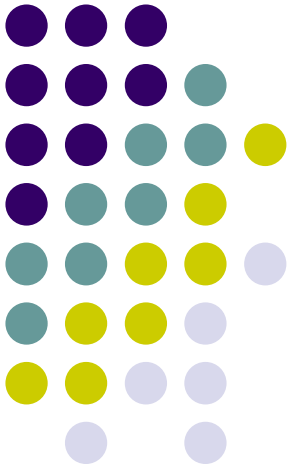
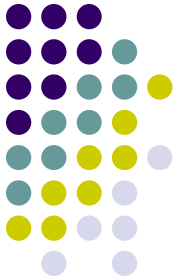
Nutrisi pada pasien critical ill

Oleh : dr. Susmiati



Perubahan metabolisme pada pasien multiple trauma



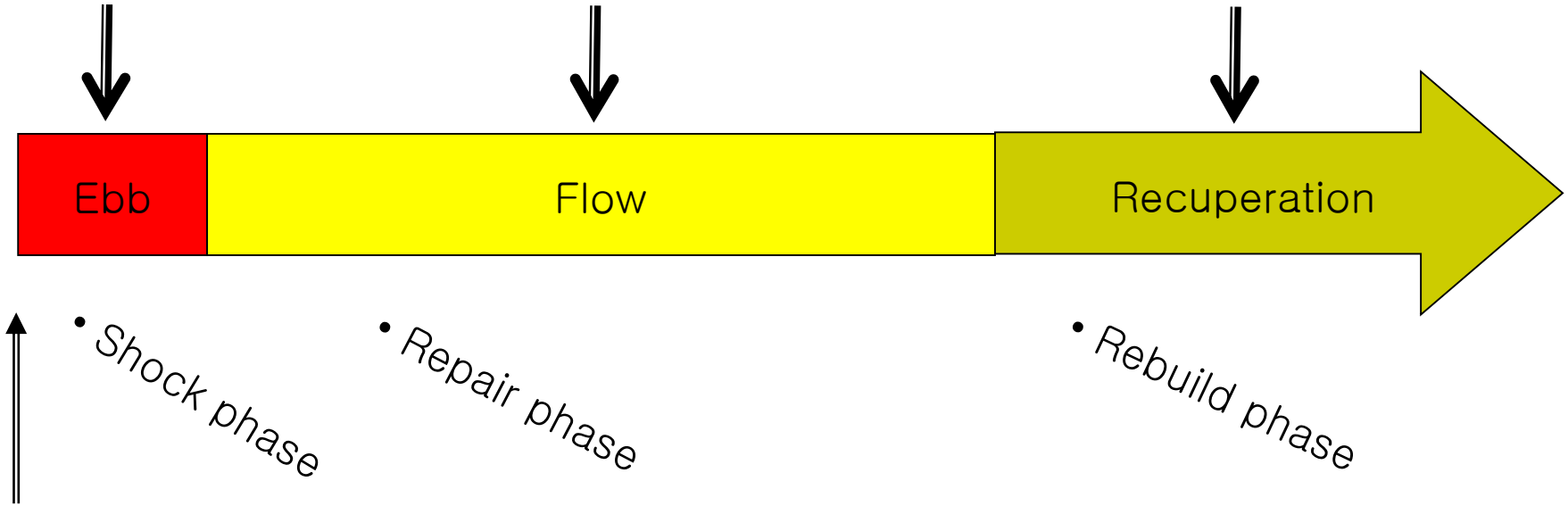
- Peningkatan kebutuhan energi dan zat gizi lain
- Perubahan metabolisme karbohidrat, lipid dan protein

Fase post trauma

Hemodynamic stabilization
-Fluid resuscitation

Hypercatabolism control & support
-Anti-inflammation
-Nutrition support

Anabolism support
-Nutrition
-Rehabilitation



Respon metabolik pada trauma

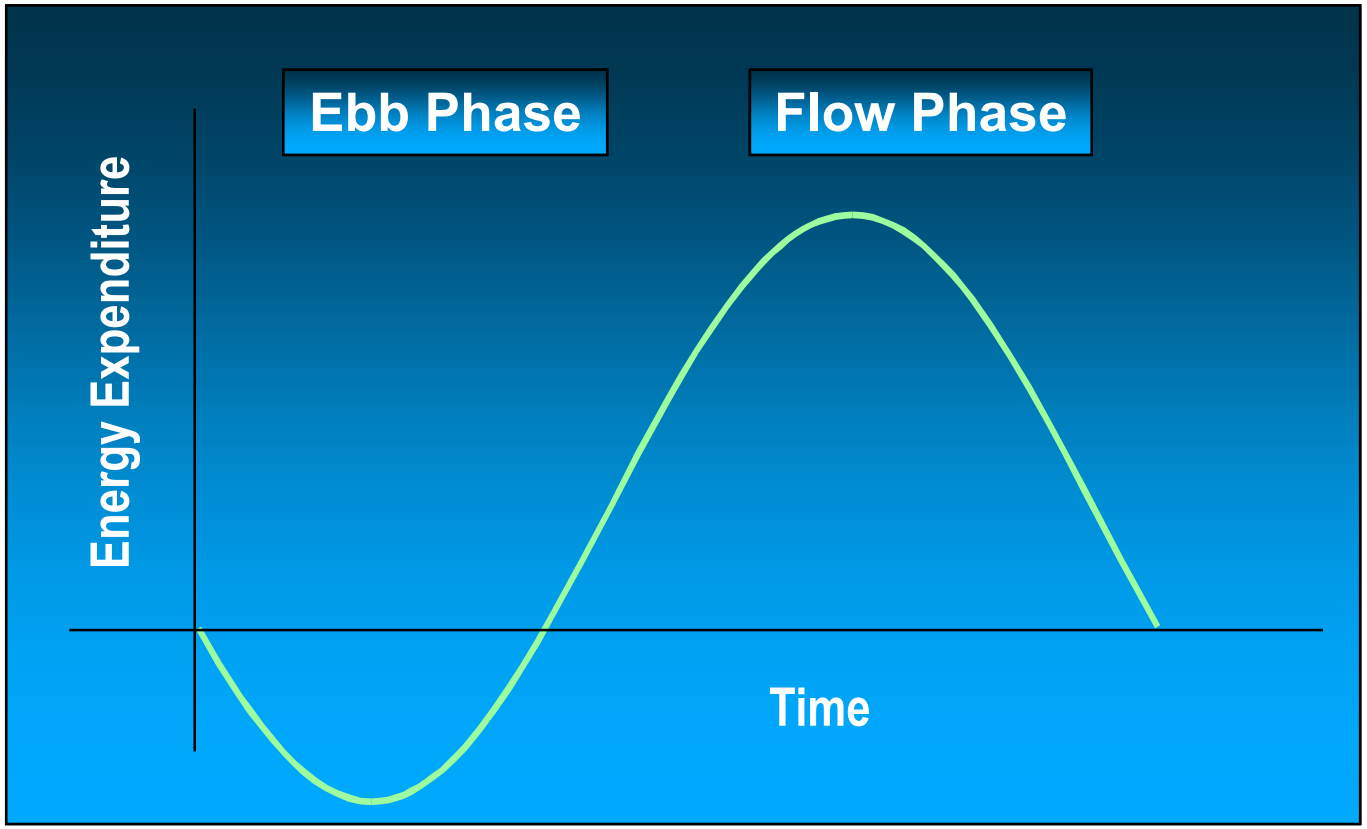
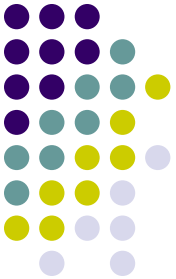


