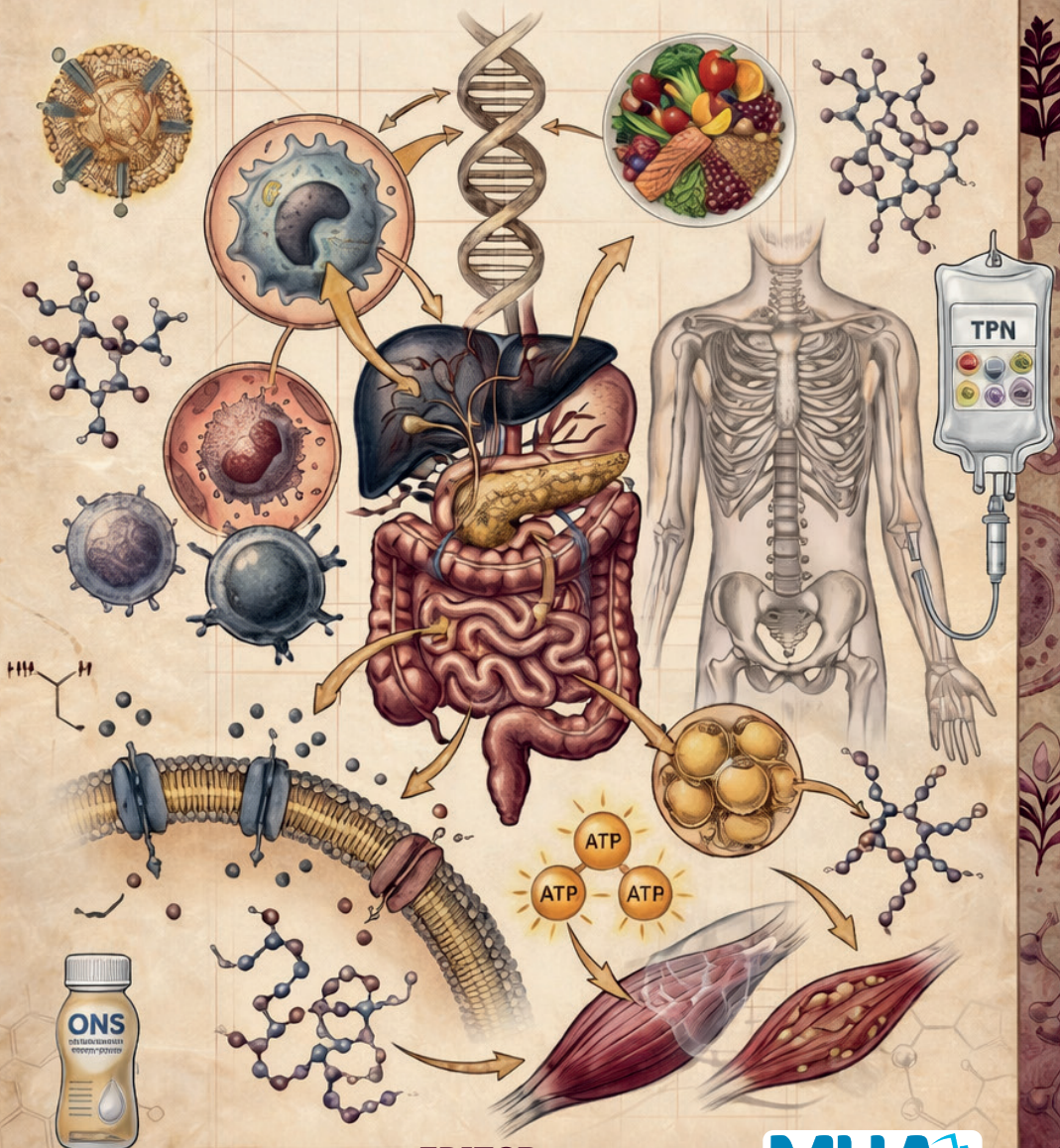
The Art of Clinical Nutrition



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Preface

Nutrition is a discipline that is often overlooked by clinicians, largely because it is not exclusively claimed by any single medical specialty. However, in almost all pathological conditions, the presence of accompanying malnutrition inevitably worsens clinical outcomes. Optimal nutritional management is essential for the proper functioning of everything from micro-level enzymatic reactions to macro-level tissues, organs, and systems. Today, even many ostensibly healthy individuals pave the way for chronic diseases by adopting a monotonous, "unhealthy" dietary pattern based solely on personal preferences, rather than the "balanced nutrition" approach the body truly requires.

