INTRODUCTION

The fundamental goal of nutritional support is to provide individual patients with their daily nutritional requirements, so nutrients and energy needs of each patient should be determined. The important categories of nutritional elements include carbohydrates, lipids, proteins, vitamins, minerals and water ***(Guyatt et al., 2011).***

Objective assessment of nutritional status is difficult in intensive care. Anthropometric measures such as triceps skin-fold thickness and mid-arm circumference may be obscured by edema. Voluntary hand-grip strength, a test of functional capacity, is impractical in unconscious patient. Laboratory measures, including trasnsferrin, albumin, pre-albumin levels, lymphocyte counts and skin-prick test reactivity are abnormal in critical illness ***(Davis et al., 2012).***

Malnutrition, including the depletion of essential micronutrients and erosion of lean body mass, is very common in patients who are critically ill, with 20-40% of such patients showing evidence of protein energy malnutrition. Also malnutrition has been established as an independent risk factor for morbidity, leading to increased rate of infections, length of hospital stay and number of days on mechanical ventilation. As well as difficult wound healing and ultimately an increase in mortality ***(Jensen et al., 2013).***

Enteral nutrition is preferred over parentral nutrition for most patients. It is an evidence-based practice supported by numerous clinical trials upon patients with trauma, burn, head injury, major surgery, acute pancreatitis and others ***(Kudsk et al., 2014).***

The broad guidelines for optimal nutritional support include starting nutrition when benefits are exceeding risks, then providing adequate caloric goals. Next to this is choosing a method for administrating the nutrients either enteral or parentral. Then designing a formula suitable for a particular patient. Finally monitoring the patient for adequacy of nutrient intake aiming at minimizing complications and improving outcome ***(McClave et al., 2016).***

Aim of the Work

This work will discuss the principles and new guidelines for provision of nutrition, in addition to highlights for specific nutritional therapy in different forms of critical illness and finally to understand the overall outcome in critically ill patients.

CHAPTER (1)
NUTRITION AND CRITICALLY ILL PATIENT

Hypermetabolism is the trademark of critical illness, a phenomenon originally described as "ebb and flow". But now these phases became shock and post-resuscitation hyper-metabolism, respectively. The third phase is anabolic phase that follows resolution of the stress response and persists for weeks to months ***(Monk et al., 2011).***

The metabolic stress response to critical illness represent the body's physiological survival mechanism to restore homeostasis. It is multi-systemic response and characterized by Changes that affect the entire body (figure 1). The most common stimuli that trigger this compensatory response are decreases in blood pressure, plasma volume, blood pH, increased serum osmolarity, and hypoxemia. The degree of response is variable and depends on factors such as type and severity of insult and prior nutritional state of the host ***(Douglas, 2016).***

Influences of hypermetabolic state on different body systems:-

Neurologically

During stress, glucose, amino acids and lactate metabolism increases by brain. The encephalopathy of critical illness is believed to be related to the presence of elevated levels of aromatic amino acids and their metabolites. In some cases, global cerebral function is impaired, as evidenced by alterations ranging from delirium to overt coma ***(Jeevanandam et al., 2013).***

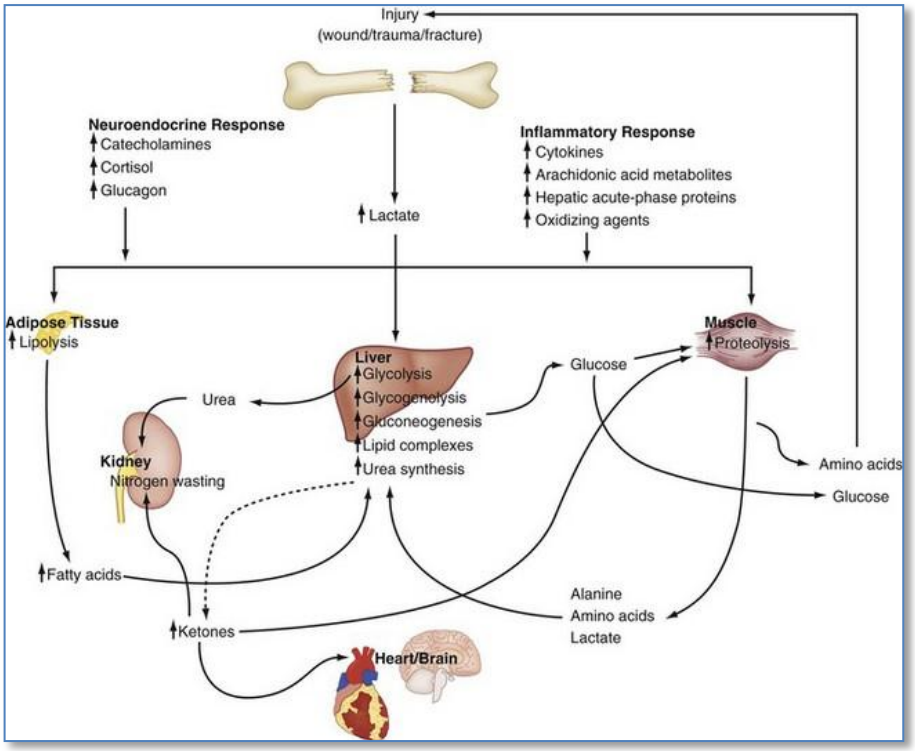

Figure (1): Metabolic stress response in critically ill patient ***(Douglas, 2016).***

