Nutritional support in chronic obstructive lung disease

Why?
In the last decade increasing attention has been focused on nutritional problems in respiratory disorders, and particularly in chronic obstructive lung disease. This stems from an awareness that malnutrition is an important clinical problem in a subpopulation of patients with chronic obstructive lung disease. The picture of the emaciated emphysematous patient, or pink puffer, is known to every clinician. This common perception is substantiated by several reports. In a study from the United States National Institutes of Health on the effects of intermittent positive pressure breathing nearly a quarter of 779 men with stable chronic obstructive lung disease weighed less than 90% of their ideal body weight, a simple albeit imperfect index of malnutrition. The prevalence of malnutrition increases with the severity of airways obstruction, and approaches or exceeds 50% in series of patients admitted to hospital. Moreover, several studies in the 1960s showed that malnutrition in itself contributed to mortality. The more recent National Institutes of Health trial also showed that mortality was higher in malnourished patients, independently of the severity of airways obstruction. Reduced respiratory muscle mass and function, as well as increased susceptibility to infection, are recognised as deleterious consequences of malnutrition.

Because malnutrition appears to be common and life threatening in patients with chronic obstructive lung disease, the idea of providing them with nutritional support has logically emerged. Several questions need to be answered, however, before nutritional support can be efficiently implemented.

How much?
The question of how much energy needs to be provided perhaps depends on the mechanism of weight loss in chronic obstructive lung disease: is this due to reduced energy intake or to increased energy output? Earlier studies, based on dietary histories, showed that the energy intake of malnourished patients with chronic obstructive lung disease was either adequate in relation to the recommended daily allowances or at least similar to that of well nourished patients. Using indirect calorimetry, however, several groups have recently reported a 10–20% increase in resting energy expenditure in patients with stable chronic obstructive lung disease. This excessive energy expenditure contrasts with the reduced metabolic rate usually observed in malnutrition and has been attributed to the increased cost of breathing. According to these findings, weight loss would seem to be the consequence of increased energy output rather than of reduced intake. Despite a higher resting energy expenditure, however, total energy expenditure was found to be normal in patients, as measured during 24 hours in a metabolic chamber. Spontaneously reduced physical activity could save energy in these patients and compensate for their resting hypermetabolism. Thus weight loss probably results primarily from inadequate energy intake in relation to energy needs, which is likely to occur during many exacerbations of the disease.

How much support is necessary also depends on the metabolic response to nutritional supplementation. Hypermetabolism caused by stress, burns, or injuries commonly requires a very high energy and nitrogen intake to stabilise nitrogen retention. Although hypermetabolic at rest, malnourished patients with chronic obstructive lung disease achieve a positive nitrogen balance when energy intake is adequate for expenditure. Thus they react like nutritionally depleted individuals and should theoretically benefit from nutritional support.

Several controlled studies have assessed the effect of nutritional support in chronic obstructive lung disease, either in outpatients or in inpatients. In addition to changes in nutrition, the peripheral and respiratory muscle performance and exercise capacity have been evaluated. The outcome of these studies was directly related to the actual increment in energy intake. Weight gain could be achieved only by substantially increasing energy intake, by more than 30% above the usual intake, amounting to 60 more than 45 kcal (0.19 MJ)/kg per day. Moreover, improvement in muscle function or exercise tolerance occurred only with concomitant weight gain.

In one of these controlled studies oral supplementation was given for three months to ambulatory malnourished patients with chronic obstructive lung disease. Daily energy intake increased by 48% above the usual intake and corresponded to 47 kcal (0.196 MJ)/kg on average. The authors reported a mean weight gain of 4.2 kg, an increase in maximal respiratory pressures and in handgrip and sternomastoid strength, and a decrease in sternomastoid muscle fatigability; similar improvements were not observed in a control group. Unfortunately these improvements waned or disappeared once the patients had returned to their usual diet. In another study six malnourished patients with chronic obstructive lung disease received nocturnal supplemental feeding via a nasoenteric tube for 16 days. Energy intake increased by 75% above the usual and corresponded to 49.5 kcal (0.207 MJ)/kg a day on average. Body weight increased by 2.4 kg and was accompanied by an improvement in respiratory muscle strength and endurance. None of these changes occurred in four similar patients receiving sham supplementation. In contrast, no change in weight or muscle performance was observed in patients whose increment in energy intake was smaller.
What?

The composition of nutritional supplementation for patients with lung disease has received some attention. Carbon dioxide production (VCO₂) is higher when carbohydrates are the main energy sources and lower when fat is mainly oxidised. Thus, the respiratory quotient (RQ = VCO₂/VO₂) tends towards 1.0 with carbohydrate-based diets and towards 0.7 with fat-based diets. For arterial carbon dioxide tension (Paco₂) to remain constant an increased VCO₂ is dependent on an increase in alveolar ventilation. Thus, carbohydrate loads could induce or worsen hypercapnia in patients with severe ventilatory limitation. This has been observed with high-energy supplements administered to patients during mechanical ventilation or weaning.32-33

Patients with chronic obstructive lung disease who are in a stable clinical state, however, usually appear to tolerate carbohydrates without difficulty. At rest both normocapnic and hypercapnic patients were able to maintain a constant Paco₂ after a 920 kcal (3.85 J) carbohydrate load.34 No clinically-significant difference could be noted with low or high carbohydrate diets in a group of hypercapnic patients with chronic obstructive lung disease. Furthermore, respiratory failure was not reported in stable patients while they were receiving nutritional support, even with diets containing up to 54% carbohydrates.20-21,36

Thus low carbohydrate, high fat formulas do not seem necessary for nutritional support in patients with stable chronic obstructive lung disease.

How?

The failure of nutritional support that was observed in some trials conducted in patients with chronic obstructive lung disease seems mainly due to the inability of the patients to increase their energy intake sufficiently.27,28,30

Spontaneous reduction in the usual energy intake and intolerance of supplemental formulas were frequently encountered. Nocturnal supplemental feeding via a nasoenteric tube may obviate these difficulties and proved to be effective in inpatients.31 When oral supplements are used they should be given at the end of meals or between meals so that the usual dietary intake is maintained. Both patients and outpatients must have close monitoring and counselling if they are to maintain their energy intake and learn various strategies for coping with meal-related complaints such as anorexia, early satiety, bloating, and dyspnea.32 From these recent trials, nutritional intervention appears to be feasible but difficult in chronic obstructive lung disease, requiring dedicated and specially trained personnel.

For whom?

At the moment we know several things about nutritional deficiency in chronic obstructive lung disease. Firstly, malnutrition is known to represent an important clinical problem, with its high prevalence and its aggravating influence on mortality. Secondly, we are beginning to understand the disruption of energy balance that leads to weight loss in these patients. Thirdly, malnutrition has been shown to be improved by nutritional intervention. Finally, we know how nutritional support should be provided in chronic obstructive lung disease, and also that it represents a major undertaking.

We still lack some important information, however. Firstly, if nutritional support appears intuitively logical for patients with chronic obstructive lung disease its impact on morbidity, quality of life, number of hospital admissions, and mortality has yet to be seen. Once this has been done it will be essential to define selection criteria to recognise those patients likely to benefit from nutritional support. Because of the labour and cost required this intervention should not be applied indiscriminately. These questions need to be answered and a cost-benefit analysis must be performed before nutritional support can be recommended on a wide scale in chronic obstructive lung disease.

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Editor
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