TABLE 42-1 Characteristics of Metabolic Phases Occurring After Severe Injury

EBB-PHASE RESPONSE	FLOW PHASE	
	ACUTE RESPONSE	ADAPTIVE RESPONSE
Hypovolemic Shock	**Catabolism Predominates**	**Anabolism Predominates**
↓ Tissue perfusion	↑ Glucocorticoids	Hormonal response gradually diminishes
↓ Metabolic rate	↑ Glucagon	↓ Hypermetabolic rate
↓ Oxygen consumption	↑ Catecholamines	Associated with recovery
↓ Blood pressure	Release of cytokines, lipid mediators	Potential for restoration of body protein
↓ Body temperature	Production of acute-phase proteins	Wound healing depends in part on nutrient intake
	↑ Excretion of nitrogen	
	↑ Metabolic rate	
	↑ Oxygen consumption	
	Impaired use of fuels	

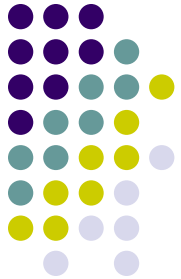
From *Enteral nutrition support in critical care*, Columbus, Oh, 1994, Ross Products Division, Abbott Laboratories.

Respon metabolik pada trauma :
Ebb Phase



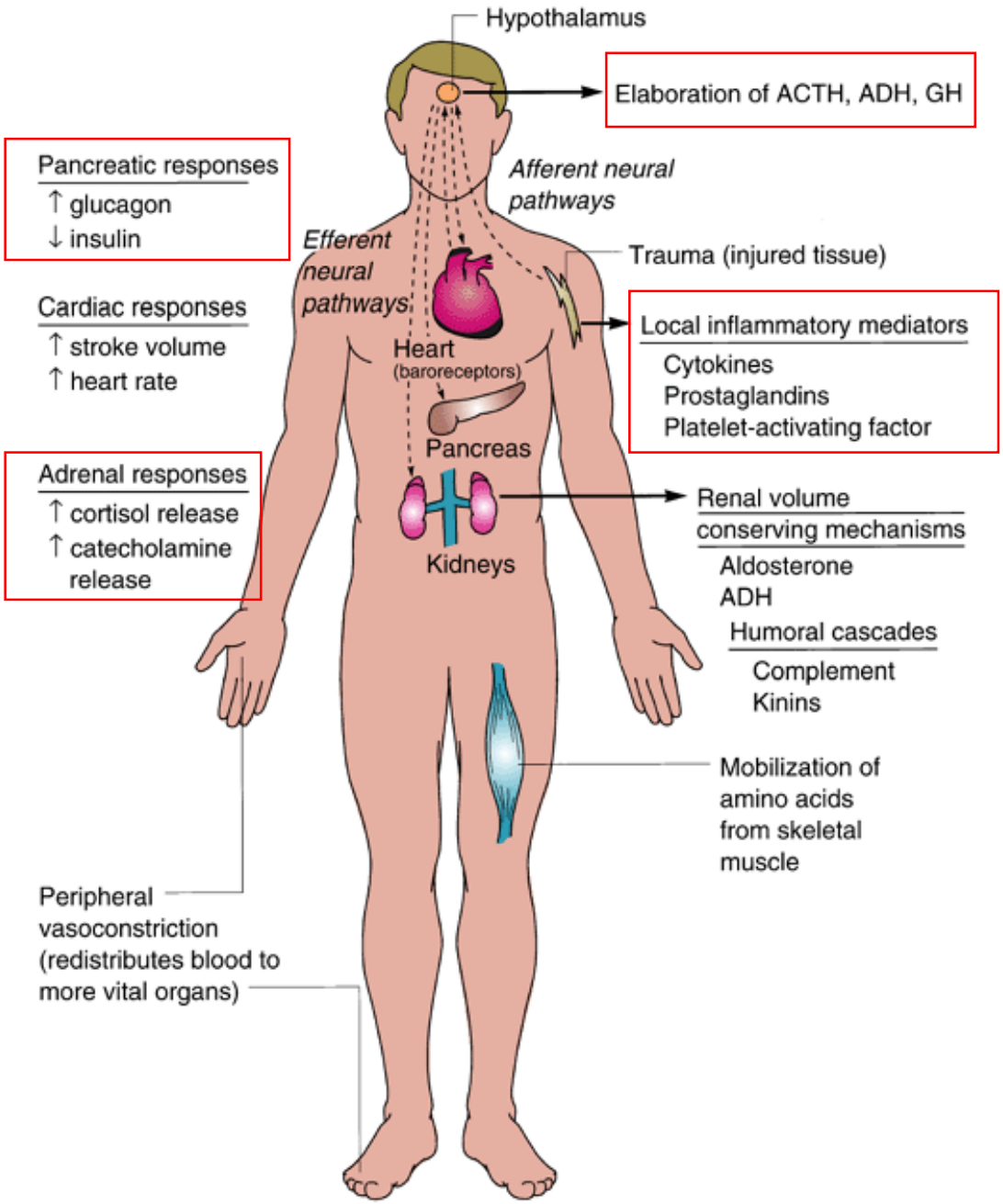
- Hipovolemic shock
- Terjadi penurunan
  - cardiac output
  - konsumsi oksigen
  - Tekanan darah
  - Perfusi jaringan
  - Suhu tubuh
  - Metabolik rate

Respon metabolik pada trauma : Flow Phase



- Peningkatan
  - catekolamin
  - Glukokortikoid
  - Glukagon
- Release citokin. Lipid mediator
- Produksi akut phase protein