Cardiovascular

Stress increases the need for oxygen and nutrients in the periphery resulting in circulatory hyperdynamic response. Cardiac output increases, and peripheral vascular tone decreases, augmenting blood flow to peripheral tissues, at the expense of loss of flow to other vascular beds. Oxygen consumption is highest in tissues with the highest levels of leukocytes (cells that repair tissue and control infection).In some patients, myocardial damage ensues. This may lead to a failure to supplement oxygen delivery that is associated with a high mortality ***(Monket al., 2011).***

Fluids, Electrolytes, Nutrition and Energy

Intravascular resuscitation and leakage from capillary beds (due to the loss of tight junctions) increase body water, and patients gain weight. The balance between fluid extravasation and reabsorption favors formation of edema because plasma proteins accumulate outside vessels walls, pulling fluid and electrolytes with them. The extracellular (increased by 15–20%) and vascular compartments expand, but intracellular water is lost ***(Boonen et al., 2014).***

Hyponatremia from water retention with vascular expansion is possible. Hypokalemia, hypomagnesemia, and hyperphosphatemia are frequent findings as the response abates because of shifts of water back into the intracellular space. Hypophosphatemia also can result from exhaustion of phosphate during hypermetabolism or as a part of the refeeding syndrome ***(Hildebrandt et al., 2016).***

Carbohydrates

Carbohydrate metabolism increases in tissues such as immune cells and decreases in many other tissues, resulting in a syndrome of glucose intolerance. The driving force for increased glucose production and decreased utilization results from the demand imposed by leukocytes in areas of injury leading to increase in whole-body glucose ***(Harsanyi et al., 2011).***

Hyperglycemia is due to release of counter-regulatory hormones, resistance in peripheral muscle to the effects of insulin despite increased insulin secretion and

mobilization of glycogen stores in the liver and skeletal muscle to meet hypermetabolic demands. These stores are quickly depleted within the first 24 hours of critical illness. Another contributor is the increased hepatic gluconeogenesis from amino acids, lactate, and glycerol from the peripheral metabolism of muscle proteins and triglycerides ***(Schulman et al., 2014).***

Proteins

Protein catabolism is accelerated and outpaces protein production. It occurs because of not only gluconeogenesis, but also thermogenesis, immune function, tissue repair and liver protein synthesis decreases and is shifted toward production of acute-phase proteins (such as C-reactive protein, fibrinogen, and haptoglobin) and enzymes involved in gluconeogenesis. Lean muscle is broken down rapidly (majority of these losses occur in skeletal muscle) to provide the necessary glucose and amino acids for immediate energy needs ***(Long et al., 2014).***

This leads to a profound nitrogen deficit (negative nitrogen balance) due to increased urinary nitrogen excretion and decreased peripheral amino acid uptake Nitrogen excretion in critically ill patients has been shown to be 9 to 10 g/day in patients undergoing elective surgery or minor stress to more than 20 g/day in patients with large burn wounds or major trauma ***(Hasselgren et al., 2011).***

The main difference between carbohydrate and protein metabolic changes is that providing enough protein

can limit lean muscle catabolism, but administering exogenous carbohydrate does not suppress the gluconeogenesis (refractory toexogenous nutritional interventions) as it does in healthy patients, and it may further exacerbate hyperglycemia**.** This is the main reason why providing adequate protein via nutritional support is more important than calories ***(Dhaliwal et al., 2014).***

Lipids

Lipid metabolism increases in the stress response. Glucagon, epinephrine and synergistic effect of cortisol increase circulating free fatty acids from lipolysis. The contribution of fat oxidation to energy production is increased. The fatty acids liberated by lipolysis are oxidized as a primary source of adenosine triphosphate during stress ***(White et al., 2012).***

Re-esterification of triglycerides in the liver is increased due to abundance of free fatty acids and glycerol. This increases the hepatic concentration of very low density lipoproteins (VLDL) and can cause hepatic steatosis. Surprisingly, the plasma concentration of cholesterol is low despite the increase in circulating free fatty acids and triglycerides, possibly because of the hypermetabolic state. This increase in lipolysis is not suppressed by hypercaloric carbohydrate administration ***(Kondrup et al., 2013).***

