

Shock and Metabolic Response to Injury

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SHOCK

Shock

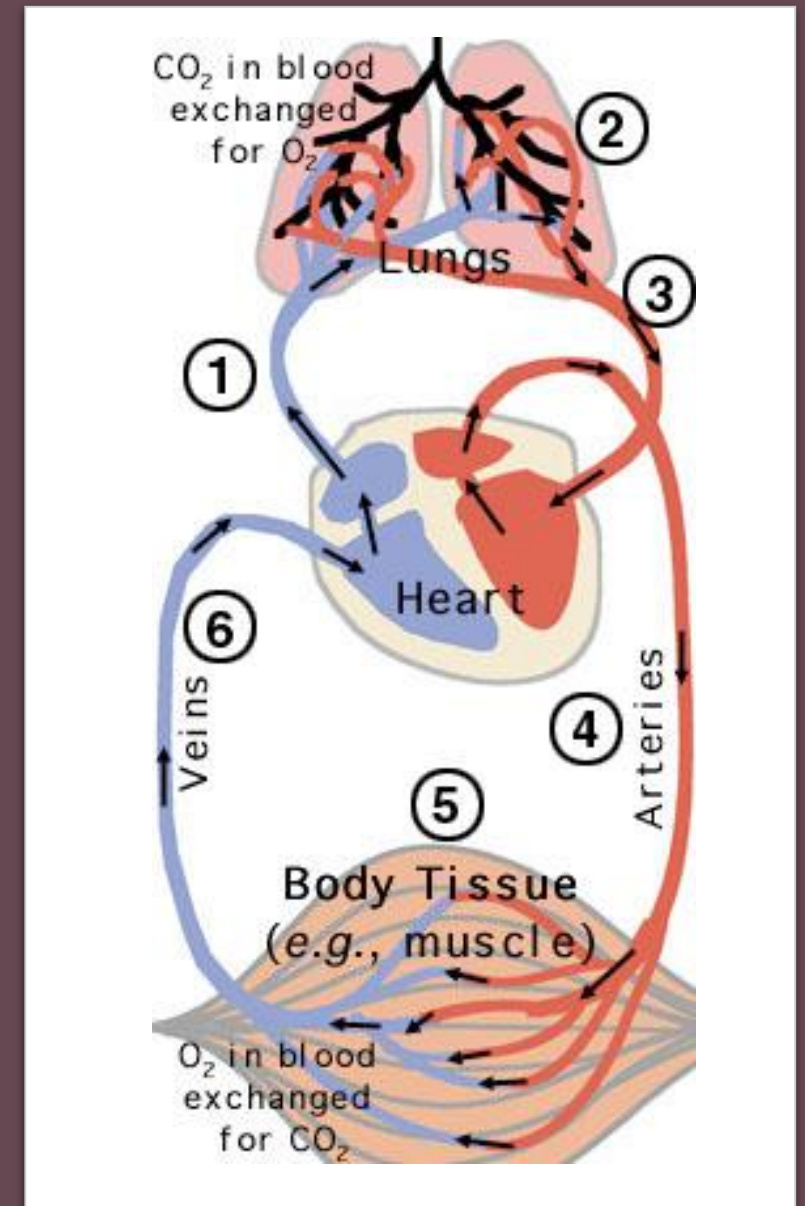
- What is Shock?

Shock

- **Inadequate oxygen delivery to meet metabolic demand.**
- Results in global tissue hypoperfusion and metabolic acidosis
- Shock can occur with a normal blood pressure, and hypotension can occur without shock

Shock

- Oxygen delivery is the function of the circulatory system.
- This system is basically:
 - Pump (heart)
 - Pipes (vessels)
 - Solution (blood)
- Needs to function at adequate pressure, volume and carrying capacity.



Understanding Shock

- Inadequate systemic oxygen delivery activates autonomic responses to maintain systemic oxygen delivery
 - Sympathetic nervous system
 - NE, epinephrine, dopamine, and cortisol release
 - Causes vasoconstriction, increase in HR, and increase of cardiac contractility (cardiac output)
 - Renin-angiotensin axis
 - Water and sodium conservation and vasoconstriction
 - Increase in blood volume and blood pressure

Understanding Shock

- Cellular responses to decreased systemic oxygen delivery
 - ATP depletion → ion pump dysfunction
 - Cellular edema
 - Hydrolysis of cellular membranes and cellular death
- The body tries to maintain cerebral and cardiac perfusion
 - Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow
- Global cellular reliance on anerobic glycolysis and increased lactate production.
- Systemic metabolic lactic acidosis

Multiorgan Dysfunction Syndrome (MODS)

