

Metabolic Response
to
Trauma (Injury)

Lecture 1

Objectives

1. To know the conditions triggering the metabolic response.
2. To illustrate the benefits and extent behind the metabolic response.
3. To illustrate the factors mediating the metabolic response.
4. To study the changes during phases of the metabolic response.
5. To illustrate the hormonal during the metabolic response.

Contents

1. Conditions triggering the metabolic response.
2. Benefits and extent behind the metabolic response.
3. Factors mediating the metabolic response.
4. Changes during phases of the metabolic response.
5. Hormonal during the metabolic response.

Following trauma or injury

There are immediate local changes at the site of trauma or injury.

This is followed by **a series of systemic changes in the body** in order to revert the body to its original state prior to the occurrence of the trauma or injury.

Conditions triggering the metabolic response to injury

- ☐ **Severe fracture**
- ☐ **Severe infection**
- ☐ **Major surgery**
- ☐ **Severe injuries**
- ☐ **Extensive burn**

Injuries of any type

(fracture, surgery, burns and infections)

- Are associated with catabolic response (triggered via neural and hormonal pathways) which is characterized by:
 - an enhanced protein breakdown, lipolysis, gluconeogenesis and hyperglycaemia.

Aims of the catabolic response

1. Restoration of adequate tissue perfusion.
2. Providing adequate energy for vital metabolic pathways.
3. Providing substrate for vital body tissues and organs, used for the biosynthesis of important substances for defense mechanisms as well as the regeneration of injured and damaged tissue.

Summary of the metabolic response to injury

❑ **Site of Injury:**

- ❖ Afferent nerve stimulation
- ❖ Endothelial activation
- ❖ Increased blood flow
- ❖ Inflammation
- ❖ Oedema

Summary of the metabolic response to injury

❑ Hypothalamus:

- ❖ Pyrexia

❑ Pituitary:

- ❖ Increased ACTH
- ❖ Increased ADH

Summary of the metabolic response to injury

❑ Adrenal cortex:

- ❖ Increased cortisol
- ❖ Increased aldosterone

❑ Adrenal medulla:

- ❖ Increased epinephrine

❑ Cardiovascular system:

- ❖ Increased sympathetic activation
- ❖ Tachycardia

Summary of the metabolic response to injury

- ❑ **Kidneys:**
 - ❖ Increased renin angiotensin system activation
 - ❖ Increased Na reabsorption
 - ❖ Decreased K reabsorption
 - ❖ Decreased urine volume
 - ❖ Poor erythropoietin response to anaemia

Summary of the metabolic response to injury

❑ Liver:

- ❖ Increased glycogenolysis
- ❖ Increased gluconeogenesis
- ❖ Increased lipolysis
- ❖ Increased ketone body formation
- ❖ Increased acute-phase protein production:
(C-reactive protein, Serum amyloid A, Ferritin,
Fibrinogen, Haptoglobin, α 2-Macroglobin,
Ceruloplasmin)

Summary of the metabolic response to injury

❑ Pancreas:

- ❖ Decreased insulin secretion
- ❖ Increased glucagon secretion

❑ Skeletal muscles:

- ❖ Increased muscle breakdown
- ❖ Increased amino acid release from muscles

❑ Bone marrow:

- ❖ Impaired red cell production

Factors mediating the metabolic response to injury

1. Acute inflammatory response: Inflammatory cells
Cytokines [TNF- α , ILs (IL-1, IL-6, IL-8)].
2. Endothelial cell activation: Adhesion of inflammatory cells,
vasodilatation, increased permeability.
3. Nervous system: Afferent nerve stimulation.
4. Endocrine response: Increased secretion of catabolic hormones.
Decreased secretion of anabolic hormones.
5. Bacterial infection:

The metabolic response to injury is divided into 2 major phases

1. EBB phase :

A short lived phase corresponding to the period of hypovolemia and sympathetic activity, occurs immediately after injury or trauma lasts from **24-48 hours**, followed by the flow phase.

2. FLOW phase:

A more prolonged lasts from **3-10 days or more** depending on the type and severity of the injury. This phase is characterized by negative nitrogen balance.

Then: The **ANABOLIC phase** ultimately occurs

Ebb Phase

Characterized by:

hypovolaemic shock and **haemodynamic** compromise.

- ❖ The most important issue at this time is to maintain survival and the function of the vital organs.
- ❖ The **ebb phase** is associated with a reduction in cardiac output, oxygen consumption and systemic blood pressure. These changes result in a decrease in tissue perfusion

Ebb Phase

Other changes during Ebb Phase:

1. Hypothermia.
2. Decreased BMR. This reduction may be a protective mechanism.
3. Hypoinsulinaemia also occurs.

Flow Phase

- ❑ Occurs following the **ebb phase** after fluid resuscitation and restoration of the circulatory volume.
- ❑ Cardiac output, body temperature and energy expenditure are increased.
- ❑ The flow phase is characterized by an accelerated tissue catabolism.

Flow phase

The flow phase is associated with:

1. Increased production of catabolic hormones (catecholamines, glucocorticoids and glucagon) .
2. Increased production of cytokines and other inflammatory mediators.
3. Increased muscle protein breakdown producing amino acid substrates to the liver for gluconeogenesis and the synthesis of acute phase proteins.

Hormonal changes in response to injury

1. Increased production:

Pituitary: ACTH, PRL, ADH

Adrenal cortex: Glucocorticoids, Mineralocorticoids

Adrenal medulla: Catecholamines

Pancreas: Glucagon

Others: Renin-angiotensin

Hormonal changes in response to injury

2. Decreased production:

Pancreas: Insulin

Gonads: Oestrogens, Testosterone

Thyroid: Thyroid hormones (T_4 , T_3)

Hormonal changes in response to injury

3. Unchanged production:

Pituitary: TSH, FSH, LH

Thank You