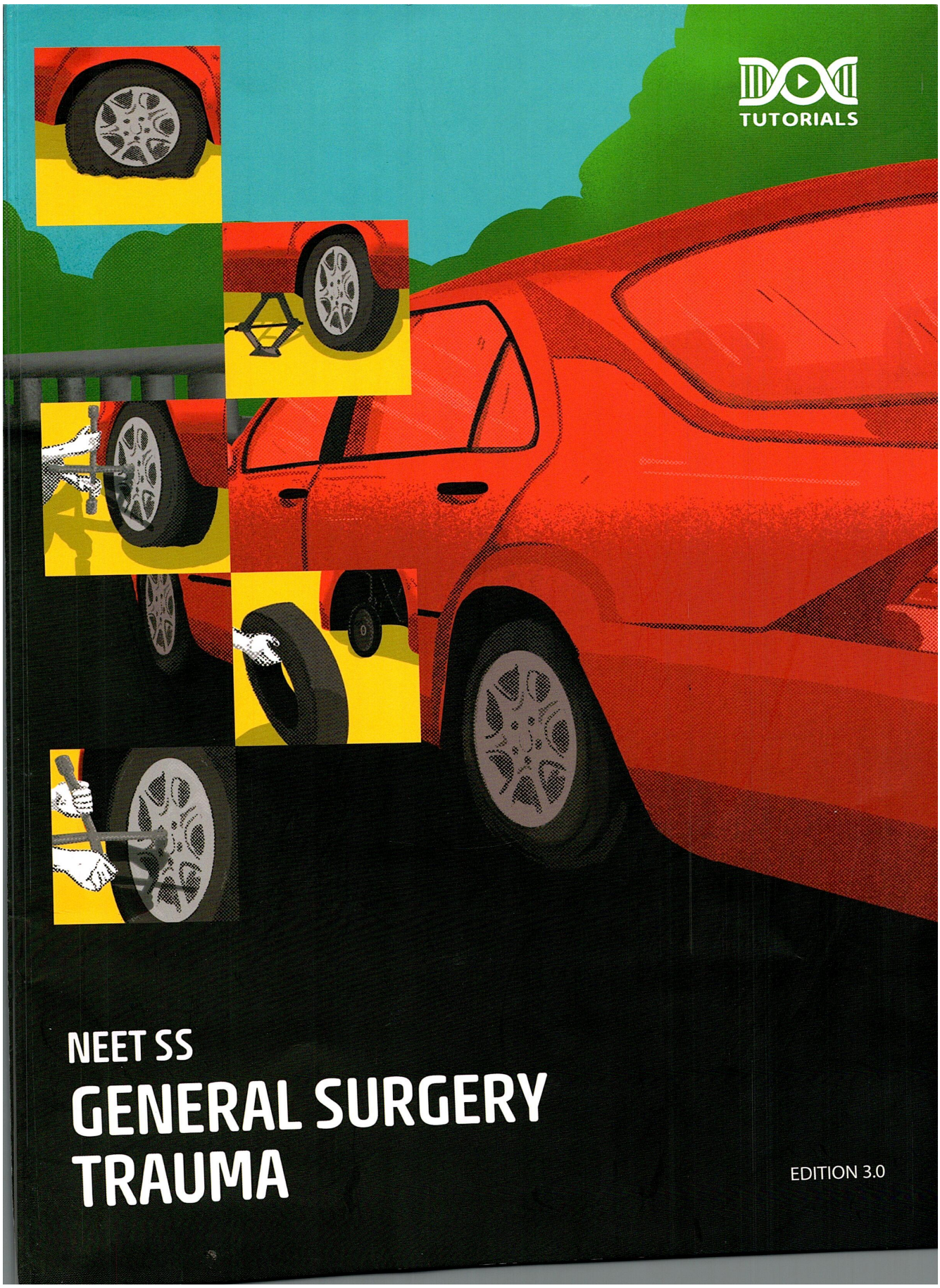


TUTORIALS

NEET SS

GENERAL SURGERY
TRAUMA

EDITION 3.0

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METABOLIC RESPONSE TO INJURY



INTRODUCTION

00:02:15

1. Metabolic Response to Injury/Surgery

Injury/Surgery
↓
Local tissue inflammation
↓
Systemic Changes affecting organ function

