Thorax 1992;47:66–67

LETTERS TO THE EDITOR

Body composition and exercise performance in patients with chronic obstructive pulmonary disease (COPD)

The original article of Dr A M W J Schols and others in the October issue of *Thorax*[1] shows that exercise performance, as indicated by the distance walked in 12 minutes, is related to the fat free mass of the patient. Fat free mass was taken to be an indicator of muscle mass, but the investigators found that arm muscle size was not well related to other measures of muscle mass.

Several possible reasons for the relationship, two of which were related to respiratory muscle performance, were considered. There are, however, two other obvious possibilities.

Firstly, muscle loss may well be from the lower limbs. If this were so, then exercise capacity could be reduced because of the smaller capacity for this type of exercise, irrespective of respiratory muscle function.

Secondly, the patients who performed badly might take little regular exercise, or indeed may be limited by their symptoms in their exercise. Such patients would display lower limb muscle wasting. Consequently, performance may be related to muscle mass (particularly in the legs) by loss of activity. In the clinical evaluation of patients who are housebound with severe lung or cardiac disease wasting of the quadriceps femoris and calf muscles is often a striking feature.

Dr Schols and his colleagues suggest that a compromised nutritional state may contribute to impaired performance, and that muscle mass determines exercise performance. Unwary readers may be led to conclude that patients do badly because of malnutrition, or even that improved nutrition might be beneficial. This is not necessarily so. Correlation does not indicate cause and effect: even if there is such a relationship, which is cause and which is effect may not be self evident.

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1 Schols AMWJ, Mostert R, Soeters PB, Wouters EFM. Body composition and exercise performance in patients with chronic obstructive pulmonary disease. *Thorax* 1991;46:695–9.

AUTHOR'S REPLY

Nutritional depletion commonly occurs in patients with COPD. It is well established that body mass depletion exerts detrimental effects on both respiratory and skeletal muscle function. In this article we also found a strong negative effect of body mass depletion, measured by body weight and fat free mass, on exercise performance in a group of patients with COPD, including a substantial proportion of underweight patients.

Dr Drummond suggests in his letter that independently of body mass depletion a selective loss of mass and strength of the lower limb muscles, irrespective of respiratory muscle function and due to inactivity, may be an important reason for exercise impairment in these patients.

We recognise that inactivity may adversely influence muscle mass and function in COPD. Our findings, however, do not confirm that muscle wasting in the patients was confined to the lower limb muscles.

1) Arm muscle circumference and fat free mass were significantly interrelated ($r = 0{\cdot}51$, $p < 0{\cdot}001$).

2) The mean value of arm muscle circumference in the whole group was *below* normal (90% of the reference value).

3) Mean maximal inspiratory mouth pressure (5·8 kPa) *and* expiratory mouth pressure (8·0 kPa, not shown) were below normal. Although inspiratory mouth pressure in COPD may partly be influenced by a mechanical disadvantage, expiratory mouth pressure reflects only respiratory muscle weakness.

The strong positive association between fat free mass and walking distance in the subgroup of underweight patients indicates that only when fat free mass drops to very low values is it critical for exercise performance. The results further suggest that in these patients nutritional intervention (in combination with reactivation) may enhance physical performance.

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Effect of positive expiratory pressure breathing in patients with cystic fibrosis

We read the study of positive expiratory pressure (PEP) breathing in patients with cystic fibrosis by Dr C P van der Schans and his colleagues in the April issue of *Thorax*[1] with interest. In their penultimate paragraph the authors speculate on high pressure PEP mask physiotherapy, a technique we have developed and investigated.[2,3] Although we are inclined to agree with some of his speculations, we are puzzled by his unsubstantiated statement that high pressure PEP might cause complications. We have considerable clinical experience of this method and believe that this speculation is wrong; unfortunately, such statements carry the risk of discouraging other centres to adopt an effective and well studied technique.

Since we developed this technique in 1982 our accumulated clinical experience adds up to 3866 patients treatment months, mostly in patients with cystic fibrosis. In these nine years there has been one spontaneous pneumothorax in an 11 year old girl, four hours after her morning PEP session. After treatment by tube drainage and pleural sclerosis the child recommenced her high pressure PEP and since then has cleared her lungs exclusively with this technique. This was the only case of spontaneous pneumothorax in our 104 patients with cystic fibrosis, which argue strongly against an increased risk of pneumothorax with high pressure PEP.

Airway distension, due to the back pressure of forcefully exhaling against a resistive load, might impose some stress on airway walls. Although this would theoretically increase the risk of bronchial artery bleeding, we have had only one serious bleeding episode that required bronchial artery embolisation.

Another effect of high pressure on airway walls is evident clinically and has recently been documented in a comparative study of different chest physiotherapy techniques[4]: it occasionally induces bronchospasm in patients with airway hyperreactivity. Nevertheless, such patients frequently prefer to use high pressure PEP because of the technique's superior speed and efficacy; in such cases we prescribe a bronchodilator.