This book aims to provide a comprehensive perspective on "nutritional management"-a vital branch of the medical art-for physicians, who are essentially the artists practicing it. Guided by current guidelines and recent scientific literature, this work, comprising 7 sections and 48 chapters, dives into the vast ocean of nutrition starting from the cellular level through a pathophysiological lens. As the chapters progress, it expands to the tissue, organ, and system levels, ultimately culminating in targeted nutritional approaches and malnutrition management tailored to specific clinical scenarios. We extend our deepest gratitude to all the specialists and faculty members from various disciplines who contributed to the creation of this exceptional and comprehensive work. We dedicate this work, which we hope will become an essential bedside reference for all clinicians striving to improve patient outcomes through optimized nutritional management, to the physicians who lost their lives in the line of duty while devotedly caring for patients, particularly those we lost during the recent pandemic.

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Chapter 1

Body Composition

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ABSTRACT

Body composition describes the ratio and placement of fat, lean tissue, bone, and active tissues in the human body, serving as an important marker of nutritional health and overall well-being. Precise assessment of body composition is vital for recognizing the nutritional status, metabolic health and functional ability in adults. As both clinical practice and research recognize the limitations of traditional anthropometric methods, a variety of advanced techniques have been developed to more accurately measure adipose tissue distribution, lean mass, and muscle function. This chapter examines the composition of the body's key elements; fat mass, lean mass, bone mass, and metabolically active tissues. It highlights their physiological importance and offers an overview of modern assessment methods, ranging from traditional anthropometry to advanced imaging techniques. These encompass bioelectrical impedance analysis, dual-energy X-Ray absorptiometry, magnetic resonance imaging, emerging positron emission/computed tomography technologies, detailing their principles, advantages, and clinical uses. By combining structural and functional evaluation tools, the chapter advocates a multidimensional approach to nutritional assessment, enhancing accuracy in diagnosis, monitoring, and personalized clinical decisions.

INTRODUCTION

An individual's nutritional status, as described by the World Health Organization (WHO), reflects the condition of the body, which is influenced by the equilibrium between nutrient consumption, absorption, and use, along with the impact of various physiological and pathological factors.[1] Body composition refers to the analysis of total body mass by dividing it into its components, including fat mass (FM), fat-free mass (FFM), skeletal muscle mass (SMM), total body water (TBW), and bone mineral content (BMC). Body composition assessments are routinely used to identify or diagnose various important nutritional conditions, including obesity, malnutrition, osteoporosis, sarcopenia, and sarcopenic obesity.[2] Acute or chronic diseases can alter body composition through catabolic effects, which may negatively influence disease outcomes. It is well established that chronic inflammatory activity leads to tissue loss characterized by a reduction in body cell mass.[3]

body fat varies depending on the degree of obesity, and in severe obesity, adipose tissue can become the largest compartment in the body. Fat tissue is not merely an energy reservoir; lipids also serve as essential structural elements of cell membranes, participate in steroid hormone synthesis, and play a paramount role in the functioning of the central nervous system. FFM includes the body's structural and functional components, such as skeletal muscle, bone, and visceral organ mass. The primary constituent of FFM is water (approximately 72%), while about 21% consists of protein and 7% of minerals, mainly derived from bone. Since FFM represents the majority of metabolically active tissues, it plays a pivotal role in assessing energy consumption, functional capacity, and nutritional status.[11]

Subtypes of Adipose Tissue and Their Clinical Effects

Body fat tissue is divided into two main subtypes: visceral adipose tissue (VAT), located in the ventral intra-abdominal area, and subcutaneous adipose tissue (SAT). While subcutaneous fat primarily functions as an energy reservoir and plays a role in thermoregulation, visceral fat is situated around the intra-abdominal organs (omentum, mesentery, and retroperitoneal space) and exhibits markedly higher lipolytic activity. After lipolysis, visceral adipocytes release free fatty acids directly into the portal circulation, which increases hepatic lipid load and consequently leads to insulin resistance, triglyceride overproduction, and dyslipidemia.[12]

