

EARLY CAREER AWARD ADDRESS

Probing the psychophysiology of the airways: Physical activity, experienced emotion, and facially expressed emotion

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Abstract

This article reviews research on airway reactivity in health and asthma within a psychophysiological context, including the effects of physical activity, emotion induction, and manipulation of facial expression of emotion. Skeletal muscle activation leads to airway dilation, with vagal withdrawal being the most likely mechanism. Emotional arousal, in particular negative affect, leads to airway constriction, with evidence for a vagal pathway in depressive states and ventilatory contributions in positive affect. Laboratory-induced airway responses covary with reports of emotion-induced asthma and with lung function decline during negative mood in the field. Like physical activity, facial expression of emotion leads to airway dilation. However, these effects are small and less consistent in posed emotional expressions. The mechanisms of emotion-induced airway responses and potential benefits of emotional expression in asthma deserve further study.

Descriptors: Airways, Respiratory resistance, Skeletal muscle tension, Emotion, Facial expression, Respiration, Asthma, Respiratory sinus arrhythmia

Despite its central role in maintaining the basic life processes of the organism, the respiratory system has attracted only limited attention in psychophysiology (Harver & Lorig, 2000). The conducting airways as part of the respiratory system have been studied mainly in the context of respiratory diseases such as bronchial asthma, and only recently have measurement issues relevant to general psychophysiology been discussed (Ritz, Dahme, et al., 2002). However, the study of the airways can generate unique insights into the autonomic regulation of psychological phenomena, and the analysis of airway interoception is essential for an understanding of breathing-related symptoms in health and disease. Following a few brief introductory passages on airway regulation and lung function measurements, I will summarize experimental research on effects of static facial muscle tension, emotion, and facial expression of emotion on the airways.

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Physiology of the Airways in Health and Asthma

The airways are a complex system of tubes with progressive branching from the main bronchi to the end of the bronchioles. Smooth muscles are located in the walls of the trachea, bronchi, and part of the bronchioles. The tone of these smooth muscles is regulated by the autonomic nervous system and hormonal activity (Barnes, 1986; Canning & Fischer, 2001). Most importantly, activation of parasympathetic cholinergic nerves contracts the smooth muscles, whereas sympathetic activation leads to a relaxation of airway smooth muscles. Because there is little evidence for a functional sympathetic innervation of the human airway smooth muscle, the main pathway for sympathetic effects is through the action of circulating adrenaline on β_2 -receptors expressed on airway smooth muscles. There are additional pathways beyond the classical antagonism of vagal and sympathetic systems. Peptides secreted from afferent nerve fibers and efferent noncholinergic parasympathetic nerves trigger airway smooth muscle constriction and relaxation, respectively. Because noninvasive dynamic measures of these regulatory aspects are not yet available, the psychophysiological exploration of airway activation (without pharmacological blockade) is currently restricted to the functional antagonism of cholinergic and adrenergic pathways. With the latter pathway being largely dependent on circulating adrenaline, the main focus will be on vagal activation, when experimental stimulation is restricted to milder forms of physical activity and emotion induction.

Changes in airway smooth muscle tone can also be triggered by changes in ventilation (for a review, see Ritz, Dahme, et al., 2002). For example, lung inflation leads to reflex smooth muscle relaxation via slowly adapting stretch receptors and cholinergic nerves. Other ventilatory influences are more potent in airway diseases such as asthma. Cooling and drying of the airway mucosa can lead to lasting increases in airway smooth muscle tone. Hypocapnia elicited by hyperventilation leads to contraction of the airways. Stimulation of rapidly adapting stretch receptors with irritants or stronger increases in airflow can also trigger contraction of the airway smooth muscles. In addition, animal studies suggest a positive correlation between central inspiratory drive and bronchoconstriction (Jordan, 2001).

It has long been recognized that spasms of the bronchial smooth muscles (*bronchoconstriction*) is an important component of the pathophysiology of asthma, among other factors such as mucus secretion, edema, and remodeling of the airway walls (National Heart, Lung, and Blood Institute [NHLBI], 2002). Stronger bronchoconstriction is often accompanied by unpleasant symptoms such as shortness of breath or chest tightness and can limit physical performance and daily life activities of patients dramatically. Asthmatic airways are also hyperresponsive to a variety of stimuli such as cold temperature, allergens, irritants, or pharmacological agents, and vagal dysfunction is thought to be an important contributor to these abnormalities (Canning & Fischer, 2001). The vagal system has also been discussed as the major pathway for behavioral influences on asthmatic bronchoconstriction (e.g., Alexander, 1950; Isenberg, Lehrer, & Hochron, 1992; McFadden, Luparello, Lyons, & Blecker, 1969; Miller & Wood, 1997), but factors governing psychophysiological influences on the airways in health and disease have not been explored sufficiently.

Noninvasive Measurement of Lung Function

Increases in tension of the airway smooth muscles reduce the diameter of the airway passages and thereby lead to a higher resistance to airflow. This resistance can be measured in a number of ways (for an overview of techniques, see Ritz, Dahme, et al., 2002). Indirect methods, such as spirometry or peak-flow measurements, estimate the amount of airway constriction by the maximum airflow that can be generated by the individual or by the volume of air that can be exhaled forcefully. In addition to allowing only limited inferences on resistance, these methods have a number of disadvantages for experimental use in psychophysiology. They provide only momentary “snapshots” of lung function, they are time consuming as they have to be repeated a few times to obtain valid measurements, and the forced expiratory maneuvers are effort dependent and thus potentially they introduce systematic biases by the participant and experimenter. In addition, the deep inspiration and forced expiration influence the airway smooth muscle tone and can thereby eliminate task-induced airway responses.

As an alternative to that, the forced oscillation technique has been developed for measuring respiratory resistance more directly. The method requires no effort and in some applications provides continuous measurements; thus it is particularly useful in psychophysiology. Rather than measuring airflow of the individual, the technique generates an external oscillating flow that is “forced” into the airways. The pressure and flow of the signal are then recorded at the mouth. Because the frequency of the

generated flow is high (> 2 Hz) compared to the low frequency of breathing, the flow is barely perceived. Typically, resistance is measured across a range of frequencies (e.g., 2–32 Hz) using pseudorandom noise or brief impulses. A simplified technique has been developed by Korn, Franetzki, and Prestele (1979), in which only one frequency (10 Hz) is used. In this application, the pressure generated by the flow signal and measured at the opening of the mouth is roughly equivalent to respiratory resistance, or, more correctly, respiratory impedance (Z_{rs}), because the signal also includes contributions from the compliance of the respiratory system (the obtained index is often abbreviated as R_{os} for oscillatory resistance). Although the technique has clear advantages in terms of more direct, continuous, and effort-independent measurements of airway constriction, care must be taken to account for possible upper airway effects caused by swallowing, subvocalization, or coughing. In addition, breathing through a respiratory apparatus with a mouthpiece and nose clip is known to alter the basal respiratory pattern (e.g., Askanazi et al., 1980), and can only be tolerated for a limited time.

