Thorax 2002;**57**(Suppl II):ii31–ii35

DYSFUNCTIONAL BREATHING IN ASTHMA: IS IT COMMON, IDENTIFIABLE AND CORRECTABLE?

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Introductory article

Prevalence of dysfunctional breathing in patients treated for asthma in primary care: cross sectional survey

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*Objectives: To estimate the prevalence of dysfunctional breathing in adults with asthma treated in the community. Design: Postal questionnaire survey using Nijmegen questionnaire. Setting: One general practice with 7033 patients. Participants: All adult patients aged 17–65 with diagnosed asthma who were receiving treatment. Main outcome measure: Score ⩾23 on Nijmegen questionnaire. Results: 227/307 patients returned completed questionnaires; 219 (71.3%) questionnaires were suitable for analysis. 63 participants scored ⩾23. Those scoring ⩾23 were more likely to be female than male (46/132 (35%) v 17/87 (20%), p=0.016) and were younger (mean (SD) age 44.8 (14.7) v 49.0 (13.8), p=0.05). Patients at different treatment steps of the British Thoracic Society asthma guidelines were affected equally. Conclusions: About a third of women and a fifth of men had scores suggestive of dysfunctional breathing. Although further studies are needed to confirm the validity of this screening tool and these findings, these prevalences suggest scope for therapeutic intervention and may explain the anecdotal success of the Buteyko method of treating asthma. (**BMJ** 2001;322:1098–100)*

BACKGROUND

One remarkable thing about the physiology of the human body is the ability to adapt to constantly changing circumstances and adverse environments. In the respiratory system arterial blood gas tensions are usually held within tight limits of normality in the face of gravitational changes in posture, the requirements of speech and exercise, as well as many other human activities. Any tendency for ventilation to stray beyond these limits is rapidly corrected by appropriate homeostatic mechanisms that operate imperceptibly. In theory, abnormal ventilatory equilibrium can be produced by sustained hyperventilation or hypoventilation. However, the latter is only likely to occur in serious organic disease or drug induced narcosis when the cause is usually obvious. Nevertheless, the potential for variability exists and it has been recognised for decades that the pattern of ventilation that develops in advanced chronic obstructive pulmonary disease (COPD) is independent of the airway pathology. Some patients will defend their arterial carbon dioxide tensions as pink puffers while others will passively accept the comfort and consequences of hypercapnia as blue bloaters. This behaviour in COPD is not voluntary or psychogenic but represents an intrinsic—possibly genetically determined—reaction to illness. Since in other circumstances the strong intrinsic physiological defence mechanisms would make sustained voluntary hypoventilation unachievable, it has become accepted in this instance that the phenotypic appearance of the patient in respiratory failure is due to an interaction of lung pathology and a predetermined pattern of respiratory drive. As a consequence, the presence of an altered breathing pattern in COPD is seen as an epiphenomenon rather than the root cause. This view is not necessarily accepted in asthma where the impression of linkage between breathing pattern and pathology appears less acceptable. Sustained hyperventilation, surplus to physiological requirement, can be induced by conscious action, behavioural change, pharmacological stimulation, or neurological disease. As every child will have learnt, deliberate conscious and temporary interference with homeostasis by hyperventilation is relatively easy to achieve. Acute deliberate or psychogenic hyperventilation can be associated with distressing physical symptoms that are usually recognised as such and traditionally treated by rebreathing or anxiolysis. Chronic or recurrent changes in breathing pattern have also been associated with physical symptoms of breathlessness, chest tightness, chest pain, dizziness, and anxiety that are

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variously described as dysfunctional breathing or hyperventilation syndrome.[1–4] This behaviour can masquerade as asthma or occur in association with genuine asthma or other airways disease.This association has always presented difficulties for clinicians who tread a fine line between inadequately treating naturally anxious people with asthma or prescribing unnecessary and excessive treatment where it will be ineffective. It would therefore be helpful to have a method of distinguishing the contribution of possible dysfunctional breathing from the inflammatory or bronchoconstrictor components of genuine asthma. One obvious solution is to titrate preventative corticosteroid treatment to the inflammatory response so that patients only receive the asthma treatment that they need. This approach is promising and is currently being explored with a variety of markers of airway inflammation.[5] The bronchoconstrictor component of asthma is traditionally managed by a combination of physiological monitoring and symptom control by bronchodilator. Although the latter strategy is adequate for most situations, it will not be helpful where symptoms of dysfunctional breathing are interspersed with those of airway narrowing. For that reason it would be helpful to be able to identify and distinguish the effects of dysfunctional breathing so that the true contribution can be assessed and alternative or additional treatment offered where appropriate or possible.