- Progression of physiologic effects as shock ensues
 - Cardiac depression
 - Respiratory distress
 - Renal failure
 - DIC
- Result is end organ failure

Types Of Shock

- **Low Cardiac Output states**

- Hypovolemic shock (↓↓ solution)
 - bleeding
 - Dehydration
- Cardiogenic shock (↓↓ pump)
 - Impaired inflow
 - Primary pump dysfunction
 - Impaired outflow

- **Low peripheral resistance states**

(↑↑ pipes)

- Neurogenic shock
 - Loss of sympathetic tone
- Vasogenic Shock
 - Septic
 - Anaphylactic

Types Of Shock

Shock type	Examples	HR	BP	CO	Capillary refill	Extremity temperature	SVR	Treatment
Hypovolemic	Hemorrhage Dehydration	↑	↓	↓	Delayed	Cool	High	Stop bleeding Fluid resuscitation
Cardiogenic	Myocarditis Dysrhythmia	↑	↓	↓	Delayed	Cool	High	Inotropes Caution with fluids ECMO
Distributive	Sepsis Anaphylaxis	↑	↓	↓ or ↑	Flash or delayed	Warm or cool	Low or high	Antibiotics, fluids Epinephrine
Neurogenic	Spinal cord injury Traumatic brain injury	↓	↓	↓	Flash or normal	Warm	Low	Fluid resuscitation Vasopressors
Obstructive	Tamponade Tension pneumothorax	↑	↓	↓	Delayed	Cool	High	Pericardiocentesis Chest tube
Dissociative	Carbon monoxide Cyanide	↑	Normal or ↑	↑	Normal	Normal	Low to normal	Antidotes Hyperbaric therapy

HR, heart rate; BP, blood pressure; CO, cardiac output; SVR, systemic vascular resistance.

Classes of Hypovolemic Shock

	I	II	III	IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	> 2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	> 40
Pulse rate (per minute)	< 100	100–120	120–140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	> 35
Urine output (mL/hour)	> 30	20–30	5–15	Negligible
Central nervous system/ mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Treatment of Shock

- Goal: Restore perfusion
- Method: Depends on type of Shock
- Reverse the cause.

End Points of Resuscitation in Shock management

- Normal vital signs (can be misleading)
- Normal serum lactate levels
- Evidence of adequate tissue perfusion!!
 - **normal mental status.**
 - **normal urine output.**
 - normal liver function.
 - etc.

What Type of Shock is This?

- 68 yo M with hx of HTN and DM presents to the ER with abrupt onset of diffuse abdominal pain with radiation to his low back. The pt is hypotensive, tachycardic, afebrile, with cool but dry skin.

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Hypovolemic shock

What Type of Shock is This?

- A 34F presents to the ER after dining at a restaurant where shortly after eating the first few bites of her meal, became anxious, diaphoretic, began wheezing, noted diffuse pruritic rash, nausea, and a sensation of her “throat closing off”. She is currently hypotensive, tachycardic and ill appearing.

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Anaphylactic

Metabolic Response to Injury

Mediating the Response

- The Acute Inflammatory Response
 - Cellular activation
 - Inflammatory mediators (TNF, IL1, etc)
 - Paracrine Vs endocrine effects

Mediating the Response

- The Endothelium
 - Selectins, Integrins, and ICAMs
 - Nitric Oxide
 - Tissue Factor

Mediating the Response

- Afferent Nerve Stimulation
 - Sympathetic Nervous System
 - Adrenal Gland Medulla

