinergic agents in asthma (Barnes, 1986). However, our findings of equally strong airway responses in BII phobia patients with healthy airways suggests that vagal excitation rather than sensitivity at the end-organ level determined the outcome in R_{OS} . Alternatively, vagal excitation may have dominated in BII phobia patients whereas airway hyperreactivity may have been the factor in asthma patients. The former interpretation is supported by another recent study in which we did not find an association between airway hyperresponsiveness to methacholine and airway constriction to the same BII stimuli in asthmatic individuals, but were able to attenuate the airway response by cholinergic blockade with ipratropium bromide (Ritz et al., 2010).

The present study is unique in demonstrating that a particular type of stimulus material elicits direct, immediate, strong, and at least 1-min long effects on airway obstruction, a major clinical end-point in asthma diagnosis. The effect sizes of airway responses to the surgery films were comparable or greater than those observed in studies using the forced oscillation technique of varying stimulus materials (Levenson et al., 1979; McQuaid et al., 2000; Ritz, 2004). Compared to the typical absolute threshold for the perception of airway obstruction from added resistive load studies (Dahme, Richter & Maß, 1996), 33% of our asthma patients, 58% of BII phobia patients, and 14% of healthy controls experienced resistance increases during the average surgery film that were of potential clinical relevance. These numbers were even larger when the pre-experimental baseline, rather than the neutral film condition was taken as a reference (53, 67, and 21%, respectively). (Footnote 1) Such resistance changes in the range of just noticeable differences did not necessarily translate into typical asthma symptoms, such as shortness of breath in our study. However, in the daily life of symptomatic asthma patients, such effects could aggravate bronchoconstriction elicited by other asthma triggers and bring these patients closer to airway obstruction levels typical for asthma exacerbations. Patients comorbid with asthma and BII phobia might be at a particular risk, especially considering the BII-related stimuli impact on airways that are already precontracted by other triggers. Although there is evidence for an elevated prevalence of psychiatric comorbidity, including anxiety disorders, in asthma and an associated greater health care burden (Goodwin, 2003), direct effects of specific anxiety disorders on the airway response to thematically relevant stress have rarely been explored.

Autonomic and ventilatory correlates of airway responses

A multitude of psychosocial pathways to asthma have been considered to date, including direct effects on airway pathophysiology through the autonomic nervous system regulation (e.g. Isenberg et al., 1992; Lehrer et al., 1996; Ritz et al., 2000), ventilation (e.g. Clarke, 1982; Ritz et al., 2008), inflammatory processes (e.g., Joachim et al., 2003; Kullowatz et al., 2008; Liu et al., 2002), endocrine pathways (Buske-Kirschbaum et al., 2003; Wamboldt, Laudenslager, Wamboldt, Kelsay, & Hewitt, 2003), modification of receptor gene expression

¹Whereas the comparison of an emotional with the neutral films provided the effect of the emotion minus the nonspecific effect of film viewing, the comparison with baseline informed about the total effect of viewing emotional films relative to a state of relaxed inactivity. Thus, researchers studying the differential impact of specific emotional states on the airways would find the comparison with the neutral film most relevant, whereas those interested in the total effect of a daily life activity such as watching an emotional film would find the comparison with the baseline most relevant.

(Miller & Chen, 2006), and upper respiratory tract infection (Wright, Rodriguez, & Cohen, 1998), as well as indirect effects through asthma management (e.g. Feldman et al., 2005; Kaugars, Klinnert, & Bender, 2004). These pathways vary greatly with respect to the timing of their impact on asthma-relevant outcome measures. In the present study, we explored a number of ventilatory and autonomic parameters that might have provided clues to mechanisms involved in the fast-onset airway responses observed. In general, R_{OS} increases followed an arousal pattern similar to SCL and V'_E , with emotional and disease-relevant films significantly exceeding neutral film levels. This is in line with prior studies that demonstrated arousal modulation of respiratory resistance increases (e.g. Ritz, George, et al., 2000; Ritz, Steptoe, et al., 2000; von Leupoldt & Dahme, 2005), V'_E (Gomez, Zimmermann, Guttormsen-Schär, & Danuser, 2005) and skin conductance (e.g. Lang et al., 1993; Winton, Putnam & Krauss, 1984). V'_E and SCL were also found to be associated consistently with R_{OS} changes in correlational analyses, particularly in asthma and BII phobia patients who responded with stronger bronchoconstriction. Participants showing stronger overall R_{OS} increases also showed significantly stronger increases in SCL and V'_E in more than one film presentation. Despite the consistencies, the sign of the associations varied across between- and within-individual correlations. In particular, within-individual correlations also yielded negative associations of SCL and V'_E with R_{OS} .

Whereas a positive association between R_{OS} and V'_E could be interpreted as coupling between respiratory drive and resistance (Baker & Don, 1988) or increases in ventilation affecting the airways negatively (and thus increasing R_{OS} , by drying, cooling, or irritation), it is more difficult to conceptualize the association between R_{OS} and SCL in terms of underlying physiological mechanisms. While common cholinergic neurotransmission and a potential alteration of this system by allergic processes could be invoked as an explanation (Kaliner, 1976; Lehrer et al., 1996; Marshall, 1989), the seemingly divergent directions of autonomic activation may simply be another instance of fractionation of response direction discussed previously by psychophysiologists (Lacey & Lacey, 1974). They could be part of an integrated pattern of response to environmental challenge not following the traditional view of reciprocity in sympathetic and parasympathetic activity, but may be of functional significance (e.g., defensive protection of the airways and skin). In any case, our findings encourage further exploration of the link between these two activation parameters at two different organ sites to improve our understanding of stress-related airway constriction. It is still possible that the positive between-individual associations of R_{OS} with SCL and V'_E were because the induced stress raised levels of all three through separate mechanisms.