Visceral fat also promotes chronic low-grade inflammation through mechanisms involving macrophage infiltration, hypoxia, and oxidative stress. During this process, inflammatory signaling molecules including C-reactive protein, IL-1β, IL-8, and monocyte chemoattractant protein-1 (MCP-1) are secreted, contributing to endothelial dysfunction, stiffness of the arteries and disruption of insulin signaling pathways. As a result, increased secretion of proinflammatory cytokines is strongly linked with elevated risk of cardiovascular disease, insulin resistance, hypertension, atherosclerosis, metabolic dysfunction associated with steatotic liver disease, and type 2 diabetes mellitus.[13]

Ectopic fat accumulation (in the liver, pancreas, epicardium and skeletal muscle) plays a significant role in metabolic toxicity. It contributes to the progression from hepatic steatosis to fibrosis, while myosteatosis in skeletal muscle is linked with decreased strength and physical performance.[14]

Muscle Mass, Quality, and Function

The muscular system is a core component of metabolic activity, serving as a glucose reservoir, source of amino acids, and producer of myokines. Both muscle mass and muscle quality, the latter typically assessed by radiodensity on computed tomography (CT), are closely correlated with increased risks of surgical complications, chemotherapy toxicity, infections, falls and frailty, and mortality. Because muscle function is the earliest parameter affected, the EWGSOP2 consensus identifies muscle strength as the primary criterion for diagnosing sarcopenia.[15]

Skeletal muscle, independent of FM, represents a key indicator of energy storage capacity and can be quantitatively measured. In cancer patients, weight loss is often accompanied by muscle wasting (cachexia).[16] Age-linked loss of muscle mass, resulting in reduced physical movements, defines sarcopenia, which is commonly associated with increased fat infiltration within muscle tissue.[17]

Chapter 2

Definition, Screening, and Evaluation of Malnutrition

Tayfun Börta

ABSTRACT

Malnutrition is a prevalent and multifactorial clinical condition that significantly impacts patient outcomes across all age groups and healthcare settings. It arises from an imbalance between nutrient requirements and intake, often exacerbated by disease-related inflammation, impaired absorption, and increased metabolic demands. Despite its strong association with increased morbidity, mortality, and healthcare costs, malnutrition remains underrecognized in clinical practice. Early identification through validated screening tools such as malnutrition universal screening tool (MUST), nutritional risk screening 2002 (NRS-2002), and mini nutritional assessment (MNA-SF) is essential for timely intervention. Comprehensive assessment-including clinical, anthropometric, biochemical, and dietary evaluation-plays a critical role in accurate diagnosis and management. The Global Leadership Initiative on Malnutrition (GLIM) criteria provide a standardized global framework for diagnosing and grading the severity of malnutrition. Importantly, malnutrition may occur even in individuals with normal or elevated body-mass index (BMI), emphasizing the need for a broader clinical perspective beyond body weight alone. Implementing systematic screening and individualized nutritional strategies can substantially improve clinical outcomes and reduce the burden on healthcare systems.

INTRODUCTION

Today, malnutrition is still a huge problem all over the world. It is especially a big challenge for countries with less money or developing economies, and poses an ongoing challenge for healthcare systems.[1-9] It significantly influences disease susceptibility, clinical outcomes, prognosis, and the effectiveness of therapeutic interventions.[2-15] Children, the elderly, people with long-term illnesses, and patients staying in hospitals are especially vulnerable to malnutrition[3,11,15] Despite its well-established association with increased morbidity and mortality, malnutrition frequently remains underrecognized in clinical settings, staying longer in a hospital bed and increasing the

dialysis-related losses, and severe burns can result in substantial depletion of proteins, electrolytes, and trace elements.[6]

If these losses are sustained and not adequately replaced, progressive negative nitrogen balance and micronutrient depletion occur, contributing to the development or worsening of malnutrition.

Multifactorial Interaction

In clinical practice, malnutrition rarely results from a single isolated factor. Instead, it typically reflects the cumulative effect of reduced intake, inflammation-induced catabolism, malabsorption, and ongoing nutrient losses. This multifactorial nature explains why early identification and comprehensive nutritional assessment are essential components of patient management in both acute and chronic care settings.