Physical Activity and the Airways: Evidence for Bronchosomatic Coupling

The early behavioral medicine of asthma has dealt with a prominent lay hypothesis. It was assumed that behavioral relaxation would improve lung function. In the extreme, it was even argued that muscle relaxation would relax the bronchi (e.g., Lask, 1991). However, there is little evidence from physiology that this assumption is tenable. Tension of the skeletal muscles typically leads to a vagal withdrawal and a reciprocal sympathetic discharge (e.g., Berntson, Cacioppo, & Quigley, 1991). Given the specifics of the autonomic regulation of the airway smooth muscles, this would translate into a dilation of the airways. This has been demonstrated in animal studies during dynamic and static muscle contraction (Kaufman, Rybicki, & Mitchell, 1985; Padrid, Haselton, & Kaufman, 1990). In studies with humans, evidence for such a mechanism comes from dynamic exercise protocols (e.g., Kagawa & Kerr, 1970; Mansfield, McDonnell, Morgan, & Souhrada, 1979). Typically, within the first 10–20 min of exercise, a bronchodilation is observed. Average effect size of these changes varies around $d = 0.50$ – 0.80 for both healthy individuals and patients with asthma (Ritz, 1998).¹ Experiments with pharmacological blockade have suggested that vagal withdrawal is an important underlying mechanism (Kaufman et al., 1985; Warren, Jennings, & Clark, 1984). Given the consistency of airway dilation to skeletal muscle activity across studies and its assumed functional role in reducing the work of breathing at higher levels of somatic activation (Kaufman et al., 1985), these observations could be termed as incidents of a *bronchosomatic coupling* (with reference to the classical analysis of behavioral factors in cardiovascular activation; Obrist et al., 1974). Thus, no benefits can be expected for the airways from a behavioral task that is focused on somatic relaxation. Rather, it seems as if

¹In contrast, a commonly observed phenomenon in asthma is exercise-induced bronchoconstriction (Cockcroft, 1997). However, this phenomenon typically develops in later stages of exercise, or following cessation of exercise. Cooling and/or drying of the airway mucosa seems to be the major trigger, but the mechanisms of these constrictions have not yet been fully elucidated. Interestingly, reinstatement of exercise can abolish the exercise-induced bronchoconstriction at least partly (Beck et al., 1994; Schnall & Landau, 1980).

activation of the skeletal muscles would elicit the desired bronchodilation. It is not surprising that the outcome of studies on relaxation techniques in asthma has been mostly disappointing (Richter & Dahme, 1982; Ritz, 2001).

The Impact of Static Facial Muscle Tension on the Airways

The frontal-pulmonary reflex hypothesis. Relaxation of the frontalis muscles has been used in EMG biofeedback studies in the 1970s and early 1980s to achieve whole body relaxation, until Fridlund, Fowler, and Pritchard (1980) demonstrated that no substantial transfer effects can be expected from the frontalis muscle site to other muscle sites. Frontalis EMG biofeedback has also been used in asthma research in the hope of triggering positive effects on the airways. Following initial studies with asthmatic children and adolescents that suggested improvements in lung function after frontalis EMG relaxation, Kotses, Glaus, Bricel, Edwards, and Crawford (1978) developed a specific hypothesis about a reflex mechanism linking trigeminal afferents with vagal efferents at the brainstem level, which should be responsible for improvements in lung function. A reduction in somatic afferents from the facial area would thereby reduce vagal outflow and relax airway smooth muscles, whereas an increase in somatic afferents would increase vagal discharge and constrict the airways. This mechanism was thought to be active in both health and respiratory disease. However, repeated attempts to demonstrate the expected lung function changes in healthy individuals were only partly successful (Glaus & Kotses, 1983; Kotses & Miller, 1987).

Methodological problems precluded any firm conclusion from these studies (for detailed discussions, see Ritz, Dahme, & Roth, 2004; Ritz, Mass, Dahme, & Richter, 1995). Also, the ecological validity of the reflex hypothesis is questionable. Although a number of somato-autonomic reflexes or response patterns linking trigeminal afferents with vagal efferents are known, their typical purpose is to protect the organism from harm. One of the better known examples is facial cold stimulation that elicits a typical pattern of heart rate deceleration that is suggestive of the diving response (Scholander, 1963), which has been linked to energy conservation in diving vertebrates. In healthy individuals, facial cooling also leads to an increase in R_{os} (Ritz, Thöns, & Dahme, 2000). However, activation of skeletal muscles of the face, specifically the frontalis muscles, does not contribute prominently to the afferent arm of any of these response patterns. Although functional links between facial afferents and the vagus

have been suggested for feeding and communication (Porges, 1995), little systematic research is available on that. We therefore held the more conservative hypothesis that facial muscle tension will not elicit a fundamentally different autonomic response pattern than skeletal muscle activation in other regions of the body. This hypothesis formed the basis of our studies on frontal muscle tension and respiratory resistance, which will be described in greater detail in the following.

Empirical findings: Frontal muscle tension and respiratory resistance. In a critical replication of the frontal pulmonary reflex studies, we assigned healthy students ($N = 48$) to one of four conditions of a frontal EMG biofeedback training (Ritz et al., 1995). They were instructed to increase or decrease the pitch of a tone, and the tone was either contingent or noncontingent to their actual muscle tension levels of the forehead region. In none of the conditions was the source of the auditory feedback revealed to the participant until after the training. R_{os} was measured before training and after two consecutive 5-min training sessions. The results suggested that only the group receiving contingent feedback of their EMG levels with the instruction to increase the pitch of the tone (thus to increase muscle tension) had lower levels of R_{os} after the two training sessions (Figure 1). However, the critical Contingency \times Instruction \times Time interaction was only present as a trend, $F(1,44) = 2.73, p = .105$. But when participants were reallocated into subgroups according to their actual changes in frontal EMG across the training, those who actually increased their frontal muscle tension throughout the training had significantly lower R_{os} following the two training sessions. Thus, static facial muscle tension leads to lower levels in respiratory resistance. The results are in line with predictions from respiratory physiology, but are in the opposite direction of the predictions from a frontal-pulmonary reflex hypothesis.

Given these contradictions, we designed a second study to investigate the phasic effects of facial muscle tension changes on the airways (Ritz, Dahme, & Wagner, 1998). We used brief tense-release sequences of forehead (frontal) and forearm (flexor) muscle sites, comparable to maneuvers used in a Progressive Muscle Relaxation exercise. Four blocks of six tense-release sequences were administered, with each participant receiving all four combinations of forehead or forearm muscle sites at 40% or 80% of their maximum individual force assisted by EMG feedback from the target muscle site. One sequence consisted of 10 s

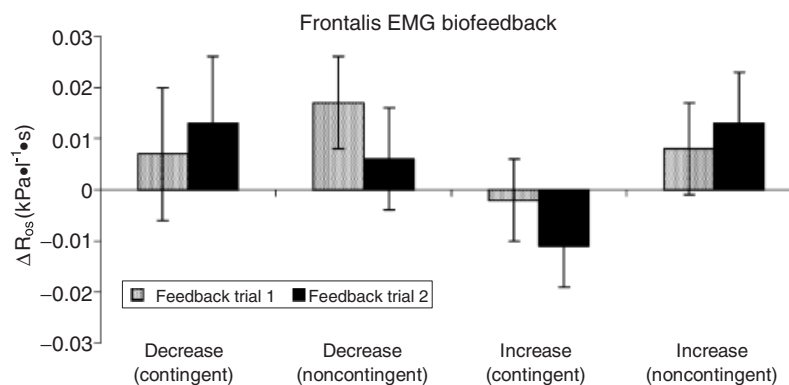


Figure 1. Effect of two 5-min trials of frontalis EMG biofeedback training on subsequent measurements of oscillatory resistance (depicted as changes from pretraining baseline) in groups instructed to increase or decrease tension with contingent or noncontingent feedback (adapted from Ritz, Mass, et al., 1995).