Vagal excitation has long been thought to be the major mechanism of airway constriction to psychological stimuli (Boushey, 1981; Isenberg et al., 1992; Miller & Wood, 2003; Miller, Wood, Lim, Ballow & Hsu, 2009). Studies with pharmacological blockade have demonstrated involvement of the vagal pathway in airway obstruction when suggestions of bronchoconstriction were made to experimental participants (McFadden, Luparello, Lyons, & Bleeker, 1969) and more recently in our research, when film or picture stimuli were presented (Ritz et al., 2010). However, the present findings do not suggest a strong overall vagal excitation during surgery films, which would have been indexed by a pronounced bradycardia. Only healthy controls showed lower minimum HR during surgery films than all

other films. In none of the groups was participants' minimum HR related to their extent of airway obstruction. [Footnote 2]

At least three factors could have contributed to the lack of association between R_{OS} and HR. First, vagal components may indeed not have been very substantial at least in BII phobia and asthma patients during the 5-min surgery films. Only healthy controls showed significant decreases in minimum HR during the surgery films, whereas asthma patients showed only nonsignificant decreases. Longer exposure may be necessary to observe a more solid eventual transition into vagal excitation, in particular in BII phobia patients. These patients showed mainly pronounced SBP, DBP and HR increases during the surgery films similar to a classic sympatho-adrenergic anxiety response and some refused to continue viewing the surgery episode. It should also be noted that the role of the vagal component in BII phobic responses is increasingly being debated (Sarlo, Buodo, Munafò, Stegagno, & Palomba, 2008). Second, the vagal system may not act in a unitary fashion across organ systems. Vagal excitation may have constricted airways more than it lowered HR. Instances of organ-specific vagal activation have been reported in the literature and in our own studies, which compared R_{OS} changes with changes in indices of cardiac vagal control (for review, see Ritz, 2009). Third, dissociations between observable instances of vagal excitation at various organ sites could also result from differences in gain and time parameters in different branches of the vagal system or different end-organs. Vagal excitation elicited by our BII-relevant material may thus have translated into small cardiac decelerations and greater airway constrictions. Effects also may take longer to be manifested in the airways than in the heart, which would be consistent with the negative prospective within-individual correlation between HR and R_{OS} observed for the healthy control participant. Finally, if vagal and sympathetic co-activation had taken place, which is supported by SCL increases, the greater importance of vagal vs. sympathetic motor control of the airway smooth muscle (Barnes, 1986; Canning & Fischer, 2001) could also have resulted in stronger effects on R_{OS} than HR. In general, reciprocity of sympathetic and parasympathetic activation is increasingly considered as only one of many possible activation patterns of these systems (Berntson et al., 1991).

Overall, a working model of autonomic and ventilatory pathways to psychologically induced airway responses will require further exploration and refinement by future studies. Major open questions deal with the exact impact of sympathetic arousal indexed both by adrenergic and cholinergic postganglionic transmission (the latter explored here and in previous studies by skin conductance) as well as circulating catecholamine effects, which may also come into play in more extreme cases of emotional arousal. Further exploration of the vagal pathway is also indicated. A limitation of the current study was that we could not include an analysis of respiratory sinus arrhythmia for technical reasons. However, given the high degree of specificity in the vagal system (Ritz, 2009), pharmacological blockade of cholinergic airway receptors by anticholinergic inhalers would be a more promising strategy to explore airway-

²However, as shown in the prospective analyses for the one healthy participant close to fainting, higher HR at time 0 predicted lower R_{OS} at time 0+10 s. This lag association could be related to activation of the baroreflex, which has been associated with bronchodilation in animal studies (Nadel & Widdicombe, 1962; Schultz, Pissari, Coleridge, & Coleridge, 1987). Consistent with this finding, we observed in an earlier study (Ritz, Steptoe, et al., 2000) a negative association between an index of baroreflex sensitivity and respiratory resistance change in asthma patients during a mental arithmetic task.

specific vagal excitation (see e.g. Ritz et al., 2010). Finally, the impact of major ventilatory changes such as hyperventilation may be more visible in extreme emotional situations or stronger symptomatic episodes in asthma patients. This may be due to the influence of stronger ventilatory changes on airways that are hyperreactive to irritation, drying, cooling, or fall in PCO_2 (McFadden & Gibert, 1994; van den Elshout et al., 1991).

Effect of leg muscle tension on airway responses to films

In this study we had shown one set of five films under the instruction to tense leg muscles. Voluntary muscle contractions could theoretically be a simple and effective behavioral maneuver to counteract emotion-induced airway constriction by vagal withdrawal (Ritz, Dahme et al., 1998). Despite reasonable expectations, we did not observe any substantial influence of static leg muscle tension on R_{os} . On average, only BII phobia patients showed lower R_{os} values during viewing and tension instructions across all films. Perhaps increases in ventilation counteracted potential decreases in R_{os} . Typically, increase in ventilation have been linked to increases in resistance, but reflex bronchodilation to skeletal muscle tension, which also leads to ventilation increases, has been shown to override this relationship in studies with cats (Baker & Don, 1988). Given interspecies variations in airway regulation, it is conceivable that these opposing response directions cancel each other out in humans, at least under some conditions. Indeed, we were able to demonstrate bronchodilation in our static muscle tension study in all conditions, except for the arm muscle tension conditions in asthma patients, in which we also observed substantial increases in ventilation (Ritz, Dahme, et al., 1998). Another possibility could be the longer duration of static muscle tension. Other studies that have examined tonic effects of static arm, shoulder, or facial muscle tension over minutes found no changes in resistance or even increases on average (Lehrer, Generelli & Hochron, 1997; Ritz, Wiens & Dahme, 1998). Muscle tension levels may also have been too low or the muscle mass that was tensed too small to lead to sustained changes in R_{os} . Finally, dynamic rather than static exercise could yield stronger effects on the airways, given that exercise-induced bronchodilation has been demonstrated in humans almost exclusively using the former.

