

Anger and lung function

# Anger, stress, dysregulation produces wear and tear on the lung

P Lehrer

## A link between chronic anger and age related deterioration in pulmonary function

The relationship between psychological and physical states has been known to medicine since the time of the ancient Greeks.<sup>1</sup> Although at one time patients with diseases of unknown origin (such as tuberculosis in the 19th century) were labelled as suffering from a psychological or moral malady, this error (and often injustice) does not negate truths about the mind/body relationship in disease. Indeed, scientific interest in and speculation even about mind/body effects on tuberculosis remains high.<sup>2</sup>

In this issue of *Thorax*, Kubzansky *et al*<sup>3</sup> have published an important paper on the relationship between decline in lung function in older men and the emotional characteristic of anger. This coincides with a considerable literature on the relationship between anger and cardiovascular disease.<sup>4</sup> As the “fight” component in the fight-flight reflex, the psychophysiology of anger overlaps with that of stress. Stress related factors are known to depress immune function<sup>5</sup> and increase susceptibility to or exacerbate a host of diseases and disorders including asthma,<sup>6</sup> hypertension,<sup>7</sup> upper respiratory infection,<sup>8</sup> various skin diseases,<sup>9</sup> chronic fatigue syndrome,<sup>10</sup> irritable bowel syndrome,<sup>11</sup> vasovagal syncope<sup>12</sup> and, more obviously, various psychiatric disorders. Indeed, it is hard to find a disease for which emotion or stress plays absolutely no part in symptom severity, frequency, or intensity of flare-ups.

Stress appears particularly related to the occurrence and severity of asthma. Asthma exacerbations have been linked to the occurrence of life stressors.<sup>13</sup> Certain psychiatric disorders, including anxiety disorders (particularly panic symptoms or panic disorder<sup>14</sup>) and affective disorders,<sup>15</sup> are unusually common among asthma patients. Asthma severity and exacerbations have been linked to the presence of psychiatric disease through several pathways including: (1) direct physiological effects of emotion or stress on the airways, (2) the effect of emotion on self-care, and (3) the effect of having a chronic physical disease on emotional state. Laboratory induced

interpersonal stress,<sup>16</sup> anger,<sup>17</sup> and sadness<sup>6</sup> have also been linked to decreases in pulmonary function, even though the acute effect of physical mobilisation and fight-flight preparedness causes sympathetic arousal and bronchodilation.<sup>18</sup> A particularly poignant and unexpected example of anger induced asthma exacerbation was reported among children at a summer asthma camp who had been given a course in assertion training.<sup>19</sup> Although, at the time, such training for greater interpersonal effectiveness was thought to carry the salutary qualities of stress reduction, children given this intervention showed a deterioration in asthma, perhaps because of greater expression of anger. There is considerable evidence for a relationship between negative emotion of all sorts in asthma (anger, anxiety, sadness) and deterioration in pulmonary function.<sup>20</sup>

Thus, one connection between lung disease and anger may be through the physiological effects of chronic anger. Reported psychophysiological accompaniments of chronic anger include sympathetic arousal,<sup>21</sup> an increased nor-epinephrine:epinephrine ratio and vasoconstriction,<sup>22</sup> increased serum lipids and low density lipoproteins,<sup>23</sup> decreased expression of the anti-inflammatory cytokine interleukin-10 in response to exposure to influenza virus,<sup>24</sup> and an increased expression of the pro-inflammatory tumour necrosis factor- $\alpha$  following lipopolysaccharide stimulation.<sup>25</sup> No research has yet been reported on the effects of anger on airway or vascular inflammation, but basic research on inflammatory cytokines certainly suggests that anger may increase inflammation.

Healthy adaptation requires not only the capacity to change blood flow, ventilation, etc in order to meet the metabolic needs of behavioral demands (such as in the fight-flight reflex), but also the ability to return to normal limits, to allow tissue regeneration, and to relieve physical and chemical stresses on various body organs. In the absence of such balance, dysregulation occurs, producing symptoms and exacerbation of disease and sometimes death. Physiological regulation entails a

combination of set points, reactivity, and gain in various modulatory reflexes (well quantified, for example, in the baroreflex).<sup>26</sup> These reflex parameters can be reset by various physical and emotional stressors<sup>27</sup> which may lead to chronic dysregulation. Thus, dysregulation of airway cytokine expression<sup>28</sup> and of vagus nerve function<sup>29</sup> are both thought to occur in chronic obstructive pulmonary disease and asthma. Decreased heart rate variability also occurs in asthma,<sup>30</sup> indicating autonomic dysregulation and impaired adaptability which may be either a contributing cause or effect of the lung disorder.