Some of the negative clinical experience with positive pressure ventilators in patients with obstructive airway disease is occasionally and uncritically transferred to high pressure PEP. There are, however, important differences between the two techniques: whereas externally developed positive pressure introduces distending forces across the airways, expirtory muscle contraction against a resistive load increases pressure in the system homogeneously. The alveolar-pleural pressure gradient is thus determined exclusively by the static elastic recoil pressure of the lung, irrespective of transpulmonary pressure. Another consequence of homogeneous increase in intrathoracic pressure is the lack of compression of the alveolar capillary bed, plus an unaltered transmural pressure on the pulmonary arteries.

In summary, there is substantial clinical experience that testifies to the safety of the technique. Speculation on potential complications may be based on ill understood concepts and beliefs rather than on hard data, as collected by controlled investigations and continued analysis of bedside clinical work.

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1 Van der Schans CP, van der Mark ThW, de Vries G, Piers DA, Beckhuis H, Dankert-Roelse JE, *et al.* Effect of positive expiratory pressure breathing in patients with cystic fibrosis. *Thorax* 1991;46:252–6.
2 Oberwaldner B, Evans JC, Zach MS. Forced expirations against a variable resistance: a new chest physiotherapy method in cystic fibrosis. *Pediatr Pulmonol* 1986;2:358–67.
3 Oberwaldner B, Theißl B, Rucker A, Zach MS. Chest physiotherapy in hospitalized patients with cystic fibrosis: a study of lung function effects and sputum clearance. *Eur Respir J* 1991;4:152–8.
4 Theißl B, Pfleger A, Oberwaldner B, Zach M. Chest physiotherapy (PT) in cystic fibrosis (CF)—a comparative study of high-pressure PEP and autogenic drainage. *Pediatr Pulmonol* 1990 (suppl 5):259.

AUTHOR'S REPLY

We would like to thank Professor Zach and Dr Oberwaldner for their response to our article. Our warning that high positive airway pressures may cause complications is not based on an "ill understood concept or beliefs," as they suggest; it is supported by their own statement that their method is "potentially harmful" (their ref 2). That high expiratory pressure does not cause any complications is based on their observation on the incidence of complications, such as pneumothorax and bronchial arterial bleeding, in a group of 64 patients. Moreover, the haemodynamic effects of increased airway pressures,[1] which may in some patients have negative effects, are ignored. We also think that a group of 64 patients, most with cystic fibrosis, is too small for a claim that the technique is always safe in all patients.

The supposition that high positive expiratory pressure treatment is beneficial is interesting, but the introduction of new physiotherapeutic methods should be accompanied by a critical consideration of their effectiveness and possible negative side effects, and the type of patient to whom this technique can be safely applied. Comparison between results for different groups of

patients, or with control measurements, is in these circumstances in our opinion essential.

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1 Cabrera MR, Nakamura GE, Montague DA, Cole RP. Effect of airway pressure on pericardial pressure. *Am Rev Respir Dis* 1989;140:659–67.

Pneumocystis carinii pneumonia complicating low dose methotrexate treatment for rheumatoid arthritis

We read with interest the paper by Dr A Wollner and his colleagues (March 1991; 46:205–7) as we have recently reported two cases of pneumocystis pneumonia in immunocompromised patients with rheumatoid disease.[1] A 42 year old woman with severe seropositive rheumatoid arthritis was started on oral methotrexate treatment 7·5 mg per week and developed pneumocystis pneumonia after 16 weeks of treatment. Despite a stormy course, requiring ventilation for almost three weeks, she made a full recovery. It is of interest that at the onset of pneumonia her total white cell count was $14{\cdot}6 \times 10^9/l$ with relative lymphopenia (total) lymphocytes $290 \times 10^6/l$, 2% of total). Our second patient was a 55 year old man treated with cyclophosphamide 2·5 mg/kg plus prednisolone 40 mg for microscopic polyarteritis nodosa. After eight months of treatment he developed pneumocystis pneumonia. Again, despite a normal total white cell count of $4{\cdot}9 \times 10^9/l$, he had profound lymphopenia (1%, $43 \times 10^6/l$). This patient also required assisted ventilation but responded well to treatment, with complete resolution of symptoms.

With the increasing use of immunosuppression for patients with rheumatoid arthritis and similar conditions, the guidelines on falling total white cell counts would seem to be ineffective. The lymphopenia found in our two patients and in all three of the cases recorded by Dr Wollner and colleagues would suggest that this makes a substantial contribution to the immunodeficiency resulting in opportunistic infections. We advise that the absolute lymphocyte count should also be monitored in patients treated with cytotoxic drugs and that the dose should be adjusted promptly if profound lymphopenia develops.

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1 Porter D, Marshall DAS, Madhok R, Capell HA, Sturrock RD. *Pneumocystis carinii* infection complicating cytotoxic therapy in two patients with lymphopenia, but a normal total white cell count. *Br J Rheumatol* (in press).

BOOK NOTICES

Respiratory Medicine in Clinical Practice. Peter Howard. (Pp 281; £29·50.) London: Hodder and Stoughton, 1991. ISBN 0-340-54560-7.

