General Surgery - Topic 1 - Metabolic response to injury

Homeostasis

- **Homeostasis** is the maintenance of a reasonably stable internal environment despite changes inside or outside the body.

What Homeostasis Means

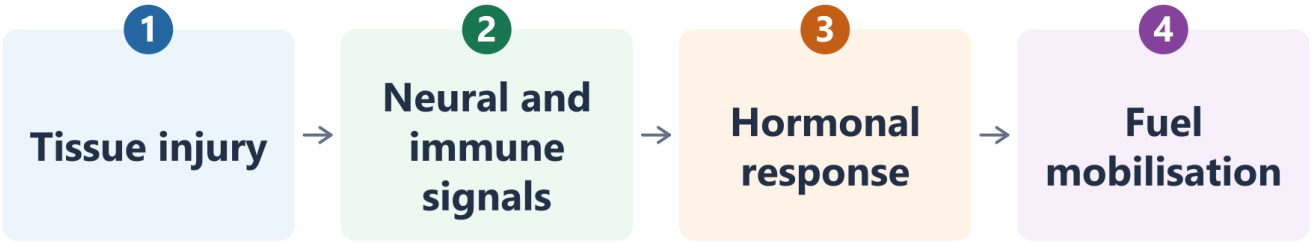
Physiological Regulation

- The body regulates blood pressure, oxygen delivery, temperature, glucose, electrolytes and acid–base balance through linked feedback systems.
- A normal blood pressure alone does not prove that every tissue is receiving enough blood.
- A patient can have early compensated shock while vasoconstriction temporarily maintains the blood pressure.

Effects of Injury and Prolonged Stress

- Injury disturbs this balance through pain, tissue damage, bleeding, inflammation and sometimes infection.
- The initial response redirects blood and fuel toward survival, immune defence and tissue repair.
- A severe or prolonged response can instead produce muscle wasting, poor healing and organ dysfunction.

How the Response Begins



Neural, Inflammatory and Volume Signals

- Pain and other sensory signals from the injured tissue activate the sympathetic nervous system and hypothalamus.
- Damaged cells release signals that activate local immune cells and inflammatory pathways.
- Reduced circulating volume also stimulates pressure-sensitive and kidney-mediated compensatory mechanisms.

Overlapping Responses and Injury Severity

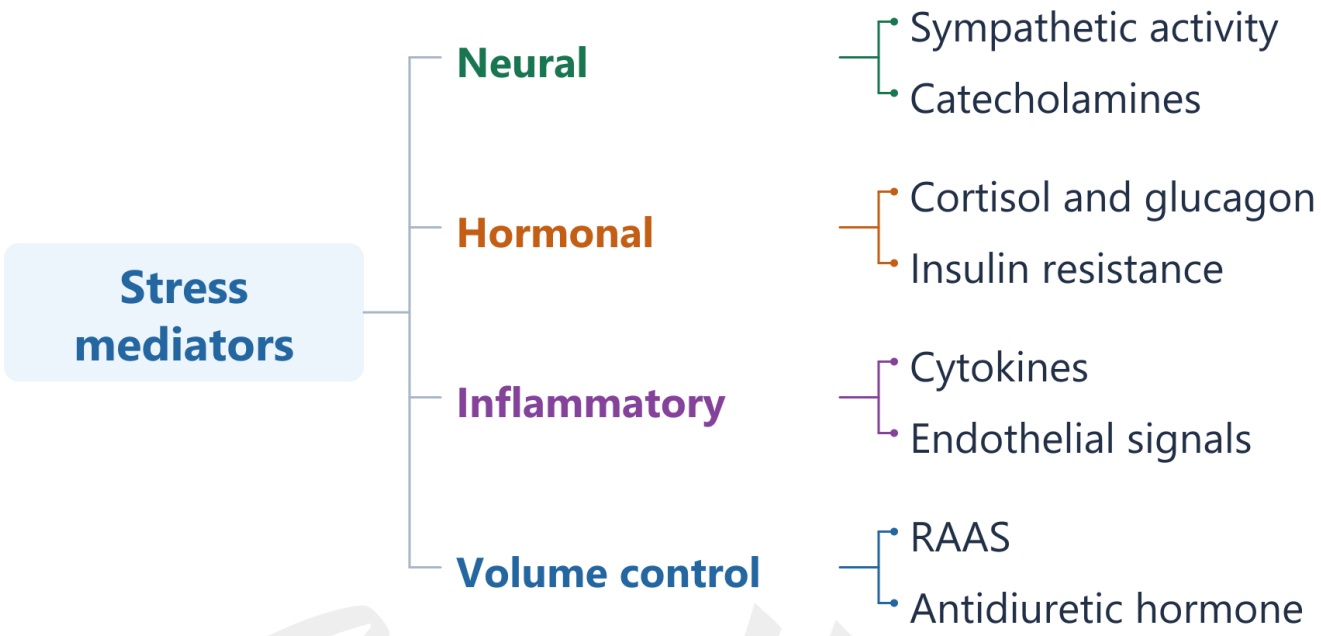
- These neural, hormonal and inflammatory signals overlap rather than acting as separate stages.

