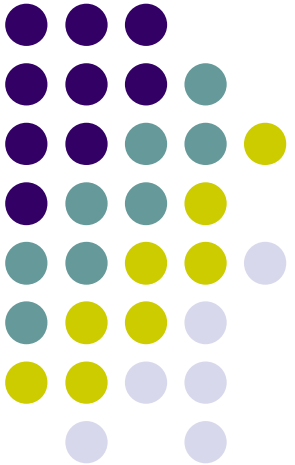
Nutritional support in surgery

Bradulskis S. MD

Kaunas Medical University of Medicine



“Although no one would dispute that drinking and eating are basic requirements of healthy man, there are still who believe that an unhealthy man need do neither”

Editorial: Nourishment in illness
Br J Anaesth 1973;45:935

It is now nearly 70 years since the first report about effect of accidental injury on nitrogen loss.

Cuthbertson DP. Q J Med 1932; 1: 233-246

In 1936 Studley established - postoperative mortality rate patients which lost more than 20% of body weight before operation achieve 30%, without malnutrition - only 3.5%.

Studley HO, JAMA ; 106:458, 1936

Prevalence of Malnutrition in Hospitalized Patients

Medicine	Kyle	2004	1273	43
	Wyszynski	2003	1000	48
	Waitzberg	2002	4000	48
	Kyle	2001	995	38
	Larsson	1993	382	29
	Willard	1990	200	32
	Coats	1983	228	38
	McWhirter	1994	300	45
	Weinsier	1974	134	48
	Robinson	1987	100	56
	Reilly	1988	365	59
	Bistrian	1980	251	44
	x		*9428*	*44*

Prevalence of Malnutrition in Hospitalized Patients

Surgical	Postma	1993	422	23
	Hall	1990	367	29
	Pettigrew	1983	198	32
	McWhirter	1994	200	33
	Bistrian	1974	131	40
	Detsky	1987	202	42
	Reilly	1988	406	48
	Buzby	1980	100	62
	x		*2026*	*41*

Prevalence of Malnutrition in Hospitalized Patients

Geriatric	Wyszynski	2003	466	50-76*
	Kyle	2002	172	61
	Larsson	1990	500	29
	Füllöp	1991	552	34
	Contans	1992	324	37
	Sullivan	1994	110	38
	Shaver	1980	115	85
	x		*2239*	*50*

** >60 y: 50; > 70 y: 53, > 80 y: 77 %*

ECONOMIC IMPACT of MALNUTRITION in 771 HOSPITALIZED PATIENTS

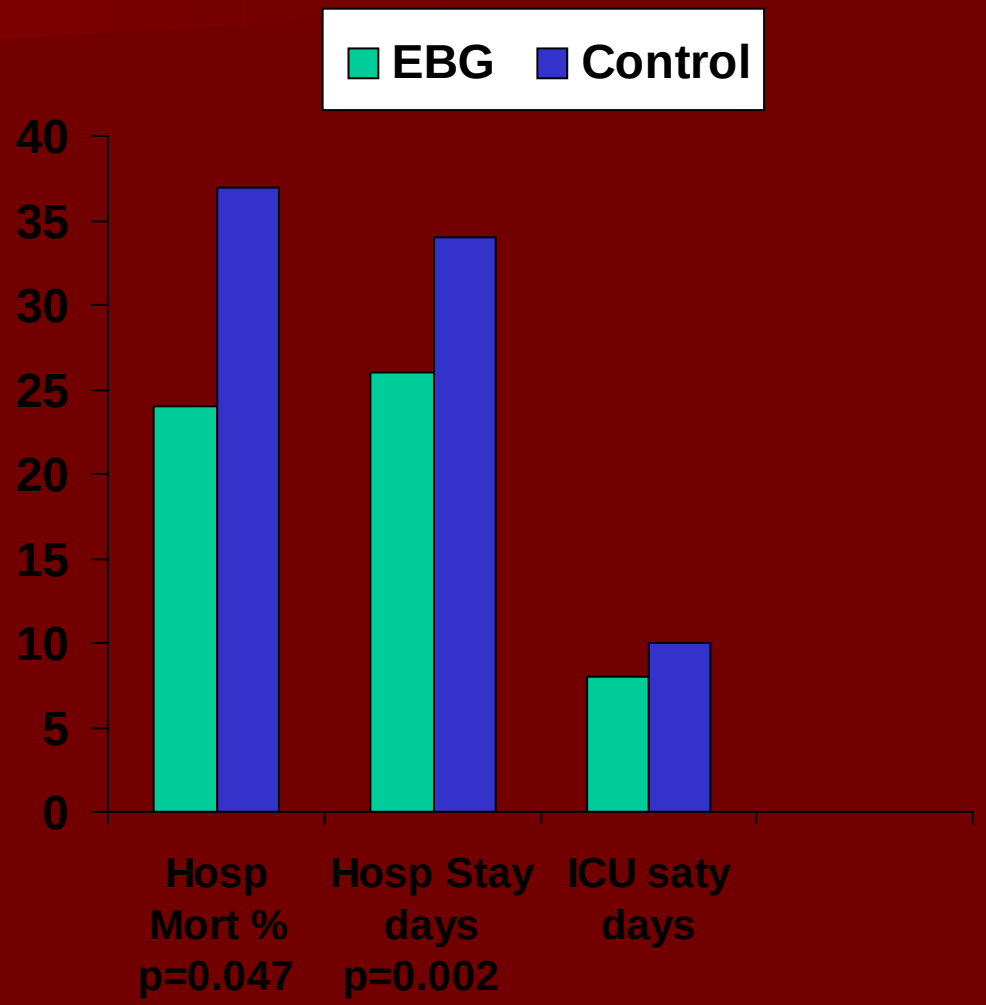
Reilly J.J. et al. J Parent Enteral Nutr 12(4), 371-376, 1988

	p	Malnourished	Well-nourished
All	771	5519 ± 300	3372 ± 138
	0.001		
Medecine	365	2945 ± 242	1783 ± 124
	0.0001		
Surgery	406	7335 ± 513	4579 ± 182
	0.001		*in US$*

Nutrition improve outcome

- 489 ICU patients in 14 hospitals RCT (cluster)
- 7 hospitals - EB guidelines
  - early nutrition
  - enteral preferred
- 7 hospitals - control
- Better outcome
  - increased EN
  - less TPN

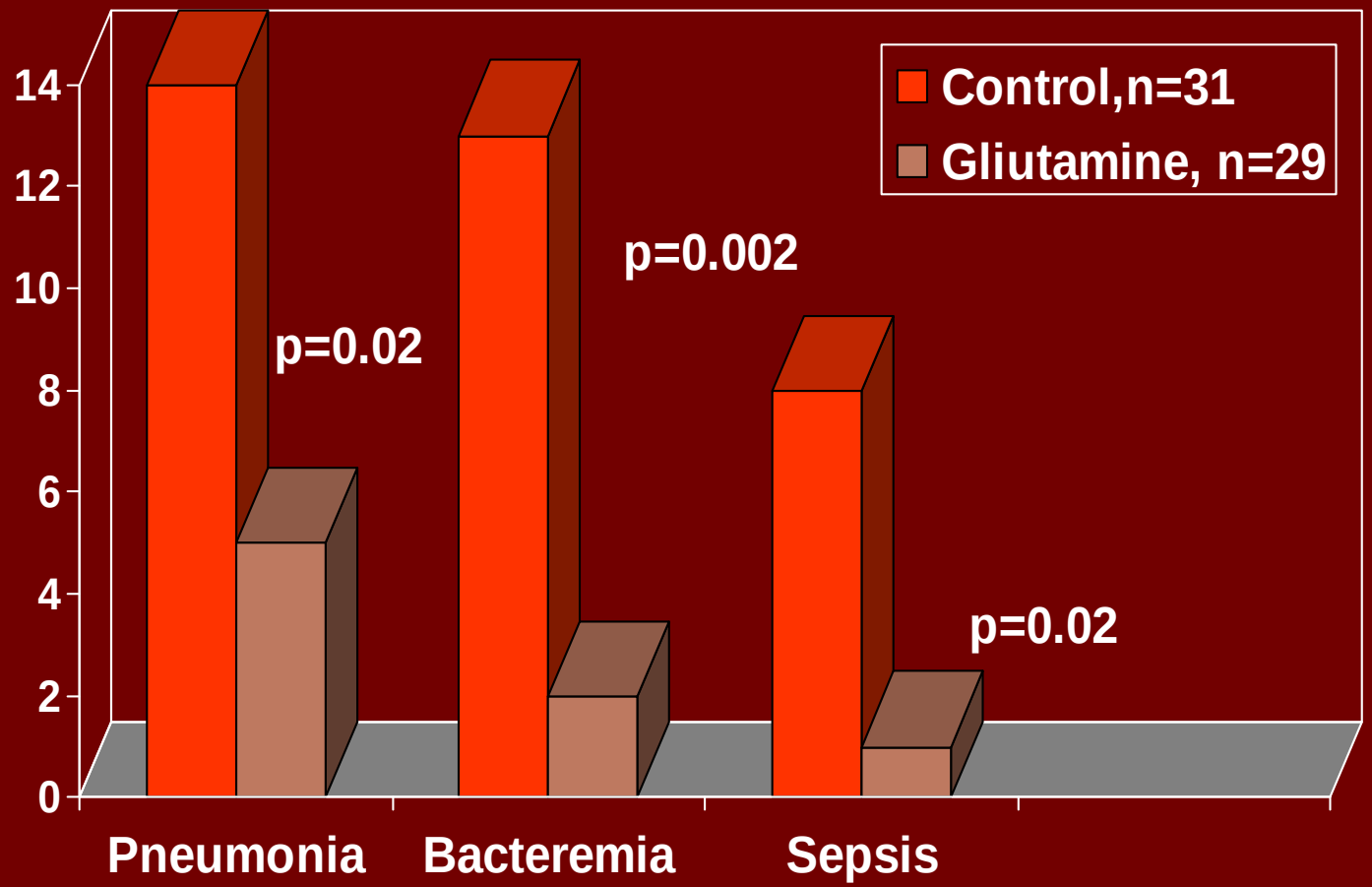
Dois GS et al Am J Respir Crit Care Med 2000; 161: A93



Enteral gliutamine& multitrauma

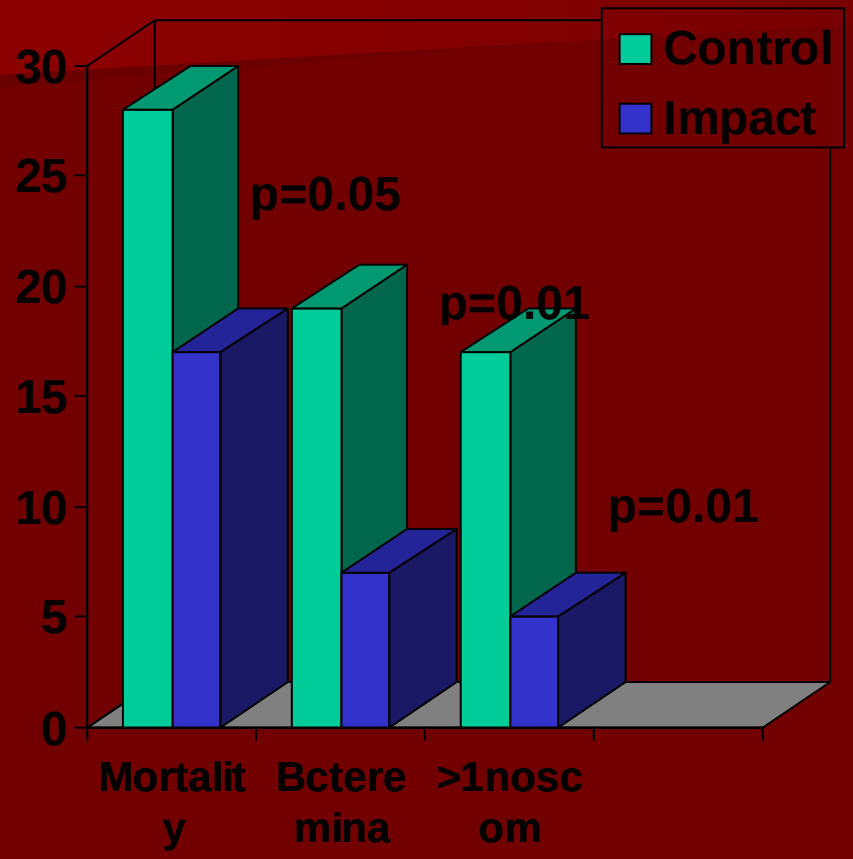
Nasojejunal tube f. < 48hrs, >25g Gliutamine supp.

Houdijk AP, et al. Lancet 1998 Sep 5; 352(9130):772-776



Enteral “Immunonutrition” in the ICU

Galban C et al. Crit Care Med 2000; 28:643-648



- Septic ICU patients

APACHE II > 10

111 with pneumonia

NG,NJ feeding within 36hrs, total calories met by day 4

- 89 Impact v 87 High Protein control

1231v1414kcal/day

equivalent nitrogen intake

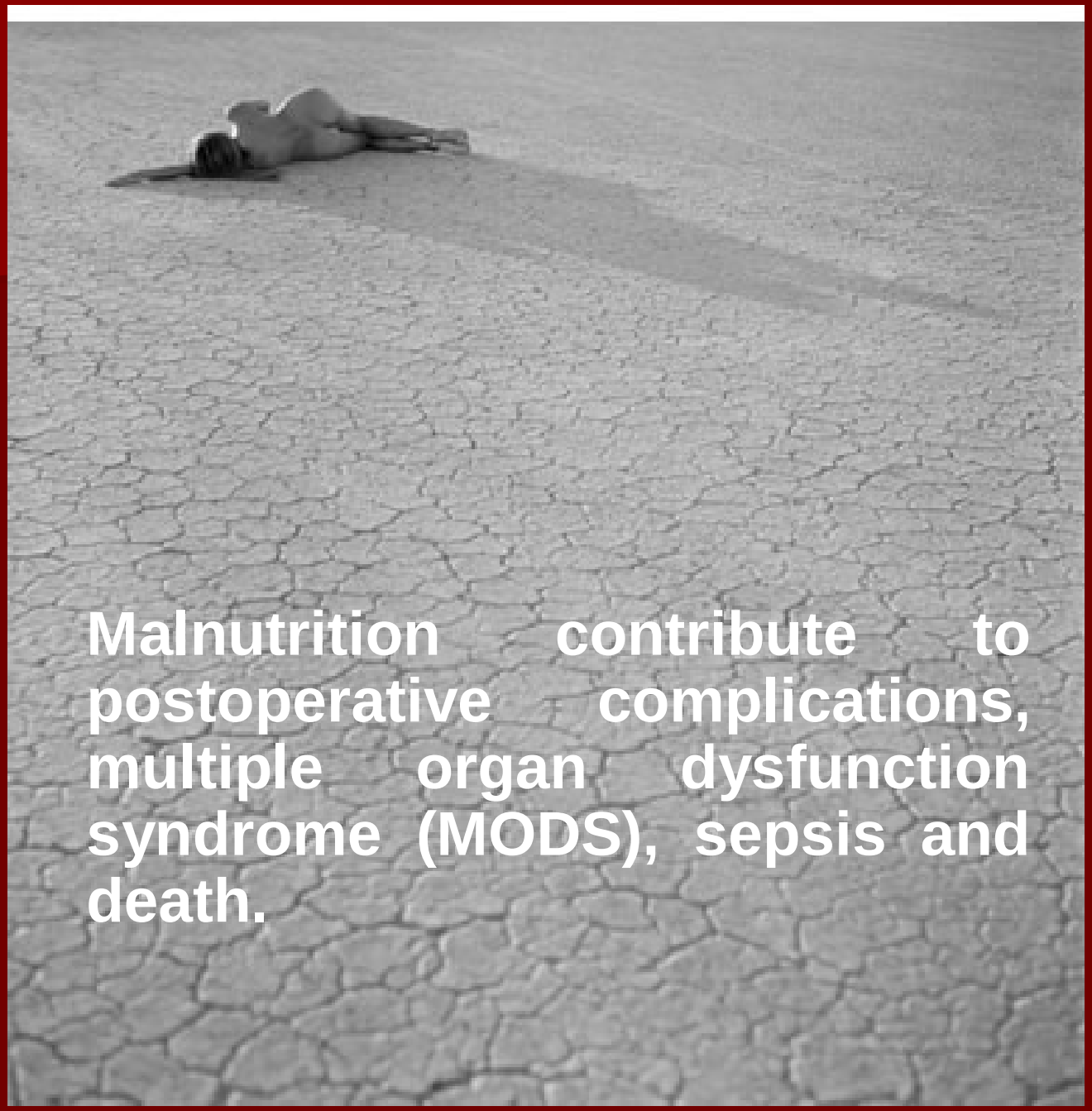
ICU LOS (18.2 v 16.6)

MVD (12.2 v 12.4)