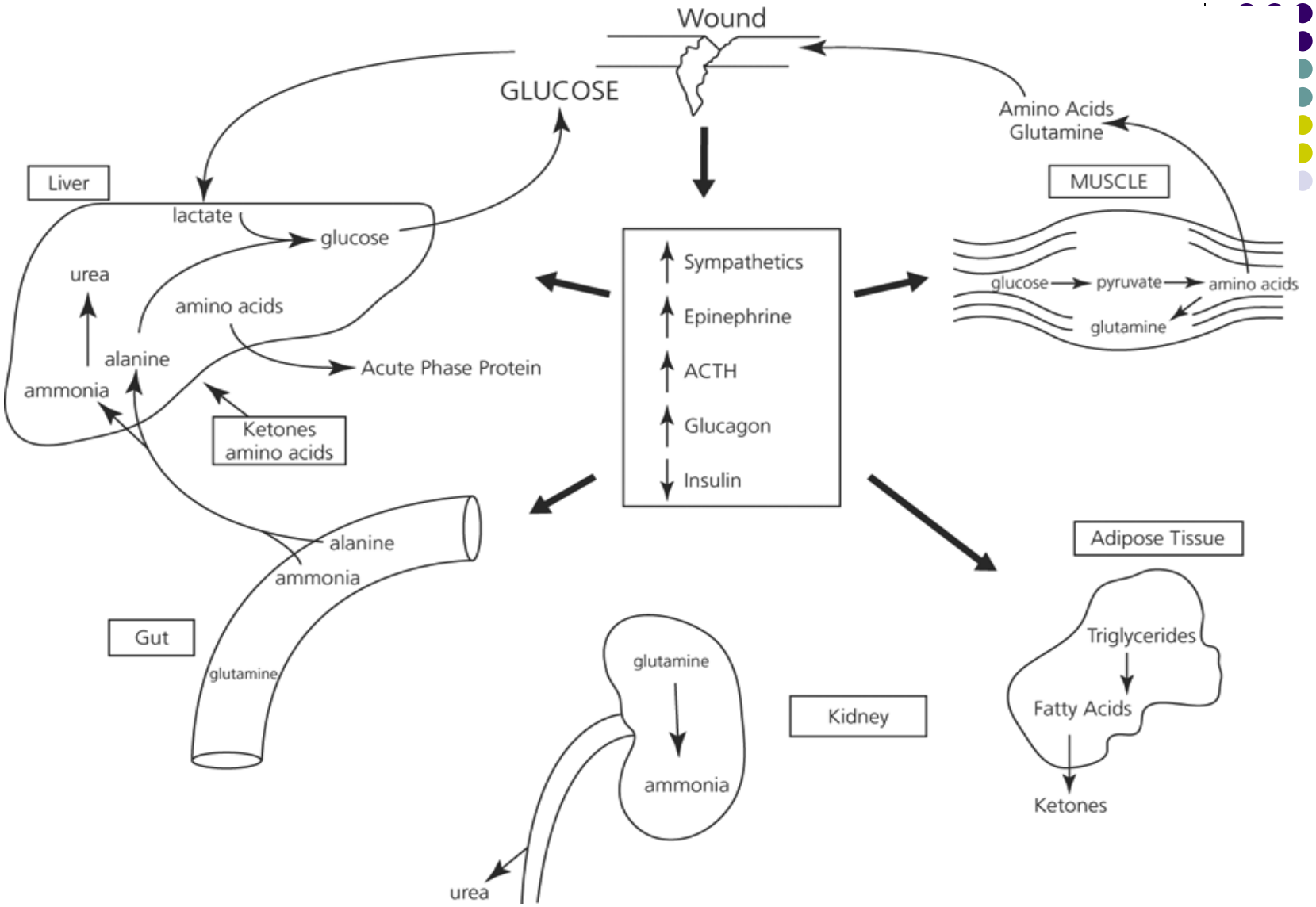
Homeostatic Adjustments Initiated after Injury.



Respon metabolik pada trauma

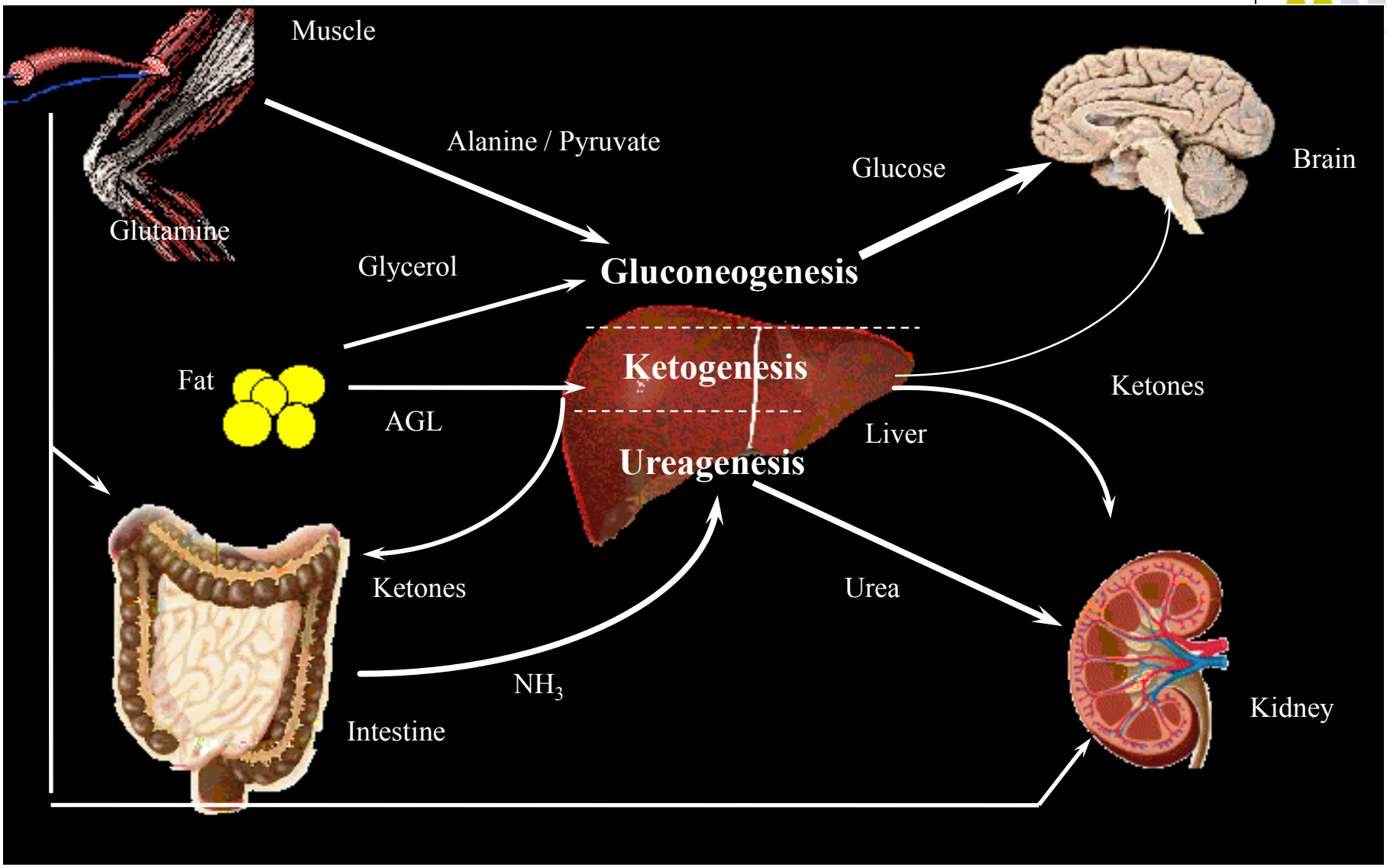
Organ	Response
liver	↑ glucose production , AA uptake , acute-phase protein synthesis trace metal sequestration
Central nervous system	Anorexia , fever
Circulation	↑ Glucose , TG ,urea ↓ AA, iron, zinc
Skeletal muscle	↑ AA efflux (especially glutamine) leading to loss of muscle mass
Intestine	↓ AA uptake from both luminal and circulating sources , leading to mucosal atrophy
Endocrine	↑ ACTH, cortisol , GH, epinephrine , norepinephrine , glucagon , insulin

Respon metabolik pada trauma



Konsekwensi Neuroendocrine & metabolic dari trauma

Perubahan metabolik setelah trauma



Pengaruh perubahan endokrin

1. Catecholamines (epinephrine and norepinephrine)
   - merangsang glycogenolysis dan gluconeogenesis di hati
   - merangsang katabolisme otot (proteolysis)
   - merangsang lipolysis
   - menghambat sekresi insulin dan uptake glucosa oleh jaringan

2. Glucocorticoids (cortisol) : dihasilkan oleh kortex adrenal dirangsang oleh ACTH (adrenocorticotropic hormone)

- merangsang lipolysis
- merangsang katabolisme otot (proteolysis
- merangsang gluconeogenesis (hepatic use of AA)
- menghambat protein synthesis
- menghambat sekresi insulin
- merangsang sekresi glucagon

3. Glucagon

- merangsang gluconeogenesis and glycogenolysis
- merangsang lipolysis and proteolysis

Cytokine – Interleukins(IL-1,IL-6),tumor necrosis factor (TNF)

