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Stress-induced breathlessness in asthma

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ABSTRACT

Background. A majority of patients with asthma believe that psychological factors (particularly stress) can induce asthma attacks, but empirical support for actual stress-induced airways obstruction is controversial. This study tested the hypothesis that stress induces breathlessness and not airways obstruction.

Methods. Stress was induced by a frustrating computer task in 30 adolescents with asthma and 20 normal controls, aged 14–19 years. Stress measures were self-reported emotions, heart rate, blood pressure. Respiratory measures were respiratory rate (RR), end tidal CO_2, deep inspirations and sighs. Asthma measures were lung function, wheeze, cough, breathlessness.

Results. All measures confirmed high levels of negative emotions and stress. None of the participants developed airways obstruction; they had no reduction in lung function, wheeze was absent and cough negligible. However, breathlessness increased in all participants with asthma and excessively in many. The mean breathlessness was higher than during induction of actual airways obstruction with provocative agents in previous studies. End tidal CO_2 showed that breathlessness could not be explained by hypocapnia.

Conclusion. Stress can be sufficient to induce breathlessness in patients with asthma.

INTRODUCTION

Empirical support for the existence of stress-induced airways obstruction in asthma is controversial. Asthma has long been considered prototypical for psychosomatic disease, and fluctuations in lung function were related to changes in emotional state. Extreme assumptions were that patients with asthma learnt to inhibit emotional expressions in order to prevent stress-induced asthma attacks, and that stress-induced asthma in children can be prevented by separating them from their parents (Purcell *et al*. 1969; Hollaender & Florin, 1983). There have been studies suggesting the influence of psychological factors on changes in lung function throughout the century, although they have generally been criticized for methodological flaws (Mackenzie, 1886; Luparello *et al*. 1968; McFadden *et al*. 1969; Clarke, 1970; Lewis *et al*. 1986; Hyland, 1990). In two more recent studies, peak expiratory flow rate (PEF)[1]† and mood state of patients were regularly measured in their homes, to assess relationships between lung function and emotional state over intervals of several weeks. In a study of seven patients, Steptoe & Holmes (1985) found a diminished PEF in two asthmatics when they were angry, tense or depressed, and in a third patient when either calm or elated.

Hyland (1990) was less successful and warned that mood could influence PEF, but that a diminished PEF would also affect mood. In neither of these studies did patients evidence actual airways obstruction[2] nor were results for breathlessness or physiological variables reported.

The cautious attitude among specialists regarding stress-induced airways obstruction is probably related to uncertainty about the potential causal mechanism involved (Isenberg *et al*. 1992; Busse *et al*. 1995). Emotional arousal is associated with sympathetic dominance and adrenergic activity, and the major pathophysio-

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† The notes will be found on page 1366.

logical mechanism in asthma is airways obstruction, with parasympathetic dominance and cholinergic activity (Feldman & McCrimmon, 1999). Epinephrine secretion during acute stress should relieve and not induce asthmatic reactions. Moreover, prolonged stress would trigger cortisol release with a reversing effect on airways obstruction (Feldman & McCrimmon, 1999). These paradoxes point to a complex involvement of, for example, alpha and beta adrenergic responses, different feedback loops as enervated by the vagal nerve, or immunological and neuroendocrine interactions which are not well understood. However, exercise-induced airways obstruction, one of the problems of many patients with asthma, seems to be associated with a distorted adrenergic/cholinergic balance after exposure of hypersensitive airways to rapid inhalation of relatively cold or dry air (Busse *et al*. 1995). Airways obstruction in asthma has also been documented as a result of rapid breathing during hyperventilation or lung-function testing (Jurenec & Ullman, 1984; Moran, 1991). Emotional breathing patterns could be the mechanism underlying stress-induced airways obstruction. Increases in respiratory rate and depth of breath are common during anxiety and fear, whereas anxious anticipation causes the respiratory rate to increase in conjunction with shallow breathing without pauses (Boiten *et al*. 1994).