Limitations

Our study was also limited by the small sample size, which may have rendered some effects nonsignificant due to a lack of power. In addition to the muscle tension effects, this may have affected group differences in airway responses and associations of R_{os} with other variables. Another limitation could have been potential order effects. The fact that film presentations were randomized could have led to some nonspecific effects of tense expectation in BII phobia patients depending on the position of the feared stimulus in the film order: Although participants were not informed on the number of BII relevant films, late presentation of this material within the series of film clips could have led to more tension and thus elevated levels in some physiological parameters, such as blood pressure.

Variations in length of our films could have been another limitation, however, this limitation is shared with most film studies of emotion induction (Rottenberg, Ray & Gross, 2007). In addition, pre-mature termination of the surgery films by some of our BII phobia patients also contributed to variations in film length. Given the lack of consensus among theorists about

the duration of an emotion and probable variation between individual emotional states in parameters of activation such as latency, peak, duration, and rise time, the averaging across a certain period of time is a pragmatic approach. A more promising method for future studies may involve continuous tracking of particular aspects of emotional experience of participants using manual tracking devices (e.g. Levenson, 1988) and off-line extraction key scenes across channels.

The arousal matching of film clips was also not optimal. In BII phobia patients and healthy controls, the arousal values for the BII relevant film exceeded those of most other films. The high surgery film R_{OS} values in both groups could thus be a function of the higher arousal value, rather than being a BII content specific effect. On the other hand, in asthma patients, levels of arousal were comparable between all emotional films, including the surgery film, yet R_{OS} values were again higher for surgery than for all other films.

Finally, our 1-min recovery periods following film presentations were relatively short. Longer recovery periods would have allowed for a more detailed exploration of how emotion-induced airway responses resolve following stimulus presentation. However, in planning the study we sought to balance gain in information with potential adverse effects of boredom on physiology in those participants who were less affected by the films. The short duration of the recovery most likely did not lead to any notable carry-over effects following films. After each 1-min recovery recording, participants filled in the emotions and symptoms rating sheet and briefly interacted with the experimenter who typically explored whether they felt okay, were relaxed, and were ready to continue with the next film. Participants with BII phobia were given extra time to recover after the surgery film ratings

Conclusion

We have shown that BII stimuli are particularly potent in eliciting airway responses in asthma patients, BII phobia patients, and healthy controls. This stimulus material is more potent than material with general positive or negative emotional valences or with asthma-relevant themes depicting asthma attacks. In general, a heightened sensitivity to BII stimuli leads to particularly strong airway constriction, as seen in our BII patient group and in two participants who were close to fainting. Excitation of the sudomotor system seems to have a particularly tight association with airway regulation during emotional stimulation. Further research on autonomic and ventilatory mechanisms of airway constriction to stress, in particular BII-related distress, and potential behavioral countermeasures against ensuing airway obstructions is indicated.

Acknowledgements

This study was supported by the German Research Society (DFG Ri 957/2–1), the National Institute of Mental Health (MH56094), the Palo Alto Research Institute (PAIRE), and the Department of Veterans Affairs. We thank David Rosenfield and Bernhard Dahme for helpful comments, Tana Bliss, Mark Rothkopf, and Alysha Khavarian for their assistance in recruitment and data collection, and Antje Kullowatz, Nino Wessolowski, Chris Burrows, and Barnes McKenzie for their help in data reduction procedures.

List of abbreviations

ANOVA	analysis of variance
-------	----------------------

BII	blood-injection-injury
DBP	diastolic blood pressure
EMG	electromyogram
HR	heart rate
MANOVA	multivariate analysis of variance
NHLBI	National Heart, Lung, and Blood Institute
PCO₂	partial pressure of carbon dioxide
Ros	oscillatory resistance
RR	respiration rate
SBP	systolic blood pressure
V'E	minute ventilation
VT	tidal volume
SCL	skin conductance level

References

- Bagby RM, Parker JDA, & Taylor GJ (1994). The twenty-item Toronto Alexithymia Scale - I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, 38, 23–32. [PubMed: 8126686]
- Barnes PJ (1986). Neural control of human airways in health and disease. *American Review of Respiratory Disease*, 134, 1289–1314. [PubMed: 3538958]
- Baker DG & Don H (1988). Reversal of relation between respiratory drive and airway tone in cats. *Respiration Physiology*, 73, 21–30. [PubMed: 3175355]
- Basmajian JV, & Blumenstein R (1982). Electrode placement in electromyographic biofeedback In Basmajian JV (Ed.), *Biofeedback. Principles and practice for clinicians* (3rd ed.) (pp. 369–382). Baltimore: Williams & Wilkins.
- Beck KC, Offord KP & Scanlon PD (1994). Bronchoconstriction occurring during exercise in asthmatic subjects. *American Journal of Respiratory and Critical Care Medicine*, 149, 352–357. [PubMed: 8306029]
- Berntson GG, Bigger JT, Eckberg DL, Grossman P, Kaufmann PG, Malik M, Nagaraja HN, Porges SW, Saul JP, Stone PH, & Van der Molen MW (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, 34, 623–648. [PubMed: 9401419]
- Berntson GG, Cacioppo JT, & Quigley KS (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review*, 98, 459–487. [PubMed: 1660159]
- Boushey HA (1981). Neural mechanisms in asthma In Weiner H, Hofer MA, & Stunkard AJ (Eds), *Brain, behavior, and bodily disease* (pp. 27–44). New York, NY: Raven Press.
- Buske-Kirschbaum A, von Auer K, Krieger S, Weis S, Rauh W, & Hellhammer D (2003). Blunted cortisol responses to psychosocial stress in asthmatic children: a general feature of atopic disease? *Psychosomatic Medicine*, 65, 806–810. [PubMed: 14508024]
- Canning BJ & Fischer A (2001). Neural regulation of airway smooth muscle tone. *Respiration Physiology*, 125, 113–127. [PubMed: 11240156]