The author's preface states that this book is not intended to compete with comprehensive textbooks but is a survey of salient topics and controversies, aimed primarily at MRCP candidates. In this object it only partially succeeds. The book is organised in a standard way. Basic anatomy and physiology are covered adequately in the first five chapters; chapter 6 discusses basic investigations, and the middle chapters deal with clinical topics. The coverage is adequate but from the MRCP candidate's viewpoint there are important omissions. Recent developments are popular topics in membership examinations and coverage of these is patchy. For example, discussion of atypical mycobacterial infections is brief, and no mention is made of the recent improvement in the understanding of the genetics of cystic fibrosis or of bisphosphonates in the management of malignant hypercalcaemia. The use of antineutrophil cytoplasmic antibody in the diagnosis of Wegener's granulomatosis is not mentioned. Fine detail on radiographs is difficult to see owing to reproduction on print quality paper. The radiographs in figures 11.6 and 15.5 look very similar although the attached diagnoses are different. The book concludes with case histories, which are rather brief and would be improved if more information were given and questions were more akin to the "grey" cases posed in the membership examination. The MCQ questions are a useful aid to revision. Thus, although most standard topics are covered well, there are important omissions in the coverage of recent changes in diagnosis and management of topical respiratory conditions.—JAR

Immunologically Mediated Pulmonary Diseases. Edited by Joseph P Lynch III and Richard A DeRemee. (Pp 547; $95.) Philadelphia: Lippincott, 1991. ISBN 0-397-51051-9.

This multi-author volume deals with a wide range of lung diseases in which immunological processes are thought to play a part in pathogenesis. The authors are exclusively American or Canadian and most are acknowledged experts in their spheres. The topics covered range from asthma, including occupational asthma, occupational lung disease, various disorders in which lung vasculitis is a component (four chapters), eosinophilic pneumonia, granulomatous lung diseases, and diseases characterised by the presence of fibrosing alveolitis. Rarer lung problems, such as pulmonary alveolar proteinosis, pulmonary haemorrhage syndromes, and eosinophilic granuloma, receive comprehensive attention and I welcome the detailed chapter on the complex disorders in which low grade lymphoproliferative processes operate, such as lymphomatoid granulomatosis. The editors' aim has been to integrate advances in the basic sciences with a clinical approach to diagnosis and management and each chapter addresses each of those issues separately. The text is directed mainly at practising clinicians and each chapter has been written, successfully in my view, with this in mind. No major area has been ignored. Each chapter is well referenced numerically, though there are few references more recent than 1989. The quality of the illustrations is somewhat variable. Particularly informative and well written chapters include those on pulmonary complications in collagen vascular disease, lymphomatoid granulomatosis and lymphoproliferative disorders of the lung, pulmonary haemorrhage syndromes, and idiopathic pulmonary fibrosis. This book has achieved its aim of combining basic science with clinical practice in a very readable form. Anyone concerned with the management of pulmonary disease, either as pulmonary physicians or as rheumatologists, clinical immunologists, or other clinicians seeing patients with pulmonary problems, should find much of interest here; and at $95 the book is extremely good value for money. Medical students and staff in training would find this an invaluable source of reference—RMduB

Asthma—its Pathology and Treatment. MA Kaliner, PJ Barnes, CGA Persson. (Pp 808; $165, USA and Canada; $189.75, all others.) New York: Dekker, 1991. ISBN 0-8247-8217-8.

This book comprises several reviews with discussion sections derived from a three day symposium and attempts to cover comprehensively the pathology and treatment of asthma. In the main the book is well written and has drawn on the expertise of several acknowledged experts. Of particular note are the sections on asthma epidemiology and airway epithelium. Occasional chapters suffer from a disproportionate emphasis on the authors' own work. There are one or two subjects that are not covered particularly well, including cytokines, occupational asthma, and certain of the new asthma treatments, such as the potassium channel activators, in addition to some of the more practical aspects of asthma management. It would also have been useful to have an overview chapter, either at the beginning of the sections on asthma treatment or at the end. All things considered, however, this book represents a comprehensive review of asthma, which would be a useful addition to the bookshelf of the asthma specialist. It would make a useful reference book for general respiratory physicians, but for this group the price may prove prohibitive.—AK

NOTICES

International meeting on pulmonary mechanics and chest physiotherapy

The Fourth International Meeting on Pulmonary Mechanics and Chest Physiotherapy will take place in Brussels on 30 May 1992. The main topic will be breathlessness, and the free communications will relate to this. Details from Professor R Sergysels, Clinique de Pneumologie, Hôpital Universitaire St-Pierre, rue Haute 322, 1000 Brussels, Belgium.

Course on lung pathology

A comprehensive course of lectures on lung pathology and practical, hands on microscopy sessions will be held at the National Heart and Lung Institute, London, on 1–4 June 1992. The course is aimed at pathologists in training and consultant pathologists wishing to update their knowledge. The fee will be £195 (US $335). Further information from Professor B Corrin, Lung Pathology, Brompton Hospital, London SW3 6NP (Tel: 071-351 8420, Fax: 071-351 8443).