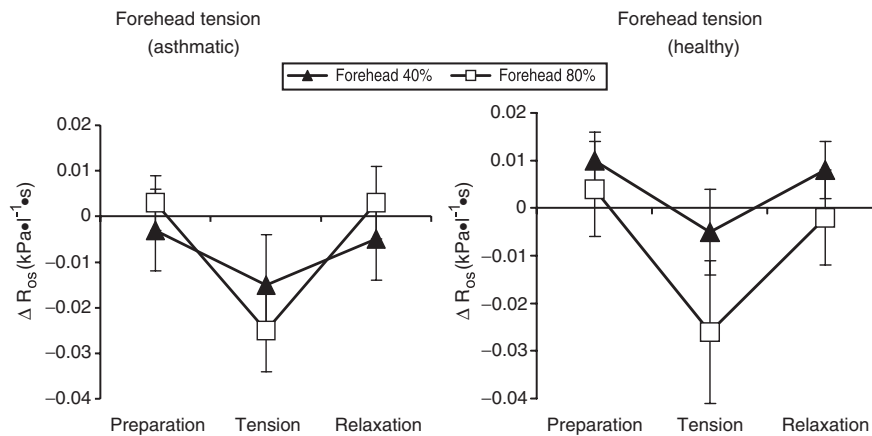


Figure 2. Changes in oscillatory resistance (from baseline) during tense–release sequences of the forehead muscles at 40 and 80% of the maximum individual force in healthy and asthmatic participants (adapted from Ritz, Dahme, et al., 1998).

preparation, 15 s tension, and 20 s relaxation. Each block was preceded by a brief 1-min baseline. This time, R_{os} was measured continuously throughout the exercises to observe possible phasic effects on R_{os} concomitant with the brief tension periods. Both healthy and asthmatic participants were included ($N = 24$ each). As expected, results for R_{os} showed phasic decreases during forehead tension epochs and a return to pretension levels during the subsequent relaxation epochs (Figure 2). Across 1-min baseline measurements, tonic R_{os} remained on a stable level, suggesting no substantial effect of the complete tense–release protocol on the airways. Results were comparable for healthy and asthmatic participants. The within-individual effect sizes of R_{os} changes ranged between $d = 0.26$ and 0.50 across groups and tension levels, and were comparable to the effect sizes obtained for heart rate (HR), which showed significant increases under 80% tension conditions.

Mechanisms and influences. As an indication for potential underlying mechanisms of the airway responses, we studied the effects of the experimental protocol on respiratory sinus arrhythmia (RSA). Using an index of cardiac vagal activity to infer changes in vagal outflow to the bronchi is not optimal, but no noninvasive measure of vagal outflow to the airways is currently available. Also, a valid interpretation of RSA as an index of vagal activation requires the control of respiration rate and tidal volume (V_T), which can both have an independent influence on RSA (Berntson et al., 1997; Grossman, Karemaker, & Wieling, 1991). Therefore, we developed a within-individual correction procedure using paced breathing baseline calibrations of RSA to estimate the amount of RSA changes independent from these factors (Ritz, Thöns, & Dahme, 2001; Ritz, Wagner, & Dahme, 1996). Using this corrected index of RSA (RSA_c), we found robust evidence for decreases during tension epochs. This is compatible with earlier findings that vagal withdrawal is responsible for airway dilation during skeletal muscle tension. No changes in RSA were found across the session, indicating that vagal tone returned to baseline levels following tension periods. Other authors have observed increases in RSA during progressive relaxation sessions (Lehrer, Hochron, et al., 1997); however, ventilatory influences were not controlled. Because slower and deeper breathing can increase RSA, findings may have been due to relaxation-induced changes in ventilation alone.

Additional factors were identified that had influenced the changes in R_{os} . First, increases in functional residual capacity (FRC) are known to reduce airway resistance (Forster, DuBois, Briscoe, & Fisher, 1986). Changes in FRC can be estimated using the drift-corrected end-expiratory levels from the volume curve of the pneumotachograph. The results suggested that FRC was significantly increased during forehead tension epochs, echoing earlier findings that voluntary facial muscle activation (in tasks of posed emotional expression) leads to brief increases in FRC (Boiten, 1996). When we controlled for FRC changes by choosing from each block only one tense–release sequence with minimal or no FRC change, R_{os} decreased during tension epochs were still visible, but significance was reduced to the condition with 80% of the maximum force in healthy participants. In asthma patients, an additional effect of medication on airway responses was observed. When the asthma group was divided into patients who took nonsteroidal medication (such as β -adrenergic bronchodilators) versus those taking additional inhaled corticosteroids, significant decreases in R_{os} during forehead tension (using the sequences with minimal or no FRC changes) were only found in the former group. Although these results showed that airway response to skeletal muscle tension is determined by a number of factors including medication that aims to reduce airway inflammation and hyperreactivity in asthma, the general direction of our findings remained intact: If anything, decreases in R_{os} were observed as a consequence of facial muscle activation rather than increases in R_{os} . The results again contradicted the mechanism outlined in the frontal-pulmonary reflex hypothesis, and were more in line with expectations derived from studies in respiratory physiology.

Static Tension Effects of Other Skeletal Muscle Sites on the Airways

Our experiment using tense–release sequences (Ritz, Dahme, et al., 1998) also included muscle tension of the forearm. During tension epochs, R_{os} decreased in a fashion similar to that during forehead tension, with few changes in ventilation. This effect was only found for healthy participants, whereas no substantial R_{os} changes were found for asthma patients. Compared to healthy individuals and to forehead muscle tension, a substantially stronger response in ventilation with higher inspiratory flow (V_T/T_1 , or “respiratory drive”; Milic-Emili & Grunstein, 1976) and

minute ventilation was observed in asthma patients. This is consistent with other research demonstrating stronger ventilatory activation of asthma patients to normal physical activity (Ritz, Meuret, Wilhelm, & Roth, 2002; Varray & Préfaut, 1992). We suspected that these increases in ventilation could mask the effects of muscle tension on the airways, by a central link between respiratory drive and vagal outflow to the airways, by known physiological effects of ventilatory adjustments on the airways, or by interaction with the forced oscillatory flow of the equipment (more turbulent flow profiles can lead to increased values in R_{os}). Similar effects could have been operative in another study with 3-min tension sequences of facial (frontalis), shoulder (trapezius), and forearm (extensor) muscle sites at 75% of the individual maximum, which yielded no effects on mean Z_{rs} (Lehrer, Generelli, & Hochron, 1997). No measurements of respiration were reported.

In a further study with healthy participants we investigated the effect of a 1.5-min isometric exercise of both arms at 30% of the individual maximum force (Ritz, Wiens, & Dahme, 1998). Against our expectations, R_{os} increased significantly during tension periods. The ventilatory adjustments that accompanied the extended tension periods could have accounted for these findings, or the proximity of the tensed muscles to the thorax (the resistance and compliance of the thoracic wall contribute to measures of R_{os}) and the respiratory muscles that participate in the mechanics of breathing. Thus, it is too early to extend the findings from dynamic exercise and static facial muscle tension to static tension of other skeletal muscle sites.

