DT SS SURGERY

2nd edition (Latest)

VOL 1

SURGERY GROUP NOTES FOR NEET SS

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6. Endocrine

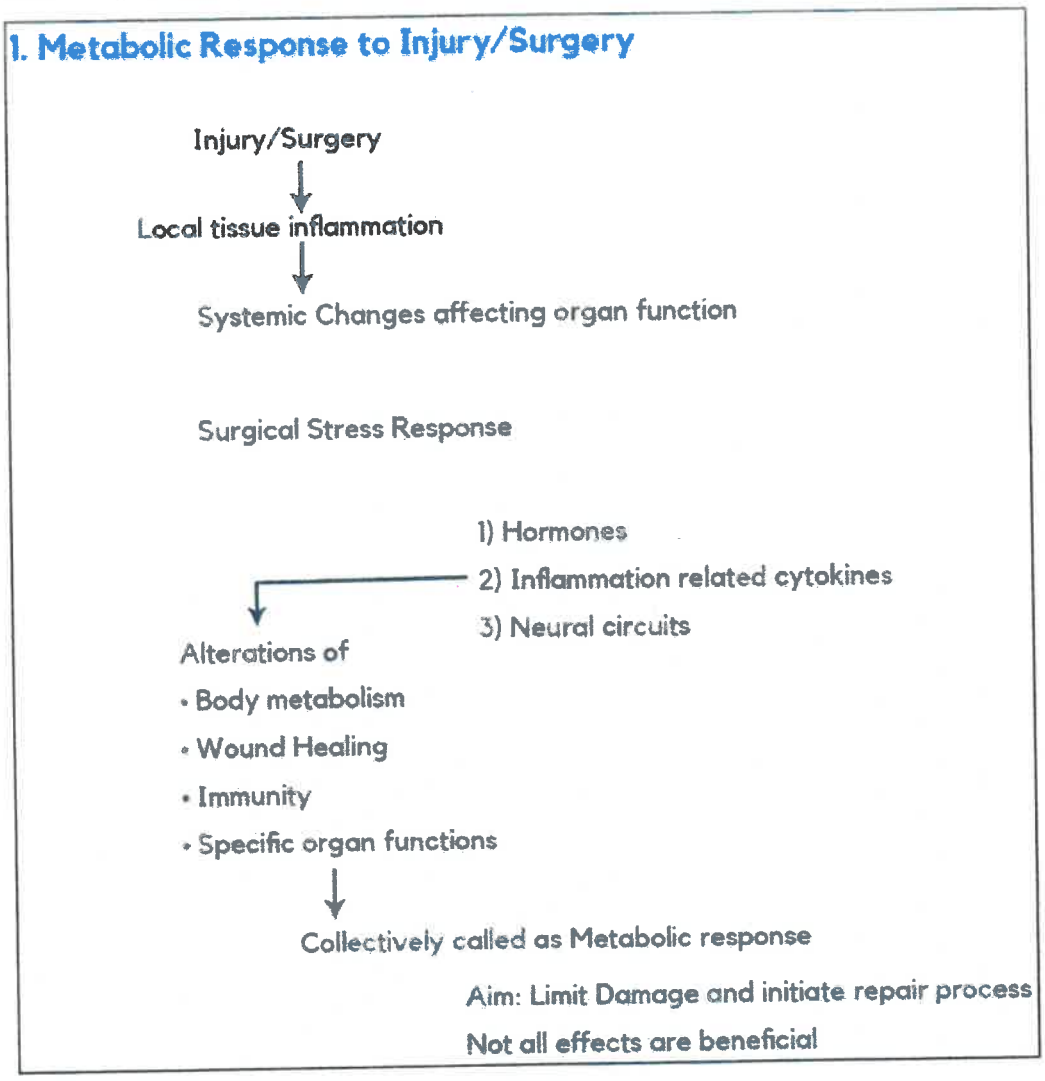
7. Oncosurgery

METABOLIC RESPONSE TO INJURY

INTRODUCTION

00:02:15

- Homeostasis- milieu interieur



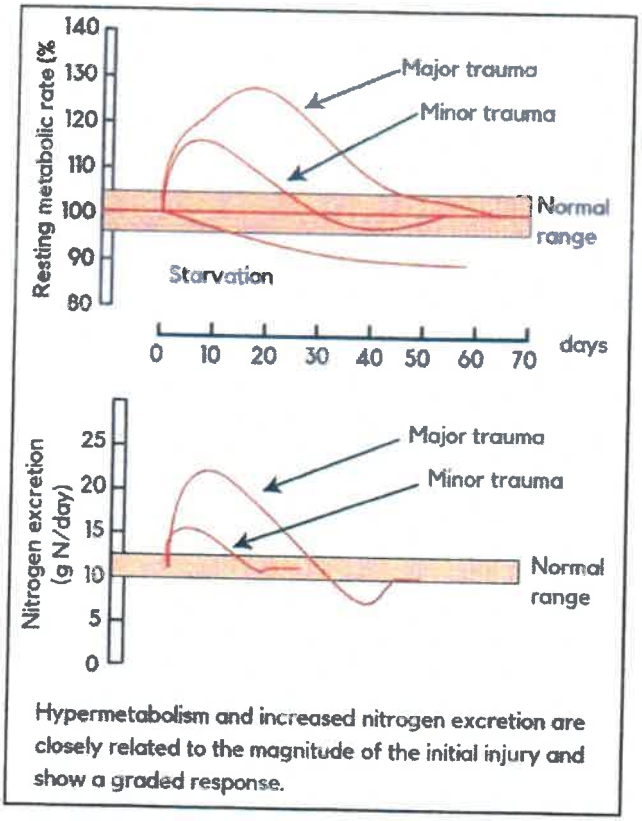
- Aim- Limit the damage and initiate repair process
- Not all the effects are beneficial- can cause infection, sepsis→ can impair wound healing
- Depends upon the extent of injury or surgery
- Metabolic response to injury is divided into-

1. Initial period of catabolism- May include shock
2. Anabolic phase of repair and tissue healing
   - Ebb/flow phases- removed from latest bailey 28th edition

   ↓ ↓

   Shock hypermetabolism
3. Initial period of catabolism- May include shock

- To conserve circulating volume and energy sources
- characterised by hypovolemia; decreased BMR; decreased 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



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

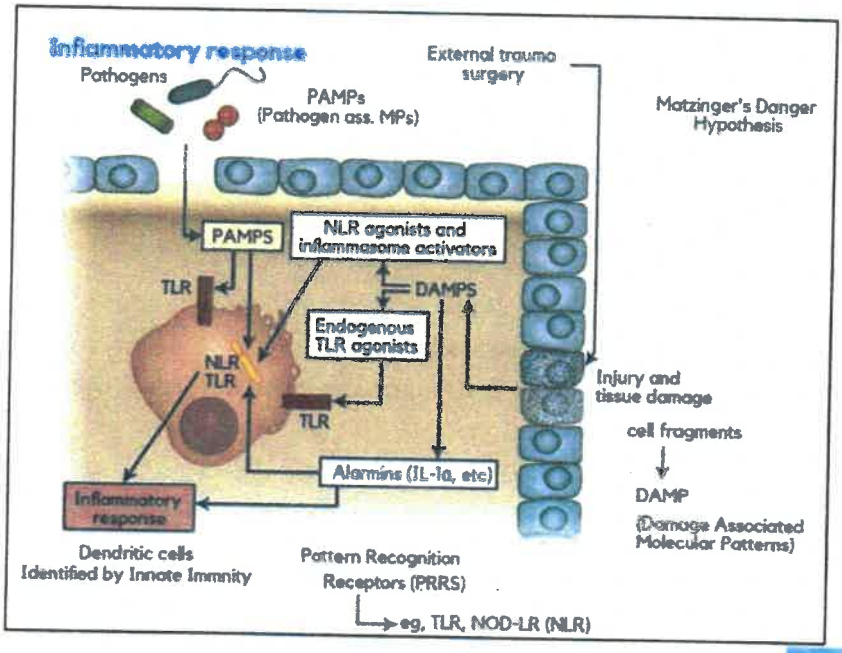
- *Response is graded in nature*
- *Varies with time*
- *Varies between genetic individuals*

