factor, and most of the deaths caused by smoking are in fact due to respiratory diseases.[6] Smoking cessation in patients with COPD reduces the accelerated rate of decline of forced expiratory volume in one second (FEV_1) and is one of only two interventions which improve the long term prognosis, the other being long term oxygen therapy.[13 14] Active support and treatment of nicotine addiction in these patients should therefore be considered to be an essential component for medical management of this disease.

If nicotine addiction is accepted to be a fundamental issue in preventing smokers from successfully stopping smoking, it is clear that simple advice alone is not always adequate to address the problem. Active help and support such as psychological counselling and pharmacological treatments need to be made available to increase the chances of success. Other addictions such as those to heroin and alcohol are already treated and funded by the NHS, and although funding is now being made available to establish smoking cessation services in selected areas of Britain, it is time to make the treatment of nicotine addiction available and affordable for all smokers through the NHS. Smoking cessation interventions give good value for money and the cost implications of providing smoking cessation services in the UK are well established.[15] The report by the Royal College of Physicians adds further urgency to the need for the implementation of smoking cessation services.

It is also important for all health professionals to take an active role in advising and helping smokers to stop.[16] For this to be achieved, there must be better education and training for health professionals in both the knowledge of the adverse effects of active and passive smoking and in smoking cessation methods. Unfortunately, marked deficiencies in knowledge of tobacco control and prevention have been shown amongst medical students from all over the world.[17] Few medical schools include education on tobacco issues in their undergraduate curriculum and the prevalence of smoking amongst medical students increased during their medical school careers. Even for specialist registrars training in respiratory medicine, the current curriculum does not include a recommendation for training in smoking cessation as an essential part of the syllabus.[18]

What messages can respiratory physicians take from the latest Royal College of Physicians report? Many of our patients need help and support to stop smoking and, because so many of our patients have smoking related diseases, it would be appropriate for respiratory specialists to take ownership of the problem and become lead physicians for tobacco control within their NHS trusts or districts. This would involve a more active role in the education and training of medical students, junior doctors, general practitioners, and other health care professionals in smoking issues especially, and for respiratory physicians to become expert in smoking cessation interventions. Respiratory physicians should actively campaign for and request the appointment of smoking cessation counsellors in all NHS hospitals to provide advice and psychological support for patients and staff. They could work closely with such individuals who could have their base within respiratory medicine departments. The pharmacological aspects of smoking cessation could also become part of the expertise of the respiratory specialist.

ANGELA HILTON

Consultant Respiratory Physician
& Chairman of the BTS Tobacco Committee,
North West Lung Centre,
Wythenshawe Hospital,
Manchester M23 9LT,
UK

1 Royal College of Physicians. *Smoking and health. A report on smoking in relation to lung cancer and other diseases*. London: Pitman Medical, 1962.
2 Royal College of Physicians. *Smoking and health now. A new report on smoking and its effects on health*. London: Pitman Medical, 1971.
3 Royal College of Physicians. *Smoking or health*. London: Pitman Medical, 1977.
4 Royal College of Physicians. *Health or smoking. A follow-up report*. London: Pitman Medical, 1983.
5 Royal College of Physicians. *Smoking and the young*. London: Pitman Medical, 1992.
6 Royal College of Physicians. *Nicotine addiction in Britain*. London: Pitman Medical, 2000.
7 Fiore MC. Trends in cigarette smoking in the United States: the epidemiology of tobacco use. *Med Clin North Am* 1992; **76**: 289–303.
8 Silagy C, Mant D, Fowler G, *et al.* The effect of nicotine replacement therapy on smoking cessation. Cochrane Library (Issue 1), 1997.
9 British Thoracic Society. Comparison of four methods of smoking withdrawal in patients with smoking related diseases. *BMJ* 1983; **286**: 595–7.
10 British Thoracic Society. Smoking withdrawal in hospital patients: factors associated with outcome. *Thorax* 1984; **39**: 651–6.
11 British Thoracic Society. Smoking cessation in patients: two further studies. *Thorax* 1990; **45**: 835–40.
12 Prathiba BV, Tjeder S, Phillips C, *et al*. A smoking cessation counsellor: should every hospital have one? *J R Soc Health* 1998; **118**: 356–9.
13 Fletcher CM, Peto R. The natural history of chronic airflow obstruction. *BMJ* 1977; 1: 1645–8.
14 US Department of Health and Human Services. *The health benefits of smoking cessation. A report of the Surgeon General*. Washington: US Government Printing Office, 1989.
15 Parrot S, Godfrey G, Raw M, *et al.* Guidance for commissioners on the cost effectiveness of smoking cessation interventions. *Thorax* 1998;**53**(Suppl 5, Part 2):S1–38.
16 Raw M, McNeill A, West R. Smoking cessation guidelines for health professionals. *Thorax* 1998;**53** (Suppl 5, Part 1):S1–19.
17 Richmond R. Teaching medical students about tobacco. *Thorax* 1999;**54**: 70–8.
18 Joint Committee for Higher Medical Training (JCHMT). *Curriculum for higher specialist training in respiratory medicine*. Royal College of Physicians, July 1998.