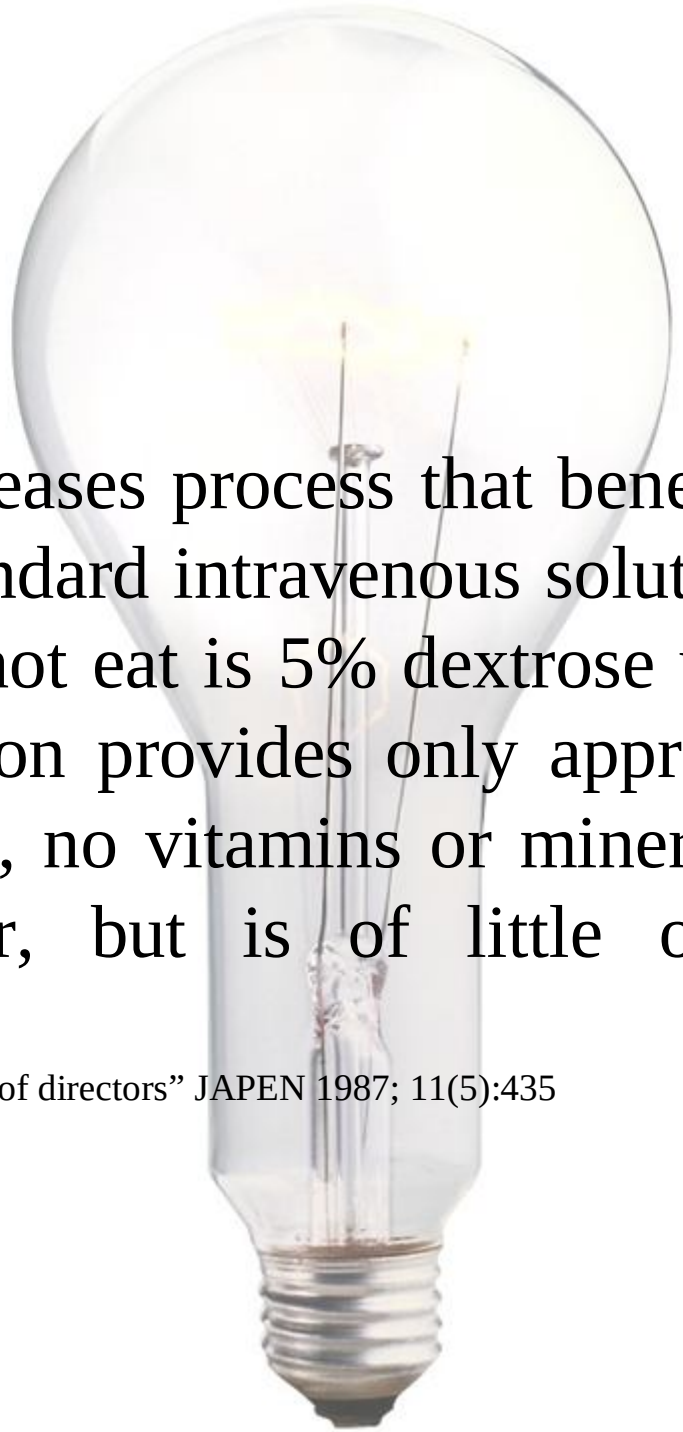
Aggravation of Malnutrition

DURING

the Hospital Stay



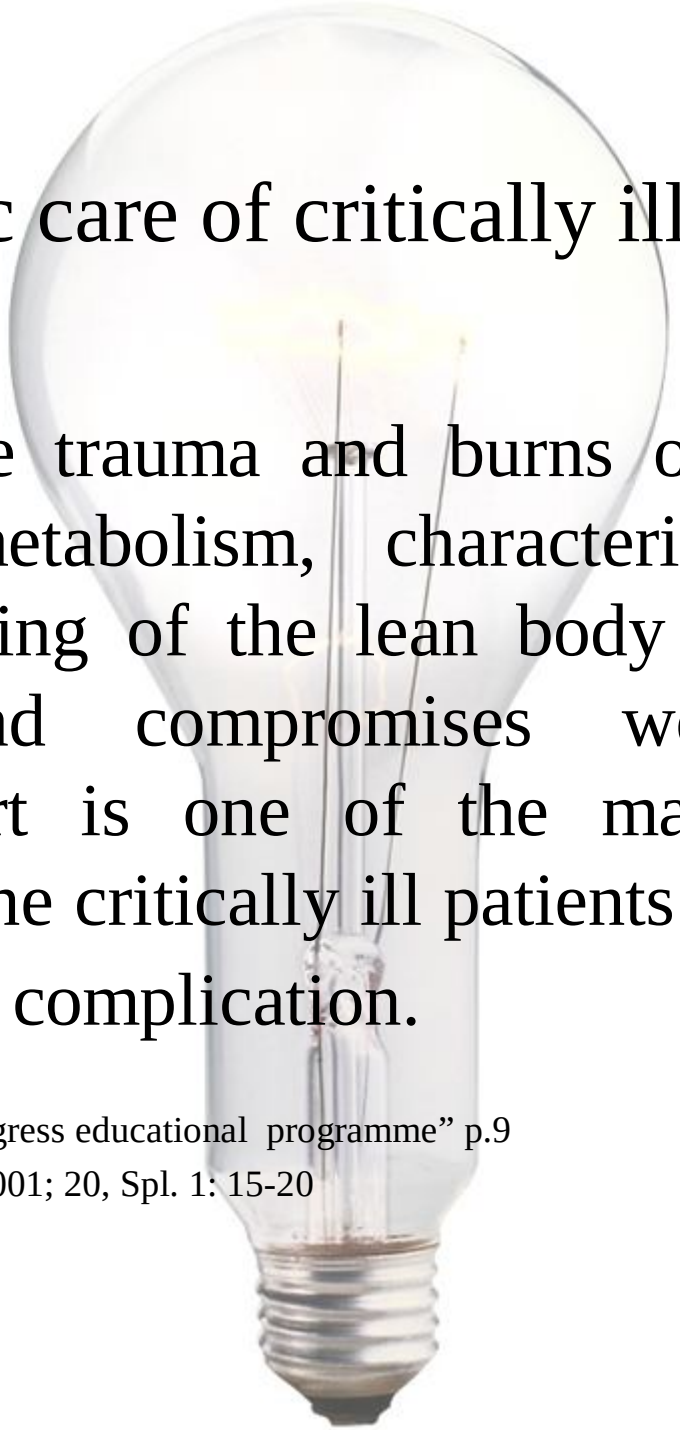
Malnutrition contribute to postoperative complications, multiple organ dysfunction syndrome (MODS), sepsis and death.



There is no diseases process that benefits from starvation. The standard intravenous solutions given patients who cannot eat is 5% dextrose with electrolytes. This solution provides only approximately 500 kcal as glucose, no vitamins or minerals. It provide adequate water, but is of little other benefits.

“Special Report ASPEN board of directors” JAPEN 1987; 11(5):435

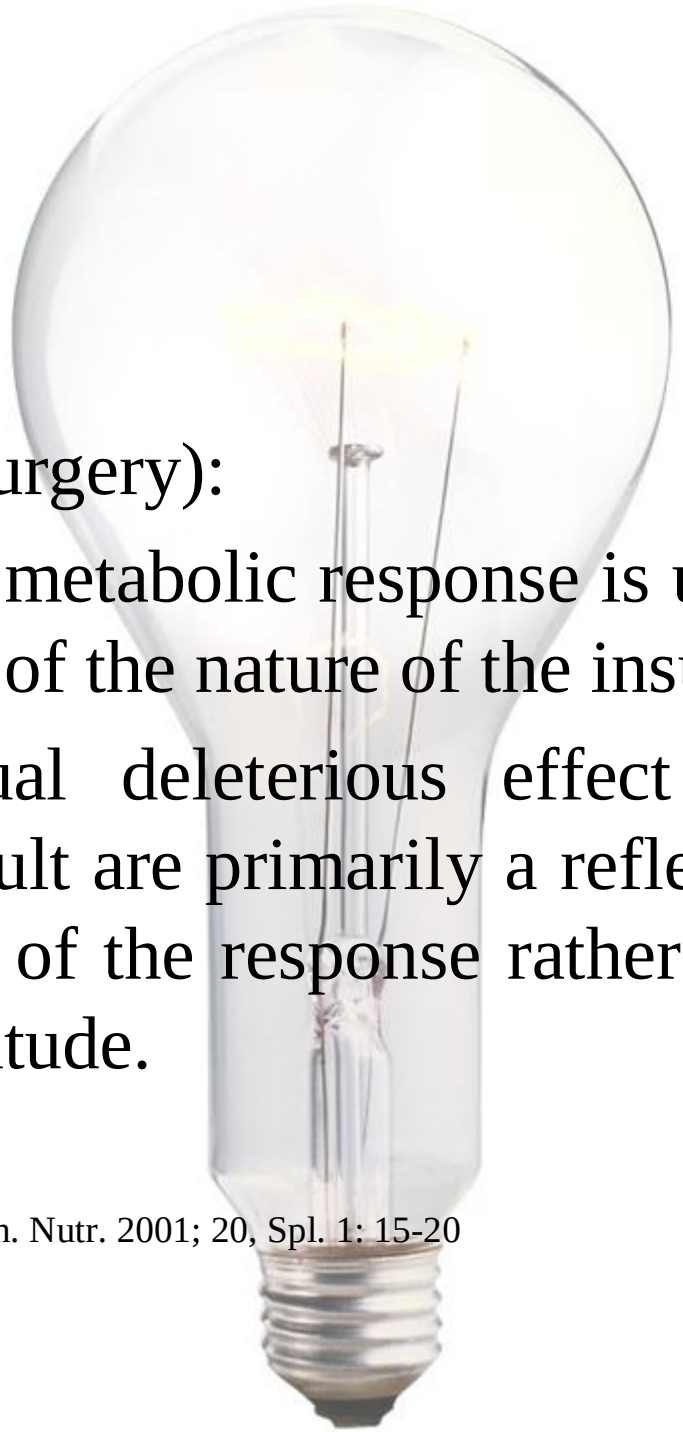
Metabolic care of critically ill patients



Sepsis , multiple trauma and burns often associated with altered metabolism, characterized by sever catabolism, wasting of the lean body mass, immune dysfunction, and compromises wound healing. Nutrition support is one of the mainstays in the management of the critically ill patients and is aimed at minimizing these complication.

Popova T. 2000, "22nd ESPEN congress educational programme" p.9

Reeds PJ., Jahoor F. Clin. Nutr. 2001; 20, Spl. 1: 15-20

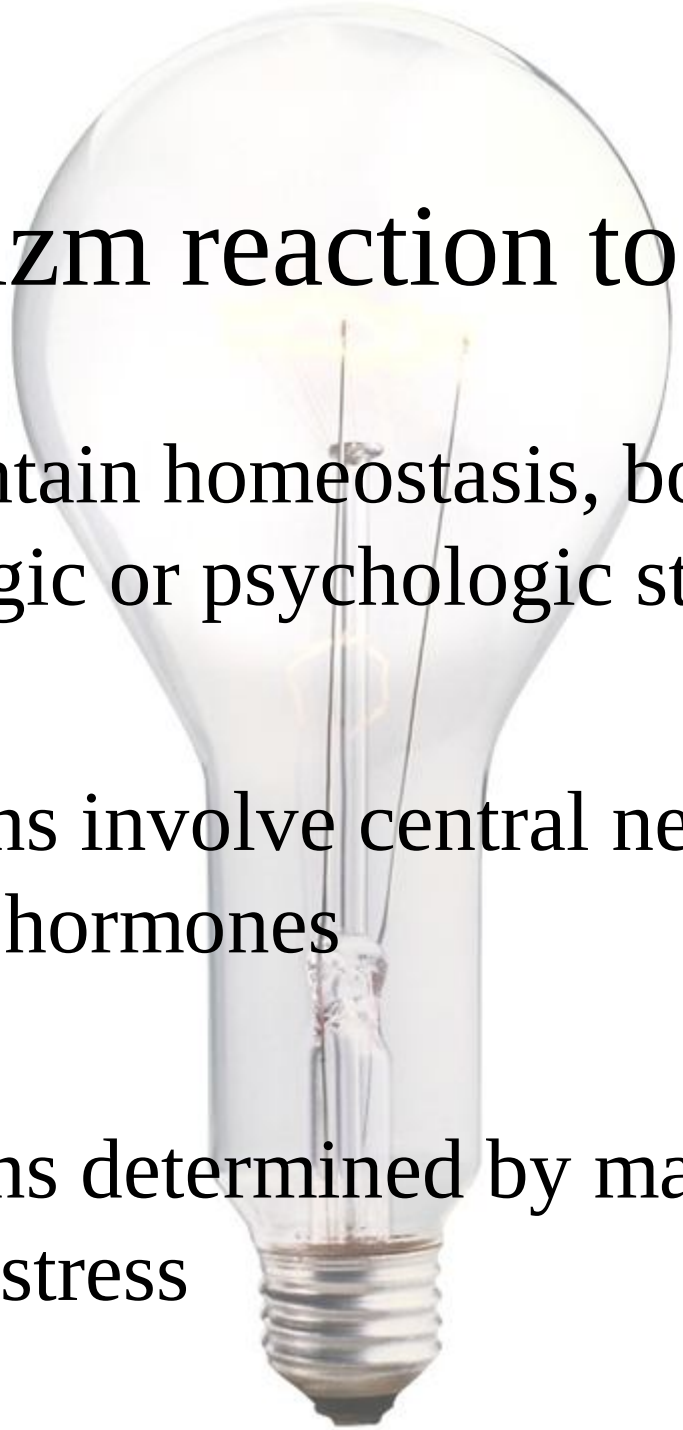


In trauma (surgery):

•the general metabolic response is uniform, inrespective of the nature of the insult

• the eventual deleterious effect of the different insult are primarily a reflection of the duration of the response rather than its initial magnitude.

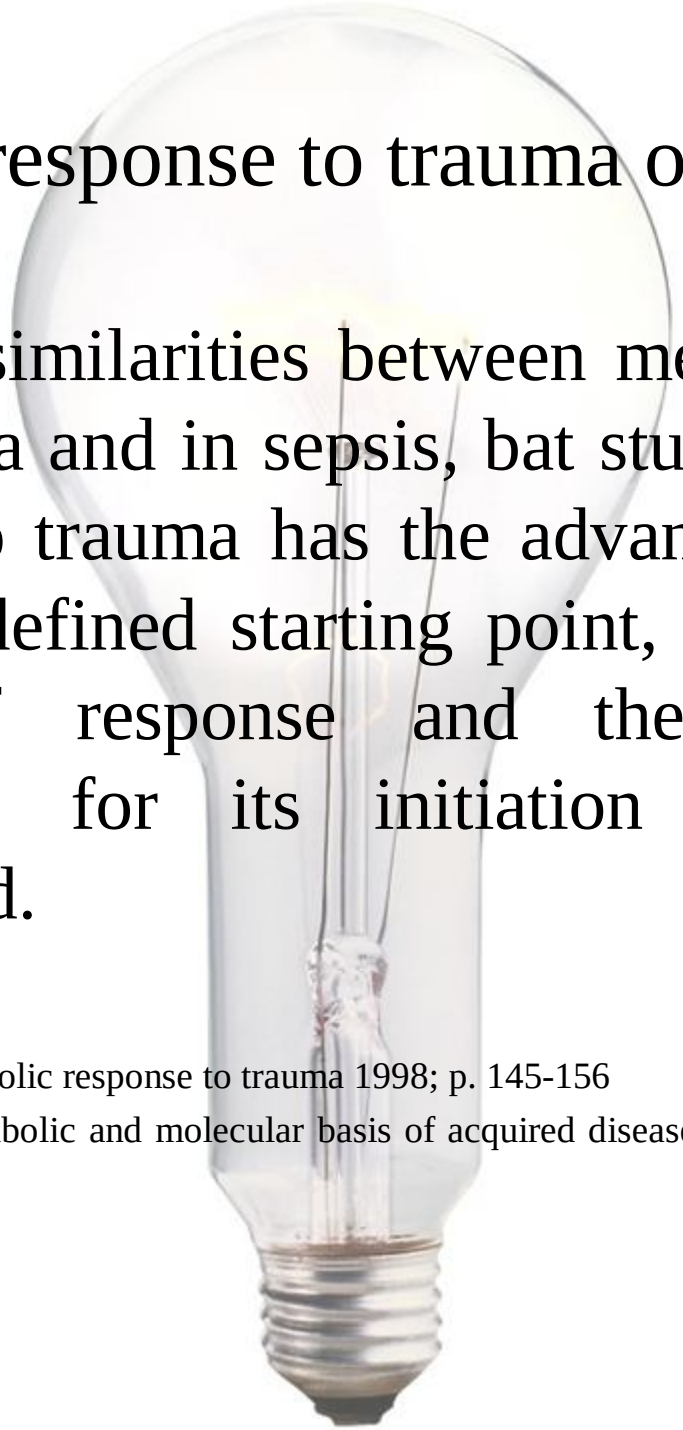
Reeds PJ., Jahoor F. Clin. Nutr. 2001; 20, Spl. 1: 15-20



Organizm reaction to stress

- A. To maintain homeostasis, body responds to physiologic or psychologic stress
- B. Reactions involve central nervous system and hormones
- C. Reactions determined by magnitude and duration of stress

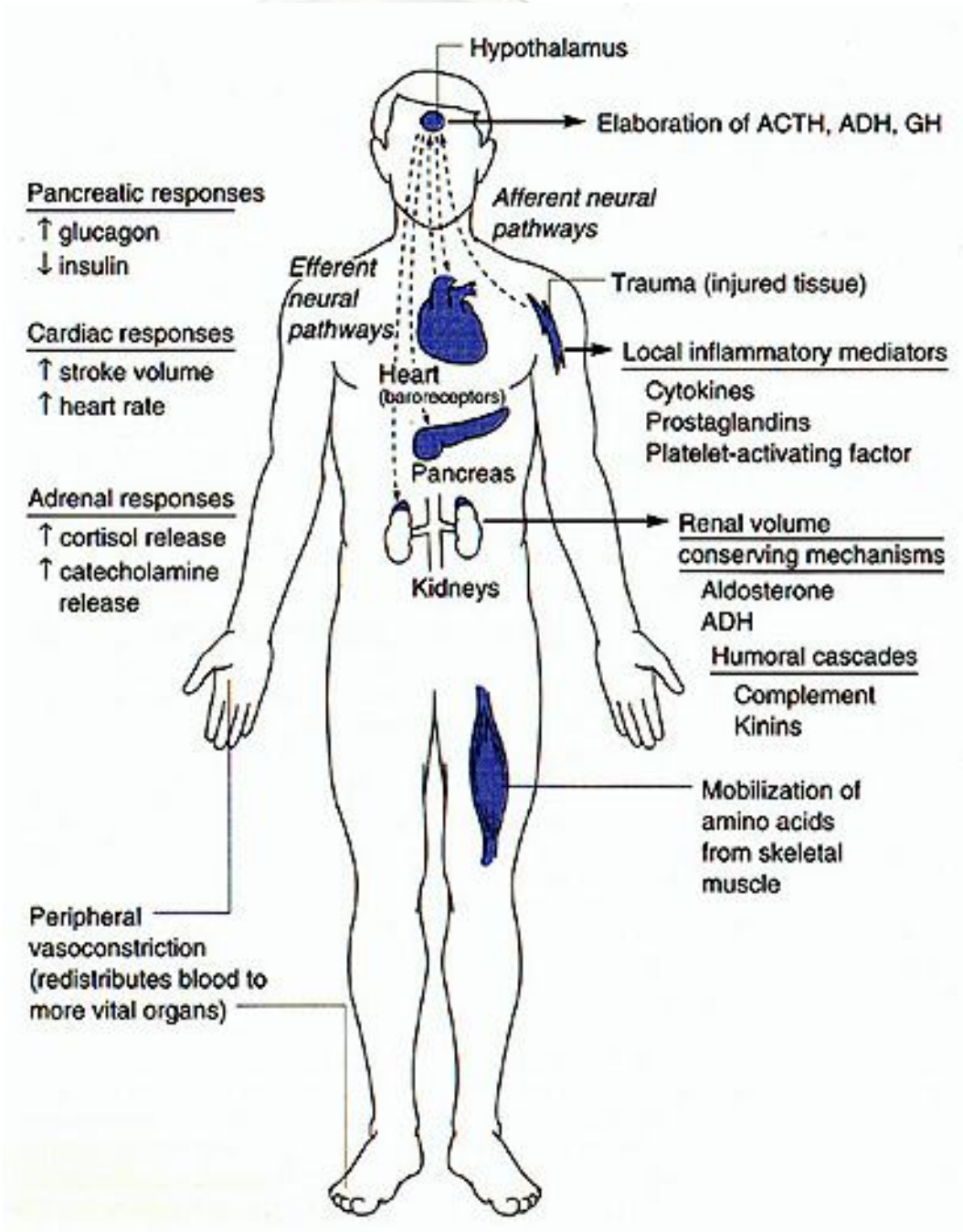
Metabolic response to trauma or infection



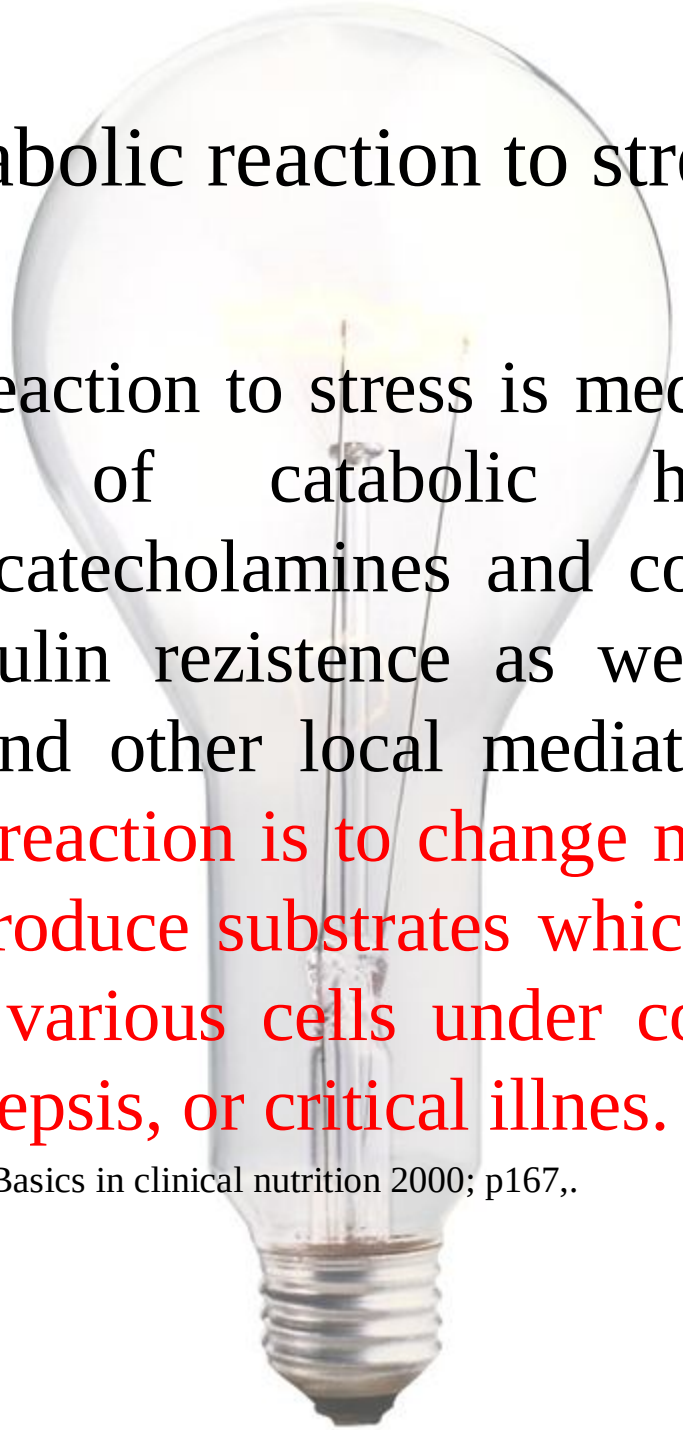
There are similarities between metabolism after trauma and in sepsis, bat study of the response to trauma has the advantage that is clearly defined starting point, the early pattern of response and the factors responsible for its initiation can be investigated.