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 impaired immunity*

Inflammatory Response

00:12:10



MATZINGER DANGER HYPOTHESIS

External trauma/surgery

↓

Cell fragments

↓

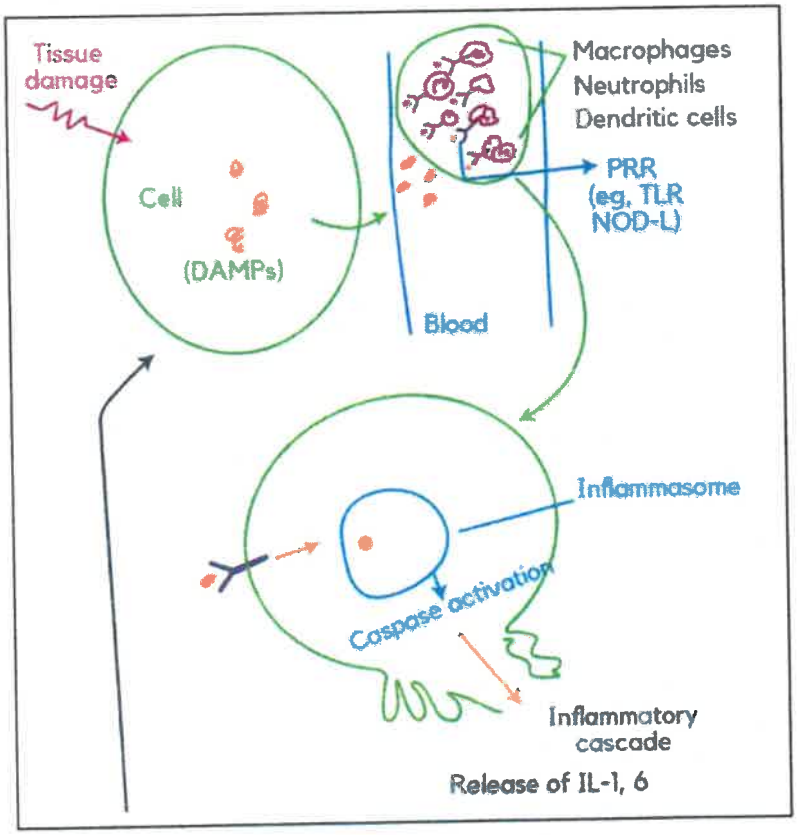
DAMPS (Damage associated molecular patterns)

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

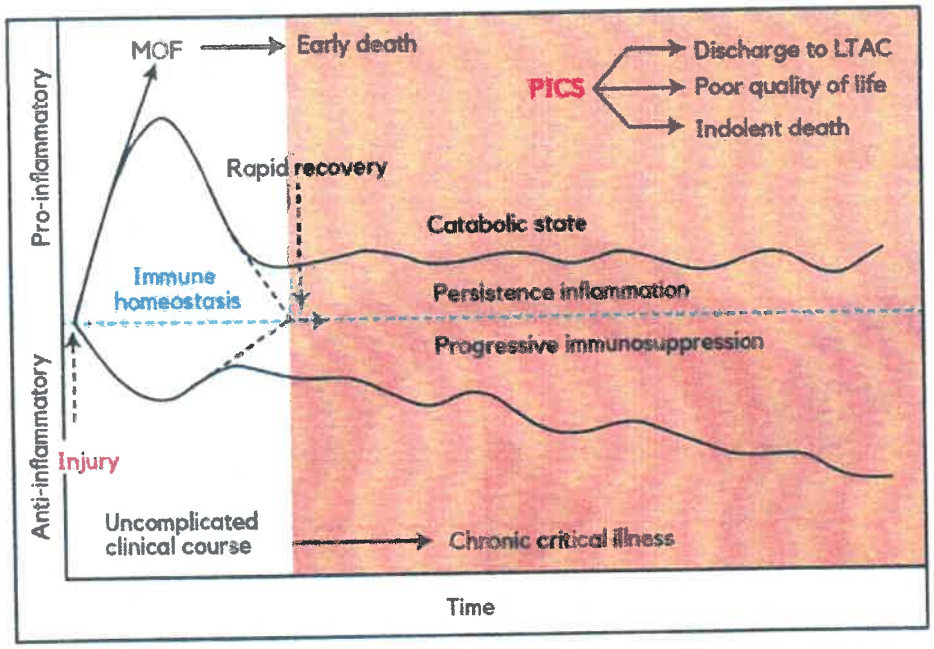
00:22:26

Inflammatory response

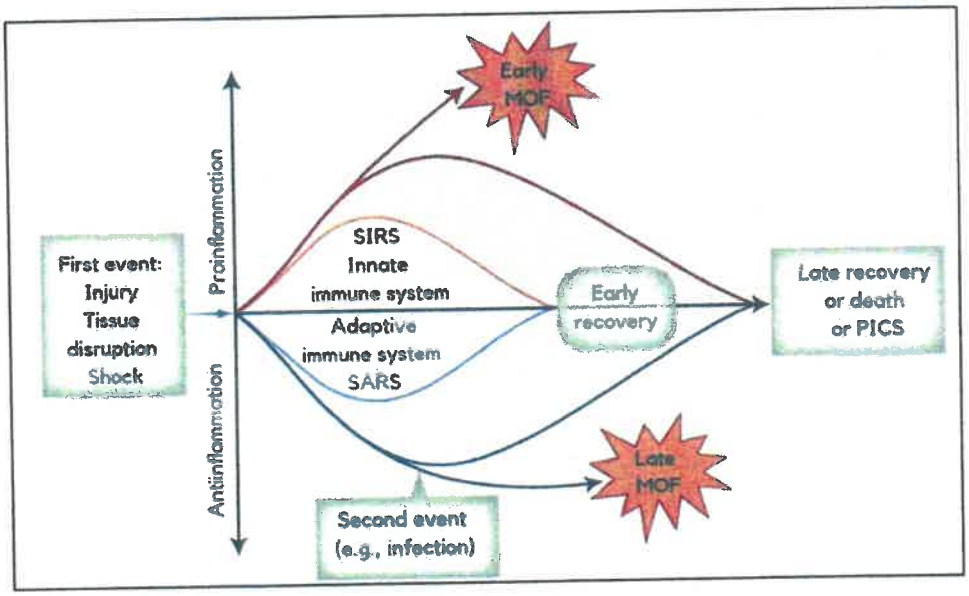
First 24 hours	After 24 hours	Further changes
Pro inflammatory Mediators	Balancing	Adaptive changes CARS
Cytokines-TNF-alpha, IL-1, IL-6 and IL-8, IL-12, IFN Y	Endogenous cytokine antagonists IL-1Ra TNF-sR 55 and 75	By IL-4, 5, 9, 10 and 13 TGF-beta Immunosuppression Infections
Nitric Oxide Prostanoids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