Emotion and the Airways

Because airway smooth muscle tone is regulated by the autonomic nervous system, it is logical to expect that emotional states will influence the diameter of the airway passages. On a very general level, two competing predictions can be made regarding the pattern of airway responses to be expected from emotional stimulation. On the one hand, a naïve biobehavioral stress model predicts that sympatho-adrenergic arousal (and parasympathetic withdrawal) will lead to a relaxation of airway smooth muscles and thus a dilation of the airways. This mechanism is most prominently reflected in the widely used β -adrenergic asthma medication that produces fast relief from symptoms of airway obstruction. On the other hand, clinical evidence suggests that stress and emotions, including positive excitement and sexual arousal, produce a deterioration in lung function in asthma patients (e.g., Purcell, 1963; Rees, 1980). Evidence for the importance of psychosocial factors in asthma exacerbations varies widely across studies (Weiner, 1977), but using a psychometrically sound questionnaire measure we found that approximately 25% of patients of a larger primary care sample perceived

emotions and stress as an important factor (Ritz, Steptoe, Bobb, & Edwards, 2001). In addition, a number of earlier studies using stressful tasks have shown that airway resistance generally increases under experimental challenge (e.g., Levenson, 1979; Mathé & Knapp, 1971; see Lehrer et al., 1996, for an exception with an active coping task). A number of mechanisms could account for these increases, such as vagal excitation that is concomitant with sympathetic discharge (Berntson et al., 1991), or ventilatory influences through stress-induced hyperventilation, laughing, sighing, or crying (Clarke, 1982; Liangas, Morton, & Henry, 2003; Weinstein, 1985).

It is now widely accepted that a unidimensional stress-arousal model cannot capture the richness of autonomic activation patterns of emotional states (Cacioppo, Klein, Berntson, & Hatfield, 1993; Levenson, 1992; Stemmler, 1989). For a more complete understanding of emotional effects on the airways it is important to induce a wider range of emotional states covering both states of a negative and positive valence. Therefore, we conducted a number of basic emotion induction experiments to study (a) the direction of airway response under emotional challenge, (b), whether mainly negative states would lead to airway responses (equivalent to a valence modulation), (c) or whether negative and positive states would lead to airway responses, and finally, (d) to interpret potential airway responses within a wider framework of autonomic psychophysiology.

Affective Stimulation: Phasic Influences on Respiratory Resistance

In a first series of experiments, we studied phasic airway responses to processing of brief affective stimuli (Table 1). We mainly used affective pictures, most of them taken from the International Affective Picture System (IAPS; Center for the Study of Emotion and Attention, 1999). In two of the studies, we had preevaluated and preselected these stimuli for inducing specific affects, such as depressive or happy states; in two others we selected stimuli according to global categories of positive, neutral, and negative affect. Studies also varied regarding the mode of picture presentation, with presentation in either random series or in affectively homogeneous blocks. Depending on the individual study, each stimulus was shown for 10 to 20 s. In addition, in two of the studies, viewing of each picture was followed by a brief imagery period of identical length, during which the participants were instructed to recall the picture and form a vivid mental elaboration of it. In one study, we also used stimuli of the Velten mood induction procedure (Velten, 1968). These are brief self-referring sentences designed to induce feelings of happiness (e.g., "I'm pleased that most people are so friendly to me"), depression (e.g., "I feel terribly tired and indifferent to things today"), or affectively neutral states (e.g., "My attitude towards things has changed with the years"). Throughout the presentation (and

Table 1. Four Studies Using Brief Affective Stimuli: Modulation of R_{os} According to a Dimensional Model of Affect

	Stimulus qualities	Mode of presentation	R_{osTOT}	R_{osI}	R_{osE}
Ritz, George, et al. (2000)	happy, neutral, depressing	homogeneous block	(Arousal)	Arousal	n.s
Ritz, Alatupa, et al. (2002)	positive, neutral, negative	random series	Valence	n.s	(Valence)
Ritz & Thöns (2002)	positive, neutral, negative	homogeneous block	(Valence)	n.s	Valence
Ritz, Dahme, et al. (2001)	happy, content, erotic, neutral, anxious, disgusting, depressing	random series	(Valence)	n.s	n.s

Note: Valence and arousal modulation of R_{os} was determined by polynomial trend analysis across positive, neutral, and negative stimulus categories; effects in parentheses indicate statistical trends ($p < .10$).

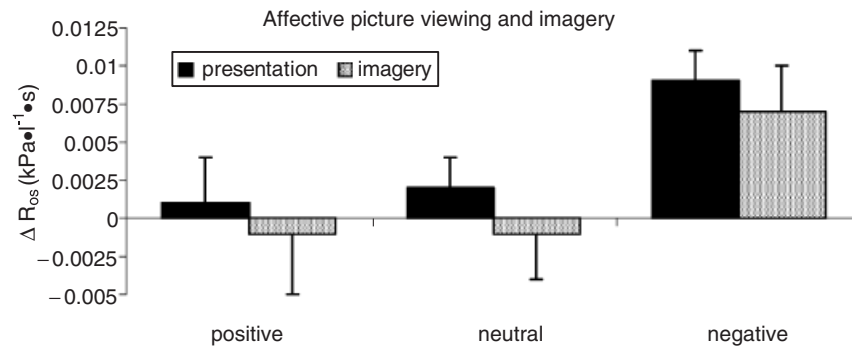


Figure 3. Changes of oscillatory resistance during viewing and imagery of affective pictures (adapted from Ritz, Alatupa, et al., 2002).

imagery) trials, R_{os} was measured continuously, along with the V_T trace from a pneumotachograph, EKG, and skin conductance response (SCR). In three studies we also measured EMGs of distinct facial muscles sites that have been shown to be active during affective processing (e.g., Brown & Schwartz, 1980; Cacioppo, Petty, Losch, & Kim, 1986). Emotional experience of the stimuli was measured on rating scales of valence, arousal, and interest. Because participants were breathing through a mouthpiece and tube for resistance measurements, EMGs were only recorded over corrugator supercilii and orbicularis oculi sites.² The four experiments involved a total of 148 participants.

Results on the rating scales showed that stimuli were experienced as expected, and findings on other physiological and behavioral measures (such as viewing time) often reflected findings from previous IAPS studies (e.g., Bradley, Cuthbert, & Lang, 1990; Lang, Greenwald, Bradley, & Hamm, 1993). R_{os} , the main parameter of interest, mostly increased during affective stimulation. Increases were most prominent during negative affective stimuli, with three studies showing evidence for a valence modulation of R_{os} (e.g., Figure 3). In one study with random presentation of pictures (Ritz, Dahme, Thöns, & Fahrenkrug, 2001) these increases were mainly restricted to disgust stimuli (e.g., picture of a dirty toilet), whereas stimuli of other emotional qualities such as depression, anxiety, happiness, or erotic feelings did not elicit substantial changes. In this study both healthy individuals and asthma patients participated, and little difference in airway response between groups was observed. Only one study (Ritz, George, & Dahme, 2000) yielded evidence for an arousal modulation, with higher R_{os} during viewing and imagery of happy and depressing stimuli (pictures and statements). We were not able to identify a role for either mode of presentation or stimulus quality alone in modulating R_{os} across all studies. It could be speculated, however, that for positive states these factors interact, so that more persistent exposure with a homogeneous stimulus series of a specific positive affect are needed to lead to substantial R_{os} increases. In any case, the smallest common denominator of these studies was an increase in R_{os} during negative states. Thus, brief affective stimulation with unpleasant

material leads to a small airway narrowing, both in health and asthma. The mean within-individual effect size for R_{os} increases (from prestimulus levels) for negative pictures was $d = 0.37$.