Energy

Critical illness and severe stress can cause a significant increase in basal metabolic rate and overall

energy requirements. Elective surgeries cause a 10% to 15% rise in basal metabolic rate, but trauma, sepsis, and burns can raise a patient's rate up to 120%. Energy expenditure may be increased to 150–200% of normal in the most severely injured or ill patients, whereas most patients in the ICU maintain REEs of 100–150% of normal. This increase in energy requirements is due to the metabolic consequences and synergistic effects of epinephrine, cortisol, and glucagon that are secreted to mount a response to the critical stress ***(Fraipont et al., 2013).***

Pulmonary

Increases in oxygen consumption and carbon dioxide production put greater demand on the pulmonary system. Changes in capillary permeability and perivascular fluid flux force fluids and proteins into alveoli. At the same time, altered immune function and risk for aspiration increase the likelihood of pulmonary infection. These changes can lead to pulmonary failure {either type I (oxygenation) or type II (ventilation) failure}, including the adult respiratory distress syndrome (ARDS) ***(Chen et al., 2011).***

Gastrointestinal

Protein turnover manifests in the gastrointestinal system by increased organ edema and atrophy of villi. Gastric or large bowel ileus frequently signals worsening stress. These changes can confound attempts to provide enteric nutrition and potentially lead to bowel obstruction. excretion of bilirubin and other metabolites is impaired ***(Klein et al., 2016).***

Renal

With peripheral vasodilation, perfusion can be "stolen" from the kidneys. Multiple renal insults that decrease perfusion and circulating mediators may produce a syndrome of oliguria and impaired tubular function up to acute kidney injury. Metabolically active tubular cells suspend function and become quiescent until the stress has long resolved. With recovery, renal function often returns ***(McClave et al., 2016).***

Immunologic

The stress response involves complex immunologic and neurologic signaling. Numerous changes occur in the activity of plasma cytokines, chemical mediators and immune function. They include increased production of proinflammatory cytokines, such as interleukin-1, interleukin-6, and tumor necrosis factor which can cause fever, hypotension, hyperglycemia, and alterations in macronutrient metabolism. Cell-mediated immunity is suppressed and susceptibility to infection increases as systemic inflammatory signals are elevated ***(Douglas, 2016).***

Endocrine

The endocrine axis is integral in signaling changes that accompany the stress response. Hyper-metabolism results from upregulation of catabolic signals and suppression of anabolic hormone signaling. The hypothalamic-pituitary-adrenal axis and pancreas play vital

roles. These neuro-endocrine pathways secrete counter-regulatory hormones (adrenocorticotropic, cortisol, glucagon, and catecholamines) to support the patient during times of severe stress. Relative cortisol deficiency, described in some patients, might worsen vasodilatory shock and stall recovery ***(Corrigan et al., 2011).***

Altered peripheral response to insulin is probably result of immunologic signaling to peripheral tissues such as muscle and fat. Quantity and pulsatility of growth hormone secretion diminish, whereas prolactin secretion is elevated. Vasopressin secretion from the posterior pituitary increases in response to shock but can become depleted, possibly as a form of neuroendocrine exhaustion. The thyroid axis is disturbed, not by changes in thyroid-stimulating hormone as much as by altered peripheral conversion to rT_3 instead of T_3 ***(Boonen et al., 2014).***

The results of changes in endocrine signaling include stress hyperglycemia, the euthyroid sick syndrome, disorders of sleep cycles, and altered immunologic function. In broad terms, the stress response results in pituitary hypersecretion and altered peripheral sensitivity that may give way to exhaustion ***(Dhaliwal et al., 2014).***

Malnutrition

Up to 50% of patients in ICU are malnourished. It is suspected in patients with body mass index (BMI) <18.5kg/m, unintentional weight loss >10% in 3 – 6 months, BMI <20kg/m and unintentional weight loss >5% in 3 – 6 months, impaired intake, digestion and absorption, altered nutritional requirements or excess nutrient losses***(White et al., 2012).***

Metabolic and physiologic changes can be protective mechanisms, but also put patients at risk for developing malnutrition. This acquired malnutrition often is overlooked because of the severity of the initial illness ***(Jeevanandam et al., 2013).***

Often patients have malnutrition before seeking medical attention and if not already malnourished, will become malnourished very quickly (figure 2). It is because of the metabolic demands of stress, anorexia, unable to feed volitionally and the loss of specific vitamins and trace elements ***(Jensen et al., 2013).***

