

Airflow and autonomic responses to stress and relaxation in asthma: The impact of stressor type

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Abstract

The impact of stress on respiratory airflow in asthmatics is unclear. Part of the uncertainty may spring from the different physiological effects of different stressors. Given their potential to elicit increases in parasympathetic vagal activity, stressful situations that present few opportunities for coping (passive coping stressors) may be particularly problematic for people with asthma. Thirty-one adult asthmatics participated in a protocol including a widely used passive coping stressor (the cold pressor test), an active coping stressor (mental arithmetic), an interview about an upsetting asthma-related incident (viewed as a potential passive coping stressor given the exposure to unpleasant memories), and progressive muscle relaxation. Repeated measurements of airflow (via peak expiratory flow), vagal tone (via heart rate variability), and other variables were obtained. The cold pressor test, asthma interview *and* progressive muscle relaxation produced significant decreases in airflow compared to the baseline period. The cold pressor test and progressive muscle relaxation produced significant, complementary increases in vagal tone. These results suggest that passive coping stressors and other stimuli (e.g., certain forms of relaxation) that elicit increased vagal tone may be associated with poorer asthma control, a view consistent with a significant negative correlation between the participant's mean vagal tone response to the tasks and score on a measure of asthma self-efficacy.

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1. Introduction

Clinicians and researchers have discussed and studied possible psychological influences on the course of asthma for many years. The idea that negative emotions or psychological stress may exacerbate asthma is widespread among those who suffer from this problem and many professionals. Yet despite anecdotal and some epidemiological evidence relating such states to symptoms, many have noted that those who propose a relationship between stress and impaired respiratory function in asthma face what appears on the surface a fundamental paradox

(Isenberg et al., 1992; Lehrer et al., 1996; Ritz et al., 2000). That is, several aspects of the generic stress response should, in principle, improve rather than impair respiratory function in asthmatics. For example, glucocorticoid release by the adrenal cortex might reduce respiratory inflammation and stress-related sympathetic nervous system activity should in principle produce bronchodilation rather than bronchoconstriction.

On the other hand, investigators have also known for many years that the idea of a generic stress response is an oversimplification (e.g., Ax, 1953; Engel and Moos, 1967) and that there are a number of stimulus-specific aspects of physiological reactions to challenging stimuli, some of which may interact with specific illnesses (e.g., Ditto, 1986; Steptoe et al., 1984). Some of these stimulus-specific effects may relate to asthma. For example, Miller et al. (2002)

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recently observed a significant impairment in the ability of a synthetic glucocorticoid to suppress pro-inflammatory interleukin-6 in a group of people exposed to long-term chronic stress (parents of children with cancer) and proposed that chronic stress and release of endogenous glucocorticoids may produce a state of glucocorticoid resistance and worsen problems related to inflammation.

Others have studied the effects of different kinds of acute emotions (e.g., Ritz et al., 2000) and stimuli (e.g., Lehrer et al., 1996) on respiratory function in asthma. A potentially important distinction may be the degree to which the stressor allows the person to exert some control over their situation. “Active” coping stressors that allow some control are well-known to elicit greater increases in beta- vs. alpha-adrenergic activity and decreased vagal parasympathetic activity while “passive” coping stressors that do not allow the person to control the situation generally elicit greater increases in alpha- vs. beta-adrenergic activity and less dramatic decreases, and occasionally increases, in parasympathetic vagal activity (Obrist, 1981). The most dramatic example of the impact of uncontrollable stress on parasympathetic activity is probably the vasovagal reaction, in which an uncontrollable stimulus such as an injection needle elicits strong vasoconstriction, an increase in vagal activity and a decrease in heart rate to the point where loss of consciousness may occur (Ditto et al., 2003; Engel, 1978). These patterns of autonomic activity suggest that passive coping stressors might be more problematic for asthmatics, a view consistent with the limited research on stimulus-specific effects in asthma.

For example, although Mathé and Knapp (1971) found that mental arithmetic, a potentially controllable stressor, produced reduced airflow in asthmatics, others have found either no effect (Micklich et al., 1973) or evidence of improved airflow in asthmatics following arithmetic (Lehrer et al., 1996). Lehrer et al. (1996) observed significant decreases in respiratory impedance during two active coping stressors—arithmetic and a reaction time task.

In contrast, a number of studies have observed significant decreases in airflow in asthmatics after passively watching unpleasant films (Levenson, 1979; Mathé and Knapp, 1971; Miller and Wood, 1997; Ritz et al., 2000). For example, Levenson (1979) found that asthmatics displayed significant increases in respiratory resistance while watching films about an industrial accident and hospitalized asthmatic children. One limitation of this important early study was that there was no measure of vagal tone. In the first study to explicitly test the idea that active and passive coping stressors might have differential effects on airflow in asthmatics, Lehrer et al. (1996) were also the first to assess vagal tone in this context via spectral analysis of heart rate variability (respiratory sinus arrhythmia). However, while they observed a significant decrease in respiratory impedance during the active coping stressors, they did not observe a significant change in impedance during films about an accident and thoracic surgery. On the other hand,

the films also did not produce a significant change in respiratory sinus arrhythmia. These films may have been insufficiently stressful or viewed as personally irrelevant by this particular audience. Thus, while the results of this study do not support the hypothesis that passive coping stressors are particularly problematic for asthmatics via their impact on vagal tone, they are not inconsistent with the hypothesis. The present study sought to evaluate changes in respiratory resistance among asthmatics in response to a somewhat more intense and immediately threatening passive coping stressor, i.e., the cold pressor test, as well as several other tasks, that is, mental arithmetic, a stress interview, and progressive muscle relaxation.

The cold pressor test is a widely used passive coping stressor which elicits pain and a defensive physiological response emphasizing vasoconstriction as opposed to the cardiac activation typically produced by active coping stressors (Allen et al., 1992; Peckerman et al., 1994). In a small study, Neild et al. (1984) found that the cold pressor reduced airflow in six asthmatic subjects. It was predicted that asthmatics would experience increases in respiratory resistance following the cold pressor. In contrast, it was predicted that mental arithmetic would produce decreases, or at least no increases, in respiratory resistance. The arithmetic task used in the present study was modeled on previous tasks used in this laboratory found to produce increases in beta-adrenergic activity (Miller and Ditto, 1991) and decreases in vagal tone (France and Ditto, 1992).

Given less previous research concerning the physiological effects of interviews, the predictions for this task were less firm. However, it was tentatively predicted that this task would also produce increases in respiratory resistance given that it was essentially a passive coping stressor involving recollection of an unpleasant, anxiety-provoking asthma-related event. Using similar procedures (Adler and Ditto, 1998), we observed significantly greater decreases in finger pulse amplitude, suggesting greater vasoconstriction, during interviews about anxiety-provoking events than anger-provoking events.

Finally, a significant increase in respiratory resistance following progressive muscle relaxation (PMR) was predicted. This might seem surprising given that PMR is neither a passive coping stressor or a stressor at all, but a generic stress-reduction technique that has been used widely with asthmatics (Kotses, 1998). However, relaxation has been found to produce increases in measures of vagal activity (e.g., Sakakibara et al., 1994) and the clinical effects of PMR on asthma are quite inconclusive (Huntley et al., 2002). Indeed, Lehrer et al. (1997) have suggested that in the short-term relaxation may actually lead to reduced airflow due to its impact on vagal tone, although this may be replaced by improvement after long-term practice due to a reduction in sympathetic activity and parasympathetic rebound. The present study sought to further examine the effects of this popular stress management technique on autonomic activity and airflow in asthmatics.

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