

Metabolic Stress & Starvation

Module Objectives:

- Describe benefits of nutrition intervention in critically ill patients.
- Characterize how the hormonal changes of stress affect nutrient metabolism.
- Explain the alterations in macronutrient metabolism in stress and starvation.
- Assess protein status and requirements of patients during stress and starvation.
- Describe fatty acid function and factors influencing absorption, transport, and utilization.

Module Outline:

Getting Started

Module Objectives

The Importance of Nutrition

Critically Ill Patients

Patients At Risk For Malnutrition

Ebb & Flow

Metabolic Alterations

Physiological Response to Stress

Effects of Cytokines

Ebb Phase

Flow Phase

Hypercatabolism

Metabolic Response to Stress

Normal Protein Metabolism

Protein Metabolism in Stress

Normal Carbohydrate Metabolism

Carbohydrate Metabolism in Stress

Normal Lipid Metabolism

Lipid Metabolism in Stress

Fasting & Starvation

Metabolic Response to Starvation

Carbohydrate Metabolism in Starvation

Carbohydrate Requirements

Protein Metabolism in Starvation

Lipid Metabolism in Starvation

Physiological Response to Starvation

Integrated Practice (Trauma Case)

Nitrogen Balance

Nitrogen Balance

- Laboratory Assessment
 - Serum Proteins
 - Protein Indicators
- Patient Scenarios
 - Patient Scenarios
- Protein Requirements
 - Normal Protein Requirements
 - Factors Influencing Protein Requirements
 - How Stress or Starvation Influences Protein Needs
 - Protein Needs in Critical Care
- Amino Acid Requirements
 - Essential and Non-essential Amino Acids
 - Importance of Providing Essential Amino Acids
 - Amino Acid Pool
- Lipid Assimilation
 - Fat Absorption
 - Fatty Acid Chain Length and Saturation
 - Absorption by Fatty Acid Chain Length
 - Carnitine
- Fatty Acid Requirements
 - Essential Fatty Acids
 - Essential Fatty Acid Deficiency
 - Attributes of Eicosanoids
- Integrated Practice (Nutrition Support Case)

Objectives, Key Concepts, and Key Concept Summaries by Topic

Topic: The Importance of Nutrition

Objective:

State the role of nutrition in the care of critically ill patients.

Key Concept:

It is important to initiate feeding as early as possible in critically ill patients.

The stress response is characterized by secretion of hormones and mediators which results in an increased metabolic rate. Without careful nutrition support and feeding, this hypercatabolism can lead to loss of lean body mass and damage to vital organs and functional impairment. Malnutrition in a hospitalized patient is associated with increased morbidity and mortality, and delayed wound healing. It is the physician's obligation to identify, prevent, and treat malnutrition and undernutrition.

Topic: Ebb & Flow

Objective:

Explain the metabolic effects of the stress response.

Key Concept:

Cytokines and other mediators alter metabolism during the “ebb” and “flow” phases of the metabolic stress response.

During stress, there is increased secretion of cytokines (interleukins and tumor necrosis factor), catecholamines, glucocorticoids, glucagon, insulin, and other mediators. Many of these mediators have anti-insulin activity, causing hyperglycemia and insulin resistance. At the same time, cardiac output, body temperature, and oxygen consumption increase. The common theme is increased metabolic rate. The response to trauma typically evolves in two stages. First comes the ebb phase, in direct response to the initial secretion of cytokines and stress hormones (cortisol, catecholamines, and glucagon). After a few days, in the flow phase, blood levels of cortisol and glucagon remain high while catecholamines decrease. As a result, body protein is rapidly broken down to synthesize acute-phase proteins and glucose; fat oxidation increases, and free fatty acid levels rise.

Topic: Hypercatabolism

Objective:

Explain the effects of hypercatabolism during the stress response.

Key Concept:

Stress induces a hypercatabolic state, increasing a patient's energy and protein needs.

Stress hormones stimulate muscle proteolysis and liver synthesis of acute-phase proteins. Liver glycogen is broken down; glucose released into blood causes hyperglycemia. Insulin resistance impairs glucose transport into tissues so the brain gets glucose preferentially because it does not require insulin. Muscle and organ proteins are broken down; amino acids are released into blood, transported to the liver, and used for gluconeogenesis. Stress releases lipolysis-stimulating mediators. Triglycerides in adipose are cleaved by hormone-sensitive lipase. Fatty acids are oxidized in the liver for energy or can generate ketone bodies, which enter circulation. Because there are not enough ketone bodies present to inhibit gluconeogenesis from amino acids, the end result is hyperglycemia and muscle wasting.

Topic: Fasting & Starvation

Objective:

Characterize the changes in metabolism that occur during starvation.

Key Concept:

In starvation, fat is the primary energy source; muscle and organ proteins are degraded to provide needed amino acids.

