



# I. Introduction

Adequate nutrition plays an important role in maintaining optimal health. The consequences of malnutrition have been well documented and contribute significantly to morbidity and mortality. Malnutrition increases health care costs by prolonging hospital length of stay due to the increased probability of medical complications which result from compromised immune function and impaired wound healing.

In general, malnutrition may be envisioned as a continuum with four basic stages. Initially, decreases in nutrient intake (e.g. poor diet, eating difficulty) or excessive losses (e.g. chronic diarrhea, abnormal bleeding, large draining wounds) limit nutrient availability. Next, nutrient stores are depleted as nutrients are required to meet metabolic demands. In the third stage, metabolic or biochemical changes occur, leading to marginal malnutrition. These are followed by deficiency symptoms. In this last stage, referred to as clinical or symptomatic malnutrition, cell or tissue damage is present and nutrient deficiencies are manifest by specific, observable symptoms. Malnutrition that results in the depletion of body cell mass and impaired function frequently accompanies acute and chronic diseases, as well as trauma. Perhaps as many as 40 to 50 percent of hospitalized medical or surgical patients either have or are at risk for developing malnutrition, with 5 to 10 percent of patients classified as severely malnourished.

This manual is not intended to be a comprehensive textbook on nutrition but rather is intended to provide guidelines for the evaluation and management of patients with exceptional nutritional requirements. The goal of this resource manual is to maximize patient benefits from parenteral or enteral nutrition therapy while minimizing potential complications.

## II. Nutrition Assessment

### A. Risk Factors

There are numerous risk factors for poor nutritional status, including major trauma, burns, sepsis, substance abuse, recent weight loss, and many gastrointestinal disorders. Additional information learned through a careful medical history can also suggest possible risk factors for malnutrition. The factors listed below may place a patient at risk for developing, or may denote the presence of, nutrient deficiencies.

- ◆ Age < 18 years or > 65 years (increased risk age >75 years)
- ◆ Recent significant, unintentional weight loss: > 5% in 1 month or >10% in 6 months

Weight loss calculated as follows:

$$\text{Percent weight loss} = (\text{UBW} - \text{CBW}) / \text{UBW}$$

Where: UBW = usual body weight, CBW = current body weight

- ◆ Excessive alcohol intake, other substance abuse
- ◆ Homelessness, limited access to food
- ◆ Limited capacity for oral intake (dysphagia, odynophagia, stomatitis, mucositis)
- ◆ NPO > 3 days
- ◆ Increased metabolic demands: extensive burns, major surgery, trauma, fever, infection, draining abscesses, wounds, fistulae, pregnancy
- ◆ Protracted nutrient losses: malabsorption syndromes, short gut syndrome, draining abscesses, wounds, fistulae, effusions, renal dialysis
- ◆ Intake of catabolic drugs: corticosteroids, immunosuppressants, antineoplastics

- ◆ Protracted emesis: anorexia nervosa, bulimia, hyperemesis gravidarum, radiation, cancer chemotherapy
- ◆ Chronic disease (especially AIDS, diabetes, cystic fibrosis, stroke, cancer)

## **B. Diet History**

A detailed diet history provides insight into a patient's baseline nutritional status and may detect subclinical nutrient deficiencies or toxicities. Assessment includes questions regarding chewing or swallowing problems, avoidance of eating related to abdominal pain, changes in appetite, taste, or intake, as well as use of a special diet or nutritional supplements.

## **C. Medical History**

A review of past medical history includes identifying existence of conditions resulting in increased metabolic needs, altered gastrointestinal function and absorptive capacity, chronic disease states, organ failure, and levels of physical activity. A review of current medications may further elucidate at-risk nutrient status. (See **Appendix A**).

## **D. Physical Examination**

Physical examination should focus on assessment of muscle mass and strength, evidence for chronic liver disease and signs of vitamin or mineral deficiency. In the United States it is uncommon, though not rare, to find patients with classical manifestations of far-advanced vitamin or mineral deficiencies (see **Appendix B**), though short term, acute vitamin deficiencies are more common than appreciated.

## E. Subjective Global Assessment

A systematic bedside assessment of nutritional status has been shown to accurately categorize patients as well nourished, moderately malnourished or severely malnourished. A worksheet adapted from the original research publication (JPEN 1987; 11:8-13) can be found in **Appendix C**.

## F. Laboratory Tests

Biochemical measurements are useful to assess organ function, fluid status and electrolyte balance, confirm nutritional deficiencies, and monitor the adequacy of nutritional therapies. Useful baseline laboratory data includes basic chemistries (e.g. electrolytes, glucose, BUN and creatinine), liver function tests, hemogram, albumin, and transthyretin. C-Reactive protein should be measured concomitantly with transthyretin in patients with suspected metabolic response to injury/infection. For further comments on the use of laboratory tests see Section: VII. Monitoring Nutrition Therapy (page 37). Laboratory tests for individual nutrients are available (see **Appendix D**).

# III. Estimating Nutritional Requirements (Adults)

## A. Determine Usual or “Adjusted” Body Weight

If the patient is severely underweight (<80% of IBW) then use Current Weight for nutrient calculations.

If the patient is obese, use:

Adjusted Body Weight =  $IBW + 0.25(Usual\ Weight - IBW)$ .

Ideal body weight can be used for nutritional assessment purposes. See **Appendix E** for IBW tables based on height for males and females.

## **B. Energy Requirements**

Most hospitalized patients will require 30 kcals/kg/d. Refer to **Appendix F** for detailed information on estimating energy requirements. Dietitians can also provide more refined estimates of nutrient requirements. At Harborview Medical Center, a metabolic cart is available for estimation of energy requirements. Overfeeding should be avoided.

## **C. Protein Requirements**

Protein needs may vary greatly with the metabolic status of the patient. The average patient receiving nutritional intervention requires 0.8 – 2.0 g protein/kg usual body weight. The obese patient is unusual. Use of usual body weight can result in overfeeding. It is recommended to use Adjusted Body Weight (ABW) for reasonable estimation of nutrient requirements. The goals of nutrition support are to minimize protein breakdown, preserve lean body mass, promote protein synthesis, and optimize immune responses. The factors listed in **Table I** can be used to estimate protein requirements.

**Table I: Estimating Protein Requirements**

Clinical Status	Protein Requirements (g/kg/day)*
Maintenance	0.8-1.0
Mild to Moderate Depletion	1.0-1.5
Post-operative	1.2-2.0

\*Note: Based on usual body weight except in obese patients.

## D. Fluid Requirements

A healthy adult ingests approximately 1 mL free water/kcal of energy, or 35-50 mL/kg body weight/day. Hospitalized patients usually require 30-35 mL/kg/day. Fluid needs may also be approximated as 1500 mL per m<sup>2</sup>BSA. However, wide variations in fluid intake are normally well tolerated without producing hypo- or hypernatremia or fluid overload. Patients with liver disease, renal failure, cardiac or pulmonary diseases or closed head injuries may require restricted fluid intakes while patients with nasogastric output, diarrhea, hypovolemia secondary to burns or trauma, diuresis, fistulae, and insensible losses may require additional fluids. Insensible losses are the result of respiration, fecal loss, evaporation, and fever. Replace diarrhea output volume per volume with normal saline, nasogastric or fistulous output with 1/2 normal saline, and evaporation due to fever (250 mL/day for each °C above 37°C) with sterile water or D5W.

Volume depleted patients should be rehydrated and electrolytes repleted **before** initiating PN, i.e. fluid deficits should not be corrected with amino acid and dextrose solutions. PN solutions are extremely hyperosmolar and cannot be converted to an equivalent iso-osmolar volume or volume of free water. Additional fluids (e.g. normal saline, sterile water) can be added directly to PN bags, some enteral feeding bags, or can be administered as boluses in tube fed patients.

## E. Electrolyte Requirements

Electrolyte requirements for the average adult patient **without** significant cardiovascular, hepatic, or renal disease, or an underlying electrolyte abnormality, or significant electrolyte loss (e.g. fistulae) are shown in **Table II**. Electrolyte needs are adjusted daily based on lab results and current clinical status of the patient.

**Table II: Typical Adult Baseline Electrolyte Requirements During Nutritional Repletion**

Electrolyte	Daily Requirement	Comments
sodium (chloride, acetate, or phosphate)	60-150 mEq	basal catabolism: 1-4 mEq/kg mild- moderate catabolism: 2-3 mEq/kg severe catabolism: 3-4 mEq/kg
potassium (chloride, acetate, or phosphate)	70-150 mEq	basal catabolism: 0.7-0.9 mEq/kg mild- moderate catabolism: 2 mEq/kg severe catabolism: 3-4 mEq/kg
chloride (sodium or potassium)	60-150 mEq	replaced 1 mEq per 1 mEq Na <sup>+</sup> or K <sup>+</sup> unless other salt form specified
calcium (gluconate)	10-15 mEq	monitor ionized calcium
magnesium (sulfate)	8-24 mEq	monitor serum Mg concentration
phosphate (sodium or potassium)	7-10 mMol per 1000 kcal	severe catabolism or prolonged absence of nutritional intake: 15-25 mMol per 1000 kcal of glucose

Nutritional repletion therapy increases electrolyte requirements. During the first 3 to 5 days of re-feeding, patients typically pass through three phases of electrolyte utilization. During the first 24 to 48 hours, total body deficits must be replaced. In the second phase, which may last for several days, anabolic processes are induced which result in increased intracellular uptake of potassium and phosphate. After approximately one week of providing nutritional therapy, electrolyte requirements become relatively stable.



## F. Fat Requirements

A wide range of fat intake is generally well tolerated by most individuals. Current national guidelines recommend limiting fat intake to less than 30% of total kcals. A higher percent fat intake may be desired for patients with poor appetites/limited food intake to increase caloric density of foods (fat contains 9 kcal/g vs. 4 kcal/g in carbohydrates and protein). A minimum of 2-4% kcals as linoleic acid is required daily to prevent essential fatty acid deficiency. See Section VI: Initiating Parenteral Nutrition (page 25) for guidelines on parenteral lipid administration.

## G. Micronutrient Requirements

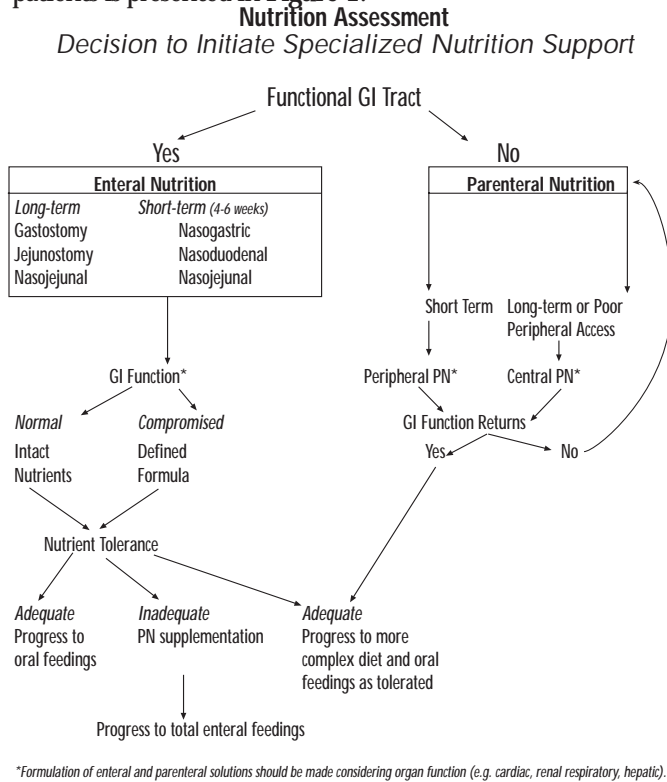
Twelve vitamins and six trace elements are known to be essential in human metabolism (**Appendix G**). Hospital diets (except liquid diets) and enteral formulas are designed to provide at least the recommended daily allowance of each known essential nutrient.