↓

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 >98° 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

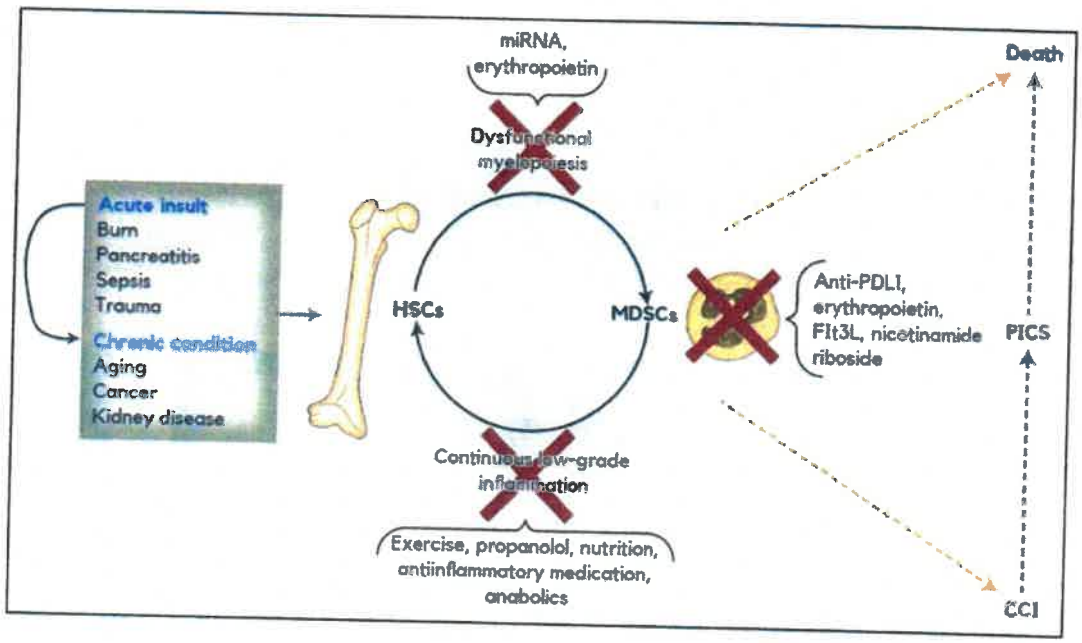
↓

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

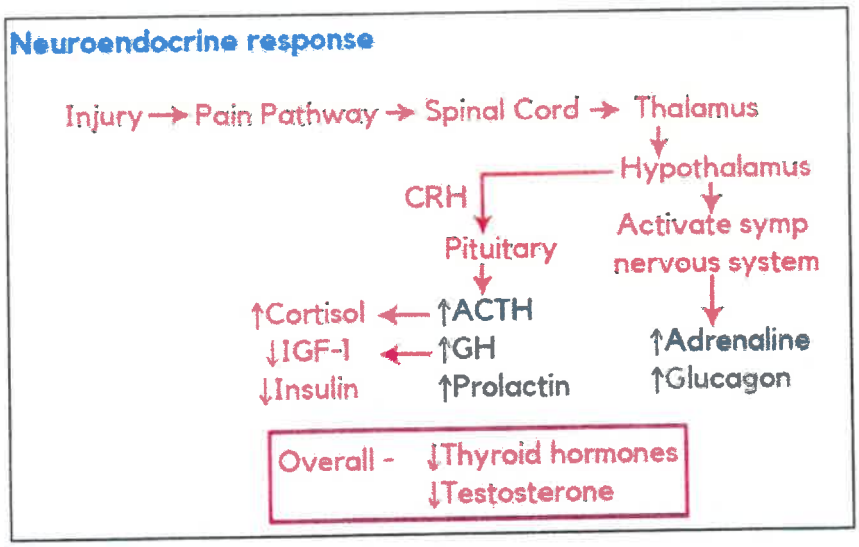
↓

Catabolism

Neuroendocrine Response

00:39:11

- Responsible for the catabolic effects



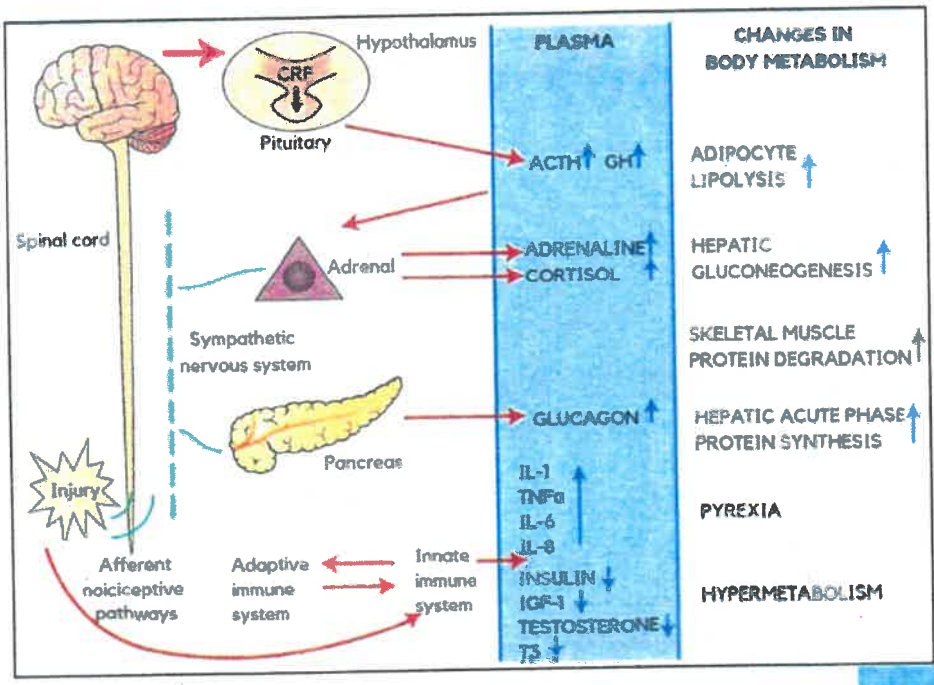
- Acute phase response- ↑Cortisol ↑adrenaline ↑glucagon
  ↓
- Mobilise body stores
- Chronic phase- hypothalamic suppression
  ↓
  ↓thyroid hormones
  ↓ testosterone

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

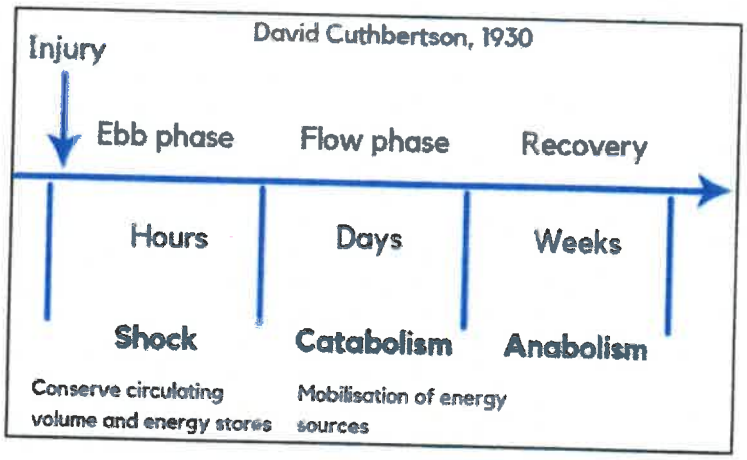
These changes may be helpful in the short term, but may be harmful in the long term, especially to the severely injured or critically ill patient.