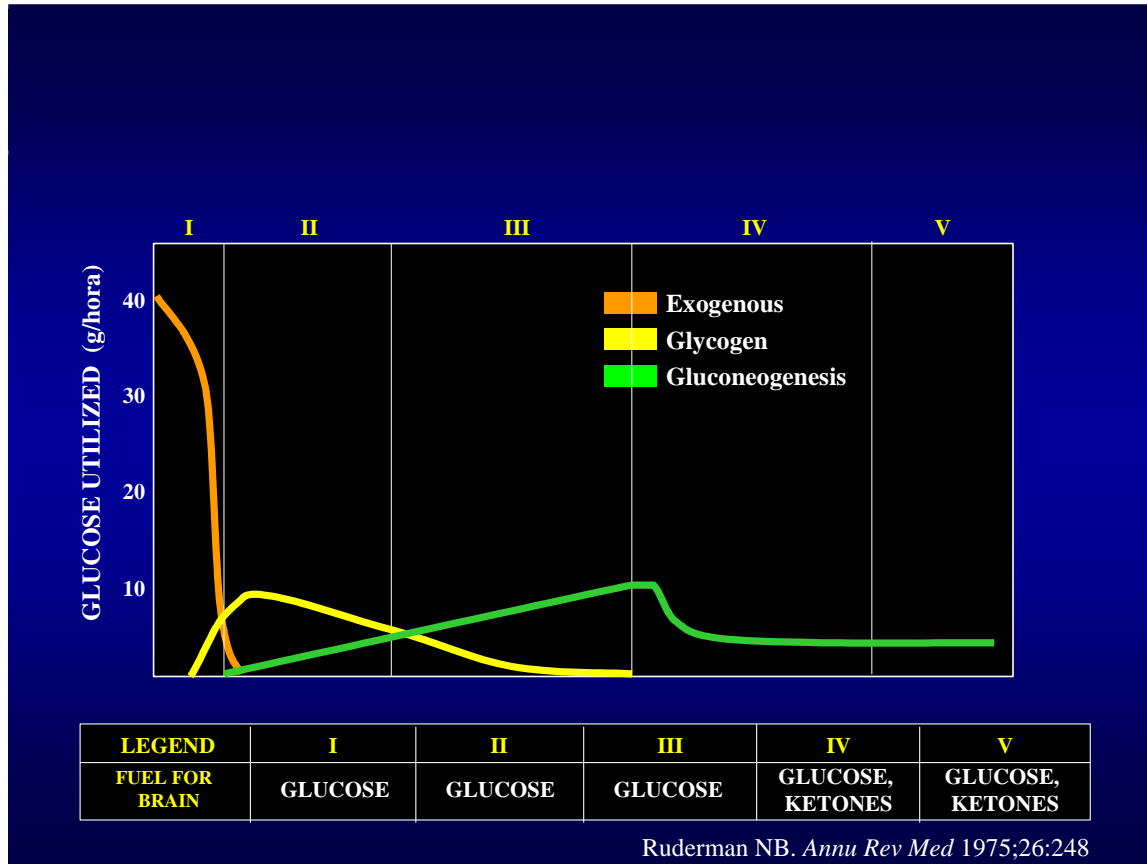
Mediating the Response

- The Endocrine System
 - Pituitary Gland (GH, ACTH, ADP)
 - Adrenal Gland (Cortisol, Aldosterone)
 - Pancreatic (Glucagon, ↓ Insulin)
 - Others (Renin, Angiotensin, ↓ Sex hormones, ↓ T4)

Consequences of the Response

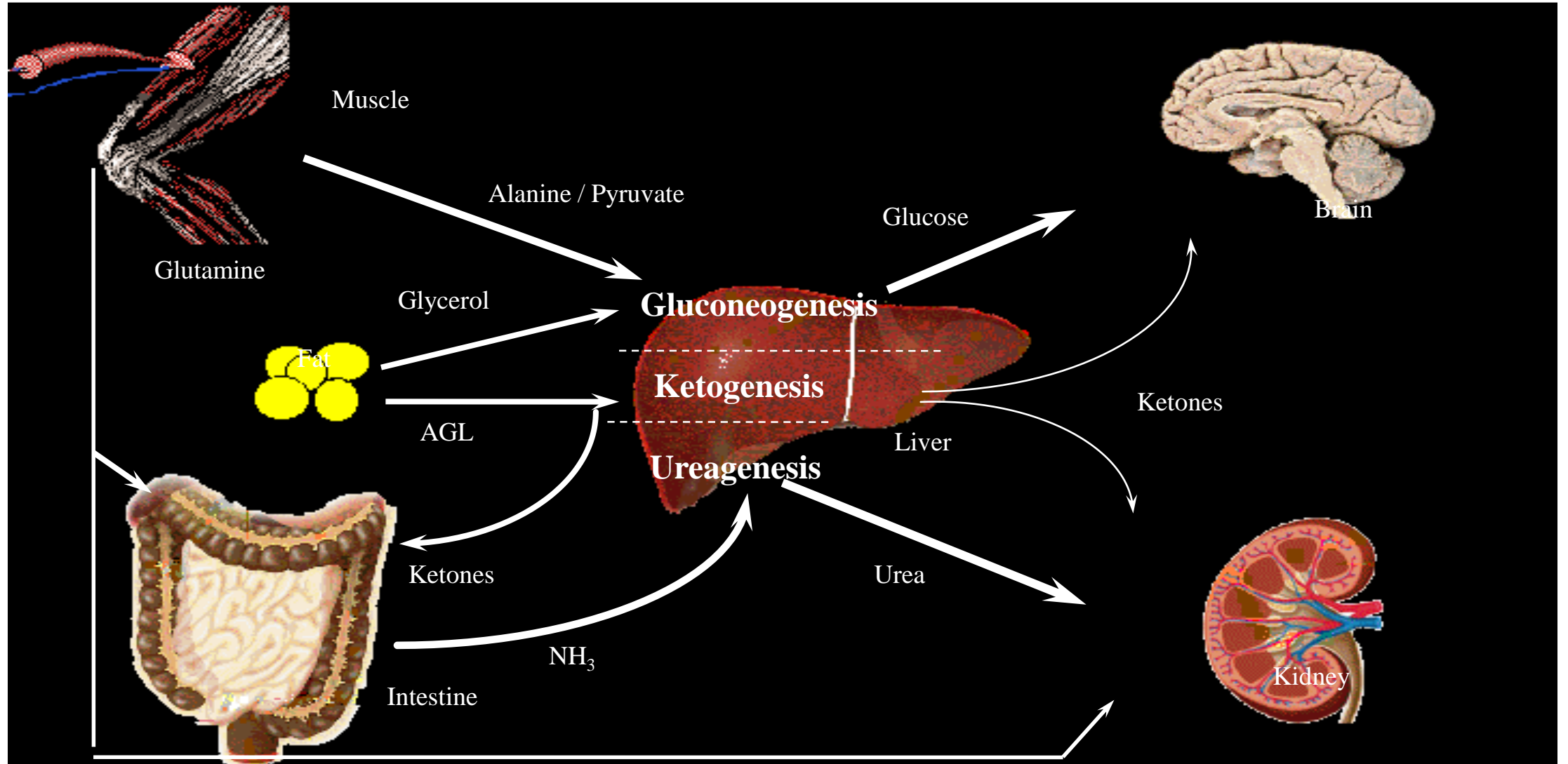
- Limiting injury
- Initiation of repair processes
- Mobilization of substrates
- Prevention of infection
- Distant organ damage