Surgical Stress Response

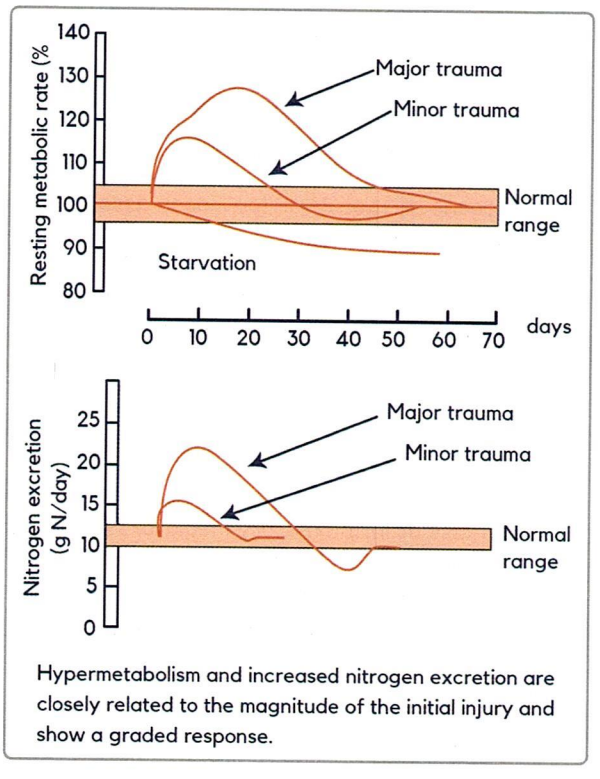
1) Hormones
2) Inflammation related cytokines
3) Neural circuits

↓
Alterations of
- Body metabolism
- Wound Healing
- Immunity
- Specific organ functions

↓
Collectively called as Metabolic response

Aim: Limit Damage and initiate repair process
Not all effects are beneficial

- Aim- Limit the damage and initiate repair process
- Not all the effects are beneficial- can cause infection, sepsis→ can impair wound healing
- Depends upon the extent of injury or surgery
- Metabolic response to injury is divided into-
  1. Initial period of catabolism- May include shock
  2. Anabolic phase of repair and tissue healing
  3. Initial period of catabolism- May include shock
- To conserve circulating volume and energy sources
- Characterised by hypovolemia; BMR; low cardiac output; hypothermia and lactic acidosis
- Accompanied by neurohormonal response
- Catabolism-To mobilise the body stores for recovery and repair
- Varies with severity of trauma
- Response is graded in nature
- Varies with time
- Varies between genetic individuals



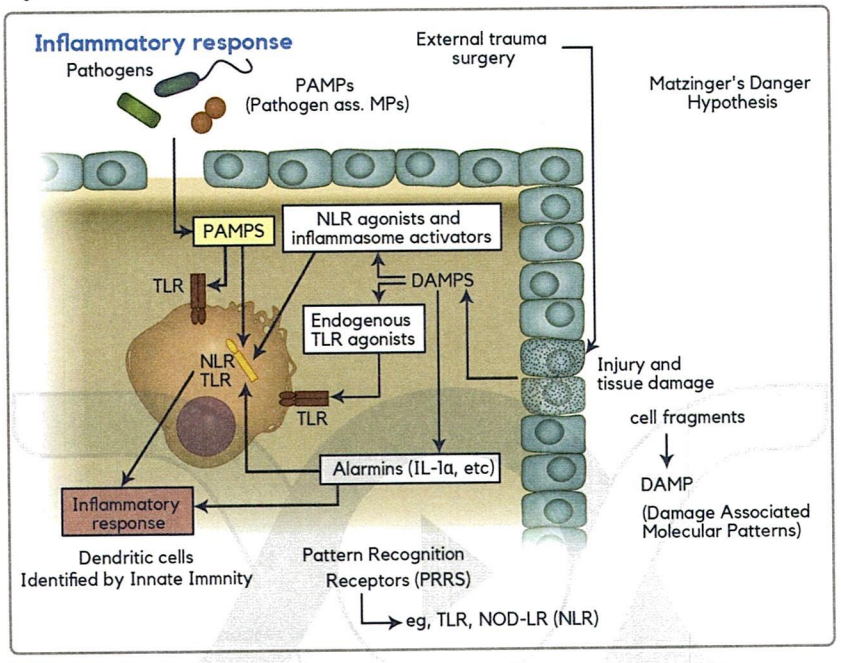
Hypermetabolism and increased nitrogen excretion are closely related to the magnitude of the initial injury and show a graded response.