Arnolds J et al, Metabolic response to trauma 1998; p. 145-156

Barton et al, The metabolic and molecular basis of acquired disease" 1990; vol 1, pp 684-717,


Hypothalamus
Elaboration of ACTH, ADH, GH
Pancreatic responses
↑ glucagon
↓ insulin
Afferent neural pathways
Efferent neural pathways
Trauma (injured tissue)
Cardiac responses
↑ stroke volume
↑ heart rate
Heart
(baroreceptors)
Local inflammatory mediators
Cytokines
Prostaglandins
Platelet-activating factor
Pancreas
Adrenal responses
↑ cortisol release
↑ catecholamine release
Kidneys
Renal volume conserving mechanisms
Aldosterone
ADH
Humoral cascades
Complement
Kinins
Mobilization of amino acids from skeletal muscle
Peripheral vasoconstriction (redistributes blood to more vital organs)

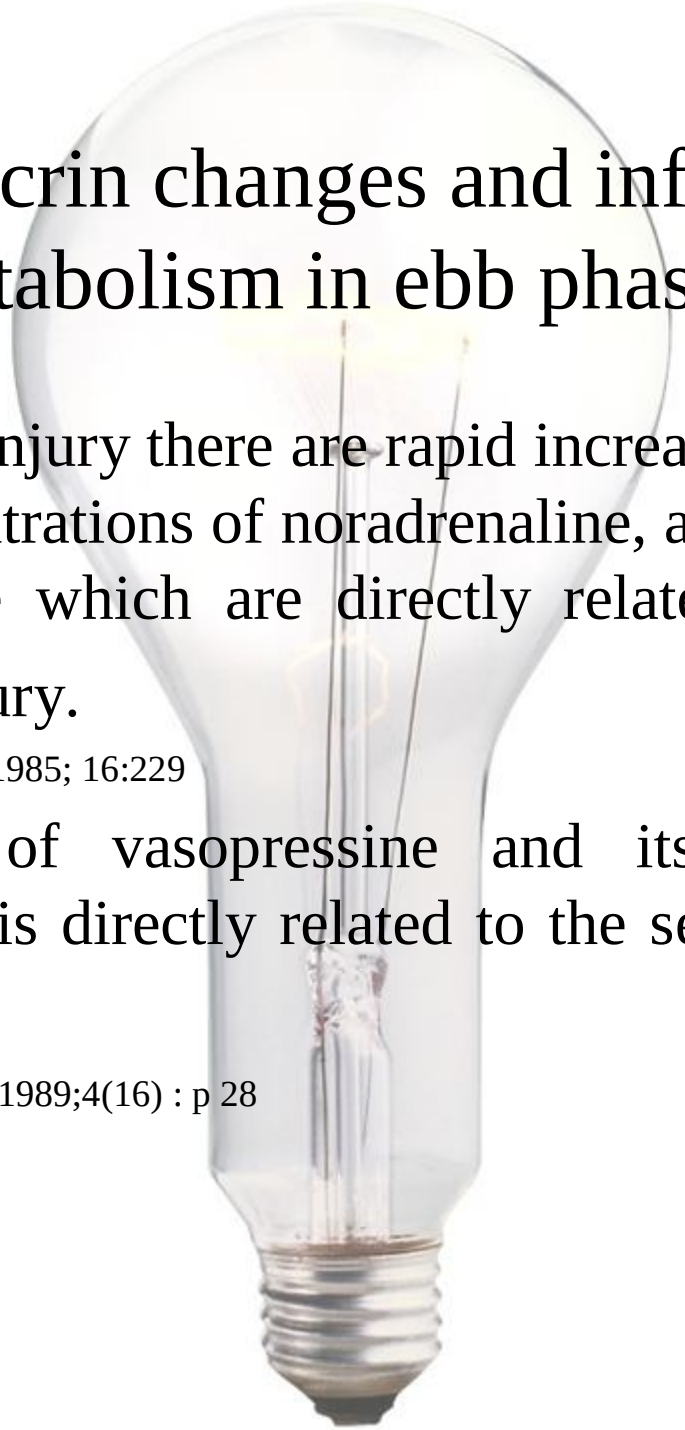
Metabolic reaction to stress



Organism reaction to stress is mediated by stimulation of catabolic hormones (glucagon, catecholamines and corticoids) and by insulin rezistence as well as by cytokines and other local mediators. The aim of this reaction is to change metabolic pathways produce substrates which can be utilized by various cells under conditions of trauma, sepsis, or critical illnes.

Sobotka L, Soeters PB Basics in clinical nutrition 2000; p167,.

Neuroendocrin changes and influence to metabolism in ebb phase



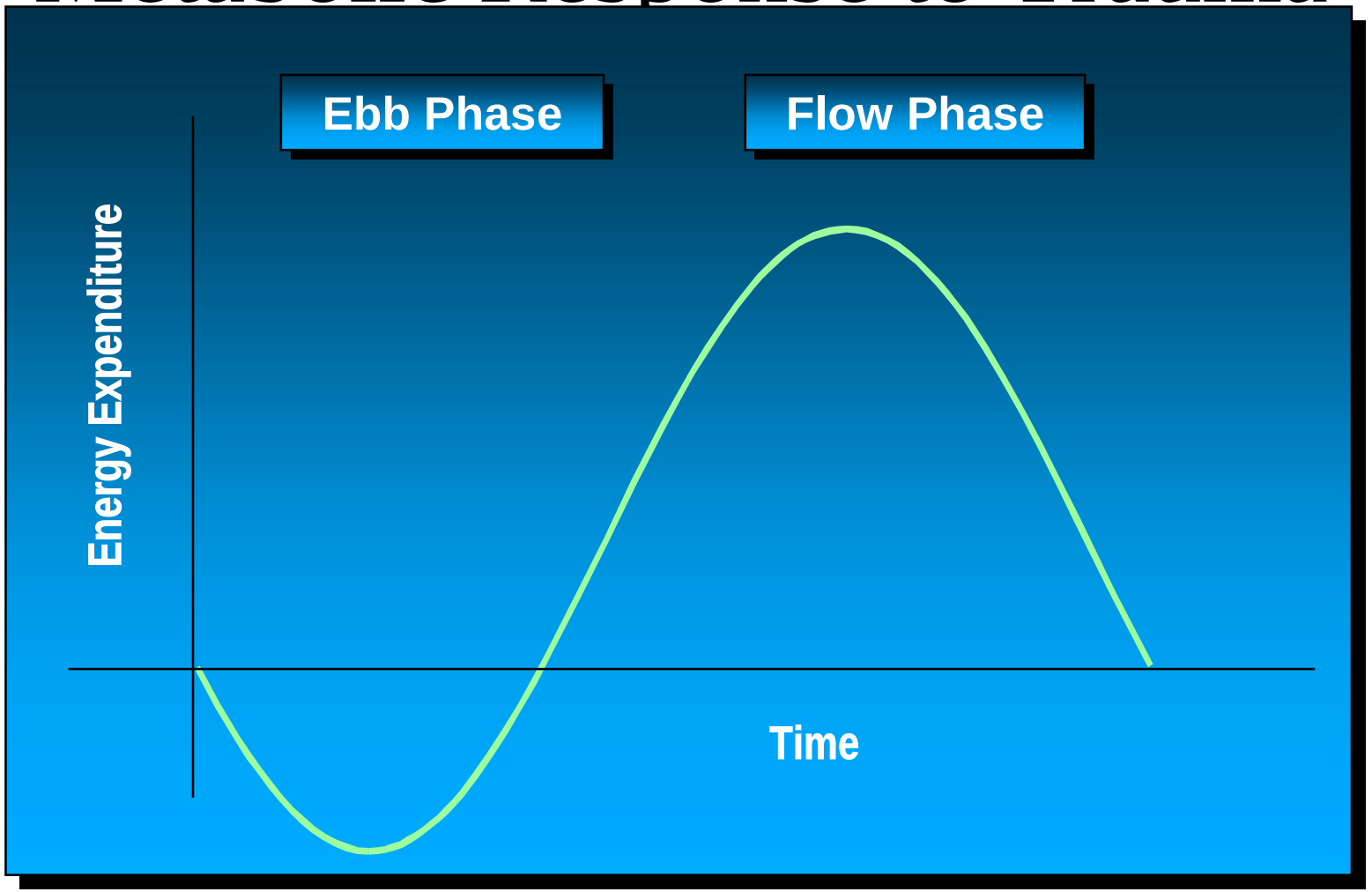
Acutely after injury there are rapid increases in the plasma concentrations of noradrenaline, adrenaline and dopamine which are directly related to the severity of injury.

Frayn et al Cyrc Shock 1985; 16:229

The release of vasopressine and its plasma concentration is directly related to the severity of injury too.

Anderson et al J Physiol 1989;4(16) : p 28

Metabolic Response to Trauma

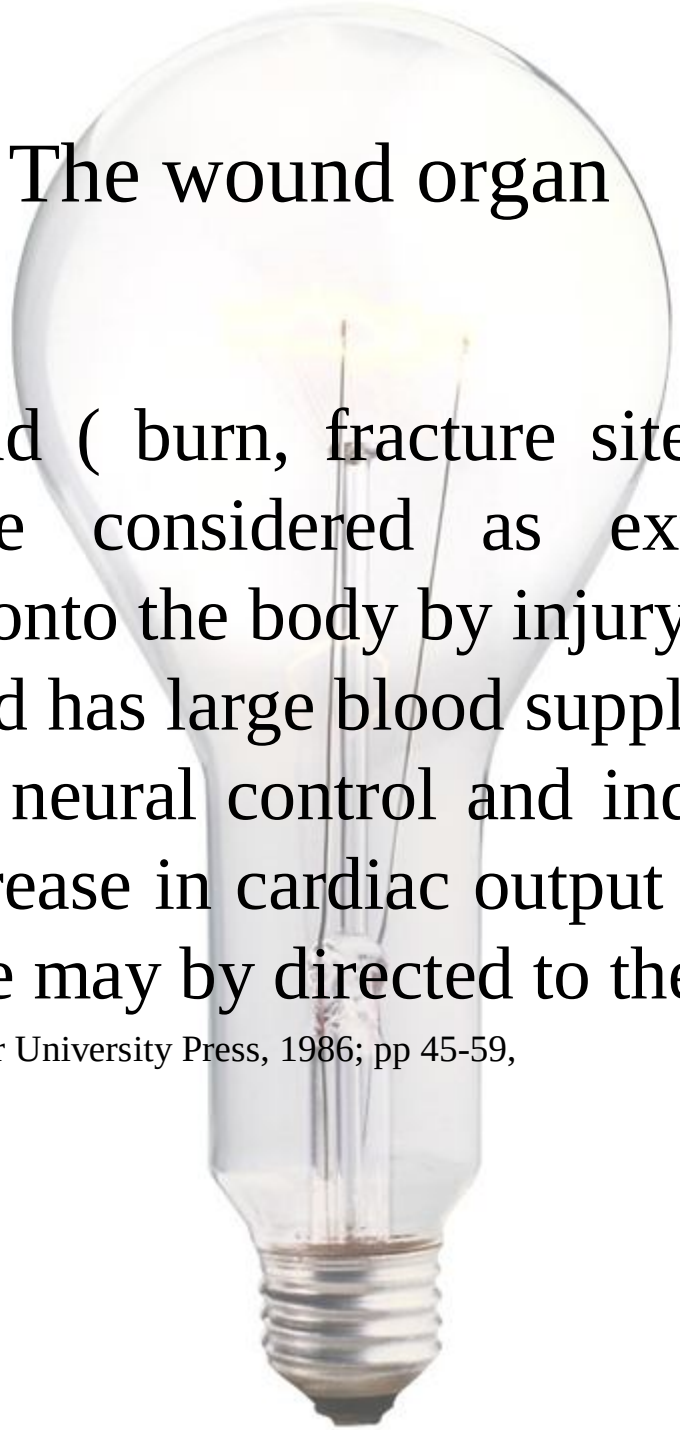


Cutherbertson DP, et al. *Adv Clin Chem* 1969;12:1-55

Table 15-3
Metabolic Responses to Severe Stress

Ebb Phase	Flow Phase
↓Oxygen consumption	↑Oxygen consumption
↓Cardiac output	↑Cardiac output
↓Plasma volume	↑Plasma volume
Hypothermia	Hyperthermia
	↑Nitrogen excretion
↓Insulin levels	Normal or elevated insulin levels
Hyperglycemia	Hyperglycemia
Hypovolemia	
Hypotension	
↑Lactate	Normal lactate
↑Free fatty acids	↑Free fatty acids
↑Catecholamines, glucagon, cortisol	↑Catecholamines, glucagon, cortisol
Insulin resistance	↑Insulin resistance

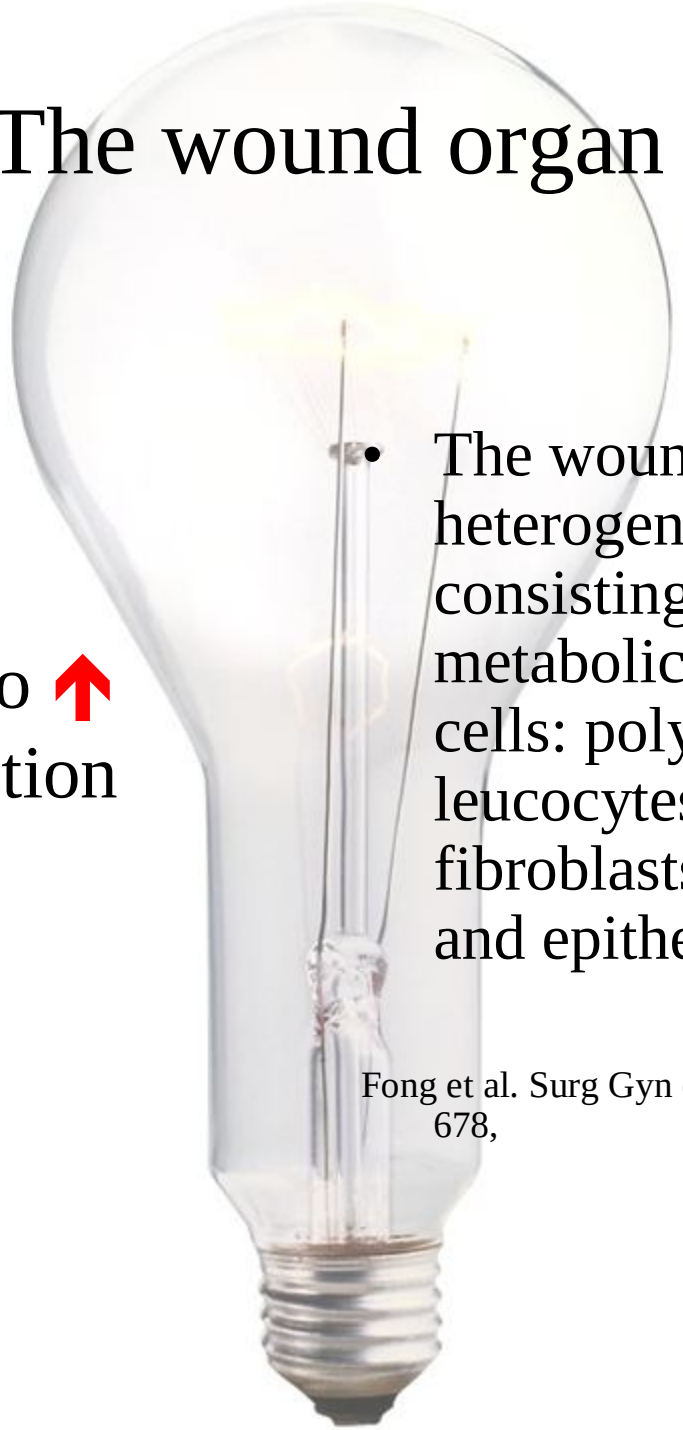
The wound organ



The wound (burn, fracture site, abscess) should be considered as extra organ “grafted” onto the body by injury/infection. The wound has large blood supply which is not under neural control and indeed much of the increase in cardiac output during the flow phase may by directed to the wound.

Wilmore Manchester University Press, 1986; pp 45-59,

The wound organ

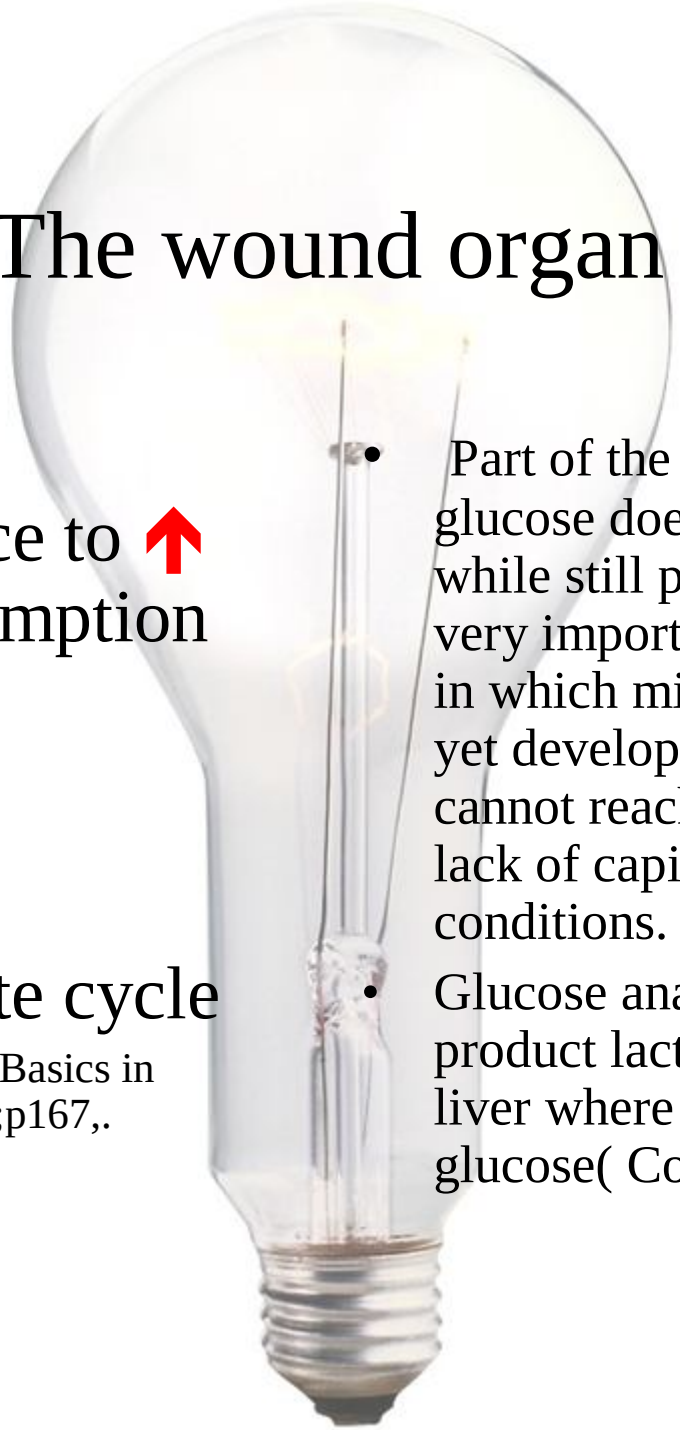


Wound influence to ↑ energy consumption

- The wound is a heterogeneous tissue consisting of metabolically very active cells: polymorph nuclear leucocytes, monocytes, fibroblasts, endothelial and epithelial.