- Carruthers M & Taggart P (1973). Vagotonicity of violence: Biochemical and cardiac response to violent films and television programmes. *British Medical Journal*, 3, 384–389. [PubMed: 4730188]
- Clarke PS (1982). Emotional exacerbation in asthma caused by overbreathing. *Journal of Asthma*, 19, 249–251. [PubMed: 7174601]
- Dahme B, Richter R & Maß R (1996). Interoception of respiratory resistance in asthmatic patients. *Biological Psychology*, 42, 215–229. [PubMed: 8770380]
- Engel GL (1978). Psychological stress, vasodepressor (vasovagal) syncope, and sudden death. *Annals of Internal Medicine*, 89, 403–412. [PubMed: 99068]
- Feldman JM, Siddique MI, Morales E, Kaminski B, Lu SE, & Lehrer PM (2005). Psychiatric disorders and asthma outcomes among high-risk inner-city patients. *Psychosomatic Medicine*, 67, 989–996. [PubMed: 16314605]
- First MB, Spitzer RL, Gibbon M, & Williams JBW (1994) Structured clinical interview of DSM IV: Patient edition (SCID-I/P), Version 2.0. New York, NY: Biometrics Research Department.
- Freed AN (1995). Models and mechanisms of exercise-induced asthma. *European Respiratory Journal*, 8, 1770–1785. [PubMed: 8586137]
- Gardner W (1994). Measurement of end-tidal pCO₂ and pO₂. *Biofeedback and Self-Regulation*, 19, 103–113. [PubMed: 7918749]
- Gerlach AL, Spellmeyer G, Vögele C, Huster R, Stevens S, Hetzel G, & Deckert J (2006). Blood-injury phobia with and without a history of fainting: disgust sensitivity does not explain the fainting response. *Psychosomatic Medicine*, 68, 331–339. [PubMed: 16554401]
- Gomez P, Zimmermann P, Guttormsen-Schär S, & Danuser B (2005). Respiratory responses associated with affective processing of film stimuli. *Biological Psychology*, 68, 223–35. [PubMed: 15620792]
- Goodwin RD (2003). Asthma and anxiety disorders. *Advances in Psychosomatic Medicine*, 24, 51–71. [PubMed: 14584347]
- Graham DT, Kabler JD, & Lunsford L (1961). Vasovagal fainting: A diphasic response. *Psychosomatic Medicine*, 23, 493–507. [PubMed: 13901013]
- Gross JJ, & Levenson RW (1995). Emotion elicitation using films. *Cognition and Emotion*, 9, 87–108.
- Hodes RL, Cook EW III, Lang PJ (1985). Individual differences in autonomic response: Conditioned association or conditioned fear? *Psychophysiology*, 22, 545–557. [PubMed: 4048355]
- Isenberg SA, Lehrer PM, & Hochron S (1992). The effects of suggestion and emotional arousal on pulmonary function in asthma: A review and a hypothesis regarding vagal mediation. *Psychosomatic Medicine*, 54, 192–216. [PubMed: 1565756]
- Joachim RA, Quarcoo D, Arck P, Herz U, Renz H, & Klapp B (2003). Stress enhances airway reactivity and airway inflammation in an animal model of allergic bronchial asthma. *Psychosomatic Medicine*, 65, 811–815. [PubMed: 14508025]
- Kagawa J & Kerr HD (1970). Effects of brief graded exercise on specific airway conductance in normal subjects. *Journal of Applied Physiology*, 28, 138–144. [PubMed: 5413299]
- Kaufman MP, Rybicki KJ & Mitchell JH (1985). Hindlimb muscular contraction reflexly decreases total pulmonary resistance in dogs. *Journal of Applied Physiology*, 59, 1521–1526. [PubMed: 4066582]
- Kaugars AS, Klinnert MD, Bender BG (2004). Family influences on pediatric asthma. *Journal of Pediatric Psychology*, 29, 475–491 [PubMed: 15347697]
- Kleinknecht RA, Thorndike RM, Walls MM (1995). Factorial dimensions and correlates of blood, injury, injection and related medical fears: Cross validation of the Medical Fears Survey. *Behavior Research and Therapy*, 34, 323–331.
- Knapp PH & Nemetz SJ (1960). Acute bronchial asthma. I. Concomitant depression and excitement, and varied antecedent patterns in 406 attacks. *Psychosomatic Medicine*, 22, 42–56. [PubMed: 14410060]
- Kozak MJ, & Miller GA (1985). The psychophysiological process of therapy in a case of injury-scene-elicited fainting. *Journal of Behavior Therapy and Experimental Psychiatry*, 16, 139–145. [PubMed: 4044863]

