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Nutritional Assessment, Requirements, and Monitoring in Critically ill Patients

**An Essay Submitted for Partial Fulfillment of
The Master Degree in Anaesthesiology**

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Contents

I. Introduction	1
II. Nutrition requirements for different groups	3-8
III. Body fluids and electrolytes distribution and requirements	9-28
IV. Protein and fuel metabolism during starvation and illness	29-38
V. Selection of patient in need for nutritional support and timing of nutritional intervention	39-42
VI. Organization of nutritional devices	43-53
VII. Monitoring of nutritional support	54-57
VIII. English Summary	58-59
IX. References	60-66
X. Arabic Summary	--



Introduction



Introduction

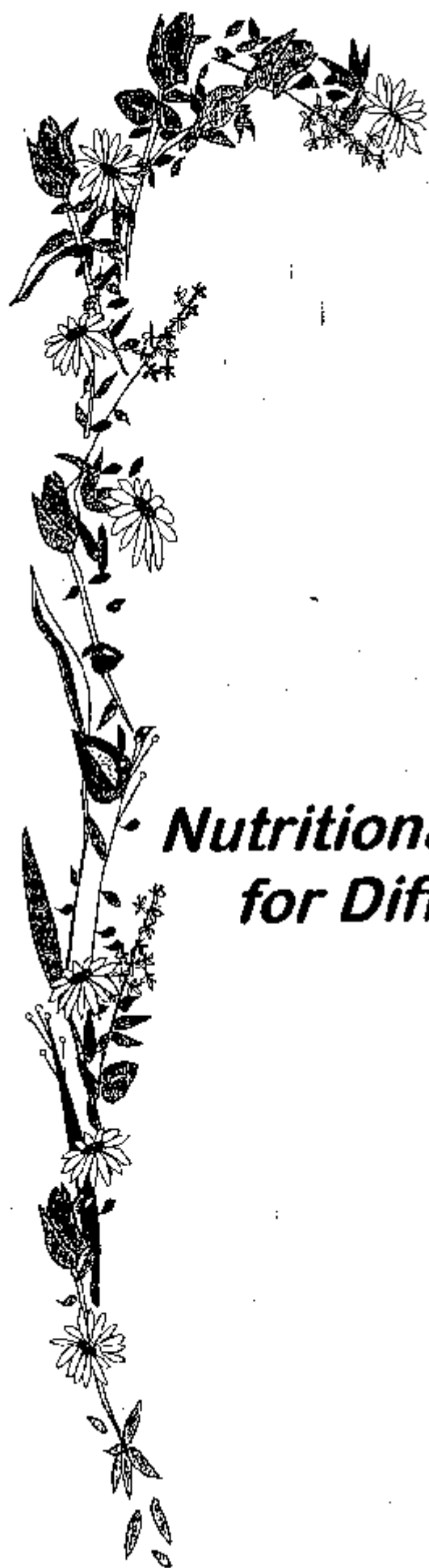
Starved, critically ill patients generally became malnourished much more rapidly than do normal fasting humans. Recognition of this phenomenon and of the relationship between nutritional status and certain host defenses has led many physicians to conclude that early nutritional intervention may favourably influence the course of a critical illness. It has generally been assumed that nutritional therapy may influence the clinical course by affecting the rate of protein loss and therefore the strength of endogenous host defense (*Robert Schlichtig et al., 1986*).

The acceleration of protein catabolism appear to have the most marked influence on survival. When the protein host defenses become depleted, the patient may become more vulnerable to the additional insults that are usually unavoidable during a prolonged ICU stay. Noxious stimuli include bacteria and fungi introduced by invasive devices, intravascular volume overload and consumption of clotting factors; these stimuli challenge the integrity of immune barrier, haemodynamic and respiratory reserves and hepatic synthetic capacity, respectively. The previous factors are of course central to nutritional monitoring and assessment. The aim of the work is to study which nutritional dependent parameter we should follow and attempt to modulate and which aspects of nutritional status are associated with the survival of critically ill patient.

This subject will be discussed under the following items :

1. Nutritional requirements for different groups.
2. Body fluids and electrolytes distribution and requirements.
3. Protein metabolism during starvation and illness.

4. Selection of patient in need for nutritional support and timing of nutritional intervention.
5. Organization of nutritional devices.
6. Monitoring of nutritional support.