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

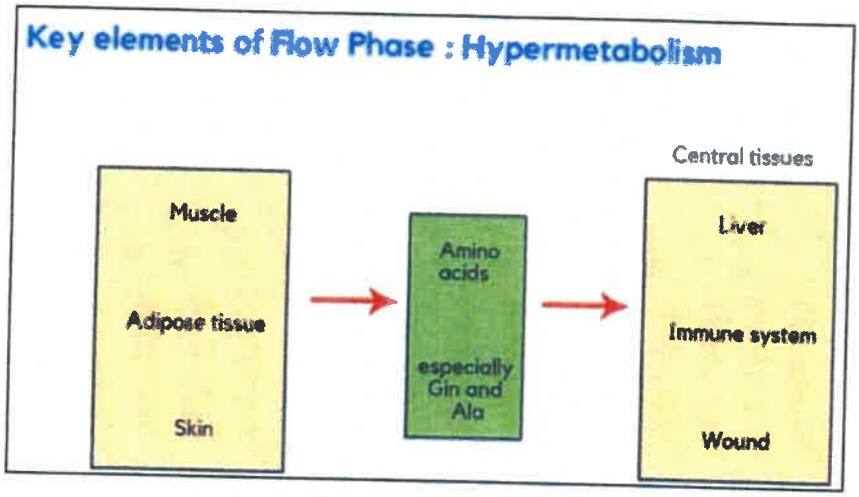
Metabolic Response

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?

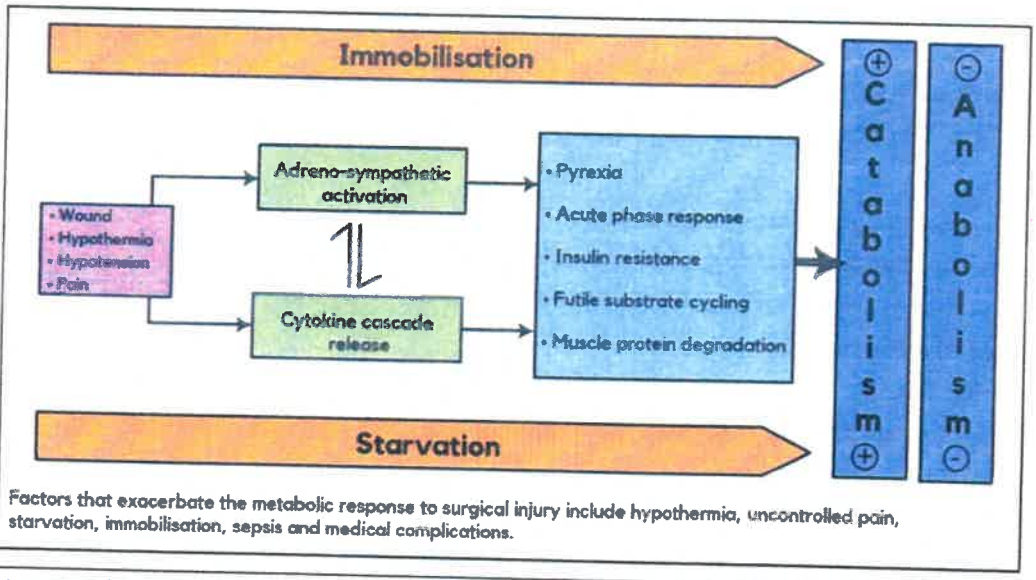
- BEE 15-25 % Above predicted healthy resting values
- Due to
  1. Central Thermo dysregulation(cytokines)
  2. Increased sympathetic activity(catecholamines)
  3. Abnormalities in wound circulation (ischemia-lactate-ATP dependent metabolism)
  4. Increased protein turnover
  5. Nutritional support
- 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
- 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

SURGICAL NUTRITION

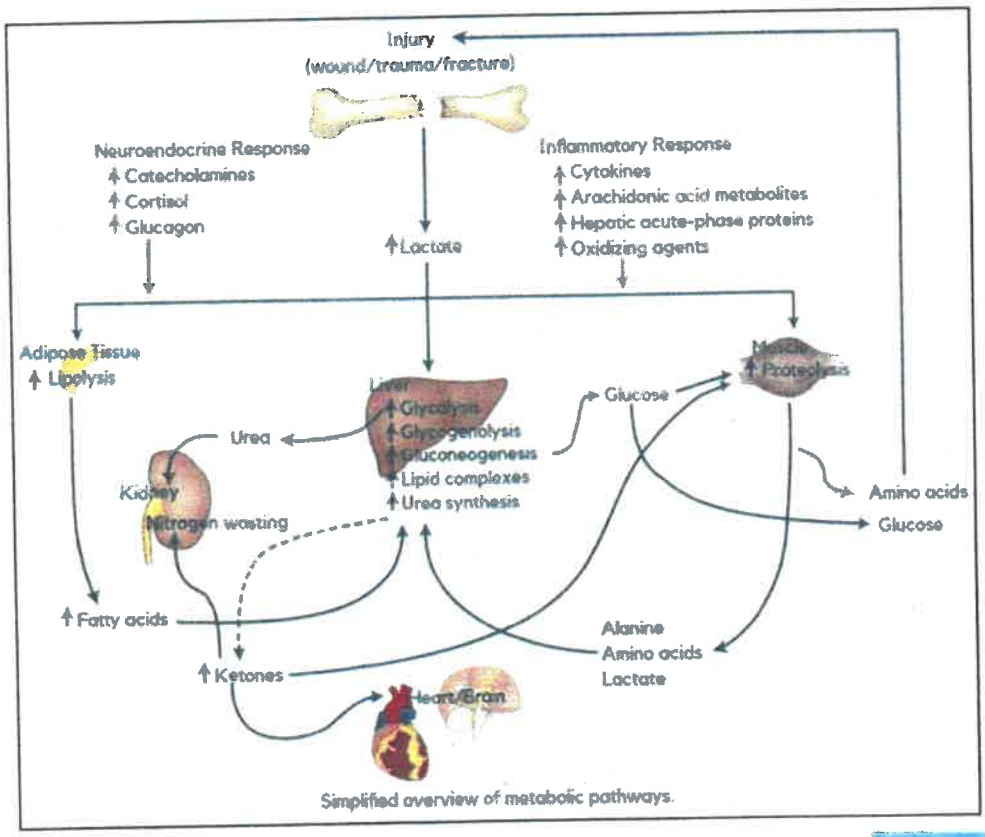
Injury response vs starvation on nutrition

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 (increased fat oxidation)-overall decrease in protein and carbohydrate oxidation
- Adaptive ketogenesis
- Reduction in resting energy expenditure (from approximately 25-30 Kcal/Kg per day to 15-20 Kcal/kg per day



Simplified overview of metabolic pathways.