Fong et al. Surg Gyn ecol 1990: Obstet 170:363-678,

The wound organ



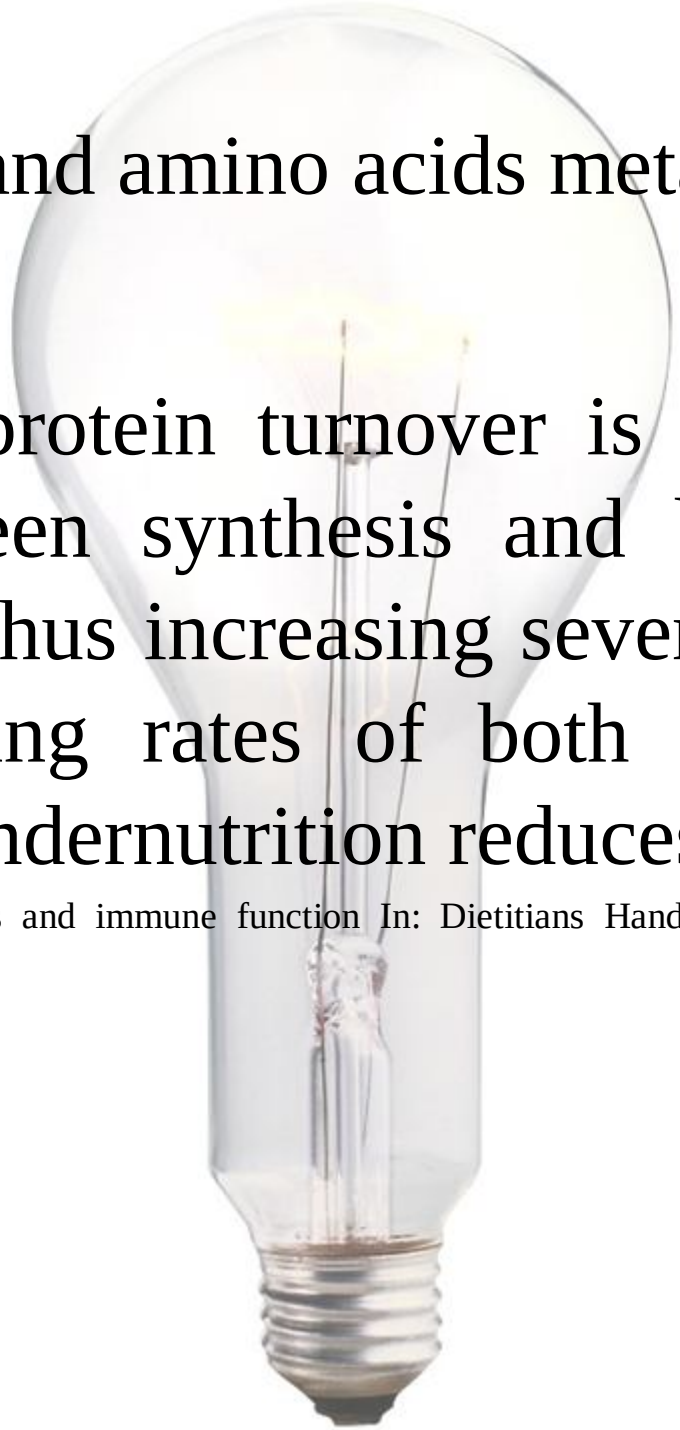
Wound influence to ↑ energy consumption

- Increasing of glucose/lactate cycle

Sobotka L, Soeters PB Basics in clinical nutrition 2000;p167,.

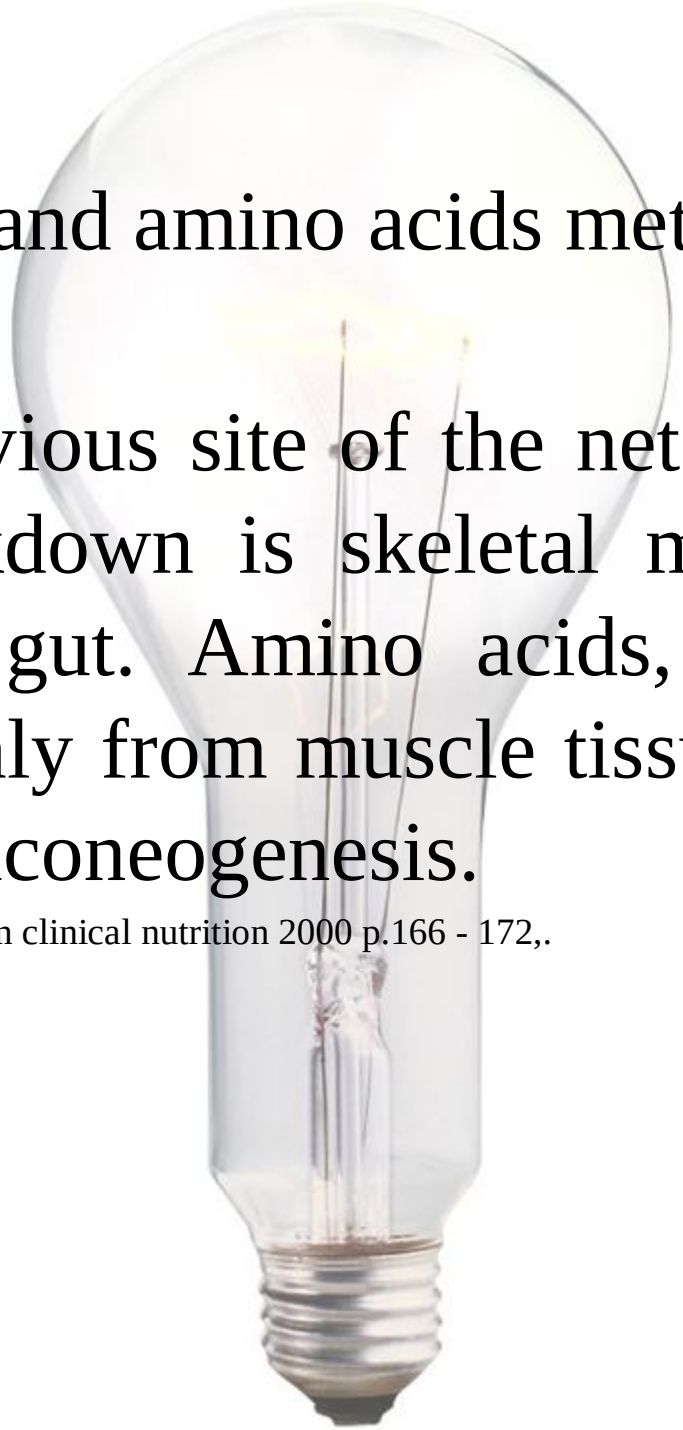
- Part of the breakdown of glucose doesn't require oxygen, while still provide energy. It is very important. In the wound in which mitochondria are not yet developed, or where fat cannot reach the cells due to lack of capillaries are hypoxic conditions.
- Glucose anaerobic degradation product lactate is carried to liver where it is converted to glucose(Cori cycl)

Protein and amino acids metabolism



Whole body protein turnover is increased and balance between synthesis and breakdown in flow phase. Thus increasing severities of injury cause increasing rates of both synthesis and breakdown. Undernutrition reduces synthesis.

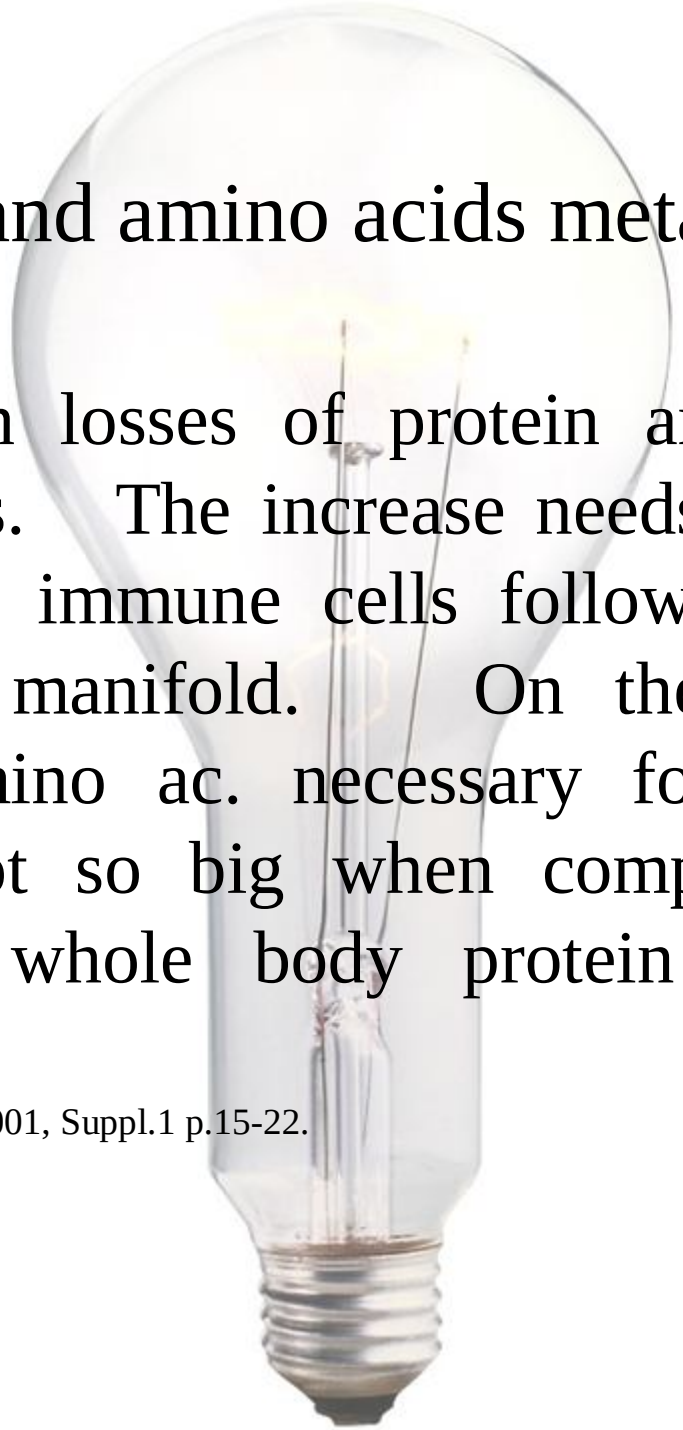
Hutchins AM Metabolic stress and immune function In: Dietitians Handbook of enteral and parenteral nutrition 1998;p.353-383



Protein and amino acids metabolism

The most obvious site of the net increase in protein breakdown is skeletal muscles, the wall of the gut. Amino acids, which are released mainly from muscle tissue are main source for gluconeogenesis.

Sobotka L, Soeters PB Basics in clinical nutrition 2000 p.166 - 172,.

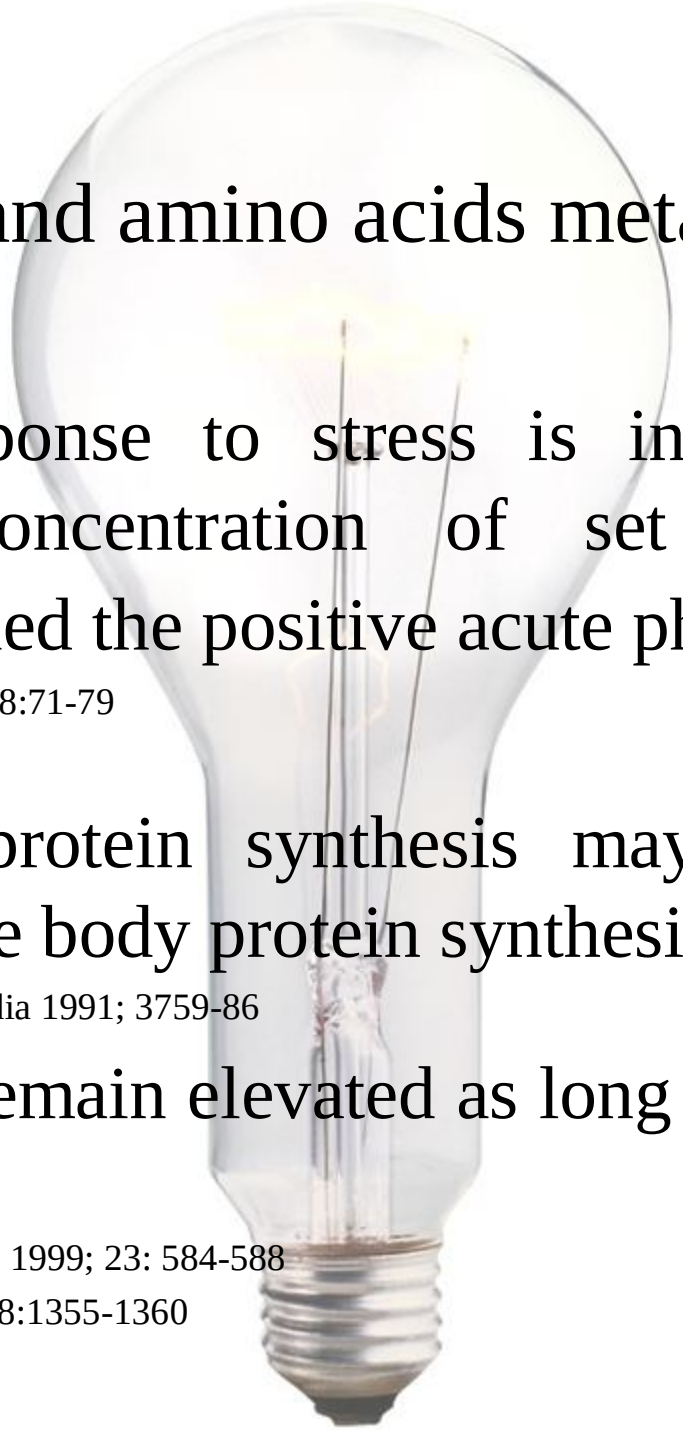


Protein and amino acids metabolism

The main losses of protein are not only in gluconeogenesis. The increase needs for amino ac. for circulatingg immune cells following injury and infection can by manifold. On the other hand quantity of amino ac. necessary for new protein synthesis is not so big when compared with the magnitude of whole body protein turnover and nitrogen loss.

Reeds PJ, Jahoor F Clin Nutr. 2001, Suppl.1 p.15-22.

Protein and amino acids metabolism



A critical response to stress is increase in the circulatingg concentration of set of proteins collectively called the positive acute phase proteins.

Hall GM et all Clin Sci 2000; 98:71-79

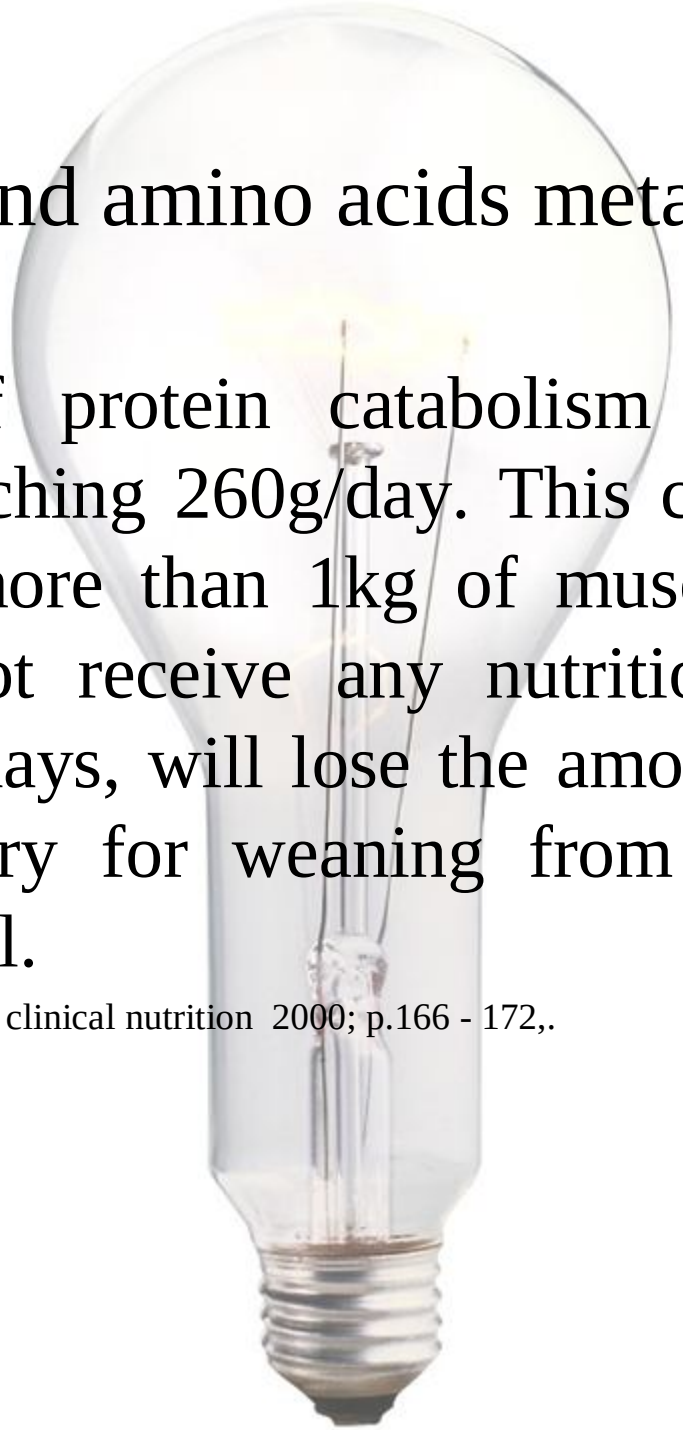
Acute phase protein synthesis may account for quarter of whole body protein synthesis.

Waterlow JC Proc Nutr Soc India 1991; 3759-86

The synthesis remain elevated as long as organism is sressed.

Fearon KC et al. World J Surge 1999; 23: 584-588

Preston T et al. J Nutr 1998: 128:1355-1360



Protein and amino acids metabolism

The extend of protein catabolism in sepsis is tremendous reaching 260g/day. This corresponds to daily loss of more than 1kg of muscles tissue. If patient does not receive any nutrition al support more than ten days, will lose the amount of muscle protein necessary for weaning from ventilator or even for survival.

Sobotka L, Soeters PB Basics in clinical nutrition 2000; p.166 - 172,.