- Kullowatz A, Rosenfield D, Dahme B, Magnussen H, Kannies F, Ritz T (2008). Effects of stress on lung function in asthma are mediated by changes in airway inflammation. *Psychosomatic Medicine*, 70, 468–475. [PubMed: 18480192]
- Lacey BC & Lacey JI (1974). Studies of heart rate and other bodily processes in sensorimotor behavior. In Obrist PA, Black AH, & Brener J (Eds.), *Cardiovascular psychophysiology* (pp. 538–564). Chicago: Aldine.
- Lang PJ, Greenwald MK, Bradley MM & Hamm AO (1993). Looking at pictures: Affective, facial, visceral, and behavioral reactions. *Psychophysiology*, 30, 261–273. [PubMed: 8497555]
- Larsen RJ & Diener E (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, 21, 1–39.
- Lehrer PM, Hochron S, Carr R, Edelberg R, Hamer R, Jackson A, & Porges S (1996). Behavioral task-induced bronchodilation in asthma during active and passive tasks: A possible cholinergic link to psychologically induced airway changes. *Psychosomatic Medicine*, 58, 413–422. [PubMed: 8902893]
- Lehrer PM, Isenberg S, & Hochron SM (1993). Asthma and emotion: a review. *Journal of Asthma*, 30, 5–21. [PubMed: 8428858]
- Levenson RW (1979). Effects of thematically relevant and general stressors on specificity of responding in asthmatic and nonasthmatic subjects. *Psychosomatic Medicine*, 41, 28–39. [PubMed: 432361]
- Levenson RW (1988). Emotion and the autonomic nervous system: a prospectus for research on autonomic specificity. In Wagner HL (Ed.), *Social Psychophysiology and emotion: Theory and clinical application*. (pp. 17–42). Oxford: Wiley.
- Lewis SE, Snell PG, Taylor WF, Hamra M, Graham RM, Pettinger WA & Blomquist CG (1985). Role of muscle mass and mode of contraction in circulatory responses to exercise. *Journal of Applied Physiology*, 58, 146–151. [PubMed: 3968005]
- Liangas G, Morton JR & Henry RL (2003). Mirth-triggered asthma: is laughter really the best medicine? *Pediatric Pulmonology*, 36, 107–112. [PubMed: 12833489]
- Liu LY, Coe CL, Swenson CA, Kelly EA, Kita H, & Busse WW (2002). School examinations enhance airway inflammation to antigen challenge. *American Journal of Respiratory and Critical Care Medicine*, 165, 1062–1067. [PubMed: 11956045]
- Longhurst JC (1984). Static contraction of hindlimb muscles in cats reflexly relaxes tracheal smooth muscle. *Journal of Applied Physiology*, 57, 380–387. [PubMed: 6469808]
- Mansfield L, McDonnell J, Morgan W, & Souhrada JF (1979). Airway response in asthmatic children during and after exercise. *Respiration*, 38, 135–143. [PubMed: 515538]
- Marshall P (1989). Attention deficit disorder and allergy: a neurochemical model of the relation between the illnesses. *Psychological Bulletin*, 106, 434–446. [PubMed: 2682719]
- McFadden ER Jr, & Gilbert IA (1994). Exercise-induced asthma. *New England Journal of Medicine*, 330, 1362–1367. [PubMed: 8152449]
- McFadden ER, Luparello T, Lyons HA, Bleeker E (1969). The mechanism of action of suggestion in the induction of acute asthma attacks. *Psychosomatic Medicine*, 31, 134–143. [PubMed: 5784000]
- McQuaid EL, Fritz GK, Nassau JH, Lilly MK, Mansell A, & Klein RB (2000). Stress and airway resistance in children with asthma. *Journal of Psychosomatic Research*, 49, 239–245. [PubMed: 11119780]
- Miller BD, & Wood BL (2003) Emotions and family factors in childhood asthma: psychobiologic mechanisms and pathways of effect. *Advances in Psychosomatic Medicine*, 24, 131–160 [PubMed: 14584352]
- Miller BD, Wood BL, Lim J, Ballow M, & Hsu C (2009). Depressed children with asthma evidence increased airway resistance: “vagal bias” as a mechanism? *Journal of Allergy & Clinical Immunology*, 124, 66–73. [PubMed: 19523670]
- Miller GE & Chen E (2006). Life stress and diminished expression of genes encoding glucocorticoid receptor and beta2-adrenergic receptor in children with asthma. *Proceedings of the National Academy of Sciences USA*, 103, 5496–5501.

- Nadel JA & Widdicombe JG (1962). Effect of changes in blood gas tension and carotid sinus pressure on tracheal volume and total lung resistance to airflow. *Journal of Physiology*, 163, 13–33. [PubMed: 14477795]
- National Heart, Lung, and Blood Institute und World Health Organisation (2002). NHLBI/WHO workshop report: Global strategy for asthma management and prevention. NIH Publication 02–3659.
- Öst LG & Sterner U (1987). Applied tension. A specific behavioral method for treatment of blood phobia. *Behaviour Research and Therapy*, 25, 25–29. [PubMed: 3593159]
- Padrid PA, Haselton JR, & Kaufman MP (1990). Ischemia potentiates the reflex bronchodilation evoked by static muscular contraction in dogs. *Respiration Physiology*, 81, 51–62. [PubMed: 2218107]
- Ritz T (2004). Probing the psychophysiology of the airways. Physical activity, experienced emotion, and facially expressed emotion. *Psychophysiology*, 41, 809–821 [PubMed: 15563334]
- Ritz T (2009). Studying non-invasive indices of vagal control: the need for respiratory control and the problem of target specificity. *Biological Psychology*, 80, 158–168. [PubMed: 18775468]
- Ritz T, Dahme B, DuBois AB, Folgering H Fritz GK, Harver AR, Kotses H, Lehrer PM, Ring C, Steptoe A, & Van de Woestijne KP (2002). Guidelines for mechanical lung function measurements in psychophysiology. *Psychophysiology*, 39, 546–567. [PubMed: 12236321]
- Ritz T, Dahme B & Wagner C (1998). Effects of static forehead and forearm muscle tension on total respiratory resistance in healthy and asthmatic participants. *Psychophysiology*, 35, 549–562. [PubMed: 9715099]
- Ritz T, George C & Dahme B (2000). Respiratory resistance during emotional stimulation: Evidence for a nonspecific effect of emotional arousal? *Biological Psychology*, 52, 143–160. [PubMed: 10699353]
- Ritz T, & Kullowatz A (2005). Effects of stress and emotion on lung function in health and asthma. *Current Respiratory Medicine Reviews*, 1, 208–219.
- Ritz T, Kullowatz A, Bobb C, Dahme B, Kannies F, Magnussen H, & Steptoe A (2008) Psychological asthma triggers and symptoms of hyperventilation. *Annals of Allergy, Asthma, and Immunology*, 100, 426–432.
- Ritz T, Kullowatz A, Goldman GD, Smith H-J, Kannies F, Dahme B, & Magnussen H (2010). Airway response to emotional stimuli in asthma: The role of the cholinergic pathway. *Journal of Applied Physiology*, 108, 1542–1549. [PubMed: 20360438]
- Ritz T & Steptoe A (2000) Emotion and pulmonary function in asthma: Reactivity in the field and relationship with laboratory induction of emotion. *Psychosomatic Medicine*, 62, 808–815. [PubMed: 11139001]
- Ritz T, Steptoe A, Bobb C, Harris A, & Edwards M (2006). The Asthma Trigger Inventory: Development and evaluation of a questionnaire measuring perceived triggers of asthma. *Psychosomatic Medicine*, 68, 956–965. [PubMed: 17132841]
- Ritz T, Steptoe A, De Wilde S & Costa M (2000). Emotions and stress increase respiratory resistance in asthma. *Psychosomatic Medicine*, 62, 401–412. [PubMed: 10845354]
- Ritz T, Thöns M, Fahrenkrug S, & Dahme B (2005). The airways, respiration, and respiratory sinus arrhythmia during picture viewing. *Psychophysiology*, 42, 568–578. [PubMed: 16176379]
- Ritz T, Wiens S & Dahme B (1998). Stability of total respiratory resistance under multiple baseline conditions, isometric arm exercise, and voluntary deep breathing. *Biological Psychology*, 49, 187–213. [PubMed: 9792493]
- Ritz T, Wilhelm FH, Gerlach A, Kullowatz A, & Roth WT (2005). End-tidal pCO₂-levels in blood phobia during viewing of emotional and disease-relevant films. *Psychosomatic Medicine*, 67, 661–668. [PubMed: 16046385]
- Ritz T, Wilhelm FH, Meuret AE, Gerlach A & Roth WT (2009). Do blood phobia patients hyperventilate during exposure by breathing faster, deeper, or both? *Depression and Anxiety*, 26, E60–67. [PubMed: 19085969]
- Rottenberg J, Ray RR, & Gross JJ (2007). Emotion elicitation using films In Coan JA & Allen JJB (Eds.), *The handbook of emotion elicitation and assessment* (pp. 9–28). New York: Oxford University Press.