No adaptive ketogenesis-

Metabolic response to trauma and sepsis
• Increased counter-regulatory hormones: adrenaline (epinephrine), noradrenaline (norepinephrine), cortisol, glucagon and growth hormone • Increased energy requirements (up to 40 kcal/kg per day) • Increased nitrogen requirements • Insulin resistance and glucose intolerance • Preferential oxidation of lipids • Increased gluconeogenesis and protein catabolism • Loss of adaptive ketogenesis • Fluid retention with associated hypoalbuminaemia

Effect of metabolic response to surgery on nutrition

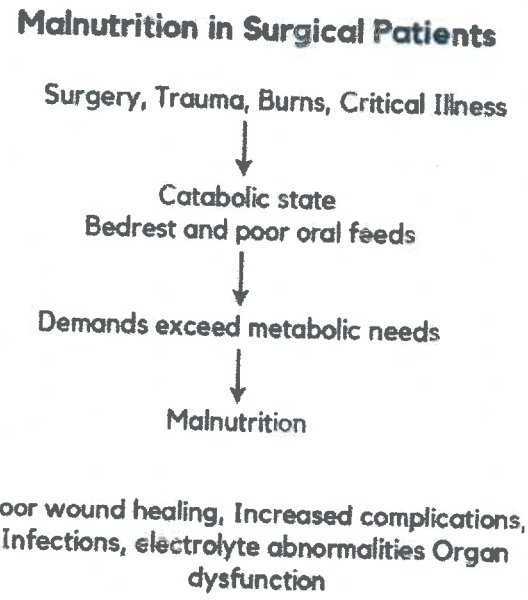
00:08:06

- Prolonged fasting
- Insulin resistance
- Hyperglycemia- Reduced peripheral glycolysis
  - Increased gluconeogenesis
  - Reduced GLUT 4 proteins
- Hence catabolic state cannot be resolved by glucose provision
- Prevention- Preoperative carbohydrate drinks
  Minimal access surgery
  Early mobilisation
  Avoid prolonged fasting
  Prompt early nutrition after surgery

Nutritional assessment

00:09:51

- ABCD
- Anthropometry
- BMI= WT IN KG/ (HT IN M)^2
- Quick screening -- BMI (indirect assessments and not reliable in critically ill)
- BMI <18.5 and unintentional wt loss >10% in 6 months
- BMI <20 and unintentional wt loss >5% in 3 months
- Require pre op nutrition support
- Mid Upper Muscle Circumference = Mid Upper Arm Circumference (cm) - 3.14 x TSF (cm)
- Biochemistry – Albumin-not reliable in all pts
- Hemoglobin gives us the status of anemia and glucose control(hbA1c)
- Clinical Evaluation - Symptoms, Past history and comorbidities
- Dietary Assessment Methods
- Caloric intake assessment
- 25-35 Kcal/kg LBM per day x Stress and Activity factor



Anthropometry indices-not reliable

- Body Weight (takes the wet weight)
- Ideal Body weight for Height-better

- Lean Body Mass-best way-prognosticator of post op outcome
- Body Mass Index
- Skin Fold thickness

Body composition analysis

1. DEXA scan- Dual energy x ray absorptiometry
   - Lean body mass
   - Non Bone tissue
   - Gold standard- not reproducible
2. Bedside USG- rectus femoris at the midpoint of femur
3. CT-assess the psoas muscle at the level of L3 vertebra

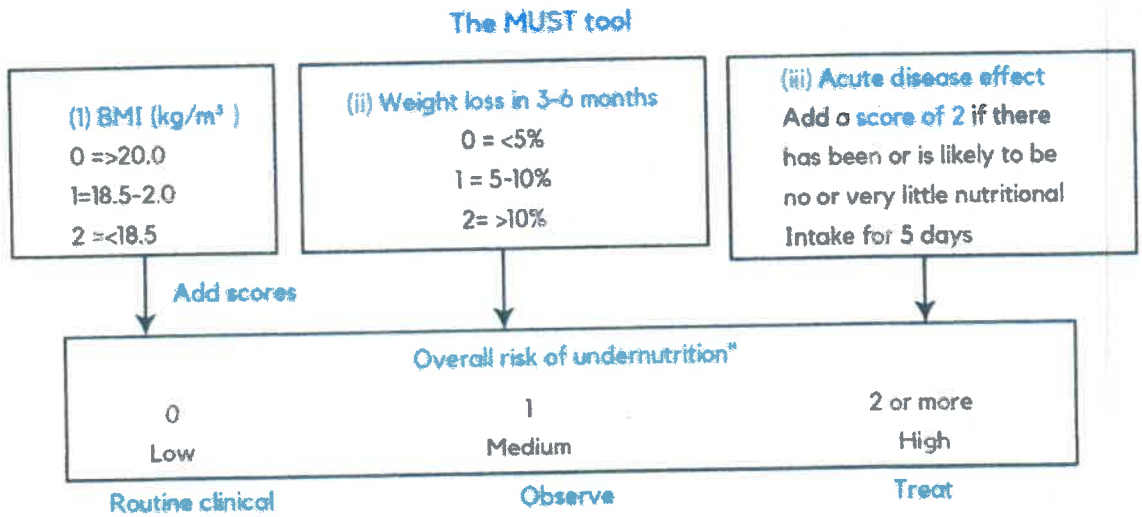
} Check the quality and quantity of the mass

Biochemical analysis

1. **Albumin**

Association between preoperative serum albumin and surgical outcome.		
SERUM ALBUMIN (g/dL)	**30-DAY MORTALITY RATE (%)**	**30-DAY MORBIDITY RATE (%)**
>4.5	≤1	≤10
3.5	5	25
3	9	35
2.5	15	45
<2.1	≈30	65

   - Not a very good indicator
2. Pre albumin
3. Transferrin
   - Varies with body measures
   - MUST TOOL-malnutrition universal screening tool



Repeat screening
Hospital - every week
Care homes - every month
Community- every year for special groups, e.g. those >75 years

Hospital - document dietary and fluid intake for 3 days
Care homes (as for hospital)
Community-repeat screening, e.g. from <1 month to >6 months (with dietary advice if necessary)

Hospital - refer to dietician or implement local policies. Generally food first followed by food fortification and supplements
Care homes (as for hospital)
Community (as for hospital)

- If height, weight or weight loss cannot be established, use documented or recalled values (if considered reliable). When measured or recalled height cannot be obtained, use knee height as a surrogate measure.

If neither can be calculated, obtain an overall impression of malnutrition risk (low, medium, high) using the following:

(i) Clinical impression (very thin, thin, average, overweight);

(iia) Clothes and/or jewellery have become loose fitting;

(iib) History of decreased food intake, loss of appetite or dysphagia up to 3–6 months;

(iic) Disease (underlying cause) and psychosocial/physical disabilities likely to cause weight loss.

† Involves treatment of underlying condition, and help with food choice and eating when necessary (also applies to other categories).

Nutritional Risk Screen (NRS-2002) tool			
	IMPAIRED NUTRITIONAL STATUS		SEVERITY OF DISEASE (=INCREASE IN REQUIREMENTS)
Absent Score 0	Normal nutritional status	Absent Score 0	Normal nutritional requirements
Mild Score 1	Weight loss >5% in 3 months or food intake below 50%-75% of normal requirement in preceding week	Mild Score 1	Hip fracture chronic patients, in particular with acute complications: cirrhosis, COPD.* Chronic hemodialysis, diabetes, oncology
Moderate Score 2	Weight loss <5% in 2 months or BMI 18.5-20.5 + impaired general condition or food intake 25%-60% of normal requirement in preceding week	Moderate Score 2	Major abdominal surgery*
			Stroke
Severe Score 3	Weight loss >5% in 1 month (>15% in 3 months) or BMI <18.5 + impaired general condition or food intake 0%-25% of normal requirement in preceding week	Severe Score 3	Severe pneumonia, hematologic malignancy
			Head injury*
			Bone marrow transplantation**
Score: Age	+ if 70 years: add 1 to total score above	Score: = Age-adjusted total score	Intensive care patients (APACHE >10). = Total score

Score 23: the patient is nutritionally at-risk and a nutritional care plan is initiated.