Protein Metabolism During Starvation and Critical Illness

	Starvation	Stress reaction
Proteolysis	-	+++
Protein synthesis	-	+
Amino acid oxidation	-	+++

AA should be increased up to 1.5-2 g/kg/d

Glucose Metabolism During Starvation and Critical Illness

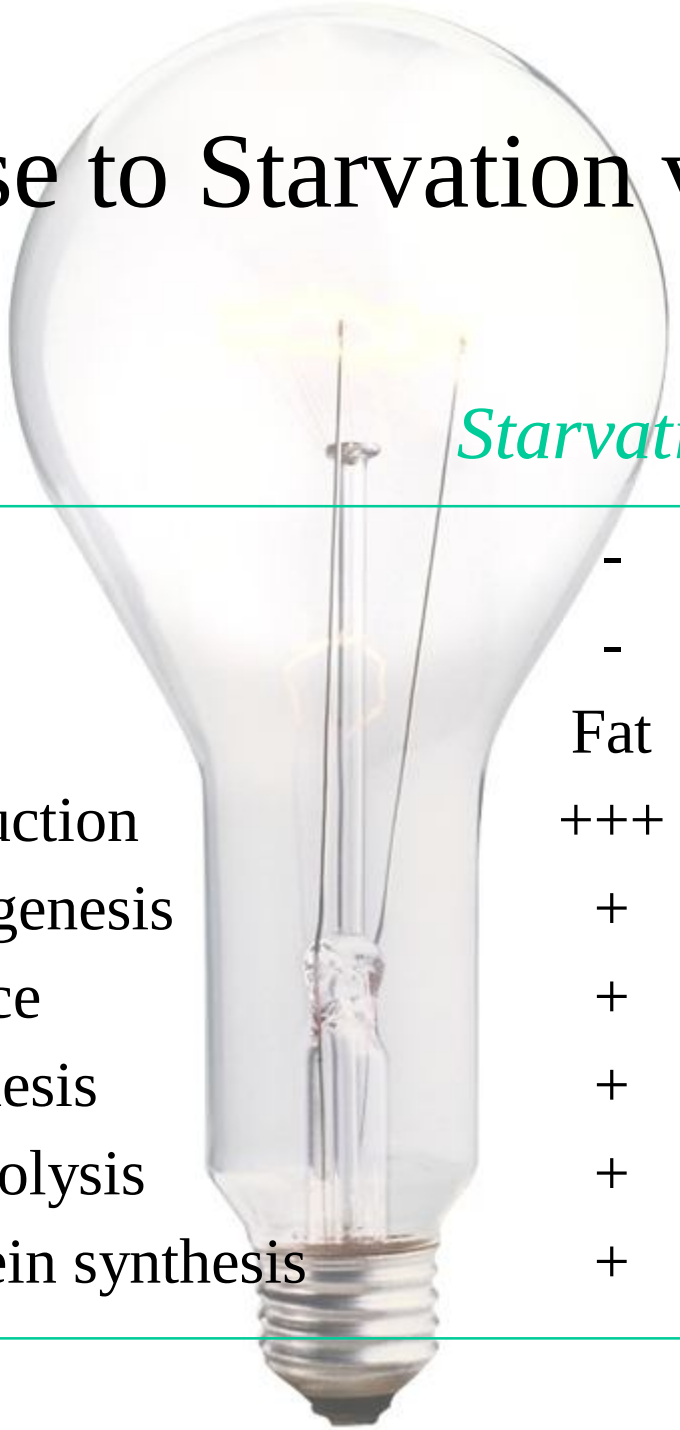
	Starvation	Stress reaction
Gluconeogenesis	+	+++
Glycolysis	-	+++
Glucose oxidation	-	-
Glucose cycling	-	+++

Glucose oxidation decrease to 2-2.5 mg/kg/min (3-4 g/kg/d)

Lipid Metabolism During Starvation and Critical Illness

	Starvation	Stress reaction
Peripheral lipolysis	+++	++
Lipid oxidation	+++	+
Fatty acid cycling	-	++

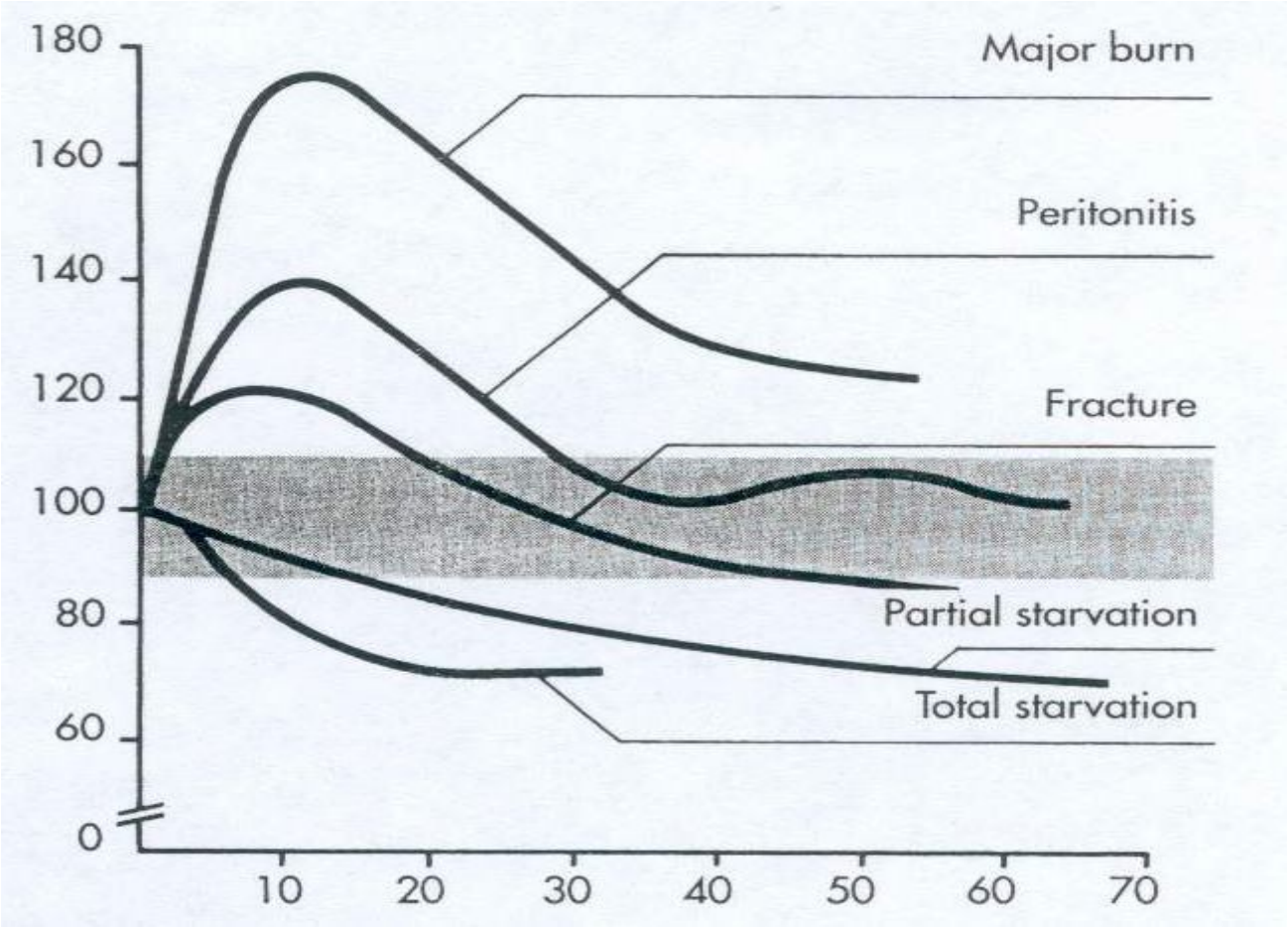
Response to Starvation vs Injury

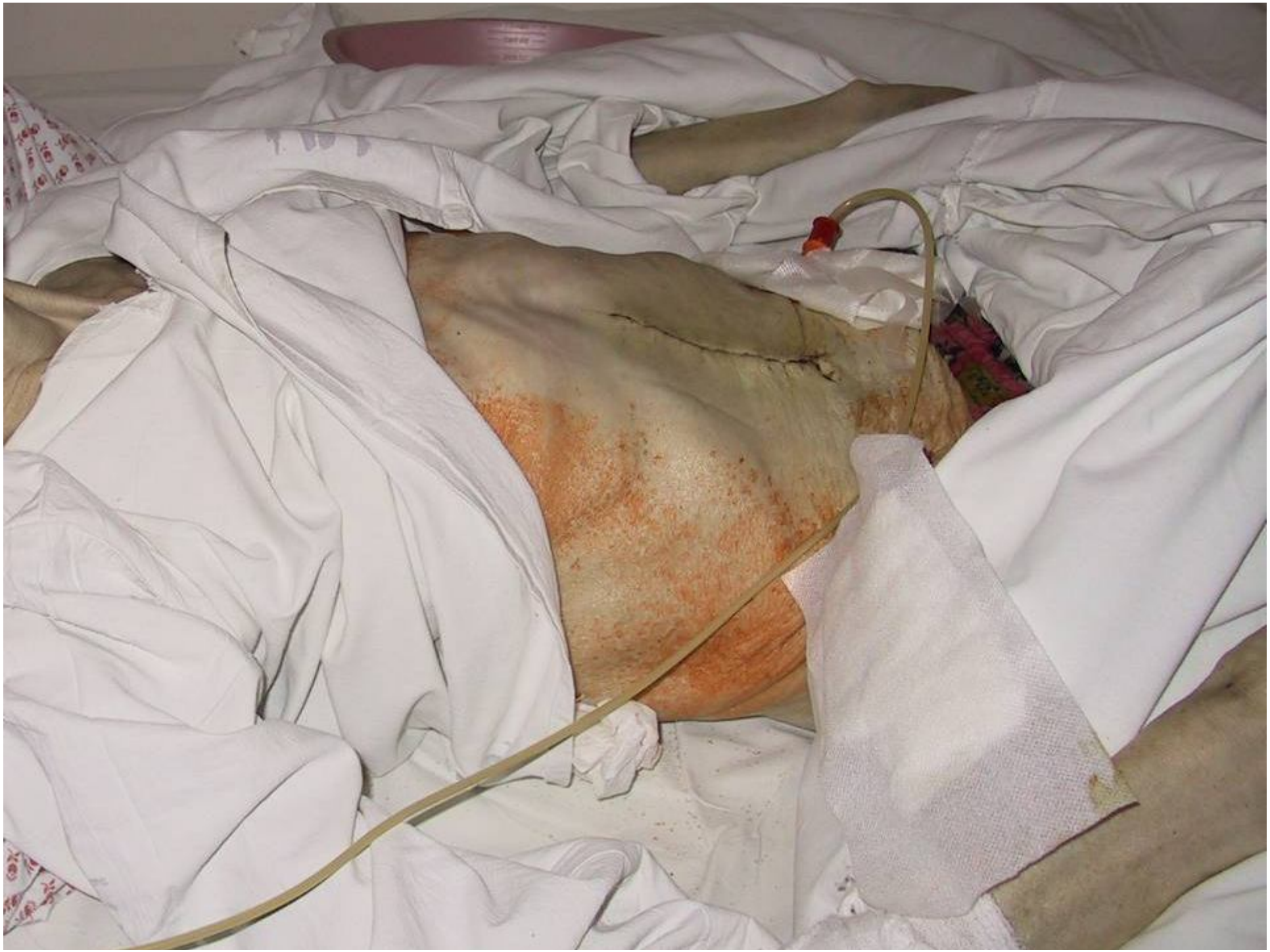


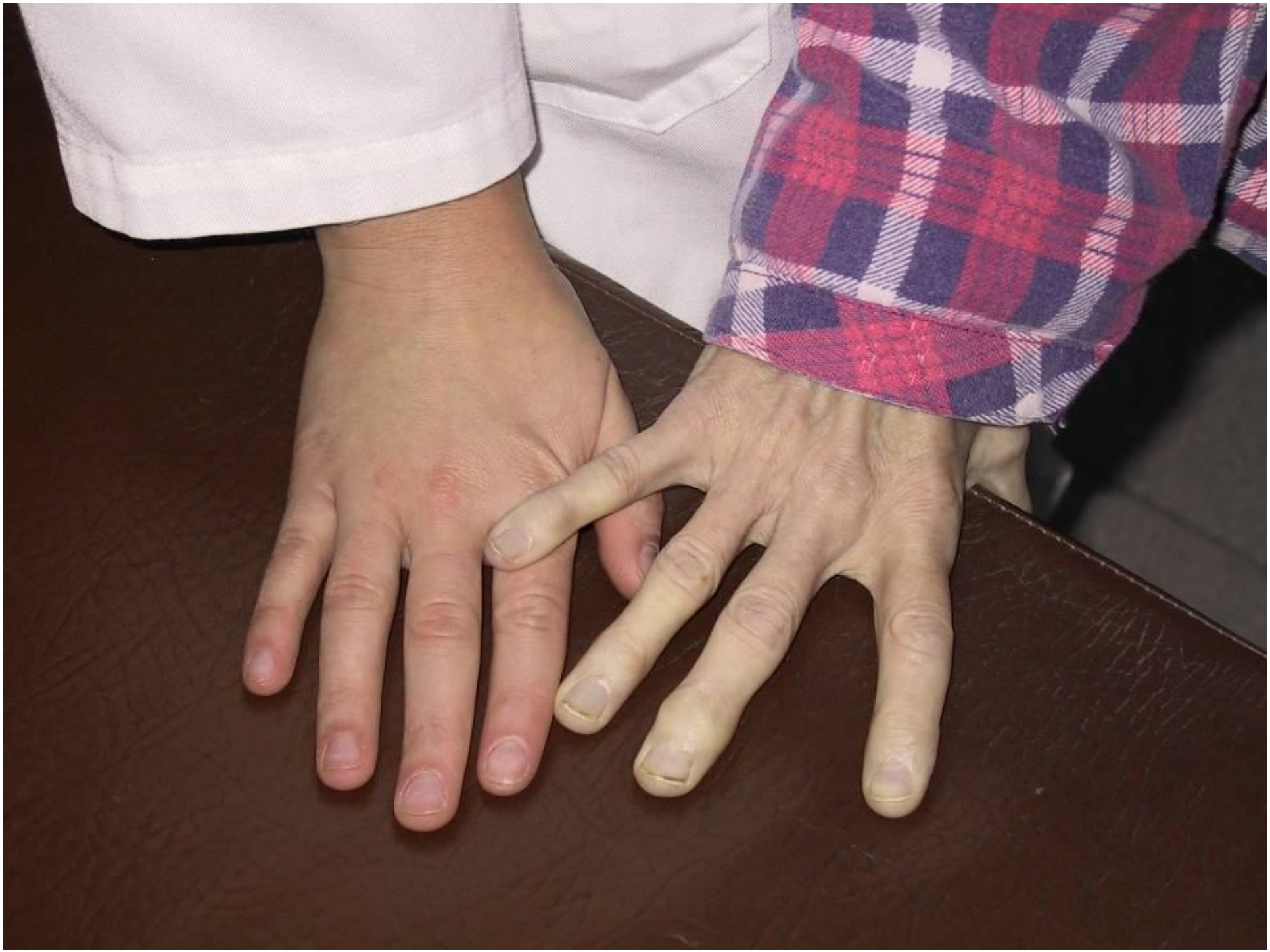
Parameter	*Starvation*	*Trauma*
BMR	-	++
Mediators	-	+++
Major fuel	Fat	Mixed
Ketone production	+++	+/-
Hepatic ureagenesis	+	+++
Neg N balance	+	+++
Gluconeogenesis	+	+++
Muscle proteolysis	+	+++
Hepatic protein synthesis	+	+++

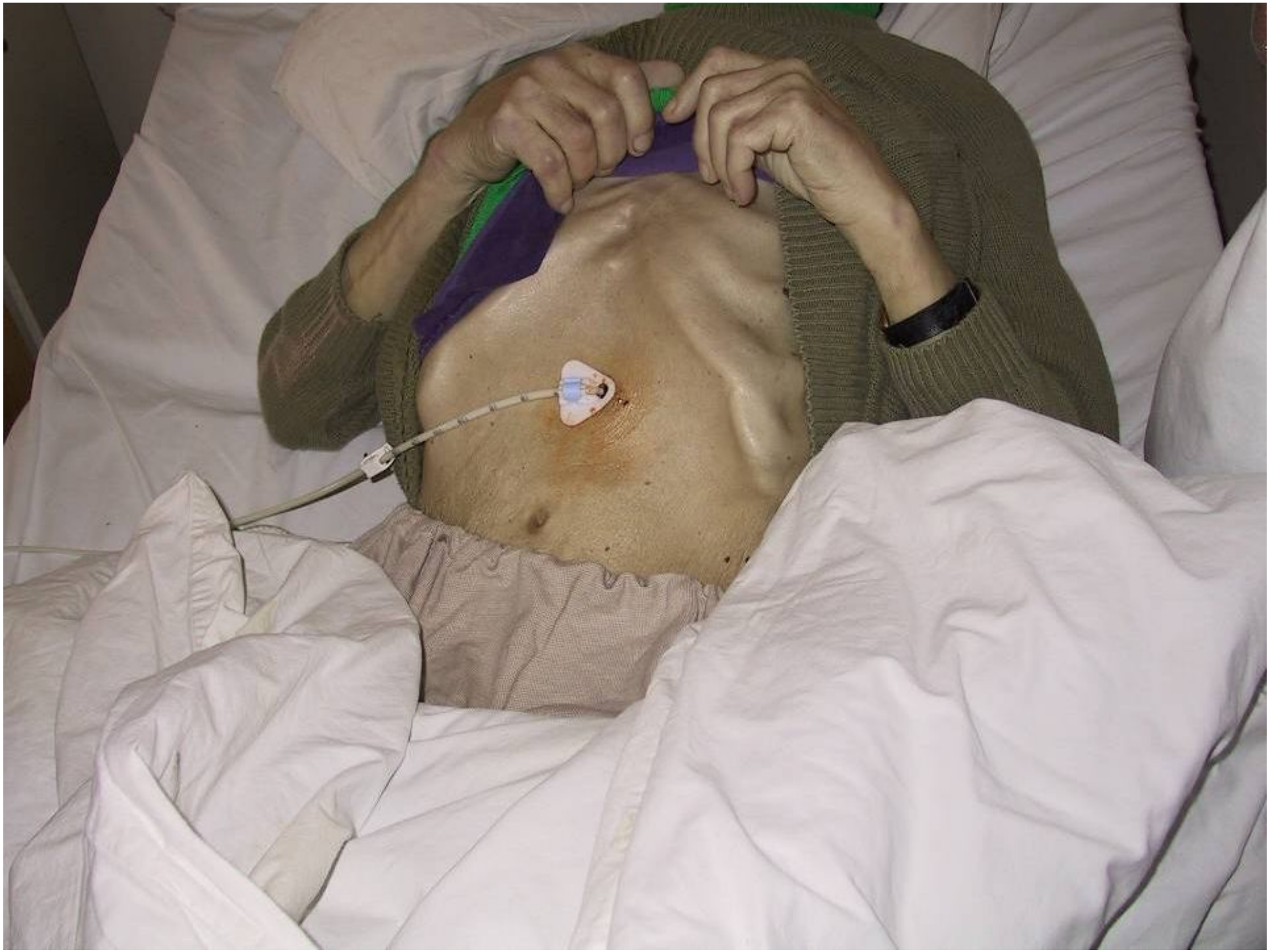
Hyper metabolism occurs in proportion to the severity and duration of the critical illness