A plausible alternative explanation for stress-induced airways obstruction would be a shift in immunological response after prolonged stress (O'Leary, 1990). However, there is no contemporary research available that includes emotional, respiratory and immunological variables to substantiate stress-induced airways obstruction. Nonetheless, most patients are convinced that emotions or stress often precede asthma attacks. Rumback *et al*. (1993) reported that 65% of asthmatic adults from a low economic background believed that feelings of anxiety or depression triggered their asthma symptoms. Almost 40% of mothers in a study by Weinstein (1984) complained that 5 min of crying induced cough or wheeze in their asthmatic child. However, from the patient's perspective, an asthma attack means breathlessness, rather than airways obstruction. Although breathlessness and airways obstruction in asthma generally coincide, there are physiological and psychological explanations for breathlessness without airways obstruction. First, individuals in a negative emotional state may breathe very quickly (hyperventilation) and cause hypocapnia, or breathe with prolonged expiratory phase towards hyperinflated lungs (dynamic hyperinflation). Both conditions evoke sensations including breathlessness, irrespective of airways obstruction (Cohen *et al*. 1975; Bass & Gardner, 1985; Goreczyn *et al*. 1988; Saisch *et al*. 1996).

Secondly, recent studies have shown that experimental manipulation of relevant information to patients with asthma in an ambiguous situation induces breathlessness without airways obstruction. Patients reported substantially more breathlessness after exposure to false asthmatic wheezing sounds, negative lung-function values of 30% below their actual value, or general sensations (Rietveld *et al*. 1997, 1999*a*; Rietveld, 1998).

In summary, empirical studies on the effects of stress in airways obstruction remained inconclusive. Patients' opinions are clear and so are recent findings about the psychological induction of breathlessness.

This study tested the hypothesis that stress in patients with asthma enhances breathlessness, not airways obstruction. This hypothesis was tested in adolescents with asthma and normal controls. Since a history of asthma would be essential for biased perceptions favouring stress-induced breathlessness, there was no effect expected in controls. Stress was defined as acute emotional arousal as indicated by self-reports and physiological measures.

METHOD

Participants

The study comprised adolescents because it was expected that pulmonary pathology due to environmental or long-term asthma would be less severe in adolescents. Moreover, some studies suggested that stress-induced asthma would be particularly pervasive in young people (Tal & Micklich, 1976; Weinstein, 1984). There were 30 adolescents with asthma and 20 normal controls, 13/10 boys and 17/10 girls, respectively. They were 14 to 19 years old (mean = 16·6, S.D. = 1·5).

The asthma patients were referred (without

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particular selection criteria) by general physicians in Amsterdam. The inclusion criteria were: a current diagnosis of asthma by a physician; and prescribed medication according to the classification of asthma severity (British Thoracic Society, 1993). The severity of asthma ranged from class one (prescribed bronchodilator medication) to class four (large concentrations of inhaled corticosteroids). The four classes were equally represented in the sample. None of the patients used long-term bronchodilators. Furthermore, patients were instructed not to use (short-term) bronchodilator medication on the test day. Normal controls were enroled via advertisements in a local newspaper. They were free of any known disease and had no history of respiratory complaints.

The experiment was officially approved by the ethics committee of the Department of Psychology. Participants and their parents were fully informed about the procedures of the study. However, expectations about changes in asthma symptoms after stress were not mentioned to the participants before testing. All participants gave informed signed consent. The participants were debriefed by a clinical psychologist. The participants were assured that they had performed very well but that the task had been extremely difficult and that the research assistants had not been friendly in order to maintain a stressful climate. Apart from the usual financial payment, all participants received a bonus.

Induction of stress

The participants were informed that they could earn 100 Dutch guilders by reaching the goal in a computer game. Money was visible during testing, and each minute a bank note of 10 guilders was withdrawn. The game was surprisingly difficult and lasted 10 min. Errors, or lack of progress, in the computer task were acknowledged by an 'unfriendly' assistant. Pilot testing had shown that the task was very stressful with a climax during minutes 8–9.

Measures

There were three groups of measures in this study: stress measures to confirm a successful induction of stress; respiratory measures to test the mediating effects of respiration on changes in asthma measures; and asthma measures to test the effect of stress on objective and subjective asthma variables.

Stress measures

Assessment of self-reported emotions

State anxiety was measured before and after the computer task (before debriefing) with the Spielberger State Anxiety Inventory (Spielberger, 1983). There were 20 statements with responses varying from 1 (not at all) to 4 (very much). The total score ranged from 20 to 80 points.

In addition, four items were presented in the same scale format, two items measuring test-specific disappointment/frustration, and two items measuring irritability/anger. Each total score ranged from 2 to 8 points.