Very little information is known about the micronutrient requirements of catabolic or hospitalized patients. Hypermetabolism, infections, and anabolic processes increase micronutrient requirements above those needed for health maintenance. Furthermore, many patients may present with micronutrient deficits due to poor food intake, absorption or utilization of micronutrients associated with disease processes.

# IV. Nutrition Therapy Options

## A. Nutrition Modalities

A clinical decision algorithm that outlines the selection process for choosing the route of nutrition support in adult patients is presented in **Figure 1**.



**Figure 1.** Nutrition Therapy Decision Algorithm



**Figure 1. Nutrition Therapy Decision Algorithm**

**1. Oral Diet**

The oral route is always the preferred route for providing nutritional intake. Many different types of oral diets are available (see *Manual of Clinical Dietetics* located on each Nursing Unit). In addition, commercially prepared liquid oral supplements can be used in conjunction with an oral diet to promote adequate nutrient intake. Dietitians can perform a nutrient intake analysis (calorie/protein count) to evaluate the adequacy of daily oral nutrient intake if needed.

**2. Enteral Nutrition**

Tube feedings are indicated for patients who are unable to ingest adequate nutrients normally and safely by mouth but who have at least a partially functional GI tract. **Enteral nutrition support is preferred to parenteral nutrition** because it facilitates maintenance of intestinal structure and function, improves immunity, and avoids catheter-related complications associated with the use of PN. Enteral nutrition support is significantly less expensive than parenteral nutrition.

**3. Parenteral Nutrition (PN)**

Parenteral nutrition support is indicated in the presence of compromised nutritional status when adequate protein and calories cannot be provided by oral or other enteral routes. Parenteral nutrition includes both peripheral parenteral nutrition (PPN) and central or total parenteral nutrition (TPN).

# V. Enteral Nutrition

## A. Tube Placement Options

### 1. Nasoenteric Feeding Tubes

Tubes are passed through the nose to various points in the GI tract and are named with reference to the location of the terminal end of the feeding tube. Examples include **nasogastric**, **nasoduodenal**, and **nasojejunal** tubes.

#### Advantages

- ◆ avoids general anesthesia or surgical procedure
- ◆ low incidence of complications

#### Disadvantages

- ◆ risk of aspiration (may be less with nasoduodenal and nasojejunal tube)
- ◆ X-ray confirmation of correct tube placement required
- ◆ suited only to short-term (less than 6 weeks) use

### 2. Tube Enterostomy

Tubes are placed either laparoscopically, operatively, or percutaneously. Examples include **esophagostomy**, **gastrostomy**, **jejunostomy**, **percutaneous endoscopic gastrostomy (PEG)**, **percutaneous endoscopic jejunostomy (PEJ)**, **needle catheter jejunostomy (NCJ)**, **operative laparoscopic gastrostomy**, **operative laparoscopic jejunostomy**.



### **Advantages**

- ◆ may be used immediately or within hours of placement
- ◆ may be used for long-term support
- ◆ may be used in presence of significant disease of upper GI tract (esophagus, stomach and duodenum)
- ◆ percutaneously placed tubes avoid risks of surgery and general anesthesia
- ◆ laparoscopic feeding tubes allow patients to return home the same day after procedure

### **Disadvantages**

- ◆ may require endoscopy, abdominal ultrasound, or radiologic procedure with contrast media
- ◆ endoscopy may be difficult or impossible in presence of tumor or stricture, altered anatomy or severe obesity
- ◆ laparoscopically or operatively placed tubes require general anesthesia
- ◆ potential for chronic wound complications

## **B. Formula Selection**

When selecting an appropriate enteral formulation both formula characteristics and patient-specific factors should be considered. Formula variables include: digestibility/availability of the nutrients, nutritional adequacy, viscosity, osmolality, ease of use, and cost. Patient variables include: nutritional status and requirements, electrolyte balance, digestive and absorptive capacity, disease state, renal function, medical or drug therapy, and possible routes available for administration. Adult enteral formula products fall into one

of the following categories: general use, high nitrogen, high nitrogen and high calorie, fiber enriched, semi-elemental, fat modified, and specialty. Dietitians are available to assist with formula selection. HMC and UWMC RD's prepare and distribute enteral formulary pocket reference cards containing up-to-date product information for physician use.

## **C. Administration Guidelines**

### **1. Initiation and Progression**

To promote tolerance, enteral tube feedings should be initiated at rates of 50 cc/hr in adults. Most currently available formulas are isotonic (300 mOsm/L) and are well tolerated at full strength when delivered into the stomach or small intestine. The rate of administration of isotonic formulas can usually be advanced in 20-25 cc/hr increments every 8 hours until goal rate is achieved. It is often more realistic to calculate goal rates based on 20-22 hours/day allowing for interruptions in delivery.

### **2. Calculate Additional Free Water Requirements**

Most patients on enteral nutrition therapy will require additional fluid to meet minimum fluid requirements. To calculate additional fluid requirements begin by determining the patient's total fluid needs (see Section III part D). Then determine the amount of free water provided by the tube feeding formula by multiplying the percent free water content (information available in diet manual, on enteral formulary cards, or by contacting a dietitian) by the total volume of tube feeding formula to be administered each day. Subtract the free water supplied by the formula from the calculated total free water requirement which equals remaining volume of free water and divide the remaining into 3 or 4 boluses per day.

**Example:**

*A 50 year old female weighing 55 kg requires full strength Isosource HN at 55 mL/hr to meet her energy and protein requirements and 1800-2000 mL fluid/d. Isosource HN is 82% free water. Then  $55 \text{ mL/hr} \times 24 \text{ hr} \times 0.82 = 1082 \text{ mL free water/d}$  provided by the enteral formula. Her additional fluid needs are  $1800 - 1082 = 718 \text{ mL/d}$  or about 3 boluses of 250 mL each.*

### **3. Transition to Cyclic or Bolus Feedings**

Hospitalized patients may initially benefit from a continuous infusion to establish tolerance to enteral nutrition therapy and later transition to an intermittent infusion schedule. Intermittent infusion (bolus feedings) can be administered by gravity dip or syringe bolus for those patients with gastric feeding tubes.

Cyclic feeding using a pump can be used in patients with intestinal feeding tube sites (duodenum or jejunum). Cyclic feeding infuse formula for a set number of hours (e.g. 8-12 hours overnight). A cyclic feeding schedule should be considered for patients for whom free time off the pump is desired.

### **4. Vitamin and Mineral Requirements**

Enteral formulations contain varying amounts of vitamins and minerals and may not be sufficient to meet patient needs. Consult a dietitian for the micronutrient content of specific products and for assistance in determining supplementation for these patients.

## 5. Monitoring

Routine nursing care includes checking gastric residuals every 4-6 hr in patients receiving gastric feedings until desired rate is established. Infusions are held for 1 hour if gastric residual is  $>(1.0 \text{ to } 1.5) \times$  hourly rate or  $>150$  mL before bolus or intermittent feeding.

Daily weights, I/O records, serum electrolytes, phosphorus, magnesium, and ionized calcium should be monitored until tolerance is established and patient is stable (see Section VII).

### D. Writing Enteral Nutrition Orders

- a. Confirm feeding tube placement by KUB before initiating enteral nutrition.
- b. Document location of the terminal end of the feeding tube in the orders for initiating tube feeding.
- c. Order desired formula and indicate initial strength, initial rate (mL/hr), desired progression regimen and goal strength and rate.
- d. Order additional fluid (e.g. sterile water or normal saline) to flush tube with at least 30 mL every eight hours and provide needed hydration.
- e. Note that 20-30 mL sterile water should be used to flush feeding tube before and after administering medications via the feeding tube.
- f. Order all routine monitoring parameters, baseline and routine lab tests.

**Example:**

1. Place feeding tube into stomach
2. Get KUB
3. Initiate Isosource HN full strength at 30 mL/hr.  
Increase rate by 20 mL/hr every 6 to 8 hours or as tolerated to goal of 70 mL/hr.
4. Give additional 250 mL H<sub>2</sub>O TID per feeding tube
5. Mini panel, P, Mg, iCa and Nutrition Monitoring Panel in a.m.

**E. Medications and Enteral Nutrition**

The following are general guidelines for ordering and administering medications to patients receiving enteral nutrition.

- ◆ If the patient is able to take medications by mouth, the **oral route is preferred** over administration via the feeding tube
- ◆ For certain medications that pose a particular problem for enteral administration (e.g. repeatedly clogs tube, unavailable in suitable liquid or crushable form, unpredictable absorption, etc.), **alternative routes of administration that bypass the tube or even therapeutic alternatives should be considered.** Alternative routes may include IV, IM, PR, SL, or transdermal.
- ◆ For individual doses of most medications, the **tube should be flushed** with at least 30 mL of sterile water before **and** after administration. This serves to clear the tube for drug delivery, facilitate drug transport to the intestine, and indicates whether the tube is cleared.

- ◆ When several medications are to be administered, all medications should be **given separately** and **the tube flushed** with at least 5 mL of water after **each** dose.
- ◆ Most drugs in suspension, elixir, or other liquid form are **hypertonic**. Highly concentrated drug solutions and suspensions **should be diluted with at least 60 mL of water** before administration to decrease gastric mucosal irritation and prevent osmotic diarrhea.
- ◆ For patients receiving many medications via the feeding tube or for critically ill patients, **the volume required for diluting, flushing, and administering medications may be significant**. Alternative routes of administration may need to be considered.
- ◆ **Medications should never be added directly to the feeding formulation**. The potency, stability, and availability of the medication as well as the stability of the enteral formulation cannot be ensured.
- ◆ For most medications, the **enteral feeding should be stopped for at least 15 minutes before and after drug administration**. Certain drugs have increased bioavailability, produce more predictable blood levels, and/or are better tolerated on an empty stomach. Some drugs may require feedings to be held for longer intervals. For example, dilantin administration requires that the feeding be stopped for one hour prior to and after dosage. **Multiple interruptions in formula delivery may compromise nutrition support and should therefore be avoided..** Other routes of drug administration may need to be considered.
- ◆ Stop gastric feedings 1/2 hour prior to and after treatment or procedures requiring the Trendelenberg position (e.g. chest physiotherapy, central line insertion).