Basic concepts
Homeostasis is the foundation of normal physiology 'Stress-free' perioperative care helps to preserve homeostasis following elective surgery Resuscitation, surgical intervention and critical care can return the severely injured patient to a situation in which homeostasis becomes possible once again The metabolic response to surgery influences these processes profoundly, particularly through catabolic effects, MODS and impired immunity

Inflammatory Response

00:12:10

- Varies between genetic individuals



MATZINGER DANGER HYPOTHESIS

External trauma/surgery

↓

Cell fragments

↓

DAMPS (Damage associated molecular patterns)

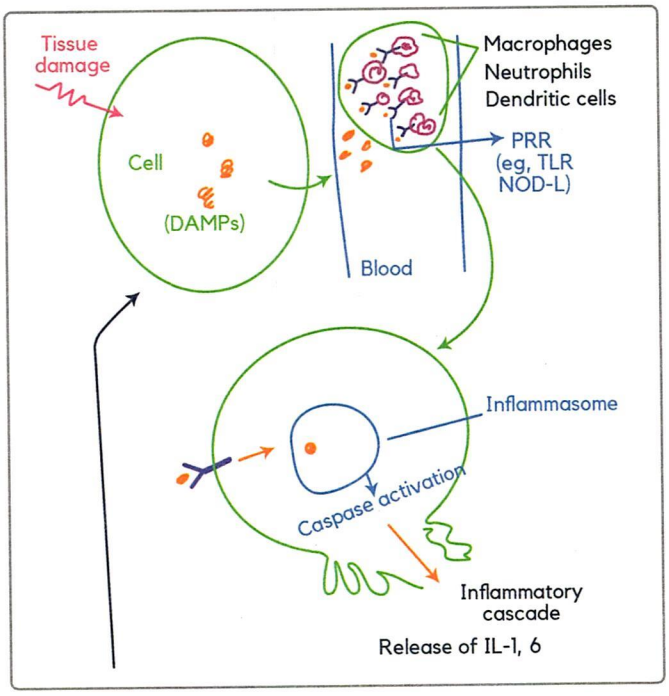
Pathogens release PAMP (pathogen associated molecular patterns)

DAMPS – Identified by innate immunity via Pattern recognition receptors (TLR, NOD-LR)-present on Antigen presenting cells

↓

Inflammatory response

- CASPASE ACTIVATION→ Inflammatory Cascade→ Release of inflammatory mediators (IL-1,6 and TNF α- primary cytokines) → elicit local tissue inflammation→ helps in repair
- When becomes systemic and uncontrolled→ SIRS due to cytokine storm – ARDS → MODS
  - AKI
  - DIC



Functions of inflammatory mediators

- Cell death
- Tissue damage
- Immune suppression
- Leaky capillaries
- Coagulopathy→ microthrombi→ local ischemia→ cellular injury→ damps→ inflammatory response

Damage-associated molecular pattern (DAMPs) and their receptors	
DAMP molecule	Putative receptor (S)
HMGB-1	TLRs (2,4,9), RAGE
Heat shock proteins	TLR2, TLR4, CD40, CD14
S100 protein	TLR4, RAGE
Mitochondria DNA	TLR9
Hyaluron	TLR2, TLR4, CD44
Biglycan	TLR2, and TLR4
Formyl peptides (mitochondrial)	Formyl peptide receptor I
IL-1α	IL-1 receptors

- HMGB1- High mobility group protein B1
  - Most common DAMP
  - Prognosticator of inflammatory response
- Heat shock protein 70-2nd most common
- S100-specific damp released in traumatic brain injury

Some secondary triggers of the metabolic response to injury.
Secondary triggers of inflammatory pathways in trauma and surgery • Sepsis • Haemorrhage • Massive transfusion • Acidosis • Surgery • Crush syndrome • Ischaemia-reperfusion
These events can amplify or prolong the catabolic phase, leading to organ failure or immune dysfunction.

