General Surgery

Nutrition and Fluid Therapy

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<u>Intro</u>

Malnutrition is common illness; it occurs in about 30% of surgical patients with gastrointestinal disease and up to 60% of prolonged hospital stayed patients because of postoperative complications. Patients who suffer malnutrition have a higher risk of complications and an increased risk of death in comparison with patients who have adequate nutritional reserves. Long-standing protein-calorie malnutrition is easy to recognize. Short-term undernutrition, although less easily recognized, frequently occurs in association with critical illness, major trauma, burns or surgery. The aim of nutritional support is to identify those patients at risk of malnutrition and to ensure that their nutritional requirements are met by the most appropriate route and in a way that minimizes complications.

Metabolic Response to Starvation

After a short fast (12 h or less), most food from the last meal will have been absorbed. Plasma insulin levels fall and glucagon levels rise, converting of hepatic glycogen (200 g) into glucose. The liver, therefore, becomes an organ of glucose production under fasting conditions. Many organs, including brain tissue, blood cells and the kidneys utilize only glucose for their metabolic needs. Additional stores of glycogen exist in muscle (500 g), but these cannot be utilized directly. Muscle glycogen is broken down (glycogenolysis) and converted to lactate, which is then exported to the liver where it is converted to glucose (Cori cycle). With increasing duration of fasting (>24 h), glycogen stores are depleted and glucose production from non-carbohydrate precursors (gluconeogenesis) takes place predominantly in the liver. Most of this glucose is derived from the breakdown of amino acids, particularly glutamine and alanine as a result of catabolism of skeletal muscle (up to 75 g per day). This protein catabolism in simple starvation is readily reversed with the provision of exogenous glucose. With more prolonged fasting there is an increased reliance on fat oxidation to meet energy requirements. Increased breakdown of fat stores occurs, providing glycerol, which can be converted to glucose, and fatty acids, which can be used as a tissue fuel by almost all of the body's tissues. Hepatic production of ketones from fatty acids is facilitated by low insulin levels and, after 48-72 hours of fasting, the central nervous system may adapt to using ketone bodies as their primary fuel source. This conversion to a 'fat fuel economy' reduces the need for muscle breakdown. Another

important adaptive response to starvation is a significant reduction in the resting energy expenditure.

Summary

- Low plasma insulin and high plasma glucagon
- Hepatic glycogenolysis
- Protein catabolism
- Hepatic gluconeogenesis
- Lipolysis
- Adaptive ketogenesis
- Reduction in resting energy expenditure

Metabolic Response to Trauma and Sepsis

From a nutritional point of view, focus on two factors; First, in contrast to simple starvation, patients with trauma have impaired formation of ketones, and gluconeogenesis cannot be prevented by glucose administration. Second, although metabolic response to trauma and sepsis is always associated with "hypermetabolism" or "hypercatabolism" but there is no indication for high energy intake (may be harmful).

Nutritional assessment

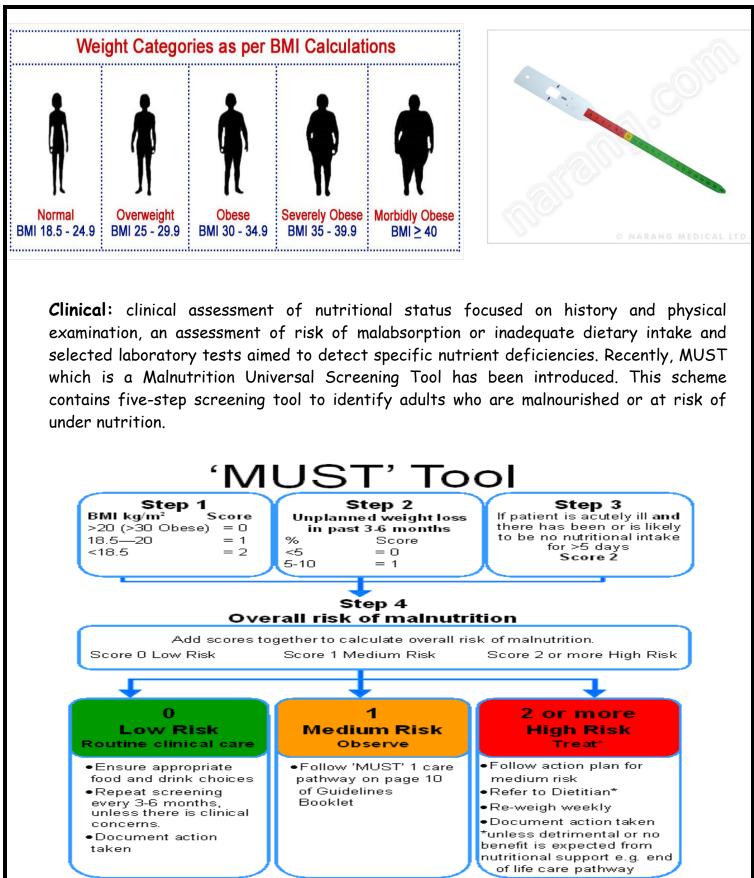
Lab. Technique: there is no biochemical test that reliably identifies malnutrition. Some test could be helpful:

- 1. Serum albumin
- 2. Lymphocytes count and skin test (delayed hypersensitivity) measure immune system.

Body Weight and Anthropometry

Estimating weight loss is done by comparing the measured body weight with ideal body weight. BMI Body Mass Index is weight in kg divided by height in squared meters. A BMI of less than 18.5 indicates nutritional impairment, and below 15 associated with significant hospital mortality.

Anthropometric techniques include measurements of skinfold thickness and mid-arm circumference permit estimations of body fat and muscle mass, these are indirect measures of energy and protein stores.



This tool is to assist your assessment. If in doubt, use your professional judgement

Fluids and Electrolytes

Fluid intake is derived from both exogenous (consumed liquids) and endogenous (released during oxidation of solid foodstuffs) fluids.

Fluid losses occur by four routes:

- 1. Lungs: 400 ml of water lost in expired air per 24 h.
- 2. Skin: 600-1000 ml/day lost by sweating (depends on climate temperature)
- 3. Feces: 60-150 ml/day
- 4. Urine: 1500ml/day. The minimum urine output is 400ml/day is required to excrete the end products of protein metabolism.

Maintenance of fluid requirements are estimated of insensible and obligatory losses, the nature and type of fluid replacement therapy will be determined by individual patient needs.

COMPOSITION OF COLLOIDS ...

The daily requirement of electrolytes is as following:

- 1. Sodium 50-90 mM/day
- 2. Potassium 50 mM/day
- 3. Calcium 5 mM/day
- 4. Magnesium mM/day

Colloid Fluid K+ Ca+2 CI-Na⁺ Osmoti solutions Pressur ٧ ٧ Composition Notes Albumin 5% 130-160 <1 27 Types of isotonic Gelatin 154 <0.4 <0.4 125 34 NaCl 0.9% Na 150 mmol/L Use to correct ECF loss and for initial resuscitation of (Gelofusin) CI 150 mmol/L intravascular volume. Hydroxyethy 154 0 0 154 28 Ringer's Lactate Na 131 mmol/L It is physiological solution. After administration the I Starch 6% (Haartman) 5mmol/L lactate is metabolised, resulting in bicarbonate Κ Dextran 60 130 4 2 110 22 2mmol/L generation. It will decrease the risk of Ca (3%) CI 111mmol/L hyperchloraemia HCO3 29mmol/L Gelatin 145 5.1 6.25 145 27 (Haemaccel) Dextrose 5% dextrose 50g/L Glucose is rapidly metabolized. The remaining water 200kcal/L distributes rapidly throughout the body's fluid Ionic 135-145 3.8-5.1 2.2-2.6 100-110 27 composition compartments therefore not suitable for resuscitation. of n-plasma

The most common crystalloid

The fluid therapy contains three main strategies:

- 1. Maintenance fluid
- 2. Replacement fluid (required to correct pre-existing deficiencies)
- 3. Supplemental fluid (required to compensate for additional intestinal or other losses

The nature and volumes of fluid therapy are determined by

- A careful assessment of the patient including, pulse, blood pressure and central venous pressure. Clinical examination to assess hydration status (peripheries, skin turgor and urine output), urine and serum electrolytes and hematocrit.
- Estimation of ongoing losses and nature (vomiting, ileus, diarrhea, excessive sweating and burns)
- Estimation of supplemental fluids required for future losses from drains, fistulae, nasogastric tubes or abnormal urine or fecal losses.
- When volumes have been estimated, the appropriate replacement fluid can be determined from a consideration of the electrolytes composition of gastrointestinal secretions. Most intestinal losses are adequately replaced with normal saline containing supplemental potassium.