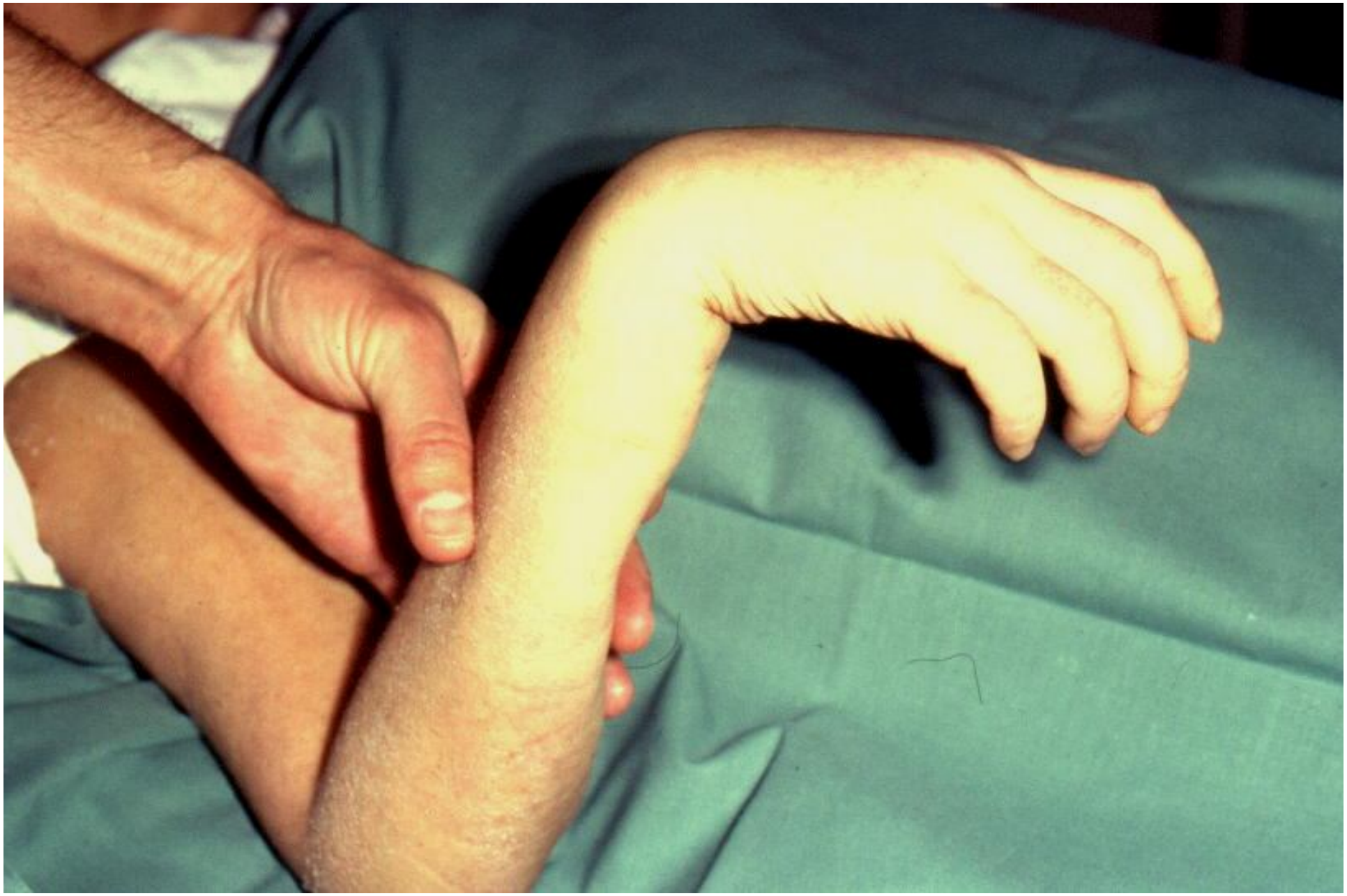
Handbook of clinical nutrition ed by Heimburgers DC, Weinsier RL, 1997; p. 445-457

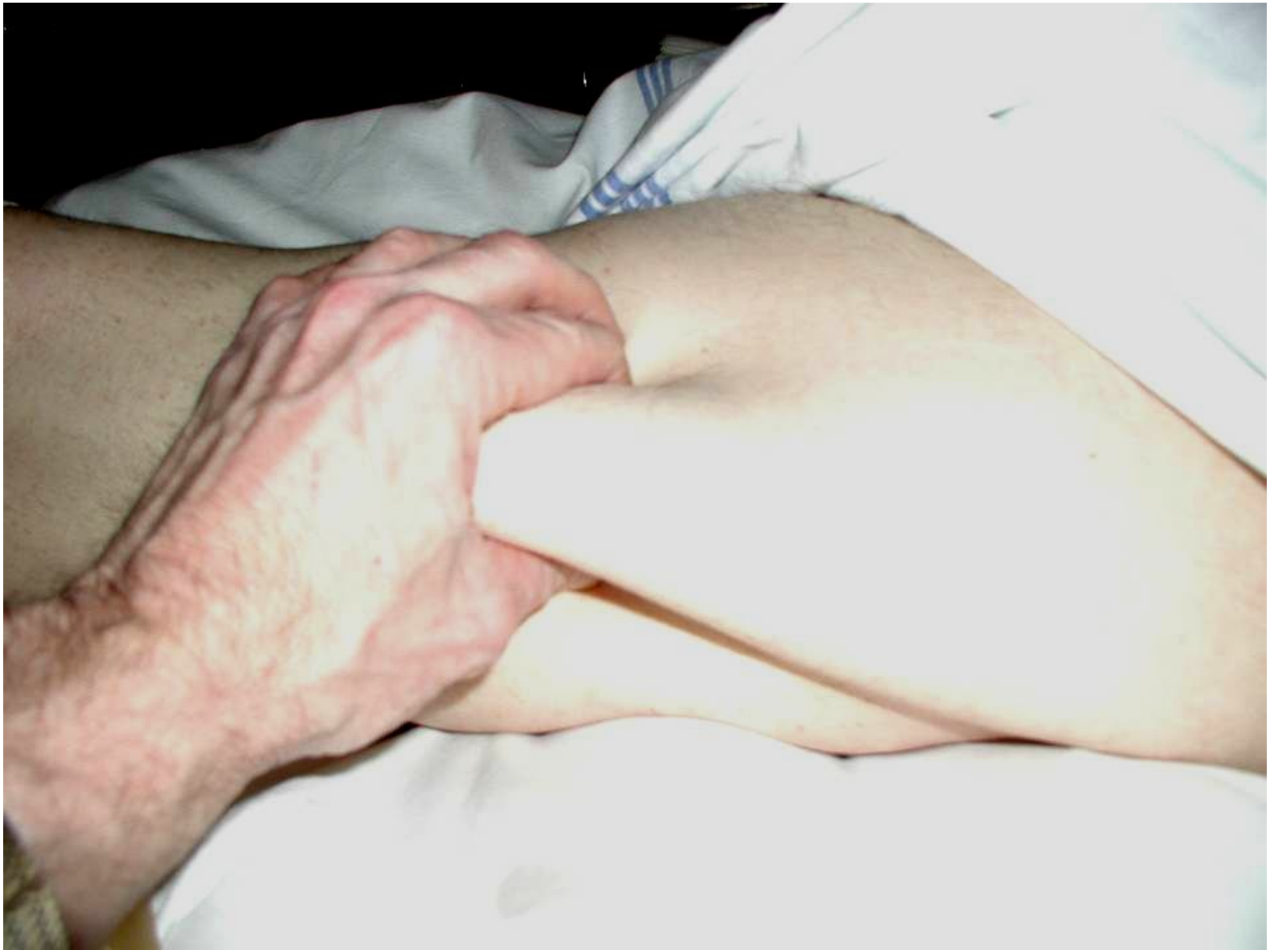


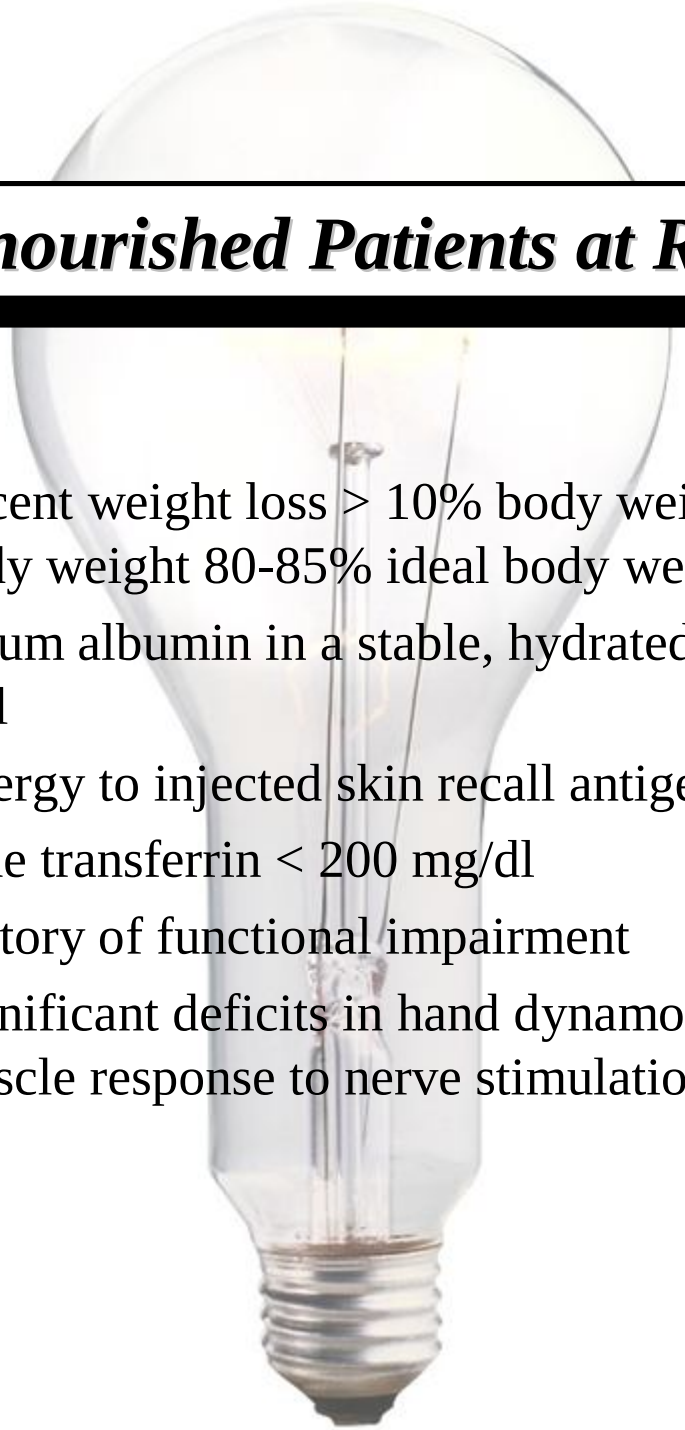






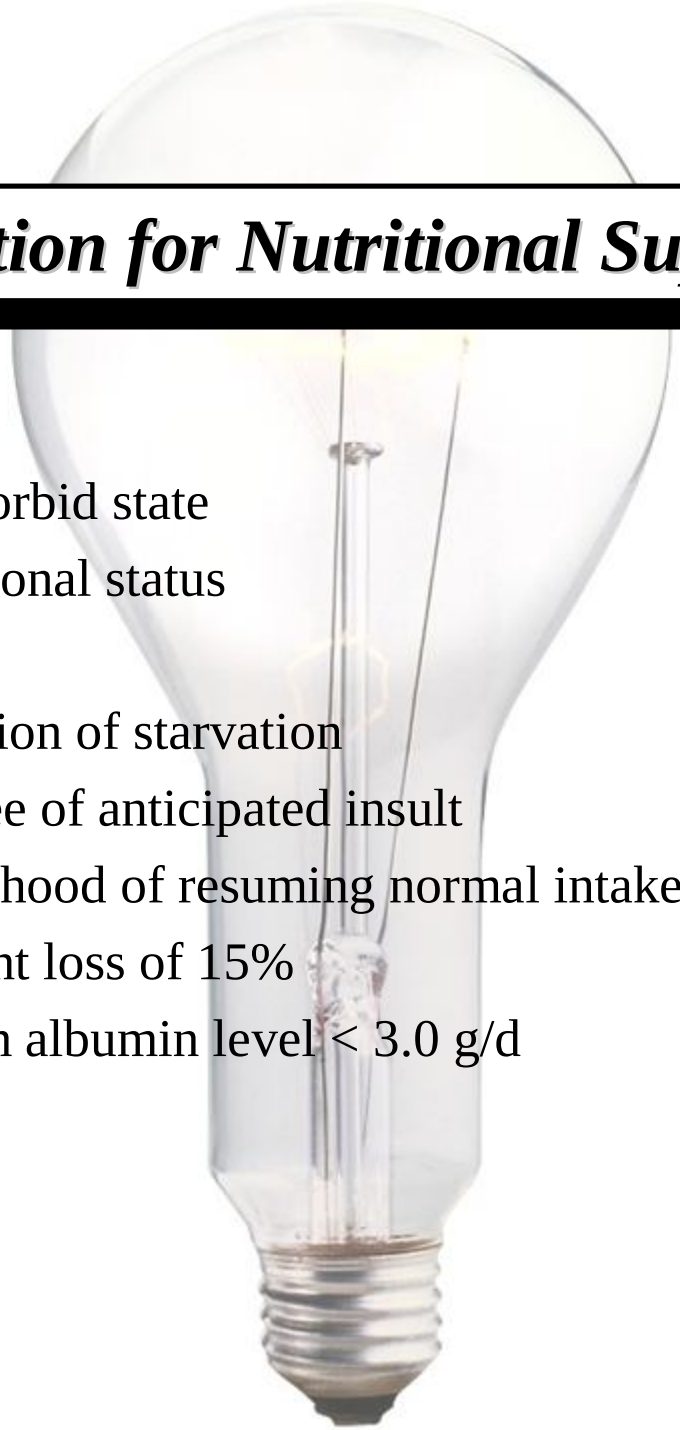






Malnourished Patients at Risk

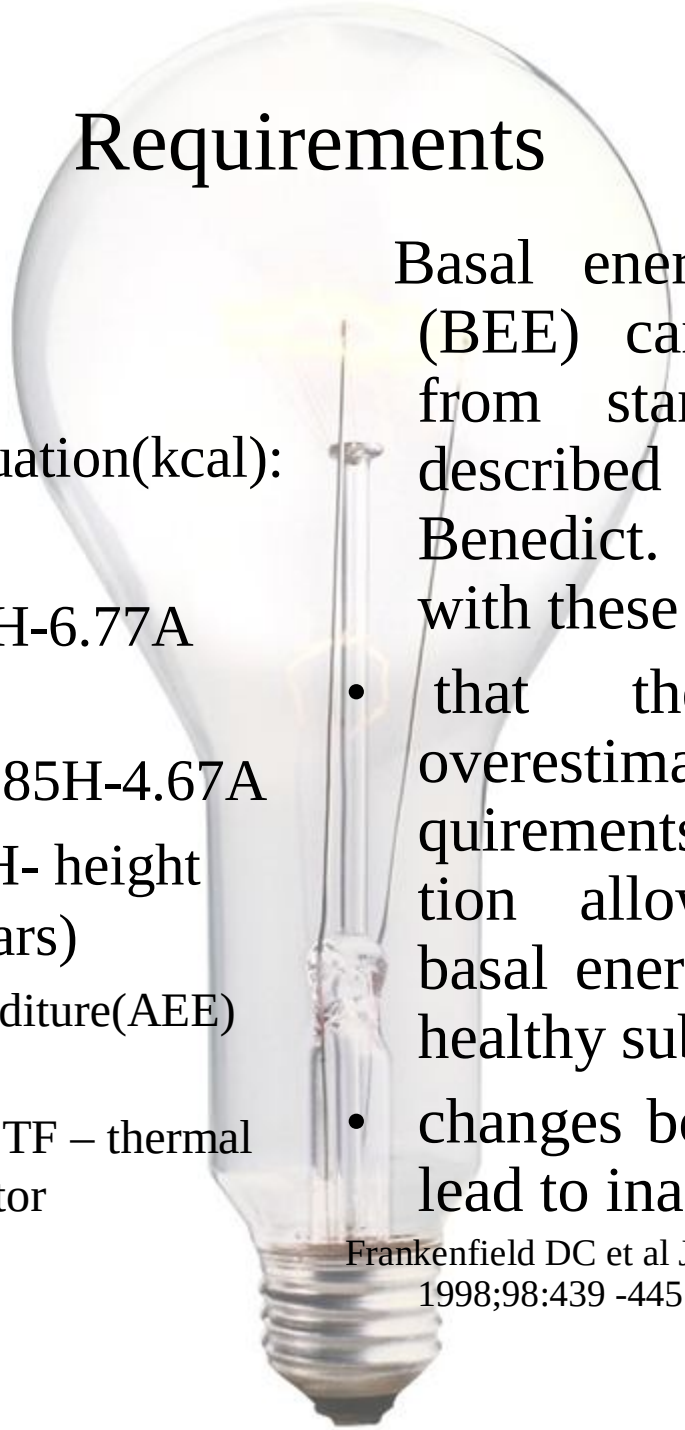
- Recent weight loss > 10% body weight and/or body weight 80-85% ideal body weight
- Serum albumin in a stable, hydrated patient < 3.0 g/dl
- Anergy to injected skin recall antigens
- True transferrin < 200 mg/dl
- History of functional impairment
- Significant deficits in hand dynamometry or muscle response to nerve stimulation



Indication for Nutritional Support

- Premorbid state
- Nuritional status
- Age
- Duration of starvation
- Degree of anticipated insult
- Likelihood of resuming normal intake soon
- Weight loss of 15%
- Serum albumin level < 3.0 g/d

Requirements



Harris–Benedict equation(kcal):

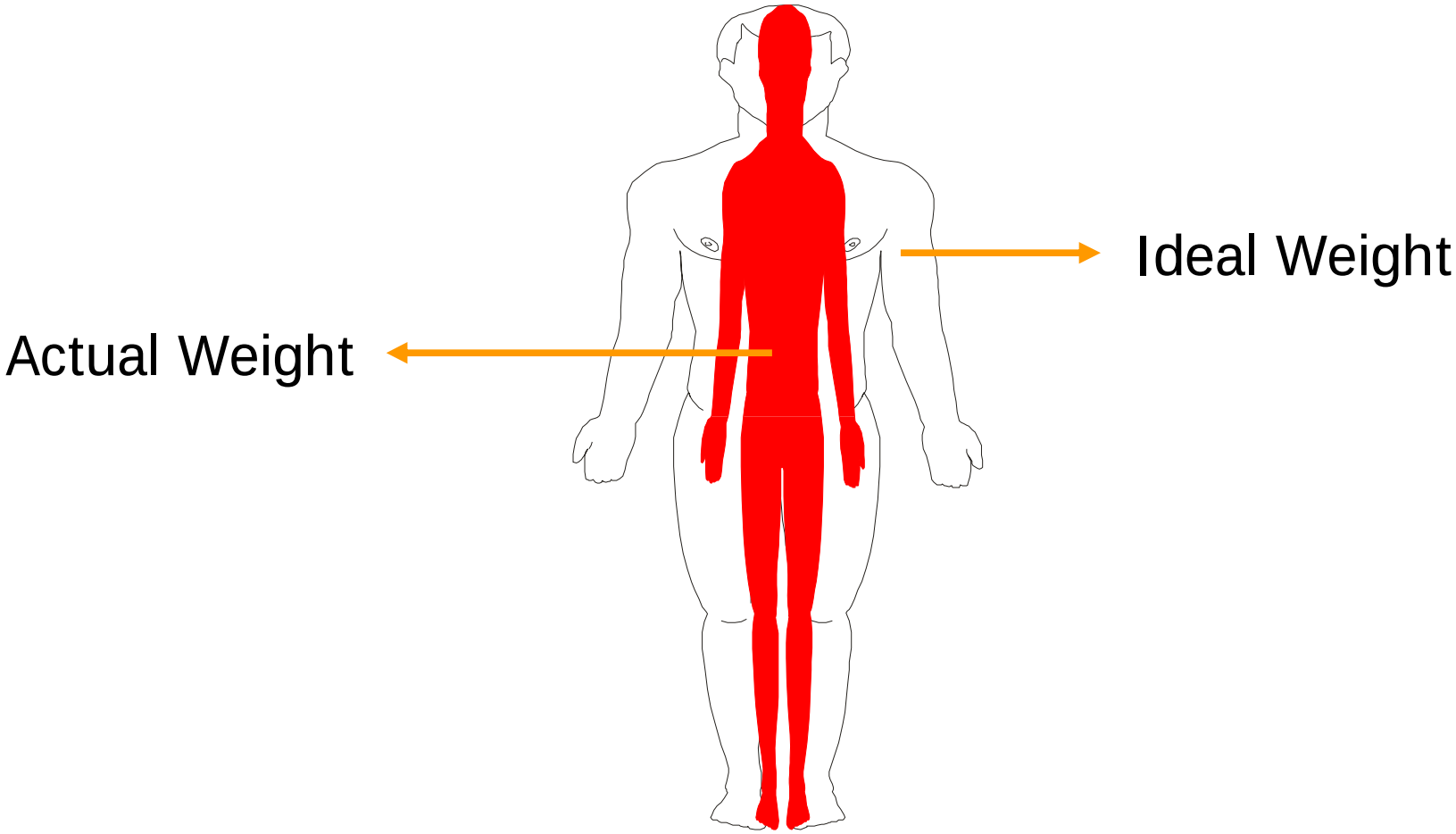
- Male = 66.5+13.75W+5H-6.77A
- Female= 655.1+9.56W+1.85H-4.67A
- W- weight(kg), H- height (cm), A- age (years)
- Actual energy expenditure(AEE) =BEExAFxIFxTF
- AF – activity factor, TF – thermal factor, IF- injury factor

Basal energy expenditure (BEE) can be estimated from standard equation described by Harris and Benedict. The problems with these are:

- that they tend to overestimate energy requirements, because equation allow to estimate basal energy expenses for healthy subjects
- changes body weight may lead to inaccuracies.