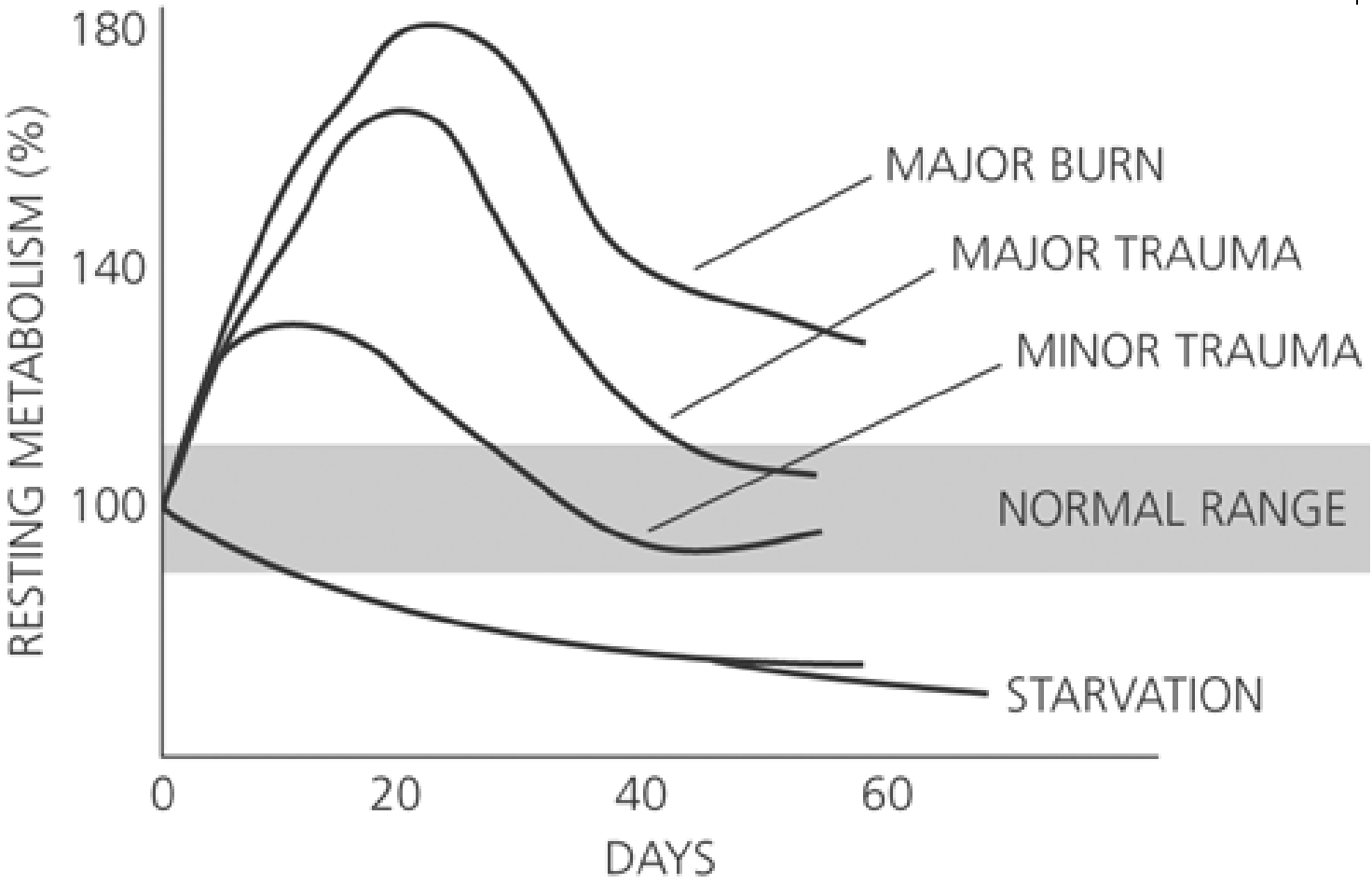
- Dihasilkan oleh sel fagosit sebagai respon kerusakan jaringan, infeksi, obat, bahan kimia
- Cytokines memberi eek metabolik
  - * merangsang uptake AA oleh hati (protein synthesis)
  - * mempercepat pemecahan otot (muscle breakdown)
  - * meningkatkan eksresi nitrogen
  - * meningkatkan leukocyte count
  - * anorexia
  - * fever
  - * redistribusi trace minerals dalam plasma

Major Cytokines Involved in Hypermetabolic Response

Cytokine	Cell source	Metabolic effects
TNF-α	**Monocytes/macrophages**, lymphocytes, Kupffer, glial, endothelial, natural killer, & mast cells	↓ Decrease free FA. synthesis ↑ lipolysis ↑ peripheral AA.s efflux ↑ hepatic AA uptake & acute-phase protein synthesis ↑ body temperature ↑ insulin-resistance
IL-1	**Monocytes/macrophages**, neutrophils, lymphocytes, keratinocytes, Kupffer cells	↑ ACTH hormone ↑ acute-phase protein synthesis ↑ body temperature
IL-6	**Monocytes/macrophages**, keratinocytes, fibroblasts, endothelial, T, & epithelial cells	↑ acute-phase protein synthesis ↑ body temperature
IFN-γ	**Lymphocytes**, pulmonary macrophages	↑ TNF-α production ↑ monocyte respiratory burst

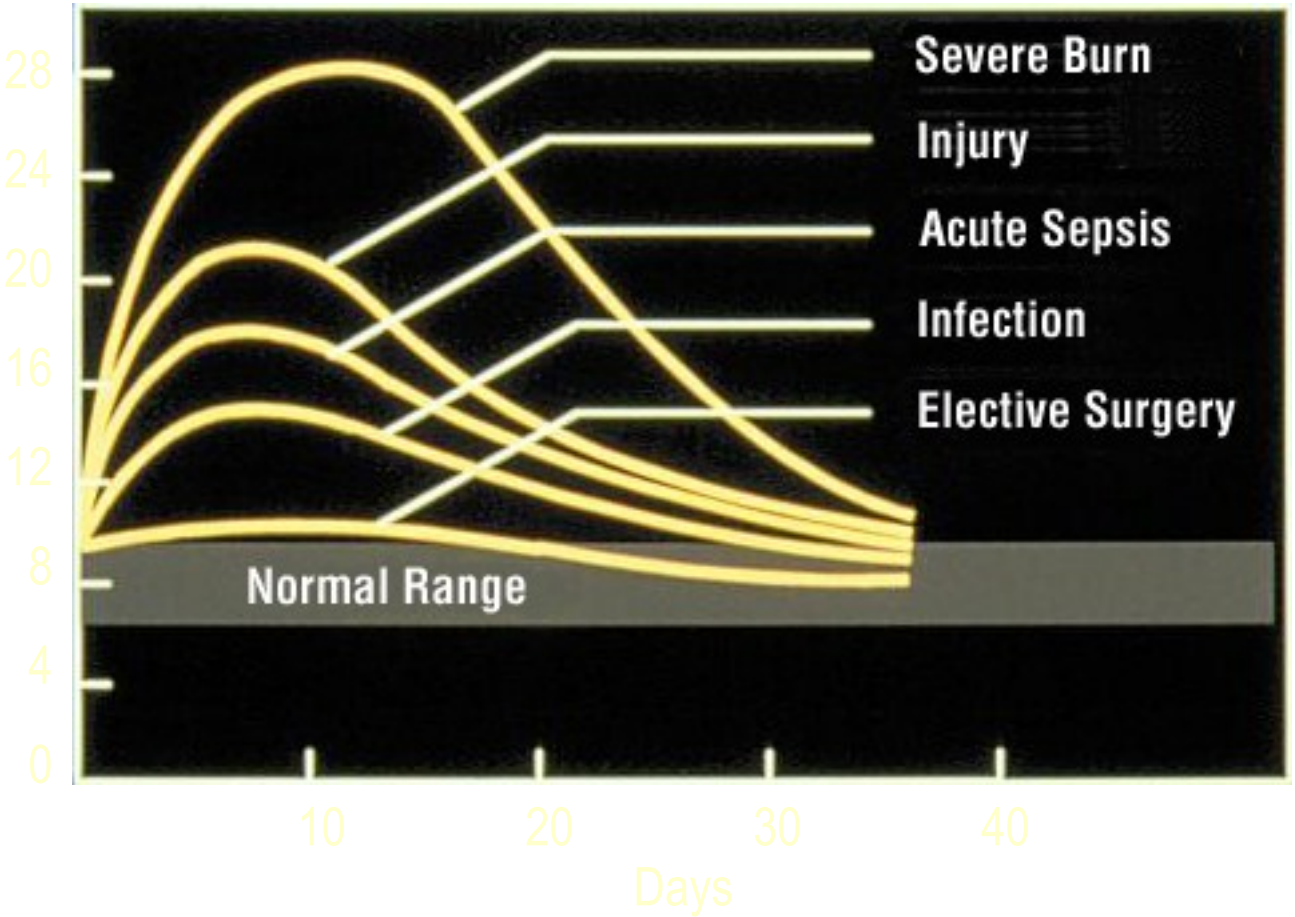
From Matarese G, La Cava A. The intricate interface between immune system and metabolism. Trends Immunol 2004;25:195–;6, with permission.

