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## Enteral nutrition in patients with respiratory disease

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**ABSTRACT: Nutritional assessment and management is an important therapeutic modality in patients with respiratory disease. Malnutrition adversely affects respiratory function. Nutritional therapy for the spontaneously breathing patient should include an appropriate diet plus the consideration of nutritional supplements. Complete nutritional support should be undertaken with enteral nutrition in critically ill patients with respiratory failure. Nutritional complications occur. Overfeeding can lead to nutritionally associated hypercapnia.**

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Nutrition is an important aspect of patient care in any patient with respiratory disease. Malnutrition adversely affects lung function by diminishing respiratory muscle strength, altering ventilatory capacity, and impairing immune function. Repletion of altered nutritional status or refeeding results in improvement of altered function and may be important in improving outcome. When spontaneous oral intake is inadequate, enteral feeding is preferred over parenteral feeding in all but those with nonfunctional gastrointestinal tracts. Unfortunately, as with any therapy, complications of nutritional support exist. Those complications presenting special problems to the patient with respiratory disease are nutritionally-related hypercapnia and aspiration of enteral feedings. This article considers the association of respiratory disease and malnutrition, the determinants of appropriate nutritional support in respiratory disease, the use of enteral nutritional support to reverse malnutrition, and the complications associated with enteral feeding. Although patients with a variety of respiratory diagnoses are appropriate targets for this discussion, the article will deal largely with patients with chronic obstructive pulmonary disease (COPD), as this is the respiratory disease most commonly studied. General principles involved in the nutritional care of the COPD patient can be applied to patients with other respiratory diagnoses.

### Adverse effects of malnutrition

A substantial proportion of patients with COPD are malnourished. The incidence depends largely upon disease severity. As many as 25% of out-patients with COPD may be malnourished while almost 50% of patients admitted to hospital have evidence of malnutrition [1, 2]. Critically ill COPD patients with acute respiratory

failure (ARF) have a 60% incidence of malnutrition [3]. Disease severity can be assessed by the degree of pulmonary function and gas exchange abnormalities. Malnutrition occurs in 50% of patients with chronic hypoxaemia and normoxaemic patients with severe airflow obstruction (forced expiratory volume in one second (FEV<sub>1</sub>) <35% of predicted); however, it is also present in 25% of patients with moderate airflow obstruction [4].

Poor nutritional status can adversely affect thoraco-pulmonary function in spontaneously breathing as well as mechanically-ventilated patients with respiratory disease by impairment of respiratory muscle function, ventilatory drive, and pulmonary defence mechanisms [5] (table 1). The adverse effects of malnutrition occur independently of the presence or absence of primary lung disease; however, they can be additive in some patients with ARF, such as those with respiratory failure due to COPD. In COPD, primary abnormalities of decreased inspiratory pressure and increased work of breathing are found. Inspiratory muscle weakness, as assessed by maximal inspiratory pressure, results both from mechanical disadvantage to inspiratory muscles consequent to hyperinflation and generalized muscle weakness [6, 7]. In COPD, inspiratory muscle weakness must be severe for hypercapnia to occur. In patients with myopathy, hypercapnia occurs when inspiratory pressures are less than one third [7]. However, hypercapnia is found in the majority of COPD patients when inspiratory pressures are only less than half normal [8]. Thus, hypercapnia occurs with a much lower level of respiratory

Table 1. – Adverse effects of malnutrition on thoraco-pulmonary function in patients with respiratory disease

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Decreased respiratory muscle strength  
Altered ventilatory drive  
Impaired immunological function

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muscle weakness when other mechanical abnormalities are present that increase the work of breathing. Thus, malnutrition may further compromise an already compromised lung function. Dyspnoea may worsen in the spontaneously breathing COPD patient. Hypercapnic respiratory failure and/or difficulty in weaning from mechanical ventilation may be more easily precipitated in the malnourished patient with COPD than in the normally nourished patient with COPD.