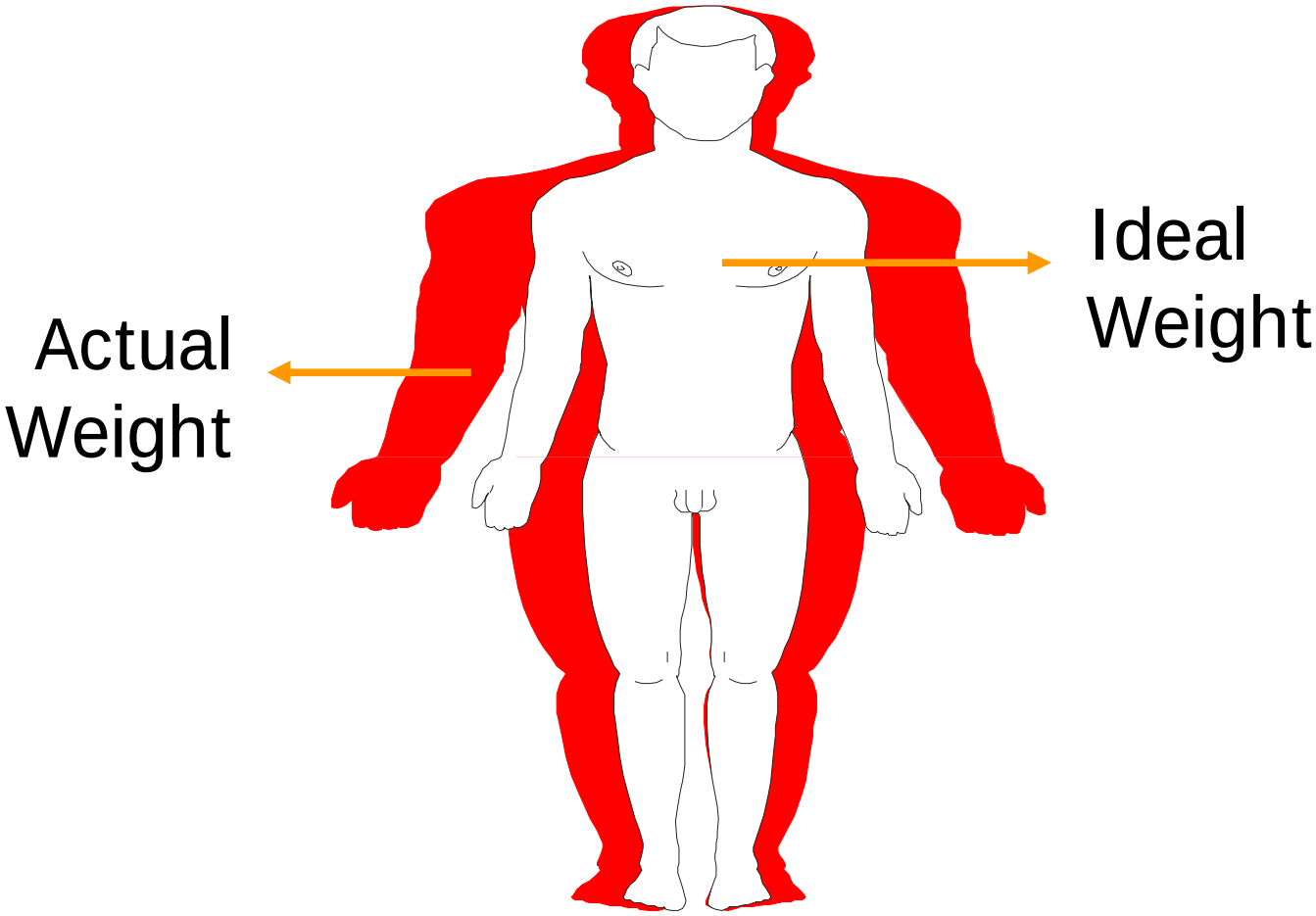
Frankenfield DC et al J Am Dietetic Ass. 1998;98:439 -445

Malnutrition



In malnutrition, energy expenditure must be calculated based on actual body weight.

Obesity



In obesity, energy expenditure must be calculated on adjusted weight

Requirements

Most simple method is:

to consider an average value of 1kcal/kg/h for unstressed and resting male subjects(minus 5-10% in female subjects), and to make some adjustments for level of activity, injury and stress factors.

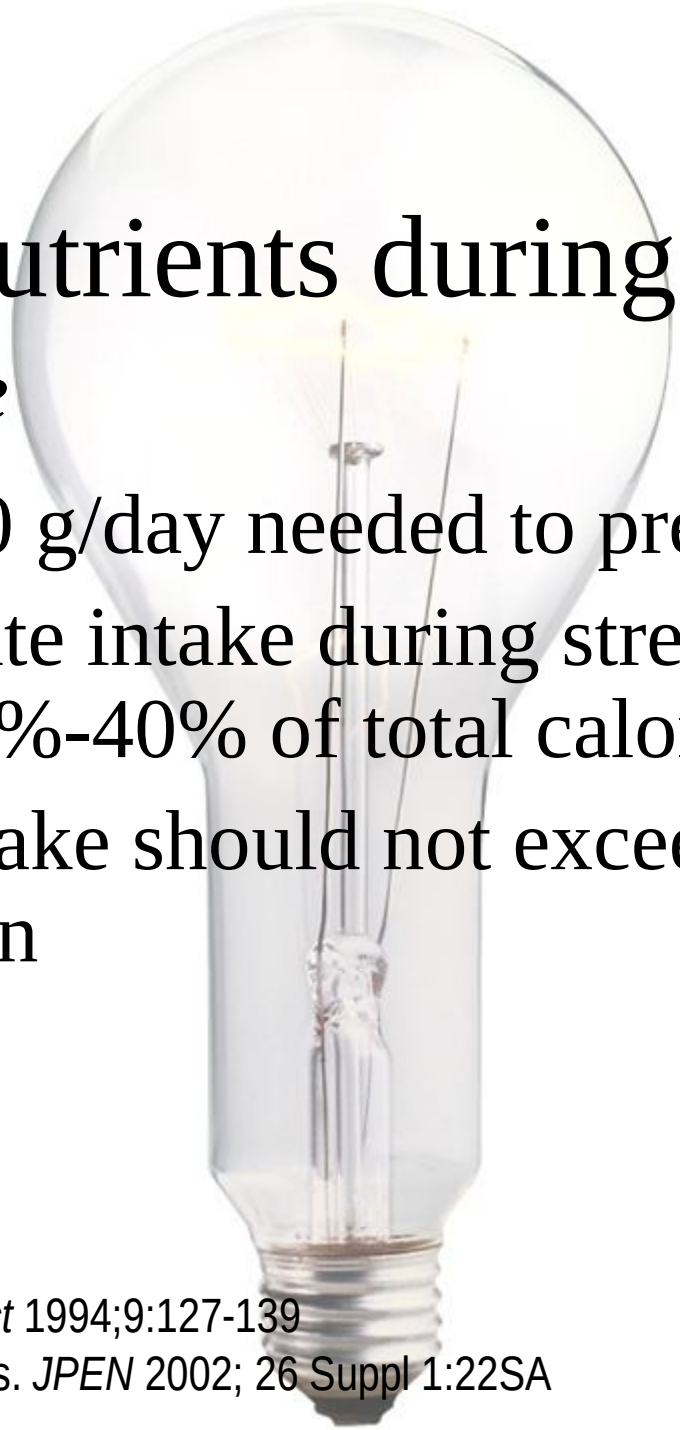
Y. Carpentier Basics in clinical nutr. 1999;p. 39-42

Requirements

by Clinical nutrition in practice Braun Melsungen AG1997; p.30

Actual energy expenditure(AEE) =BEE x AF x IF x TF			
AF(Activity factor)		IF (Injury factor)	1.0
In bed	1.1	Uncomplicated patient	1.1
In bed, but mobile	1.2	Postoperative state	1.2
Mobile	1.3	Fracture	1.3
		Sepsis	1.4
TF(Thermal factor)		Peritonitis	1.5
38°C	1.1	Multiple trauma	1.6
39 °C	1.2	Multiple trauma + sepsis	1.7
40 °C	1.3	Burns 30-50%	1.8
41 °C	1.4	Burns 50-70%	1.9
		Burns 70-90%	2.0

°C



Macronutrients during Stress

Carbohydrate

- At least 100 g/day needed to prevent ketosis
- Carbohydrate intake during stress should be between 30%-40% of total calories
- Glucose intake should not exceed 5 mg/kg/min

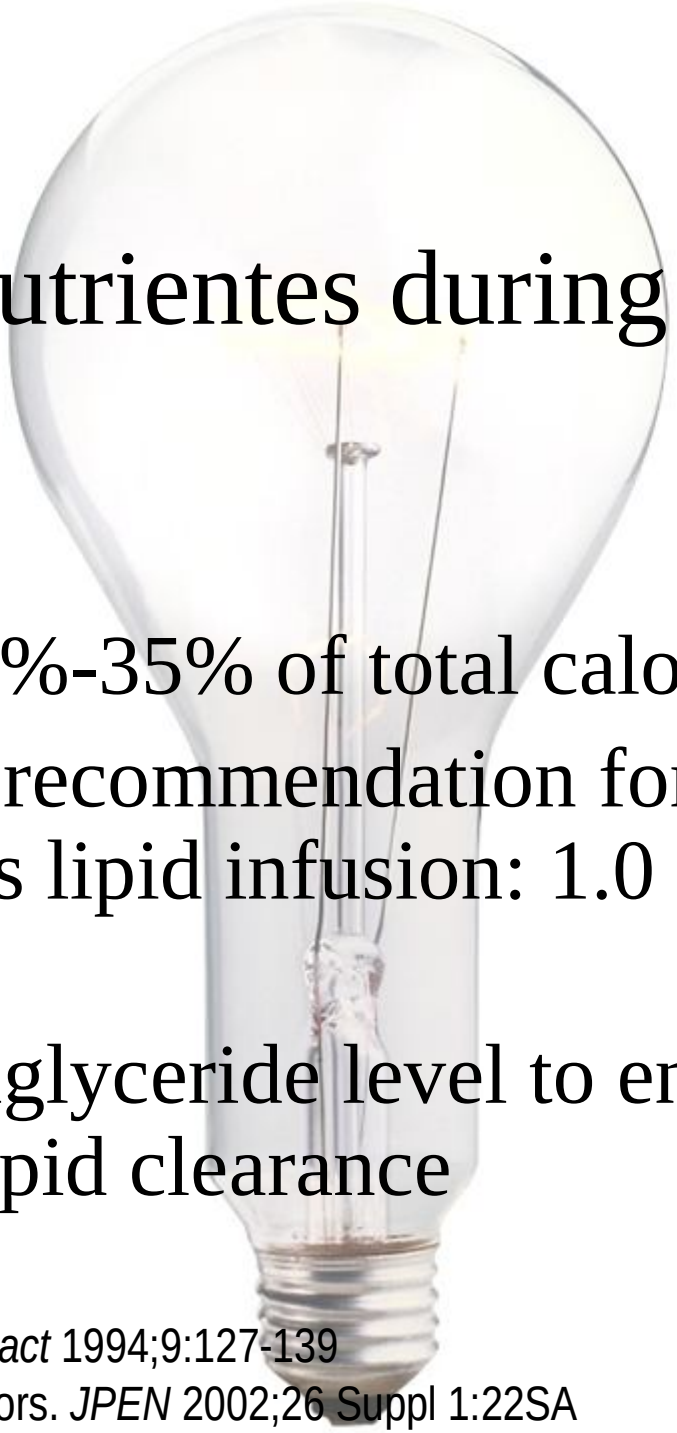
Barton RG. *Nutr Clin Pract* 1994;9:127-139
ASPEN Board of Directors. *JPEN* 2002; 26 Suppl 1:22SA

Macronutrients during Stress

- Glucose is never recommended as sole source of energy substrate in injured or septic patients

Y. Carpentier Clinical Nutrition-Parenteral nutrition 2^{ed}ed. 1993, p.35

Hutchins AM et al Metabolic stress and immune function In: Handbook of clinical nutrition ed by Heimburgers DC, Weinsier RL, 1997; p.73-74



Macronutrientes during Stress

Fat

- Provide 20%-35% of total calories
- Maximum recommendation for intravenous lipid infusion: 1.0 -1.5 g/kg/day
- Monitor triglyceride level to ensure adequate lipid clearance

Barton RG. *Nutr Clin Pract* 1994;9:127-139
ASPEN Board of Directors. *JPEN* 2002;26 Suppl 1:22SA

Recommended daily substrate intake in critical illness

Sobotka L., Soeters PB Nutrition support in critically ill patients. In: Basics in clinical nutrition 2000; p. 166-172

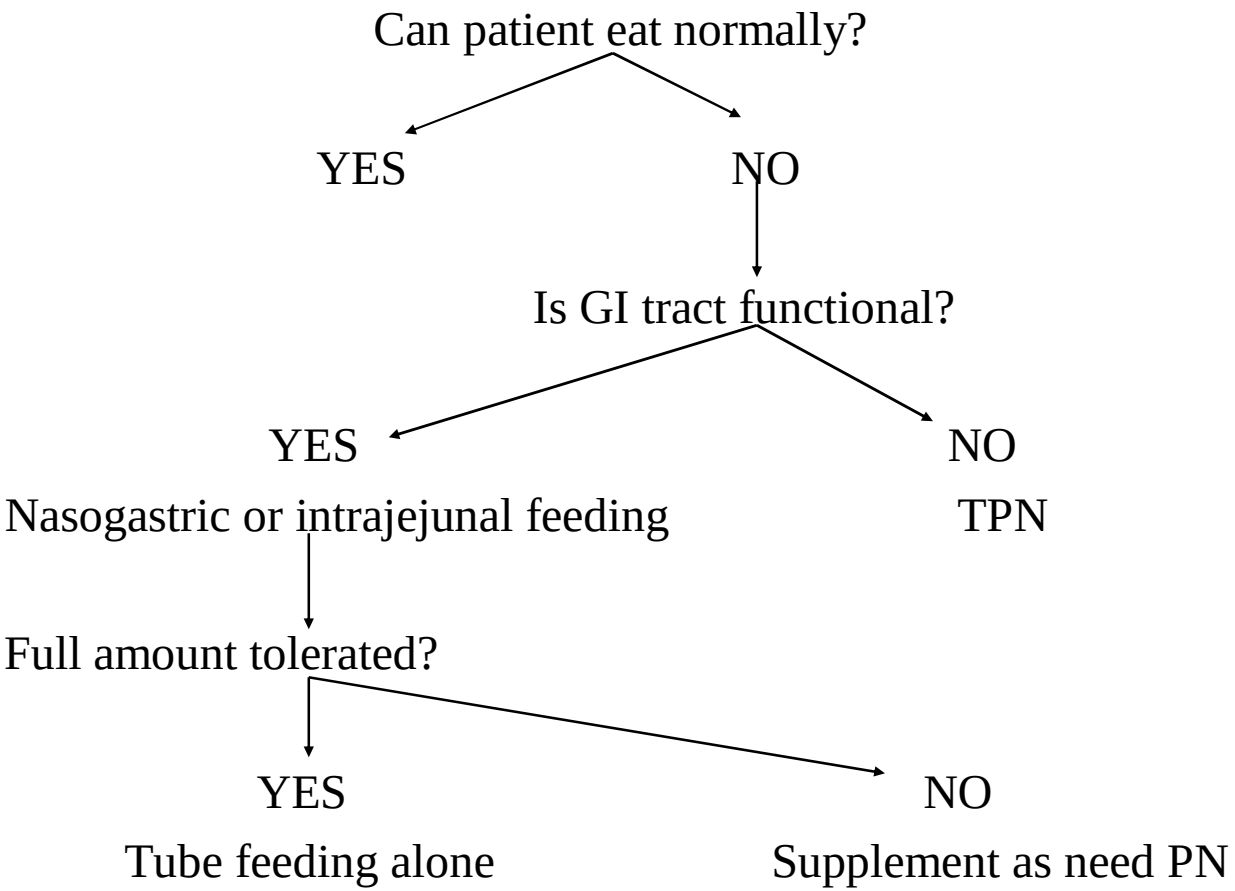
	Minimal dose (g/kg/d)	Maximal dose (g/kg/d)	Comments
Glucose	2	6	Give insulin if necessary
Lipid emulsion	0.5	1.5	MTC/LTC emulsions is preferred
Amino ac.	1.2	2.0	Special formulas

Determining Fluid Requirements

- Fluid requirements (H_2O):
  - Young athletic adult: 40 ml/kg
  - Most adults: 35 ml/kg
  - Older adults (55 to 65 y): 30 ml/kg
  - Elderly adults (> 65 y): 25 ml/kg
- Or 1 ml/kcal energy expenditure
  - Fluid requirements increase with pregnancy, infants, fever, high altitude, low humidity, profuse sweating, diarrhea, vomiting, hemorrhage, fistula drainage, surgical drains, and loss of skin integrity
  - For example, a 58 y old weighing 60 kg requires 1800 ml fluid/d

Decision tree for nutritional support in IC

by Berger R lecture 2001



Route

TEN

Advantages:

- Physiologic
- Maintains mucosal integrity, minimizing risk of bacterial colonization
- Fewer septic complications

Disadvantages:

- Requires adequate gastric emptying
- Risk of aspiration
- Frequent interruptions in feeding necessitated by multiple trips to the OR

TPN

Advantages:

- Does not require adequate gastric motility
- No risk of aspiration

Disadvantages:

- Intestinal mucosal atrophy
- Catheter related sepsis
- Expensive in relation to TEN

Aim of early enteral feeding

- Anabolism/ Reduction of Catabolism
- Avoidance of Hypermetabolism
- Nurturing of gut micro-organisms
- maintenance of gut mucosal health
- Improving outcome?