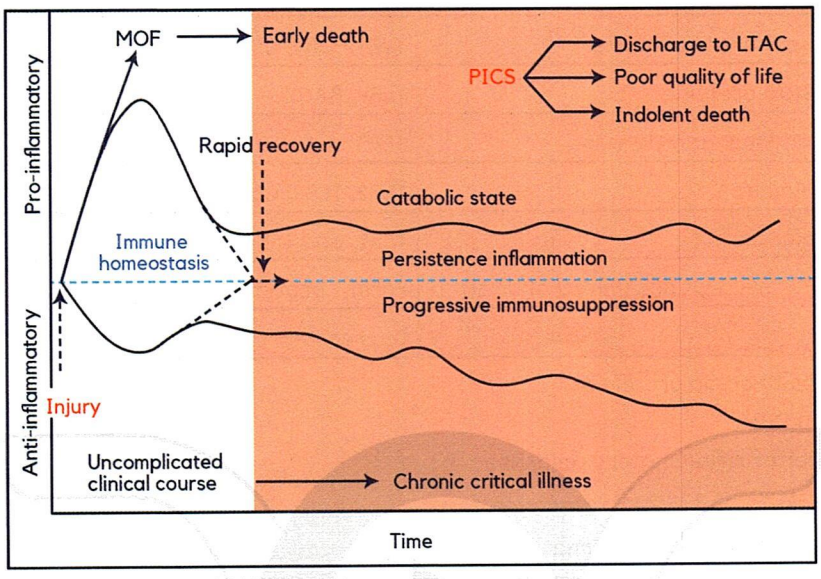
INFLAMMATORY RESPONSE

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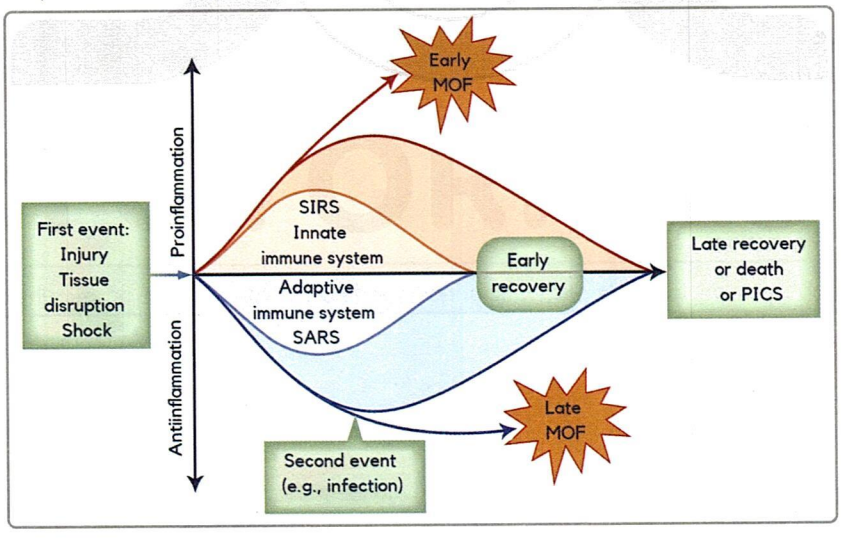
Inflammatory response

First 24 hours	After 24 hours	Further changes
Pro inflammatory Mediators	Balancing	Adaptive changes CARS
Cytokines-TNF-alpha, IL-1, IL-6 and IL-8, IL-12, IFN Y	Endogenous cytokine antagonists IL-1Ra TNF-sR 55 and 75	By IL-4, 5, 9, 10 and 13 TGF-beta Immunosuppression Infections
Nitric Oxide Prostanoids Endothelin-1	Local-Specialised Proresolving Mediators Lipoxins, Resolvins, Protectins and Maresins	

- Hyperacute mediators- TNF α, IL-1
  - Released into circulation within mins to hrs
  - Short half life
- Subacute mediators - IL-6 IL-8
  - Can be measured
  - Key accurate prognosticator of outcome
- Endogenous cytokine antagonists & proresolving mediators- Decide the magnitude and duration of inflammatory response
- CARS-compensated anti-inflammatory response syndrome- body will be in immunosuppression and prone for infections



- PICS -Persistent inflammation immunosuppression catabolic syndrome
  - Anti-inflammatory system is brought out by adaptive immunity
- Adaptive immunity has to be suppressed to get overwhelming anti-inflammatory response



- SIRS- Due to overwhelming innate immunity system
  -IL-1, IL-6, TNF α

  ↓

  Local Tissue injury /edema/ vasodilation/ microthrombi
- When limiting measures fail → these cytokines released into systemic circulation→ Cytokine storm →SIRS- Multiple actions at multiple organ levels (ARDS, AKI, DIC)
- SIRS-1. temp >38° C/ <36° C
  2. TC >11,000/<4000/>10 % band forms
  3. RR>20/min
  4. HR>90/min

- Any of the 2 criteria + ->SIRS
- CARS- compensated anti-inflammatory response syndrome
  - Simultaneous and opposing response to SIRS
  - ↓ in HLA DR molecules
  - Suppression of adaptive immune system
  - LYMPHOPENIA

