Principles and Indications of Hypocaloric Parenteral Nutrition

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Postoperative metabolism is characterized by increased energy demand and nitrogen loss. Whereas autoregulatory mechanisms are able to meet the energy needs by enhanced lipolysis without endogenous problems, nitrogen loss mainly affects functionally and metabolically important proteins. In order to prevent these protein losses during the acute postoperative period, hypocaloric parenteral nutrition is suggested. The concept of hypocaloric nutrition consists of adequate amino acid substitution of 1.2–1.5 g/kg body weight per day and a low-dosed carbohydrate supply not to exceed 150–200 g per day. According to our own experience, this carbohydrate supply is best done with glucose substitutes like polyoles, since these substrates do no greater damage to the endogenous autoregulatory mechanism and give additional support to hepatic gluconeogenesis so that amino acids can be spared. Analyzing a 6-year period of using both hypocaloric and caloric-adequate postoperative parenteral nutrition we believe that hypocaloric nutrition is as beneficial as higher caloric parenteral nutrition, which in its application and monitoring is more expensive and time-consuming.

The term hypocaloric parenteral nutrition is generally applied to mean a system of parenteral nutrition that is specifically adapted to the requirements of postoperative metabolism. As the adjective, hypocaloric, clearly indicates, an adequate caloric supply through parenteral nutrition is not the objective of this procedure; on the contrary, the dosage of carbohydrates is consciously kept to a minimum. A practical advantage resulting from this process is that it can be applied via a peripheral vein, thus avoiding the danger of central venous catheterization [1–3]. At the same time, however, one condition for effective application of hypocaloric parenteral nutrition is that the organism be able to meet the energy demands itself by mobilizing endogenous reserves. Severe malnutrition or the necessity for long-term parenteral nutrition consequently does not provide a foundation for the application of this system. Thus, hypocaloric parenteral nutrition is restricted to the early postoperative or posttraumatic phase [4, 5]. During this phase the following special characteristics of metabolism form the logical basis for the application and composition of such a regimen which, in fact, represents a specific substrate substitution.

Postoperative Energy Metabolism

Triggered by trauma or surgery, a far-reaching hormonal switchover of the organism occurs whose maximal phase of effectiveness encompasses 2–5 days [6]. The dominant mechanism in this phase is the forced release of free fatty acids during increased lipolysis by the secretion of catecholamines [7], combined with the simultaneous antagonization of the insulin effect, which inhibits this process, by increased glucagon secretion [8]. As a result of these hormonal changes, free fatty acids are utilized almost exclusively as an energy substrate in the peripheral and most visceral organ systems. In this way the pool of freely available glucose is made available or replenished for those organ systems that necessarily need glucose, independent of insulin, as their energy substrate. These are the brain, the erythrocytes, the reticuloendothelial system and, to a certain extent, the kidneys [9, 10]. All other organs, especially the liver, are switched over to the utilization of free fatty acids as their energy source. Recent investigations have confirmed that the predominant use of free fatty acids in the liver after resection, for example, is detectable for a
period of 4–5 days, and that only after that period does an increasing utilization of glucose set in again [11, 12]. This temporal sequence also corresponds to changes that have been observed in the musculature and other peripheral organs [13].

Assuming that in a normally nourished patient with about 15 kg fat tissue approximately 150,000 kcal energy are stored, it becomes clear that the endogenous energy supply to the organism during this acute phase neither poses a problem nor is metabolically detrimental. The prerequisite for the clinical application, however, is that this energy release from the fat tissue not be inhibited—or even greatly reduced—by counter-regulatory effects which can be initiated by massive insulin stimulation or exogenous substitution. The basic reasoning for a limited carbohydrate supply is derived from this concept.

Postoperative Protein and Glucose Metabolism

Characteristic of the postoperative protein and glucose metabolic situation is the increased substrate demand with nonexistent protein or insufficiently available glucose reserves. The amount of glycogen that is normally available (250–300 g or 1,000–1,200 kcal) is only enough to meet the requirements of half a day [9], and all available protein, in muscle tissue as well, must fulfill important functional tasks [14]. The basic mediators for glycogen mobilization and protein breakdown are the increases in glucagon and glucocorticoid secretions, which additionally antagonize the insulin effect [8]. If the glycogen stores are temporarily exhausted due to an increased glucose demand or turnover of approximately 200–400 g/day in the postoperative phase [15], gluconeogenesis from amino acids along with conversion of lactate, pyruvate, and glycine become the main mechanism for the provision of the glucose supply [16]. This endogenous provision of glucose is performed almost solely by the liver. Assuming that about 150–180 g glucose must be made available from amino acids, this amounts to a daily protein loss of approximately 300–350 g [17]. Added to these endogenous losses are additional losses in tissue and blood and from secretions. This potentially results in a total daily deficit of approximately 400 g. Thus, it becomes clear that the protein balance, finally, represents the weakest link in the chain of autoregulatory mechanisms following trauma or surgery and, as such, must be given therapeutic priority.

According to Waterlow [18], the mechanism of these acute protein losses is based primarily on an increased breakdown of visceral, short-lived functional proteins accompanied by the maintenance of synthesis or resynthesis (Fig. 1). The sufficient and adequate substitution of amino acids becomes extremely significant under these conditions. The principal objective of a concomitant infusion of carbohydrates must be to reduce additional endogenous protein losses or at least to compensate for them as best as possible. Only in this way can a beneficial protein-sparing effect be expected. To put these ideas into clinical practice the following points must be determined: the dosage of the carbohydrate supply, the choice of appropriate carbohydrate source, and the dosage of amino acid substitution.

Dosage of the Carbohydrate Supply

Gamble’s classic investigations [19] have shown that in a fasting state even a slight increase in glucose (100 g) suffices to achieve an effective (roughly 50%) reduction of protein losses. McDougal [20] confirmed these findings for the postoperative and posttraumatic phase, although in this study the reduction of nitrogen loss was only about 25%. In a comparison with exclusive amino acid infusion [21], Elwyn [22] reported that supplementary administration of 150 g carbohydrate significantly improved postoperative nitrogen bal-