Body Response to Stress

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THE STRESS RESPONSE

- is the name given to the:
  - hormonal changes
  - metabolic changes

  which follow injury or trauma.

The terms 'ebb' and 'flow'

Describe:

- an initial decrease in metabolic activity
- subsequent increase in metabolic activity.

Physiological response to injury

Natural response to injury include:

- **Immobility/ rest**
- **Anorexia**
- **Catabolism**

To aid survival of mild to moderate injury in the absence of medical intervention.

trauma - burns - operation -
bacterial infection -
ischemia/reperfusion

LOCAL REACTION:

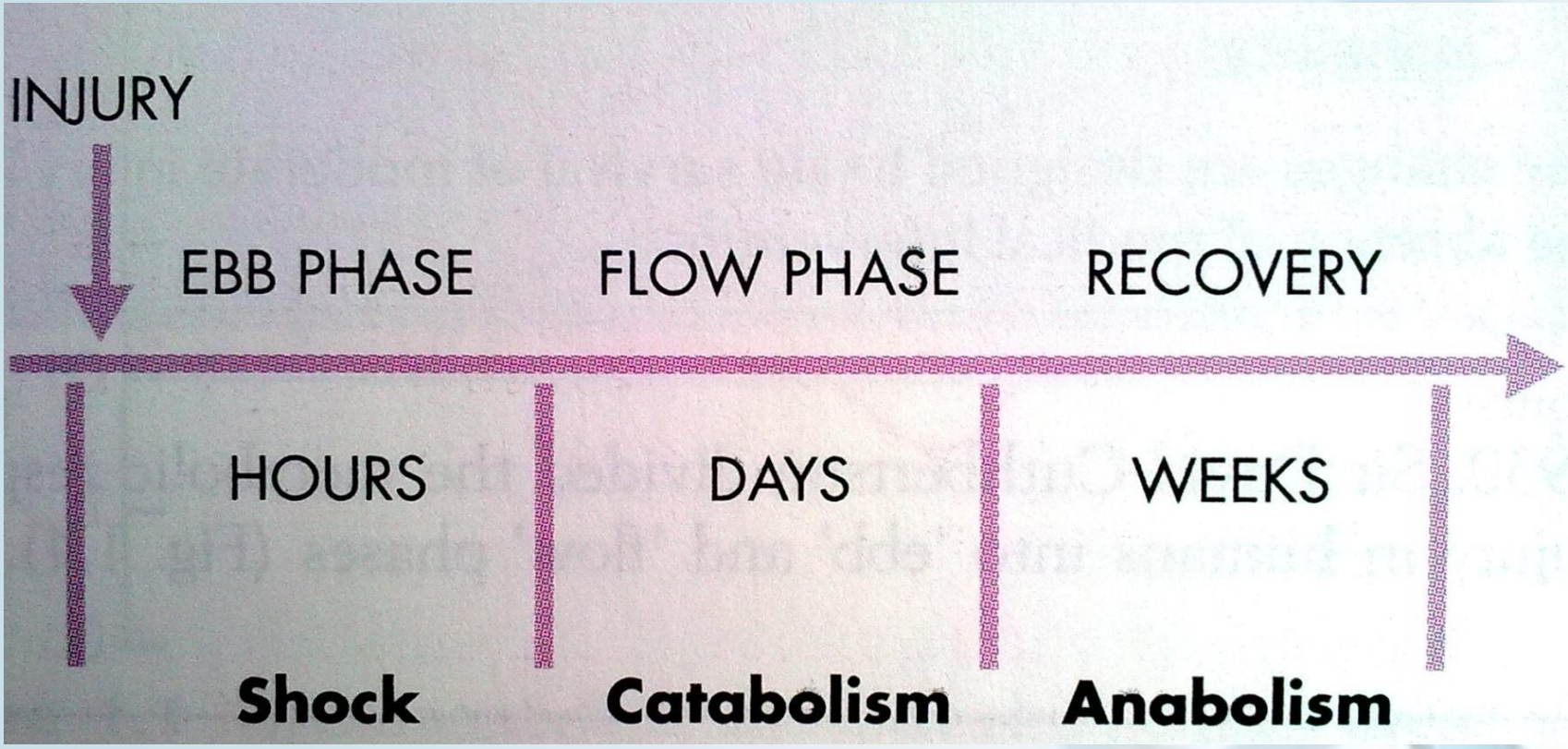
IL-1; IL-6;IL-‡8
prostaglandin;
leukotriene; TNF;
catecholamines;
histamine; kinin;
cortocoïds

SYSTEMIC REACTION:

SIRS

- Aid in survival of moderate injury

- Ebb and Flow Model



INJURY

AFFERENT SIGNALS

EFFERENT SIGNALS

HYPOTHALAMIC –PITUITARY – ADRENAL AXIS

A: Ebb Phase [24 – 48hrs]

Physiological Role:
Conserve both circulating volume and energy stores for recovery and repair

- characterized by:
  - depression of local metabolism
  - reduction in energy expenditure .