Assessment of heart rate

The heart rate was continuously measured with a Polar Fitwatch (Semex Medische Techniek, Nieuwegein, The Netherlands). A band was strapped around the chest of each participant. The data were expressed in mean values during three intervals of 1 minute each: before the computer task, during minutes 8–9,[3] and after the task (after debriefing).

Assessment of blood pressure

Blood pressure was measured with an Imron HEM-711 sphygmomanometer (Fuzzy Logic, USA) and expressed in systolic and diastolic values on three occasions: before the computer task; immediately after the task; and after debriefing, approximately 10 min after the task.

Respiratory measures

Assessment of respiratory rate

The respiratory rate (RR) was measured by a Capnogard $etco_2$ Monitor (Novametrix, Medical Systems, Walingford, CT, USA). A tube was inserted in each of the participant's nostrils. The scores were transformed by a 12-bit Keithley Model 580 analog-to-digital converter and expressed in mm Hg. There were mean values during three intervals of 1 min each: before the computer task; during minutes 8–9; and after the task (after debriefing).

Assessment of deep inspirations and sighs

Tracheal sounds were continuously recorded for the assessment of deep inspirations and sighs (as

well as cough and wheeze; see paragraph *Assessment of cough and wheeze*) by wireless telemetry (Emco Electronics, Assendelft, The Netherlands). These respiratory variables were measured because they would influence the respiratory rate. Deep sighs were defined as obvious deviations from the normal breathing pattern. The number of deep inspirations and sighs in the sound records was scored by an experienced examiner and expressed in mean values during three intervals of 1 min each: before the computer task; during minutes 8–9; and after the task (after debriefing). These data were considered to be exploratory.

Assessment of end tidal CO_2

The end tidal carbon dioxide (CO_2) was continuously measured to detect hypocapnia with the Capnogard $etco_2$ Monitor (see paragraph *Assessment of respiratory rate*), and expressed in mean mm Hg during three intervals of 1 min each: before the computer task; during minutes 8–9; and after the task (after debriefing). The accuracy of the assessment was within 2 mm Hg from 0–40 mm Hg, and within 5% from 40–99 mm Hg.

Asthma measures

Assessment of lung function

Lung function was measured before and after the computer task (before debriefing) with a spirometer (Spirosense, Tamaraco Systems, Lode BV, Groningen, The Netherlands). The forced expiratory volume in one second (FEV_1) was used because it is the gold standard of lung physicians (British Thoracic Society, 1993). A second parameter of lung function used was, the forced vital capacity (FVC). The values were expressed as a percentage of the predicted value.

Assessment of cough and wheeze

Cough, was defined as an explosive burst of air followed by an expiration (Rietveld & Rijssenbeek-Nouwens, 1998). Although cough is a prominent symptom in asthma, its linear relationship with airways obstruction is marginal. Cough was measured because it may provide additional information about asthma in some patients (Rietveld & Rijssenbeek-Nouwens, 1998).

Wheeze, was defined as a secondary sound in the normal tracheal sound, for example high-pitched wheeze (Rietveld & Dooijes, 1996; Rietveld *et al*. 1999*b*). Wheeze is a sensitive and very specific marker of airways obstruction. Because of continuous sound recording, short-term wheezing would indicate event transient periods of airways obstruction.

An experienced examiner scored cough and wheeze on-line. A second examiner scored cough and wheeze by listening to recorded sounds and had no knowledge of the origin of these records. Previous research showed that inter-examiner reliability for scoring cough and wheeze during relatively short intervals is usually 100% (Rietveld & Dooijes, 1996; Rietveld *et al*. 1999*c*).

Assessment of breathlessness

Breathlessness, was defined and explained to the participants as laboured breathing, shortness of breath, or tightness of the chest. The degree of breathlessness was measured before the computer task and immediately after the task (before debriefing) with a self-report Likert-type scale. The responses ranged from 0 (not breathlessness) to 9 (most severe breathlessness). The scale has been used extensively in previous studies. A reduction of $\geqslant 20\%$ in FEV_1 generally coincided with a mean rise of $\geqslant 2{\cdot}5$ scale points in patients with asthma (Rietveld, 1998; Rietveld *et al*. 1997).

Statistics

The tests were conducted with an analysis of variance for repeated measures. A Greenhouse-Geisser epsilon correction for degrees of freedom was used when the sphericity assumption was not met. The significance criterion for statistical computations was set at $< 0{\cdot}05$.