BMR pada berbagai tingkat trauma

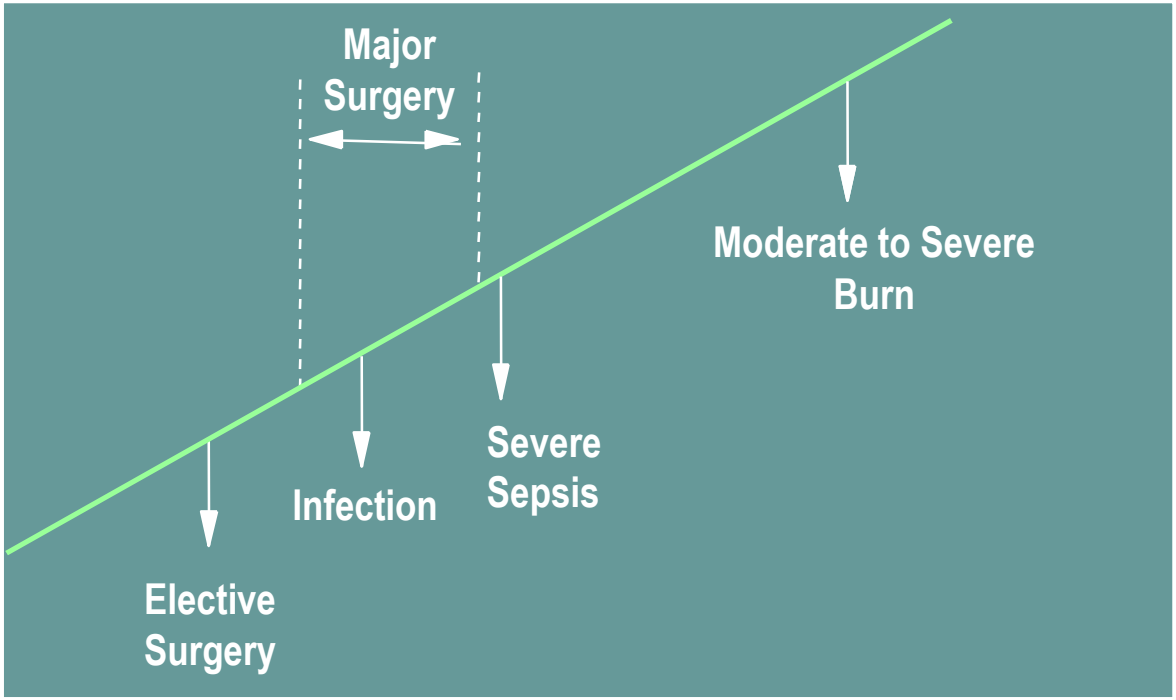


Effect of injury on metabolic rate. (Adapted from Wilmore DW. The Metabolic Management of the Critically Ill. New York: Plenum Medical Book, 1977)

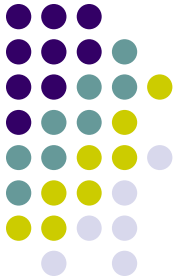
Respon metabolik pada trauma



Tingkat keparahan trauma : efek nitrogen Losses dan laju metabolisme



Penentuan kebutuhan kalori



- Kalorimetri indirect
- Harris-Benedict x stress factor x activity factor
- 25-30 kcal/kg body weight/day

Kebutuhan energi

- **TEE (total energy expenditure)**

(1) BMR (basal metabolic rate)

(2) efek aktifitas

> efek miimal pd pasien critical ill

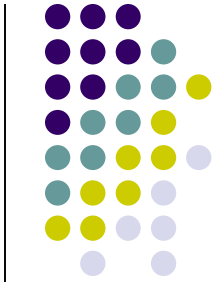
> except self-ventilating , tachypnoea , severely agitated.

> penurunan kebutuhan pd muscular paralysis 30% ,.

(3) SDA

> 10 % untuk diet campuran

Perhitungan berdasarkan BB



- 25-35 kcal / kg
  (1) 25-30 kcal / kg
  (well-nourished , elective operation)
  (2) 35 kcal / kg
  (multiple trauma)
- 25-35 kcal / kg actual BW
  (1) 30 –35 kcal /kg (septic and SIRS)
  (2) 25 –30 kcal /kg (non-septic and SIRS)
- ABW (adjusted BW) =
  (acutual BW - IBW * 0.25) + IBW
- Cachetic, marasmic
  → actual BW to assess needs

Kebutuhan nutrisi pada berbagai keadaan

Injury	Stress Factor
Minor surgery	1.00 – 1.10
Long bone fracture	1.15 – 1.30
Cancer	1.10 – 1.30
Peritonitis/sepsis	1.10 – 1.30
Severe infection/multiple trauma	1.20 – 1.40
Multi-organ failure syndrome	1.20 – 1.40
Burns	1.20 – 2.00

Activity	Activity Factor
Confined to bed	1.2
Out of bed	1.3

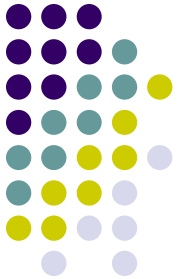
Contoh :

Kebutuhan energi untuk penderita cancer(in bed)

= BEE x 1.10 x 1.2

Kebutuhan kalori

(rata-rata untuk laki-laki 70 kg)

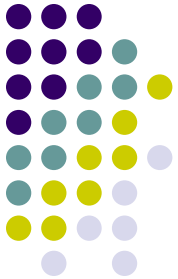


Proses penyakit	**kcal/day**
Basal	1,450
Post-op. (uncomplicated)	1,500–1,700
Sepsis	2,000–2,400
Multiple trauma (ventilator)	2,200–2,600
Major burn	2,500–3,000