In simple starvation or undernutrition, fat and protein are lost, but the loss of protein is minimized by reducing the need to use it as a source of energy [9]. Nitrogen loss is modified by mobilization of fat, and enhanced fat oxidation is the principal source of energy in the starving individual. Some protein wasting does occur, despite the availability of fat as a source of energy, and it becomes markedly accelerated when fat stores are used up. When body weight drops to less than 80% of ideal body weight, protein catabolism occurs in the spontaneously breathing COPD patient. In critical illness, protein catabolism occurs to provide energy. With inadequate caloric intake in critically ill patients, energy sources are derived from protein breakdown and glyconeogenesis. Of various protein "pools" available, the muscle protein pool is susceptible to catabolism to provide fuel [10]. Inspiratory and expiratory muscles, primarily the diaphragm and intercostals, are skeletal muscles and therefore susceptible to this catabolic effect. Because the diaphragm is the principal respiratory muscle, the following discussion will focus on it, although these considerations are generally valid for all respiratory muscles. It is important to note that little, if any, data exist directly examining respiratory muscle function and malnutrition in critically ill, mechanically-ventilated patients with COPD.

Malnutrition reduces diaphragmatic muscle mass in health and disease [11, 12]. In necropsy studies, body weight and diaphragmatic muscle mass were reduced, respectively, to 70 and 60% of normal in underweight patients dying of a variety of diseases [12]. Animal studies confirm the loss of diaphragmatic strength in prolonged and acute nutritional deprivation [13, 14]. Respiratory muscle function is also impaired in poorly nourished humans. When malnourished patients without lung disease were studied, respiratory muscle strength, maximum voluntary ventilation and vital capacity were reduced by 37, 41 and 63%, respectively [15]. Respiratory muscle strength in patients without a systemic disease is also decreased. Maximal inspiratory pressures were lower in malnourished postoperative patients compared to normally nourished patients [16]. Recently, similar data have also been described in anorexia nervosa patients, a relatively pure model of malnutrition without systemic disease [17]. Transdiaphragmatic pressures elicited by phrenic nerve stimulation, were markedly diminished in anorexia patients before institution of enteral nutritional support.

The effect of nutritional status on respiratory muscle function in patients with COPD is controversial. In COPD, primary abnormalities of decreased inspiratory pressure and increased work of breathing are found. Inspiratory muscle weakness, as assessed by maximal inspiratory

pressure, results from mechanical disadvantage to inspiratory muscles consequent to hyperinflation and perhaps generalized muscle weakness [18]. Controversy exists as to the additive role of denutrition in the aetiology of the measured inspiratory muscle weakness. Cystic fibrosis (CF) patients with hyperinflation and malnutrition were compared to asthmatics with hyperinflation but no malnutrition and to anorexia nervosa patients with malnutrition but no hyperinflation, as well as control patients with neither [19]. Peak inspiratory pressures in CF with hyperinflation were decreased as were pressures in anorexia nervosa patients. With volume correction, however, the difference in inspiratory strength in the CF group disappeared. These data suggest that hyperinflation may be a major cause of diminished respiratory muscle weakness in COPD. In contrast to these data, renutrition studies in COPD as well as CF patients documenting improved muscle strength suggest that malnutrition is an important cause of diminished muscle strength [20, 21].

Malnutrition also affects ventilatory drive [22]. The interaction of nutrition and ventilatory drive appears to be a direct function of the influence of nutrition on metabolic rate [23]. In general, conditions which reduce metabolic rate reduce ventilatory drive. A decrease in metabolic rate occurs with starvation. A parallel fall in metabolic rate and hypoxic ventilatory response has been documented in humans [23]. A 58% reduction in the ventilatory response to hypoxia was found in volunteers placed on a balanced 550 kcal·day<sup>-1</sup> diet for 10 days. The ventilatory response returned to normal with refeeding. Ventilatory response is also affected by constituents of the diet. After a 7 day protein-free diet, a blunted ventilatory response to carbon dioxide was noted [24].

Consequences of decreased respiratory strength and decreased ventilatory drive could include decreased cough and, thus, increased likelihood for atelectasis and subsequent pneumonia in spontaneously breathing patients with any type of respiratory disease. A decrease in respiratory muscle strength and drive may also possibly prolong the duration of mechanical ventilation in patients who are otherwise candidates for weaning. Thus, the potential for adverse outcomes is present in patients who are initially malnourished from their disease as well as in patients with respiratory disease who develop malnutrition as a consequence of other intercurrent diseases.

Malnutrition has also been shown to alter immune function. Protein calorie malnutrition is the most frequent cause of acquired immunodeficiency in humans [25]. Polymorphonuclear leucocytes are normal in number, and chemotaxis, opsonic function and phagocytic function usually remain or are mildly depressed, whilst intracellular killing is reduced [26]. Thymus, spleen and lymph nodes become markedly atrophic, and lymphocytes may decrease. Whilst immunoglobulins remain normal or slightly increased, antibody response may be depressed [26].

