

Vesalius SCALpel™ : Nutrition/metabolism

Stress hypermetabolism: change thermoregulation, increase T, altered nutrition, metabolism increase proportional to injury up to 2X BMR max (physiologic reserve = how close to max), gut barrier dysfunction (translocation), increased O₂ consumption (VO₂), increased cardiac output

Early (ebb) phase: cardiac instability, hypovolemia (incr cap perm, 3rd space), impaired O₂ transport, increased autonomic activity, increased catechols and glucagons, increased glucose (nl. glu production), increase free fatty acids, low insulin, increased lactate, decreased O₂ consumption, hypothermia

Late (flow): increased glucose production and glucose, increased FFA, incr insulin, incr catechol, glucagons, lactate normal (being used for glucose production), O₂ consumption and cardiac output increased, increase core T
beta blockade to resting heart rate may decrease energy expenditure 20%

Thermoregulation:

reset/elevate central reference temp., increase heat production, temp warm environment saves patient energy, doesn't have to work as hard to maintain new temp.

Protein

muscle wasting: skeletal muscle main source of protein -> nitrogen (N) loss via kidneys generalized, proportional to injury, stress; peaks early 1-2d

other N loss: wd (esp. burn), blood, exudates, atrophy GI mucosa (slough), increase in protein synthesis by d2

free aa's released from skel M, mostly alanine (-> glutamine production in liver), glutamine (gut fuel, trophic for gut mucosa)

accelerated breakdown

benefits

precursors for glucose production

precursors for NH₃ production in kid (buffer for acidosis from early stress resp)

fuel (glutamine) for gut and other tissues

negative

M breakdown, debility

irreversible N loss from the kid as urea, NH₃, Cr, uric acid

oxandrone (synthetic testosterone analog) may increase protein synthesis, no effect on breakdown, no change in energy expenditure

Carbohydrate metabolism

glucose production increased from ebb to flow

glucose uptake increases, especially in injured areas

liver uses alanine and lactate as precursors for glucose production

glycogen stores depleted rapidly
insulin resistance develops even as insulin rises, glucose rises
glucose utilization won't meet caloric needs because can't get into cells, especially sepsis

N sparing

increasing carbohydrate intake (alternate source of energy) reduces N losses (greatest in sepsis)
upper limit 60-70% of total calories as carbs, max benefit

Fat metabolism

triglyceride hydrolysis -> FFAs, 2X increase, alternate fuel source
FFA oxidation rate doubles -> increase FFA production -> lactate
fatty acid infusion needed to meet caloric needs, good fuel source (except in early stress phase when can't anabolize)

Hormones

most trophic hormones increased in stress
increase in counterregulatory hormones: cortisol, glucagons, epi/norepi (sympathetic-adrenal axis important to stress response, if inadequate production -> circulatory collapse)

Cytokines

TNF: first cytokine released in stress, most important, lead hormone starts/orchestrates cascade -> IL1 -> IL2 -> IL6; role in proteolysis
IL1: major role in acute phase response, increases proteolysis via incr PGE2, increases vascular permeability
IL2: (role not fully understood) appears relatively early, may act as alternative or amplifying path; systemic effects similar to endotoxin/TNF (inappropriate increase -> systemic response/septic shock, circulatory collapse)
IL6: later release by tissue macrophages, important accessory path to IL1; aids in stimulating hepatic synthesis of acute phase proteins; at site of injury; activates leukotrienes; activates WBCs -> breakdown devitalized tissue, overcome infection

Modulators of stress response

good surgical care, nutritional support, thyroid hormone, GH?, antibiotics, ibuprophen (block eiconoid production via arachidonic acid cascade) (cyclooxygenase v lipooxygenase deleterious blockade; some prostaglandins lead to higher level leukotrienes, caution); adrenergic block, beta block, warm environment