- Stage of shock
  - Hypovolemia
  - ↓ BMR
  - ↓ CO
  - Hypothermia
  - Lactic Acidosis

Hormones

- Catecholamines
- Cortisol
- Aldosterone

Magnitude? Depends upon

- Degree of blood loss
- Stimulation of somatic afferent nerves at site of injury

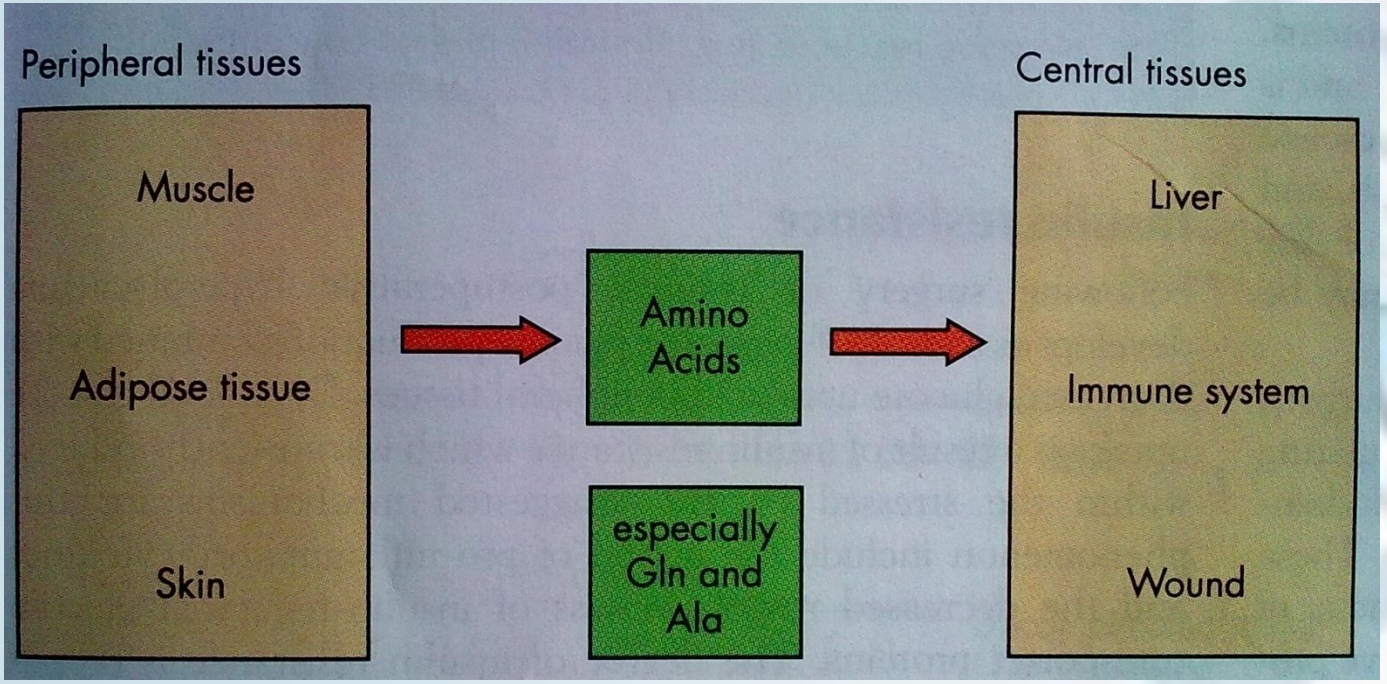
B. The catabolic (flow) phase

Flow Phase [3-10days]

Stage of Catabolism

Mobilization of body energy stores

Replacement of lost tissue



B. The catabolic (flow) phase

- mediated by an increased catecholamine drive
  - which mobilizes energy reserves from adipose tissue & carbohydrate stores in liver & muscle.

- There is an increase in protein breakdown
- The increase in protein degradation > protein synthesis ,so there is marked nitrogen loss that is proportional to the pre-injury nutritional status –
- In this period ,no amount of exogenous protein supplementation will obviate the negative nitrogen balance & nitrogen positivity should not be attempted
- This proteolysis results in:
  - muscular weakness
  - decreased enzymatic function
  - decreased immune competence
  - excessive hepatic gluconeogenesis
  - rapid weight loss occurs.

B. The catabolic (flow) phase

- increasing gluconeogenesis & decrease in insulin sensitivity = hyperglycaemia .
- This accompanied by increased energy expenditure & a negative balance .
- Hepatic Acute phase response
  - +ve reactant = CRP increased
  - -ve reactant = Albumin decreased

- The size & duration of the response are directly related to the severity of the trauma or surgical insult.

