Body response to Trauma Stress Sepsis, Burns, Surgery

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Trauma

 Trauma is the Greek word mean "wound". Although the Greeks used the term only for physical injuries, generally is psychological & emotional response to an event or experience that is deeply distressing or disturbing ... Among them are complex trauma is PTSD (Post Traumatic Stress Disorder)











Restore tissue function & maintain organ perfusion (Homeostasis)

Eradicate invading Microorganisms.

Response Components

Physiological Consequences

Metabolic Manifestations

Clinical Manifestations

Laboratory Changes

Response Components

PHYSIOLOGICAL

METABOLIC

- Membrane Transport
- Weight loss
- Wound Healing

- Hypermetabolism
- Acclerated Gluconeogenesis
- Enhanced Protein breakdown
- Increased Fat oxidation

Response Components

CLINICAL

LABORATORY

- FeverTachycardia
- Tachypnoea
- Presence of wound or Inflammation
- Anorexia

- Leucocytosis/Leucop enia
- Hyperglycemia
- Elevated CRP/Altered acute phase reactants
- Hepatic/Renal dysfunction

Mediators of Injury Response

Neuro – Endocrine [Hormonal]

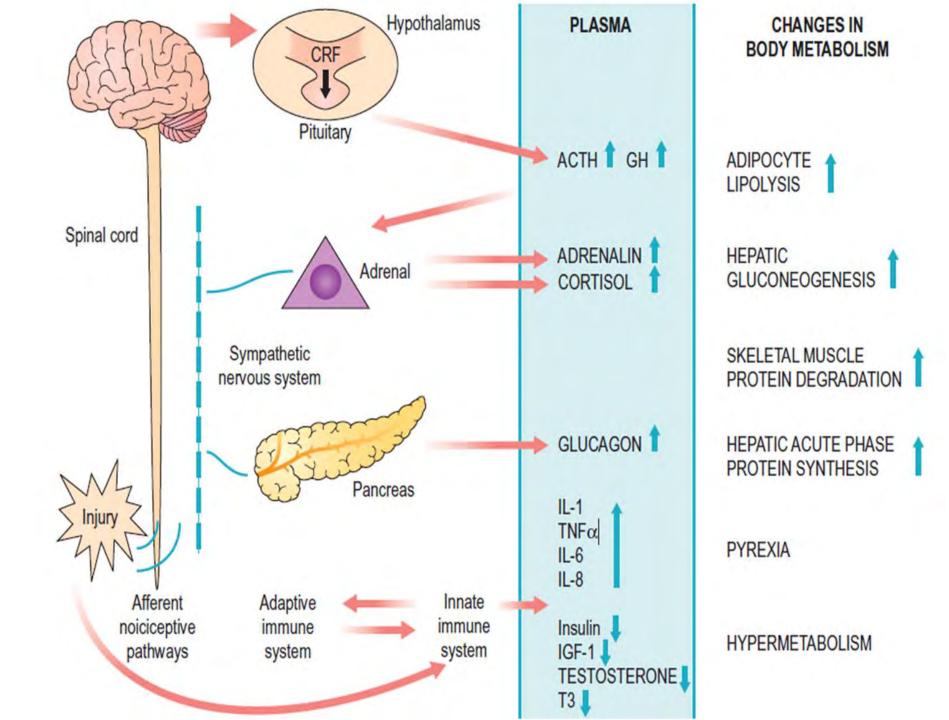
Immune System [Cytokines]

Neuro-endocrine response to injury/critical illness

Biphasic :

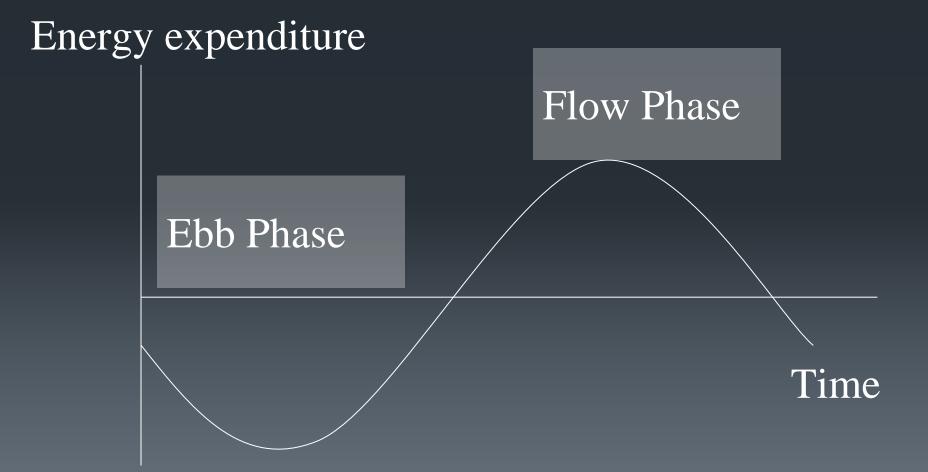
Acute phase - An actively secreting pituitary & elevated counter regulatory hormones (cortisol, glucagon, adrenaline). Changes are thought to be beneficial for short-term survival.

Chronic phase - Hypothalamic suppression & low serum levels of the respective target organ hormones. Changes contribute chronic wasting.