In search of evidence for potential mechanisms we analyzed ventilation, RSA_c , inspiratory fraction of R_{os} (R_{osI}), and expiratory fraction of R_{os} (R_{osE}). Respiratory parameters extracted from the V_T curve did not show any consistent pattern of changes across the four studies and did not parallel the overall findings in R_{os} . Findings such as increasingly shorter timing indices with greater pleasure (Ritz, Dahme, et al., 2001) were not replicated. V_T/T_I was lower during positive and negative stimuli in one study (Ritz, Alatupa, Thöns, & Dahme, 2002), which was compatible with an arousal modulation, whereas changes in another study were more compatible with a valence modulation, with flow increasing with pleasure (Ritz, George, et al., 2000). Changes in FRC were not significant. Correlational analysis yielded some relationship between R_{os} and timing parameters, in particular a negative correlation with T_I and T_I/T_{TOT} for happiness in healthy participants. RSA_c yielded evidence for increases in cardiac vagal tone for erotic pictures, but the overall pattern of changes did not follow changes in R_{os} (Ritz, Thöns, Fahrenkrug, & Dahme, 2004). However, a positive correlation between R_{os} and RSA_c changes was found in asthmatics during depressing stimuli. R_{osI} and R_{osE} were analyzed in an attempt to uncover evidence for a possible greater involvement of the upper airways (larynx, vocal cords) as compared to the lower airways (bronchi). R_{os} measurements cover the whole respiratory tract, and approximately 15% of the values is due to the upper airways in regular measurements (Ritz, Dahme, et al., 2002). Because the upper airway contribution is typically greater during expiration, changes exclusively seen in R_{osE} would be of less importance for asthma (although they may be interesting from a basic research point of view and clinically for disorders such as vocal cord dysfunction; see, e.g., Newman, Mason, & Schmaling, 1995). Again, no consistent pattern across studies emerged: One study yielded evidence for R_{osI} (Ritz, George, et al., 2000), one for R_{osE} (Ritz & Thöns, 2002), whereas the other two studies remained equivocal. Thus, additional parameters did only yield sporadic evidence for a ventilatory or vagal mediation of R_{os} or for systematic effects attributable to the upper airway system.

Emotion and Tonic Levels of Respiratory Resistance

Although the study of phasic airway responses to brief affective stimuli can further our basic understanding of airway dynamics under affective challenge, the effect of longer lasting emotional stimuli on airway tone could be more relevant for emotion-

²Orbicularis oculi EMG is measured in many IAPS studies to capture startle response. Our studies did not include this parameter, and placements of the EMG electrodes followed the recommendations of Fridlund and Cacioppo (1986). Although tension levels of the orbicularis oculi are not expected to reflect affective processing by some researchers (Lang et al., 1993), others have provided evidence that the site is particularly active in positive emotional states, possibly in states of genuine pleasure (Ekman, Davidson, & Friesen, 1990; Hess, Banse, & Kappas, 1995).

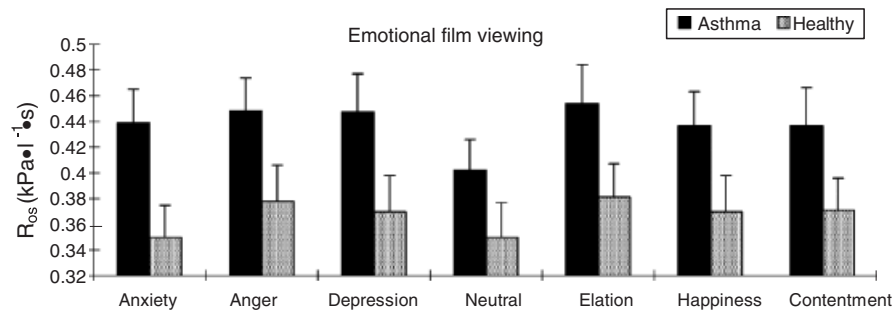


Figure 4. Oscillatory resistance during viewing of emotional film clips in healthy and asthmatic participants (adapted from Ritz, Steptoe, et al., 2000).

induced asthma. Clinical reports of asthma-relevant psychosocial situations almost always imply sustained levels of emotional stimulation, such as periods of angry or anxious feelings, elation, or erotic arousal (e.g., Knapp & Nemetz, 1960). We therefore studied effects of longer lasting experimental stimuli on the airways in both healthy and asthmatic individuals (Ritz, Steptoe, De Wilde, & Costa, 2000). In addition, as autonomic patterns can vary beyond global categories of positive and negative affect, we selected the stimulus material according to distinct emotional states: We used three film clips for unpleasant states (sadness, anger, anxiety), three clips for pleasant states (elation, happiness, contentment), and one clip for a neutral emotional state. R_{os} was measured along with ventilation, HR, blood pressure, and skin conductance level (SCL) throughout the film clips (1.5–5 min duration) and for 1 min following the end of the film. Results showed robust R_{os} increases during all emotional films compared to the neutral film in healthy and asthmatic participants ($N = 24$ in each group), suggesting an arousal modulation of airway tone (Figure 4). During the 1-min recovery, R_{os} gradually approached neutral film levels. Rating scales for categories of emotion filled in after each film mostly confirmed that participants had experienced the film clips as intended.

The only other physiological parameter showing a similar response pattern across emotional qualities was SCL, with higher values for all emotional films compared to the neutral film. Baroreflex sensitivity (BRS), employed as an estimator of vagal activity,³ remained unaffected by emotional films. Respiration rate was positively correlated with R_{os} during the anger film in asthmatics and during the happiness film (also minute ventilation) in healthy participants. Thus, only sporadic evidence for potential mechanisms of R_{os} changes across emotional categories emerged. Increases in R_{os} that could be indicative of greater vagal outflow to the airways coexisted (on the group level) with increases in electrodermal activity that typically indicates greater sympathetic outflow. This could be interpreted as an instance of fractioning of response directions across organ systems (Lacey & Lacey, 1974) or, alternatively, as the result of sympathetic and

vagal coactivation with a predominance of vagal effects (Berntson et al., 1991) on the airways. Involvement of peptidergic pathways in the regulation of the airways may also play a role.