#### **Effect of renutrition on malnutrition**

Nutritional repletion can improve diminished respiratory muscle strength in some patients. A 37% increase in maximal inspiratory pressure and a 12% increase in

body cell mass was found in 21 of 29 hospitalized patients given parenteral nutrition for 2–4 weeks [16]. Short-term oral refeeding in malnourished COPD patients can also improve respiratory muscle function, although it appears to depend on the presence of weight gain [20]. When six ambulatory patients with COPD were given oral nutritional repletion for 2 weeks, body weight increased by 6% and transdiaphragmatic pressures increased by 41% [20]. In contrast, when 8 weeks of nutritional supplementation in 21 malnourished COPD patients produced no change in weight, no change in respiratory muscle function was found [27]. Intensive, nocturnal, nasoenterally-administered nutrition in COPD and cystic fibrosis can result in weight gain and improved respiratory muscle and pulmonary function [28]. Renutrition has also been found to improve diaphragmatic contractility in a more "pure" model of malnutrition, that of anorexia nervosa [17]. After 1 month of enteral nutrition (weight gain 15%), stimulated transdiaphragmatic pressure ( $P_{di}$ ) was increased from  $16 \pm 5$  to  $23 \pm 7$  cmH<sub>2</sub>O, documenting improved diaphragmatic function with renutrition. With long-term nocturnal enteral feeding, CF patients were found to have improved pulmonary function in conjunction with significant weight gain [21].

The mechanisms of improved muscle performance with renutrition is not known with certainty. In animal and human studies, chronic hypocaloric dieting produces changes in skeletal muscle that may be important in the genesis of muscle dysfunction. In addition to protein catabolism, these changes include depletion of glycolytic and oxidative enzymes, reduction in high-energy phosphate stores and increases in intracellular calcium [29, 30]. The electrophysiological properties of the muscle can also be altered by modification of the cell membrane properties, which decrease the sodium potassium pump activity, alter ionic permeability and, thus, lead to an imbalance in the intercellular electrolyte composition [29]. These data suggest that alterations in muscle contractility and endurance properties are not simply or solely due to changes in lean tissue. Indeed, renutrition studies in hypocaloric dieting and fasting and in the severe starvation of anorexia nervosa patients document improvement in muscle performance at a time when significant changes in body composition could not be detected [31]. Changes in intracellular electrolytes may be responsible for early improvement in muscle contractility and endurance properties.

### Nutritional support

The optimum mode of nutrition in any patient is oral, spontaneous intake of an appropriate balanced diet. Unfortunately, patients with respiratory disease may require supplementation or even complete nutritional support, depending on the severity and intensity of illness. However, the principles of nutritional support are independent of the type of respiratory disease, the mode of nutritional administration, or the severity of respiratory illness. Whether nutritional support requires either supplementation or total support, the following discussion will focus on

enteral nutrition, as the enteral route is preferred whenever nutritional support is indicated.

### Energy needs

Several methods exist for estimating caloric requirements of patients with respiratory disease. Levels of energy expenditure can be estimated, calculated with formulae or nomograms, or determined by using measurements of energy expenditure (table 2). In mechanically-ventilated patients with respiratory disease, guidelines of 25 kcal·kg<sup>-1</sup> daily have been suggested [32]. Estimates of basal metabolic rate (BMR) *via* a resting energy expenditure (REE) can be obtained from the Harris-Benedict equation, which relates energy expenditure to sex, weight in kilograms (W), height in centimetres (H), and age in years (A).

$$\begin{aligned} \text{BMR (males)} &= 66.47 + 13.75 (W) + 5.0 (H) - 6.76 (A) \\ \text{BMR (females)} &= 655.1 + 9.65 (W) + 1.85 (H) - 4.68 (A) \end{aligned}$$

A "stress factor" or percentage increase in energy requirement is then added to this determination, based on the severity of the patient's illness. Stress factors are based on estimated metabolic needs over and above resting needs, and will vary with respect to body temperature, degree of physical activity, or extent of injury [33]. Most critically ill patients with respiratory disease require a stress factor of 1.2. The utility of the Harris-Benedict equation in clinical practice is controversial. Caloric needs may be inaccurate, with overestimation of caloric requirements [34]. It is, however, a relatively simple method of estimating caloric requirements, especially in critically ill patients.