CLINICAL CONSEQUENCES OF MALNUTRITION

Malnutrition adversely affects nearly all organ systems and is associated with impaired immune function.[9,15]

Increased susceptibility to infections: A weakened immune system heightening the risk of secondary infections by impairing the body's natural protective barriers and immune responses like bacteria and viruses. This increased susceptibility often results in more frequent illnesses and longer recovery times for common infections. Determinants such as protracted stress, nutritional deficiencies, or pre-existing clinical pathologies can significantly impair your natural defenses. Taking proactive steps to boost your immunity is essential to protecting your long-term health and well-being.

Delayed wound healing: Delayed wound healing occurs when the body's natural repair process is slowed down by internal or external factors. This condition is often linked to poor blood circulation, which prevents essential nutrients and oxygen from reaching the injury site.

Decreased muscle strength and reduced functional capacity: Decreased muscle strength often leads to a significant decline in an individual's ability to perform everyday tasks independently. As muscles weaken, functional mobility decreases, which can increase the overall risk of falls and physical injuries.

Prolonged length of hospital stay: A prolonged length of hospital stay significantly increases the risk of hospital-acquired infections and other clinical complications. This situation often leads to a substantial rise in healthcare costs and places a heavy burden on medical resources. Various factors, such as the severity of the illness or delayed recovery progress, can keep a patient hospitalized longer than initially expected.

Increased morbidity and mortality: Increased morbidity and mortality rates represent a significant decline in overall public health and individual life expectancy. These statistics often rise due to the combined effects of chronic diseases, poor access to healthcare, and environmental risk factors.

SCREENING FOR MALNUTRITION

Nutritional screening is a rapid, structured process used to identify individuals at risk of malnutrition. Screening should be performed at hospital admission, during outpatient visits, and at regular intervals in high-risk populations.[5,14]

Characteristics of an Effective Screening Tool

- An effective screening tool should be easy to apply,
- Time-efficient,
- Reliable and valid,
- Applicable by different healthcare professionals.

Commonly Used Screening Tools

Malnutrition universal screening tool (MUST): A broad consensus supports the use of MUST for the rapid identification of malnutritional status, facilitating consistent screening across the continuum of care-from hospital admission to long-term residency.[6]

- **Based on BMI:** BMI is one of the most commonly used anthropometric indicators for evaluating nutritional status in adults in both clinical practice and epidemiological studies. Individuals are classified into categories such as underweight, normal weight, overweight, and obesity according to BMI thresholds defined by organizations like the World Health Organization. BMI is calculated by dividing body weight in kilograms by the square of height in meters, and it provides a simple screening tool for identifying potential malnutrition or obesity-related health risks.
- **Percentage of unintentional weight loss:** Unintentional weight loss is a critical clinical indicator used to assess nutritional status and identify patients at risk of malnutrition. In clinical practice, the percentage of weight loss over a defined time period (e.g., 1, 3, or 6 months) is commonly used to evaluate the severity of nutritional depletion. Higher percentages of unintended weight loss are associated with increased morbidity, mortality, and poorer clinical outcomes, particularly in hospitalized or chronically ill patients.
- **Presence of acute disease with little or no nutritional intake for five days or more (≥5 days):** The presence of an acute disease or injury can significantly increase metabolic demands while simultaneously reducing nutritional intake, thereby predisposing patients to rapid nutritional deterioration. In hospitalized or critically ill individuals, inadequate or absent nutritional intake for five days or longer is considered a significant risk factor for the development of malnutrition. Clinical guidelines from the European Society for Clinical Nutrition and Metabolism emphasize that patients experiencing acute illness with minimal or no food intake for ≥5 days should be promptly assessed and considered for early nutritional support to prevent further metabolic complications.