- Sarlo M, Buodo G, Munafò M, Stegagno L, & Palomba D (2008). Cardiovascular dynamics in blood phobia: evidence for a key role of sympathetic activity in vulnerability to syncope. *Psychophysiology*, 45, 1038–1045. [PubMed: 18823415]
- Schultz HD, Pisarri TE, Coleridge HM, & Coleridge JC (1987). Carotid sinus baroreceptors modulate tracheal smooth muscle tension in dogs. *Circulation Research*, 60, 337–345. [PubMed: 3581443]
- Scichilone N, Marchese R, Soresi S, Interrante A, Togias A, & Bellia V (2007). Deep inspiration-induced changes in lung volume decrease with severity of asthma. *Respiratory Medicine*, 101, 951–956. [PubMed: 17049828]
- van den Elshout FJJ, van Herwaarden CLA, Folgering HTM (1991). Effects of hypercapnia and hypocapnia on respiratory resistance in normal and asthmatic subjects. *Thorax*, 46, 28–32. [PubMed: 1908137]
- von Leupoldt A & Dahme B (2005). Emotions and airway resistance in health and asthma: Study with whole body plethysmography. *Psychophysiology*, 42, 92–97. [PubMed: 15720584]
- Wamboldt MZ, Laudenslager M, Wamboldt FS, Kelsay K, & Hewitt J (2003). Adolescents with atopic disorders have an attenuated cortisol response to laboratory stress. *Journal of Allergy and Clinical Immunology*, 111, 509–514. [PubMed: 12642830]
- Warren JB, Jennings SJ, & Clark TJH (1984). Effect of adrenergic and vagal blockade on normal human airway response to exercise. *Clinical Science*, 66, 79–85. [PubMed: 6228370]
- Wasserman K, Whipp BJ & Casaburi R (1986). Respiratory control during exercise In Cherniack NS & Widdicombe JG (Eds.), *Handbook of physiology, Sec.3. The respiratory system: Vol. 2. Control of breathing* (pp. 595–619). Bethesda, MD: American Physiological Society.
- Wilhelm FH, Trabert W, & Roth WT (2001). Characteristics of sighing in panic disorder. *Biological Psychiatry*, 49, 606–614. [PubMed: 11297718]
- Winton WM, Putnam LE, & Krauss RM (1984). Facial and autonomic manifestations of the dimensional structure of emotion. *Journal of Experimental Social Psychology*, 20, 195–216.
- Wright RJ, Rodriguez M, & Cohen S (1998). Review of psychosocial stress and asthma: an integrated biopsychosocial approach. *Thorax*, 53, 1066–1074. [PubMed: 10195081]

Figure 1.
 R_{os} during (upper panel) and following (lower panel; 1-min recovery) emotion and disease-relevant films

Figure 2.
Impact of emotion and disease-relevant films on experienced affective valence (upper panel)
and arousal (lower panel)

Figure 3.

Autonomic and respiratory response (in consecutive 10-s means) of the one healthy control who felt close to fainting during one surgery film (viewing only); upper panel: heart rate and blood pressure, lower panel respiratory resistance and breathing pattern; break with vertical line indicates beginning of recovery

Figure 4.

Autonomic and respiratory response (in consecutive 10-s means) of the one BII phobia patient who felt close to fainting during one surgery film (viewing with tension); upper panel: heart rate and blood pressure, lower panel respiratory resistance and breathing pattern; break with vertical line indicates beginning of recovery; break before that in R_{os} indicates data loss by repeated swallowing

Table 1.