Metabolic Response to Fasting

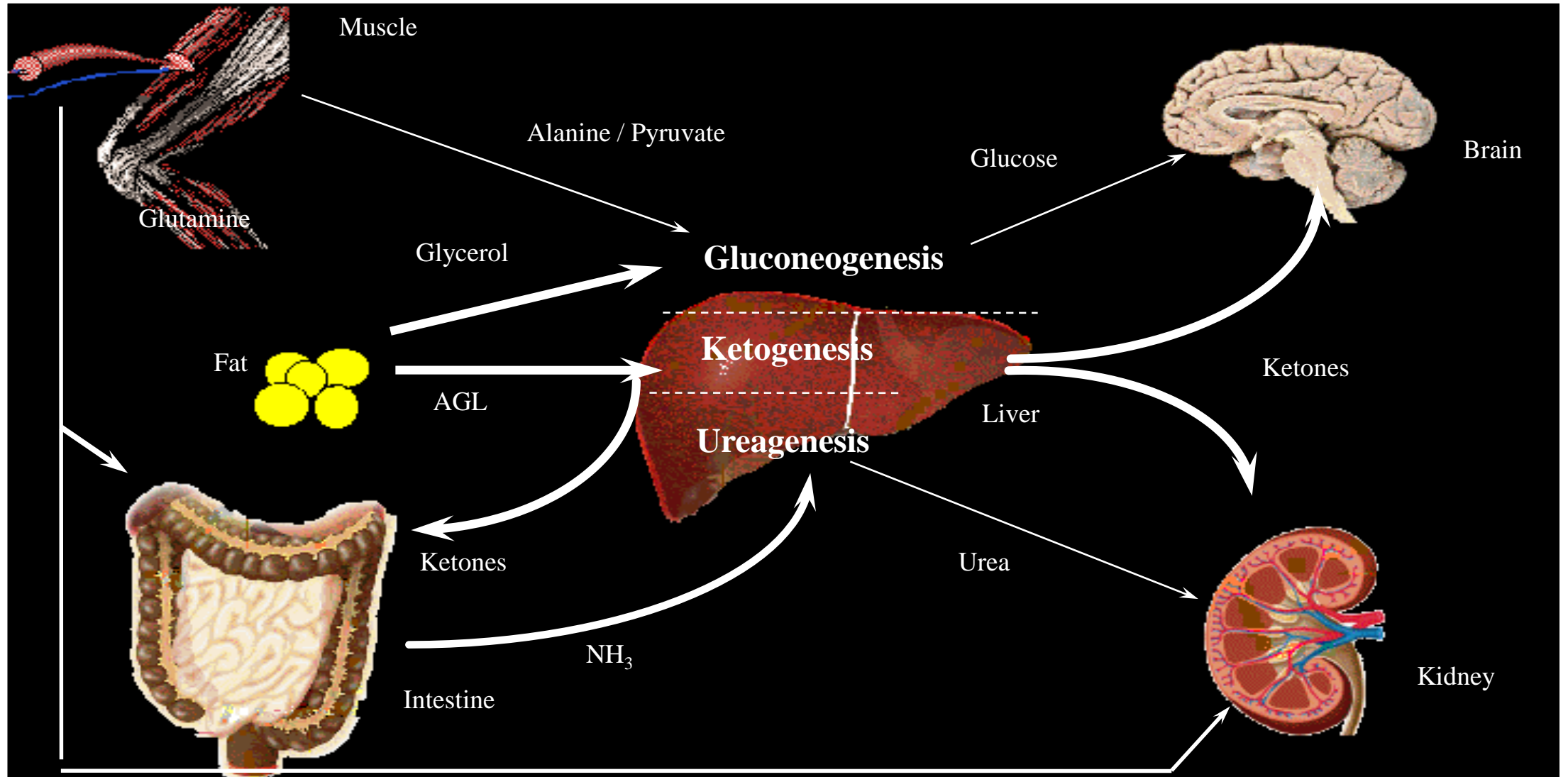


- The carbohydrate deposits of the body last about 18 to 20 hours and new glucose is produced through gluconeogenesis of amino acids from the lean body mass