The most accurate method of determination of energy requirements is indirect measurement of actual energy expenditure with a metabolic cart. In this case, caloric requirements can be indirectly determined by measuring the rate of oxygen consumption, each litre representing approximately 4–5 kcal. Metabolic carts can be used to measure oxygen consumption both in mechanically-ventilated and spontaneously breathing patients but are expensive and require technical expertise. Unfortunately, the stringent conditions that must be imposed during these study periods are not the ordinary conditions of clinical care. Also, it should be noted that while indirect calorimetry may accurately reflect the energy requirements over the 30–60 min time-period of measurements, it is difficult to know how to extrapolate this measure to a 24 h time-period.

Table 2. – Determination of daily expenditure in patients with respiratory disease

Estimation	25 kcal·kg <sup>-1</sup> daily for respiratory failure
Calculation	Resting energy expenditure Harris-benedict plus "stress" factor
Measurement	Indirect calorimetry Pulmonary artery catheter measurements

Energy requirements in COPD patients follow general guidelines, with several caveats. Malnourished spontaneously breathing COPD patients have increased resting energy requirements, approximately 15% above values predicted by Harris-Benedict equations, resulting in far greater expected energy requirement [35]. The "relative hypermetabolism" is explained by the increased energy needs of the ventilatory muscles [36]. The energy cost of respiratory muscles can be approximated from the severity of lung hyperinflation. Assessment of these points should be made in the perspective of whether the COPD patient is spontaneously breathing or being mechanically-ventilated. Nutritional support in the spontaneously breathing COPD patient should also take in account the limitations that COPD patients have in augmentation of caloric intake, such as early satiety, anorexia, bloating and fatigue.

When calculating, estimating, or measuring total daily energy needs, it is important to remember that the nutritional goal is appropriate total daily calories, *i.e.* neither underfeeding nor overfeeding the patient. Whether the intake is spontaneous, supplemented or completely controlled, physicians caring for the patient with respiratory disease should determine appropriate daily calories. Underfeeding the patient over a long period of time or during hypermetabolic states, such as critical illness, risks the adverse effects of malnutrition on thoracopulmonary function. Overfeeding the patient risks metabolic complications, especially nutritionally related hypercapnia.

*Substrate mix*

Once total energy requirements are determined, the next question relates to the most appropriate substrate mix, that is the percentage of total calories that are carbohydrate, fat and protein. Protein (nitrogen) requirements in the patient with pulmonary disease are not significantly different from that in other patients. Optimal support would establish neutral or positive nitrogen balance, depending on the need for repletion. In the critically ill patient with ARF, this can be accomplished by giving 1–3 g of protein·kg<sup>-1</sup> daily [32]. Generally, this amounts to approximately 20% of total calories being administered as protein.

The most appropriate carbohydrate/fat substrate mix for COPD patients is complicated and controversial. The precise substrate mix is largely an issue for respiratory disease patients in the Intensive Care Unit, where nutritional support is totally controlled and adverse sequelae are theoretically more likely. Spontaneous oral intake is less problematical, except for those occasions when the intake is supplemented by prepared oral formulations.

Although the critically ill patient with respiratory failure does use lipid preferentially as a fuel source, glucose oxidation is not impaired, and lipid infusion probably does not change patterns of fuel oxidation [37]. Thus, there is no theoretical metabolic reason to choose one fuel over the other. There is also no benefit of glucose over lipids and *vice versa* in the "sparing" effect of proteins.

Clear disadvantages of carbohydrate administration exist. Hyperglycaemia, especially in diabetics or patients receiving corticosteroid therapy, can be exacerbated by high dextrose concentrations. Elevated blood glucose can negatively affect humoral immune function and potentiate the growth of *Candida albicans* [38]. Excess glucose administration is not oxidized but stored as body fat. Clinically, this can result in increased fatty deposition in the liver, as well as nutritionally associated hypercapnia.

Fat calories are required in total nutritional support to provide essential fatty acids. Intravenous lipids, even during slow administration, may cause pulmonary haemodynamic changes in injured lungs [39]. The clinical significance of these changes may be small. Lipids, especially long-chain triglycerides, can impair reticuloendothelial clearance functions, even when hypertriglyceridaemia is absent [40]. Hepatic steatosis is significantly influenced by the proportion of fat calories as well as glucose calories in excess of caloric needs [41]. Despite many disadvantages of intravenous lipids, fats in enteral feeding formation are well-tolerated with few adverse effects.

While recommendations for an appropriate substrate mix of carbohydrates and fats vary, generally 60–70% carbohydrates are given with 20–30% fats (table 3).

