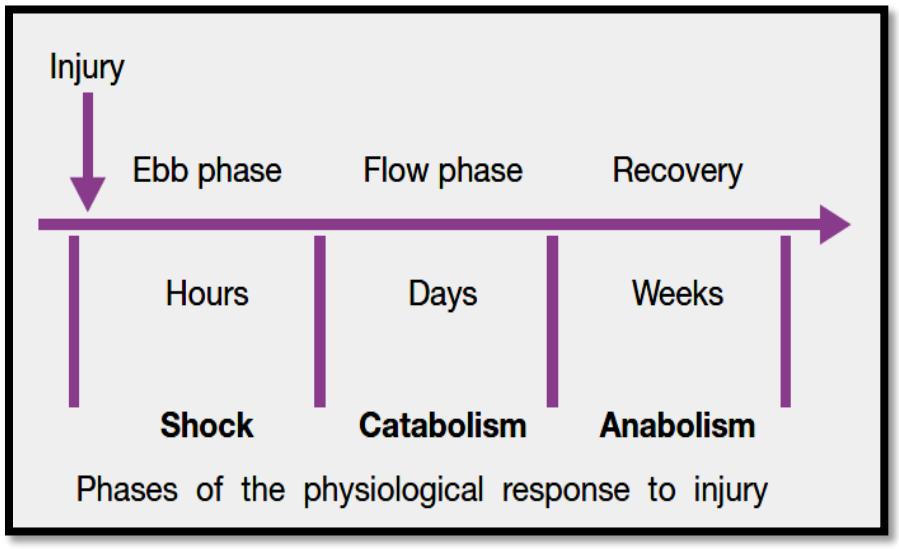
# Metabolic Response to Trauma - p. 2

## Phases of the Metabolic Response

In the natural world, if an animal is injured, it displays immobility, anorexia and catabolism. These are manifestations of the 1st phase of metabolic response.
To understand the detailed manifestations of metabolic response; the response to injury in humans was divided into two phases: **'ebb' and 'flow' phases**.

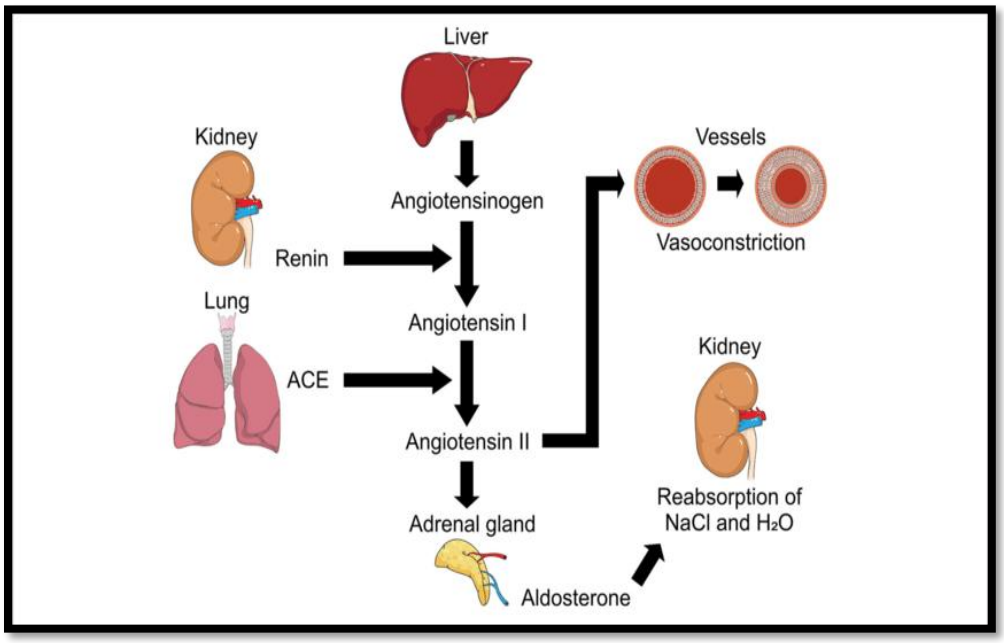


Phases of the physiological response to injury

### The Ebb Phase

- The ebb phase begins at the time of injury and lasts for approximately 24–48 hours.
- It may be attenuated by proper resuscitation, but not completely abolished.
- The ebb phase is characterized by:
  - ✓ **Hypovolemia**
  - ✓ **Decreased basal metabolic rate**
  - ✓ **Reduced cardiac output**
  - ✓ **Hypothermia**
  - ✓ **Lactic acidosis**.