RESULTS

Stress measures (Table 1)

Self-reported state anxiety, disappointment/frustration and irritability/anger all increased during the stress-inducing computer task. The testing effects were significant: $F(1,48) = 4{\cdot}15$, $P < 0{\cdot}01$; $F(1,48) = 10{\cdot}84$, $P < 0{\cdot}01$; and $F(1,48) = 23{\cdot}75$, $P < 0{\cdot}001$, respectively. State anxiety, disappointment/frustration and irritability/anger were higher in participants with asthma than in controls throughout the experiment. The group effects were significant: $F(1,48) = 193{\cdot}41$, $P < 0{\cdot}001$; $F(1,48) = 110{\cdot}21$,

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Table 1. *Means (and standard deviations) of stress measures in participants with asthma and controls*

	Asthma ($N = 30$)			Control ($N = 20$)		
	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)
Anx	31·43 (3·34)	— (—)	43·47 (7·18)	30·05 (3·09)	— (—)	40·20 (3·52)
Frus	2·17 (0·38)	— (—)	5·50 (1·76)	2·05 (0·22)	— (—)	4·05 (1·54)
Ang	2·10 (0·31)	— (—)	5·83 (1·80)	2·00 (0·00)	— (—)	3·75 (1·02)
HR	81 (5·82)	107 (6·89)	82 (5·96)	79 (3·52)	105 (6·73)	77 (4·36)
Sys	126·45 (10·86)	132·86 (10·90)	128·00 (12·35)	120·50 (9·42)	130·80 (11·39)	127·53 (10·50)
Dias	72·17 (7·98)	82·75 (7·84)	77·55 (6·32)	70·70 (7·86)	80·10 (9·81)	75·07 (7·76)
RR	18·93 (1·51)	27·50 (1·41)	18·30 (1·26)	17·45 (1·67)	27·70 (1·17)	16·50 (1·85)

Peak, mean of minutes 8–9; Anx, state anxiety; Frus, test-specific disappointment/frustration; Ang, test-related irritability/anger; HR, heart rate; Sys, systolic volume; Dias, diastolic volume; RR, respiratory rate.

Table 2. *Means (and standard deviations) of respiratory measures in participants with asthma and controls*

	Asthma ($N = 30$)			Control ($N = 20$)		
	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)
RR	18·93 (1·51)	27·50 (1·41)	18·30 (1·26)	17·45 (1·67)	27·70 (1·17)	16·50 (1·85)
Ins	0·07 (0·25)	0·20 (0·41)	0·10 (0·31)	0·05 (0·22)	0·05 (0·22)	0·10 (0·31)
Sig	0·03 (0·18)	1·20 (0·66)	0·30 (0·47)	0·00 (0·00)	1·20 (0·52)	0·05 (0·22)
CO_2	35·10 (2·11)	35·14 (2·16)	34·17 (2·28)	36·35 (1·93)	36·55 (1·83)	35·60 (1·75)

RR, respiratory rate; Ins, deep inspirations; Sig, deep sighs; CO_2, end tidal CO_2.

$P < 0·001$; and $F(1, 48)147·25$, $P < 0·001$, respectively. According to the interaction effect, irritability/anger increased significantly more after the stress-inducing task in participants with asthma than in controls: $F(1, 48) = 19·26$, $P < 0·01$. There were no significant interaction effects for state anxiety and disappointment/frustration ($P > 0·05$).

In addition to these self-report measures, virtually all physiological measures increased from baseline to stress-induction. Heart rate, systolic and diastolic blood pressure increased significantly during the stress-inducing task: $F(1, 48) = 7·36$, $P < 0·01$; $F(1·91, 87·84) = 40·35$, $P < 0·001$; and $F(2, 46) = 2891·49$, $P < 0·001$, respectively.

Throughout the experiment heart rate was significantly higher in participants with asthma than in controls and diastolic blood pressure was nearly so: $F(1·77, 48·76) = 386·72$, $P < 0·001$; and $F(1, 47) = 3·67$, $P = 0·061$, respectively. There was no significant group effect for systolic blood pressure and there were no significant interaction effects for heart rate, systolic or diastolic blood pressure ($P > 0·05$).