Vesalius SCALpel™ : Nutrition

goal: provide substrates to maintain or restore physiologic homeostasis, eustasis, improve vital organ functn, restore immunocompetence
who needs: 30-40% below baseline weight M, 50% F, >15% past 3 mo benefit at outset;
anticipated illness >10d, incomplete gut recovery 7d PO
pre-op parenteral if indicated -> 10% decrease in morbidity if given min 7d
unselected pts.: increased infectious complications
pre-op enteral decrease M & M if given 10d
indicators of malnutrition: decreased lymphocyte count, alb < 2.5, anthropomorphic testing, (anergy: rarely used)
indications for support:
physiologically stable (don't use in early sepsis, shock), ABC's, O2, lytes, acid/base
estimate caloric needs:
30Kcal/Kg/d normally, 50 with stress (2,100Kcal/d for 70Kg M)
Harris-Benedict formula
disease v nutritional requirement
routine post op increase 10% over BMR
mild peritonitis or fracture 25%
severe injury, mult org dysfnctn syndr 50%
head injury 60%
burn >40% BSA 100%
protein needs
nl 0.8g/Kg/d, illness 2-3X, 1.5-2g/Kg/d, no benefit beyond 1.5 in stressed
typical N/cal ration 1:150
fatty acids:
linoleic & linolenic essential, exogenous only
deficiency -> dermatitis, alopecia
minimal amts req weekly to prevent deficiency
replace in anyone NPO > 1w
current commercial lipid preparations use omega 6 FAs, overload bad
omega 3's more beneficial, fishs oils: lower BP, protect against athero, increase insulin sensitivity, decrease resistance, increase appropriate immune response (decrease Reynauds, RA)

Vitamins

C: supports hydroxylation of proline and collagen matrix; free radical scavenger
D: hepatic stores minimal, depleted quickly in malnutrition
A, B, D, K: safe up to 10X RDA before toxicity; others (B12, niacin, C, E, biotin, folic acid, pantothenic acid) safe up to 50-100X
E: important antioxidant; deficiency may => anemia
trace elements:
Zn: alopecia, dermatitis, loss of taste; wound healing abnormalities
(fatty acid deficit also assoc w alopecia and dermatitis, but not loss of taste)
Fe: anemia, immune incompetence

Cu: anemia, neutropenia
Chr: hyperglycemia, neuropathy, encephalopathy
Se: cardiomyopathy

Routes of administration

Enteral: safer, cheaper, protect gut, 1Kcal/cc
in critically ill decreases infectious complications, fewer episodes hyperglycemia,
preserves gut-associated lymphoid tiss (GALT), decreases BT translocation
overall mort enteral=parenteral
in pancreatitis (J-tube enteral) decreases infection, no. ops, hosp stay
* too early enteral feeding associated with bowel ischemia (don't start if pt. shocky,
hypotensive, poor perfusion)
contraindications: GI output > 600/d, intestinal obstruction, intestinal ischemia, GI
bleed, (BS or flatus not required)
elemental not more beneficial
glutamine and fiber decrease bacterial translocation
hepatic formula: increased branch chain aa's, lower aromatic aa's (precursors of
neurotransmitters associated w encephalopathy)
renal formula: only beneficial for renal insufficiency, not pt on dialysis*
lack nonessential aa's promoting reuse of urea
necessary so don't lose so much protein
pulmonary formula: increased fat/carbohydrate ratio to reduce CO2
gut tolerance: begin slow, trial 30cc/h X 24
parameters suggesting failure: vomit, severe cramps, gastric residual > 50% of
previous 4h infusion, increasing distention, increasing diarrhea
aspiration risk: decreased sensorium (head, sedation, shock, encephalopathy)
severe GE reflux (theophylline, anticholinergics, Ca channel blockers, B
agonists, alpha antagonists), hx of prior aspiration
prevention: elevate head of bed, prokinetic agents, food coloring (blue) to ID
aspiration (cough up, ET tube aspirate)
complications
GI: diarrhea common
diarrhea risk: antibiotics, prokinetic agents, hyperosmolar formula, bolus
to small intestine, prolonged bowel rest, severe protein deficiency, c. diff,
malabsorption of fat
diarrhea Rx: kaopectate 30cc Q3h X 48; paragoric 1cc/dl formula X
24h, 50% decrease delivery rate, convert to TPN
metabolic: hyperglycemia
mechanical: clog, dislodge, leakage
infection: don't leave hanging at room T > 24h