Thorax 2000;**55**:257–259

Orthostatic increase of respiratory gas exchange in hyperventilation syndrome

William Gardner

The paper by Malmberg *et al* in the current issue of *Thorax*[1] deals with the difficult subject of the hyperventilation syndrome and finds that these patients have a disproportionately high ventilatory response to change of body position from supine to standing. The authors suggest that this can be used as a diagnostic criterion for hyperventilation syndrome. Hyperventilation is a confused and poorly documented subject and the publication of this paper provides an opportunity to review some of the particularly controversial aspects of this subject.

The first issue concerns the basis for the labelling of these patients as "hyperventilation syndrome". Some of the controversies about the use of this term have recently been reviewed by Folgering[2] and by Gardner.[3] The physiological definition of hyperventilation is alveolar ventilation that is inappropriately high for the metabolic production of carbon dioxide, leading to reduction of arterial P_{CO_2} (Pa_{CO_2}) below the normal range (hypocapnia) and respiratory alkalosis. The combination can lead both to vasoconstriction in selected vascular beds and to neuronal

hyperexcitability producing symptoms involving most systems of the body. Many psychosomatic syndromes have been described in the past in which hyperventilation has a variable and uncertain role but the term "hyperventilation syndrome" was first used in 1938 to describe patients with the somatic symptoms of both hypocapnia and anxiety.[4] This theme was extended by subsequent authors[5–7] and the definition arrived at by Lewis and Howell in 1986 on the basis of a questionnaire of delegates at a psychophysiology meeting[8] was "a syndrome induced by physiologically inappropriate hyperventilation and usually reproduced in whole or in part by voluntary hyperventilation". However, the term "hyperventilation syndrome" is now used in so many different contexts that it could be argued that it has ceased to have any universal meaning. Some physicians diagnose it in the presence of the somatic symptoms of hypocapnia either at rest or induced by voluntary overbreathing without assumptions about aetiology,[9] or regard it primarily as an abnormality of respiratory control[10] or as a variant of disproportionate breathlessness.[11 12] Folgering accepts that anxiety may be absent and has recently suggested a new definition as "a dysregulation of ventilation causing hypocapnia in the absence of organic causes for hyperventilation, with symptoms and complaints not exclusively associated with hypocapnia". Many refuse to recognise it as a separate entity[13] or regard it as secondary to organic disease and especially to asthma.[14 15] Many would not use the term in the presence of any organic cause of hyperventilation, yet organic and psychiatric factors are usually difficult to separate. Lum[16] regards hyperventilation as a form of conditioned response and avoids use of the term "hyperventilation syndrome". Gardner[3] believes that it is not useful in the clinical context to label a patient with hypocapnia as "hyperventilation syndrome" and that the term should be abandoned. He believes that hyperventilation is often due to a complex interaction between a range of organic, psychogenic, and physiological factors and that, in all cases, the initiating and sustaining cause or causes of the increased respiratory drive causing the hyperventilation should be sought and documented. Use of a label such as "hyperventilation syndrome" tends to preclude further search for underlying aetiological factors and can be dangerous in the context of the emergency room. It is difficult to assess which of the current definitions applies to the patients in the study by Malmberg *et al*. There was no clear evidence of anxiety or organic causes of hyperventilation and the end tidal P_{CO_2} was recorded as being no different from the control value at rest. However, the history suggested hypocapnia at other times although the presence of a low P_{CO_2} at that time was not documented. About the only certain statement that can be made is that these patients do not have chronic hyperventilation.[17]