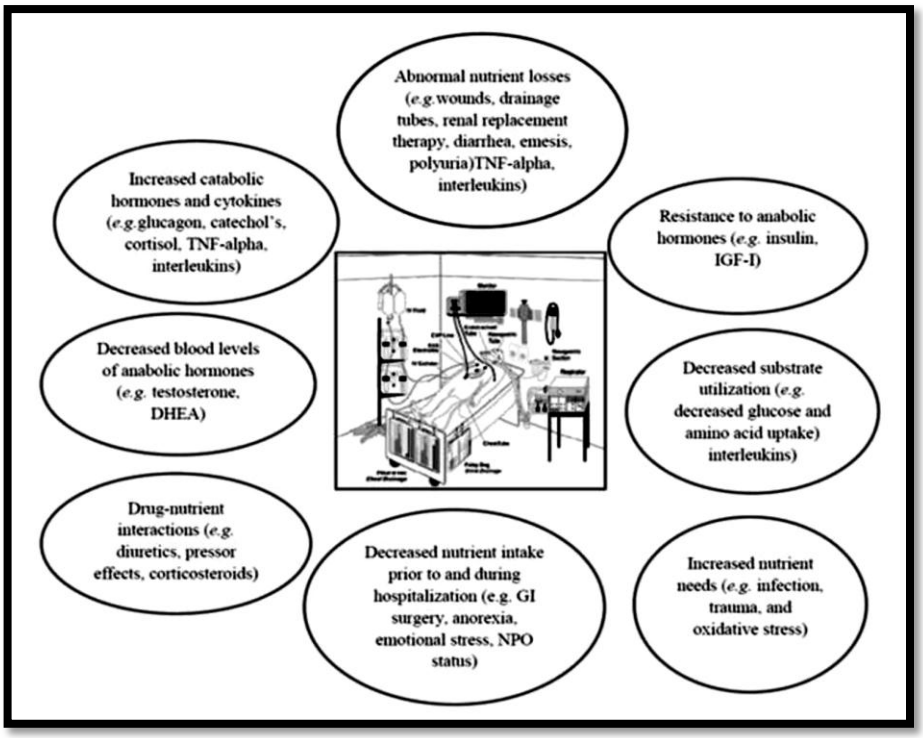

Figure (2): Different causes of malnutrition in ICU patients ***(Jensen et al., 2013).***

Malnutrition represents the deficiency(underfeeding) or excess(overfeeding; if more 40 kcal/kg/day are administered) of energy, protein, vitamins, or minerals with its complications (hepatic dysfunction, abnormal liver function tests, cholestasis,fatty deposits in the liver, excess CO_2 production, fluid overload, respiratory compromise, hyperlipidemia, azotemia (increased urea), hyperglycaemia ***(White et al., 2012).***

Consequences of malnutrition include; Weight loss, Weakness and fatigue, loss of body cell mass, alterations in mineral homeostasis, and derangements in organ system function, impaired ventilatory drive, Depression / apathy, Poor wound healing, Impaired immune function,prolonged dependence on mechanical ventilation, increased rates of infection and death ***(Jensen et al., 2013).***

Chapter (2)
Assessment of nutritional requirements and status

Energy Requirements:-

Energy requirements should be determined to establish the goals of nutrition therapy. These requirements continue to be debated over the past 20 years. Energy requirements in critically ill patients differ per patient and per day and thus should be individually estimated on a daily basis ***(Cresci et al., 2016).***

Determination of the caloric requirements depends on measuring and/or calculating (estimating) the resting energy expenditure which is the amount of calories burnt in 24 hour during a non-active period ***(Kan et al., 2011).***

Factors that influence REE include:-

- The amount of lean body mass is the strongest determinant of resting energy expenditure.
- Age {REE declines 1–2% per decade after the third decade of life}.
- Medications {For example, adrenergic blockades (decrease metabolism by about 12 kcal/m^2/h), epinephrine (increase energy demand up to 2.5 times the basal metabolic rate) and high-dose barbiturate (decrease energy need by up to 34%)}.

- Temperature {fever increase the basal metabolic rate 10% for every 1°C increase above normal temperature}.
- Stress Factors and the underlying disease increase oxygen consumption and REE.
- Sex, thyroid function and nutritional status before illness ***(Frankenfield et al., 2015).***

The gold standard for determining a patient's caloric needs is by indirect calorimetry (with less than a 4% error in critically ill patients) and to which the prediction equations are compared. IC is performed by a trained respiratory therapist to measure oxygen consumption (VO2) and carbon dioxide excretion (VCO2) (both in mL/min), using a closed respiratory circuit ***(Kee et al., 2012).***

For best results, the individual is interfaced by a metabolic measurement system (facemask canopy or mouth piece with nose clip). Then steady-state measurement are obtained for at least 30 minute to determine the REE (Table 1)by **Weir formula**; REE (kcal per day) = 1.44 (3.94 VO_2 + 1.11 VCO_2) and REE (kcal per minute) = 3.94 VO_2 + 1.11 VCO_2 ***(Long et al., 2014).***