Enteral feeding - the downside

By Griffits RD lecture" Controversies in nutrition on the ICU" 2001

- Not as good as normal eating
  - -lacks cephalic neurohumoral stimuli (taste etc)
  - -gastro-oesophageal and colonic dysfunction
  - - failure to develop post-prandial motility in small bowel
- Delivery considered "easy"
  - - too easily discontinued , not seen as essential to well-being
  - - gastric feeding considered the same as jejunal feeding
- Complications targets of delivery unrecognised
  - - aspiration may be more common than realised
  - - abdominal distension can impair ventilatory capacity
- Nutrition targets often not achieved
  - - NG feeding leads to inadequate delivery

EN: Contraindications

- Malfunctioning GI tract or conditions requiring extended bowel rest:
  - SBS
  - Mechanical obstruction or GI motility disorder
  - Prolonged ileus
  - Severe GI bleeding, diarrhea or vomiting
  - High output fistula (> 500 ml/d)
  - Severe inflammation or enteritis
  - GI ischemia
  - Severe pancreatitis

Access for Enteral Nutritional Support

Access Option	*Comments*
Nasogastric tube	Short-term use only; aspiration risks; nasopharyngeal trauma; frequent dislodgment
Nasoduodenal/nasojejunal	Short-term use; lower aspiration risks in jejunum; placement challenges (radiographic assistance often necessary)
Percutaneous endoscopic gastrostomy (PEG)	Endoscopy skills required; may be used for gastric decompression or bolus feeds; aspiration risks; can last 12–24 months; slightly higher complication rates with placement and site leaks
Surgical gastrostomy	Requires general anesthesia and small laparotomy; may allow placement of extended duodenal/jejunal feeding ports; laparoscopic placement possible
Fluoroscopic gastrostomy	Blind placement using needle and T-prongs to anchor to stomach; can thread smaller catheter through gastrostomy into duodenum/jejunum under fluoroscopy
PEG-jejunal tube	Jejunal placement with regular endoscope is operator dependent; jejunal tube often dislodges retrograde; two-stage procedure with PEG placement, followed by fluoroscopic conversion with jejunal feeding tube through PEG
Direct percutaneous endoscopic jejunostomy (DPEJ)	Direct endoscopic placement with enteroscope; placement challenges; greater injury risks
Surgical jejunostomy	Commonly applied during laparotomy; general anesthesia; laparoscopic placement usually requires assistant to thread catheter; laparoscopy offers direct visualization of catheter placement
Fluoroscopic jejunostomy	Difficult approach with injury risks; not commonly done

Enteral Formulas

- Standard tube feeding formulas are like your see in our hospital :
  - Assume normal organ function
  - No allergies
  - No fluid restrictions
  - Contain fibre
  - 1.0 – 1.2 kcal/ml; isotonic

Enteral Formulas

- Volume-restricted/nutrient dense formulas:
  - Generally 1.5 – 2.0 kcal/ml
  - 400 – 700 mOsm/kg
  - Useful for CHF, renal failure, home tube feeds (less time)

Enteral Formulas

- Disease specific formulas:
  - Useful for diabetes (less CHO), renal failure (less Na, K, PO_4, Mg)
  - Often higher in fat
  - 375 – 700 mOsm/kg

Enteral Formulas

- Chemically defined formulas (semi-elemental and elemental):
  - Promote rapid absorption of nutrients for patients with GI impairment
  - Contain free a.a., hydrolyzed whey, casein, short chain peptides, MCT f.a.
  - 460 – 650 mOsm/kg
  - Useful for pancreatic disorders, malabsorption syndrome, Crohn's

Ordering EN

- Initiate full strength formula at 20 – 40 ml/h
- Increases of 10 – 20 ml/h are OK if feeds tolerated
- Continue to progress feeds until goal reached
  - If tolerated, increase 20 ml/h q 8 h to goal of 80 ml/h
  - 24 h to get to goal rate

Transitioning Enteral Feeds

- To change to overnight feeds:
  - Increase flow rate
  - Decrease infusion duration
  - For example, 80 ml/h x 24 h → 105 ml/h x 18 h → 130 ml/h x 15 h → 160 ml/h x 12 h
- Bolus feeds:
  - 2 cans infused over 3 hours
  - Gradually decrease infusion time
  - Do not bolus into the jejunum

Practical Enteral Feeding

- Goals of Nutritional Support
  - ✓ Use the gut if possible
  - ✓ Administer at least 20% of caloric and protein requirement by gut
- Smalllest possible nasgastric tube, tip at the duodenum
- Constant infusion except at bed time, head up 30°
- For gastric feeding, first osmolality and then volume,

  reversed for jejunal feeding
- Complications
  - ✓ Malposition and/or aspiration
  - ✓ Diarrhea, dehydration, hyperglycemia and ions
  - ✓ Pneumaosis intestinalis with perforation
  - ✓ Hyperosmolar nonketotic coma
  - ✓ perforation

Monitoring EN

- Monitoring parameters vary with patient acuity, duration of feeds and institutional practice
- Weekly weights
- Bowel function
- Fluid and electrolyte balance
- Visceral protein (albumin, prealbumin)
  - Consider half-life, change in fluid status, organ function and presence of infection

Complications of EN

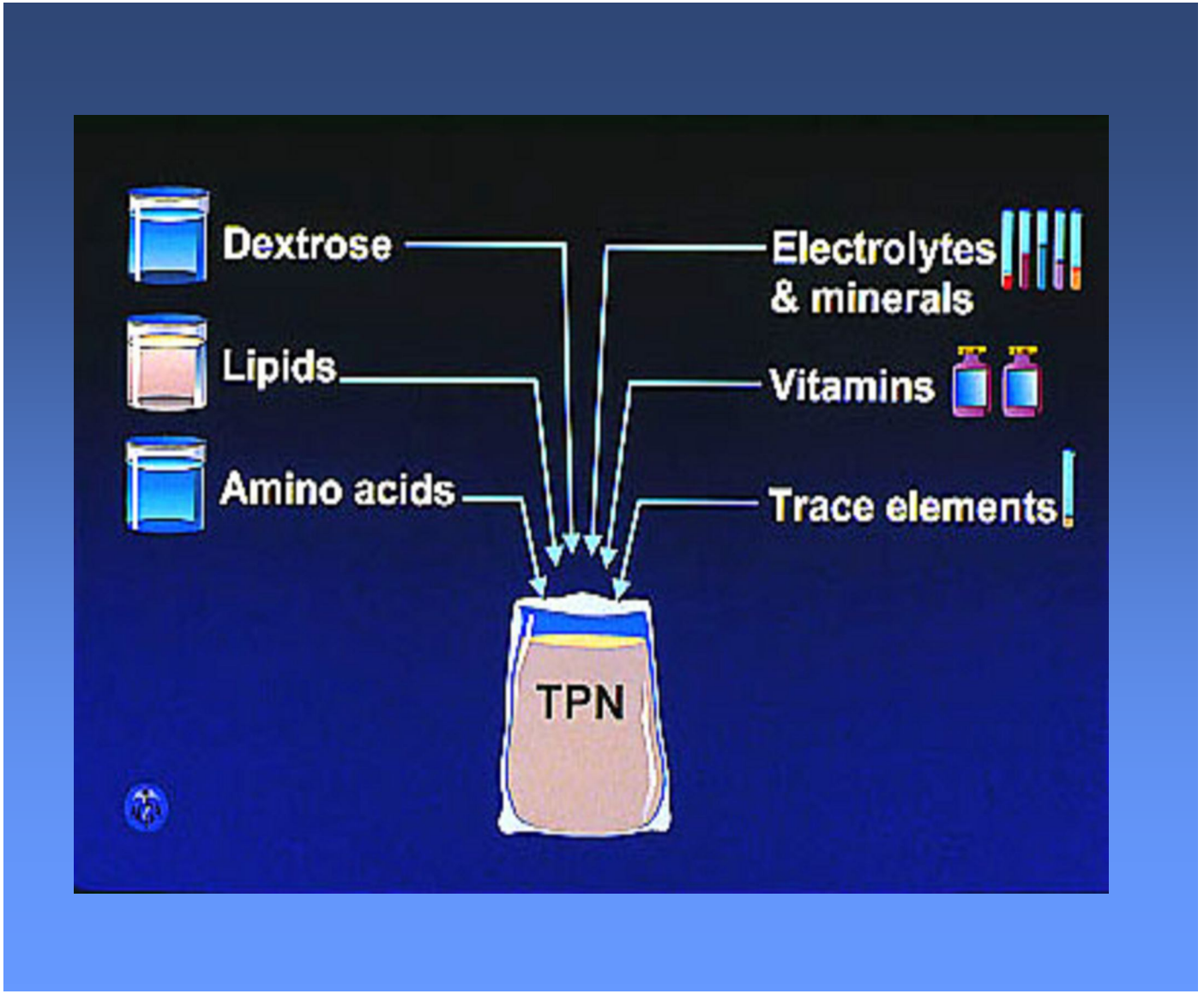
- Diarrhea: 2 – 63 % incidence
  - Formula responsible for diarrhea ~ 20% of cases
- Constipation
- Aspiration: 0.8 – 95 % incidence
  - Clinically significant aspiration resulting in pneumonia 1 – 4 %
- GI intolerance: N & V, abdo discomfort
- Clogged tubes
- Procedure related complications

PARENTERAL NUTRITION

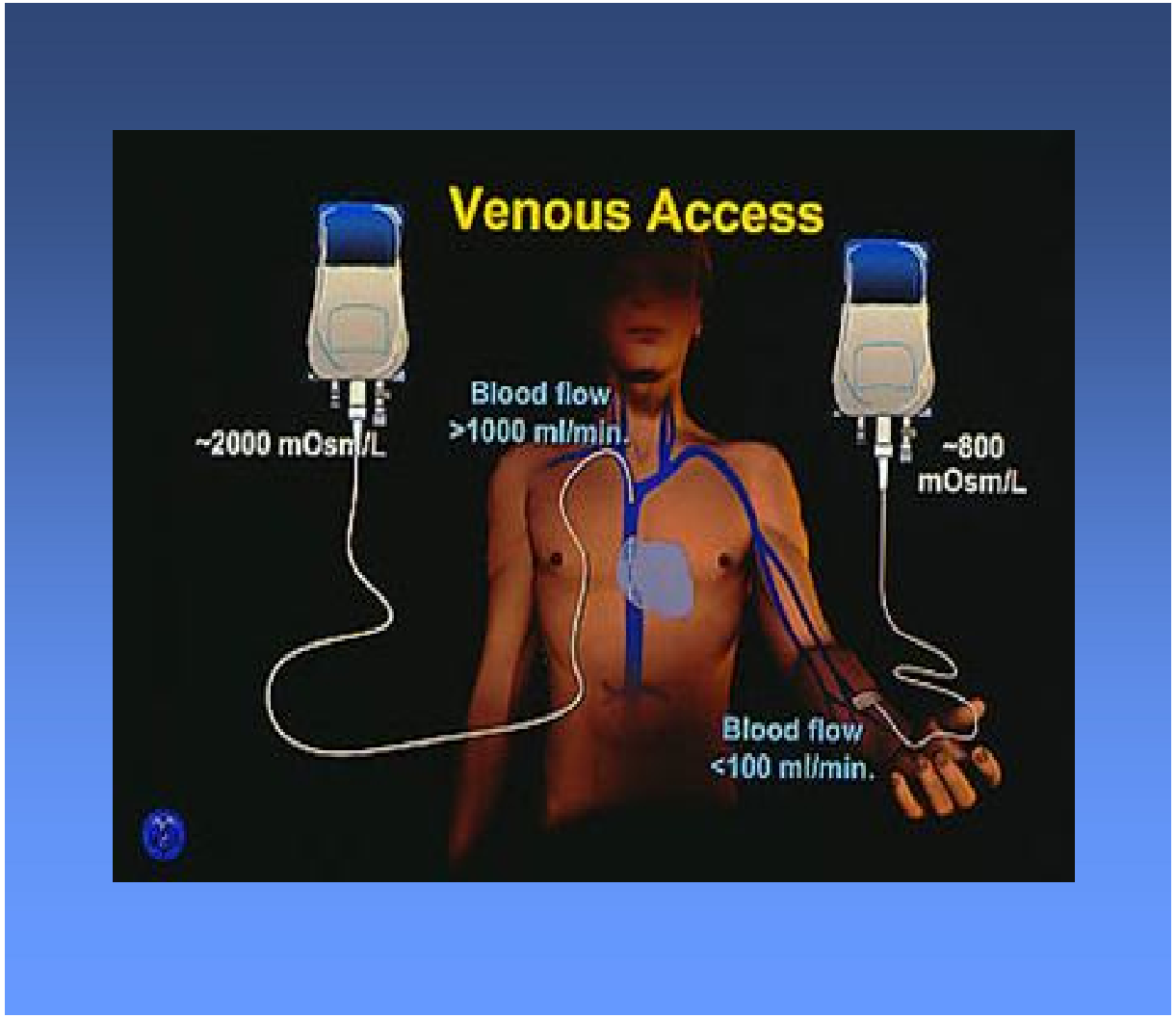
Parenteral nutrition involves the continuous infusion of a hyperosmolar solution containing carbohydrates, proteins, fat, and other necessary nutrients through an indwelling catheter inserted into the superior vena cava.

In order to obtain the maximum benefit, the ratio of calories to nitrogen must be adequate (at least 100 to 150 kcal/gnitrogen), and both carbohydrates and proteins must be infused simultaneously.

TPN


Dextrose
Lipids
Amino acids
Electrolytes & minerals
Vitamins
Trace elements
TPN

PN Access – Peripheral vs. central



PN Access – Peripheral vs. central

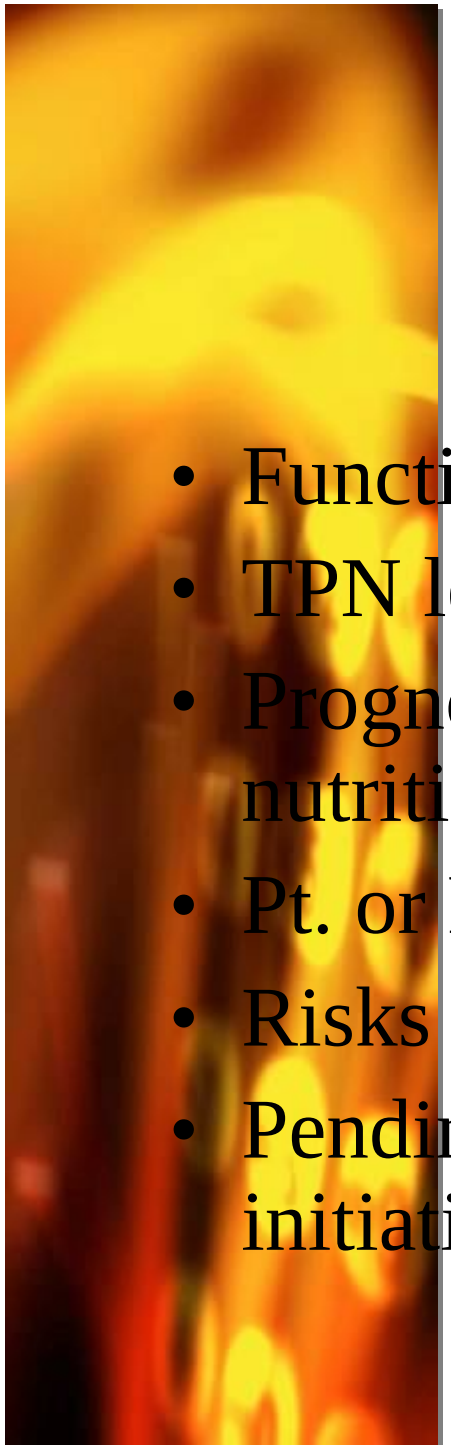
- The higher the osmolarity, the larger the vein needed to accommodate the solution
- A solution with high osmolarity infused into a small peripheral vein will cause irritation, pain, damage to the vessel, which requires frequent changes to the IV site
- Peripheral TPN not recommended > 7 d
- Peripheral TPN < 1100 mOsmol/l
- PICC preferred

Parenteral Nutrition - Indications

- Primary Therapy
  - Efficacy shown
    - ✓ GI-cutaneous fistula
    - ✓ Renal failure
    - ✓ Short bowel syndrome
    - ✓ Acute burns
    - ✓ Hepatic Failures
  - Efficacy not shown
    - ✓ Crohn's disease
    - ✓ Anorexia nervosa

Supportive therapy

- Efficacy shown
  - ✓ Acute radiation enteritis
  - ✓ Acute chemotherapy toxicity
  - ✓ Prolonged ileus
  - ✓ Weight loss preliminary to major surgery
- Efficacy not shown
  - ✓ Before cardiac surgery
  - ✓ Prolonged respiratory support
  - ✓ Large wound losses



Parenteral Nutrition - Contrindications

- Functional GI tract
- TPN less than 1 week in a well-nourished Pt.
- Prognosis does not warrant aggressive nutrition support
- Pt. or POA decline nutrition support
- Risks exceed potential benefits
- Pending surgery delayed to accommodate the initiation of TPN

Monitoring TPN

- Weekly weights
- Daily fluid balance, vital signs
- Visceral protein (albumin, prealbumin)
  - Consider half-life, change in fluid status, organ function and presence of infection
- Electrolyte and acid-base balance

Complications of TPN

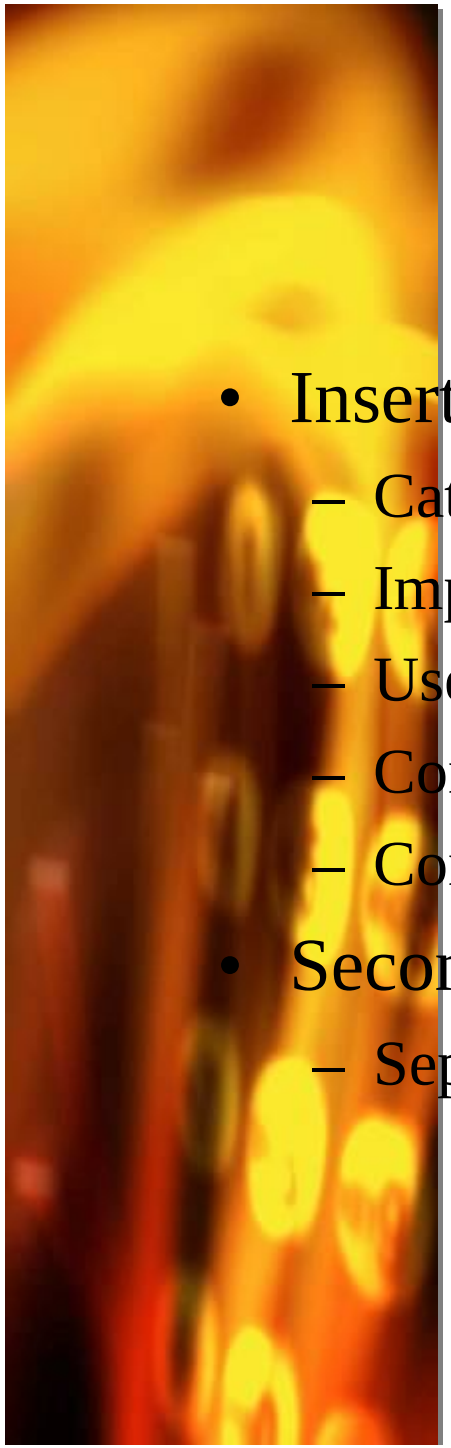
- Mechanical
- Infectious
- Metabolic

Mechanical Complications of TPN

- Related to vascular access technique
  - Pneumothorax
  - Air embolism
  - Arterial injury
  - Bleeding
  - Brachial plexus injury
  - Catheter misplacement
  - Catheter embolism
  - Thoracic duct injury

Mechanical Complications of TPN

- Related to catheter insitu
  - Venous thrombosis
  - Catheter occlusion
  - Dislodgement or breakage



Infectious Complications of TPN

- Insertion site contamination
  - Catheter contamination
  - Improper insertion technique
  - Use of catheter for non-feeding purposes
  - Contaminated TPN solution
  - Contaminated tubing
- Secondary contamination
  - Septicemia

Metabolic Complications of TPN

- Abnormalities related to excessive or inadequate administration:
  - Hyper and/or hypoglycemia
  - The refeeding syndrome
  - Electrolyte/acid-base disorders
  - Hyperlipidemia
  - Hepatic complications
  - Metabolic bone disease

The Refeeding Syndrome – What is it?

- The physiological alterations that are observed when an individual is refed after a period of starvation, either parenterally, enterally or orally
- The metabolic and physiologic consequences of the depletion, repletion, compartmental shifts and interrelationships of the following: PO_4, K, Mg, glucose metabolism, fluid resuscitation and vitamin deficiency

The Refeeding Syndrome

- The sudden provision of adequate or excess calories causes the body to convert to CHO metabolism as an energy source
- This precipitates a surge in the release of insulin
- Metabolic rate increases, as does O_2 consumption and CO_2 production
- Insulin stimulates the shift of PO_4, K and Mg from the serum into the cells as these minerals are required for energy metabolism

The Refeeding Syndrome

- As body stores are depleted, minerals that have moved from the serum into the cells cannot be replaced
- Critical levels of hypophosphatemia, hypokalemia and hypomagnesemia may develop with resulting cardiac and/or neuromuscular compromise
- Arrhythmia, CHF, acute respiratory failure and even sudden death may result
- Thiamine deficiency and intolerance of the glucose and fluid load administered contribute to the adverse results

The Refeeding Syndrome – How to feed patients at risk

- Do not attempt to immediately meet estimated energy and fluid goals
- Malnutrition does not develop over night and cannot be corrected in a matter of days
- Rapidly switching from a catabolic starved state to an anabolic refed state can overwhelm the functional capacity of the body

The Refeeding Syndrome – How to feed patients at risk

- Replete serum PO_4, K and Mg before initiating EN/PN
  - Hypomagnesemia may also result in hypokalemia
- Goal to meet requirements over a few days
- Use a “starter” solution which provides less calories and dextrose
- Progress volume of EN/PN after assessment of labs, ability to tolerate fluid volume, etc.