In this respect, the Introductory article by Thomas *et al*[6] should be considered alongside the accompanying editorial by Keely and Osman.[7] Thomas *et al* have used a simple questionnaire to identify the prevalence of dysfunctional breathing in a general practice population that has previously been defined as asthmatic. They identified a high prevalence of dysfunctional breathing across all grades of severity of asthma, particularly in women. Superficially, the study is fairly innocuous and appears to make a useful contribution to the body of knowledge in this area. It seems to define the size of the problem and point the way to alternative treatment for those who are inappropriately treated. However, there are several assumptions within the methodology and the discussion which make the conclusions difficult to generalise. As a result, the study raises almost as many questions as it answers and unscrambling the eggs from the chickens is difficult. The main issues can largely be considered under three headings.

Can dysfunctional breathing be defined and identified?

The term "dysfunctional breathing" is not a precise or definable entity. It ascribes causality to a pattern of abnormal breathing that may result at worst in a symptom complex of breathlessness, chest tightness, paraesthesiae, anxiety, and dizziness. This may include overt hyperventilation and hypocapnia but also includes more subtle and subjective features that are difficult to characterise. This new term of description presumably includes overt episodic hyperventilation, chronic hyperventilaton syndrome, and disproportionate breathlessness. This creates something of a difficulty since, in the context of this paper, the condition is defined by the method used to recognise it. Dysfunctional breathing is therefore just the latest imprecise description of a behaviour and symptom complex which remains unexplained.

Physiological alveolar hyperventilation leads to hypocapnia and respiratory alkalosis that may lead to neuronal hyperexcitability. The initial description of hyperventilation syndrome was used to identify people who developed somatic symptoms and anxiety associated with overbreathing. This description has since become corrupted to include those with anxiety and appropriate somatic symptoms even when hypoventilation is not obvious or constant. In fact, the relationship between the apparently recognisable symptom complex and hypocapnia is far from clear. Although characteristic symptoms can be reproduced by voluntary hyperventilation, the demonstration of hypocapnia is not a reliable diagnostic test.[8 9] Hypocapnia in symptomatic people may be episodic and may not relate directly to the symptoms. Ambulatory monitoring of arterial $Paco_2$ or end tidal CO_2 has not yet provided evidence of a relationship between symptoms and hypocapnia. In fact, it is evident that hyperventilation and hypocapnia can occur in the complete absence of symptoms. If there is no physiological observation that characterises the syndrome, it may be possible that a provocation test might reproduce the symptoms. In normal people voluntary hyperventilation will produce tetany or paraesthesia but not the wider range of symptoms. Some simple provocation like the 20 deep breath test may reproduce the symptoms and suggest the diagnosis. More formal observation of prolonged hypercapnia associated with short periods of hyperventilation has also been proposed. Unfortunately the validity of this approach is partly undermined by the observation that symptoms can still occur when normal $Paco_2$ is artificially maintained.[10] It is now generally acknowledged that the syndrome need not necessarily be linked to hypocapnia but more to the act of overbreathing.[2] The term hyperventilation remains, but other descriptors such as disproportionate breathlessness may be more apt. Other physiological approaches have examined the erratic ventilatory response to exercise that can disclose inappropriate ventilation. Another concept is that the syndrome may result from a disorder of physiological control or autonomic failure. So far, no specific abnormality of CO_2 regulation has been identified, but changes in ventilation with postural challenge have been observed.[11]

If there is no satisfactory physiological definition of dysfunctional breathing, perhaps the answer can be found in a psychological explanation. The patient with obvious air hunger, sighing respiration, or episodic hyperventilation may well have a psychiatric disorder and respond to appropriate treatment. However, the waters are very muddied and dismissal of dysfunctional breathing as a manifestation of panic attack is considered by both psychiatrists and physiologists as an oversimplification.[1] It is established that some people with asthma may have impaired perception of dyspnoea that could be potentially dangerous. It is also possible that the spectrum extends to those who develop an enhanced sensitivity to airway responsiveness or airway conductance and react by hyperventilating. More overtly, people with anxiety may overbreathe, but then distressing asthma is a perfectly understandable source of anxiety.