In starvation states, the body tries to preserve itself by using less energy for basic metabolic functions; thus, overall metabolic rate decreases. Metabolism shifts to use fat as a primary energy source, Fatty acids are transported to the liver and converted to ketone bodies. Ketones are used after about a week of starvation by the brain and other tissues, including muscles, where they are oxidized for energy. This spares body protein and decreases gluconeogenesis. In starvation, the high levels of ketones present inhibit gluconeogenesis. However, body tissues still must be broken down to supply amino acids for other critical functions, eventually leading to loss of lean body mass and vital organ wasting, and possibly death.

Topic: Nitrogen Balance

Objective:

Explain how a nitrogen balance study is used to assess protein status in critical care settings.

Key Concept:

Assessment of protein status can include a nitrogen balance study.

Nitrogen balance (obtained by comparing nitrogen intake in dietary proteins with nitrogen losses mainly in the urine) can be used to estimate protein requirements and protein catabolism. Positive balance is seen during growth and gain of lean body mass. Negative balance indicates inadequate protein intake or excessive catabolism. Stressed patients may not be able to achieve a positive nitrogen balance; instead the goal is to minimize losses.

Topic: Laboratory Assessment

Objective:

Specify laboratory methods used to assess protein status in critical care settings.

Key Concept:

Laboratory assessment of protein status utilizes serum protein levels.

Albumin and transthyretin levels are used for nutritional assessment and monitoring. These proteins are synthesized only in the liver, and their synthesis rate decreases with lack of amino acid precursors. Albumin reflects long-term protein status. However, serum albumin is not a sensitive nor specific indicator of nutrition status because it changes in response to non-nutritional factors such as hydration status. Transthyretin (prealbumin) responds to short-term changes in protein status.

Topic: Protein Requirements

Objective:

Describe the factors influencing protein requirements.

Key Concept:

Many factors determine dietary protein requirements.

Age, sex, and lifecycle stage determine dietary protein requirements, as this usually indicates how much muscle and lean body mass a person has, and whether they have additional protein requirements for rapid growth (such as in children or pregnant women). The Recommended Dietary Allowance (RDA) for protein in healthy adults is 0.8 g/kg/day. Factors that will affect dietary protein requirements are protein losses or the presence of metabolic stress (both of which increase protein needs), quality of the protein source (ratio of essential vs. non-essential amino acids), and adequacy of energy intake (with inadequate energy intake, some dietary protein will be burned for energy).

Topic: Amino Acid Requirements

Objective:

State the source of the body's amino acid pool and when the need for certain non-essential amino acids is increased.

Key Concept:

Both dietary and tissue protein contribute amino acids to the body's pool.

Amino acids are essential (have carbon skeletons that cannot be synthesized in the body) or non-essential (can be made in the body, although under certain conditions some of them may not be synthesized in sufficient amounts). Quality of a dietary protein depends on its proportions of essential vs. non-essential amino acids. Both dietary protein and tissue protein can contribute amino acids to the body's amino acid pool. Breakdown of an amino acid separates the carbon skeleton and the amino group. The carbon skeleton can acquire another amino group to become an amino acid again for another use. Amino acids can also go directly into muscles where they are used for protein synthesis. The carbon skeletons of all amino acids can be utilized for energy if needed. The amino groups are eventually converted into urea and excreted in the urine.

Topic: Lipid Assimilation

Objective:

Explain how fatty acids of different lengths are absorbed and the relevance for feeding.

Key Concept:

Long- and short-chain fatty acids are absorbed by different routes.

Dietary triglycerides are cleaved in the intestine by lipase and taken up by the intestinal mucosa as fatty acids and monoglycerides. Fatty acids with > 12 carbons are incorporated into chylomicrons, transported through the lymphatics via the thoracic duct into peripheral circulation. Fatty acids with less than or equal to 12 carbons are transferred directly into portal blood, transported to the liver, and taken up by hepatocytes. Short-chain fatty acids can provide dietary fat to patients who cannot efficiently absorb longer chain fats. Long-chain fatty acids are transported into the mitochondria for oxidation by an energy-consuming process that requires coenzyme A and carnitine. Short- and medium-chain fatty acids can enter the mitochondria without carnitine. Carnitine is produced by the body and eaten with meats. In some disease states, endogenous carnitine may be insufficient.

Topic: Fatty Acid Requirements

Objective:

Explain the body's requirements for the two types of essential fatty acids

Key Concept:

Essential fatty acids must be provided in the diet.

Essential fatty acids (alpha-linolenic acid and linoleic acid) are polyunsaturated fats that cannot be synthesized by the human body and must be provided in the diet to prevent deficiency. These fatty acids are the dietary precursors for the omega-3 and omega-6 series of fatty acids. The eicosanoids are 20-carbon endogenous products of omega-3 or omega-6 fatty acids that act as biochemical mediators and are important in blood clotting and immune function. Clinical signs of essential fatty acid deficiency include skin lesions, alopecia, thrombocytopenia, and poor wound healing. About 0.5% of calories should be from omega-3 fatty acids and 1 to 2% from omega-6 fatty acids to meet requirements.

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