↓

Profound immunosuppression

↓

Impaired wound healing

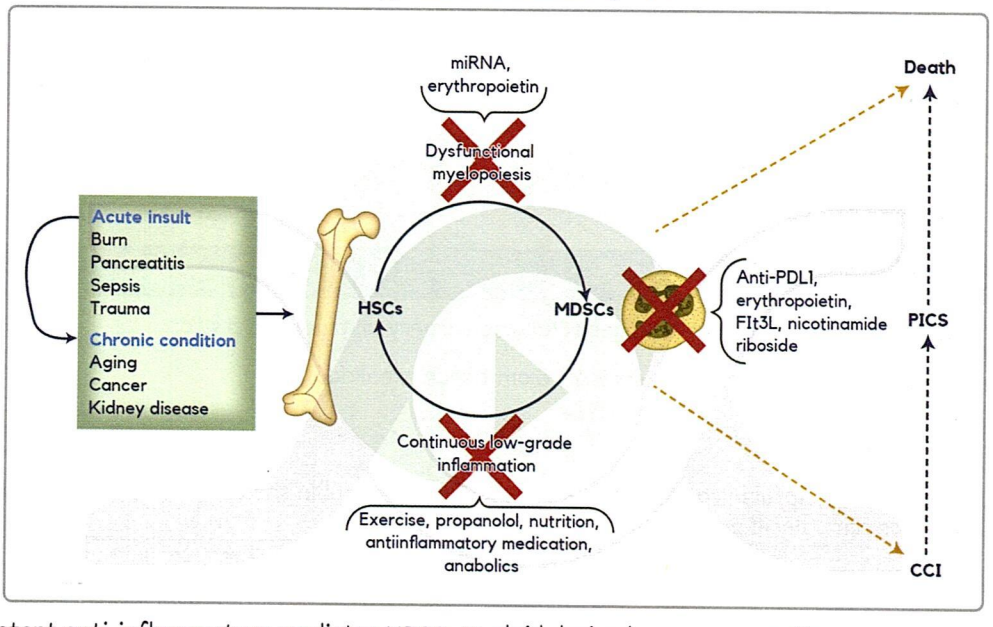
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Increased infections

↓

Late MOF/late death by sepsis

- PICS- Persistent inflammation immunosuppression catabolic syndrome



TGF β- Most potent anti-inflammatory mediator MDSC- Myeloid derived suppressor cells

↓

Constantly release TGF β

↓

CARS

↓

Profound lymphopenia

↓

Profound immunosuppression

↓

Catabolism

Neuroendocrine Response

- Responsible for the catabolic effects

Neuroendocrine response

Injury → Pain Pathway → Spinal Cord → Thalamus → Hypothalamus

Hypothalamus → CRH → Pituitary
- ↑ACTH → ↑Corisl, ↓IGF-1, ↓Insulin
- ↑GH → ↓IGF-1
- ↑Prolactin

Hypothalamus → Activate symp nervous system → ↑Adrenaline, ↑Glucagon

Overall - ↓Thyroid hormones, ↓Testosterone

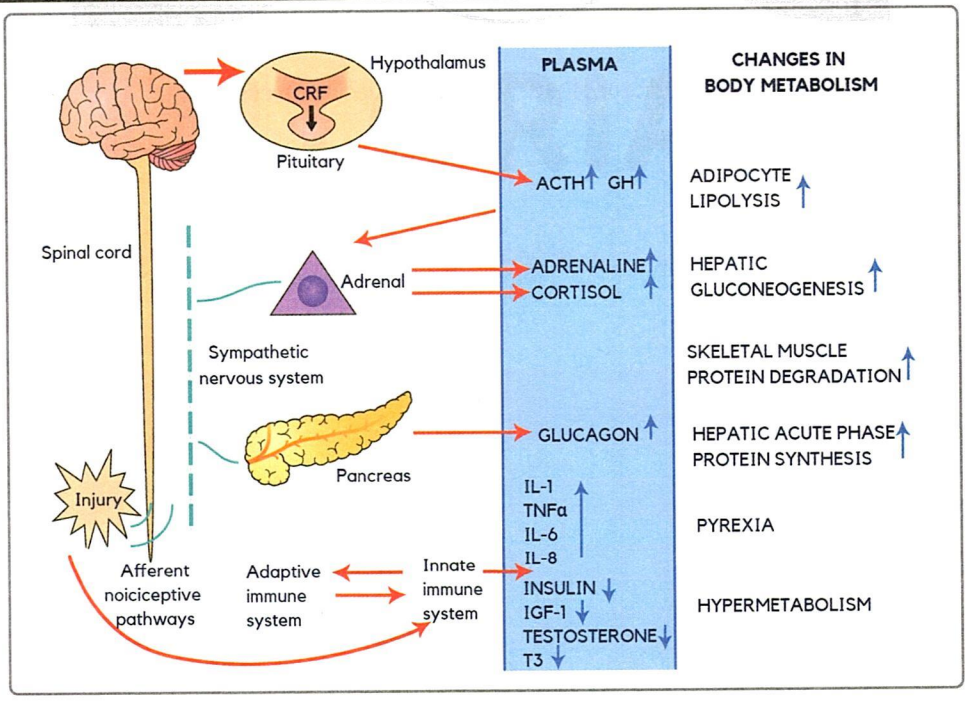
- Acute phase response- ↑Cortisol ↑adrenaline ↑glucagon
  ↓
- Mobilise body stores
- Chronic phase- hypothalamic suppression
  ↓
  ↓thyroid hormones
  ↓ testosterone