Score <3: weekly rescreening of the patient. If, for example, the patient is scheduled for a major operation, a preventive nutritional care plan is considered to avoid the associated risk status.

NRS-2002 is based on an interpretation of available randomized clinical trials, Indicates that a trial directly supports the categorization of patients with that diagnosis. Diagnoses shown in italics are based on the prototypes given below.

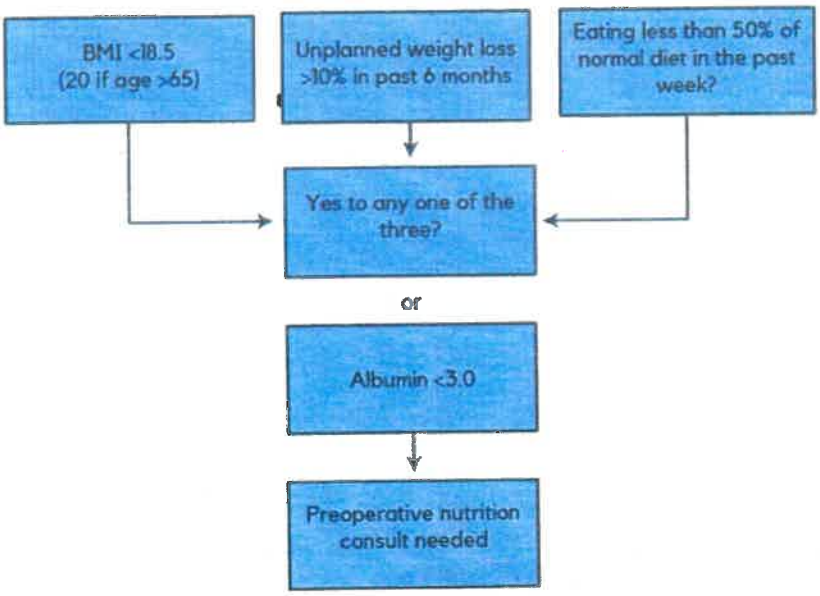
Nutritional risk is defined by the present nutritional status and risk of impairment of present status due to increased requirements caused by stress metabolism of the clinical condition.

A nutritional care plan is indicated in all patients who are:

(1) severely undernourished (score = 3), (2) severely ill (score 3), (3) moderately undernourished + mildly ill (score 2+1), or (4) mildly undernourished + moderately ill (score 1 + 2).

Prototypes for severity of disease. Score = 1: a patient with chronic disease admitted to hospital due to complications. The patient is weak but out of bed regularly. Protein requirement is increased but can be covered by oral diet or supplements in most cases. Score 2: a patient confined to bed due to illness e.g., following major abdominal surgery). Protein requirement is substantially increased but can be covered, although artificial feeding is required in many cases. Score = 3: a patient in intensive care with assisted ventilation, etc. Protein requirement is increased and cannot be covered even by artificial feeding. Protein breakdown and nitrogen loss can be significantly attenuated.

- Nutritional risk screen (NRS-2002) tool
- NRS 3 or > nutritional care plan initiated
- Perioperative nutrition screen tool (PONS)



The Perioperative Nutrition Screen (PONS) is a simple tool to

The Nutrition Assessment in Critically Ill (NUTRIC) score.		
VARIABLE	CRITERIA	POINTS
Age	<50 years	0
	50 to <70 years	1
	≥75 years	2
APACHE II	<15 points	0
	15 to <20 points	1
	20 to 28 points	2
	≥28 points	3
SOFA	<6 points	0
	6 to <10 points	1
	≥10 points	2
Number of comorbidities	0 to 1	0
	22	1
Days from hospital admission to ICU admit	0 to <1	0
	21	1
Total	0 to 4	Low malnutrition risk
	5 to 9	High malnutrition risk, need nutritional plan

Harris benedict energy equation

- Men-
  - BMR = (10 x weight in kg) + (6.25 x height in cm).-
  - (5 x age in years) + 5
- Women-
  - BMR = (10 x weight in kg) + (6.25 x height in cm) -
  - (5 x age in years) – 161

REE

- Men-
  - 66.5 + (13.75 x Weight (kg))
  - + (5.003 x Height (cm)) - (6.755 x Age (in years))
- Women-
  - 655. 1 + (9.563 x Weight kg))
  - + (1.85 x Height (cm)) - (4.676 x Age (years))

Harris-Benedict energy expenditure multipliers	
Scenario	Energy expenditure multiplier
Resting (AF)	1.1
Confined to bed (AF)	1.2
Out of bed (AF)	1.3
Minor operation (IF)	1.2
Skeletal trauma (IF)	1.35
Cancer cachexia (IF)	1.3-1.5
Major sepsis (IF)	1.6
Severe thermal injury (IF)	2.1
Febrile (IF)	1+0.09 per 0.5° C > 38.5

- Indirect calorimetry-gold std only with the help of CPET-cardiopulmonary exercise test

Modified Weir calculation

- REE (kcal/d) = (Vo2 x 3.94) + (Vco2 x 1.1)} x 1.44
- Also assess adequacy of feeding by calculating respiratory quotient
- Normal VCo2/Vo2 0.7 to 1.0
- <0.7 - Underfeeding
- >1.0 - Overfeeding

Pre-op Nutrition

00:27:39

- Where Severe nutrition risk expected (ATLEAST ONE)
  1. Severe undernutrition or chronic disease
  2. BMI < 10.5
  3. Weight 20% less than Ideal body weight
  4. Involuntary weight loss > 10%-15% in 6 months or >5% in 1 month
  5. Expected blood loss >500ml in surgery
  6. Severe catabolic state (Trauma, burns, sepsis)
  7. Albumin <3, Transferrin < 200mg/dl in absence of renal/hepatic dysfunction or inflammatory state
  8. Failure to thrive
  9. Anticipate patient unable to meet caloric needs in 7 to 10 days perioperatively
- At least 7 days of goal directed EN/PN