Means, *SEs* (in parentheses) and ANOVA effects for film presentation in asthma patients ($n=14-15$), BII phobia patients ($n=11-12$) and healthy controls ($n=12-14$)

	Pleasant	Neutral	Unpleasant	Surgery	Asthma	Film effect	Group effect	Film x Group effect
R_{os} (kPa $\cdot$ l $\cdot$ s)								
Asthma	0.482 (.284)	0.470 (.303)	0.502 (.315)	0.522 (.328)	0.484 (.307)	$F(4,152)=24.59$, $p<.001$, $e=.66$, $\eta^2=.393$	$F(2,38)=7.29$, $p=.002$, $\eta^2=.277$	$F(8,152)=1.55$, $p=.178$, $e=.66$, $\eta^2=.075$
BII Phobia	0.327 (.318)	0.319 (.339)	0.344 (.352)	0.389 (.367)	0.324 (.343)			
Controls	0.342 (.294)	0.353 (.314)	0.353 (.326)	0.381 (.340)	0.341 (.318)			
RR (breaths/min)								
Asthma	16.3 (.75)	15.5 (.76)	17.6 (.85)	16.3 (.94)	15.5 (.80)	$F(4,152)=7.31$, $p<.001$, $e=.68$, $\eta^2=.161$	$F(2,38)=0.56$, $p=.946$, $\eta^2=.003$	$F(8,152)=1.08$, $p<.001$, $e=.68$, $\eta^2=.054$
BII Phobia	17.6 (.84)	16.0 (.85)	17.1 (.95)	16.8 (1.05)	17.2 (.90)			
Controls	16.0 (.78)	15.9 (.79)	17.6 (.88)	>15.7 (.97)	16.1 (.82)			
V_T (ml)								
Asthma	421.6 (65.0)	428.8 (60.2)	397.1 (58.5)	423.8 (76.9)	429.0 (65.4)	$F(4,152)=9.19$, $p<.001$, $e=.51$, $\eta^2=.195$	$F(2,38)=0.13$, $p=.883$, $\eta^2=.007$	$F(8,152)=5.58$, $p<.001$, $e=.51$, $\eta^2=.227$
BII Phobia	435.7 (72.7)	419.1 (67.3)	435.7 (65.5)	614.0 (86.0)	423.3 (73.2)			
Controls	472.0 (67.3)	442.3 (62.3)	437.6 (60.6)	474.7 (79.6)	478.3 (67.7)			
V_E (l/min)								
Asthma	6.8 (0.95)	6.4 (0.95)	6.9 (1.03)	6.6 (1.10)	6.6 (0.95)	$F(4,152)=8.91$, $p<.001$, $e=.51$, $\eta^2=.190$	$F(2,38)=0.26$, $p=.769$, $\eta^2=.014$	$F(8,152)=8.47$, $p<.001$, $e=.51$, $\eta^2=.380$
BII Phobia	7.1 (1.10)	6.8 (1.10)	7.1 (1.15)	10.1 (1.23)	7.4 (1.06)			
Controls	7.1 (1.06)	6.8 (0.99)	7.3 (1.06)	7.0 (1.14)	7.2 (0.98)			
Sighs (freq/min)								
Asthma	0.14 (0.055)	0.16 (0.077)	0.20 (0.065)	0.18 (0.103)	0.09 (0.040)	$F(4,152)=5.09$, $p=.003$, $e=.69$, $\eta^2=.118$	$F(2,38)=7.47$, $p=.002$, $\eta^2=.282$	$F(8,152)=2.23$, $p=.051$, $e=.69$, $\eta^2=.105$
BII Phobia	0.33 (0.061)	0.28 (0.086)	0.39 (0.072)	0.67 (0.115)	0.23 (0.044)			
Controls	0.07 (0.057)	0.14 (0.080)	0.11 (0.067)	0.13 (0.107)	0.04 (0.041)			
PCO ₂ (mmHg)								
Asthma	33.3 (1.68)	33.8 (1.60)	33.0 (1.59)	33.2 (1.72)	33.4 (1.64)	$F(4,148)=7.98$, $p<.001$, $e=.65$, $\eta^2=.177$	$F(2,37)=2.26$, $p=.119$, $\eta^2=.109$	$F(8,148)=6.49$, $p<.001$, $e=.65$, $\eta^2=.260$
BII Phobia	39.1 (1.82)	39.9 (1.72)	38.0 (1.72)	35.3 (1.86)	38.4 (1.77)			
Controls	37.1 (1.68)	37.4 (1.60)	35.9 (1.59)	37.6 (1.78)	36.2 (1.64)			

	Pleasant	Neutral	Unpleasant	Surgery	Asthma	Film effect	Group effect	Film x Group effect
HR (beats/min)								
Asthma	73.8 (2.17)	74.0 (2.17)	72.9 (2.29)	73.4 (2.45)	73.7 (2.33)	$F(4,148)=3.47$, $p=.022$, $e=.69$, $p\eta^2=.086$	$F(2,37)=0.11$, $p=.898$, $p\eta^2=.006$	$F(8,148)=4.59$, $p<.001$, $e=.69$, $p\eta^2=.199$
BII Phobia	72.0 (2.54)	71.1 (2.53)	70.0 (2.67)	75.5 (2.86)	72.1 (2.72)			
Controls	74.2 (2.25)	74.9 (2.24)	73.0 (2.37)	72.4 (2.54)	73.4 (2.41)			
HR _{min} (beats/min)								
Asthma	68.2 (2.20)	67.8 (2.14)	66.7 (2.23)	66.9 (2.31)	68.1 (2.31)	$F(4,148)=2.88$, $p=.047$, $e=.65$, $p\eta^2=.072$	$F(2,37)=0.36$, $p=.698$, $p\eta^2=.019$	$F(8,148)=4.39$, $p<.001$, $e=.65$, $p\eta^2=.191$
BII Phobia	62.7 (2.57)	64.5 (2.50)	63.6 (2.61)	67.3 (2.70)	65.5 (2.70)			
Controls	66.8 (2.28)	68.9 (2.22)	66.0 (2.31)	65.0 (2.39)	67.0 (2.39)			
SBP (mmHg)								
Asthma	148.9 (4.53)	146.7 (5.27)	149.5 (4.70)	147.4 (5.17)	149.7 (4.70)	$F(4,140)=0.82$, $p=.493$, $e=.80$, $p\eta^2=.023$	$F(2,35)=0.24$, $p=.789$, $p\eta^2=.013$	$F(8,140)=1.94$, $p=.076$, $e=.80$, $p\eta^2=.100$
BII Phobia	147.0 (5.29)	149.7 (6.15)	144.7 (5.48)	153.5 (6.03)	148.5 (5.49)			
Controls	148.3 (5.06)	141.8 (5.89)	143.7 (5.29)	142.9 (5.78)	144.4 (5.26)			
DBP (mmHg)								
Asthma	85.4 (3.04)	83.7 (3.52)	85.8 (3.52)	85.4 (3.45)	85.9 (3.70)	$F(4,140)=0.46$, $p=.713$, $e=.84$, $p\eta^2=.013$	$F(2,35)=1.39$, $p=.264$, $p\eta^2=.073$	$F(8,140)=0.88$, $p=.519$, $e=.84$, $p\eta^2=.048$
BII Phobia	83.0 (3.55)	86.2 (4.12)	82.3 (4.11)	87.0 (4.03)	84.7 (4.32)			
Controls	79.7 (3.40)	76.8 (3.94)	77.0 (3.94)	77.8 (3.86)	77.8 (4.14)			
SCL (microS)								
Asthma	11.9 (2.68)	11.9 (2.59)	12.1 (2.82)	12.5 (2.83)	12.4 (2.86)	$F(4,144)=5.42$, $p<.001$, $e=.81$, $p\eta^2=.131$	$F(2,36)=0.26$, $p=.775$, $p\eta^2=.014$	$F(8,144)=1.43$, $p=.206$, $e=.81$, $p\eta^2=.074$
BII Phobia	14.1 (3.13)	13.7 (3.03)	14.7 (3.29)	16.5 (3.30)	15.2 (3.34)			
Controls	12.0 (2.88)	11.7 (2.78)	11.9 (3.03)	12.3 (3.03)	12.6 (3.07)			