Purpose of neuroendocrine changes following surgery or trauma

The constellation of neuroendocrine changes following surgery or trauma acts to:

- Provide essential substrates for survival from tissue breakdown
- Postpone anabolism
- Optimise host defence

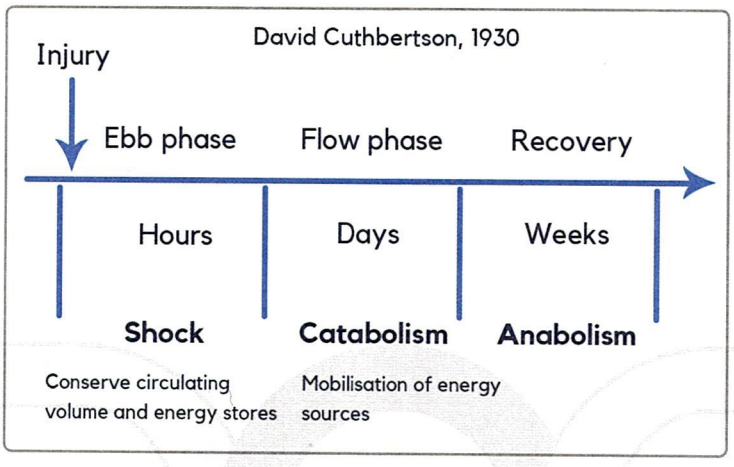
These changes may be helpful in the short term, but may be harmful in the long term, especially to the severely injured or critically ill patient.



The metabolic response to surgery and injury: key characteristics
• Rapid onset driven by proinflammatory cytokines (e.g. IL-1, IL-6 and TNFα) • Broadly related to injury severity: most severe in sepsis, burns and major trauma • Varies in severity between individuals (genetic) • Causes catabolism, muscle breakdown, immunosuppression and organ dysfunction/failure • Counterbalanced by antagonist response but the balance may be imperfect • Prolonged by sepsis and other secondary insults • Can become chronic • Associated with most late deaths from injury or surgery in developed health systems

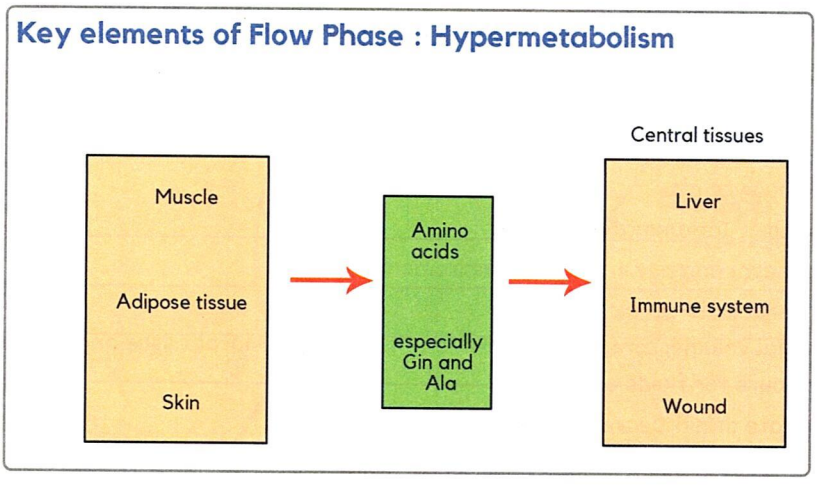
Metabolic Response

00:47:40



Hypermetabolic flow phase

- Tissue oedema
- Increased basal metabolic rate
- Increased cardiac output
- Raised body temperature- due to cytokines
- Leucocytosis-inflammatory response
- Increased oxygen consumption
- Increased gluconeogenesis



- Protein breakdown requires ATP (ATP dependent ubiquitin proteosome pathway)
- AA → hepatic gluconeogenesis (require ATP)
- Lactate conversion requires ATP dependent Cori cycle

Why hypermetabolism?