In this case the syndrome of dysfunctional breathing therefore defines itself by the self-reinforcing symptom check list which was developed from those with a clinical diagnosis of hyperventilation. The Nijmegen questionnaire is therefore different from other disease specific health status measures where the primary diagnosis is predefined by independent criteria. In the original validation of the questionnaire there was some crossover with a healthy reference group.[12] No validation was performed with people who had other causes of breathlessness. It is therefore difficult to determine the discriminative properties of the questionnaire when many of the 16 relevant symptoms might appear in genuine respiratory disease. Furthermore, people with a positive

Nijmegen result may or may not exhibit spontaneous hypocapnia or response to provocation.[9] It seems therefore that a clinical syndrome of symptoms exists which may be loosely linked to overbreathing and formally described by the structured clinical history of the Nijmegen questionnaire. However, physiological definition or recognition of the syndrome does not exist and clear separation of the condition by the questionnaire from genuine organic disease has not yet been made. This does not mean that dysfunctional breathing is not an entity, but does make it difficult to quantify in the suspected presence of asthma.

How does dysfunctional breathing relate to asthma?

Acute and chronic hyperventilation are the components of dysfunctional breathing that are most likely to relate to asthma. Acute asthma attacks are often associated with a reduction in $Paco_2$ in association with developing hypoxaemia. This is expected as the natural attack develops and reverses with effective treatment. Similar acute hyperventilation also occurs with induced bronchoconstriction through bronchial challenge and is again reversible. The converse is observed when acute bronchospasm is induced by deliberate hyperventilation or increased ventilation associated with physical exercise. In this case the mechanism of bronchial challenge is thought to include airway cooling and drying. These acute situations are well recognised and appropriate treatment or advice can usually resolve the problem.

The relationship between chronic hyperventilation and asthma is more complicated. Several papers have reported a high incidence of covert respiratory disease in patients who have been clinically diagnosed with the hyperventilation syndrome. In particular, underlying asthma has been identified as the most common cause of confounding illness. In these cases the identification of asthma has been made by bronchial challenge, a bronchodilator response, or improvement with treatment. In one series 80% of subjects with the hyperventilation syndrome turned out to have asthma.[13]

The reverse situation of hyperventilation occurring naturally in mild asthma does not appear to be true except during periods of bronchial hyperresponsiveness.[11] Interestingly, there is a circumstance where the airways might be exposed to prolonged high levels of ventilation where airway cooling and drying may occur. Athletes, particularly those involved in winter sports, may be exposed to airway challenge in this way. There is evidence that asthma is more common in these people and that increased airway responsiveness is associated with the environmental challenge.[14] Experimentally, repeated hyperventilation has also led directly to peripheral airway inflammation, hyperreactivity, and impaired bronchodilation in dogs.[15]

It therefore appears that hyperventilation can occur in association with acute asthma and possibly contribute to a hyperreactive state in extreme conditions. It is more difficult to uncover a relationship between asthma and other features of dysfunctional breathing. Some of the symptoms will obviously overlap. Breathlessness, chest pain, chest constriction, and accelerated breathing are common to asthma and the Nijmegen questionnaire. Even some of these somatic symptoms could reasonably be experienced by any person with a worrying illness. It is possible that some of the somatic symptoms could be an expression of an enhanced perception of dyspnoea. However, when this has been examined in severe asthma the opposite appears to be true. Some patients with severe asthma have a poor perception of dyspnoea and therefore put themselves at risk.[16]

The main emphasis of the Introductory article is not to suggest that hyperventilation or dysfunctional breathing is the cause or result of asthma. The suggestion is that the diagnosis of asthma may be incorrect in some cases, or at least only part of the problem. This is a perfectly reasonable hypothesis but, in the absence of a specific and discriminatory test, it is difficult to prove.

Can dysfunctional breathing be treated effectively?

There is an assumption in the paper by Thomas *et al*[6] that dysfunctional breathing can be effectively treated after it has been recognised. The evidence for this assumption needs to be explored. In spite of the apparent definition of the condition by the clinical syndrome or the Nijmegen questionnaire, there is little good quality research to support effective treatment. There is some limited evidence for the effect of beta blockade and breathing retraining in overt hyperventilation, but very little information about treatment in the context of other additional lung disease. If hyperventilation is directly related to underlying illness such as asthma or pulmonary embolism, then treatment of the primary condition may be all that is required. Specific independent treatment for dyspnoea or hyperventilation should only be necessary if these features cannot be controlled by primary therapy. COPD is a good example of a condition where the specific symptom of dyspnoea can be effectively detached from the underlying disease. In this case, exertional dyspnoea can be treated beyond efforts to produce bronchodilation by a number of treatment modalities.[17 18] Pulmonary rehabilitation modulates dyspnoea predominantly through the vehicle of physical exercise training. Additional benefit may be obtained by cognitive behavioural therapy in the context of rehabilitation, although this does not appear to work on its own. Some patients with emphysema naturally learn to change the pattern of breathing by pursing their lips to delay airway closure. For this reason, breathing retraining exercises have been attempted in COPD to try to correct inefficient or uncomfortable breathing patterns.[19] Although there is some logic in this approach that may produce short term benefits, the effects do not seem to be sustained. One other approach in COPD is the specific pharmacological modification of dyspnoea. Several agents including opioids and antidepressants have been tried with some success.