Respiratory measures (Table 2)

The respiratory rate increased significantly during the stress-induced task and was significantly higher in participants with asthma than in controls. The testing and group effects were significant: $F(1, 48) = 11·74$, $P < 0·001$; and $F(0·98, 95·24) = 681·43$, $P < 0·001$, respectively. When looking at the interaction effect, it was evident that participants with asthma breathed faster before and after the test, whereas the groups breathed equally fast during emotions or stress: $F(1·98, 95·24) = 9·62$, $P < 0·01$.

The number of deep inspirations remained stable but marginal throughout the experiment and there were no significant differences between assessments or between groups ($P > 0·05$).

The number of deep sighs also remained marginal, but increased significantly during the stress-inducing task. There was a significant testing effect: $F(1·51, 72·49) = 125·56$, $P < 0·001$. There was no significant difference between groups and no interaction effect ($P > 0·05$).

End tidal CO_2 was rather low throughout the study and significantly lower in participants

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Table 3. *Means (and standard deviations) of asthma measures in participants with asthma and controls*

	Asthma ($N = 30$)			Control ($N = 20$)		
	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)	Pre-test Mean (S.D.)	Peak Mean (S.D.)	Post-test Mean (S.D.)
FEV_1	92·67 (8·30)	— (—)	92·63 (8·41)	99·65 (3·30)	— (—)	98·95 (3·39)
FVC	96·70 (6·10)	— (—)	96·63 (5·74)	99·85 (3·50)	— (—)	99·55 (3·07)
Cgh	0 (0)	0 (0)	4 (—)	0 (0)	0 (0)	1 (—)
Wh	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Br	0·33 (0·61)	— (—)	4·93 (1·74)	0·30 (0·57)	—(—)	0·50 (0·83)

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; Cgh, cough; Wh, wheeze; Br, breathlessness.

with asthma than in controls. The group effect was significant: $F(1{\cdot}97, 87{\cdot}18) = 14{\cdot}36$, $P < 0{\cdot}05$. Although there was also a significant testing effect, this was the opposite of that expected: the difference was accounted for by the post-test assessment (Table 2): $F(1, 47) = 9{\cdot}18$, $P < 0{\cdot}01$. There was no significant interaction effect ($P > 0{\cdot}05$).

Asthma measures (Table 3)

The lung function (FEV_1, FVC) remained stable throughout the experiment ($P > 0{\cdot}05$). The FEV_1 and FVC were consistently lower among participants with asthma than in controls throughout the experiment. The group effect was significant: $F(1, 48) = 11{\cdot}52$, $P < 0{\cdot}001$; and $F(1, 48) = 4{\cdot}42$, $P = 0{\cdot}04$, respectively. Following these results, the interaction effects between testing and group were not significant ($P > 0{\cdot}05$).

Breathlessness increased significantly during the stress-inducing task. The testing effect was significant: $F(1, 48) = 87{\cdot}68$, $P < 0{\cdot}001$. There were significant group and interaction effects, showing that participants with asthma reported significantly more breathlessness during the whole test, but particularly after the stress-inducing task. The group and group × testing interaction effects were significant: $F(1, 48) = 127{\cdot}12$, $P < 0{\cdot}001$; and $F(1, 48) = 106{\cdot}81$, $P < 0{\cdot}001$.

The presence of cough was negligible, and wheeze was not observed.

DISCUSSION

Stress was successfully induced, as was evident from increased physiological and self-report measures. None of the 30 participants with asthma or the 20 without asthma developed airways obstruction as reflected in a decreased lung function (FEV_1 or FVC). Other measures – end tidal CO_2 and absence of cough and particularly wheeze – confirmed that pulmonary condition remained stable from baseline to stress-induction. However, breathlessness at least doubled in all participants with asthma and excessively so in many of them. Breathlessness did not increase in the controls.

The mean values on end tidal CO_2 did not decrease during stress-induction, which precluded the influence of hypocapnia in excessive breathlessness.