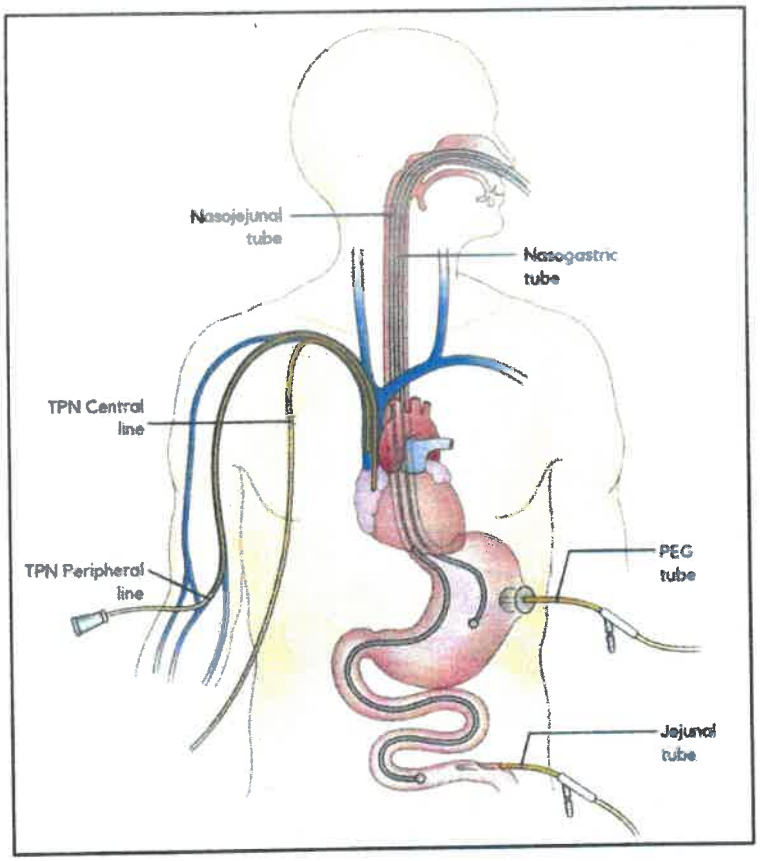
ENTERAL NUTRITION

Advantages-

- Physiologic
- Preserve gut integrity, gut immunity,
- blood flow to intestine, Provide butyrate to mucosal cells to grow
- Release enzymes and endogenous hormones,
- Preserve intestinal microbia
- Low cost
- Less complications compared to TPN

Cannot Use in

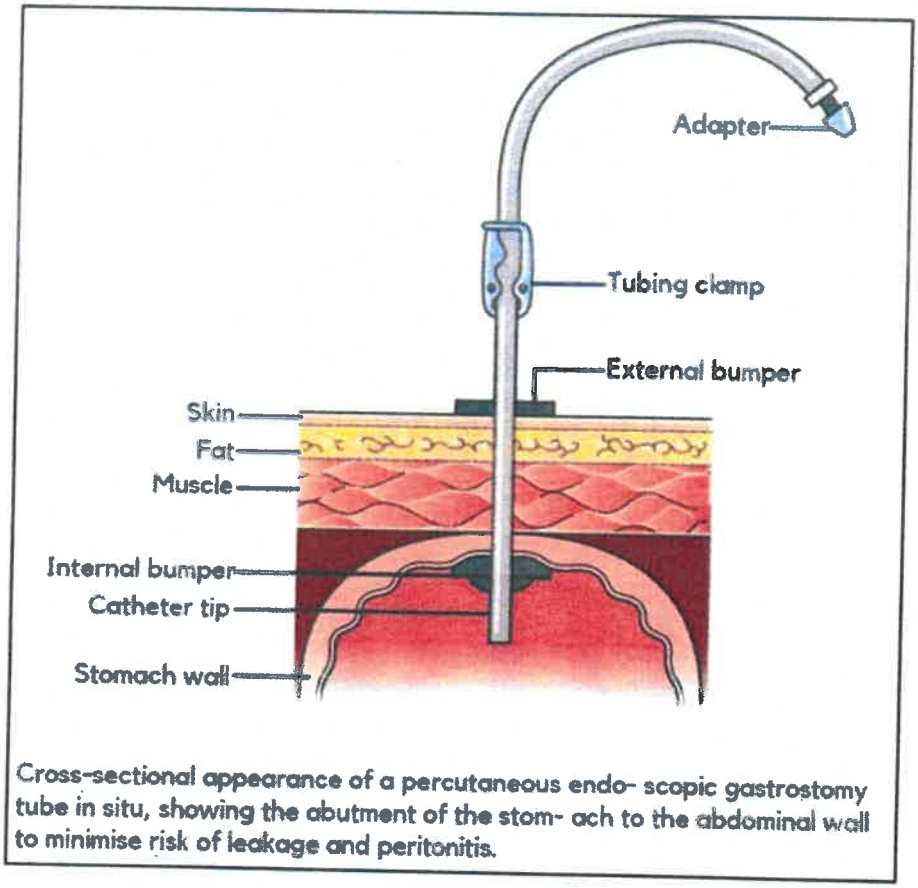
- Ileus, obstruction, ischemia, bleeding, fistula
- Hemodynamically unstable, bowel discontinuity, Peritonitis
- SBS, Intractable vomiting, diarrhoea
- Severe malabsorption



- *Pre pyloric feeds*
  - *Stomach*
  - *Bolus is possible*
  - *Polymeric feeds*
  - *Digestion is possible*
  - *↑ Risk of aspiration*
- *Post pyloric feeds*
  - *Small bowel*
  - *No bolus (continuous infusion)*
  - *Semi elemental feeds*
- *Short term-nasogastric/nasojejunal feeds*
- *Long term-PEG/FJ*

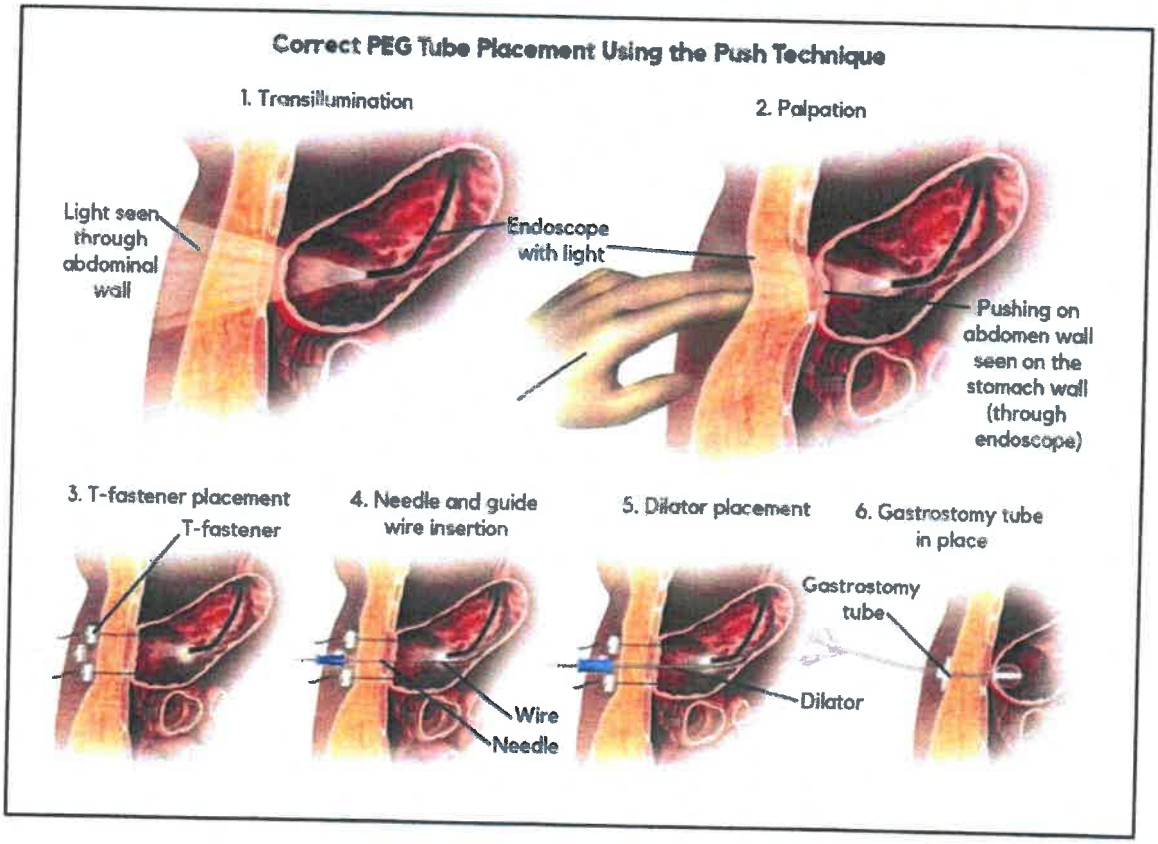
Routes for tube feeding.