Biasanya kebutuhan energi meningkat sebanding dengan tingkat keparahan penyakit

Kebutuhan protein

(rata-rata untuk laki-laki 70 kg)

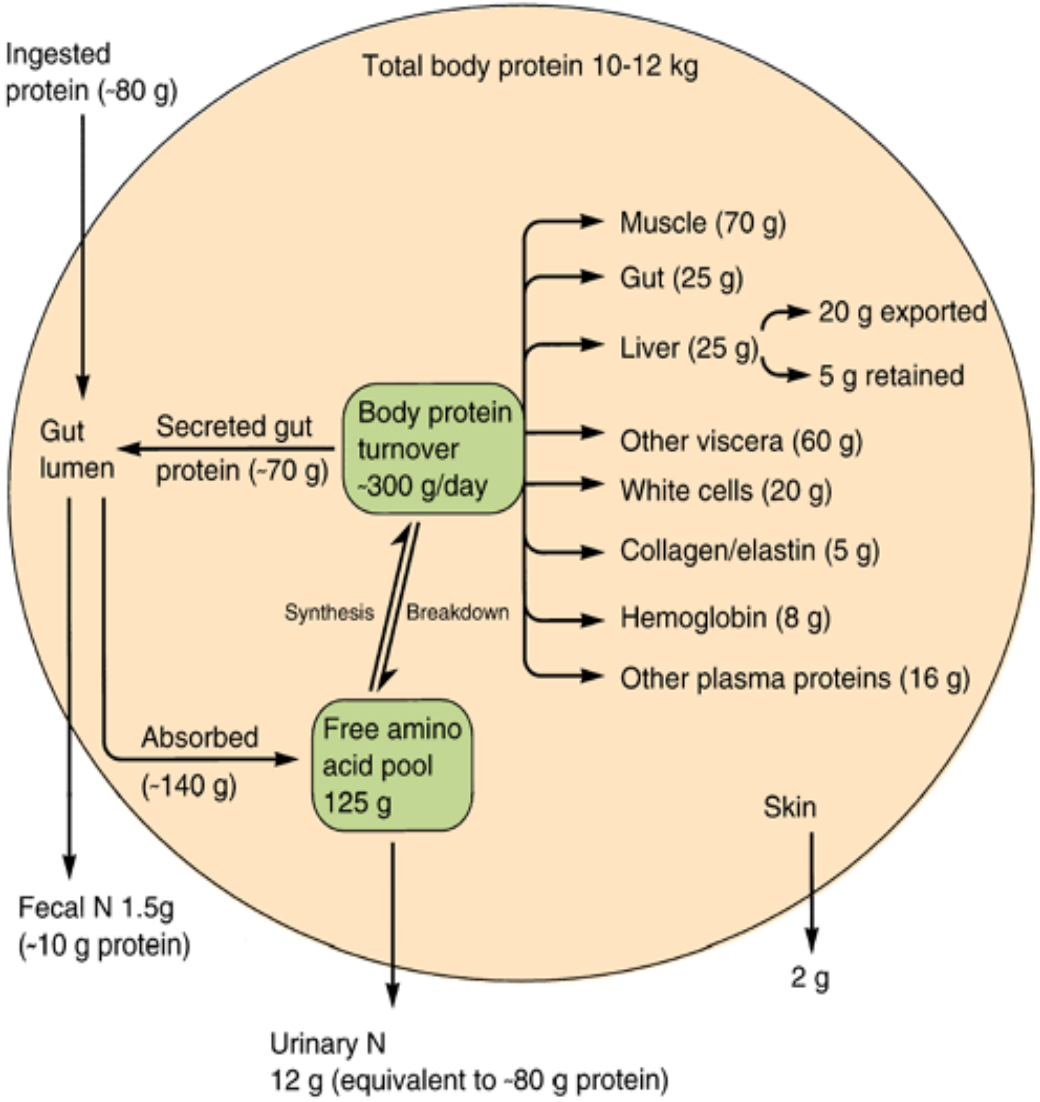


Proses penyakit	**Amino acids (kg/day)**
Basal	0.8–1.0
Postop (uncomplicated)	1.0–1.5
Sepsis	1.5–2.0
Multiple trauma (ventilator)	1.5–2.0
Major burn	2.0–3.0

(1 g of N_2 = 6.25 g of protein)

Metabolisme protein normal

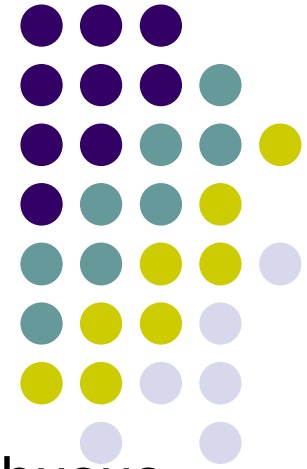
(rata-rata untuk laki-laki 70 kg)



Kebutuhan nutrisi

Prinsip :

- Hindari overfeeding
- Kebutuhan energi
- Kebutuhan protein
- Kebutuhan karbohidrat
- Vitamins and Minerals
- Kebutuhan Energy and protein pada penyakit khusus
- Makanan khusus untuk pasien critically ill



Hindari overfeeding

- **Respiratory quotient (RQ)**

CHO	→	**1**
Fat	→	**0.7**
Protein (PT)	→	**0.81**
Alcohol	→	**0.67**

Kelebihan CHO dapat menyebabkan

(1) Steatosis dari hati

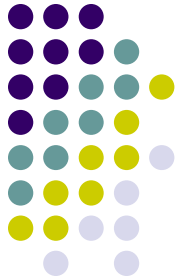
Glucose → glycogen

(stores are replete ,about 400 g)

Glucose → fat (lipogenesis , CO_2 production)

(2) hyperglycemia

(3) keterlambatan weaning dari ventilator



Kelebihan fat > 50 % of total calories

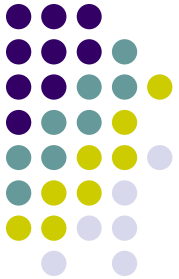
(1) overload the reticulo-endothelial system (RES)

TG → glycerol + free fatty acids

reduce RES clearance

(2) kegagalan pertukaran gas alveolar

Kebutuhan protein



- 1.2 –2 g protein /kg BB

- Kcal : N ratio

  300: 1 (healthy adults)

  150: 1 (moderate stress)

  80 –100 : 1 (severe stress)

UUN(urine urea nitrogen)

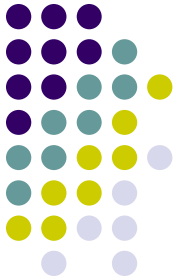
- **Assess the degree of hypermetabolism (stress)**
  - **UUN : 0 – 5 no tress**
  - **UUN : 5 – 10 mild hypermetabolism/level 1 stress**
  - **UUN : 10 –15 moderate hypermetabolism/level 2 stress**
  - **UUN : > 15 severe hypermetabolism/level 2 stress**
- **Estimate protein requirement**
  - **UUN : 10 (1.2 –1.3 g protein/ kg BW)**
  - **UUN : 25 (2 g protein/ kg BW) (Kcal :N ratio :90:1)**

Perkiraan kebutuhan nitrogen per kg actual BB/hari

		Nitrogen (protein)
• Normal		0.17g (1.0625 g)
• Hypermetabolic	5-25 %	0.2 g (1.25 g)
	25 –50%	0.25 g (1.5625g)
	> 50 %	0.3 g (1.875 g)

- *Note: maksimum jumlah nitrogen yang dapat dimetabolisme 18 g /hari (112.5 g protein).*