- ◆ **Call Pharmacy** for any questions regarding the availability of dosage forms suitable for tube administration, alternative dosage forms and routes, contents of a specific formulation, specific drug administration techniques, or timing of drug administration with enteral feedings.
- ◆ Drugs can not be put down NCJ.

## F. Complications of Enteral Nutrition Therapy

Patients should be monitored frequently for evidence of complications from enteral nutrition support. **Table III** lists potential complications of tube feedings and offers suggestions for intervention.

**Table III: Complications of Enteral Nutrition Therapy: Possible Causes & Management**

Complication	Possible Cause	Suggested Management
<b>Gastrointestinal</b>		
diarrhea (> 4 BM per day or large loose stool)	medications	<ul style="list-style-type: none"> <li>◆ eliminate antibiotics or antacids if possible</li> <li>◆ eliminate liquid formulations containing sorbitol</li> </ul>
	fat intolerance	◆ change to low fat formula
	bacterial overgrowth	<ul style="list-style-type: none"> <li>◆ stool culture for pathogens</li> <li>◆ Rx L. acidophilus/L. bulgaricus (Lactinex™) if patient receiving antibiotics</li> </ul>
contaminated formula		<ul style="list-style-type: none"> <li>◆ DC current formula</li> <li>◆ replace bag and tubing using aseptic techniques</li> <li>◆ adhere to clean standard when changing or manipulating feeds</li> </ul>

Complication	Possible Cause	Suggested Management
<b>Gastrointestinal cont.</b>		
	osmotic overload	<ul style="list-style-type: none"> <li>◆ decrease concentration of formula</li> <li>◆ change to isotonic formula</li> <li>◆ further dilute hypertonic medications</li> <li>◆ administer medications by alternate route</li> </ul>
	decreased bulk	<ul style="list-style-type: none"> <li>◆ change to high fiber formula</li> <li>◆ administer bulking agents (e.g. psyllium) but not through small bore (&lt;10 French) feeding tubes</li> </ul>
nausea or vomiting	patient position	<ul style="list-style-type: none"> <li>◆ position patient on right side to facilitate passage of gastric contents through pylorus</li> </ul>
	volume overload	<ul style="list-style-type: none"> <li>◆ decrease total volume</li> <li>◆ decrease delivery rate to one tolerated previously</li> <li>◆ advance delivery rate slowly over 12 to 24 hours</li> </ul>
	delayed gastric emptying	<ul style="list-style-type: none"> <li>◆ stop feeding for 2 hours &amp; check residuals</li> <li>◆ change to low-fat formula</li> <li>◆ administer prokinetic agent (metoclopramide, cisapride) to stimulate GI motility</li> </ul>
	specific nutrient intolerances	<ul style="list-style-type: none"> <li>◆ change to lactose-free or low-fat formula</li> </ul>
	GI tract obstruction	<ul style="list-style-type: none"> <li>◆ stop feeding</li> </ul>
constipation (no stool x 3 days)	dehydration & impaction	<ul style="list-style-type: none"> <li>◆ provide free water</li> <li>◆ remove impaction</li> </ul>
	decreased fiber	<ul style="list-style-type: none"> <li>◆ change to high fiber formula</li> <li>◆ administer bulking agents (e.g. psyllium) but not through small bore feeding tubes (&lt;10 French)</li> </ul>

Complication	Possible Cause	Suggested Management
<b>Gastrointestinal cont.</b>		
	GI tract obstruction	◆ stop feeding
<b>Mechanical</b>		
pulmonary aspiration	patient lying flat	◆ elevate head of bed 30 to 45° during continuous feeds or for 30 to 60 minutes after bolus feeds
	absent or depressed gag reflex	◆ infuse feedings into duodenum or jejunum
	reflux	◆ change to smaller bore tube (<12 French) (large bore tubes can reduce LES competence) ◆ infuse feedings into duodenum or jejunum
	improper tube placement	◆ confirm proper placement by X-ray after insertion, after severe coughing, vomiting, or seizure ◆ reconfirm placement prior to each feeding by checking residuals ◆ tape tube in place & mark tube at exit point for reference ◆ restrain patient if unable to keep from pulling
tube obstruction	acid precipitation of formula	◆ flush tube with water before & after check gastric residuals ◆ infuse feedings into duodenum or jejunum ◆ do not mix medications with enteral formula
	insufficient tube irrigation	◆ flush tube with warm water before & after each bolus feeding, every 8 hours during continuous feeding, or whenever feeding is stopped

Complication	Possible Cause	Suggested Management
<b>Mechanical</b> <i>cont.</i>		
	medications	<ul style="list-style-type: none"> <li>◆ adequately crush medications and mix powder with water</li> <li>◆ use liquid medications where possible or administer by alternative route</li> <li>◆ flush tube before &amp; after medication administration with at least 20 mL warm water</li> <li>◆ avoid administering bulk forming agents via small bore tubes</li> </ul>
mucosal damage	extended use of large bore tubes	<ul style="list-style-type: none"> <li>◆ alternate nares</li> <li>◆ change to small bore tube (&lt;10 French)</li> <li>◆ change to a permanent gastrostomy or jejunostomy tube for extended enteral support</li> <li>◆ tape in place to minimize rubbing</li> </ul>
<b>Metabolic</b>		
overhydration	refeeding	◆ decrease delivery rate
	fluid overload	<ul style="list-style-type: none"> <li>◆ restrict free water</li> <li>◆ change to concentrated formula</li> <li>◆ administer diuretics</li> </ul>
dehydration	high osmolality formula	◆ change formula
	diarrhea	<ul style="list-style-type: none"> <li>◆ change formula</li> <li>◆ see above management of diarrhea</li> </ul>
	excessive protein intake with inadequate fluid intake	<ul style="list-style-type: none"> <li>◆ change decreased protein content formula</li> <li>◆ provide additional water</li> </ul>

Complication	Possible Cause	Suggested Management
<b>Metabolic cont.</b>		
hyperglycemia	insulin deficiency	<ul style="list-style-type: none"> <li>◆ give insulin (insulin drip used more successfully for enterally tube fed patients)</li> <li>◆ change formula to higher fat/ lower carbohydrate content</li> <li>◆ change to high fiber formula</li> </ul>
hypoglycemia	sudden cessation of feedings	<ul style="list-style-type: none"> <li>◆ taper feedings</li> <li>◆ monitor blood sugar if feeding interrupted</li> </ul>
hyperkalemia	metabolic acidosis	<ul style="list-style-type: none"> <li>◆ reduce K intake/use reduced K formula</li> <li>◆ Rx Kaexalate™</li> </ul>
	renal insufficiency	<ul style="list-style-type: none"> <li>◆ reduce K intake/use reduced K formula</li> <li>◆ Rx Kaexalate™</li> <li>◆ assess renal function</li> </ul>
	anabolic metabolism	<ul style="list-style-type: none"> <li>◆ reduce potassium intake/use reduced K formula</li> </ul>
hypokalemia	refeeding syndrome	<ul style="list-style-type: none"> <li>◆ monitor serum K daily and replete until stable</li> </ul>
	insulin administration	<ul style="list-style-type: none"> <li>◆ lower dose or discontinue</li> </ul>
	diuretics	<ul style="list-style-type: none"> <li>◆ discontinue if possible</li> </ul>
	diarrhea	<ul style="list-style-type: none"> <li>◆ see management above</li> </ul>
hyperphosphatemia	renal insufficiency	<ul style="list-style-type: none"> <li>◆ use reduced PO<sub>4</sub> formula</li> <li>◆ administer phosphate binder</li> </ul>
hypophosphatemia	refeeding syndrome, insulin administration	<ul style="list-style-type: none"> <li>◆ monitor serum PO<sub>4</sub> daily and replete until stable</li> </ul>
hypomagnesemia	refeeding syndrome, alcoholism	<ul style="list-style-type: none"> <li>◆ monitor serum Mg daily and replete until stable</li> </ul>
hyponatremia	fluid overload	<ul style="list-style-type: none"> <li>◆ restrict free water</li> <li>◆ Use NS to flush tube and provide hydration instead of water</li> </ul>

Complication	Possible Cause	Suggested Management
<b>Metabolic cont.</b>		
elevated BUN	renal failure excess protein (nitrogen) intake dehydration medications (diuretics, steroids)	<ul style="list-style-type: none"> <li>◆ reassess medications</li> <li>◆ increase free water</li> <li>◆ reassess renal function</li> <li>◆ reassess protein needs</li> </ul>
rapid, excessive weight gain	excess calories, excess fluid, electrolyte imbalance	<ul style="list-style-type: none"> <li>◆ change formula or decrease delivery rate</li> <li>◆ evaluate electrolytes</li> </ul>
insufficient weight gain	inadequate calories	◆ change formula or increase delivery rate
	malabsorption	◆ change to semi-elemental formula
	catabolic state	◆ provide nutrition support for weight maintenance while addressing medical issues
depression, withdrawal, non-compliance	altered body image	<ul style="list-style-type: none"> <li>◆ encourage socialization at mealtimes</li> <li>◆ provide emotional support</li> </ul>
	loss of oral gratification	<ul style="list-style-type: none"> <li>◆ provide ice chips, sugar-free gum or hard candies</li> <li>◆ provide oral care every shift</li> </ul>

## VI. Parenteral Nutrition

### A. Access Routes and Catheter Placement

#### 1. Access Routes

A variety of locations can serve as sites for catheter insertion including subclavian, internal jugular, external iliac, and cephalic veins. Due to the hypertonicity of parenteral solutions, the catheter tip must always be positioned into the superior vena cava so that the solutions are immediately diluted to tolerable concentrations. Solutions containing 10% or less dextrose (final concentration) plus amino acids (750-900 mOsm/L) can be infused into a peripheral vein (peripheral parenteral nutrition, PPN). However, this practice is associated with a high risk of phlebitis and is therefore reserved for short-term therapy in individuals with robust veins. Simultaneous infusion of lipid emulsions will dilute the osmotic load and thereby improve tolerance to peripherally administered parenteral nutrition. PPN is useful for preserving somatic and visceral protein reserves in patients with limited tolerance of enteral nutrition support.

#### 2. Catheter Placement Requests

Central catheter placement for parenteral nutrition administration is **never** an emergency and, to minimize risk of complications, should only be done under planned circumstances by experienced personnel using strict aseptic technique.

**UWMC:** A variety of catheters can be placed to provide parenteral nutrition. For inpatients, Hohn and Triple Lumen Catheters can be placed by most medical and surgical services. Peripheral Inserted Central Catheters (PICC) are placed by PICC nurses. To schedule a PICC line, phone 548-8702. Hickman and Porta Catheters can be placed in Interventional Radiology or by the General Surgery Service if necessary. For outpatients, catheter placement can

be scheduled by the referring physician. PICC lines can be placed if an advanced clinic visit is scheduled with a PICC nurse (548-8702). For other catheters, complete the “Long-Term Central Venous Access Request” form and page the General Surgery Nurse Coordinator. A pre-anesthesia testing clinic visit may be required and labs including a coagulation profile and CBC are necessary before catheter placement.