Hyperventilation occurs in many different specialties including neurology, cardiology, chest medicine, and psychiatry. The patient population to which the term is applied will vary greatly between specialties. Without a clear understanding of the patient population being studied it is difficult to understand the significance of any findings concerning this subject, and the source of the patients and the clinical context from which they were recruited into the study requires particular emphasis in any study about hyperventilation. There is considerable ignorance among physicians about psychiatry and the precise criteria that are used for psychiatric diagnoses. Many physicians in medical specialties assume that the demonstration of a low arterial P_{CO_2} automatically diagnoses hyperventilation syndrome with an assumption that the patient has some unspecified psychiatric condition. Such patients are then considered to be no longer of interest to the physician. This is often unfair to the patient, and ignores the wide range of organic, behavioural and physiological causes of hyperventilation, many of which can and usually do coexist. These uncertainties reflect the complexity of this subject which falls between psychiatry, clinical medicine, and physiology.

It is difficult to determine the significance of the suggestion in the paper by Malmberg *et al* that their findings should be used as the basis for a diagnostic test for hyperventilation syndrome. At a physiological level this finding requires a more detailed physiological study to evaluate mechanisms before its significance can be ascertained. At a clinical level ventilation is difficult to measure and most routine lung function laboratories do not have tilt tables or the facilities for measurement of respiratory control variables, especially while the patient is moving from one position to another. The methodology for diagnosing hyperventilation is even more controversial than the issues surrounding the definition, and it could be argued that diagnostic criteria cannot adequately be defined unless the issue of definition has been clarified. Strict adherence to the physiological definition would require documentation of hypocapnia, but hyperventilation may be sporadic and there are few current techniques for measuring P_{CO_2} over prolonged periods of time outside the laboratory.[18 19] As in the present study, hyperventilation is often diagnosed with, not only no evidence of a low arterial, end tidal or transcutaneous P_{CO_2}, but even with evidence of a normal P_{CO_2} on spot sampling. While it is possible to provide convincing evidence of hyperventilation on behavioural grounds, using the term hyperventilation in the presence of a normal P_{CO_2} puts an onus on the authors to be more meticulous than usual in documenting the criteria by which hyperventilation was diagnosed. Many such descriptions are sadly unconvincing.

Hypocapnia induces a range of symptoms, and symptom checklists such as the Nijmegen questionnaire[20] have often been used for diagnosing the hyperventilation syndrome. However, many would argue that most of these symptoms are non-specific and do not provide an adequate basis for diagnosis when used alone. It could be argued that the only symptoms specific to hypocapnia are paraesthesiae and tetany, possibly combined with symptoms due to cerebral vasoconstriction and hypoxia. In clinical practice the clinical finding of a low arterial or end tidal P_{CO_2} is of little relevance in itself if there are no associated symptoms of hypocapnia, or if the symptoms are of minor importance compared with the symptoms of the disorder causing the hyperventilation. In other situations the symptoms of hyperventilation are pivotal to the clinical presentation of the patients. The reporting of familiar symptoms during voluntary hyperventilation is often used as a basis for diagnosis, but this has been criticised by Hornsveld *et al*[21] in that similar symptoms are also reported when the Pa_{CO_2} is artificially maintained at normal levels during voluntary overbreathing.

The role of anxiety disorders in the paper by Malmberg and colleagues is unclear. No psychiatric disorders were reported but their patients nevertheless had symptoms of "episodic dyspnoea or air hunger" and "palpitations, sweating, trembling, dryness of the mouth or other symptoms of overactivity of the autonomic nervous system . . . suggestive of panic disorder". This definition of panic is imprecise and no formal psychiatric assessment was apparently performed. Contrary to the statement in the introduction to the paper, the association between anxiety and panic is still controversial.[22 23] Although anxiety was a core component in the original description of hyperventilation syndrome, the relation of hyperventilation to anxiety is not simple and hyperventilation can occur without

anxiety[17 24 25] or anxiety may be induced by hyperventilation.[26] Anxiety can be associated with both mild hyperventilation and abnormalities of breathing pattern.[27] Endogenous non-retarded depression can be associated with hyperventilation[28] and phobic patients have a high prevalence of breathing difficulties.[29 30] The predisposition to overbreathe in response to stress may be dependent on biological vulnerability, personality, and cognitive variables[22] as well as individual interpretation of the hyperventilation induced somatic symptoms,[31] and may become a conditioned response.[22] Because of the complexities of this subject, it could be argued that any paper about hyperventilation requires a collaborative input from a psychiatrist or psychologist. At a clinical level such an input is often required for no reason other than to counter the assumptions of the referring clinician that a patient with hyperventilation must automatically have an anxiety state. Often there is a complex interaction between organic and psychiatric factors such as depression and phobic states which require a combined input from a physician and a psychiatrist.