Increased chronic anger may have particularly deleterious effects on the body because it prevents homeostatic release and may change the set points for modulatory reactions. It often reflects chronic personality maladjustment or, in some cases, chronic exposure to job or marital dissatisfaction which perpetuate anger and its physiological accompaniments. Thus, although temporary changes in emotion or stress—which occur normally in all of us—may temporarily affect our adaptability and susceptibility to various diseases, chronic anger—as assessed in the paper by Kubzansky *et al*<sup>3</sup> in this issue of *Thorax*—might be expected to produce more long lasting effects. Although a healthy ability to express a wide range of emotion is generally considered to be a sign of health and good adaptation, chronic anger may lead to chronic dysregulation.

Although the exact pathways whereby chronic anger contributes to chronic physical deterioration are not known, it is not hard to imagine how the wear and tear associated with chronic anger could produce chronic dysregulation and, ultimately, physical deterioration. The paper by Kubzansky *et al*<sup>3</sup> establishes a link between chronic anger and age related deterioration in pulmonary function. The next step is to determine the exact pathway by which this happens.

In this, as in all research, however, the reader should interpret the data cautiously. Correlation does not necessarily imply causation, even when “trait” measures of anger (or any other state) are relatively stable. Personality, as well as physiology, can change over time, and deterioration in health and physical function can lead to negative emotion as well as vice versa, including for respiratory diseases.<sup>31</sup>

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Correspondence to: Dr P Lehrer, Department of Psychiatry, UMDNJ-Robert Wood Johnson Medical School, Piscataway, NJ 08854, USA; lehrer@umdnj.edu

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Spirometric screening

## Spirometric screening: does it work?

D M Mannino

### Role of spirometric testing in smoking cessation

Pulmonary function testing offers an easy, inexpensive, and non-invasive means of diagnosing and staging chronic lung disease.<sup>1,2</sup> It provides information on both the presence of obstructive lung disease and restrictive lung disease and can provide insights on how patients might respond to treatment.<sup>3,4</sup> Spirometric testing also provides prognostic information, with lung function measures predicting mortality and the development of lung cancer.<sup>5,6</sup>

Despite the valuable information that spirometric testing provides, it is underused in medical practices in much of the world. There are several reasons for this, including (1) problems with doing the procedure,<sup>7</sup> (2) problems related to compensation, and (3) the absence of “evidence” that spirometric testing actually makes a difference in the diagnosis and treatment of patients. Advances in the design of spirometric

tests that provide quality control feedback are addressing the first reason. The second reason varies between locales and health plans. Addressing the final reason is critical to increasing the use of spirometry in a general medical practice.

A recent review commissioned by the United States’ Agency for Health Research and Quality by Wilt *et al*<sup>8</sup> concluded that “the evidence does not support widespread use of spirometry in primary care settings for all adults with persistent respiratory symptoms or having a history of exposure to pulmonary risk factors for case-finding, improving smoking cessation rates, monitoring the clinical course of COPD, or adjusting COPD interventions”. With specific regard to smoking cessation, the report’s review of four studies in the literature concluded the following:

“Spirometric testing as a motivational tool to improve smoking cessation rates is unlikely

to provide more than a small benefit. Results from observational studies of spirometry are mixed. RCT of other biomarkers used as motivational tools for smoking cessation are generally negative. The only randomized controlled trial that assessed the independent contribution of spirometry and counseling on smoking cessation rates reported a nonsignificant 1 percent greater quit rate at 12 months in the group assigned to receive spirometry ...”<sup>8</sup>

The paper in this issue of *Thorax* by Bednarek *et al*,<sup>9</sup> which is observational and not a randomised clinical trial, would not have been included in the review by Wilt *et al* and thus would not have changed the conclusion. The information in the study by Bednarek *et al* is, however, compelling. Their evidence suggests that spirometric testing, with a very quick and simple feedback consisting of a lung function decline curve marked with the patient’s value, improved smoking cessation. In the world of smoking cessation, the validated cessation rates at 1 year of 16.3% in the overall group was higher than the expected 4–6%,<sup>10,11</sup> and there was evidence that lower lung function at baseline resulted in higher cessation rates. Even though this was not a randomised trial, these results are remarkable and should be a model for designing a study to determine whether this quick and