**HMC:** Central catheter placement is performed and/or supervised by a medical house staff member with the exception of peripherally inserted central catheters (PICC lines) which are placed by Interventional Radiology. HMC does not use a standardized ordering form for catheter placements.

## **B. Initiating Parenteral Nutrition (PN)**

### **1. Formula Determination**

- a. *Determine energy requirement (see Section IIIB, page 5)*
- b. *Determine protein requirements (see Section IIIC, page 5).*
- c. *Determine fluid requirements (see Section IIID, page 6).*
- d. *Determine the proportion of calories to be provided as intravenous fat.*

Fat emulsion is used as a concentrated calorie source and to prevent essential fatty acid deficiency in patients receiving PN. When used as a source of calories for critically ill patients, 15 to 50 percent of total calories may be supplied as fat. However, to avoid the fat overload syndrome, maximum intravenous fat intake should not exceed 2.5 g/kg/day. The fat overload syndrome is a potentially lethal syndrome, consisting of lipemic serum, massive fat deposition in the lungs and liver, spleen and reticuloendothelial blockade, sepsis, and thrombocytopenia. In order to prevent fatty acid

deficiency, linoleic acid must be provided as 2 to 4 percent of total caloric intake (500 mL 10% lipid emulsion 2 to 3 times/week will supply adequate linoleic acid for most patients). The following maximum infusion rates are recommended: 500cc of 10% lipids over 8 to 12 hours; 500cc of 20% lipids over 12 to 16 hours. For critically ill patients with respiratory compromise, continuous 24 hours fat infusion provides stable energy intake. Due to potential intolerance, plasma triglyceride level should be checked prior to initial infusion and repeated 6 hours after lipid infusion is completed.

Fat emulsion should be used cautiously in patients with severe liver disease or dysfunction, or history of hyperlipidemia (e.g. AIDS) as these patients have a decreased capacity to clear the infused fat.

**e. Determine carbohydrate tolerance.**

Some studies have demonstrated that the rate at which the body can oxidize glucose as energy is limited. Exceeding this limit results in the excess glucose calories being converted into fat which requires energy and places additional stress on the patient. In order to prevent overfeeding with glucose and the resulting complications of hyperglycemia, weight gain, and fatty liver, a "maximum" carbohydrate utilization or tolerance rate should be calculated for all patients who are on total parenteral nutrition. A reasonable estimate is 5 mg CHO/kg/min. For critically ill patients, 3-4 mg CHO/Kg/Min is recommended. The following formula is used to calculate maximum daily CHO tolerance:

$$\text{Maximum CHO (g/d)} = 5 \text{ mg CHO/kg/min} \times \text{IBW}^* \times 1.44^{**}$$

\* IBW = Ideal body weight in kg

\*\* 1.44 = (60 min/hr x 24 hr/day) / (1000 mg/g)

**f. Determine electrolyte requirements.**

The daily electrolyte requirements for most patients can be met by adding one to three standard electrolyte packages to the PN (See Section III, **Table II**, pg 7). The standard electrolyte package available provides the following:

**Standard Parenteral Electrolyte Package**

Electrolyte	Amount
sodium	25 mEq
potassium	40.6 mEq
calcium	5 mEq
magnesium	8 mEq
acetate	33.5 mEq
gluconate	5 mEq
chloride	40.6 mEq

Phosphorus is NOT included in the standard electrolyte package and must be added separately. PN solutions typically contain more phosphate than calcium (as great as 6:1 molar ratio). Cramps may result from excessive phosphate administration. The solubility of calcium in PN solutions is limited by formation of calcium phosphate and carbonate, as well as magnesium salts. Unfortunately, calcium solubility is unpredictable because it depends upon factors such as the commercial sources of the PN components, the order of mixing the PN components, the solution pH, and temperature and storage conditions. In clinical practice, adherence to pharmacy recommendations on the PN order sheets rarely results in precipitation of calcium salts. Furthermore, infusion of large doses of calcium in PN solutions may cause precipitate formation.

Single electrolyte formulations are available in injectable form for individualizing patient prescriptions. Non-standard electrolyte formulations need to be designed with a balance of cations and anions. Acetate is not a “routine” component of a PN prescription as it may result in iatrogenic metabolic

alkalosis. Clinical Pharmacists are available to assist with determining electrolyte requirements of individual patients. Potassium replacement in patients with renal impairment must be done cautiously.

**g. Determine vitamin requirements.**

It is recommended that all adult PN patients, except those in renal failure, be supplemented daily with a standard multivitamin package. The standard vitamin package available provides the following:

**Standard Parenteral Multivitamin Package\***

Vitamin	Amount
Vitamin A	3300 IU
Vitamin D	200 IU
Vitamin E	10 IU
Vitamin C	100 mg
Thiamine (B <sub>1</sub> )	3 mg
Riboflavin (B <sub>2</sub> )	3.6 mg
Niacin (B <sub>3</sub> )	40 mg
Pyridoxine (B <sub>6</sub> )	4 mg
Pantothenic acid	15 mg
Folic Acid	400 mcg
Biotin	60 mcg
Vitamin B <sub>12</sub>	5 mcg

\* Meets AMA Nutritional Advisory Group and FDA Recommended Allowances

In chronic renal failure, intake of vitamins A and D should be restricted. A vitamin B + C complex is available for PN administration to patients with chronic renal failure. Additional ascorbic acid (500 - 1000 mg) may be added directly to PN solutions for meeting the increased vitamin C requirements of wound healing during critical illness or in the post-op period. Additional folic acid (1 mg/day) can be added

directly to PN solutions to meet the increased requirements of pregnancy, or of accelerated red blood cell production in patients with macrocytic anemia. Vitamin K (phytonadione) at 10 mg/week should be given to maintain prothrombin times within the normal range. Vitamin K may be given orally, IM, IV, SQ, or added to PN solution. Patients who are currently receiving warfarin should not be given vitamin K supplementation.

***h. Determine trace element requirements.***

The trace elements zinc, copper, chromium, manganese, iodine, iron, and selenium must be provided in PN to prevent clinical deficiency. It is recommended that all adult PN patients be supplemented daily with a standard trace element package. The standard trace element package available provides the following:

***Standard Parenteral Trace Elements Package***

Trace element	Amount
zinc	5 mg
copper	1 mg
manganese	0.5 mg
chromium	10 mcg
selenium	60 mcg
iodide	75 mcg

Iron is not included in the standard trace element package. Iron is not routinely added to PN solutions because it may alter the stability of other PN components. Furthermore, iron stores are usually sufficient to avoid the need for supplementation during short term use of PN. A parenteral form of iron (iron dextran) is available for intramuscular or intravenous administration to iron-deficient patients who are unable to be supplemented enterally. Iron dextran has caused adverse reactions in a few patients requiring interruption or discontinuation of the infusion. Call the Pharmacy for intravenous iron dextran administration guidelines.

Additional individual trace elements may be added to PN solutions for patients with high metabolic or replacement needs and to treat suspected or diagnosed single trace element deficiencies. Patients with gastrointestinal fluid losses may have increased zinc requirements and should receive additional zinc in their PN. Add to PN daily 5 to 10 mg zinc/L of small bowel fluid loss and 15 to 20 mg zinc/kg stool or ileostomy output. An additional 10 to 20 mg of chromium may be added daily to PN for patients with intestinal losses in excess of one liter. The following trace elements are routinely available to addition to PN solutions:

***Single Parenteral Trace Element Formulations***

Trace element	Amount
chromium	0.004 mg/mL
copper	0.4 mg/mL
manganese	0.1 mg/mL
selenium	0.04 mg/mL
iodide	0.1 mg/mL
zinc	1 mg/ml

Trace element supplementation for patients with liver failure or biliary obstruction should be limited to zinc, since copper and manganese are eliminated from the body by biliary excretion. Zinc and chromium are eliminated by renal excretion and should be administered cautiously to patients with renal dysfunction to avoid toxicity.

***i. Determine if additional additives are necessary, considering especially:***

- ◆ H2-antagonist (e.g. ranitidine)
- ◆ insulin—should be given preferably by separate drip until calorie delivery is stable and insulin requirements are known.

## 2. Writing Parenteral Nutrition Orders

- a. Confirm central catheter placement by X-ray **before** writing PN orders.
- b. Order baseline and routine labs (see Section VII, page 39)
- c. Order routine nursing monitoring parameters (see Section VII, page 37)
- d. Complete PN order form by checking appropriate boxes to select solution components and additives to meet the patient's estimated requirements. Note that orders for PN at the UWMC and HMC are written based on **initial** concentrations of the dextrose and amino acid solutions used. For PN solutions, all components including calorie, protein, fat, fluid, mineral, vitamin, electrolyte, and trace element intakes **must be prescribed daily**.

### ***An Example for Writing the PN Prescription***

A patient requires 2200 kcal and 100 g of protein.

For the *protein requirement* simply check off one liter of 10 percent crystalline amino acids (CAA). This provides 100 g protein and 400 kcal, leaving a caloric balance of 1800 kcal to be provided by carbohydrate and fat.

The minimal *fat requirement* should be considered next. This is 8 percent of total kcal (4 percent as linoleic acid)—that is,  $2200 \text{ kcal} \times 0.08 = 176 \text{ kcal/day}$ , or  $7 \times 176 = 1232 \text{ kcal/week}$ . One bottle of 10 percent lipid is 550 kcal. Therefore, the fat requirement can be met approximately with two bottles of 10 percent lipid/week (i.e.,  $2 \times 550 \text{ kcal} = 1100 \text{ kcal/week}$ ). Keep it simple! Check off the appropriate lipid order in the box on the request form.

The *caloric balance* is now 1800 minus 176 kcal = 1624 kcal to be provided as glucose. The caloric density of dextrose is 3.4 kcal/g. Thus,  $1624 \text{ kcal} / 3.4 \text{ kcal/g dextrose} = 478 \text{ g dextrose}$ . Again, keep it simple! This is approximately one liter of 50 percent dextrose (“dextrose” is actually glucose monohydrate). Check to verify that this amount of glucose is within the patient’s calculated maximum carbohydrate tolerance (see Section VI part B, page 22). For example, if the patient’s IBW is 70 kg, their carbohydrate tolerance is approximately  $70 \text{ kg} \times 5 \text{ mg CHO/kg/min} \times 1.44 \text{ (min/d)} / (\text{mg/g}) = 504 \text{ gm/d}$ . Check off one liter of 50 percent dextrose on the order sheet.

Complete the remaining parts of the order sheet, checking off trace elements and vitamins, as well as 10 mg vitamin K/week. If additional volume is required, add sterile water to the prescription. Electrolytes should be added as 1 package/L. Custom additions can be made as necessary. Phosphate should be the final item ordered; in a patient with normal renal function, phosphate should be added as 10 mmol/1000 kcal dextrose. Select the appropriate volume bag (1, 2, or 3 liters) and rate, keeping in mind that the prescription is to cover a 24 hour period.