- BEE 15-25 % Above predicted healthy resting values
- Due to
  1. Central Thermo dysregulation(cytokines)
  2. Increased sympathetic activity(catecholamines)
  3. Abnormalities in wound circulation (ischemia-lactate-ATP dependent metabolism)
  4. Increased protein turnover
  5. Nutritional support
- Skeletal muscle breakdown→ Proteins→ AA→ liver
- Liver- Acute phase protein response (APPR)
- Positive acute phase reactants-fibrinogen, CRP
- Negative acute phase reactants-albumin
- ↓ albumin – due to 1) haemodilution
  2) ↑ transcapillary escape rate of albumin

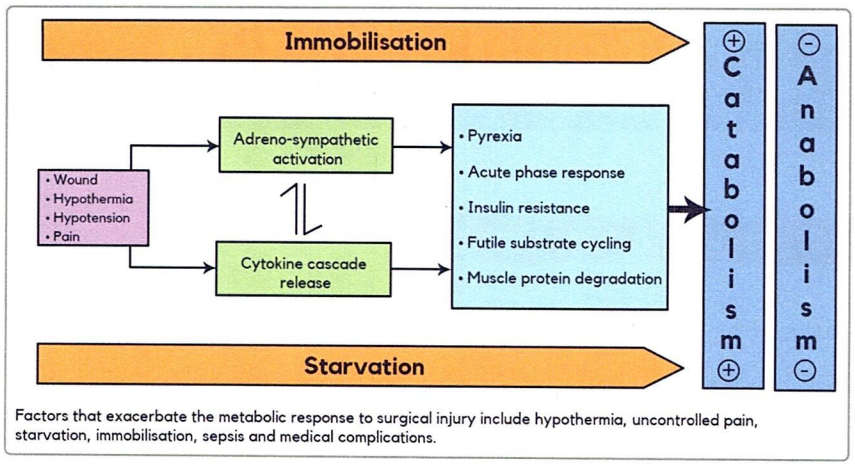
Stress Free Perioperative Care

00:56:57

Some secondary triggers of the metabolic response to injury.
Secondary triggers of inflammatory pathways in trauma and surgery • Sepsis • Haemorrhage • Massive transfusion • Acidosis • Surgery • Crush syndrome • Ischaemia-reperfusion
These events can amplify or prolong the catabolic phase, leading to organ failure or immune dysfunction.

- Decrease the secondary triggers
- Precautions to avoid compounding factors after surgery
  1. Limit crystalloids with no weight gain during elective surgery
     - Stress→ ADH→ Na, h2O retention→ Oliguria
     - Tissue edema, ↑ blood volume → ileus, weight gain
  2. Maintain normothermia using forced body air warmers
     - Decrease wound complications
     - Decrease cardiovascular complications
     - Less risk of bleeding
  3. Maintain normoglycemia
     - Hyperglycemia-Inflammatory marker
       - ➤ Endothelial damage→ tissue inflammation
       - ➤ ↑ Free radicals-cellular injury
  4. Maintain normal volume, control inflammation Tissue edema- local tissue anoxia-inflammation
  5. Fasting two hours for fluids enough
     - Carbohydrate drink- Decreased periop anxiety and thirst
       - ➤ Decreased post op insulin resistance
  6. Avoid unnecessary bed rest and Active early mobilisation When muscles are inactive→ They cannot promote anabolism

They cannot express GLUT 4 receptors



Factors that exacerbate the metabolic response to surgical injury include hypothermia, uncontrolled pain, starvation, immobilisation, sepsis and medical complications.

A proactive ERAS approach to prevent unnecessary aspects of the surgical stress response

- Minimal access techniques
- Blockade of afferent painful stimuli (e.g. epidural analgesia,
- Spinal analgesia, wound catheters)
- Minimal periods of starvation
- Early mobilisation