- Tissue Edema
- ↑ BMR
- ↑ CO
- ↑ Body Temperature
- Leukocytosis
- ↑ O2 consumption
- Gluconeogenesis

Hormones

- Catecholamines
- Cortisol
- Insulin
- Glucagon

Cytokines

- IL 1
- IL 6
- TNF α

Key catabolic elements

- *Hypermetabolism*
- *Alteration in skeletal muscle protein metabolism*
- *Alteration in hepatic protein metabolism*
- *Insulin resistance*

Hypermetabolism

- Central thermodysregulation
- ↑ sympathetic activity
- Abnormalities in wound circulation
- ↑ protein turn over

Clinically

- Asthenia
- ↑ fatigue
- ↓ functional ability
- ↓ quality of life
- Risk of morbidity and mortality

Alteration in hepatic protein metabolism

Pro inflammatory cytokines → hepatic synthesis of positive Acute Phase Proteins

[fibrinogen & C-reactive proteins]

Insulin resistance

- Pro inflammatory cytokines
- ↓ responsiveness of insulin regulated glucose transporter proteins

C. The anabolic (Recovery) phase:

If recovery from the injuries occurs ,an anabolic phase supervenes in which:

- weight gain occurs
- protein & fat stores are replenished
- metabolic rate returns to normal .

- may last for several months depending on the extent of the initial injuries .

Physiological response to infection, trauma and surgery

- Similar responses seen to trauma, burns, sepsis and surgery
- Involves both local and systemic reactions
- Extent of response proportional to severity of insult
- An appropriate response maintains homeostasis and allows wound healing
- An excessive response can produce a systemic response
- This can cause the systemic inflammatory response syndrome (SIRS)
- Multiple organs dysfunction syndrome (MODS) can result from SIRS

- **Initiation of response**
- Several factors can initiate the physiological response to trauma
- Important factors are:
  - Tissue injury
  - Infection
  - Hypovolaemia
  - Hypoxia or hypercarbia

- **Control of response**
- Four systems control the response to trauma
  - –Sympathetic nervous system
  - –Acute phase response
  - –Endocrine response
  - –Vascular endothelium

Sympathetic nervous system

- Has direct actions via the release of noradrenaline from sympathetic nerves
- Has indirect action via the release of adrenaline from the adrenal medulla
  - Produces cardiovascular, visceral and metabolic actions
  - Blood diverted from skin and visceral organs

Sympathetic nervous system

- Heart rate and myocardial contractility are increased
- Bronchodilation occurs
- gastrointestinal motility is reduced
- Insulin production is reduced and glucagons production increased
- Increased glycogenolysis increases blood sugar levels

Acute phase response

- Tissue injury results in cytokine release
- Important cytokines include TNF-alpha, IL-1, IL-2, IL-6, interferon and prostaglandins
- Important in regulating the inflammatory response
- Overflow of cytokines into systemic circulation is important factor in SIRS

Acute phase response

- Cytokines stimulate the production of acute phase proteins such as:
  - C-reactive protein
  - Fibrinogen
  - Complement C3
  - Haptoglobin

Endocrine response

- The hypothalamus, pituitary, adrenal axis is important
- Trauma increases ACTH and cortisol production
- Steroids have a permissive action in many metabolic responses
- Catabolic action increases protein breakdown
- Insulin antagonism increases blood sugar levels

Endocrine response

- Anti-inflammatory actions reduce vascular permeability
- Aldosterone increases sodium reabsorption
- Vasopressin increases water reabsorption and produces vasoconstriction
- Histamine increases vascular permeability
- Total T4, total and free T3 levels are reduced

Vascular endothelium

- Nitric oxide produces vasodilatation
- Platelet activating factor augments the cytokine response
- Prostaglandins produce vasodilatation and induce platelet aggregation

Outcome or response

- Inflammatory response produces clinically apparent
  - local effects
  - systemic effects

- The local response is usually
  - the cardinal signs of inflammation
    - Odema
    - Pain
    - Redness
    - dysfunction