- The predominant hormones regulating the ebb phase are catecholamines, cortisol and aldosterone (following activation of the Rennin-Angiotensin-Aldosterone system –RAAS).




**Renin Angiotensin Aldosterone System- RAAS**

- The main physiological role of the ebb phase **is to conserve both circulating volume and energy stores for recovery and repair**.

## The Flow Phase

- Following resuscitation, the ebb phase evolves into a hypermetabolic flow phase, which corresponds to SIRS.
- This phase **involves the mobilization of body energy stores for recovery and repair, and the subsequent replacement of lost or damaged tissue.**
- It is characterized by:

- **Tissue oedema (from vasodilatation and increased capillary leakage)**
- **Increased basal metabolic rate (hypermetabolism)**
- **Increased cardiac output**
- **Raised body temperature**
- **Leukocytosis,**
- **Increased oxygen consumption**
- **Increased gluconeogenesis.**

- The flow phase may be subdivided into an initial **catabolic** phase, lasting approximately **3–10 days**, followed by an **anabolic** phase, which may last for **weeks.**

  During the catabolic phase, the increased production of counter-regulatory hormones (including catecholamines, cortisol, and glucagon) and inflammatory cytokines results in significant fat and protein mobilization, leading to significant weight loss. The increased production of insulin at this time is associated with significant insulin resistance and, therefore, injured patients often exhibit poor glycemic control.

## Outcomes of the flow phase

### 1. Hypermetabolism with increased energy expenditure

The majority of trauma patients demonstrate energy expenditures approximately 15–25% above healthy resting values.

the factors that contribute to increase the energy expenditures are:

- **Increased sympathetic activity**
- **Abnormalities in circulation and increased cardiac output**
- **Increased protein turnover.**

Theoretically, patient energy expenditure could rise even higher than observed levels following surgery or trauma, but several features of standard intensive care counteract the hypermetabolic forces of the stress response, these factors include:

- **Bed rest**
- **Oxygenation**
- **External temperature regulation**

## 2. Skeletal Muscle Wasting

Muscle protein is continually synthesized and broken down. Under normal circumstances, synthesis equals breakdown and muscle bulk remains constant.

During the metabolic response to injury (catabolic phase), the body reprioritizes protein metabolism away from peripheral tissues and towards key central tissues such as the liver, immune system and wounds. The major site of protein loss is peripheral skeletal muscle. Muscle wasting occurs as a result of an increase in muscle protein degradation, coupled with a decrease in muscle protein synthesis.

Clinically, a patient with skeletal muscle wasting will experience:

a. **Asthenia (weakness)**
b. **Increased fatigue**
c. **Reduced functional ability**
d. **Decreased quality of life (increased morbidity) and increased risk of mortality.**

Immobility has long been recognized as a potent stimulus for inducing muscle wasting. Inactivity impairs the normal meal-derived amino acid stimulation of protein synthesis in skeletal muscle. Avoidance of unnecessary bed rest and active early mobilization are essential measures to avoid muscle wasting as a consequence of immobility.

## 3. Hyperglycemia

Following surgery or trauma, **postoperative hyperglycemia** develops as a result of:

- Increased glucose production (gluconeogenesis and Glycogenolysis) by the **effect of counter regulatory hormones**.
- The **effect of cytokines** to increase the insulin resistance (decreased glucose uptake in peripheral tissues).

The degree of insulin resistance is proportional to the magnitude of the injurious process. Following routine upper abdominal surgery, insulin resistance may persist for approximately 2 weeks. Postoperative patients with insulin resistance behave in a similar manner to individuals with diabetes mellitus and managed by insulin administration.

**This is the End of the Lecture – Good Luck**