Mood and Lung Function in Everyday Life

It is of considerable interest to study the role of emotion in airway reactivity beyond the artificial and highly structured setting of the psychophysiological experiment. As a convenient way to monitor lung function in everyday life, self-managements of peak expiratory flow (PEF) have been utilized in asthma for some time. PEF and other indices of the forced expiratory maneuver can only provide indirect estimations of airway resistance, but the equipment is currently the least cumbersome and most economical option for ambulatory monitoring of lung function. The PEF and symptom diary traditionally used in self-management of asthma have been employed in earlier efforts to study the association of emotional states and lung function in daily life (e.g., Steptoe & Holmes, 1985). In a field study of lung function and mood we used an electronic pocket spirometer combined with an electronic diary, which allowed monitoring of the correct timing of self-assessments (Ritz & Steptoe, 2000), rather than relying on PEF paper-and-pencil techniques of questionable validity (e.g., Verschelden, Cartier, L'Archeveque, Trudeau, & Malo, 1996). The equipment had also implemented a fixed measurement protocol of symptom, mood, and activity ratings followed by lung function measurements to avoid contamination of mood ratings by the measurement procedure or by results of lung function measurements. In addition to PEF the electronic spirometer also recorded forced expiratory flow in the first second (FEV_1), a flow parameter regarded as slightly less effort dependent and more sensitive for airway obstruction in asthma (Ritz, Dahme, et al., 2002). Twenty asthma patients and 20 healthy participants from the previous laboratory study (Ritz, Steptoe, et al., 2000) recorded their mood three times daily for a period of 3 weeks. From the measurement protocols we extracted episodes of extreme positive mood (mean of contentment, happiness, and elation ratings) and extreme negative mood (mean of anxiety, depression, and anger ratings) as well as episodes of a more neutral mood quality. Lung function associated with these mood states was then analyzed.

Results of this field study showed a variation of lung function with mood states in asthma (Ritz & Steptoe, 2000). In patients, FEV_1 was lower (which is roughly equivalent to higher airway resistance) during negative mood episodes and also slightly lower during positive mood episodes than during episodes of neutral mood, again suggesting a modulation of airway function by emotional arousal. Average ratings of experienced arousal, but not self-reported physical activity in the previous 30 min,

³BRS was analyzed using the spontaneous sequence method that allows a separation of down sequences (in which decreases in blood pressure are accompanied by increases in heart rate) and up sequences (in which increases in blood pressure are accompanied by decreases in heart rate). Only the latter reflects vagal excitation (in concert with sympathetic withdrawal, which is probably less relevant to the analysis of airway smooth muscle tone). There was no effect of film clips on either up or down sequences. In this study, substantial reduction in BRS was only observed during a mental arithmetic test, confirming prior research and demonstrating the adequate sensitivity of this noninvasive index of autonomic function.

Table 2. Effect Sizes d for Increases in Respiratory Resistance (R_{os}) during Laboratory Emotion Induction by Films and Decreases in Lung Function (FEV_1) during Extreme Mood States in the Field

	Laboratory		Field	
	Negative	Positive	Negative	Positive
Asthma patients	1.02	0.95	0.51	0.26
Healthy participants	0.55	0.89	0.17	-0.43

mirrored this pattern of lung function changes. Although lung function was also lower during negative episodes in healthy participants, these findings were not substantial. Regarding the laboratory-field relationship, a significant between-individual correlation of lung function changes in daily life was observed with R_{os} changes during emotional film viewing in the laboratory for asthma patients. The FEV_1 change for negative mood episodes was negatively correlated with R_{os} changes during the average negative film clip, particularly during the film eliciting sadness, suggesting that patients with a greater decline in lung function in negative mood episodes also showed stronger R_{os} increases during films inducing negative emotional states. These results corroborate the overall conclusion from studies on phasic airway responses to brief stimuli that particularly negative emotional states work toward a decline in lung function. The mean effect sizes for R_{os} changes during film clips and lung function changes during extreme mood states showed the greatest importance for negative states in asthma patients (Table 2). Methodologically, although forced expiratory flow measurements are suboptimal for psychophysiological research, an explanation of the mood-dependent changes in lung function in the field by effort dependence is less convincing, given the correlation with effort-independent R_{os} measurements in the laboratory. The results also demonstrate that experimental emotion induction techniques can elicit airway changes with relevance to patients' everyday lives. The findings argue for the validity of the long-held belief that emotions influence the airways in asthma.

Clinical Relevance of Experimentally Induced Airway Responses for Asthma

The actual size of airway responses to emotional stimulation in the laboratory is often too small to elicit symptoms in asthma patients. Full-blown asthma attacks in the laboratory have never been observed in any of our studies, and other authors have only reported instances in earlier research (e.g., Dekker & Groen, 1956). The current standard of antiasthmatic medication is probably responsible for this, as is our focus on patients with mainly mild to moderate persistent severity of their disease. From interoception studies using added resistive loads (Dahme, Richter, & Maß, 1996) we know that changes in resistance of 0.06–0.08 or 0.07–0.11 $kPa \cdot l^{-1} \cdot s$, for healthy controls or asthmatic patients, respectively, are needed to produce a just noticeable difference from baseline conditions with unobstructed airway passages. Although the ecological validity of the added resistive load paradigm for simulating asthmatic airway obstruction has been debated, and the link between visceral activation and symptom perception is only modest in general (Lang, 1994), these figures can at least provide rough guidance. The observed average R_{os} changes from pretask or baseline levels were below these levels, with 0.007 $kPa \cdot l^{-1} \cdot s$ for negative pictures in our

four experiments, and 0.020 and 0.064 $kPa \cdot l^{-1} \cdot s$, for healthy and asthmatic participants, respectively, for negative film clips. Therefore, the clinical relevance of laboratory emotion-induced airway responses for the everyday life of patients might be questioned.

However, two major facts argue in favor of their clinical relevance. First, in many studies a certain proportion of patients show airway responses that in fact can be regarded as “clinically significant,” following currently accepted (though somewhat arbitrary) criteria. In our film study (Ritz, Steptoe, et al., 2000), 7 patients (29% of the patient group) reached R_{os} increases from baseline between 0.078 and 0.181 $kPa \cdot l^{-1} \cdot s$, which exceeds the average perception threshold. Using other criteria for clinical significance, similar proportions of asthma patients (20–40%) have been reported to respond in other research with experimental psychological stimuli (Isenberg et al., 1992). These proportions also correspond with approximately 25% of patients reporting psychological triggers for their asthma symptoms in a systematic survey of primary care asthma patients (Ritz, Steptoe, et al., 2001).⁴ A problem of this extent certainly justifies further research efforts. Second, airway responses in our film-viewing study correlated with lung function changes in extreme everyday life mood episodes of patients (Ritz & Steptoe, 2000). R_{os} changes during negative as well as positive films were also positively correlated with scores on the psychological trigger subscale of the Asthma Trigger Inventory (ATI): Patients reporting a greater importance of psychological triggers for their asthma symptoms also showed greater airway obstruction while viewing the emotional films in the subsequent laboratory session (Ritz, Steptoe, et al., 2001). Thus, airway responses elicited by emotion induction are indeed relevant for the study of psychological determinants of asthma in daily life, even when these airway responses are on average below common criteria of clinical significance.

