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* 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, 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, it is cause and which effect may not be self evident.

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* with interest. In their penultimate paragraph the authors speculate on high pressure PEP mask physiotherapy, a technique we have developed and investigated.1 Although we are inclined to agree with some of his speculations, we are surprised 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 by 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. It has recently been documented in a comparative study of different chest physiotherapy techniques4; 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.

The results further suggest that in these patients nutritional, there is little advantage, of respiratory muscle weakness.

The strong positive association between fat free mass and walking distance in the sub-group 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, there is little advantage, of respiratory muscle weakness.

To conclude, exercise capacity may partly 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.

Amongst 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.

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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 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.
Letters to the Editor

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

C P VAN DER SCHANS
Division of Physiotherapy,
Department of Rehabilitation.
Academisch Ziekenhuis,
Vrije Universiteit.
Amsterdam.
The Netherlands

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 arthritis. A 42 year old man 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, he made a full recovery. It is of interest that at the onset of pneumonia her total white cell count was 14.6 x 10^9/l with relative lymphopenia (total) lymphocytes 290 x 10^9/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 4.9 x 10^9/l, he had profound lymphopenia (1%, 43 x 10^9/l). This patient also required assisted ventilation but responded well to treatment, with complete resolution of symptoms.

With the increasing use of immuno-suppression 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 immuno-deficiency 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.

DAS MARSHALL
RD STURROCK
HA CAPPEL
Centre for Rheumatic Diseases,
Royal Infirmary.
Glasgow G4 0SF

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 during 3-4 June 1992. The course is aimed at pathologists in training and consultant pathologists wishing to update their knowledge. The fee will be £60. Further information from Professor B Corrin, Lung Pathology, Brompton Hospital, London SW3 6NP (Tel: 071-351 8420, Fax: 071-351 8443).
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M S Zach and B Oberwaldner

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