Parenteral

Peripheral

initial feed until central access

supplement enteral feed if inadequate due to gut dysfunction
basal needs (1.5-1.8 Kcal/d) in a non-depleted pt who can tolerate 2.5-3L
fl/d

can't provide complete repletion peripherally because osmotic load precludes
giving enough calories

600mOsm (double serum) = sl osmolar
calorie density 0.3-0.6 Kcal/cc, 3-4L/d (1/3 of enteral)

rotate IV site Q 48-71h

rare mechanical, septic complications

Central/TPN: 3 in 1, protein, carbo, fat

osmolarity 1,900mosm/L; N/cal 1:154; cal density 1.15Kcal/cc; 2400cc/d
+lytes, vitamins, trace elements

lytes tailored to losses or renal insufficiency

severe renal, liver insuffic require less trace

alcoholics, dialysis pts, pancreatic insufficiency require additional Zn

biliary obstruction require less Cu and Mn (not losing as much in stool)

Fe contraindicated in sepsis (supports bacterial growth, restart after sepsis)

monitoring:

glucose, lytes, coags, prealbumin, transferrin, retinol binding protein

hyperglycemia sign of possible infection

triglycerides (pt can't utilize > 20% FA)

meticulous cath care:

72h trial antibiotics to normal T, if not remove

true cath infection if quant cult cath tip and blood

cult both pos, if only cath = colonization

orgs mostly from skin: s. aur, epi, candida

2% chlorhexidine more effective than alcohol or iodine

for prep and care

hydrophobic BT exopolysaccharide allows attachment,
resistance

fibrin, fibronectin receptors for attachment

femoral line increased colonization

no use for prophylactic antibiotics

withhold TPN 24h after removal for cath sepsis

branched chain aa's: leucine, isoleucine, valine

little metabolized by liver, good in liver disease

increased pro synth by 50%, decreased degradation by 25%

physiologically significant only during stress or injury -> pos N bal
and decreased mort

requires doubling aa concentration to 46%

glutamine: most abundant aa in body, gut fuel

important precursor of glutathione: most abundant/important free
radical scavenger in body

large quantity of glutamine in RBCs

massive initial release of glutamine from skel. M in stress & injury,

support, replete with exogenous
not in standard aa prepa, less stable, omitted to prolong shelf life
improves N bal, lymphocyte counts, decrease gut and M atrophy,
decrease infectious complications, decr hosp stay, mort

arginine

N rich, required for growth promoting products (glu, insulin, GH)
potent secretagogue of GH, insulin, glucagon, prolactin, somatostatin
stimulates wd healing, enhanced cellular immunity
inhibits tumor growth by nonspecific immunomodulation & by
augmenting response to immunoregulatory cytokines (IL2)

fatty acid supplement:

linoleic, linolenic essential, not synthesized, need for inflammatory
mediators, (arachidonic, leukotriene B4), immunity, decreased
infection

deficiency: alopecia, scaly skin, delayed wound healing
highest calorie content, 9Kcal/g, can supply 35% of energy requirement
in the face of insulin resistance

omega 6 FA's increase eicosanoids include PGE2 and
leukotriene B4, in XS are immunosuppressive

omega 3 increase M mass, enhance cell mediated immunity response,
opsonic indices, transferrin levels, decrease sepsis, mortality

periop TPN

some benefit GI malignancies (mainly esophagus) if the patient comes in
depleted, higher complication rate; if nutrition normal, no diff

References:

Hall J. nutritional assessment of surgery patients. JACS, 202(5), May '06: 837-843.