Outcome or response

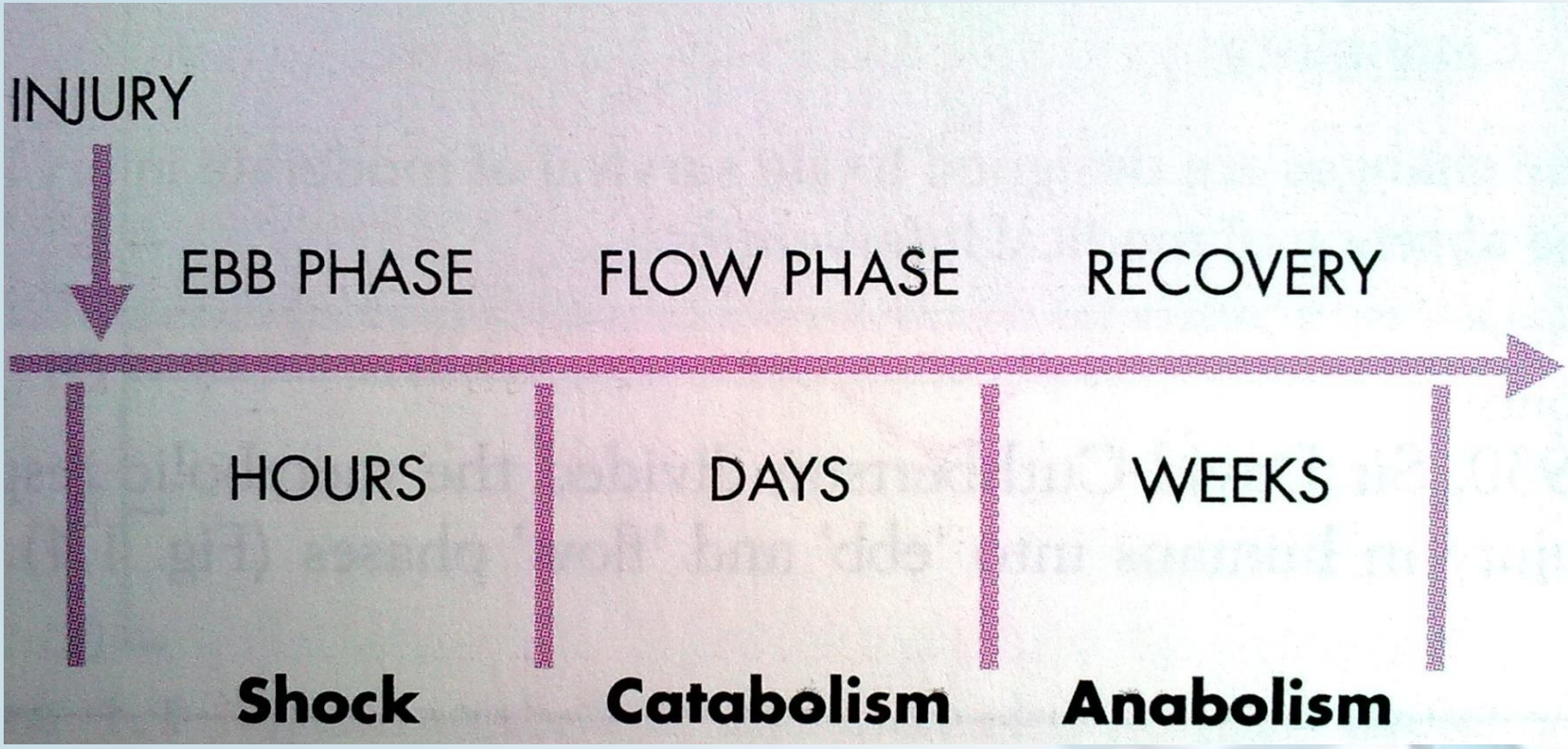
- The systemic response includes:
  - Increased ECF volume and hypovolaemia
  - Increased vascular permeability and oedema
  - Early reduced urine output and increased urine osmolality
  - Reduced ‘free’ water clearance
  - Late diuresis and increased sodium loss
  - Pyrexia in the absence of infection
  - Early reduction in metabolic rate

Outcome or response

- The systemic response includes:
  - Late increased metabolism, negative nitrogen balance and weight loss
  - Lipolysis and ketosis
  - Gluconeogenesis via amino acid breakdown
  - Reduced serum albumin
  - Hyponatraemia due to impaired sodium pump action
  - Acid-base disturbance – usually a metabolic alkalosis or acidosis
  - Immunosuppression
  - Hypoxia and coagulopathy

- Aid in survival of moderate injury

- Ebb and Flow Model



METHODS TO DECREASE THE RESPONSE TO INJURY

.1 PROMPT OPERATIVE CARE

.2 CAREFUL SURGICAL TECHNIQUE

.3 MINIMAL SURGICAL PROCEDURES

.4 EPIDURAL OR SPINAL ANESTHESIA

METHODS TO DECREASE THE RESPONSE TO INJURY

.5 NORMOTHERMIA

.6 NUTRITIONAL SUPPORT

.7 HORMONE MANIPULATIONS

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)

DEFINITION

- INAPPROPRIATE RELEASE OF MEDIATORS
- TRIGGERING REMOTE INFLAMMATION
- DUE TO :
  - INFECTIOUS OR
  - NON - INFECTIOUS cuases

trauma - burns - operation -
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