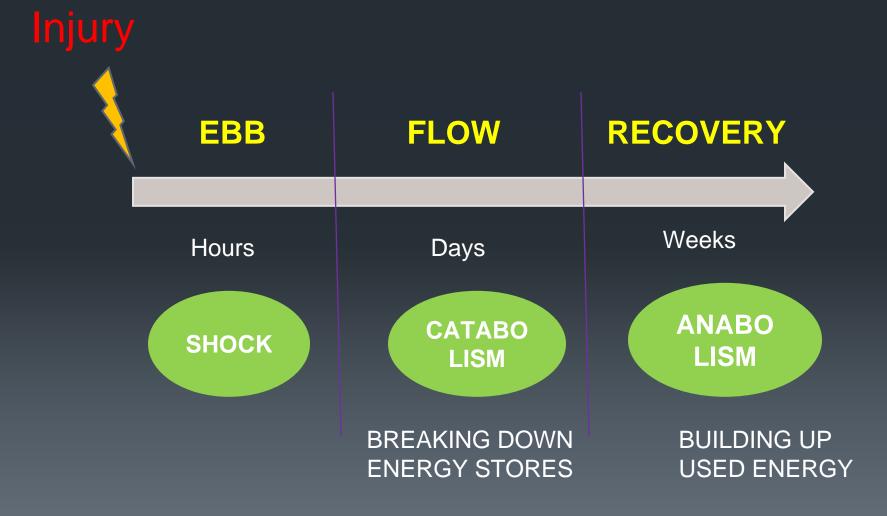


Metabolic response during stress:

 Metabolic response to stress (tissue injury, infection) is divided into the ebb and flow phase



Phases – Physiological response [David Cuthbertson – 1930]



Ebb and Flow Phases

Phase	Duration	Role	Physiological	Hormones
Ebb	24 - 48 hrs	Conserve - blood volume & energy reserves - Repair	↓ BMR, ↓ temp, ↓ CO, hypovolaemia, lactic acidosis	Catecholamines, Cortisol, aldosterone
Flow				
Catabolic	3 – 10 days	Mobilisation of energy stores – Recovery & Repair	↑ BMR, ↑ Temp, ↑ O2 consump, ↑ CO	Cytokines + ↑ Insulin, Glucagon, Cortisol, Catechol but insulin resistance
Anabolic	10 – 60 days	Replacement of lost tissue	+ve Nitrogen balance	Growth hormone, IGF

- In the ebb phase, the body 'shuts down' and the metabolic rate decreases
- Leads to hypovolemic shock:
 - Blood pressure
 - ↓ Cardiac output
 - JBody temperature
 - ↓tissue perfusion
 - ↓O2 consumption
 - ↓ metabolic rate
- Body's protective response (eg to blood loss)

 However, once the blood pressure is stabilized, the flow (recovery) phase begins

Divided into 2 response:

- Acute Response:
 - catabolism predominates
 - f glucocorticoids

 - Release cytokines, lipid mediators
 - Acute phase protein (CRP)
 - ^N2 excretion
 - ↑ metabolic rate
 - ↑ O2 consumptions
 - Impaired fuel utillization
- Adaptive Response:
 - Anabolism predominates
 - Hormonal response gradually diminished
 - ↓ hypermetabolic rate
 - Assoc with recovery
 - Restore body protein
 - Wound healing

Key catabolic elements of flow phase

- Hypermetabolism
- Alterations in skeletal muscle protein
- Alterations in Liver protein
- Insulin resistance

1. Hypermetabolism

Majority of trauma pts - energy expenditure appr.
 15-25% > predicted healthy resting values.

Factors which increases this metabolism :

- * Central thermodysregulation
- * Increased sympathetic activity
- * Increased protein turnover
- * Wound circulation abnormalities

2.Skeletal muscle – Metabolism

1. Muscle wasting – result of ↑ muscle protein degradation + ↓ muscle protein synthesis. Cardiac muscle is spared.
2. Lead - Increased fatigue, reduced functional ability, & ↑ risk of morbidity & mortality.

3.Hepatic acute phase response

 Cytokines – IL- 6 → ↑ Synthesis of Positive acute phase proteins : Fibrinogen & CRP

Negative acute reactants : Albumin decreases

4.Insulin resistance

- Hyperglycaemia is seen ↑ glucose production + ↓ glucose uptake – peripheral tissues. (transient induction of insulin resistance seen)
- Due Cytokines & decreased responsiveness of insulin- regulated glucose transporter proteins.

The degree of insulin resistance is ∞ to magnitude of the injurious process.

Factors - ↑ severity of injury (2H,2S,IP)

- Hypothermia
- Pain
- Starvation
- Immobilisation
- Sepsis
- Hypotension

Avoidable factors that compound the response to injury

- Continuing haemorrhage
- Hypothermia
- Tissue oedema
- Tissue underperfusion
- Starvation
- Immobility

Avoidable Factors

Volume loss : Careful limitation of intra operative administration of colloids and crystalloids so that there is no net weight gain.

Hypothermia : maintaining normothermia by an upper body forced air heating cover ↓ wound infection, cardiac complications and bleeding and transfusion requirements.

Avoidable Factors

Starvation : During starvation, the body is faced with an obligate need to generate glucose to sustain cerebral energy metabolism(100g of glucose per day).

Provision of at least 2L of IV 5% dextrose for fasting patients provides glucose as above.

Avoidable Factors

Tissue oedema : is mediated by the variety of mediators involved in the systemic inflammation. Careful administration of anti-mediators & reduce fluid overload during resuscitation reduces this condition.

Immobility : Has been recognized as a potent stimulus for inducing muscle wasting. Early mobilization is an essential measure to avoid muscle wasting.

- Diagnosis of Systemic Inflammatory Response Syndrome (SIRS):
 - Site of infection established and at least two of the following are present:
 - Body temperature >38° C or <36° C</p>
 - Heart rate >90 beats/minute
 - Respiratory rate >20 breaths/min (tachypnea)
 - PaCO₂ <32 mm Hg (hyperventilation)</p>
 - WBC count >12,000/mm³ or <4000/mm³
 - Bandemia: presence of >10% bands (immature neutrophils) in the absence of chemotherapy-induced neutropenia and leukopenia
 - May be caused by bacterial translocation