Kebutuhan karbohidrat



- Jumlah CHO berhubungan dengan kemampuan hati untuk oksidasi
- 60 –70 %dari energi
- Parenteral nutrition

kecepatan Maximum oksidasi glucose :

5 –7 mg /kg BB / min , 7.2 g / kg BB / hari

umumnya: 2-5 mg /kg BB/ min

Atau 3-7 g CHO /kg BB/hari

Kebutuhan lemak

- 15 –40 % dari energi
- Untuk pasien critically ill ,kebuthan 0.8 –1 g /kg BB/hari
- 3 karakteristik sbg sumber energi

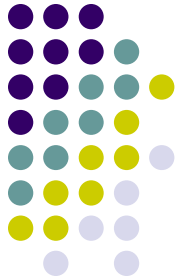
  1. concentrated

  2. isotonic (toleration of tube feedings,particularly into the lower duodenum or jejunum)

  3. nonglucose

  (terbatasnya jumlah isulin dan penggantian lemak dari CHO untuk membatasi produksi CO_2 untuk weaning ventilator)

Vitamins and Minerals



- **Tidak ada rujukan spesifik**
- **Berdasarkan RDA**
- **Perhatian :**

> peningkatan kebutuhan B complex (thiamin , niacin) bersamaan dengan peningkatan kalori

> peningkatan kebutuhan K , Mg , P , Zn

- Vit A 3300 IU
- Vit D 200 IU
- VitE 10 IU
- Vit C 100 mg
- Folacin 400 mcg
- Niacin 40 mg
- Riboflavin 3.6 mg
- Thiamin 3 mg

- Vit B6 4 mg
- Vit B12 5 mcg
- Pantothenic acid 15 mg
- Biotin 60 mcg
- Copper 0.5-1.5 mg
- Chromium 10-15 mcg
- Manganese 0.15-0.8 mg
- Zinc 2.5 - 4 mg

- **Penambahan jumlah zinc direkomendasikan pada kondisi :**
- **1. kehilangan yg banyak cairan usus**
- **2. ileostomy drainage**

Kebutuhan Energy and protein pada penyakit khusus

•Penyakit hati

Kondisi klinik	Energy (kcal/kg/day)	Protein (g/kg/day)
Compensated cirrhosis	30-40	1-1.2
Complications,inadequate intake, malnutrition	40-45	1.5
Encephalopathy grade I-II	30-40	Transiently 0.5, then 1-1.5
Encephalopathy grade III-IV	30-40	0.5-1.2

XVIII ESPEN Consensus Conference on Nutrition and Liver Disease,September 1996.

BCAA (valine,leucine,isoleucine) digunakan pd penyakit hati kronik

- **Akumulasi AAA pd plasma dan otak dapat menyebabkan kerusakan yg berat pd sintesis neurotransmitter otak => hepatic encephalopathy.**
- **BCAA berkompetisi dgn AAA pd transpor darah otak untuk mengatasi koma.**
- **Penggunaan jangka lama dapat menyebabkan penurunan tyrosine and cysteine level dan penurunan nitrogen balance**

- **AAAs(aromatic AA):**
  phenylalanine, tyrosine, tryptophan

•Penyakit ginjal

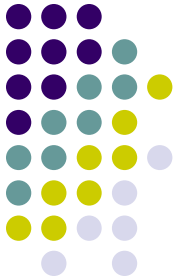
Therapy	Energy (kcal/kg/day)	Protein (g/kg/day)
Continuous haemofiltration/ diafiltration dialysis	30-35	1 – 1.2
Intermittent haemodialysis haemofiltration/diafiltration	30-35	1 – 1.2
Non-dialysed/filtered (residual renal function, minimal catabolism	30-35	0.55 – 0.6

BW: actual BW

Trauma kepala (head injury)

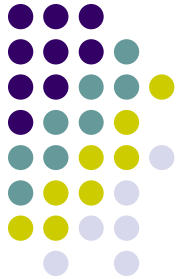
- Peningkatan BMR pd HI akut dapat mencapai 40% ,
- 1.5-2.5 g (2.2 g ,ref 3) protein / kg actual BW /day
- 20 –30 % increase in energy above BMR using formula .

Makanan khusus untuk pasien critically ill



- Glutamine
- Arginine
- Nucleotides
- W-3 fatty acids
- MCT (medium chain triglyceride)
- Structured lipids
- SCFA
- Antioxidant

Glutamine (GLN)



- **Normal intake : 4-5 g /day**
- **Fungsi :**
  > The principle fuel for rapidly dividing cells of the small intestine and immune system e.g. enterocytes , lymphocytes. (as a fuel by the gut in the criticall ill).
  > A trophic factor to maintain of the gut mucosa
  > A precusor of nucleotides , I.e. DNA and RNA