Nutritional risk screening 2002 (NRS-2002): This methodology aligns with ESPEN guidelines, representing the premier screening approach for assessing nutritional vulnerability in the adult inpatient setting. Unlike MUST, it evaluates both nutritional status and the metabolic impact of disease severity. It is a recommended screening tool, especially for adult patients, in hospitals.[5] The parameters evaluated are:

- It assesses both nutritional impairment
- Disease severity
- Risk assessment parameters for individuals in the 70+ age bracket

- **Phenotypic:** Weight loss, low BMI, muscle loss
- **Etiological:** Inflammation or low intake

Pediatric malnutrition screening tools: Pediatric nutritional screening instruments are specifically engineered for the early detection of children predisposed to malnutrition. By employing systematic and rapid evaluation protocol, these tools facilitate risk identification across both inpatient and ambulatory care settings. Several validated instruments, including the screening tool for the assessment of malnutrition in pediatrics (STAMP), the pediatric Yorkhill malnutrition score (PYMS), and the screening tool for risk on nutritional status and growth (STRONGkids), are commonly used to evaluate nutritional risk based on clinical history, dietary intake, disease severity, and anthropometric measurements. These tools facilitate early detection of nutritional risk and enable timely nutritional intervention, which is essential for supporting growth, immune function, and overall clinical outcomes in pediatric patients, as recommended by the European Society for Clinical Nutrition and Metabolism.

Tools specifically developed for children:

- STRONGkids
- PYMS (Pediatric Yorkhill Malnutrition Score)
- STAMP (Screening Tool for the Assessment of Malnutrition in Pediatrics).[11]

COMPREHENSIVE EVALUATION OF MALNUTRITION

Following a positive malnutrition screening, a detailed assessment is warranted to validate the nutritional status. This clinical protocol encompasses four fundamental pillars: clinical history, body composition metrics (anthropometry), laboratory data (biochemistry), and nutritional intake assessment.[13,14]

Clinical Evaluation

A comprehensive history and physical examination are performed:

- Changes in appetite, nausea and vomiting
- Changes in bowel movements (diarrhea, constipation)
- Difficulty swallowing and chewing
- Medical history, comorbidities
- Metabolic stress states
- Amount and duration of weight loss.[2,8,14]

Physical examination:

- Muscle wasting (deltoid, interosseous muscles, temporalis)
- Fat loss
- Presence of edema
- Dry skin, nail and hair changes
- Pressure ulcers or delayed wound healing

WATER

A water molecule (H_2O) is created through the covalent bonding of one oxygen atom and two hydrogen atoms. Owing to its polar and dipolar nature, water displays several unique physicochemical properties that make it indispensable for sustaining life. As the principal constituent of body composition, water makes up approximately 60% of body weight in adult males and about 50-55% in adult females. This sex-related difference is primarily explained by the higher proportion of adipose tissue typically observed in women. The proportion of water varies across tissues-roughly 75% of muscle and brain tissue, around 81% of blood and kidneys, 71% of liver tissue, close to 22% of bone mass, and about 20% of adipose tissue.[25]

Water is vital for metabolic reactions, molecular transport across membranes, maintenance of intracellular and extracellular homeostasis, thermoregulation, and circulatory function. Because endogenous water production is insufficient to meet physiological demands, water is regarded as an essential nutrient. All biochemical processes occur in aqueous environments. Although scientific committees and professional organizations have proposed recommended daily water intakes for different age and sex groups, there remains no universal agreement on the exact requirements for the general population. Hydration requirements differ substantially among individuals and are influenced by multiple variables, including physical activity intensity, body mass index, environmental temperature, dietary patterns, renal function, underlying acute or chronic health conditions, and pharmacological treatments.[25]

According to guidance issued by the European Food Safety Authority (EFSA), individuals living in temperate climates and engaging in moderate physical activity should aim for an average daily water intake of 2.5 liters for men and 2.0 liters for women, with additional allowances of 300 ml during pregnancy and 700 ml throughout lactation.[24]

Similarly, the National Academy of Medicine (NAM) in the United States suggests total daily water consumption levels of 3.7 liters for men and 2.7 liters for women, inclusive of fluids derived from both dietary sources and beverages. NAM recommends 3.0 liters daily during pregnancy and 3.8 liters for lactating women.[24]

Contemporary evidence suggests that hydration assessment should incorporate not only total water intake but also biochemical indicators such as plasma osmolality and antidiuretic hormone (ADH) concentrations.[26]

CONCLUSION

The adequate and balanced intake of nutrients in amounts sufficient to meet daily requirements is essential for maintaining a healthy life. Regardless of their specialty, it is important for all physicians to provide patients with appropriate guidance on nutrition. Planning and disseminating information by health authorities regarding the dietary sources of the macronutrients and micronutrients mentioned in the text, as well as their recommended daily intake levels, would help address existing knowledge gaps in the community in this field.

