PARENTERAL NUTRITION

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BY

Dr. FAHMY SAAD FAHMY TADROS
M.B.B.CH AIN SHAMS UNIVERSITY

SUPERVISED BY

Dr. RAFIK RAMSIS MORCOS
F.R.C.S.
ASSISTANT PROFESSOR OF GENERAL SURGERY
AIN SHAMS UNIVERSITY

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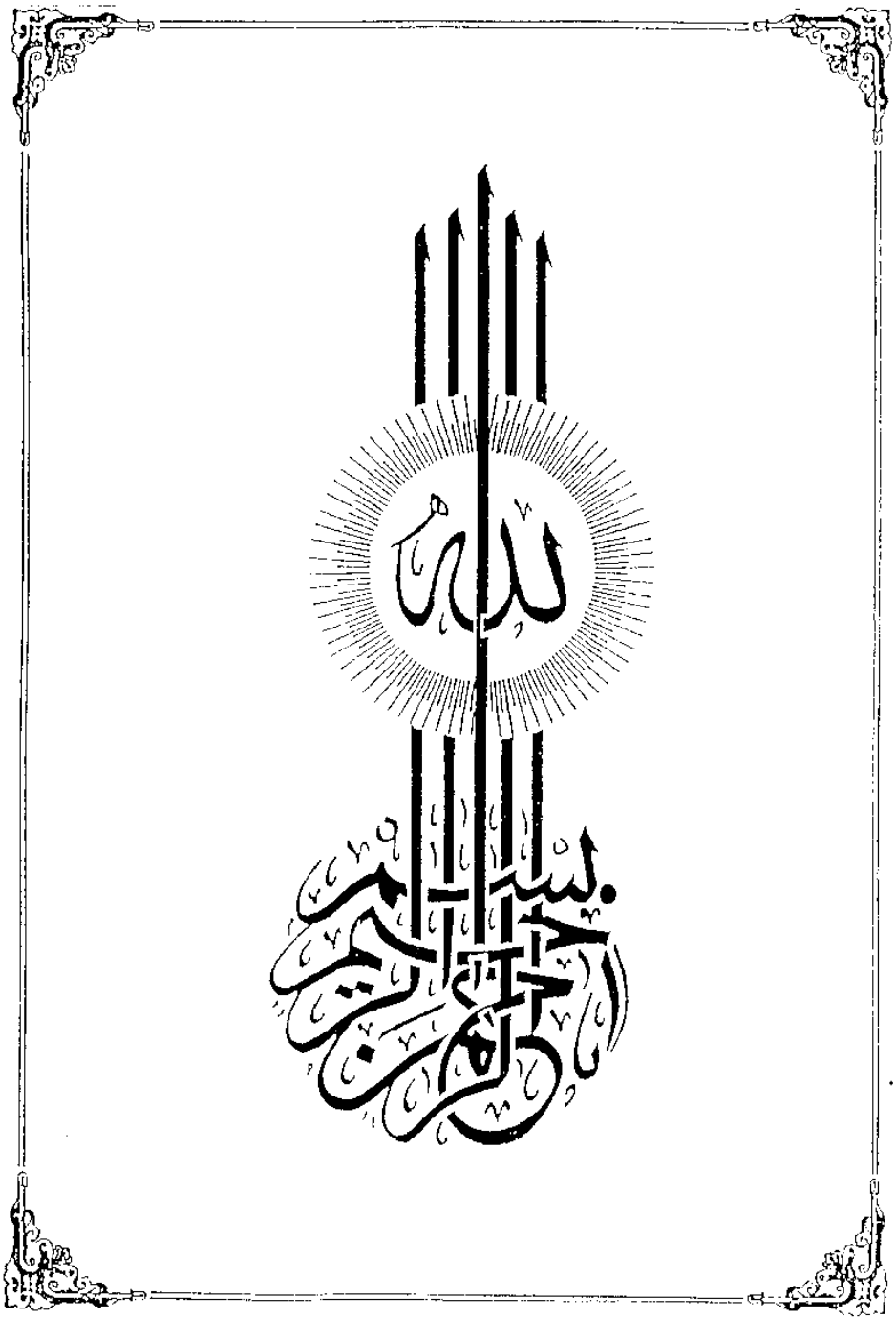

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INTRODUCTION

PARENTERAL NUTRITION

Parenteral nutrition is the administration of nutrients directly into the venous system. It aims at acheiving nutritional homeostasis in patients whose metabolic demands can not be met by enteral supplementation alone and patients whose poor mental status does not allow for oral-enteric tube feeding *(Fong and lowry, 1988)*.