The authors describe their patients as having a history of recurrent or episodic dyspnoea. Howell[11 12] regards disproportionate dyspnoea as being synonymous with hyperventilation syndrome, and he has described the characteristics of these patients. Gardner[3] has argued that the two are not synonymous and that, if a patient is breathless for reasons that are not clear, it is the breathlessness that is the primary condition for which a cause should be sought and that, if hyperventilation is also present, it is usually secondary to the breathlessness and of little clinical importance. Patients are often referred with a label of psychogenic dyspnoea when the degree of distress seems disproportionate to the clinical findings and lung function or blood gas data, but there is almost no literature on this subject.[32 33] Dyspnoea is what the patient reports and it is therefore difficult to dispute. Studies of breathlessness are impeded by uncertainty about the basic mechanisms[34] and there are probably many different forms. Air hunger, or a sensation of inability to take a satisfying breath or to fill the lungs, was reported by patients in the Malmberg study and may indicate a psychogenic component to breathlessness. It was a universal feature of the patients with chronic hyperventilation studied by Gardner and Bass,[17 25] and more recently Gardner has reported patients in whom the primary presentation is air hunger which leads to variable degrees of panic and hyperventilation.[3]

Asthma is a common underlying basis for hyperventilation[15 35 36] and Gardner has argued that many patients with a primary presentation of hyperventilation have very mild and often previously undiagnosed asthma.[37] Malmberg *et al* describe the exclusion of "physician diagnosed" asthma but give no details about how this was achieved, although patients had "histamine provocation tests and pulmonary diffusion capacity if clinically indicated". Asthma is a surprisingly difficult and controversial diagnosis and many physicians would argue that these indications should be clarified.

In summary, it would seem that the patients in the study by Malmberg and colleagues might fit more accurately into a classification of dyspnoea and air hunger with secondary intermittent hyperventilation rather than receiving an automatic label of "hyperventilation syndrome". Such a reclassification may lead to a different interpretation of these clearly interesting results and may suggest lines of enquiry for possible mechanisms of air hunger. The physiological basis for these responses requires investigation and may provide useful insights into mechanisms by which postural changes can influence control of breathing and respiratory sensations. It is probably unhelpful to suggest this as the basis for yet another indirect diagnostic test for hyperventilation syndrome.

WILLIAM GARDNER

Department of Respiratory Medicine & Allergy,
Guy's, King's and St Thomas' School of Medicine,
London SE5 9PJ,
UK