Starvation – Early Stage



Starvation – Late Stage



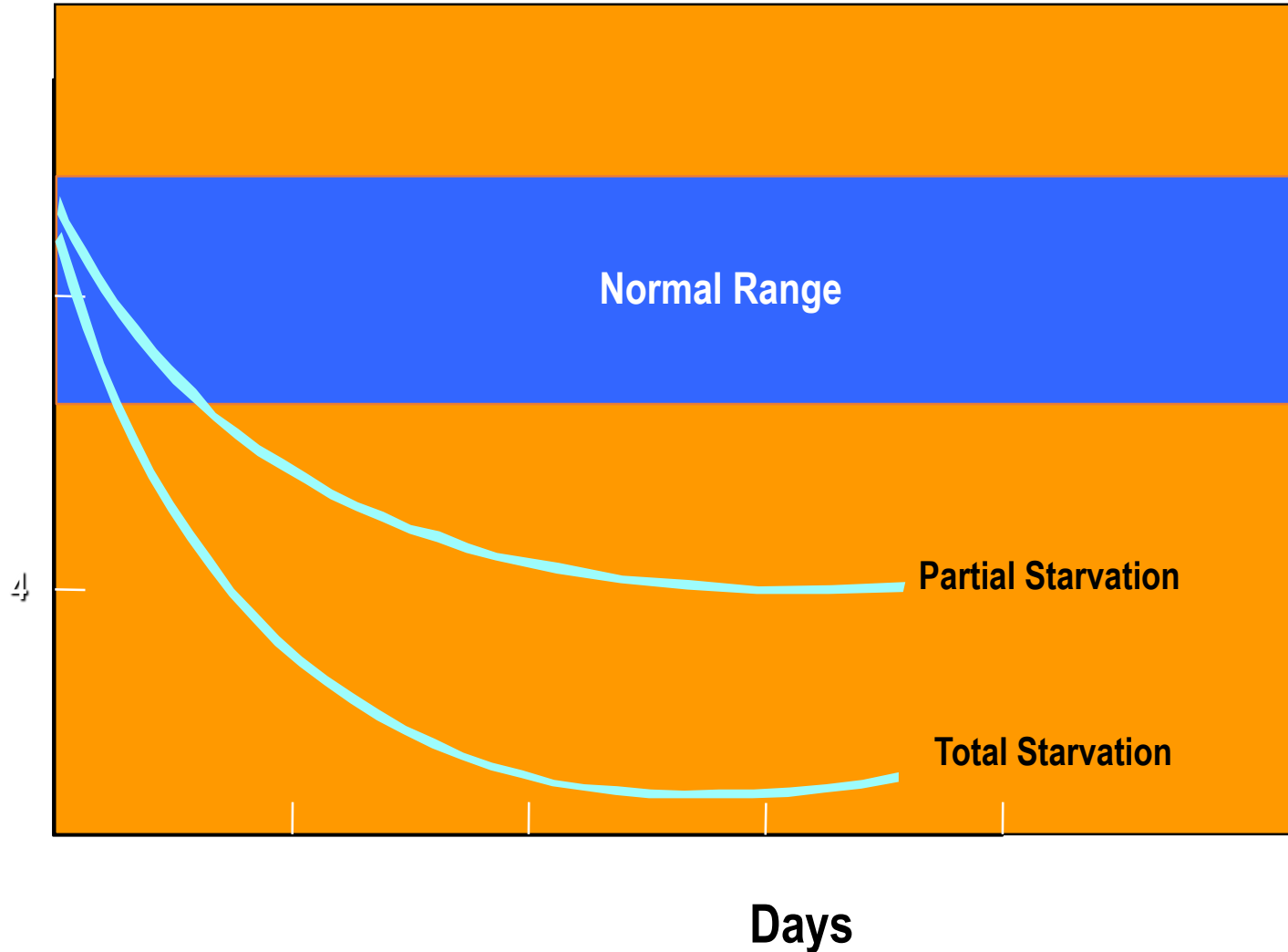
Metabolic Response to Starvation

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<u>Hormone</u>	<u>Source</u>	<u>Change in Secretion</u>
Norepinephrine	Sympathetic Nervous System	↓ ↓ ↓
