Editorials

Intensive Care Medicine

Intensive Care Med (1994) 20:463–465

Does dietary manipulation influence weaning from artificial ventilation?

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In recent years it has become apparent that the nutritional status of patients with respiratory insufficiency is an important consideration in their management [1, 2]. The incidence of malnutrition is high, both in patients with chronic respiratory disease and in those hospitalized with acute respiratory failure [3–6]. A relationship exists between nutritional status and respiratory function. One study conducted in normal subjects found that several days of a hypocaloric feeding regimen depressed the subjects' response to hypoxia [1]. Subjects with chronic obstructive pulmonary disease (COPD) and concomitant impaired nutritional status demonstrated reduced respiratory muscle strength [2], decreased cell-mediated immunity [7], nutritionally induced anaemia [8], decreased production of surfactant and decreased replication of respiratory endothelium [9–11].

The composition of nutritional support in the malnourished is critical. Provision of glucose calories in excess of metabolic handling requirements can promote fatty infiltration of the liver (due to excessive triglyceride formation) with increased oxygen consumption and carbon dioxide production [12]. Protein in amounts greater than 2 g/kg per day can result in an elevated ventilatory response to carbon dioxide and an elevated ventilatory drive to which patients with chronic obstructive airway disease (COPD) and respiratory failure are unable to respond [13, 14]. Sub-optimal protein and calorie provision can results in the metabolism of endogenous protein as an energy source [15].

Glucose is the major source of non-protein calories in parenteral solutions. Oxidation of carbohydrate as the primary energy source decreases endogenous fat utilization. This result in an increased respiratory quotient (RQ) from a normal baseline of 0.85 to values that frequently exceed 1.0. As a result of the marked increase in carbon dioxide production, ventilatory failure can occur or an inability to wean from mechanical ventilation may arise [16].

In respiratory failure, the cardiopulmonary system is unable to regulate the oxygen and carbon dioxide content of the blood appropriately. The level of P_aCO_2 could be influenced by several factors, the most important being ventilation and the state of metabolism. High carbohydrate loads in both normal subjects and patients with COPD have been shown to increase carbon dioxide production and ventilatory requirements. In normal subjects, there were significant increases in tidal volume, alveolar ventilation, carbon dioxide production, oxygen consumption and respiratory exchange ratio following a carbohydrate load of 230 g (920 cal) [17]. Furthermore, Gieseke et al. [12] observed increases in minute ventilation, carbon dioxide production, oxygen consumption, and RQ after a single carbohydrate load.

The effects of a high-carbohydrate feed were also demonstrated in a study [15] involving patients with chronic nutritional depletion. A high-fat parenteral feed containing 50% fat and 50% carbohydrate as the non-protein calorie sources was compared with a parenteral feed in which carbohydrate was the sole source of non-protein calories. The carbohydrate diet resulted in a 20% increase in carbon dioxide production, a 26% increase in minute ventilation and a 14% increase in RQ compared with the carbohydrate-fat mixture. These studies demonstrated that partial substitution of carbohydrate with fat reduces carbon dioxide production and ventilatory requirements. Askanazi et al. [15, 18] also showed that the increase in carbon dioxide production following carbohydrate ingestion is exaggerated at high caloric intakes. This, however, is dependent on the metabolic state of the patient. In normal subjects receiving caloric intakes of 150–225% of the resting energy expenditure, both oxygen consumption and carbon dioxide production increased, with the RQ rising from 0.85 to above 1.0 indicating net lipogenesis. Acutely ill hypermetabolic patients responded similarly with a 29% increase in oxygen consumption and a 56% increase in carbon dioxide production. The average RQ was 0.9 indicating that, despite the provision of glucose calories, these hypermetabolic patients continued to use fat as an energy source [15, 18]. In contrast, nutritionally depleted patients without pulmonary disease who were receiving a high-carbohydrate regimen had a 32% increase in carbon dioxide

production while oxygen consumption remained essentially unchanged, resulting in an RQ greater than 1.0.

Respiratory failure can be precipitated in patients with COPD given a high-carbohydrate diet [16, 19]. Reductions in carbon dioxide production (20%), RQ (7%), and P_aCO_2 (4%) have been seen when patients with stable COPD were switched from a standard enteral feed to one high in fat content [20].

A greater effect was seen when critically ill patients requiring artificial ventilation were studied [21]. Changing from a standard enteral feed to one high in fat and low in carbohydrate resulted in a drop in P_aCO_2 of 16% (prior to weaning) as opposed to a 4% increase. The high-fat group spent less time on the ventilator. However, in this study only 1 patient out of 20 was ventilated for acute or chronic respiratory failure due to COPD.