The overall results of the study supported the hypothesis that stress, in adolescents with asthma, induces breathlessness and not airways obstruction. The results were very convincing because of the excessive exhibited levels of emotion and stress. Several adolescents expressed disbelief when tests indicated that their lung function had not changed. The mean rise in breathlessness was 4·6 scale points, which was quite impressive. Even less breathlessness was reported during actual airways obstruction after inhalation of histamine in a previous study (Rietveld *et al.* 1997). Since there was no objective explanation for excessive breathlessness, it could be argued that breathlessness was a psychological construction. The adolescents with asthma falsely interpreted breathlessness on the basis of ambiguous physical sensations in a situation that they had learned to associate with asthma. Rietveld & Brosschot (1999) emphasized that patients have learned to associate symptoms with particular contexts. When they enter such a situation (high emotional arousal) they are likely to interpret falsely ambiguous or general sensations in terms of the expected symptoms. This seems to be a general

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phenomenon in patients and is not restricted to patients with anxiety problems (cf. Tiller, Pain & Biddle, 1987; Robbins & Kirmayer, 1991).

The current results were in line with studies based on patients' recall that asthma is often preceded by emotions or stress. However, the results were contrary to studies that substantiated a causal relationship between emotions or stress and airways obstruction (Luparello *et al*. 1968; Clarke, 1970; Tal & Micklich, 1976). It is possible that motivational and emotional factors had a negative influence of lung-function testing in previous research, affecting empirical conclusions about stress-induced airways obstruction (Hyland, 1990; Isenberg *et al*. 1992). Other methods to induce stress and different emotions may account for contradictory results between this and former studies.

There were several limitations with the current study. First, there were only 30 patients with asthma and it is possible that by mere chance none of them was sensitive to stress-induced airways obstruction. Moreover, it is possible that only emotionally stable patients (and controls) had agreed to participate in an experiment addressing 'an emotional computer game'. Secondly, although two lung-function parameters were measured, a more sensitive measure such as airways conductance would have been informative. Although induced airways obstruction can be transitional, the bulk of the literature confirms the validity and reliability of lung function testing during airways challenge tests. In fact, many studies that did show changes in 'lung function' after psychological manipulation or emotional states used the mini-peakflowmeter instead of the more reliable spirometer used in this study. The reliability of the peakflowmeter has often been disputed (Higgs *et al*. 1986; Rietveld *et al*. 1996). Regarding this influence, it remains the case that controls did not report much breathlessness, whereas the respiratory rate at the peak of induced stress in asthmatics was almost similar to that found among controls, which would argue against a strong impact of respiratory variables in excessive breathlessness (Table 2). A qualitative analysis of deep inspirations and sighs was conducted to correct for changes in respiratory rate. However, RR was not influential in either lung function or breathlessness, as evidenced by some non-significant correlations (not presented). Moreover, Table 2 shows that deep inspirations and sighs remained marginal. Further analysis was then omitted. Note that Butler & Steptoe (1986) argued that psychological induction of airways obstruction is not related to respiratory rate. As another limitation, the current methods did not include assessment of airflow and minute volume. Assessment of these variables was considered, but the equipment alters the natural breathing pattern and was also difficult to combine with all other experimental procedures (Western & Patrick, 1988). When we again examined the literature in search of cases during which airways obstruction was confirmed after psychological manipulation, only a few cases were found: for example, a girl who actually experienced asthma after a stressful film (Tal & Micklich, 1976). Situational details of this study are not mentioned. Could the experimental setting have contained airway-provoking allergens? Obviously, more research is required to solve this puzzle.

New research should be focused on breathing patterns in different (including emotional) situations. Body plethysmography is a complex method for testing lung function but would be helpful in detecting the potential involvement of dynamic hyperinflation during stress-induced breathlessness. The assessment of sympathetic and parasympathetic activity before and during airways obstruction would be interesting, particularly with regard to the autonomic control of respiration during stress. This could be done by continuously measuring respiratory sinus arrhythmia (RSA) and pre-ejection period (PEP). Assessment of sputum cortisol and possibly asthma-specific immunological parameters would be helpful to establish relationships in the complex pattern of interactions between stress, respiration, immunological responses and asthma symptoms.

Nonetheless, severe complaints of asthma symptoms may often be a subjective instead of an objective problem. Hence, excessive illness behaviour including medicinal use in emotionally vulnerable patients should often be questioned.

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NOTES

[1] One of the lung-function parameters, assessed by patients themselves with a mini-peakflowmeter. This simple plastic device is often used in the self-management of asthma.

[2] A reduction of $> 20\%$ in forced expiratory volume in 1 s (FEV_1) is generally considered too indicate a significant degree of airways obstruction. This state is clinically relevant, warranting bronchodilator medication.

[3] These minutes represented the peak of emotional stress, as evidenced from *post hoc* analysis.

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