**Complications of nutritional support**

Multiple complications are associated with enteral nutrition and are of importance to the patient with respiratory disease (table 4). Complications can be generally classified into mechanical, infectious, gastrointestinal and metabolic types. Whilst of concern to all patients requiring enteral nutrition, patients with respiratory disease are particularly susceptible to adverse sequelae of pulmonary

Table 3. – Nutritional recommendations for patients with respiratory disease

Determination of daily energy requirements (total calories)	
Substrate mix	
Protein	20% of total calories
	1–2 gm·kg <sup>-1</sup> daily
Carbohydrates	60–70%
Fats	20–30%

Table 4. – Complications of enteral nutritional support

Mechanical	
	Inadvertent tracheal intubation
	Clogging or obstruction of tube
	Aspiration of enteral feeding
Gastrointestinal	
	Vomiting
	Abdominal distension
	Diarrhoea
Metabolic	
	Hyperglycaemia
	Hypophosphataemia
	Hypercapnia

aspiration and the metabolic complication of nutritionally-related hypercapnia.

Pulmonary aspiration of enteral feeding has the potential for significant adverse sequelae in patients with respiratory disease. Large volume aspiration of enteral feeding can precipitate or worsen respiratory failure, small volume aspiration can cause nosocomial pneumonia with the potential for sepsis. The frequency of pulmonary aspiration is difficult to determine as the incidence of pulmonary aspiration reported with gastric intubation by feeding tubes varies widely, ranging 0.8–77% [42–44]. In a review of 253 hospitalized patients treated with enteral nutritional support, only two patients (0.8%) were diagnosed as suffering aspiration pneumonia [42]. When detected by glucose-positive endotracheal secretions, aspiration was diagnosed in 21% of mechanically-ventilated patients [43]. Likewise, 77% of tracheally-intubated patients were found to aspirate as determined by the presence of methylene blue in tracheal secretions [44]. It is difficult to draw general conclusions about the effect of feeding tubes from these studies because all differ in the population studied, study design, method of diagnosing aspiration, and size of enteral feeding tube.

Multiple factors can affect the frequency and severity of aspiration. Clearly, the presence of an endotracheal tube and the type of endotracheal tube influences the incidence of aspiration [45]. Other risk factors for aspiration include a reduced level of consciousness, with consequent compromise in glottic closure, the presence of an artificial airway, and ileus or gastroparesis [46].

Small bore feeding tubes are generally recommended as a means of reducing gastro-oesophageal reflux and, ultimately, pulmonary aspiration. Data supporting this recommendation are controversial. No aspiration of methylene blue dyed tube feeding was found in 30 ventilated patients with small bore (8 F) feeding tubes [42]. In contrast, bore size was not a significant variable in witnessed aspiration or aspiration pneumonia in hospitalized patients [47]. Recent data suggest that feeding tube size may not be an important variable in reflux. When reflux was assessed by gastro-oesophageal scintiscanning in normals, no difference was found in reflux between large bore (14 F) and small bore (8 F) nasogastric feeding tubes [48]. These data suggest that additional factors may be more important in reflux. One of these is clearly the position of the patient. In early work, normal volunteers with nasogastric tubes were studied in the supine, 10, 30 and 45 degree position, as well as sitting upright. After a large volume of acid had been instilled into the stomach, aspiration, as detected by a fall in oesophageal pH, occurred only in the supine position [49]. More recent data confirm the importance of patient position, with studies of pulmonary aspiration by scintiscanning of tracheal secretions after isotopic labelling of enteral nutrition [50]. Radioactive counts and, therefore, aspiration were four times higher in the supine position than in the semirecumbent position. Duration of supine position is also important. Aspiration increased 650% 1–6 h after placement in the supine position.

These data confirm the presence of aspiration with enteral feeding, but documentation of aspiration is difficult. Recent data suggest that detection of aspiration by glucose determinations of tracheal secretions may be misleading [51]. Prevention of aspiration of gastric content should be directed at minimizing the mechanical factors contributing to regurgitation, such as patient elevation and improper tube placement. Gastric residuals should be frequently checked, especially in patients at risk for slow gastric emptying.