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levels of blood chemicals such as sodium, glucose, chloride, proteins, and bicarbonate. Blood urea nitrogen (BUN) is important in calculating serum osmolality. In clinical practice, plasma osmolality can be calculated according to a formula that includes sodium, glucose, and BUN:[8,9]

Plasma osmolality = 2 × Sodium + Glucose / 18 + BUN / 2.8.

Hypothalamic osmoreceptors respond to alterations in the osmotic concentration of plasma. When plasma osmolality increases, the thirst center is activated, increasing water intake and stimulating ADH secretion. Through this mechanism, water reabsorption from the kidneys is increased, ensuring that plasma osmolality is kept within the physiological range.[10]

Antidiuretic Hormone (ADH)

ADH is mainly produced by neurons in the supraoptic nuclei of the hypothalamus and is subsequently transported to and secreted from the posterior pituitary into the bloodstream. This hormone is essential for preserving fluid homeostasis by adjusting water reabsorption, influencing sodium concentration, and contributing to the regulation of arterial blood pressure. ADH interacts with specific receptors located on the principal cells of the renal collecting ducts. Binding to these receptors triggers the intracellular cyclic adenosine monophosphate pathway, leading to the phosphorylation of aquaporin-2. This mechanism facilitates water reabsorption. As a result, urine volume decreases, and urine becomes more concentrated.[11]

The release of ADH is triggered by reduced extracellular fluid volume, elevated plasma osmolality, or changes in arterial pressure. This mechanism is an important response that limits fluid loss, especially in cases of dehydration and hypovolemia.[7,11]

Renin-Angiotensin-Aldosterone System (RAAS)

The RAAS is ubiquitous and involves many organ systems, particularly the kidneys and lungs.[12] The RAAS increases extracellular fluid volume by enhancing sodium and water reabsorption and increasing potassium excretion (**Table 2**).[13]

Table 2. RAAS and ANP: summary of antagonistic regulation

System	Trigger	Main target organs	Primary actions	Net effect
RAAS	Low blood pressure/low sodium/decreased renal perfusion	Kidney, adrenal gland, blood vessels	Renin → angiotensin II → aldosterone	Vasoconstriction; ↑ sodium and water retention
ANP	Atrial stretch due to increased blood volume/ pressure	Kidney, blood vessels, adrenal gland	↑ Natriuresis & diuresis; ↓ renin and aldosterone	Vasodilation; ↓ blood volume and pressure

RAAS: Renin-angiotensin-aldosterone system, ANP: Atrial natriuretic peptide

RAAS is a hormonal regulatory system that plays a key role in maintaining sodium balance, ECF volume, and blood pressure.[14] Activation of the RAAS begins with the secretion of renin by juxtaglomerular cells in the afferent arterioles of the kidney. Renin secretion is triggered by reduced renal perfusion pressure, lower sodium and chloride delivery, and heightened sympathetic stimulation mediated by β_1-adrenergic receptors.[7,15] Renin breaks down angiotensinogen to form angiotensin Renin enzymatically cleaves angiotensinogen to produce angiotensin I.[7] Subsequently, angiotensin I is converted into angiotensin II mainly by angiotensin-converting enzyme located on the endothelial surface of the pulmonary circulation.[16,17]

Chloride

Chloride is a negatively charged ion found predominantly in the extracellular fluid (ECF). Sodium and chloride are the main cations and anions of the ECF, together playing a fundamental role in determining plasma osmolality and extracellular volume. Due to electrophysiology, the increase in sodium in the extracellular space to maintain neutrality usually occurs together with chloride; therefore, both ions are considered biochemical indicators of volume status. Serum chloride concentration is regulated by the kidneys. Most of the chloride passing through the glomerulus is recovered in the proximal and distal tubules via both active and passive transport. Sodium and chloride reabsorption in the kidneys occurs together in most nephron segments.[38]

Calcium

Calcium is a cation found mainly outside the cell. Calcium has functions such as blood clotting, bone mineralization, muscle contraction, transmission of nerve impulses, and hormone release. Calcium uptake in the intestine is mediated by 1,25-dihydroxy vitamin D3, whereas parathyroid hormone modulates calcium handling in the renal distal tubules.[39]