Emotional Facial Muscle Activation and the Airways

Emotional Facial Expression and Autonomic Nervous System Activity in Health and Disease

The role of emotional facial expression in shaping autonomic nervous system activity has been subject to intensive investigation (e.g., Buck, 1979; Cacioppo et al., 1992; Levenson, 1992). Respiration and airway activation (as well as indices indicative of vagal efferent outflow) have been largely ignored in these studies. In the clinical context, research along these lines can ideally help to uncover the role of emotional expression in modifying disturbances of organ function, such as the airway system in asthma. The short history of psychosomatic medicine is full of speculations and observations on the potential impact of emotional expression in asthma (e.g., Alexander, 1950; Purcell, 1963). Rees (1980) concluded in an overview of his extensive research on etiological factors of asthma that “suppression or inadequate expression of emotions were far more important in the precipitation of attacks of asthma than the type of emotion experienced” (p. 252). Emotional expression has also found its way into lists of asthma triggers in contemporary consensus statements on the diagnosis and treatment of asthma (NHLBI, 2002).

⁴Reports regarding relevance of psychological triggers to asthma attacks typically vary considerably (Weiner, 1977). Methodological problems such as the lack of psychometrically validated instruments and varying reference populations probably account for these differences.

In a series of studies Florin and colleagues have collected experimental evidence for the role of emotional expression in asthmatic children undergoing challenging tasks or viewing cartoon films (for a review, see Florin et al., 1993). Their findings remained equivocal, with some experiments providing evidence for an improvement in lung function following emotional expression, whereas others showed no changes or a decline. A major disadvantage of their experimental setup was the use of peak flow meters or spirometers for measuring lung function. In two of our studies we explored the impact of spontaneous and posed facial expressions of emotion on the airways using the forced oscillation technique.

Drawing on our findings with voluntary, biofeedback-induced increases in static facial muscle tension (Ritz et al., 1995; Ritz, Dahme et al., 1998) we hypothesized that at least part of the airway response to emotional expression could be simply explained by skeletal muscle tension effects. In general, we would expect a tendency toward lower airway resistance following emotional facial expressions or negative correlations between indices of facial expression (such as EMGs) and R_{os} . Given the specific autonomic nervous system regulation of the airways, with vagal excitation leading to bronchoconstriction, complicated speculations about mechanisms that can help explain changes in organ function following emotional expression, such as catharsis or discharge models (for discussion, see Cacioppo et al., 1992), may not be necessary. Skeletal muscle activation will reduce vagal excitation (and increase sympathetic outflow), thus dilating the airways. This is in contrast with research on emotional expression and sympathetic autonomic indices, which has also produced negative (between-individual) associations between expressiveness and autonomic activation (e.g., Buck, 1979; Gross & Levenson, 1993).

One methodological problem with airway resistance measurements using mouthpieces and nose clips is that they interfere considerably with emotional facial expressions. Measurements have to be performed at the end of an expression episode, which has the apparent disadvantage of providing no parallel observation of facial muscle and airway activation. As we have observed in our study using emotional films (Ritz, Steptoe, et al., 2000), the airways recover from experimental emotional induction effects within 1–2 min. Although activation in some facial muscle sites related to emotional processing can be captured while participants use the mouthpiece and nose clip (e.g., Ritz, Alatupa, et al., 2002), this is not necessarily successful (e.g., Ritz, George, et al., 2000). Problems with parallel measurements of facial expression and lung function aside, brief presentations of experimental affective stimuli typically do not elicit more intense or longer lasting emotional states and expressions, thus reducing the possibility of detecting an impact on the airways. Given these methodological problems it is not surprising that the study of Ritz, Alatupa, et al. (2002) with healthy participants yielded no substantial between- or within-individual correlations between R_{os} and EMGs over corrugators supercillii and orbicularis oculi muscle sites.

Spontaneous Emotional Expression and Respiratory Resistance

To induce longer lasting emotional states and facial muscle activation we used a presentation of affective pictures and self-referent Velten statements in homogeneous blocks in a study with healthy and asthmatic participants (Ritz, Claussen, & Dahme, 2001). Spontaneous facial expression of emotion was inferred from EMGs over corrugator supercillii, orbicularis oculi, and

zygomaticus major muscle sites. Whereas facial EMGs were measured during stimulus presentations, R_{os} , cardiac interbeat interval (IBI), RSA_c , and ventilatory indices were measured for 1 min immediately after each block of nine happy or depressing stimuli (with one additional neutral control stimulus in each block). Each stimulus was viewed for 10 s, followed by an imagery period of 10 s. Ratings of happiness versus depression confirmed that the stimulus blocks had induced adequate variations in mood. Results in R_{os} showed relatively uniform increases from baseline following all stimulus blocks; however, this time they were only significant for asthma patients following depressing stimulation. As an indication that vagal excitation probably contributed to these changes in tonic resistance, under these conditions R_{os} changes were positively correlated with changes in RSA_c and IBI.

The average facial EMG responses to individual stimuli were only partly related to R_{os} and RSA_c changes following stimulus blocks. Although in asthma patients for target muscle sites of the happy emotional state, zygomaticus and orbicularis, a significant negative between-individual correlation was found with subsequent R_{os} changes from baseline, this correlation was mainly due to one outlier, a patient with strong facial activation and subsequently strong reductions in R_{os} . Excluding this patient the correlation was not significant anymore. More convincing were the correlational findings in RSA_c , providing partly significant evidence for a negative relationship with orbicularis or zygomaticus responses following blocks of positive stimuli. In asthma patients, greater average facial response to happiness stimuli was associated subsequently with lower cardiac vagal tone. No consistent correlational pattern was observed with average corrugator EMG response to individual depressing stimuli.

However, additional analysis of facial EMGs revealed typical gradients of corrugator EMG (see Bradley, Cuthbert, & Lang, 1996; de Jong-Meyer, Hubert, Ostkamp-Hovekamp, & Vennen, 1993): Across blocks of depressing stimuli, prestimulus levels of corrugator EMG activity increased continuously so that corrugator response to individual stimuli captured only part of the actual level of activation (Ritz, Dahme, & Claussen, 1999). Participants were divided into subgroups who produced these significant positive corrugator EMG gradients versus those who did not show these gradients, and R_{os} changes were reanalyzed for these subgroups. For both stimulus types, depressing pictures and Velten statements, R_{os} was lower for those groups who showed the significant positive corrugator EMG gradients. The group differences were significant for pictures. Thus, greater emotion-induced facial muscle activation was followed by lower respiratory resistance. There was no substantial difference between asthma patients and healthy participants in this response pattern. These findings corresponded well with our earlier results on biofeedback-assisted changes in facial static muscle tension. Again, bronchosomal coupling is the most parsimonious explanation.

Influence of Posed Emotional Expressions on Respiratory Resistance

Because the facial muscle activation was only small in our study on spontaneous emotional expression, we designed a study with posed facial expressions of emotion that are accompanied by greater muscle activation (Ritz, Dahme, Thöns, & Quast, 2003). The initial observation in our previous study (Ritz, Claussen, et al., 2001) involving the outlier in the negative correlation between facial EMGs and R_{os} led us to expect that more intense facial

expressions may also trigger stronger airway dilation. The new study mainly explored effects of emotion regulation on the airways, but I will focus on a brief description of muscle tension effects and their relation to R_{os} , and the main findings will be reported in greater detail elsewhere (Ritz, Dahme, Thöns, & Quast, 2004).