Parenteral nutrition may be considered to combat post-operative or post-traumatic negative nitrogen balance where normal feeding will be delayed for more than a few days, or pre-operatively in an emaciated patient *(Johnston, 1973)*.

Fat emulsions and ethanol provide essential calories and are necessary for correct utilization of amino-acid solutions *(Hadfiled, 1973)*.

The goals of nutritional management, therefore are: *(Benson and Fischer, 1990)*.

1. To identify patients at nutritional risk early in their course, so that further nutritional deterioration can be prevented.
2. To maintain lean body mass and provide the necessary energy and nutrients to sustain physiologic systems.
3. To select the nutritional formulations and methods of administration appropriate to the patient's condition or disease.
4. Continual reassessment of the patient, including adequacy of the patient's response to that regimen, and the changing

requirements as the patient goes through the phases of hypermetabolism, stabilization or recovery, and finally repletion.

A principle regarding the institution of nutritional support that has needed tempering is, "If you think of it, you probably should do it." It is easier to prevent the complications of malnutrition than to treat them once they have occured *(Benson and Fischer, 1990).*

METABOLIC RESPONSE TO TRAUMA, SEPSIS AND STARVATION

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From a nutritional point of view, surgical patients may be divided, arbitrarily into four groups: *(Elwyn, 1980)*.

1. **Depleted.**
2. **Hypermetabolic.**
3. **Hypermetabolic and depleted.**
4. **Chronic malabsorption.**

Each group is characterized by either or both of (a)an abnormal metabolic response to nutritional therapy, and (b)specific goals of nutritional therapy. The major goal in the depleted patient is restoration of lean body mass (LBM), or body cell mass (BCM) with or without concomitant restoration of fat. The depleted patient also demonstrates abnormal responses to changes in protein intake *(Elwyn, 1980)*.

It is to be noted that the metabolic response to starvation and injury are quite different *(Silk, 1983)*.

METABOLIC RESPONSE TO INJURY (TRAUMA, STRESS AND SEPSIS)

In the traumatized patient, the systemic metabolic and circulatory responses are directed to the support of the healing wound. These responses are proportionate to the severity of injury and when the trauma is severe, a state develops from which recovery may or may not occur; this is called **shock** *(Ryan, 1976)*.

If the patient survives, there ensue metabolic changes that terminate in complete recovery. This is called the period of **convalescence** *(Ryan, 1976)*.

Therefore the metabolic adaptations for energy production during trauma and sepsis pass through 2 phases, the **Ebb phase** and the **High flow phase** *(Ryan, 1976)*. The main characteristics of each phase are summarized in table (1):

	Ebb (or) early phase	Convalescence, High flow or flow phase
Definition	It is the phase of depressed energy metabolism, to conserve body resources for survival.	A period of resurgence of energy metabolism which had some of the features of increased metabolism seen in inflammation. This has two components: **1- Catabolic phase:** characterized by excessive protein breakdown and negative nitrogen balance. **2- Anabolic phase:** during which the body stores are replenished.
Duration	6 - 18 hours No need for nutritional support during this period.	5-90 days depending on type and severity of injury. It is the period where nutritional support is concerned.
Significance	Maintenance of blood volume and allow escape to safety.	.Maintenance of energy production .Replacement of tissue lost in previous period.
Metabolic rate	Decreased.	Increased.
Body temperature	Decreased.	Increased.
Neuro endocrine response:		
1-Catecholamine output.	Increased. Maximum shortly after injury and return to normal within 24 hours	Normal or slightly increased.
2-Blood insulin level	Decreased.	Increased (but the tissues are resistant to it).
3-Blood glucose	Increased.	Normal or slightly increased (due to unopposed action of cortisol).
4-Blood lactate	Increased.	Normal or slightly increased.
5-Blood fatty acids.	Increased. i.e. the ebb phase is associated with increased sympathetic activity and an outpouring of counter-regulatory hormones.	Decreased. (due to insulin leading to inhibition of lipolysis)

Table (1) Phases of the metabolic response to injury *(Ryan, 1976; Silk, 1983).*

The following figure is an overview of the important hormonal regulatory factors:

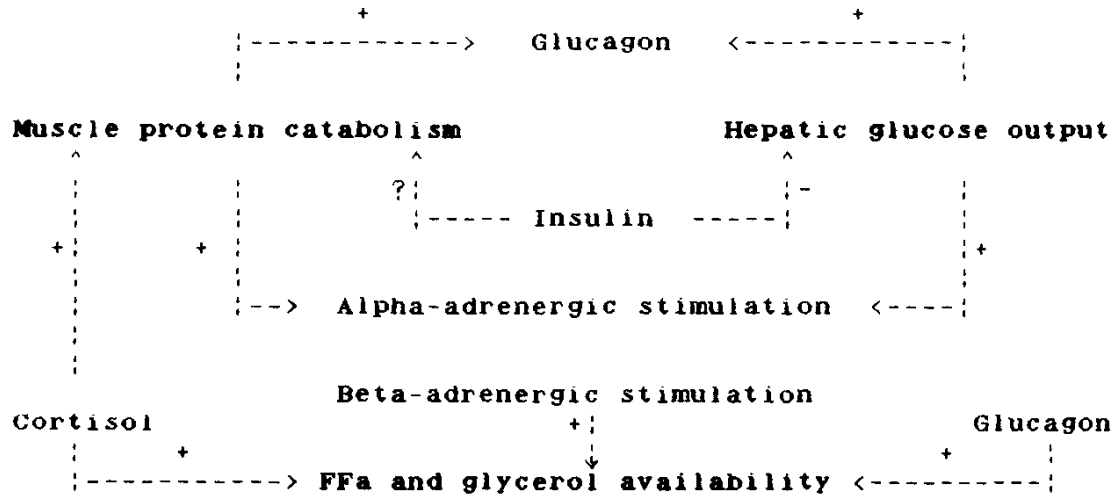

Figure (1). An overveiw of the important hormonal regulatory factors in the regulation of metabolism in septic and injured patients *(Douglas, and Shaw, 1989).*

Notes on neuro-endocrine response:

A- Catecholamines

It should be noted that there is no correlation between the injury severity score (ISS) and the degree of metabolic abnormality seen in patients after blunt trauma. The metabolic response in these patients appears to be an "all or none"response- the patient with ISS of 15 is metabolically similar to the patient with a score of 50 *(Shaw and Wolfe, 1989).*

B- Plasma cortisol

The relationship between injury severity and plasma cortisol is not direct. Cortisol levels peak at an ISS 12 and then become lower with more severe injury *(Barton and Passingham, 1981).*

The correlation between hormonal changes and metabolic abnormalities is difficult to show, for e.g. we have no

correlation between the plasma cortisol level and the degree of alteration in either glucose or protein kinetics *(Shaw and Wolfe, 1989)*.

C- Transient increase in the plasma concentration of growth hormone, prolactin, ADH, aldosterone and glucagon *(Stoner, 1986)*.

The metabolic response to injury is affected as follows:

A) ENERGY METABOLISM:

Consideration of energy metabolism in injured patients is often complicated by failure to clarify the terms by which it is defined. Therefore the following terms should be known:

1- Resting metabolic rate [Resting energy expenditure (REE)]:

It represents the sum of basal energy expenditure together with the energy expended on maintenance of body functions in the resting state. At room temperature, for eg. the body expands energy to maintain the temperature difference between itself and its environment *(Elwyn et al., 1980)*.

2- Oxygen consumption:

It is measured by indirect calorimetry and reflects total amount of oxygen consumed by chemical reactions in the body. Since energy is produced by oxidation of substrate, oxygen consumption is a good approximation of energy expenditure

3- Heat production:

It differs from oxygen consumption, since the amount of heat produced per unit of oxygen consumed depends on the substrate being oxidised.

Some showed that body temperature falls after severe injury.

There is some evidence for a reduction in oxygen consumption at the same time. However not all patients had a low oxygen consumption and it did not appear to correlate with severity of injury *(Little et al., 1981)*.

This low oxygen consumption might be a reflection of reduced tissue perfusion. After fluid replacement and restoration of a satisfactory level of delivery of oxygen to the tissues, an increase in O2 consumption is observed *(Little et al., 1981)*.

There is now little evidence to suggest that heat production is reduced during the ebb phase *(Little et al., 1981)*. It is likely that the increase in metabolic rate during the flow phase is not as large as previously thought *(Douglas and Shaw, 1989)*.

None the less a modest increase in REE in the flow phase is commonly observed and is related to the severity of the injury. The maximal increase in the REE coincides with the maximal rate of protein catabolism and occurs around one week after injury *(Douglas and Shaw, 1989)*.