### **C. Administration Guidelines and Formulations**

PN (dextrose and amino acid) should be initiated at a rate of 25 to 50 mL per hour and advanced at 8 to 12 hour intervals as tolerated, until fluid volume and caloric goals are attained. In the hospitalized patient, PN is generally administered by continuous infusion over 24 hours. Cyclic PN, in which the entire volume of daily PN solution is infused over a period of 8 to 16 hours, is typically used to prepare patients for home PN administration. Advantages include an infusion-free period for the patient to resume other activities and access for administration of incompatible medications.

At both UWMC and HMC, dextrose (glucose) is the major calorie source for PN solutions. It is available in 5, 10, 20, 25, 35, 50, and 70 percent concentrations. Most patients will tolerate 1 L of D5O/24h without complications. Glucose tolerance is highly variable, however, and difficult to predict from clinical criteria. D70W is the most calorically dense dextrose solution currently available (1190 kcal/500 cc VS 1000 kcal/500 cc 20% lipid emulsion) and is useful as a calorie source in fluid-restricted patients and patients with high metabolic (energy) demands such as trauma/burns.

Lipid emulsion is an alternative calorie source in patients with glucose intolerance or poor CO<sub>2</sub> clearance (CO<sub>2</sub> production from fat oxidation is only 70 percent that of glucose oxidation; however, avoiding excessive calorie delivery is typically more effective than adjusting carbohydrate intake).

Protein is supplied in PN as 5.5, 8.5, 10 or 15 percent solutions of crystalline amino acids (CAA). Routine use of special amino acid products such as branched chain amino acids and essential amino acids is discouraged because they are of unproven efficacy in most clinical situations and they are expensive. The caloric density of crystalline amino acids is 4 kcal/g. The nitrogen content of crystalline amino acids is 5 percent higher than that found in the typical diet due to the select mixture of amino acids. At UWMC and HMC, the g nitrogen in 1L of 15, 10, 8.5, and 5.5 percent amino acid solutions is 25.2 g, 16.8 g, 14.3 g, and 9.3 g, respectively.

#### **D. Medications and Parenteral Nutrition**

Administration of medications via PN may be beneficial when there is limited venous access and/or the patient is fluid restricted. The major problem associated with the addition of medications to PN is the potential for incompatibilities. The following issues should be considered if a medication is to be added to PN.

- ◆ Certain medications should not be mixed with any PN if intermittent infusion is necessary to achieve therapeutic serum levels (i.e. antibiotics).
- ◆ Medications that require a precise rate of infusion (i.e. cardiovascular agents) are not recommended to be added to PN solution.
- ◆ Doses of a medication cannot be readily adjusted once combined with the PN.
- ◆ Adding alkaline medications to PN admixtures may increase the potential for calcium-phosphate incompatibilities.
- ◆ Medication must be chemically stable in PN solution for over 24 hours.

Medications routinely added to PN solutions include: H-2 antagonists (e.g. ranitidine) and insulin.

The use of Y-site or piggyback drug delivery has helped prevent or avoid drug compatibility problems. The contact time of multiple solutions being administered via Y-site is short, often in the range of 15-20 minutes. There are many studies documenting the compatibility of PN and medications when administered via Y-site injection. Call the IV pharmacy for a complete list of medications that are compatible with PN and lipids.

### **E. Complications of Parenteral Nutrition**

In spite of improvements in the delivery of PN, this mode of nutrition support is associated with technical, septic and metabolic complications that warrant pursuit of enteral nutrition support when feasible. Technical complications associated with PN include: air embolism, subclavian artery puncture/hematoma/laceration, pneumothorax, hemothorax, carotid artery injury, thromboembolism, catheter embolism, catheter malposition, Horner's syndrome,

brachial plexus injury, and phrenic nerve paralysis. Septic complications associated with PN include: catheter infection, catheter tunnel infection, and sepsis. **Table IV** lists potential metabolic complications of PN and offers suggestions for intervention.

**Table IV: Metabolic Complications of PN**

Complication	Possible Cause	Suggested Management
Dehydration	Inadequate fluid support; unaccounted fluid loss (e.g. diarrhea, fistulae, persistent high fever).	Start second infusion of appropriate fluid, such as D5W, 1/2NS, NS. Re-estimate fluid requirement and adjust PN accordingly.
Overhydration	Excess fluid administration; compromised renal or cardiac function.	Consider D70 (can't use with PPN) or 20% lipid as calorie source. Initiate diuretics. Limit volume.
Alkalosis	Inadequate K to compensate for cellular uptake during glucose transport; excessive GI or renal K losses. Inadequate Cl <sup>-</sup> in patients undergoing gastric decompression.	Add KCl to PN. Assure adequate hydration. Discontinue acetate.
Acidosis	Excessive renal or GI losses of base; excessive Cl <sup>-</sup> in PN.	Rule out DKA and sepsis. Add acetate to PN.
Hypocalcemia	Excessive PO <sub>4</sub> salts, low serum albumin. Inadequate Ca in PN.	Slowly increase calcium in PN prescription.
Hypercalcemia	Excessive Ca in PN or administration of vitamin A in patients with renal failure. Can lead to pancreatitis.	Decrease calcium in PN. Ensure adequate hydration. Limit vitamin supplements in patients with renal failure to vitamin C and B vitamins.
Hypomagnesemia	Inadequate Mg in PN; excessive Mg losses; cellular uptake with induction of anabolism (Refeeding Syndrome).	Gradually increase Mg content of PN

<b>Complication</b>	<b>Possible Cause</b>	<b>Suggested Management</b>
Hypophosphatemia	Excess losses (urinary PO <sub>4</sub> in alkalosis, Mg, diabetes mellitus, steroid and diuretic therapy); cellular uptake with induction of anabolism (Refeeding Syndrome).	Increase PO <sub>4</sub> content of PN
Hyperglycemic, hyperosmolar nonketotic coma	Sustained untreated glucose intolerance. Easily prevented by frequent glucose monitoring. 40% mortality rate.	Stop PN. Initiate adequate hydration and insulin drip.
Hyperglycemia	Stress response. Occurs in approximately 25% of cases.	Rule out infection. Decrease carbohydrate in PN. Provide adequate insulin.
Hypoglycemia	Sudden withdrawal of concentrated glucose. More common in children.	Taper PN. Start D10.
Hypercarbia	Excessive calorie or carbohydrate load.	Decrease total calories or CHO load.
Essential fatty acid	Inadequate provision of linoleic acid in PN; release of linoleic deficiency acid from adipose stores prevented by continuous dextrose infusion and associated hyperinsulinemia.	Provide i.v. lipids (minimum 500 mL 10% lipid two times a week). Alternatively, hold dextrose infusion for 24 hours.
Hyperammonemia	Excessive protein load; arginine deficiency (urea cycle); hepatic dysfunction; preformed ammonia in amino acid solution. More common in children.	Decrease protein content of PN. Prescribe lactulose.
Hepatic tissue damage and fat infiltration	Unclear etiology. May be related to excessive glucose or energy administration; L-carnitine deficiency.	Rule out all other causes of liver failure. Increase fat intake relative to CHO.
Cholestasis	Lack of GI stimulation. Sludge present in 50% of patients on PN for 4-6 weeks; resolves with resumption of enteral feeding.	Promote enteral feeding.



## VII. Monitoring Nutrition Therapy

### A. Physical Examination

Malnourished patients should be reexamined regularly to assure abatement of deficiency symptoms following treatment. Weight should be measured daily in acute care patients receiving nutrition support since body weight is a simple, non-invasive monitor of hydration status, and a reasonable predictor of body fat and caloric balance.

### B. Functional Assessment

Repletion of protein and energy reserves is associated with improvement in functional abilities. Patient monitoring is enhanced by a subjective evaluation of grip strength, activity level, and physical endurance.

### C. Laboratory Tests

#### 1. Basic Test Schedule

Laboratory tests are useful for monitoring individual patient responses to parenteral and enteral nutrition support. Laboratory tests are specifically used to assess metabolic state (response to injury or infection, inflammation, starvation-adapted, etc.), protein-energy balance, at-risk micronutrient status, fluid, electrolyte and acid-base balance, and organ function. Selection of laboratory tests and testing schedules necessarily depend on individual patient circumstances. A basic menu and schedule for laboratory testing is provided in **Table V**.



## 2. Nitrogen Balance

Nitrogen balance (NB) can be useful in determining individual protein requirements. NB is calculated as follows:

$$\text{NB} = \text{Nitrogen Intake} - \text{Nitrogen Output}$$

Nitrogen intake is estimated from protein intake where g nitrogen equals g intact protein/6.25 or g crystalline amino acids/6.0. Nitrogen output is assessed as 24-hr total urinary nitrogen + 2 g nitrogen to account for normal losses via feces, skin, etc. One gram of nitrogen should be added to nitrogen output for each 500 mL of diarrhea, fistula, or gastric output (any collectable body fluid can be analyzed for total nitrogen content if extra-urinary losses are in question). To accurately assess NB a steady metabolic state needs to be achieved. Therefore, at least 3 days of steady nutrient intake are desirable prior to obtaining the TUN. Ideally, three consecutive 24 hour urine collections are analyzed and averaged for a best estimate of nitrogen output. Careful and complete urine collection and intake records are imperative.

*Example NB calculation:*

Intake Data: 125 g parenteral amino acids

Output Data: 24 hr TUN = 15 g nitrogen

Normal extraordinary losses = 2 g nitrogen

1 L fistula output/d = 2 g nitrogen

$$\text{NB} = (125/5.95) - (15 + 2 + 2) = 21 - 19 = +2 \text{ g nitrogen}$$

Nitrogen equilibrium (NB = 0) is the goal for weight maintenance in healthy individuals. A positive nitrogen balance of 2.4 g nitrogen per day is recommended for growth and other anabolic processes such as wound healing. It is unusual for patients that are critically ill (septic, MOF, burns or trauma) to achieve positive nitrogen balance.

**Table V. Laboratory Test Recommendations for Patients Receiving Nutrition Therapy**

<b>Parameter</b>	<b>Baseline</b>	<b>Follow up</b>
<b>PN and Tube Feeding</b>		
Mini panel (Na, K, Cl, CO <sub>2</sub> , Glu, BUN, Creat)	x	daily until stable
Albumin	x	weekly
Transferrin	x	weekly
C-Reactive protein	x	as needed
PO <sub>4</sub> <sup>*</sup>	x	q o d until stable
Mg <sup>*</sup>	x	q o d until stable
Vitamin C	x	as needed
Zinc	x	as needed
24 hour TUN	as needed	as needed

Table IV continued on next page

<b>Table V. Laboratory Test Recommendations for Patients Receiving Nutrition Therapy... (Continued)</b>		
<b>Parameter</b>	<b>Baseline</b>	<b>Follow up</b>
PN only		
Chemsticks	x	q 6 hr
iCa	x	q o d until stable
Alk Phos	x	weekly
AST	x	weekly
Tbili	x	weekly
Triglyceride	x	as needed
PT	x	as needed

\* Monitor daily until stable in malnourished patients.