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- The response depends on the severity of injury and the patient's physiological reserve.

Mediators of the Metabolic Response to Injury

Neural, Hormonal and Inflammatory Mediators



1. Neural and Adrenal Responses

A. Sympathetic and Catecholamine Response

- Sympathetic activity and catecholamines increase heart rate, cardiac contractility and peripheral vasoconstriction.
- Adrenaline and noradrenaline also encourage release of stored fuels.

B. Hypothalamic–Pituitary–Adrenal Response

- The hypothalamus stimulates pituitary ACTH release, which increases cortisol secretion from the adrenal cortex.
- Cortisol supports vascular responsiveness and promotes glucose production and protein breakdown during stress.
- The response is useful initially, but persistent cortisol and inflammatory activity contribute to loss of muscle protein.

2. Metabolic Hormones

A. Glucagon

- Glucagon promotes hepatic glucose production so that glucose remains available during fasting and injury.

B. Insulin Resistance

- Insulin resistance means that a given amount of insulin has less effect on glucose uptake and storage.

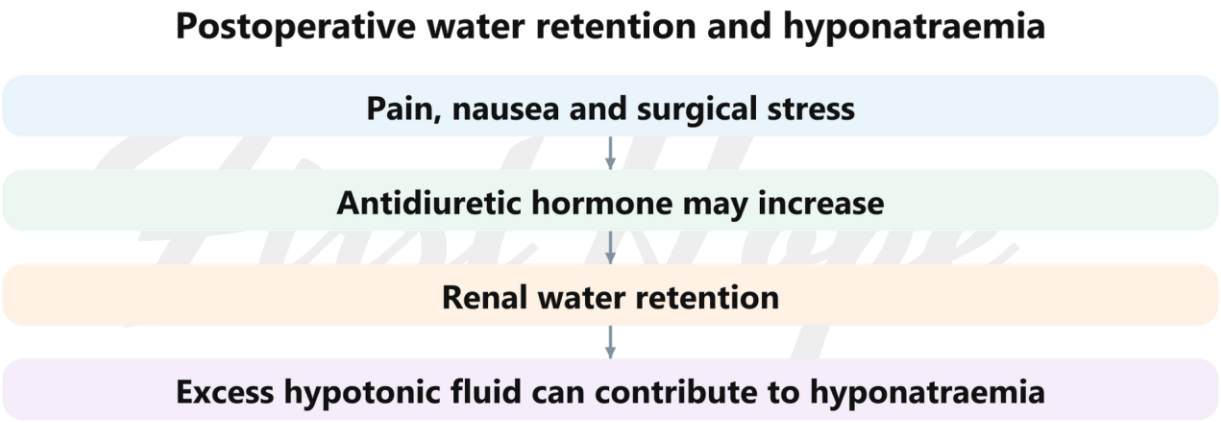
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- Insulin can be present at a normal or increased concentration even when the blood glucose is high.
- Stress hyperglycaemia therefore does not mean that all injured patients have permanent diabetes.

3. Inflammatory Mediators

- Cytokines such as interleukin-1, interleukin-6 and tumour necrosis factor help coordinate the inflammatory response.
- They influence fever, immune-cell activity, endothelial function and the production of acute-phase proteins in the liver.
- Complement, coagulation pathways and other local mediators also participate in the response.
- Excessive endothelial injury increases vascular leakage and can impair microcirculatory blood flow.

4. Salt and Water Conservation



- Reduced effective circulating volume activates the renin–angiotensin–aldosterone system.
- Aldosterone promotes renal sodium retention, while antidiuretic hormone promotes water retention.
- Pain, nausea and surgical stress can increase antidiuretic hormone even when additional water is not needed.
- Excess hypotonic fluid in this setting can contribute to postoperative hyponatraemia.

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Metabolic Changes and Their Consequences

Ebb, Flow and Recovery

Phase	Main physiological pattern	Clinical priority
Early ebb phase	Perfusion and oxygen consumption may fall when severe injury causes shock.	Control the cause and restore oxygen delivery.
Catabolic flow phase	Inflammation, fuel mobilisation and protein loss become prominent.	Treat complications and provide appropriate nutritional and organ support.
Recovery phase	Protein synthesis and functional recovery become possible as stress resolves.	Rebuild strength and nutritional reserves through rehabilitation.

1. Ebb Phase

- The **ebb phase** describes the early response to severe injury, particularly when shock and reduced perfusion are present.
- Restoring oxygen delivery and controlling bleeding are the immediate priorities during this phase.