The most severe injuries in metabolic terms appear to be large-area, full-thickness burns which can produce increase in REE up to 100% above predicted values *(Long, 1977)*. Such increase observed in burn patients could be due to:

1. Necessity to generate energy to maintain body temperature in the light of evaporative water losses from the burned area *(Stoner, 1986)*.

2. An alteration in the control of body temperature by the hypothalamus *(Little, 1985)*.

3. It is known that in trauma patients there is an increased rate of substrate recycling in which triglyceride is hydrolysed and re-esterified, and glucose and its glycolytic intermediates are recycled. As there is no net production of free fatty acids or glucose during these cycles, but ATP is hydrolysed, such recycling represents an energy drain. Recently it is concluded that it is possible that these cycles provide the principal biochemical explanation for the increased heat production seen in trauma patients *(Wolfe, et al., 1987)*.

Cycling of glucose was not affected by adrenergic blockade, confirming the suggestion that glucagon is the main stimulator of glucose turnover after injury *(Jahoor et al., 1986)*.

B) GLUCOSE METABOLISM:

The post-traumatic ebb phase and the earliest stage of sepsis are both characterized by hyperglycaemia *(Wolfe et al., 1977)*. Elevation of glucose level in the plasma could occur as a result of either increased release or decreased removal of glucose into the circulation.

1. Glucose release into the plasma:

Glucose entering plasma is derived from two sources:

a. Glycogen stored in liver.

b. Synthesized from precursor molecule.

The glucose entering the plasma in fasting humans will initially come from stored glycogen, and there is evidence that the initial hyperglycemia of injury is the result of depletion of glycogen stores.

Glycogenolysis is mediated by adrenaline release together

with a transient inhibition of insulin are the initial causes of elevated blood glucose *(Stoner et al., 1979)*. Plasma adrenaline increases within a few minutes of injury, and the extent of the rise observed is related to severity of injury *(Davies et al., 1984)*. Glucagon levels rise in the plasma within a few hours of injury and reach a peak between 12 hours and 24 hours *(Meguid et al., 1974)*. Both these hormones contribute to the elevation in plasma glucose and their effects are potentiated by elevation in blood cortisol. Within 24 hours, glycogen stores will be exhausted and the hyperglycaemic effects of adrenaline will lessen *(Burns, 1988)*.

When the injury receives approporiate treatment, plasma glucose falls slowly over the next few hours to normal levels *(Frayn et al., 1984)*. Effective anaesthesia can attenuate or even abolish these changes in elective surgery.

Continued stress arising from an unexised burn wound or unresolved sepsis will provide a reason for continued metabolic derangement.

However, the plasma glucose level gives little indication of glucose turnover. It is generally agreed that in septic and flow phase trauma patients, glucose turnover is increased and that gluconeogenesis is enhanced despite freely available plasma glucose *(Douglas and Shaw, 1989)*. This may be due to the increased availability of gluconeogenic substrates (due to muscle glycogenolysis and the metabolism of hypoxic tissues) occuring in a favourable hormonal milieu *(Wolfe et al., 1977; Ryan, 1976)* i.e. a combination of increased substrate availablity and an appropriate hormonal environment probably accounts for the

increased glucose production.

2. **Glucose removal from the plasma:**

In the uninjured state, gluconeogenesis is suppressed by glucose infusion. Injured patients are unable to respond normally to infusion of glucose and this is because insulin appears to be less efficient in enhancing the rate of clearance of glucose from the plasma.

After a transient fall, plasma insulin levels begin to rise soon after injury and are consistently elevated within a few days *(Stoner et al., 1979).*

The plasma insulin level at that time is usually inappropriately high for the plasma glucose concentration, and it appears that a degree of resistance to the effects of insulin excists *(Wolfe et al., 1979; Frayn et al., 1984).*

In addition, the role of cortisol in sustaining hyperglycaemia has been emphasized by *Bessey and his colleages (1984),* who found that the infusion of cortisol, in association with glucagon and adrenaline, significantly increased plasma glucose and caused apparent insulin resistance in normal volunteers.

Other workers have suggested that the major factor in production of insulin resistance is elevation in catecholamine levels. This suggests that insulin resistance is probably a reflection of the balance of hormones released following injury, with adrenaline and cortisol antagonising the effect of insulin on glucose clearance.

Another factor which reduces clearance of glucose from the plasma is its fate within the cell. Usually there is reduced