In asthma the main aim of pharmacological treatment is the reduction of inflammation and relief of bronchospasm. Specific isolated treatment of dyspnoea is seldom conducted on the grounds that amelioration of the underlying condition will suffice. However, there are two circumstances where the treatment of dyspnoea in asthma might be considered potentially helpful: (1) when loss of physical fitness resulting from chronic dyspnoea might be a contributing factor and (2) when the dyspnoea or its perception has another cause. Individuals with chronic asthma enter the same cycle of breathlessness and skeletal muscle deconditioning as those with COPD. When this becomes a significant component of their disability, it may be helped by physical rehabilitation in exactly the same way. The benefits in physical performance and health status for patients with disability from chronic asthma appear to be similar to those achieved in COPD.[17] Indeed, even in younger patients with asthma who would not regard themselves as disabled, there is a reduction in exercise capacity which is more closely related to lack of fitness than a ventilatory limit.[20]

Learning points

- Dysfunctional breathing (also known as hyperventilation syndrome and disproportionate breathlessness) is a syndrome of excessive breathlessness and other variable somatic symptoms that is sometimes—but not always—associated with demonstrable hypocapnia
- Dysfunctional breathing may make an important contribution to symptoms in asthma and result in overprescription of drug treatment
- There is no accepted gold standard of diagnosis of dysfunctional breathing beyond the clinical description, but the Nijmegen questionnaire is a symptom checklist that can be used to discriminate dysfunctional breathers from normal individuals
- No technique has yet been validated to identify dysfunctional breathing in the presence of other respiratory disease such as asthma
- Estimates of the frequency of dysfunctional breathing in subjects with asthma must therefore remain speculative until there is an agreed definition of the condition and validation of the investigative instrument
- To date the evidence of benefit for the treatment of dysfunctional breathing in the context of asthma remains scanty

The hypothesis of Thomas *et al*[8] is that some patients with asthma may exhibit hyperventilation or dysfunctional breathing which may compound or be confused with the original condition. It is possible that these symptoms may be helped by conventional rehabilitation and there is some evidence that this is effective even in mild cases of asthma.[21] The opportunity for specific treatment therefore lies in some form of physiotherapy or cognitive behavioural therapy that is aimed at breathing retraining or relaxation techniques. In spite of the hopeful tone of the paper, there is very little evidence for benefit of any technique in the specific management of hyperventilation or dysfunctional breathing in the context of asthma. The most well known breathing retraining technique is the Buteyko programme. The specific details of this technique are unfortunately subject to commercial sensitivities but involve some form of control of hyperventilation and mouth breathing. So far one published randomised controlled trial would suggest that, in some cases at least, there is a reduction in beta agonist use and hyperventilation.[22] However, no effect was noted on conventional measures of airway calibre or inhaled steroid use. It is highly likely that this subject will be further explored in future publications.

The use of traditional physiotherapy retraining and disease education has been explored by Thomas *et al* but so far only published as an abstract.[23] In this situation enhanced physiotherapy appears to have improved a disease specific health status measure (Asthma Quality of Life questionnaire) as well as the Nijmegen questionnaire for a period of up to 6 months. The full results of this study will be awaited with some interest.

Conclusions

It would be an attractive hypothesis to suggest that some patients with asthma had an alternative or additional explanation for their symptoms. Firstly, it offers an opportunity for treatment beyond pharmacology and, secondly, an opportunity to reduce unnecessary medication. Every clinician recognises the occasional patient with obvious hyperventilation, often in association with asthma, where alternative treatment might be of value. The scale of the problem has never been previously described but to suggest that one third of women with asthma may have a component of dysfunctional breathing is quite significant. If this is true, it will have a considerable impact on the provision of services and move treatment away from reliance on pharmacotherapy alone. However, before there is a wholesale change in practice, these results need to be verified. This is not just a matter of repeating the study or examining a wider population. In this case the definition of dysfunctional breathing and the methods for identifying it require careful validation. The application of the Nijmegen questionnaire in the context of asthma or other lung disease needs further exploration. Even if the results of this study are reproduced, there is still an outstanding difficulty. One of the guiding principles of any screening programme is that effective treatment should be available to those uncovered by the process. In this case it is far from clear whether there is yet good evidence for effective treatment of dysfunctional breathing. The authors have been brave enough to put their toes in the water but further immersion will need careful consideration.

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