In this study, participants (30 asthmatic, 30 healthy) viewed six blocks of five pictures with faces showing happy or angry emotional expressions. In a within-individual design they were instructed to respond to each picture (shown for 10 s) in one block of expressions with a congruent expression (e.g., with a happy expression to happy faces), with an incongruent expression (e.g., with an angry expression to a happy face), or with inhibition of expression (showing no expression at all). In addition, a block of control faces (surprise, nonemotional grimaces) was shown, with the instruction to mimic the expressions. During the picture sequences, EMGs were recorded again over corrugator supercilii, orbicularis oculi, and zygomaticus major muscle sites, and following the picture sequences R_{os} , RSA_c , IBI, and ventilation were measured for 2 min. Self reports of perceived effort, exerted control, and perceived authenticity of expression were also collected.

Adequate facial muscle activation under all conditions was verified by analysis of EMGs. Following a bronchosomatic coupling hypothesis we expected decreases in R_{os} from baseline following all picture blocks except those with the instruction to inhibit any expression. However, decreases in R_{os} were only observed in healthy participants and also for conditions of inhibition of expression. The a priori contrast between baseline and conditions with facial expression only yielded a statistical trend, $F(1,58) = 2.89, p = .095, d = 0.24$. For asthma patients, increases in R_{os} were found following each picture block. Between-individual correlations between average EMG activity and R_{os} showed the expected negative direction in healthy participants, with significant coefficients for orbicularis and zygomaticus under the congruent happiness condition. No other correlations were significant. Thus, skeletal muscle activation could not systematically explain the observed R_{os} changes, and evidence for bronchosomatic coupling was limited to one condition in healthy participants.

Summary and Outlook

In this series of studies, we explored the basic response of the airways to behavioral challenge, including voluntary muscle activation, emotion induction, and spontaneous and posed facial expression of emotion. Voluntary activation of the facial muscles leads to a reduction in respiratory resistance. There is good evidence that vagal withdrawal is among the mechanisms responsible for this change. This corresponds with literature on bronchodilating effects of dynamic exercise. The findings argue against the use of muscle relaxation techniques as an adjunctive behavioral treatment in asthma. Rather, it seems that brief dynamic exercise or facial muscle activation should be practiced to induce bronchodilation. Although indeed the bronchodilating effects of dynamic exercise have been employed to counteract asthmatic bronchoconstriction (e.g. Beck, Offord, & Scanlon, 1994), more research is warranted on effects of static muscle tension in areas other than the face. The location of the tensed muscle site relative to the airways and the respiratory muscles may be a critical factor. A challenge for the development of new

intervention techniques will be to find a positive balance between bronchodilating effects of brief activation periods on the one hand and possible exercise-induced bronchoconstriction of longer training periods with stronger ventilatory responses on the other hand. Interventions such as exercise training (Clark, 1992) or breathing training (Ritz & Roth, 2003) may be helpful in reducing exaggerated ventilatory response to skeletal muscle activation, thus allowing bronchodilatory effects of exercise to gain greater importance.

Negative emotions increase R_{os} ; this corresponds to clinical reports and earlier experiments with stress induction or bronchoconstrictive challenge (Isenberg et al., 1992). Although changes are qualitatively similar in healthy and asthmatic individuals, the effect size is sometimes stronger in asthma patients. Interestingly, although autonomic patterns of specific negative emotions, such as disgust, anxiety, anger, or depression, are usually not uniform (Cacioppo et al. 1993; Levenson, 1992), in terms of respiratory resistance only increases are observed across studies. For patients, laboratory-induced resistance changes correspond with daily-life lung function changes in negative emotional states and to their reports of the importance of psychological asthma triggers, thus providing validation for the concept of emotion-induced asthma. Positive emotional stimulation can also sometimes lead to resistance increases (see also von Leupoldt & Dahme, 2003), but the exact conditions of these changes require further exploration. The findings parallel observations from other researchers with HR, where sometimes only negative and other times positive and negative emotional stimulation lead to decreases or decelerations (e.g., Bradley, 1998). The onset of R_{os} changes is relatively fast, as our experiments with brief emotional stimuli have shown. Induction of tonic emotional states with film clips appeared to be more potent for triggering airway changes, reflecting findings of others with emotion induction using moving images (Simons, Detenber, Roedema, & Reiss, 1999). After film offset, R_{os} returns to baseline levels within 1–2 min.

The mechanisms of the emotion-induced airway responses are not yet fully elucidated. Using HR and/or RSA_c to estimate vagal outflow, we found that R_{os} was positively associated with vagal tone during or following depressing stimulation independently in two studies with asthma patients. It must be considered that the mechanisms leading to airway responses are not the same for different emotional states, and vary between healthy and asthmatic individuals. Whereas autonomic factors such as vagal excitation may play a role in depressive states (Miller & Wood, 1997), ventilatory changes may be more important in positive states. An association of slower breathing or longer T_1 with lower R_{os} in happiness was found for healthy participants in two studies. More research is needed on determinants of airway response to different emotional states and on emotional episodes in various contexts of daily life.

We also showed that stronger activation of facial muscles in emotional expression can be linked to lower respiratory resistance under some conditions. However, the skeletal muscle tension component can at best explain a small part of the resistance changes. Our findings on emotional expression effects on the airways are too sketchy to allow more far-reaching conclusions. Although an attractive idea, it is too early to recommend emotional expression as an element for an adjunctive behavioral treatment in asthma. Experimentally *posed* expressions were even associated with higher respiratory resistance in patients. More extreme expressive acts can also lead to bronchoconstriction by stronger changes in ventilation. Nevertheless, the instances in

which we found the predicted reductions in resistance encourage further research on the impact of emotional expression on the airways.

Although appealing from an early psychosomatic viewpoint, centrally mediated vagal excitation is by far not the only possible explanation for asthmatic bronchoconstriction. A host of other potential mechanisms have been outlined in recent years, such as muscarinergic receptor dysfunction, damage to the airway dilating NANC system, alterations in the excitability or activity in afferent pathways of the airways, or diminished ability of slowly adapting stretch receptors to dilate the airways (Barnes, 1992; Canning & Fischer, 2001). Each of these mechanisms deserves closer examination, which is hampered by the lack of noninvasive measures that would allow systematic psychophysiological experiments to estimate their importance. This also applies to other pathophysiological features of asthmatic airway obstruction, such as mucus secretion or airway edema. Our current understanding of asthma assigns a central role to processes of inflammation of the airway tissue (NIHLB, 2002). The exploration of

the psychoneuroimmunology of these processes opens an exciting new field for psychophysiology. Promising findings have been reported on links between psychosocial stress and inflammatory response in asthma in the last few years (e.g., Joachim et al., 2003; Kang et al., 1997; Liu et al., 2002; Wright et al., 2004). A challenge for future research will be the elucidation of mechanisms of emotional arousal effects on asthma-relevant immune parameters (IgE-levels, eosinophils, T-helper cell profiles, specific cytokines) and inflammatory mediators (e.g., histamine, tachykinins). Noninvasive techniques for measuring aspects of airway immune status and inflammation, such as induced sputum, exhaled nitric oxide, or breath condensate assessments, have recently been developed or are in the process of validation (Holz, Jörres, & Magnussen, 2000; Kharitonov & Barnes, 2001). A more complete understanding of emotional factors in asthma requires examination of the relationship between airway inflammation as the central pathophysiological process and lung function and respiratory symptoms as clinical endpoints of asthma both in acute exacerbations and chronic disease progression.

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