Norepinephrine	Adrenal Gland	↑
Epinephrine	Adrenal Gland	↑
Thyroid Hormone T4	Thyroid Gland (changes to T3 peripherally)	↓ ↓ ↓

Energy Expenditure in Starvation

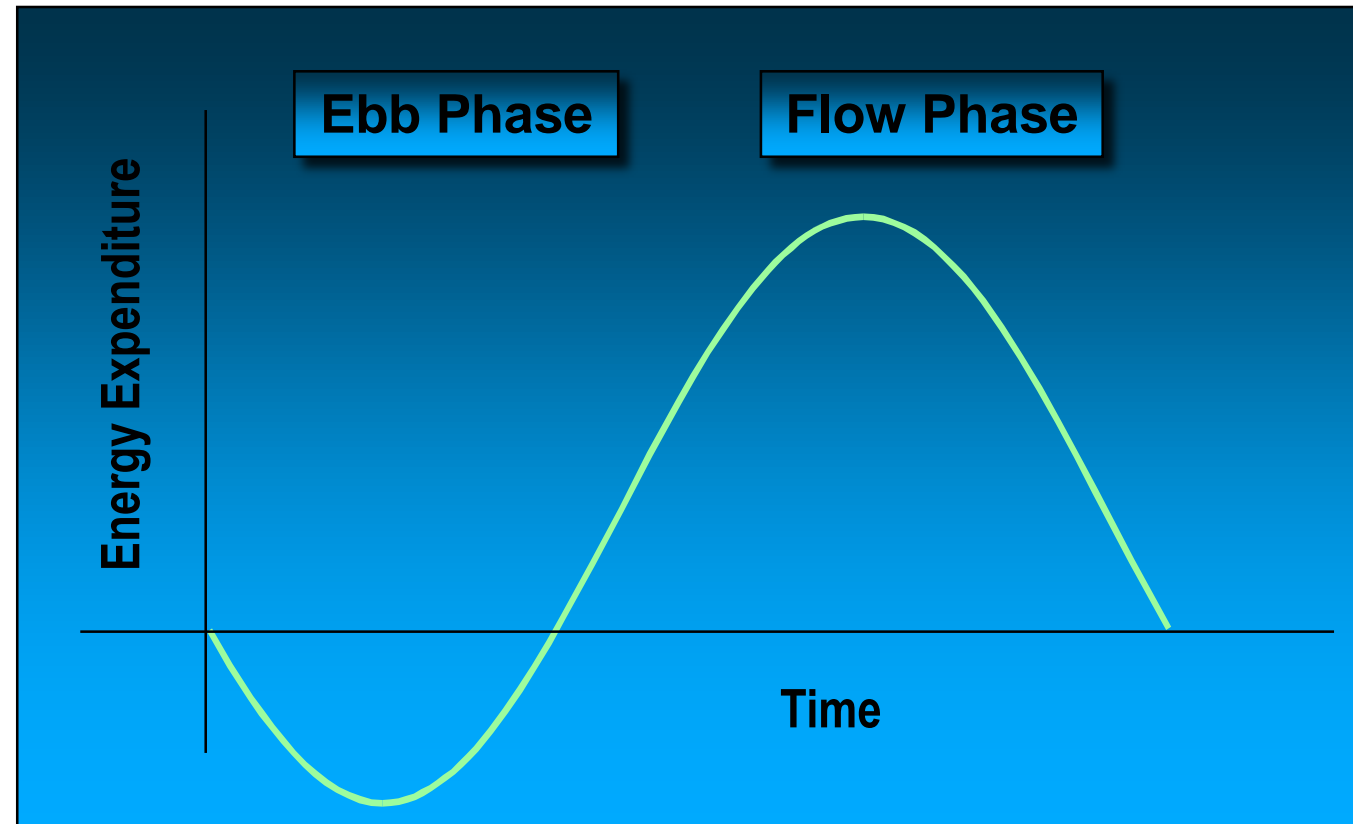
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Long CL et al. *JPEN* 1979;3:452-456