### 3. Protein-Energy Balance Markers

Several plasma proteins of hepatic origin have been suggested as dynamic indices of protein-energy balance. During periods of inadequate dietary protein or energy, a reduction in hepatic synthesis and secretion of these proteins causes plasma levels to fall. Reinstitution of an adequate diet induces protein synthesis, returning plasma concentrations to normal. Transthyretin (TTHY, previously known as prealbumin) is the currently preferred plasma protein for monitoring protein-energy balance. TTHY has a biological half-life of 2 days allowing it to rapidly respond to recent changes in nutrient balance. Transferrin (half-life = 8-10 days) and retinol binding protein (half-life = 12 h) can also reflect protein-energy balance, but are confounded by other nutritional factors (transferrin levels increase in iron deficiency and retinol binding protein levels decrease in vitamin A deficiency).

### 4. Evaluating Acid/Base Balance

The routine lab tests recommended for monitoring nutrition support provide information useful in the evaluation of acid-base status. A serum bicarbonate concentration is obtained with the “mini-panel” and a venous pH with ionized calcium. Venous pH parallels arterial pH, provided the patient is not in shock or shunting blood from the arterial to the venous system. A mild decrease in serum bicarbonate (but  $>20$  mEq/dL) or venous pH (but  $>7.30$ ) does not merit an exhaustive diagnostic workup.

## **5. Vitamins and Minerals**

A wide variety of laboratory tests for evaluating vitamin and mineral status are available through the Department of Laboratory Medicine (see **Appendices D and H**). In general, these tests are used to confirm suspected nutrient deficiencies. Additionally, some micronutrients that are involved in wound healing or immune function (e.g. vitamin C, copper and zinc) may need to be monitored to assure the adequacy of supplementation.

## **6. Liver Dysfunction**

Liver dysfunction can occur in refeeding malnourished patients as well as in routine PN therapy. Liver function tests, therefore, must be monitored periodically. Abnormal results may indicate the need to modify PN prescriptions (decrease calories, alter lipid versus glucose intake) or to discontinue PN.

## **D. Short Gut Syndrome**

Patients with an absence of the terminal ileum should be evaluated for fat soluble vitamin and fat malabsorption by measurement of either serum beta-carotene concentration and/or 72 hr quantitative fecal fat. Patients with resected ileum are at risk for vitamin B12 malabsorption requiring i.m. or aerosol supplementation and periodic serum vitamin B12 monitoring.

## **E. Refeeding Syndrome**



Starved or severely malnourished patients can undergo life-threatening fluid and electrolyte shifts following the initiation of aggressive nutritional support therapies. This phenomenon is known as the “refeeding syndrome” and can occur in patients receiving either enteral or parenteral nutrition support.



## Risk Factors for Refeeding Syndrome

- Anorexia nervosa
- Classic kwashiorkor
- Classic marasmus
- Chronic alcoholism
- Chronic malnutrition–underfeeding
- Prolonged IV hydration
- Prolonged fasting
- Morbid obesity with massive weight loss, prolonged fasting

Although not completely understood, the physiological basis of the “refeeding syndrome” is believed to stem from the following: 1) Carbohydrate repletion and insulin release enhance cellular uptake of glucose, phosphate, potassium and magnesium. Since total body stores of these minerals are depleted, blood levels fall. 2) Rapid expansion of the extracellular fluid volume occurs with carbohydrate refeeding and may predispose patients to fluid overload. 3) The reduction in cardiac mass and high energy phosphate reserves associated with malnutrition lead to cardiac insufficiency during fluid resuscitation. Alterations in cardiac function also occur as a result of severe hypophosphatemia, hypokalemia and hypomagnesemia. 4) Similarly, respiratory muscle, reduced in mass and ATP content by malnutrition, is unable to respond to the increased workload imposed by aggressive nutrition support leading to hypercarbia and in some cases respiratory failure. 5) Alterations in red blood cell shape and function occurs in hypophosphatemia which is believed to contribute to tissue hypoxia and increased respiratory drive. 6) Deficiency of B-vitamins, especially thiamin, are speculated to have a role in the refeeding syndrome since these vitamins are required in carbohydrate metabolism.



To avoid the development of the refeeding syndrome, nutrition support in patients at risk should be increased slowly while assuring adequate amounts of vitamins and minerals. In this situation, it is reasonable to start at 20 kcal/kg for the first three days and then increase to 25 kcal/kg. Carbohydrate in PN should be initiated at 2 mg/kg/minute or 150 to 200 mg/day. Nutrient can gradually be increased and be up to requirements by the end of week one. Organ function, fluid balance and serum electrolytes (especially phosphorus, potassium and magnesium) need to be monitored daily during the first week and less often thereafter.

**Appendix A  
COMMON DRUG AND NUTRIENT INTERACTIONS**

<b>Therapeutic Class</b>	<b>Drug</b>	<b>Nutrient Interaction</b>
Alcohols	Ethanol	Reduced absorption of fat, retinol, thiamin, cobalamin and folate; impaired utilization and storage of retinol; increased urinary excretion of zinc and magnesium.
Analgesics	Aspirin	Increased urinary excretion of ascorbic acid; may cause GI bleeding and subsequent iron deficiency; increased folate and vitamin D requirements.
Antacids	Al or Ca containing	Reduced iron, copper, phosphate and magnesium absorption.
Antibiotics	Penicillins Aminoglycosides Chloramphenicol	Increased urinary excretion of amino acids; reduced intestinal vitamin K and cobalamin synthesis; possible malabsorption of fat, cobalamin, calcium, magnesium and carotenoids.
Anticoagulants	Coumadin	Vitamin K decreases & tocopherol increases drug effect.
Anticonvulsants	Phenobarbital, Phenytoin	Folate antagonists; Increased vitamin D, vitamin K and pyridoxine requirements; Impaired vitamin D metabolism leading to hypomagnesemia.

continued on next page

**Appendix A continued**  
**COMMON DRUG AND NUTRIENT INTERACTIONS**

Antidepressants	Imipramine	hypocalcemia and hypophosphatemia.
Antihypertensives	Hydralazine	May induce riboflavin deficiency; increased appetite. Pyridoxine antagonist; increased urinary excretion of manganese and pyridoxine.
Antimalarials	Pyrimethamine, Sulfadoxine	Folate antagonists.
Antineoplastics	Methotrexate	Folate antagonist; may impair fat, calcium, cobalamin, lactose, folate and carotene absorption.
Antitubercular	Isoniazid	Accelerated metabolism of pyridoxine - subsequent pyridoxine deficiency blocks conversion of tryptophan to niacin leading to niacin deficiency; reduced calcium absorption; reduced conversion of Vitamin D by the liver.
Antiulcer	Cimetidine	Impaired cobalamin absorption.
Cardiac Glycosides	Digoxin	Increased urinary excretion of calcium, magnesium and zinc. Anorexia.
Corticosteroids	Hydrocortisone Prednisone Dexamethasone	Reduced calcium and phosphate absorption; increased urinary calcium, potassium, ascorbic acid, zinc and nitrogen excretion. Increased pyridoxine and vitamin D metabolic requirements.

Diuretics	Furosemide Thiazides	Increased urinary potassium, sodium, chloride, magnesium, zinc and iodine excretion; reduced calcium excretion leading to hypercalcemia and hypophosphatemia with thiazides, increased calcium excretion with furosemide.
	Spirolactone	Increased urinary sodium and chloride; reduced urinary potassium excretion.
Hypocholesterolemic Agents	Cholestyramine Colestipol	Reduced absorption of fat, fat soluble vitamins, calcium, cobalamin, folate.
Laxatives	Bisacodyl Phenolphthalein Mineral Oil	Abuse leads to general malabsorption, steatorrhea and dehydration. Malabsorption of fat soluble vitamins, electrolytes, calcium.
Oral Contraceptives	Conjugated estrogens Ethinyl estradiol Mestranol	Increased folic acid & possibly pyridoxine & ascorbic acid requirements; reduced calcium excretion, altered tryptophan metabolism.
Stimulants	Caffeine	Increased urinary calcium excretion.

continued on next page

**Appendix B**  
**PHYSICAL SIGNS SUGGESTIVE OF NUTRIENT DEFICIENCIES**

<b>Location</b>	<b>Signs/Symptoms</b>	<b>Deficient Nutrient</b>
hair	lack luster, thin, sparse, easily plucked, dyspigmentation, alternating bands light & dark	energy, protein
face	nasolabial seborrhea, diffuse dyspigmentation paleness	riboflavin iron
eyes	pale conjunctiva poor dark adaptation, Bitot's spots, corneal or conjunctival xerosis, papilledema	iron retinol
	angular blepharitis	riboflavin, niacin
	intraocular hemorrhage	ascorbic acid
lips	bilateral angular stomatitis cheilosis	riboflavin
		riboflavin, niacin
tongue	scarlet, raw tongue	niacin
	magenta tongue	riboflavin
	glossitis (beefy red tongue)	folic acid, cobalamin, iron, niacin, riboflavin
	atrophic filiform papilla (lick tongue)	folic acid, cobalamin, iron, niacin, riboflavin
teeth	caries	fluoride
gums	pale spongy, bleeding, receding, swelling	iron ascorbic acid
endocrine system	thyroid enlargement parotid enlargement	iodine protein

skin	purpura hyperpigmentation desquamatory dermatitis follicular hyperkeratosis, xerosis, mosaic dermatosis petechial hemorrhages thickening/pigmentation at pressure points and sun-exposed areas scrotal/vulval dermatosis	vitamin K energy, folic acid, cobalamin, niacin biotin, essential fatty acids, zinc, pyridoxine retinol, essential fatty acids  ascorbic acid niacin  riboflavin
nails	koilonychia (spoon nail), paleness	iron
subcutaneous tissue	fat decreased edema	energy protein, thiamin
musculoskeletal system	wasting craniotabes, frontal bossing, knock-knees, beading of ribs, bow legs, musculoskeletal hemorrhage	energy, protein vitamin D  ascorbic acid
cardiovascular system	cardiomegaly, tachycardia, CHF, wet beriberi	thiamin
nervous system	mental confusion, encephalopathy, calf tenderness, sensory loss, motor weakness, dry beriberi, bilateral absent ankle/knee jerks	thiamin, cobalamin
gastrointestinal system	hepatomegaly	protein

**Appendix C**  
**NUTRITIONAL ASSESSMENT PROTOCOL**

Name \_\_\_\_\_

Date \_\_\_\_\_

Diagnosis \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_

**A. HISTORY**

Actual Body Weight \_\_\_\_\_ kg    UBW \_\_\_\_\_ kg  
Height \_\_\_\_\_ cm    Age \_\_\_\_\_

Weight Change: past 6 mo \_\_\_\_\_ kg = \_\_\_\_\_ % loss  
past 2 wk    increase    no change    decrease

Dietary Intake (relative to normal):  
no change    change: duration = \_\_\_\_\_ weeks

type: suboptimal solids full liquids hypocaloric liquids starvation

Calories \_\_\_\_\_    Minerals \_\_\_\_\_  
Protein \_\_\_\_\_    Vitamins \_\_\_\_\_  
EFA \_\_\_\_\_    Tr Elements \_\_\_\_\_