Note: Abbreviations: R_{os} = respiratory resistance; RR = respiration rate; VT = tidal volume; V'E = minute ventilation; PCO₂ min = minimum partial pressure carbon dioxide; HR = heart rate; HR_{min} = heart rate minimum; SBP = systolic blood pressure; DBP = diastolic blood pressure; SCL = skin conductance level

Table 2.

Indices of R_{OS} change during surgery films relative to neutral films or baseline in asthma patients, BII phobia patients, and healthy controls

		Reference: neutral film			Reference: baseline				
		%	n ₀	g	%	n ₀	g		
Asthma	Viewing	0.060	13.4	5	0.34	0.092	21.5	8	0.61
	Viewing & Tension	0.045	11.7	5	0.27	0.094	23.2	8	0.64
BII phobia	Viewing	0.077	23.4	8	1.26	0.092	29.7	8	1.49
	Viewing & Tension	0.062	19.6	6	1.02	0.076	24.7	8	1.35
Control	Viewing	0.031	11.4	2	0.25	0.027	10.6	2	0.22
	Viewing & Tension	0.024	7.6	2	0.19	0.049	17.4	4	0.39

Note: = difference score; % percent change; $n(t)$ = number of participants with exceeding just noticeable difference threshold; g = within-individual effect size

Table 3.

Means and standard deviations of physiological parameters during the average film viewing vs. film viewing with tension conditions in asthma, BII phobia, and control participants

		R_{os} (kPa $\cdot$ l -l $_{\text{os}}$)	EMG $_{\text{leg}}$ (μ V)	RR (br/ min)	V $_T$ (ml)	V *_E (l/min)	PCO $_2$ (mmHg)	HR (bpm)	HR $_{\text{minimum}}$ (bpm)	SBP (mmHg)	DBP (mmHg)	SCL (μ S)
Asthma	Viewing	<i>M</i>	3.63	15.6	414.7	6.21	33.3	71.9	66.1	143.1	82.6	11.8
		<i>SD</i>	2.93	2.95	243.8	3.58	5.89	8.74	8.4	15.98	12.46	9.5
Viewing & Tension		<i>M</i>	5.56	16.8	425.5	7.04	33.4	75.2	69.0	153.7	87.9	12.5
		<i>SD</i>	2.95	3.07	250.7	4.00	6.09	8.82	8.8	21.59	13.85	11.8
BII phobia	Viewing	<i>M</i>	4.10	15.9	437.2	7.02	37.7	70.3	63.0	146.0	81.9	14.4
		<i>SD</i>	2.93	2.94	243.8	3.58	6.02	8.76	8.4	16.04	12.48	9.5
Viewing & Tension		<i>M</i>	6.04	17.3	479.8	8.35	38.6	73.9	66.5	151.3	87.4	15.3
		<i>SD</i>	2.95	3.06	249.4	4.00	6.09	8.86	8.8	21.64	13.88	11.8
Healthy	Viewing	<i>M</i>	3.67	16.1	433.3	6.53	36.9	71.7	65.0	142.0	76.6	10.7
		<i>SD</i>	2.93	2.95	244.0	3.58	6.02	8.75	8.4	16.02	12.46	9.5
Viewing & Tension		<i>M</i>	4.65	16.5	488.6	7.59	36.8	75.4	68.5	146.5	79.0	13.5
		<i>SD</i>	2.95	3.07	249.6	4.00	6.10	8.83	8.8	21.59	13.87	11.8
Instruction effect ^a	<i>P</i> - level		.544	.001	.001	.001	.223	.001	.001	.004	.006	.009
		<i>pn</i> ²	.010	.250	.300	.534	.040	.503	.464	.223	.199	.173

^a significance levels and partial eta squared (*pn*²) refer to *F*-tests for ANOVA main effects of instruction (film viewing vs. film viewing with tension), df: 1,34-38

Table 4.

Concurrent and prospective within-individual associations of autonomic and respiratory parameters with R_{os} for two participants close to fainting during one surgery film

	RR	V_T	V'_{min}	PCO ₂	HR	SBP	DBP	SCL
Participant 189 (healthy control)								
Concurrent association	.15	-.35	-.27	.11	-.01	.15	.29	-.39
Lag 1 association	.07	-.04	.03	-.26	-.41	.07	.02	-.27
Participant 175 (blood phobia)								
Concurrent association	.17	-.07	.20	-.19	.46	.61	.44	.22
Lag 1 association	-.30	.01	-.19	-.05	.26	.44	.47	-.07