Metabolic Response to Injury

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

Metabolic Response to Injury: Ebb Phase

- Characterized by hypovolemic shock
- Priority is to maintain life/homeostasis
 - ↓ Cardiac output
 - ↓ Oxygen consumption
 - ↓ Blood pressure
 - ↓ Tissue perfusion
 - ↓ Body temperature
 - ↓ Metabolic rate

Metabolic Response to Injury: Flow Phase

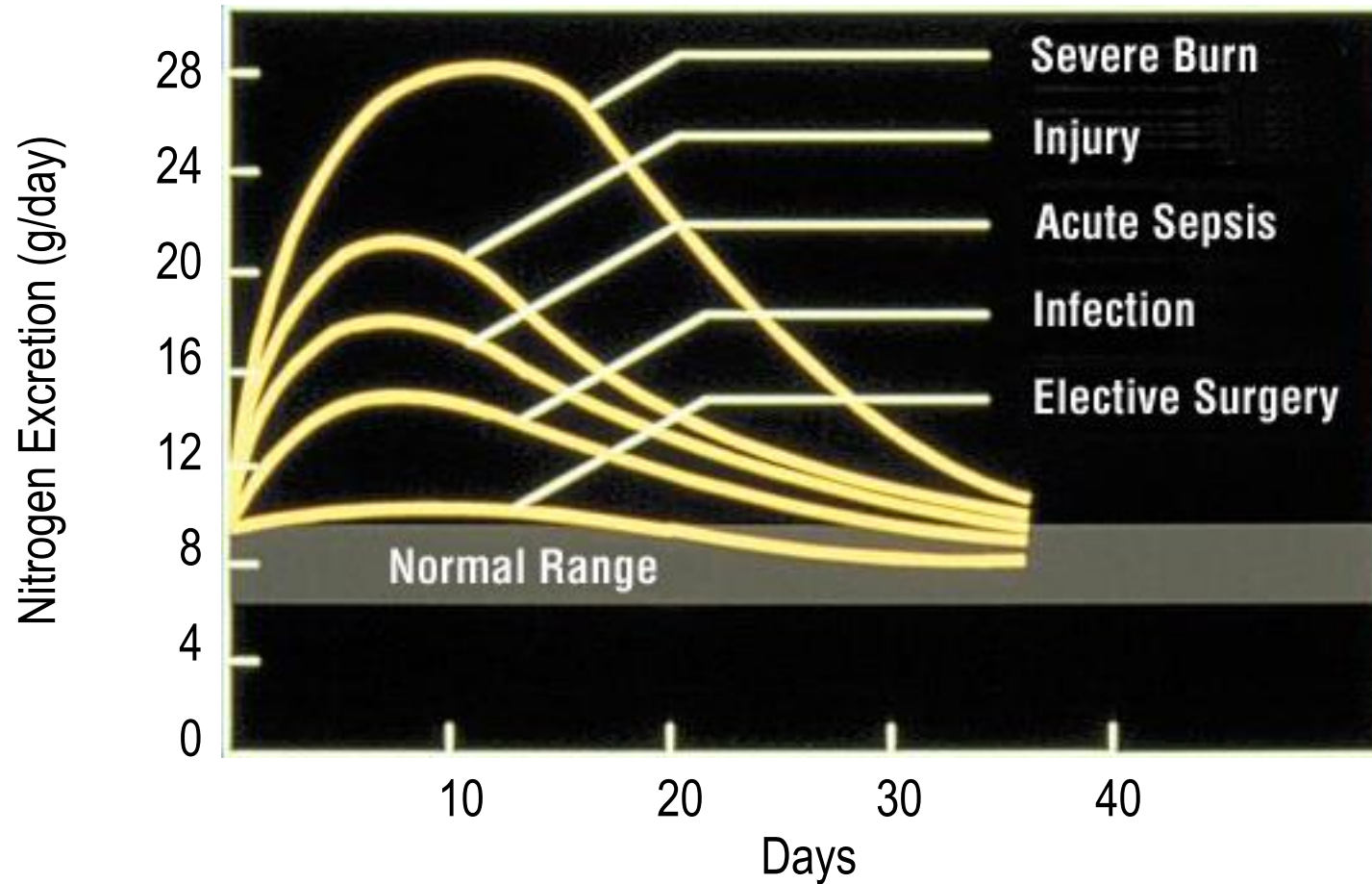
- ↑ Catecholamines
- ↑ Glucocorticoids
- ↑ Glucagon
- Release of cytokines, lipid mediators
- Acute phase protein production