Capacity for Oral Intake: normal

suboptimal: duration of disability = \_\_\_\_\_ weeks

good potential to return to normal in \_\_\_\_\_ weeks

limited potential to return to normal in \_\_\_\_\_ weeks

permanent GI disability

GI Symptoms (of >2 weeks):

none    nausea    vomiting    diarrhea    anorexia

Functional Capacity:    no dysfunction

dysfunction: duration = \_\_\_\_\_ weeks

type = work    ambulation    bedridden

Status of GI Tract:

intact	contiguous bowel
gastrectomy	ileostomy
small bowel resection	jejunostomy
duodenum _____ cm	colostomy
jejunum _____ cm	obstruction
ileum _____ cm	fistula
ileo-cecal valve	radiation exposure type _____
large bowel resection	last treatment _____
complete	
partial	

Medications:

**B. PHYSICAL** (for each trait specify: 0=normal, 1+=mild, 2+=moderate, 3+=severe)

_____ loss of subcutaneous fat	_____ ascites
_____ pallor	_____ muscle wasting
_____ glossitis	_____ dry, dull hair
_____ ankle edema	_____ cheilosis
_____ petechia	_____ sacral edema
_____ scaly dermatitis	_____ bruisability

**C. LABORATORY**

Albumin \_\_\_\_\_ g/dL                      Transthyretin \_\_\_\_\_ mg/dL

**D. METABOLIC DEMANDS**

no stress                      low stress                      high stress

**E. OVERALL NUTRITION RATING** (select one)

A = well nourished  
B = moderately (or suspected of being) malnourished  
C = severely malnourished

**F. NUTRITION SUPPORT RX:**

PN RX:

Resting energy expenditure \_\_\_\_\_ kcals

TEE \_\_\_\_\_ kcals

Nutrient deficits requiring repletion:

protein/24hr \_\_\_\_\_ gms                      fluid/24hr \_\_\_\_\_ cc

**Appendix D**  
**NUTRITION-RELATED LAB TESTS**

1,25-dihydroxycholecalciferol	carnitine
25-hydroxycholecalciferol	total carotene
amino acids (urine or plasma):	cholesterol (total, HDL, LDL)
alanine	cobalamin (vitamin B12)
aminoadipic acid	copper
amino-n-butyric acid	C-reactive protein
arginine	ceruloplasmin
asparagine	fat, quantitative (stool)
aspartic acid	fat stain (stool)
citrulline	fatty acids: complete profile, total
cystathionine	non-esterified fatty acids,
cystine	essential fatty acids
glutamic acid	ferritin
glutamine	folate (serum or red)
glycine	hemoglobin A1C (glycated
	hemoglobin)
histidine	homocysteine
hydroxyproline	iron (serum, tissue, urine)
isoleucine	magnesium
leucine	nitrogen, total
methionine	phosphate
1-methyl histidine	pyridoxine
3-methyl histidine	retinol (vitamin A)
ornithine	retinol binding protein
phenylalanine	riboflavin (vitamin B2)
phosphoserine	selenium
proline	somatomedin C (IGF-1)
serine	thiamin (vitamin B1)
taurine	tocopherol (vitamin E)
threonine	total iron binding capacity
tryptophan	transferrin and % saturation
valine	transthyretin (prealbumin)
apolipoprotein A1,B100	triglycerides
ascorbic acid (vitamin C)	zinc
	zinc protoporphyrin/heme ratio

## Appendix E

### 1983 VS 1959 METROPOLITAN HEIGHT-WEIGHT TABLES\* FOR MEN.

AGES 25-59, WEIGHT IN LBS (WEIGHT IN KGS), WITHOUT  
SHOES OR CLOTHING

1959 Men / *1983 Men			
Height	Small Frame	Medium Frame	Large Frame
5'1"	106-114	112-123	120-135
*5'1"	123-129 (55.9-58.6)	126-136 (57.3-61.8)	133-145 (60.5-65.9)
5'2"	109-117	115-127	123-138
*5'2"	125-131 (56.8-59.5)	128-138 (58.2-62.7)	135-148 (61.4-67.3)
5'3"	112-120	118-130	126-142
*5'3"	127-133 (57.7-60.5)	130-140 (59.1-63.6)	137-151 (62.3-68.7)
5'4"	115-123	121-133	129-146
*5'4"	129-135 (58.6-61.4)	132-143 (60.0-65.0)	139-155 (63.2-70.5)
5'5"	118-127	124-137	132-150
*5'5"	131-137 (59.5-62.3)	134-146 (60.9-66.4)	141-159 (64.1-72.3)
5'6"	122-131	128-141	136-155
*5'6"	133-140 (60.5-63.6)	137-149 (62.3-67.7)	144-163 (65.5-74.1)
5'7"	126-135	132-146	141-160
*5'7"	135-143 (61.4-65.0)	140-152 (63.6-69.1)	147-167 (66.8-75.9)
5'8"	130-139	136-150	145-164
*5'8"	137-146 (62.3-66.4)	143-155 (65.0-70.5)	150-171 (68.2-77.7)
5'9"	134-144	140-154	149-168
*5'9"	139-149 (63.2-67.7)	146-158 (66.4-71.8)	153-175 (69.5-79.5)
5'10"	138-148	144-159	153-173
*5'10"	141-152 (64.1-69.0)	149-161 (67.7-73.1)	156-179 (70.9-81.4)
5'11"	142-156	148-164	158-178
*5'11"	144-155 (65.4-71.4)	152-165 (69.1-75.0)	159-183 (72.3-83.2)
6'0"	146-156	152-169	162-183
*6'0"	147-159 (66.8-73.2)	155-169 (70.5-76.8)	163-187 (74.1-85.0)
6'1"	150-161	156-174	167-188
*6'1"	150-163 (68.2-74.1)	159-173 (72.3-78.6)	167-192 (75.9-87.3)
6'2"	154-165	161-179	172-193
*6'2"	153-167 (69.5-75.9)	163-177 (74.1-80.5)	171-197 (77.7-89.5)
6'3"	158-169	166-184	176-198
*6'3"	157-172 (71.4-78.2)	167-182 (75.9-82.7)	176-202 (80.0-91.8)

Source: Metropolitan Life Insurance Company

**1983 VS 1959 METROPOLITAN HEIGHT-WEIGHT TABLES\*  
FOR WOMEN, AGES 25-59, WEIGHT IN LBS (WEIGHT IN  
KGS), WITHOUT SHOES OR CLOTHING**

1959 Women / \*1983 Women

Height	Small Frame	Medium Frame	Large Frame
4'8"	89-95	93-104	101-116
4'9"	91-98	95-107	103-119
*4'9"	99-108 (45.0-49.1)	106-118 (48.2-53.6)	115-128 (52.3-58.2)
4'10"	93-101	98-110	106-122
*4'10"	100-110 (45.5-50.0)	108-120 (49.1-54.5)	117-131 (53.2-59.5)
4'11"	96-104	101-113	109-125
*4'11"	101-112 (45.9-50.9)	110-123 (50.0-55.9)	119-134 (54.1-60.9)
5'0"	99-107	104-116	112-128
*5'0"	103-115 (46.8-52.3)	112-126 (50.9-57.3)	122-137 (55.5-62.3)
5'1"	102-110	107-119	115-131
*5'1"	105-118 (47.7-53.6)	115-129 (52.3-58.6)	125-140 (56.8-63.6)
5'2"	105-113	110-123	118-135
*5'2"	108-121 (49.1-55.0)	118-132 (53.6-60.0)	128-143 (58.2-65.0)
5'3"	108-116	113-127	122-139
*5'3"	111-124 (50.5-56.4)	121-135 (55.0-61.4)	131-147 (59.5-66.8)
5'4"	111-120	117-132	126-143
*5'4"	114-127 (51.8-57.7)	124-138 (56.4-62.7)	134-151 (60.9-68.6)
5'5"	115-124	121-136	130-147
*5'5"	117-130 (53.2-59.1)	127-141 (57.7-64.1)	137-155 (62.3-70.5)
5'6"	119-128	125-140	134-151
*5'6"	120-133 (54.5-60.5)	130-144 (59.1-65.5)	140-159 (63.6-72.3)
5'7"	123-132	129-144	138-155
*5'7"	123-136 (55.9-61.8)	133-147 (60.5-66.8)	143-163 (65.0-74.1)
5'8"	127-136	133-148	142-160
*5'8"	126-139 (57.3-63.2)	136-150 (61.8-68.2)	146-167 (66.4-75.9)
5'9"	131-140	137-152	146-165
*5'9"	129-142 (58.6-64.5)	139-153 (63.2-69.5)	149-170 (67.7-77.2)
5'10"	135-144	141-156	150-170
*5'10"	132-145 (60.0-65.9)	142-156 (64.5-70.9)	152-172 (69.1-78.6)
*5'11"	135-148 (61.3-67.3)	145-159 (65.9-72.3)	155-176 (70.5-80.0)

Source: Metropolitan Life Insurance Company

**Appendix F**  
**ENERGY REQUIREMENTS**

**A. Calculated Energy Estimate Based on Body Size and Metabolic Stress**

1. Calculate Basal Energy Expenditure (BEE).  
 BEE refers to the metabolic activity necessary to sustain life (i.e., respiration, pulse, body temperature) and can be estimated using the following equation:

*Harris-Benedict equation:*

**BEE** (kcal/day): Males =  $66.5 + (13.7 \times W) + (5.0 \times H) - (6.8 \times A)$   
 Females =  $655 + (9.6 \times W) + (1.7 \times H) - (4.7 \times A)$

where: W = usual or adjusted weight in kilograms  
 H = height in centimeters  
 A = age in years

2. Calculate Total Energy Expenditure (TEE)  
 TEE can be estimated by multiplying the BEE by a factor that accounts for physical activity and clinical status (see below). Only one factor should be used (i.e. do not add multiple factors). Select the factor that corresponds to the patient's dominant situation. Most patients will require 1.3 - 1.7 times the BEE in total caloric intake or between 30 and 35 kcal/kg. Only rarely do calorie requirements exceed 2.0 x BEE or 40 kcal/kg in any patient. The TEE is adjusted as illness progresses and recovery proceeds to avoid complications of under or over feeding.

**BEE Correction Factors for Physical Activity and Clinical Status\***

<i>Physical Activity</i>	<i>Factor</i>	<i>Clinical Status</i>	<i>Factor</i>
strict bedrest	1.2	fever	1.0 + 0.13/°C
out of bed	1.3	elective surgery	1.0-1.1
shivering/thrashing	1.3	peritonitis	1.2-1.5
quadripareisis	0.8	soft tissue trauma	1.1-1.4
paralysis	0.9	multiple fractures	1.2-1.4
hemiparesis	1.2-1.3	closed head injury	1.5-1.8
		severe infection/sepsis	1.4-1.8
		cancer	1.1-1.3
		COPD	1.2-1.3
		major burns	1.5-2.0
		AIDS	1.5-1.8

\* The factors listed apply to both adult men and women.

