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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