1 Malmberg LP, Tamminen K, Sovijärvi ARA. Orthostatic increase of respiratory gas exchange in hyperventilation syndrome. *Thorax* 2000;**55**:295–301.
2 Folgering H. The hyperventilation syndrome. In: Altose MD, Kawakami Y, eds. *Control of breathing in health and disease*. New York, Basel: Marcel Dekker, 1999: 633–60.
3 Gardner WN. Review: The pathophysiology of hyperventilation disorders. *Chest* 1996;**109**:516–34.
4 Kerr WJ, Gliebe PA, Dalton JW. Physical phenomomena associated with anxiety states: the hyperventilation syndrome. *Calif Western Med* 1938;**48**: 12–6.
5 Lewis BI. Chronic hyperventilation syndrome. *JAMA* 1954;**155**:1204–8.
6 Ames F. The hyperventilation syndrome. *J Mental Sci* 1955;**101**:466–525.
7 Lewis BI. Hyperventilation syndrome: a clinical and physiological evaluation. *Cal Med* 1959;**91**:121–6.
8 Lewis RA, Howell JBL. Definition of the hyperventilation syndrome. *Bull Eur Physiopathol Respir* 1986;**22**:201–5.
9 Stoop A, de Boo T, Lemmens W, *et al*. Hyperventilation syndrome: measurement of objective symptoms and subjective complaints. *Respiration* 1986;**49**:37–44.
10 Folgering H, Colla P. Some anomalies in the control of $Paco_2$ in patients with hyperventilation syndrome. *Bull Eur Physiopathol Respir* 1978;**14**:503–12.
11 Howell JBL. Behavioural breathlessness. *Thorax* 1990;**45**:287–92.
12 Howell JB. The hyperventilation syndrome: a syndrome under threat? *Thorax* 1997;**52**(Suppl 3):S30–4.
13 Hornsveld HK, Garssen B, Fiedeldij Dop MJC, *et al*. Double-blind placebo-controlled study on the hyperventilation provocation test and the validity of the hyperventilation syndrome. *Lancet* 1996;**348**:154–8.
14 Dent R, Yates D, Higenbottam T. Does the hyperventilation syndrome exist. *Thorax* 1983;**38**:223.
15 Demeter SL, Cordasco EM. Hyperventilation syndrome and asthma. *Am J Med* 1986;**81**:989–94.
16 Lum LC. The syndrome of habitual chronic hyperventilation. *Recent Advan Psychosom Med* 1976;**3**:196–229.
17 Gardner WN, Meah MS, Bass C. Controlled study of respiratory responses during prolonged measurement in patients with chronic hyperventilation. *Lancet* 1986;ii:826–30.
18 Hibbert G, Pilsbury D. Hyperventilation in panic attacks. Ambulant monitoring of transcutaneous carbon dioxide. *Br J Psychiatry* 1988;**153**:76–80.
19 Osborne CA, Gardner WN, Varley JS. The range of end-tidal P_{CO_2} measured noninvasively using an ambulatory capnograph in normal subjects. *Eur Respir J* 1997;**10**(Suppl 25):47s.
20 van Dixhoorn J, Duivenvoorden HJ. Efficacy of Nijmegen questionnaire in recognition of the hyperventilation syndrome. *J Psychosom Res* 1985;**29**: 199–206.
21 Hornsveld HK, Garssen B, Dop MJ, *et al*. Double-blind placebo-controlled study of the hyperventilation provocation test and the validity of the hyperventilation syndrome. *Lancet* 1996;**348**:154–8.
22 Bass C, Kartsounis L, Lelliott P. Hyperventilation and its relationship to anxiety and panic. *Integrative Psychiatry* 1987;**5**:274–91.
23 Garssen B, Buikhuisen M, van Dyck R. Hyperventilation and panic attacks. *Am J Psychiatry* 1996;**153**:513–8.
24 Lewis BI. The hyperventilation syndrome. *Ann Intern Med* 1953;**38**:918–27.
25 Bass C, Gardner WN. Respiratory and psychiatric abnormalities in chronic symptomatic hyperventilation. *BMJ* 1985;**290**:1387–90.
26 Lum LC. Hyperventilation syndromes in medicine and psychiatry: a review. *J R Soc Med* 1987;**80**:229–31.
27 Tobin MJ, Chadha TS, Jenouri G, *et al*. Breathing patterns 2. Diseased subjects. *Chest* 1983;**84**:286–94.
28 Damas Mora J, Grant L, Kenyon P, *et al*. Respiratory ventilation and carbon dioxide levels in syndromes of depression. *Br J Psychiatry* 1976;**129**:457–64.
29 Arrindell WA. Dimensional structure and psychopathology correlates of the fear survey schedule (FSS-III) in a phobic population: a factorial definition of agoraphobia. *Behav Res Ther* 1980;**18**:229–42.
30 Hallam RS, Hafner RJ. Fears of phobic patients: factor analysis of self-report data. *Behav Res Ther* 1978;**16**:1–6.
31 Clark DM, Hemsley DR. The effects of hyperventilation: individual variability and its relation to personality. *J Behav Ther Exp Psychiatry* 1982; **13**:41–7.
32 Mahler DA, Harver A, Lentine T, *et al*. Descriptors of breathlessness in cardiorespiratory diseases. *Am J Respir Crit Care Med* 1996;**154**:1357–63.
33 Meek PM, Schwartzenstein RM. American Thoracic Society. Dyspnoea. Mechanisms, assessment and management: a consensus statement. *Am J Respir Crit Care Med* 1999;**159**:321–40.
34 Tobin MJ. Dyspnea. Pathophysiologic basis, clinical presentation, and management. *Arch Intern Med* 1990;**150**:1604–13.
35 McFadden ER, Lyons HA. Arterial blood gas tension in asthma. *N Engl J Med* 1968;**278**:1027–32.
36 Gardner WN, Bass C, Moxham J. Recurrent hyperventilation tetany due to mild asthma. *Respir Med* 1992;**86**:349–51.
37 Saisch SGN, Wessely S, Gardner WN. Patients with acute hyperventilation presenting to an inner-city emergency department. *Chest* 1996;**110**:952–7.