Because the majority of the body's calcium attaches to albumin, measurements of calcium should be adjusted according to albumin levels before diagnosing low calcium. For each 1 g/dl drop in albumin in the blood, total serum calcium typically falls by about 0.8 mg/dl. Hypocalcemia is defined when the adjusted serum calcium falls under 8.5 mg/dl, whereas hypercalcemia is present if the adjusted serum calcium exceeds 10.2 mg/dl Symptoms of hypocalcemia may include tetany, paresthesia, depression, anxiety, and corrected QT interval (QTc) prolongation on the electrocardiogram. Symptoms of hypercalcemia may include nausea, vomiting, musculoskeletal pain, renal colic, and abdominal pain due to constipation or peptic ulceration, polyuria, and polydipsia. Depending on the severity of hypercalcemia, QT shortening, which can lead to ventricular fibrillation, confusion, and coma, may occur.[40-42]

Magnesium

Magnesium is an intracellular cation. Magnesium is essential for the stability and proper use of ATP (adenosine triphosphate) metabolism, proper muscle function, neurological function, and neurotransmitter release. Magnesium contributes to calcium reuptake in the sarcoplasmic reticulum during muscle contraction by supporting the activity of calcium-activated ATPase.[43] Hypomagnesemia is diagnosed when magnesium in the blood is less than 1.5 mg/dl. Hypermagnesemia is defined as magnesium levels greater than 2.6 mg/dl.[44]

Magnesium deficiency may lead to low calcium and potassium levels, along with heart and nervous system symptoms. Prolonged magnesium insufficiency has also been linked to disorders like high blood pressure, diabetes mellitus, heart disease, and osteoporosis. Symptoms of hypermagnesemia include confusion, respiratory depression, bradycardia, heart block, and muscle weakness.[45]

Phosphorus

Phosphorus exists as a positively charged ion in the extracellular fluid. About 85% of the body's phosphorus is stored in bones and teeth, with the remaining 15% present in soft tissues.[46] Phosphate is essential for metabolic processes and forms part of adenosine triphosphate and nucleotides. Levels of calcium and phosphate are controlled by parathyroid hormone, active vitamin D, and calcitonin, with the kidneys being the main organ responsible for eliminating phosphorus from the body.[1]

enteral diets enriched with glutamine, omega-3 fatty acids, L-arginine, or nucleotides (containing 2.2-2.5 g/kg protein) have been shown to reduce infection rates, antibiotic use, and multiple organ failure, as well as shorten hospital stay. These diets should only be used for 7-10 days; standard diets should be resumed as longer-term use may cause energy deficiency. Although the benefits of high-fiber diets in trauma patients have not been proven, they can be used as they have no harmful effects.

Following shock resuscitation, enteral feeding should be initiated at 15 ml/hour. After 8-12 hours, it should be increased to 25 ml/hour and then increased by 25 ml/hour daily as tolerated until the target value is reached. The timing and dose of enteral feeding may vary depending on various factors such as splanchnic circulation, hemodynamic stability, acidosis, hypoxia, etc. In cases such as spinal cord injury, multiple bowel injuries, severe abdominal trauma, and pelvic fractures with large retroperitoneal hematoma, it may be more difficult to increase nutrition because it reduces bowel motility.[1]

Summary

Trauma is a complex process involving inflammatory, metabolic, and cardiovascular responses. Appropriate nutritional support during the inflammatory and metabolic phases improves patient outcomes. Compared to TPN, TEN has the significant advantage of reducing the rate of sepsis in both early and late TEN. Post-pyloric nutrition is better tolerated in the first few days in trauma patients. Immune-supported enteral diets may be beneficial in severe injuries. When planning nutrition for patients with head trauma, intracranial pressure control must be considered, and overfeeding should be avoided.