Nutritional Requirements for Different Groups



Nutritional Requirements for Different Groups

Nutrition Requirements :

The primary goal of nutrition support is to meet the patient's energy requirements for metabolic processes and tissue repair. Provision of adequate nonprotein energy sources may minimize lean-tissue loss. A second major objective is to provide substrates for protein synthesis, water essential fatty acids, minerals, vitamins, and trace elements to maintain body homeostasis (*Dark and Pingleton, 1993*).

Calories :

How many calories ICU patients should be fed and how these calories should be provided are a matter of debate. Research from the 1970s indicated that critically ill patients are markedly hypermetabolic, with energy requirements 50% to 100% above their Basal metabolic Rate (*Kinney et al., 1970; and Rutten et al., 1975*). However, later studies refuted this concept (*Roulet et al., 1983; and Daly et al., 1985*). Overfeeding is associated with fatty infiltration of the liver, hyperglycemia, depressed leukocyte function, hyperosmolality, increased carbon dioxide production, and respiratory failure. Indeed, short-term restricted nutrient intake ("Permissive underfeeding") has been shown in animal models to improve immune response, delay the onset of multiple organ system failure, and improve survival following sepsis (*Zaloga, 1994*). Ideally, however, critically ill patients should receive nutrition support that is within 10% to 20% of their Basal metabolic Rate.

A second controversy is the proportion of carbohydrate to fat in the diet. Before lipid emulsions were introduced into routine clinical practice,

all nonprotein calories came from carbohydrate. However, critically ill patients metabolize only a limited amount of glucose; excess amounts of glucose lead to hyperglycemia with its attendant problems (e.g., infectious complications). The addition of insulin to the regimen may drive glucose into the cells but does nothing to promote glucose oxidation. High intracellular levels of glucose may be detrimental in areas of reduced perfusion and hypoxia (*Rodriguez et al., 1985*).

Many physicians advocate lipids as an energy source to replace glucose in parenteral and enteral nutrition formulations. However, large lipid loads may alter pulmonary and immune functions, perhaps through the mediation of arachidonic acid metabolites. Although lipids and carbohydrates are both considered appropriate nutrients for critically ill patients, large amounts of either substrate may be harmful (*Murray et al., 1995*).

Carbohydrate :

Glucose is the carbohydrate most commonly used to meet patients' caloric requirements because it is inexpensive and the most physiologic carbohydrate. The activity of pyruvate dehydrogenase, the rate-limiting step for the oxidation of pyruvate (and glucose), is depressed in many ICU patients. Excess pyruvate is released from cells in the periphery, returns to the liver, and serves as a substrate for gluconeogenesis. In patients with regional hypoxemia, the pyruvate may be a substrate for lactic acid production and may worsen ischemic insults. Thus, stressed individuals should probably receive no more than twice the amount of glucose that can be completely oxidized (*Linshceer and Vergoresen 1994*).

For ICU patients, the total glucose load should be limited to approximately $5 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$. Even less glucose, approximately $3.5 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$, should be given to patient with more severe stress, as determined by protein balance studies, the catabolic index, the Basal metabolic Rate, and

other indicators. Fructose and glycerol can be used as caloric sources, but each has its limitations, and the clinician should be familiar with these biochemicals before adding them to a patient's nutrition regimen (*Murray et al., 1995*).

Lipids :

Because critically ill patients can metabolize only a limited amount of glucose, lipids can be used as an alternative energy source. The use of lipids does have limitations, however. Traditionally, lipids have been incorporated into patients' diets to avoid the sequelae of essential fatty acid deficiency. Lipid emulsions are osmotically inert, so they may be administered through peripheral veins. Over the past decade, increasing amounts of lipids have been used as a caloric source because of the ease of administration; the lack of toxicity, if given properly; and the effect on carbon dioxide production (decreased), which may be beneficial for patients with respiratory failure who cannot handle large carbon dioxide loads (*Linscheer and Vergoresen 1994*).

Intravenously administered dietary lipids are expensive, and their use is associated with risks. Whereas healthy volunteers may be able to metabolize lipids at up to 4 g./kg/day, a critically ill patient's ability to oxidize fat is limited to between 1 and 1.5 g./kg/day. The cells' inability to metabolize exogenous lipid leads to elevated plasma triglyceride and free fatty acid levels and hepatic deposition of these fats. Elevated fatty acid levels may directly alter immune function by inactivating membrane receptors on the surface of antigen-sensitive leukocytes or, more likely, by increasing the concentration and availability of arachidonic acid, the precursor for prostaglandins, thromboxanes, and leukotrienes, important immune modulators. The current recommendation is to limit lipids to 1-1.5 g./kg/day.