Nutritionally associated hypercapnia is a notable metabolic complication of nutritional support in patients with respiratory disease. Nutritionally associated increases in carbon dioxide production ( $V'\text{CO}_2$ ) can produce hypercapnia [52]. Clinical sequelae include worsening of dyspnoea and exercise tolerance, precipitation of ARF, and delayed weaning from mechanical ventilation [53, 54]. Hypercapnia results from increased  $V'\text{CO}_2$  for two reasons. Glucose combustion causes a greater  $V'\text{CO}_2$  than combustion of lipid, in that an isocaloric substitution of all lipid for all glucose calories results in a 22% reduction in  $V'\text{CO}_2$  [52]. More importantly, excess glucose calories result in lipogenesis and markedly increased respiratory quotient (RQ). The RQ of glucose is 1.0 and of fat is 0.7; however, the RQ of lipogenesis or fat production is approximately 8.0, reflecting the proportionally greater  $V'\text{CO}_2$  relative to oxygen consumption ( $V'\text{O}_2$ ) with lipogenesis. In normal subjects, hypercapnia is avoided by increased ventilation. Patients with compromised ventilatory status, such as COPD, or those with fixed minute ventilation due to weak respiratory muscles may not be able to increase ventilation appropriately. Hypercapnia can result and precipitate respiratory distress, ARF and difficulty in weaning from mechanical ventilation [53, 54].

The cause of nutritionally related hypercapnia is generally thought to be excess carbohydrate or glucose calories. Some confusion has existed, however, as to whether those excess glucose calories relate to an excess proportion of carbohydrate in a nutritional regimen or simple excess of total glucose calories. Prior data suggest that the cause of elevated  $V'\text{CO}_2$  and hypercapnia is simple excess total calories. A 62% increase in  $V'\text{CO}_2$  in surgical patients was found to precipitate respiratory distress [55]. Total daily calories were  $2.25 \times \text{REE}$ . ARF was precipitated after the onset of total parenteral nutrition (TPN) in three elderly patients with COPD [53]. Total calories were in excess of  $2,200 \text{ calories} \cdot \text{day}^{-1}$  in all. More recent data confirm increased  $V'\text{CO}_2$  with stepwise increases in total calories [56].  $V'\text{CO}_2$  increased in 10 mechanically-ventilated patients receiving nutritional regimens at 1.0, 1.5 and  $2.0 \times \text{REE}$  with 60% carbohydrate and 20% fat. The  $V'\text{CO}_2$  was significantly increased at 1.5 and  $2.0 \times \text{REE}$  compared to baseline. In contrast, when 10 additional patients were given isocaloric regimens that varied the percentage carbohydrate from 40–60 to 75% of total calories,  $V'\text{CO}_2$  was not different. These data suggest that total calories more clearly influence  $V'\text{CO}_2$  production than percentage of carbohydrate calories when total calories are not excessive.

Quantitation of  $V'\text{CO}_2$  is accomplished by indirect calorimetry or by analysing a timed collection of expired air.

This problem can be avoided by identifying patients at risk, especially the patient with respiratory disease, and avoiding excessive total calories.

Enteral formulations with altered carbohydrate/fat ratios have been developed and promoted, especially for patients with COPD. These formulations commonly have lower concentrations of carbohydrate calories with resultant higher fat concentrations. Data evaluating  $V'\text{CO}_2$  following administration of isocaloric nutritional regimens in COPD are limited. Eight postoperative patients received isocaloric nutritional regimens ( $1.5 \times \text{REE}$ ) of either 100% glucose or 50% glucose and 50% fat [57]. The  $V'\text{CO}_2$  increased in both nutritional regimens as compared to basal values, although it was 11% higher with glucose alone compared with glucose and lipid. In a study of exercise gas exchange in normals,  $V'\text{CO}_2$  decreased from 290 to 240 mL·min<sup>-1</sup> with a low carbohydrate (10%) concentration [58]. However, despite this decrease, mean minute ventilation was not different during rest or exercise, suggesting that numerical decreases in  $V'\text{CO}_2$  from very low percentage carbohydrate calories may not have a practical significance in decreasing minute ventilation. These data suggest there is little value for special enteral formulations in COPD that decrease carbohydrate calories when total calories are appropriate.

### Summary

In summary, malnutrition adversely affects the patient with respiratory disease by impairing respiratory function, increasing symptoms and worsening outcome. Physicians should evaluate nutritional status and intake in all patients with respiratory disease. When spontaneous oral intake is judged inadequate, consideration should be given to supplementing oral intake with enteral nutritional formulations or, in cases of critical illness, providing complete nutritional support with enteral feeding. Estimation or calculation of total energy needs is of vital importance in the patient with respiratory disease to prevent under- or overfeeding. Reduction of carbohydrate calories, especially in supplemental nutritional formulations, appears to be of little value and not necessary when appropriate nutritional support is provided.

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