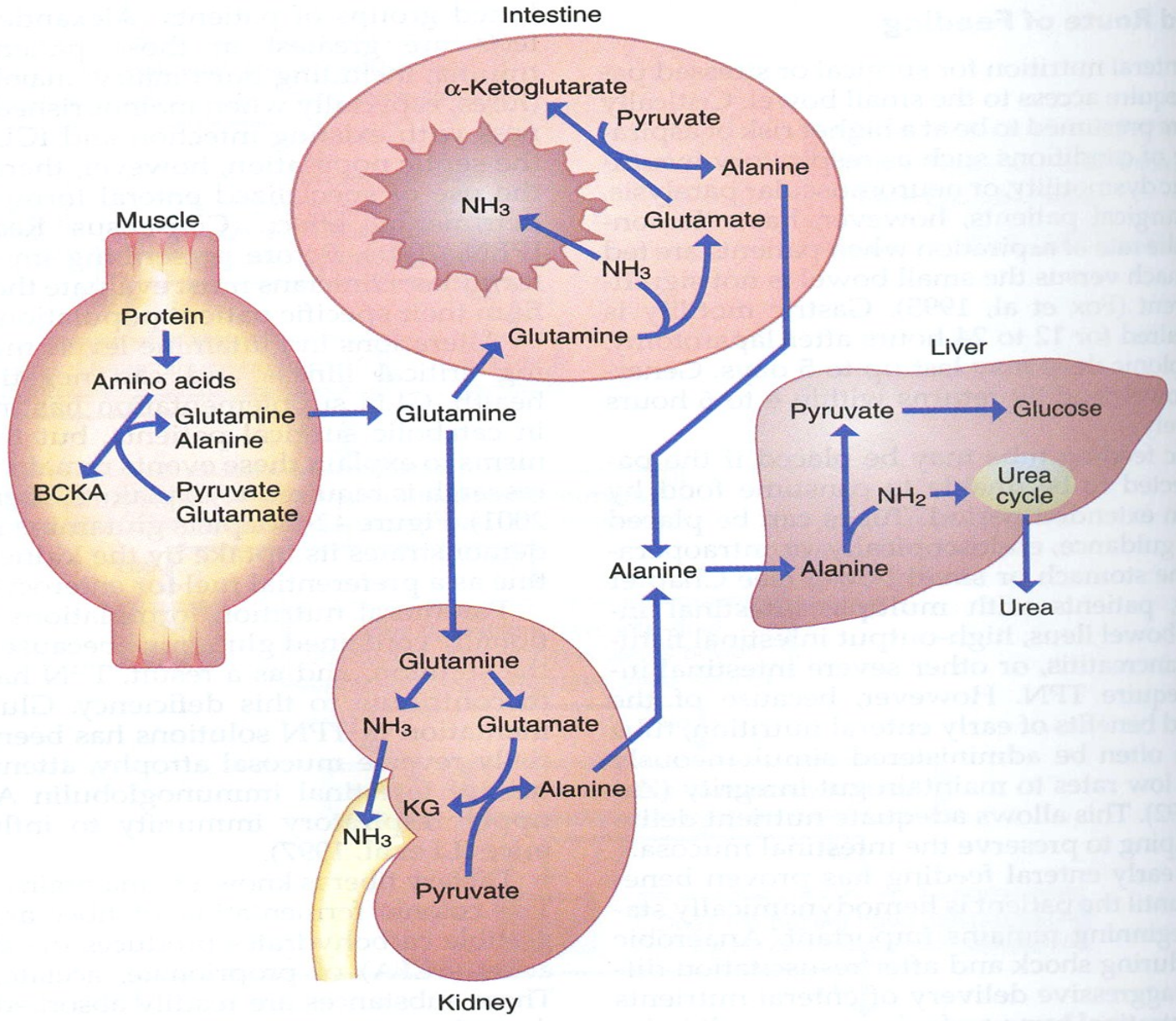


FIGURE 42-8 • Glutamine metabolism. Glutamine is generated by skeletal muscle from glutamate by transamination. Glutamine is taken up by the intestine and kidney, where deamination and ammonia elimination occur. The glutamate formed is transaminated with pyruvate to form alanine, which goes to the liver for gluconeogenesis, and α-ketoglutarate (KD), which can be used for energy production by the muscle or kidney. *NH_2,* Amine; *NH_3,* ammonia. (From Simmons RL, Steed DL: *Basic science review for surgeons,* Philadelphia, 1992, WB Saunders.)

Pathophysiology of Critical Illness

Insult

- infection
- trauma
- I/R
- hypoxemic/ hypotensive

Activation of PMN's

Role of GIT

endothelial dysfunction

activation of coagulation/complement

elaboration of cytokines, NO, and other mediators

generation of OFR (ROS + RNOS)

mitochondrial dysfunction

= oxidative stress

cellular = energetic failure

organ = failure

Death

Key nutrient deficiencies (e.g. glutamine, selenium)

Arginine

- **Normal intake : 5.4 g L-arginine /day (average)**
- **Conditional EAA (an immunomodulating effect in the critically ill to support the immune response)**

Nucleotides

- **Fungsi :**
  - **> sbg prekursor DNA and RNA**
  - **> peningkatan sintesa protein**
  - **> regulasi beberapa T-cell-mediated immune responses.**

W-3 FA

- Perbandingan W-3 FA / W-6 FA daat mempengaruhi produksi eicosanoids sebagai imun respon
- Fungsi :

> menghambat produksi prostglandin PG2.

> menurunkan thromboxane

(thromboxane adalah eicosanoid yg berperan penting dlm menjaga tekanan vaskular dan agregasi platetlet)

MCT

- **Kelebihan**

  1. menurunkan hyperlipidemia and hepatic steatosis)

  2. oksidasi cepat dan sempurna

  3. pencernaan dan absorsi tanpa memerlukan lipase pancreas dan empedu

- **Sumber : coconut oil , palm kernel oil**

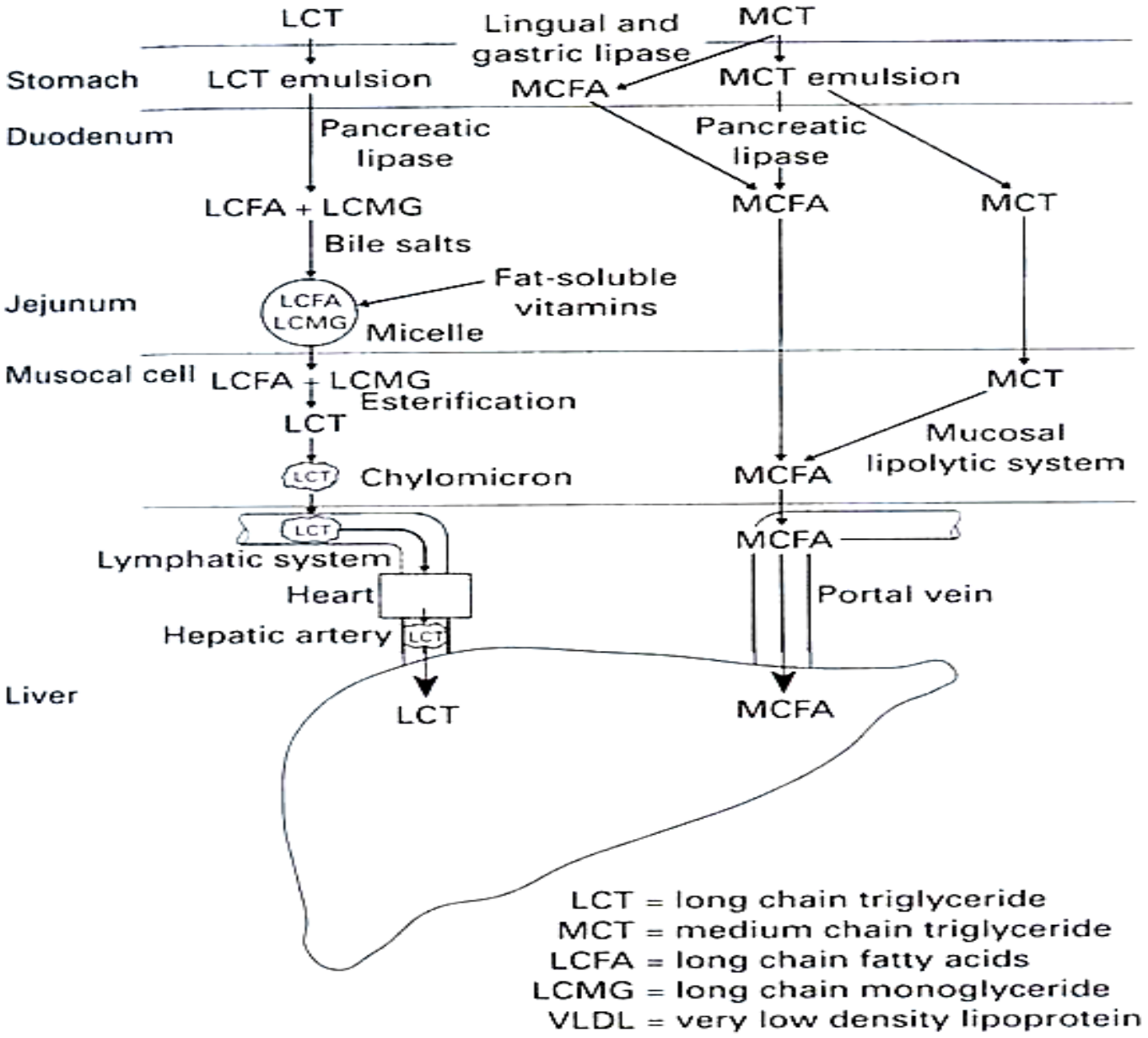


Fig. 2.8 Absorption and metabolism of long-chain and medium-chain triglycerides (adapted from Linder, M.C. (ed.) 1991: *Nutritional biochemistry and metabolism with clinical applications*, 2nd edn. Amsterdam: Elsevier).

Antioxidants

- Beta-carotene, Vit C,Vit E, Selenium
- Influence the oxidative modification of

lipoprotein in the arterial wall, and can prevent the harmful effects of the free radical chain reactions

- There have been no studies to support the supplementation of antioxidants in the critically ill.