Linoleic Acid :

Dietary lipids are triglycerides, which are composed of a glycerol molecule linked to three fatty acids. The only dietary fatty acid that is considered essential (i.e., must be provided in the diet) is linoleic acid, a long chain, polyunsaturated fatty acid with 18 carbon atoms. A deficient intake of this essential fatty acid produces a clinical disorder characterized by a scaly dermatopathy, cardiac dysfunction, and increased susceptibility to infections (*Linscheer, and Vergoresen 1994*). This disorder is prevented by providing 0.5% of the dietary fatty acids as linoleic acid. Sunflower oil is used as the source of linoleic acid in most nutritional support regimens.

Protein :

All nutrition regimens should contain enough protein to supply necessary amino acids for endogenous protein synthesis, but the amount and type of protein that should be given are controversial. The daily protein requirement for a healthy adult is 0.5 to 0.6 g./kg/day. For ICU patients, this requirement is probably 1 to 1.4 g./kg/day, although some authorities advocate regimens containing more than 2 g./kg/day. This latter regimen is more expensive and carries increased risk because it may increase blood urea and ammonia levels, and may unnecessarily stimulate respiratory drive.

The current recommendation is to start protein administration with 1 to 1.4 g./kg/day; the weight is based on the patient's hospital admission weight. If the patient is obese (> 150% of ideal body weight), the protein requirement is based on the patient's ideal body weight. The goal is not necessarily positive nitrogen balance, but is at least nitrogen parity (zero protein balance in which the amounts in and out are equal).

Nitrogen Balance :

Two-thirds of the nitrogen derived from protein breakdown is excreted in the urine (*Crim and Munro, 1994*). Because protein is 16% nitrogen, each gram of urinary nitrogen (UN) represents 6.25 g of degraded protein. The total-body nitrogen (N) balance can therefore be determined as follows :

$$\text{N Balance (g)} = \text{Protein intake (g)/6.25} - (\text{UUN} + 4)$$

Where UUN is the urinary urea nitrogen excretion (in grams) in 24 hours, and the factor 4 represents the daily nitrogen loss (in grams) other than UUN. If the UUN is greater than 30 (g/24 hours), a factor of 6 is more appropriate for the daily nitrogen losses other than UUN. The goal of the nitrogen balance is to maintain a positive balance of 4 to 6 grams.

Total Versus Urea Nitrogen :

Under normal circumstances, approximately 85% of the nitrogen in the urine is contained in urea and the remainder is contained in ammonia and creatinine. However, in certain groups of patients in the ICU (e.g., postoperative patients), urea may contain less than 50% of the total nitrogen in the urine (*Konstantinides et al., 1991*). Therefore, the UUN can underestimate urinary nitrogen losses patients in the ICU. Measuring the urinary ammonia excretion in addition to the UUN will provide a more accurate assessment of the TUN in these patients (*Burge et al., 1993*).

Nitrogen Balance and Caloric Intake :

The first step in achieving a positive nitrogen balance is to provide enough nonprotein calories to spare proteins from being degraded. When the daily protein intake is constant, the nitrogen balance becomes positive only when the intake of nonprotein calories is sufficient to meet the daily energy needs (i.e., the Basal energy expenditure) nonprotein calorie intake is insufficient, some of the protein provided in the diet will be broken down

to provide calories, which will produce a negative nitrogen balance. Therefore, when the daily intake of nonprotein calories is insufficient, increasing the protein intake becomes an inefficient method of achieving a positive nitrogen balance (*Marino, 1997*).

The catabolic index is a measure of stress in which total urea excretion is divided into that amount arising from exogenous protein administration and that arising from gluconeogenesis (glucose alanine cycle). The catabolic index (CI) is equal to the difference between the measured and predicted urine urea nitrogen (*Bistrian, 1979*).

$$\text{Catabolic Index} = 24\text{-hour urine } N_2 - (0.5 \times N_2 \text{ Intake} + 3g)$$

A CI of less than zero indicates minimal stress, 0 to 5 indicates moderate stress, and greater than 5 indicates severe stress.

Some authorities believe that nitrogen balance studies should be performed at baseline and after 3 to 7 days of nutrition support to document efficacy of the nutrition support. The ideal composition of the alimented protein is also unknown. Whereas there is agreement that essential and nonessential amino acids should be supplied, the ratios of individual amino acids are not established. Because branched chain amino acids appear to be preferentially metabolized in stress states, probably 50% of the amino acids supplied to moderately or severely stressed patients should be the branched chain type. Evidence is still insufficient to make this a definitive recommendation, however (*Murray et al., 1995*).