Metabolic Response to Injury



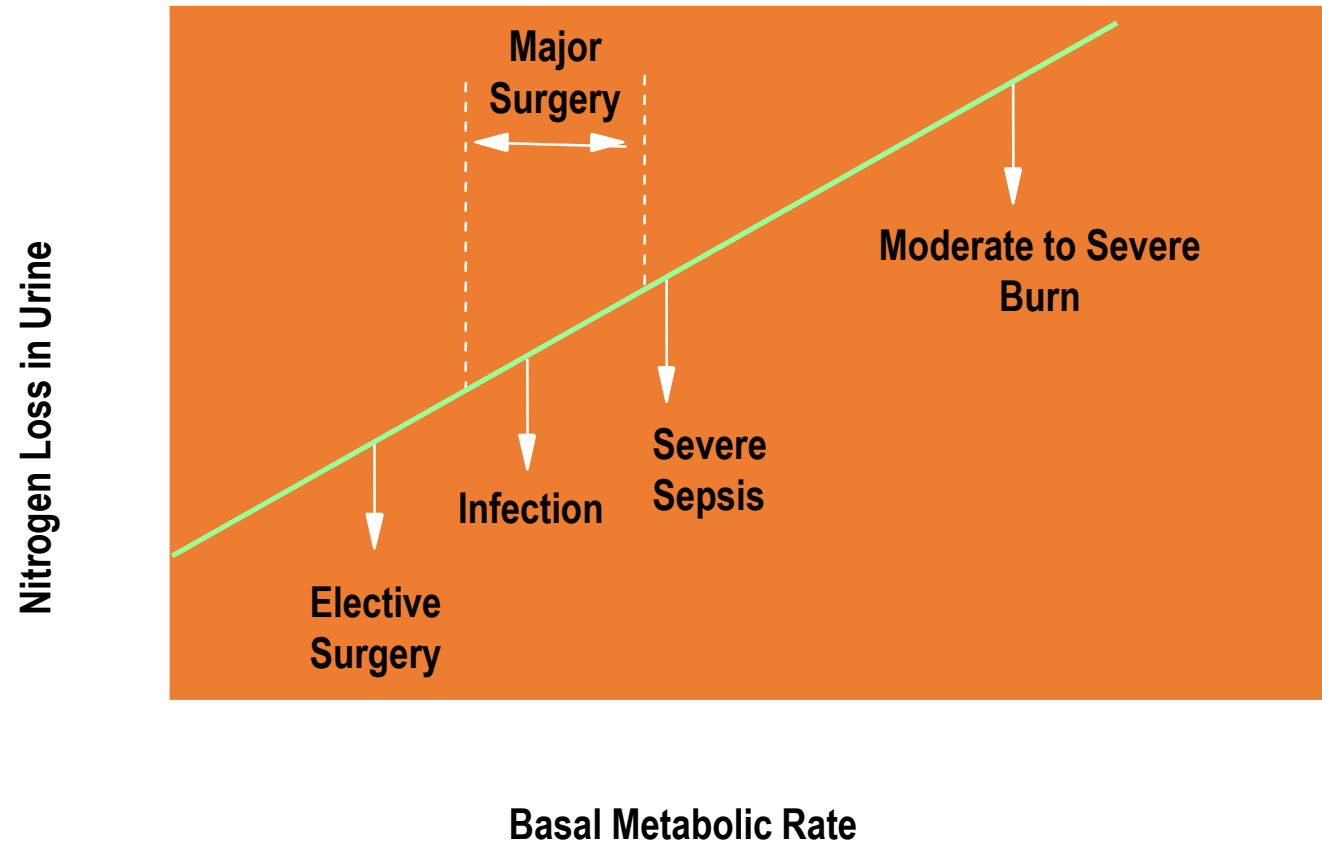
Metabolic Response to Injury

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Severity of Injury: Effects on Nitrogen Losses and Metabolic Rate

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Adapted from Long CL, et al. JPEN 1979;3:452-456

Comparing Starvation and Injury

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	<u>Starvation</u>	<u>Trauma or Disease</u>
Metabolic rate	↓	↑ ↑
Body fuels	conserved	wasted
Body protein	conserved	wasted
Urinary nitrogen	↓	↑ ↑
Weight loss	slow	rapid

The body adapts to starvation, but not in the presence of critical injury or disease.

Modifying the Response

- Medication (before or after injury)
- Nutritional status
- Severity of injury
- Temperature
- Anesthetic technique

Summary

- Injury (Trauma or Surgery) leads to a metabolic response
- Metabolic response to injury is an adaptive response
- Metabolic response could overwhelm the body and lead to increased morbidity and mortality
- We can modify the metabolic response before and sometimes after injury

Metabolic Response to Injury

Questions