THE EFFECTS OF SEPSIS ON NUTRITION

Sepsis is defined as a life-threatening organ dysfunction caused by an uncontrolled host response to infection.[5] Unlike previous classifications, the current definition focuses not only on inflammation but also on the disruption of homeostasis at many levels, including metabolism. Metabolism refers to all biochemical processes in the body, and under normal conditions, there is a balanced relationship between anabolic and catabolic reactions. Pathological conditions such as sepsis can seriously disrupt this balance. Although the relationship between metabolic disorders and sepsis has been known for a long time, most research has focused on inflammation for years. Therefore, the basic approach to treating sepsis today is still limited to antimicrobial therapy and supporting organ function. Given the need for innovative treatments in modern medicine, it is necessary to consider other components of the "dysregulated host response" besides metabolism, such as thermoregulation, hemostasis, microbiota, and circadian rhythms.[6,7] Understanding the role and impact of metabolic disorders in the pathophysiology of sepsis is critical for the effective management of patients and the identification of new treatment targets.

Main Features of the Pathogenesis of Sepsis

The primary proinflammatory cytokines in the pathogenesis of sepsis are TNF-α, IL-1, and IL-6. These mediators contribute to the increase in nitric oxide and reactive oxygen species, as well as the development of a prothrombotic response. In parallel, the compensatory activation of anti-inflammatory mediators such as interleukin-4 (IL-4) and interleukin-13 (IL-13) is also important. In the advanced stages of the disease, the pathways necessary for the production of proinflammatory mediators may be depleted,

and an immunosuppressive picture characterized by immune system dysfunction may emerge.[8-11]

Metabolic Changes in Sepsis

Metabolic dysfunction is one of the typical features of sepsis. Activation of the immune system, tachycardia, fever, and increased respiratory rate elevate energy consumption, while significant changes also occur in the endocrine and autonomic nervous systems.[12] This activation, referred to as the "neuroendocrine response," is a dynamic process that directly affects metabolism.[13] Cytokines such as TNF-α, IL-1, and IL-6 are thought to be the primary triggers of this response;[14-15] it is also known that they can directly alter metabolism.[13,16]

A pronounced hypermetabolic phase is observed at the onset of sepsis, followed by a hypometabolic phase characterized by lower energy expenditure. The hypermetabolic phase is dominated by a strong inflammatory response, accelerated catabolic processes, and suppressed anabolic pathways. This phase is thought to be an adaptive response. Increased insulin resistance and excess anterior pituitary hormones ensure that substrates for energy production are readily available.[17-19] It has been suggested that the subsequent hypometabolic phase is also an adaptation mechanism.[11,20-22] These processes cause irregularities in all aspects of protein, lipid, and carbohydrate metabolism.[13] Gluconeogenesis and glycogenolysis increase in the liver, and systemic insulin resistance results in hyperglycemia. Proteolysis increases in muscle tissue, leading to the release of amino acids into the circulation. Similarly, hepatic lipolysis also elevates plasma free fatty acid levels.[23,24] The resulting hypercatabolism not only depletes physiological reserves but also leads to the breakdown of structural or motor function proteins.[13]

Feeding Time and Method

Nutritional support can be provided via enteral or parenteral routes. Enteral nutrition is preferred in terms of preserving intestinal barrier integrity, maintaining absorption functions, and supporting the immune response, and it is more cost-effective. Early initiation of enteral nutrition may shorten the length of stay in intensive care and improve clinical outcomes. Although this benefit has been reported to be more pronounced in non-critical patients receiving long-term parenteral nutrition due to gastrointestinal dysfunction, the evidence is still inconclusive in critically ill patients at risk of multiple organ failure.

Although enteral nutrition is generally recommended, it is often difficult to meet all the energy and protein requirements of critically ill patients using this method alone. Furthermore, in severely ill patients with impaired splanchnic blood flow, enteral nutrition may increase blood flow in the proximal intestine while decreasing it in the distal regions, potentially leading to hypoxia, motility disorders, mucosal damage, reflux, and aspiration risk. To reduce aspiration risk, the patient's head can be elevated at a 45° angle. In the presence of prolonged gastrointestinal dysfunction, parenteral nutrition should be provided until enteral tolerance is achieved. The content of nutrition is as important as the route of administration; for example, studies indicate that the addition of certain substrates, such as glutamine, may be beneficial.[25]

Energy Needs and Supply

In the past, patients could be given up to 4000 kcal of energy per day because significant protein breakdown and high metabolic rates were observed during sepsis and critical illness. However, technologies that enable more accurate measurement of energy