2. Catabolic Flow Phase

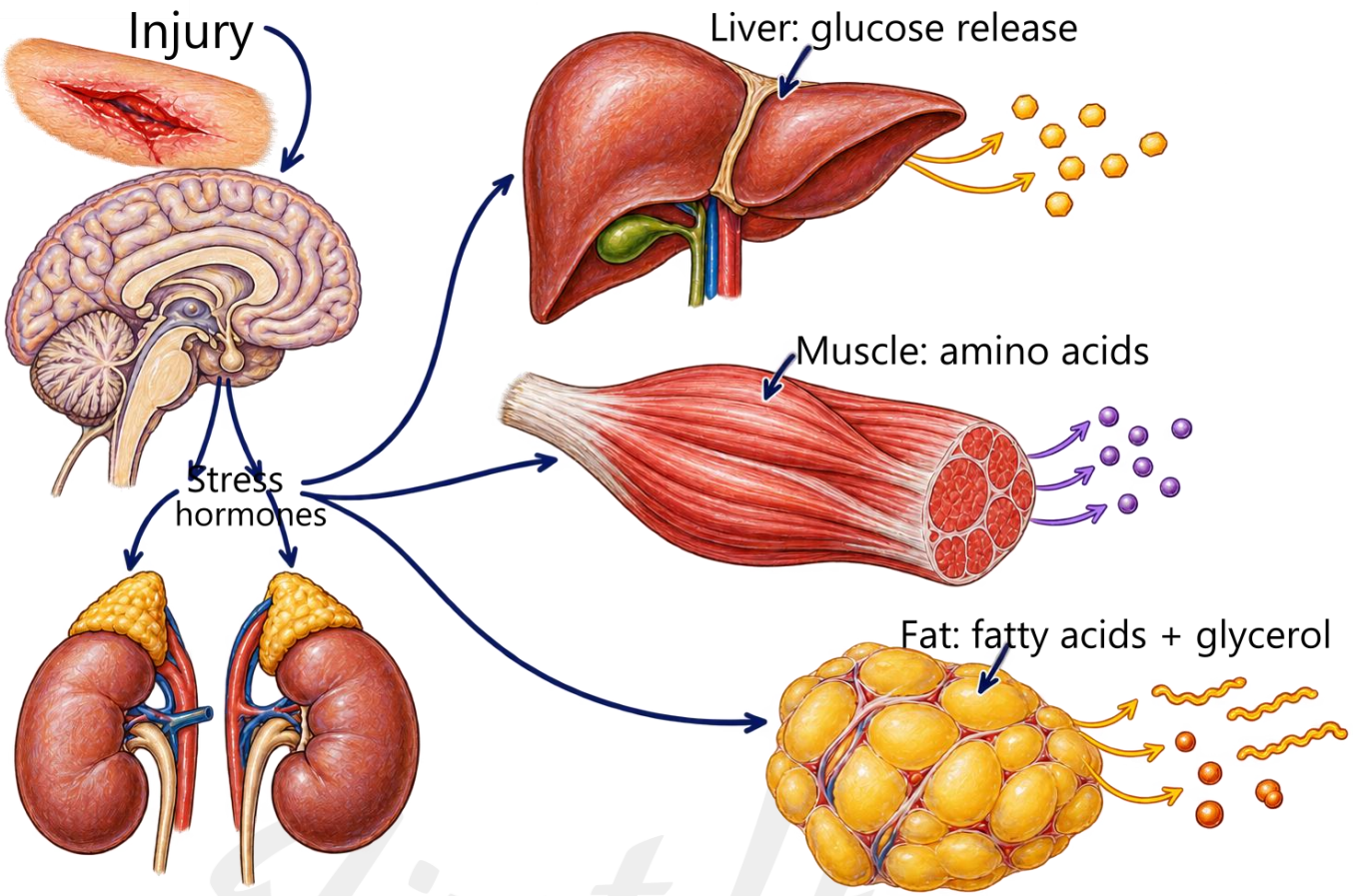
- The **flow phase** describes the subsequent hypermetabolic and catabolic response after circulation has been restored.
- The phases are a useful teaching model, but they do not occur at fixed times in every patient.
- Persistent sepsis, repeated operations or major burns can prolong catabolism and prevent a smooth transition to recovery.

3. Recovery Phase

- As inflammation settles and intake improves, net protein synthesis and restoration of lean tissue can gradually replace the earlier catabolic state.
- Mobilisation and rehabilitation help restore function, but the rate of recovery depends on the severity of illness and the patient's previous reserves.

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Changes in Carbohydrate, Protein and Fat Metabolism



Injury mobilises glucose, amino acids and fat-derived fuels through linked stress signals

1. Carbohydrate Metabolism

Hepatic Glucose Output and Insulin Resistance

- The liver initially releases glucose from glycogen and also makes glucose from substrates such as lactate, glycerol and amino acids.
- Insulin resistance reduces effective glucose uptake in some tissues and encourages continued hepatic glucose output.
- Blood glucose may rise even when the patient has eaten little.

Clinical Risks and Safe Glucose Control

- Marked hyperglycaemia can increase osmotic fluid loss and is associated with infection and poor wound outcomes.
- Glucose should be monitored and treated according to the perioperative or critical-care protocol.
- Aggressive attempts to achieve a very low glucose target can cause dangerous hypoglycaemia.

2. Protein Metabolism

- Skeletal muscle protein is broken down to provide amino acids for glucose production, acute-phase proteins and repair.

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