Q & A discussion

- Surgical stress response is brought about by all except-immunological system
- Metabolic response to injury- neurohumoral response is not responsible for anabolism and recovery
- They drive catabolism and postpone anabolism
- Modern surgical care is aimed at all except
  - Increase the need for homeostatic response
  - Minimal access elective surgeries
  - Stress free perioperative care
  - Enhanced recovery after surgery
- Decreasing the need for homeostatic response
- Response to injury-evolves with time
- Nearer endocrine response does not include-insulin. Release Glucagon is released
- Glucagon is released
- Both pro and anti-inflammatory mediator- IL-6
- Stimulates the release of cortisol
- Lipoxins-anti inflammatory
- DAMP-protectin is specialised pro resolving mediators
- Key mediator of response-neutrophils
- Accurate prognosticator of outcome- IL-6
- Damage controlled resuscitation Is not a 2° trigger
- CARS- due to suppression of adaptive immune system
- PICS-is a vicious cycle
- Ebb phase- no increased BEE
- Hypermetabolic phase/catabolic phase does not feature-SIRS
- Measures to prevent stimulating hypermetabolic or catabolic response- avoid prolonged fast
- Proactive approach in perioperative period to prevent unnecessary aspects of surgical stress response includes all except-8 hours of fast
- Max hours of fasting
  - 2 hrs for clear liquids
  - 4 hrs for breast milk
  - 6 hrs for formula feeds/cow milk
  - 8+ hrs- heavy meal
- Use of epidural analgesia- blunts neuroendocrine response

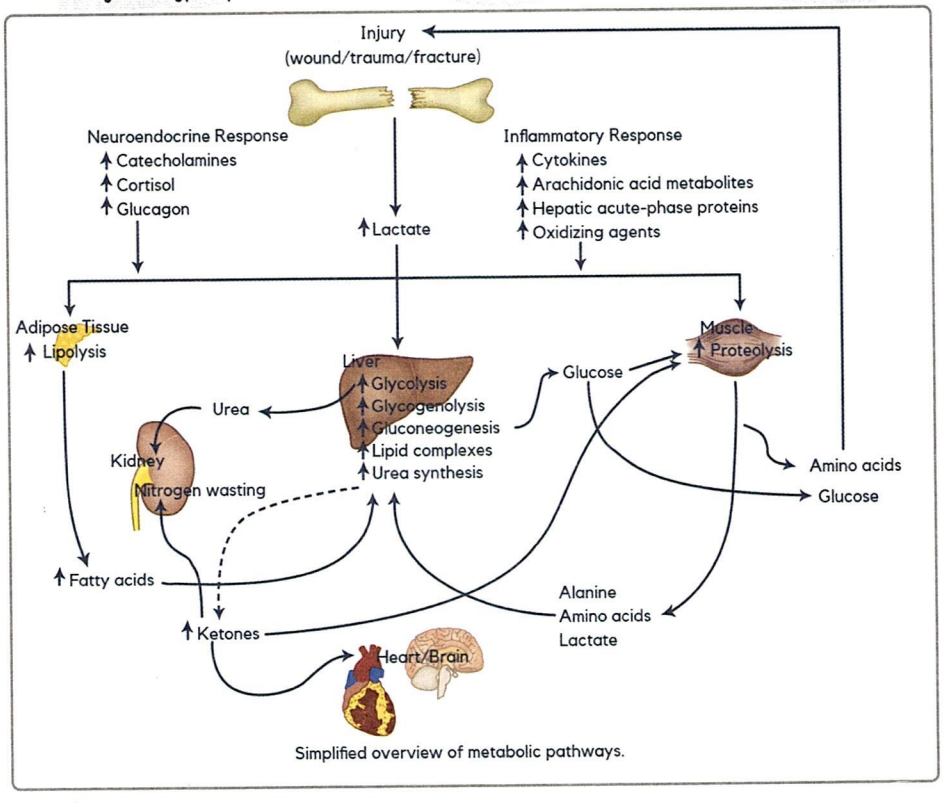
INJURY RESPONSE VS STARVATION ON NUTRITION

00:01:25

- Up to 12 hrs
  - Body gets glucose from last meal intake
- After 12 hrs
  - Liver glycogen stores will get depleted
  - 200 gms of glucose
  - Muscle Glycogen stores-500 gms of glucose
- After 24 hrs
  - Muscle breakdown-AAs-hepatic gluconeogenesis
  - Fat breakdown-Glycerol-AAs- hepatic gluconeogenesis
    - FFA-ketone bodies-fuel by heart and brain
    - Decreases the urine N2 excretion
    - Muscles are preserved
    - ADAPTIVE KETOGENESIS

Metabolic response to starvation

- Low plasma insulin
- High plasma glucagon
- Hepatic glycogenolysis
- Protein catabolism
- Hepatic glycogenesis
- Lipolysis: mobilisation of fat stores-overall decrease in protein and carbohydrate oxidation
- Adaptive ketogenesis
- Reduction in resting energy expenditure (25-30 Kcal/Kg per day to 15-20 Kcal/kg per day)



Simplified overview of metabolic pathways.