COMPOSITION OF GI SECRETIONS

Type of Secretion	Volume (mL/24 h)	Na (mEq/L)	K (mEq/L)	CI (mEq/L)	HCO ₃ ⁻ (mEq/L)
Stomach	1000–2000	60–90	10–30	100–130	0
Small intestine	2000–3000	120–140	5–10	90–120	30–40
Colon		60	30	40	0
Pancreas	600–800	135–145	5–10	70–90	95–115
Bile	300-800	135–145	5–10	90–110	30–40

Nutritional Requirement

Total enteral or parenteral nutrition necessitates the provision of the macronutrients, carbohydrate, fat, protein, vitamins, trace elements, electrolytes and water. When planning a feeding regimen, the patient should be weighed and assessed for daily energy and protein requirements. Daily needs may change depending on the patient's condition, overfeeding is the most common cause of complications, regardless of whether nutrition is provided enterally or parenterally.

Macronutrient requirement

- Energy: the total energy requirement of a stable patient with a normal or moderately increased need is approximately 20-30 kcal/kg/day. Total energy requirement is about 1300-1800 kcal/day.
- **Carbohydrate:** there is an obligatory glucose requirement to meet the needs of the central nervous system and certain haemopoietic cells, which is equivalent to about 2 g/kg/day. There is a maximum physiological oxidation of glucose which is 4 mg/kg/min (equivalent to about 1500 kcal/day in a 70 kg person). Non-oxidized glucose being primarily converted to fat.
- Fat: dietary fat is composed of triglycerides predominately four long-chain fatty acids. There are two saturated fatty acids (palmitic and stearic) and two unsaturated (oleic and linolenic). Basal requirement of essential fatty acids is 100-200 g/week.
- **Protein:** basic requirement for nitrogen in patients without pre-existing malnutrition and without metabolic stress is 0.1-0.15g/kg/day.
- Vitamins, Minerals and Trace Elements: post-operatively, the vitamin C requirement increases to 60-80 mg/day. Supplemental vitamin B12 is often indicated in patients undergone intestinal resection or gastric surgery and in alcoholic patients. Absorption of fat-soluble vitamins A, D, E and K is reduced in steatorrhea.

Fluid and Nutritional Consequences of Intestinal Resection

With extensive resection, metabolic and nutritional consequences arise, resulting in the disease entity known as short bowel syndrome.

- Small bowel motility: it is three times slower in the ileum than in the jejunum. The adult small bowel receives 5-6 liters of endogenous secretions and 2-3 liters of exogenous fluids, most of these fluids reabsorbed in the small intestine, also for sodium reabsorption (mainly ileum) occurs up to 70%. So, ileum is critical in the conservation of fluid and electrolytes.
- **Ileum:** it is the only site of absorption of vitamin B12 and bile salt (essential to absorb fats and fat-soluble vitamins).
- **Colon:** efficacy of salt and water reabsorption in the colon exceeds 90%. Another important colonic function is the fermentation of carbohydrates to produce short-chain fatty acids; these have two important functions: first, they enhance water and salt reabsorption from colon and second, they are trophic to colonocytes.

Effects of Resection

- **Resection of jejunum:** No significant alteration in fluid and electrolytes levels as the ileum and colon can adapt to absorb the increased fluid and electrolytes load. There is no malabsorption.
- **Resection of ileum:** The colon receives a much larger volume of fluid, electrolytes and bile salts, which reduce its ability for absorption resulting in diarrhea. Even the loss of 100 cm of ileum causes steatorrhea, with larger resection > 100 cm, dietary fat restriction and vitamin B12 supplement required.
- Short bowel syndrome: Most challenging patients are those with short bowel syndrome who have had in excess 200 cm of small bowel resected together with colectomy, they are usually having a jejunostomy. They are divided into two groups termed 'net absorbers' and 'net secretors'

Absorbers have more than 100 cm of residual jejunum and they absorb more water and sodium from the diet and passes through the stoma, these patients can be managed without supplementary parenteral fluids.

Secretors usually have less than 100 cm of residual jejunum and lose more water and sodium from their stoma than they take by mouth (up to 4 liters/day).

Treatment begins with restriction of hypotonic fluids (water, tea, juices, etc.) consumed and encouragement to have glucose and saline replacement solutions.

Complications of Short Bowel Syndrome

- 1. Peptic ulcer due to gastric hypersecretion.
- 2. Cholelithiasis (gall stones) due to interruption of enterohepatic cycle of bile salts.
- 3. Hyperoxaluria (renal stones) due to increased absorption of oxalate in the colon.
- 4. Some patients develop a syndrome of slurred speech and ataxia due to fermentation of malabsorbed carbohydrates in the colon and its absorption.