**B. Indirect Calorimetry by use of the Metabolic Cart**

\*\* Available at HMC only

Indirect calorimetry is the most accurate method for determining resting energy expenditure (REE) for hospitalized individuals. REE is slightly higher than BEE, accounting for the energy expended by the body at rest while awake. REE is calculated indirectly using measurements of oxygen consumption and CO<sub>2</sub> production according to the following formula:

$$\text{REE (kcal/day)} = [3.9 (\text{VO}_2) + 1.1 (\text{VCO}_2)] \times 1.44$$

where: VO<sub>2</sub> = oxygen consumption (mL/min.)

VCO<sub>2</sub> = carbon dioxide production (mL/min.)

Indirect calorimetry should be reserved for the following patients:

- ◆ hypermetabolic patients (burns, trauma, sepsis, head injury)
- ◆ starvation-adapted or malnourished patients
- ◆ extremely obese patients (≥200% of ideal body weight)
- ◆ patients with non-healing wounds
- ◆ patients with abnormal body composition (multiple sclerosis, cerebral palsy, cystic fibrosis, spinal cord injury, amputations)
- ◆ patients who can benefit from education about appropriate calorie intake.
- ◆ research study patients for whom indirect calorimetry is included in the protocol
- ◆ patients with increased minute ventilation for whom determinations of dead space ( $V_D/V_T$ ) and CO<sub>2</sub> production are informative for ventilation decisions.

The following considerations will help avoid errors and enhance the reliability of metabolic cart testing:

- ◆ Metabolic testing becomes less accurate as FiO<sub>2</sub> increases and results are not clinically useful if FiO<sub>2</sub> ≥ 0.60.
- ◆ Metabolic testing is inaccurate in patients with known system leaks (chest tube, tracheostomy cuff, endotracheal tube leaks).
- ◆ Metabolic tests do not represent steady states until ≥4 hours after hemodialysis or anesthesia.
- ◆ Non-intubated patients on supplemental O<sub>2</sub> can be tested using an external blender. These patients must have an FiO<sub>2</sub> ≥ 0.60 and must be able to tolerate a tight fitting face mask or canopy hood. Accurate tests cannot be obtained in patients with uncuffed tracheostomy tubes an supplemental O<sub>2</sub>.

- ◆ The patient should not be moving. The exception may be the patient who postures or seizes continuously throughout the day, such that movement is more his usual state than non movement.

Specific recommendations for metabolic cart studies:

- ◆ The patient should be NPO or receiving PN or continuous tube feeding for at least 12 hours before testing to assure a stable baseline. For patients who are eating or receiving bolus tube feeds, tests should not be done until 2 hours after the meal/bolus.
- ◆ Patients who are agitated or experiencing pain should be given sedatives/analgesics an hour or more before testing.
- ◆ The environment should be quiet, controlled and at normal room temperature. The patient should not be shivering.
- ◆ Any ventilator alterations should be made 30 minutes or more before metabolic testing.
- ◆ Nursing care, physical and occupational therapies should be completed at least 1 hour before testing.
- ◆ Patients under the canopy hood should not speak during testing because this falsely elevates CO<sub>2</sub> production and precludes equilibrium.
- ◆ Desiccant cartridges of the metabolic cart need to be changed when the color begins to turn pink.

### C. Fick Equation

The Fick equation can be used to calculate energy expenditure in ICU patients who have a pulmonary artery/Swan catheter. Twenty four hour energy expenditure is approximately seven times the VO<sub>2</sub> in mL/min. VO<sub>2</sub> is calculated from cardiac output (CO) in L/min, content of venous O<sub>2</sub> (CvO<sub>2</sub>) in mL/dL, and content of arterial O<sub>2</sub> (CaO<sub>2</sub>) in mL/dL according to the following formula:

Where:  $CaO_2 - CvO_2 = 1.39 \times Hb(g/dL) \times (SaO_2 - SvO_2)$

SaO<sub>2</sub> and SvO<sub>2</sub> are respectively arterial and mixed venous oxygen saturation as a fraction

$VO_2 = [(CaO_2 - CvO_2)] \times 10 \times CO$

VO<sub>2</sub> (in mL/min) x 7 = 24 hour energy expenditure in kcal/day units

Venous blood gases must be determined at approximately the same time as cardiac output. Repeated measurements performed over several days are helpful in increasing the accuracy of the estimate and in determining trends in energy expenditure.



**Appendix G**  
**ESSENTIAL NUTRIENTS IN HUMAN METABOLISM**

*Water*

*Carbohydrates*

*Amino acids*

isoleucine, leucine, lysine, methionine, phenylalanine,  
threonine, tryptophan, valine, histidine

*Fatty acids*

linoleic acid, linolenic acid

*Vitamins*

Fat soluble - retinol (A), 25-hydroxycholecalciferol (D), a-  
tocopherol (E), phylloquinone (vitamin K)



Water soluble - thiamin (B1), riboflavin (B2), pyridoxin (B6),  
niacin (B3), folic acid, cobalamin (B12), biotin, panthothenic  
acid (B5), ascorbic acid (C)

*Minerals*

calcium, chloride, magnesium, phosphate, potassium, sodium

*Trace Elements*

chromium, copper, fluoride, iodine, iron, manganese,  
molybdenum, selenium, zinc



**Appendix H**  
**DIAGNOSIS OF NUTRITIONAL ANEMIAS**

Differential diagnosis of nutritional anemias prior to nutrient supplementation is imperative for prevention of progressive neurological damage, as in the case for untreated vitamin B12 deficiency, or prevention of the undesirable side-effects of unnecessary iron supplementation (e.g. GI distress, iron overload in hemochromatosis gene carriers, increased risk of infection during acute stress). The following table classifies nutritional anemias and anemia of chronic inflammation according to red cell parameters.

<b>Red Cell Index</b>	Iron or Copper Deficiency Anemia (microcytic hypochromic)	Folate or Vitamin B12 Deficiency Anemia (macrocytic normochromic)	Anemia of Chronic Inflammation (normocytic normochromic)
MCV	low	high	normal
MCHC	low	normal	normal
MCH	low	high	normal

To confirm iron deficiency as a cause of microcytic anemia it is recommended to first evaluate the zinc protoporphyrin/heme ratio (ZPP/H). ZPP/H is an inexpensive screening test for iron deficiency. Zinc protoporphyrin is produced during heme synthesis in the developing erythron when iron availability is limited, thus iron deficiency is characterized by an increase in the ZPP/H. Confirmation of iron deficiency following an elevated ZPP/H (>80 umol/mol) requires determination of serum ferritin. Ferritin concentration <20 ng/mL is considered iron deficient. Both ZPP/H and serum ferritin can be elevated by chronic inflammation. Under conditions of chronic inflammation a serum ferritin <70 ng/mL may be considered iron deficient. ZPP/H can also be elevated in lead toxicity and protoporphyria (a rare congenital disease). Serum iron, total iron binding capacity (TIBC) and % transferrin saturation may also be used in the diagnosis of iron deficiency, however, these tests are less predictive of iron stores. Percent saturation is very useful in evaluation of iron overload. The following table outlines the changes in laboratory test results reflecting changes in iron status.

### **Indices of Iron Status**

Test	Iron Overload	Normal	Iron Depletion	Iron-deficient erythropoiesis	Iron-deficiency anemia
Bone Marrow Fe	4+	2-3+	0-1+	0	0
Serum Fe (ug/dL)	>175	115±50	115	<60	<40
TIBC (ug/dL)	<300	330±30	360	390	410
%Transferrin Saturation	>60	35±15	30	<15	<15
Ferritin (ug/L)	>300	100±60	20	10	<10
%Sideroblasts	40-60	40-60	40-60	<10	<10
ZPP/H (umol/mol)	<80	<80	80	>80	>80

Macrocytosis requires evaluation of both folate and vitamin B12 nutriture. The effect of either nutrient deficiency is impairment of genesis and maturation of red blood cells causing large, nucleated cells to be released into the circulation. Both nutrients arrest DNA synthesis by preventing the formation of thymidine monophosphate although folate and vitamin B12 are used at different steps of the synthetic pathway. Folate supplementation can compensate for vitamin B12 deficiency in DNA synthesis reversing macrocytic anemia and thereby masking vitamin B12 deficiency. Undiagnosed vitamin B12 deficiency will result in progressive permanent neurological damage.

One of the earliest clinical signs of both folate and vitamin B12 deficiency is hyper-segmentation of neutrophils. Deficiency should be suspected when >5% of cells have five or more lobes or when any six lobed cells are seen within a random sample of 100 cells. Hyper-segmentation may also occur in uremia, myeloproliferative disorders, myelofibrosis, and as a congenital lesion in 1% of the population.

Plasma vitamin B12 is the available test for evaluating vitamin B12 stores. Plasma vitamin B12 will be elevated in myeloproliferative disorders and hepatic tissue damage. A low plasma vitamin B12 is almost always indicative of deficiency. The cause of vitamin B12 deficiency must be determined for appropriate treatment. Vitamin B12 deficiency can result from either

dietary deficiency or impaired absorption. Pernicious anemia, inadequate secretion of intrinsic factor, achlorhydria, a history of gastric or ileal resections, or diseases associated with malabsorption (e.g. Crohn's disease) may cause impaired vitamin B12 absorption. The Schilling test can be used to distinguish insufficient secretion of intrinsic factor from malabsorption syndromes. In this test radioactive B12 is taken orally, and its urinary excretion is measured over 24 hours. A flushing dose of unlabeled B12 is given with the labeled B12 to saturate liver storage and enhance labeled B12 excretion. Normally, >7% of the labeled B12 is recovered in the urine. If absorption is low, it is necessary to repeat the test with administration of intrinsic factor. The following table outlines the changes in laboratory test results reflecting changes in vitamin B12 status.

### Vitamin B12

Test	Normal	Negative vitamin B12 balance	Vitamin B12 depletion	Vitamin B12 deficient erythro-poiesis	Vitamin B12 deficiency anemia
Hypersegmentation	No	No	No	Yes	Yes
Serum B12	200-900 pg/mL	150-200 pg/mL	100-150 pg/mL	80-100 pg/mL	<80 pg/mL
RBC folate	>160	>160	>160	<140	<140
MCV	Normal	Normal	Normal	Normal	elevated

Red cell folate is the preferred test for evaluation of folate stores. Falsely elevated concentrations are seen in patients with raised reticulocyte counts and low levels occur in vitamin B12 deficiency. Plasma folate can be used to assess status however, it is affected by recent folate intake. The following table outlines the changes in laboratory test results reflecting changes folate status.

<b>Folate</b>						
Test	Normal	Negative folate balance	Folate depletion	Folate deficient erythro-poiesis	Folate deficiency anemia	
Serum folate (ng/mL)	>5	<3	<3	<3	<3	<3
RBC folate (ng/mL)	>200	>200	<160	<120	<100	
Lobe average	<3.5	<3.5	<3.5	>3.5	>3.5	
MCV	Normal	Normal	Normal	Normal	elevated	
Hb	>12	>12	>12	>12	<12	



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