In this regard, the study reported by van den Berg et al. [22] in this volume is most interesting. These authors studied the effect of altering the proportion of dietary fat and carbohydrate (using similar feeds to that used by Al-Saady et al. [21]) in reducing VCO_2 during ventilatory support and weaning from the ventilator as a means of facilitating the weaning process. They examined the ventilatory and metabolic data in 32 ventilator-dependent patients using a non-invasive technique (indirect calorimetry), which allowed them to measure VCO_2, VO_2 and RQ frequently. They found that high-fat feeding was associated with significantly lower RQ values than standard feeding, during both mechanical ventilation and weaning (9% and 19%, respectively). High-fat feeding also reduced CO_2 elimination during both mechanical ventilation and weaning, but the reduction only proved to be significant during weaning. Furthermore, these investigators could not find a significant difference in P_aCO_2 during weaning between the two feeding groups.

Aside from the important issue as to whether or not the variation in dietary composition can reduce the time patients spend on the ventilator and facilitate weaning, and how the criteria of readiness for weaning from the ventilator can be defined this study raised several questions that need to be addressed before its results can be applied clinically. Firstly, it is not clear whether these patients were malnourished before the start of the study. Malnutrition produces potentially reversible changes in the contractile properties of the respiratory muscles [23]. These include early slowing of relaxation rate, alteration in force-frequency relationships, and decreased endurance. Hypermetabolism in critically ill patients also increases ventilatory requirements [18]. In this setting, moderate degrees of malnutrition and respiratory muscle weakness may prolong respiratory failure and delay the transition to spontaneous ventilation. Investigators [24] have demonstrated that there is a good correlation between inspiratory occluded mouth pressure and measurements of body-cell mass. Abnormalities of respiratory muscle function and endurance may explain the tendency towards respiratory failure in malnourished patients with COPD [25–27]. Nutrition-mediated respiratory muscle dysfunction begins to occur within days of deprivation and, similarly, will respond promptly to appropriate refeeding therapy [28]. Furthermore, there was no indication whether their patients received full caloric supplementation from the first day of feeding. This is very important, since day 3 of feeding was used as the time for collection of data for comparison between the two groups. In a previous study [21] it was shown that it takes about 18–24 h of continuous feeding to reach the optimal rate. The same authors have also shown that changes P_aCO_2 only become apparent after the third day of feeding. Nevertheless, for day 3 feeding (their Table 2) they reported a 9% decrease in RQ, 12% in VCO_2 and 10% in minute ventilation at the same P_aCO_2 concentration in the high-fat group. When these results were compared during weaning, both RQ and VCO_2 (their Table 3) were significantly lower (16% and 23%, respectively) in the high-fat group, probably a reflection of the continued fat utilization more than carbohydrate, as these patients would have been fed for a longer time. The changes were achieved despite a 16% drop in minute ventilation in the high-fat group in this study. Secondly, there was an imbalance (as mentioned in their paper) between the two feeding groups, with more hypercapnic (more acute and chronic COPD) patients in the high-fat group than in the standard-feed group. These patients would have a more exaggerated ventilation/perfusion mismatch which might affect CO_2 elimination. The mismatch also increases respiratory muscle work to compensate for reduced gas exchange efficiency. This means that even if carbon dioxide production were lower in the high-fat group, elimination would be hindered by the more severe mismatch in these patients. Thirdly, these authors also reported that the median time of their study was 4 days in the high-fat, low-carbohydrate feeding group, and 6 days in the standard-feeding group, a difference that failed to reach significance.

Thus this study and others like it, which attempt to define the role of dietary manipulation on weaning time, have shown that altering the proportion of fat and carbohydrate in enteral feeding resulted in changes in VCO_2, RQ and the time patients spent on the ventilator. However, the failure in this study to demonstrate significant changes in ventilation time could well be influenced by the various factors listed above and the number of patients studied.

This study needs to be followed by a prospective clinical trial because of the positive findings. The rationale for such studies is that since the alteration in the proportion of fat and carbohydrate in enteral feeding is potentially detrimental to the function of the respiratory system, it would be useful to study ventilator-dependent patients with COPD patients grouped separately, as the patiens in the present study represent a non-homogeneous group as regards their nutritional status and respiratory function. It is also important to define weaning criteria for different patient groups and to document the nutritional history of these patients and whether or not they were malnourished before starting nutrition.

Further clinical studies are also necessary to determine the effect of nutritional supplementation on morbidity and mortality in ventilator-dependent patients.

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