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Nutrition in respiratory failure

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Malnutrition, pneumonia, sepsis, respiratory and other organ failure are the major complications and causes of death in critically ill patients [1]. In particular, pneumonia and respiratory failure are clearly associated with protein calorie malnutrition [2–4] that can impair respiratory muscle function, ventilatory drive and pulmonary defense mechanism [5–7].

Interestingly enough most of the causes of the first two pulmonary changes can be restored by nutritional repletion [1, 8–12] even if it does not necessarily follow that improving the nutritional status will improve prognosis [13–15].

Nevertheless many authors believe that nutritional therapy should be given to patients who are or would be at risk from the metabolic complications of starvation [5–7, 16–18]. However, the feeding of patients with impending respiratory insufficiency or requiring some sort of respiratory support is a complicated matter. We have to consider the thermic effects of nutrients (DIT) i.e. the energy needed for absorption, processing and storage of nutrients that constitute the rate of energy expenditure (EE) increase above the prefeeding base line [19].

In normal volunteers, DIT is about 10%–15% of the energy intake of a meal [20], but about 10% [20] to 20% after 5–7 days [21] of a continuous enteral balanced diet at a dosage of 2–3 times above the resting EE (REE). By contrast, infusion at a rate exactly balancing the REE seems to abolish DIT [20]. DIT also depends upon the nutrients used ranging from 30%–40% for proteins to 6%–8% for carbohydrates and 2%–3% for lipids [19]. As a consequence, DIT is higher with a high carbohydrate formula enteral feed compared with a high fat formula in both malnourished (32% vs 17% after 3 days of full enteral infusion [22]) and injured patients (14%–16% vs 10% during TPN [18, 23, 24]). Thus, it is possible to have some control via nutritional means over the overall EE.

Increased EE obviously increases oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) and, as a consequence, cardiac output and ventilation; it also promotes heat dissipation leading to a possible increase in body temperature in suboptimal conditions of hydratation and circulation [25]. Gas exchange largely reflects the composition of the burned fuels; in comparison with carbohydrate, protein oxidation is associated with a 16% increase per kcal in $\dot{V}O_2$ whereas lipid produces only a 7.4% increase per kcal. By contrast, $\dot{V}CO_2$ per available kcal decreases, 6.6% with protein and 24% with fat oxidation [26]. Again, nutritional therapy is another means of modulating cardiopulmonary workload.

The $\dot{V}CO_2/\dot{V}O_2$ ratio (R) is characteristic and constant for the oxidation of a given substrate, but R being only a ratio, it cannot provide information about the absolute rate of gas exchange, the only variable of clinical relevance for cardiopulmonary function [27]. Moreover, R provides more useful information when taken together with the urinary excretion of nitrogen, to determine the net proportion of carbohydrate, lipid and protein utilized to meet E requirements. Studies in the variations in R with various different substrates, suggest a rank of preferences in drawing E from different substrates. Reviewing this topic in catabolic patients [18] it appears that with the same glucose load (less than REE) an increase in exogenous fat availability does not decrease the glucose oxidation rate which occurs when the glucose load is provided without fat. By contrast, increased glucose availability reduces fat utilization [28–29]. This preferential metabolism of glucose was isotopically confirmed in septic and cancer patients [30]. Moreover, R on its own, does not indicate whether the oxidized fuels are derived from body stores or from the diet; isotope studies have shown that the amounts of exogenous oxidized glucose and fat, are even lower than that

indicated by calorimetry [28, 29, 31–34]. Thus, fuel storage and stored fuel oxidation are also common effects of nutrition. Rapidly available calorie substrates rank from long chain saturated and polyunsaturated fatty acids (being the worse [35]), to glucose i.e. the same rank as for preferential E sources.

These findings suggest that we also have the possibility to choose between an E source mainly replacing body stores, even if oxidized after unknown time (fat), and a source more readily available (carbohydrate).

Another aspect of the problem is the protein sparing effect of each calorie substrate [18].

Returning to the patient with a poor or failing cardiopulmonary system we have the following theorical nutritional alternatives:

- to supply E at a rate below or not exceeding REE trying to avoid a net substrate storage thermic effect. This would minimize (TPN) or abolish (continuous enteral feeding) DIT.
- to give the full calorie load (about 1.2–1.5 REE) as a high fat formula diet. This approach minimizes the DIT of the selected calorie load, reduces $\dot{V}CO_2$ with respect to a more balanced diet, whereas $\dot{V}O_2$ will be increased. Exogenous lipid will mainly replenish body fat stores that are continuously mobilized to meet the EE which is not satisfied by the low glucose load. It is obvious that the patients must have enough fat stores to cope with the immediate daily E requirements.

Such an approach is well documented by Al-Saady et al. in the last issue [36]. They were able to demonstrate a shorter period of ventilatory support in a case mix of ICU patients treated by continuous enteral nutrition with a high fat vs a standard balanced formula.

This finding is important because it represents the clinical demonstration of the stoichiometric analysis of glucose and lipid metabolism and it adds yet another choice for an articulated approach to respiratory patients depending both on physiological premises and also the patient's condition and the various different therapeutic goals.

In the case of hypoxemic patients with ARDS who have a reduced healthy lung (low compliance, high shunt and dead space) coupled with an increased REE and protein catabolism, it is important not to increase $\dot{V}O_2$, $\dot{V}CO_2$, ventilation and airways pressure. However, the maintenance of E with rapidly available calories and a limitation in the loss of protein are vital. Then a possible regimen might be 1000–1300 kcal, preferentially as glucose (with insulin if necessary) and amino acids by vein, shifting to a balanced enteral feed as early as possible, whilst attempting to provide a calorie load that is close to the measured REE. We have also applied this regimen with good gas exchange results to patients with severe ARDS on extracorporeal CO_2 removal [37].

In case of ventilatory failure in well nourished, eumetabolic patients, we agree with Koretz [15] and wait 5–7 days for an improvement in the general and respiratory condition before starting nutrition. If no improvement in ventilatory capacity is observed, we have to remember that nutritional intake may increase ventilatory drive so accelerating the liberation of patients from ventilator [38] providing their lung is not compromised in some way. Finally, depleted, eu, or moderately hypermetabolic patients have to be treated immediately with at least the required E intake with the purpose of restoring as much as possible the various metabolic derangements and the lean body mass [2]. The complete net replacement of protein and E stores with the extra fuel supply required should be postponed until ventilatory failure is reversed. It might also be possible to reduce $\dot{V}CO_2$, and so ventilation-induced barotrauma by adopting the proposed high fat formula diet using the enteral route as soon as possible.

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