ROUTE	SUITABILITY	INSERTION METHOD, CONFIRMATION	ADVANTAGES	DISADVANTAGES
Nasogastric	Short term functional GI tract	Blind at bedside, fluoroscopy guided	Easy to insert, replace; can monitor gastric pH and residual volume; capable of bolus feeding	Misplacement complications, sinusitis, epistaxis, nasal necrosis, esophageal strictures, erosive esophagitis
Nasoduodenal, nasojejunal	Short term functional GI tract but poor gastric emptying, reflux, aspiration risk; begin feed only when volume resuscitated and hemodynamically stable	Blind at bedside; fluoroscopy guided, endoscopy guided	Reduced aspiration risk, some tubes enable decompression of stomach while feeding into jejunum	Easily clogged or displaced, aspiration risk, misplacement complications, displacement and reflux into stomach, sinusitis, epistaxis, nasal necrosis, requires continuous infusion, cannot check gastric residuals except with specialized gastric port
Gastrostomy	Long term good gastric emptying; avoid if significant reflux or aspiration problem	Surgical, percutaneous, endoscopic, radiologic	Bolus feeding: large-bore tube less likely to block	Procedure risks include bleeding, perforation, aspiration risk, dislodgment with peritoneal contamination, wound site infection, granulation
Jejunostomy	Long term functional GI tract but poor gastric emptying, reflux, aspiration risk, gastroparesis, gastric dysfunction	Surgical, percutaneous, endoscopic, radiologic	Theoretical reduced aspiration risk	Bleeding, infection, perforation, migration, aspiration, dislodgment and leakage into peritoneal cavity, occlusion, pneumatosis, intestinal ischemia or infarction, bowel obstruction, difficult to replace; cannot check residuals; requires continuous infusion

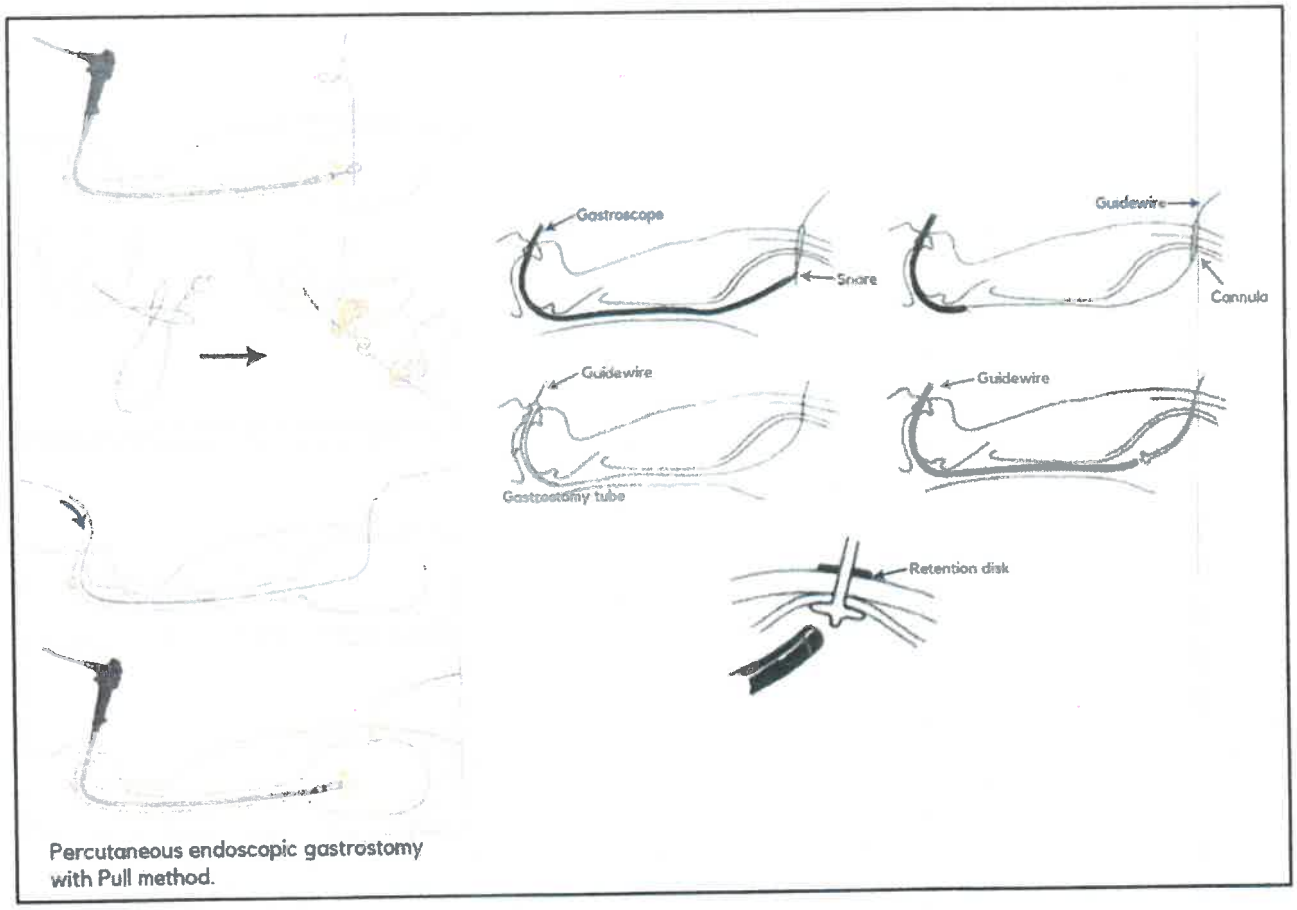


Cross-sectional appearance of a percutaneous endo- scopic gastrostomy tube in situ, showing the abutment of the stom- ach to the abdominal wall to minimise risk of leakage and peritonitis.

1. *RUSSELLS TECHNIQUE*



2. PER ORAL PULL technique (Gauderer Ponsky technique)



Percutaneous endoscopic gastrostomy with Pull method.

3. Per oral push technique-peg tube pushed over guidewire through orally

Complications of enteral feeding

00:38:35

- Tube related
  - Malposition
  - Displacement
  - Blockage
  - Breakage/leakage
  - Local complications (e.g. erosion of skin/mucosa)
- Gastrointestinal
  - Diarrhoea
  - Bloating, nausea, vomiting
  - Abdominal cramps
  - Aspiration
  - Constipation
  - Metabolic/biochemical
    - Electrolyte disorders, including refeeding syndrome
    - Vitamin, mineral, trace element deficiencies
    - Drug interactions
  - Start at 20ml/hr---up to 75ml/hr
  - Try continuous feeds for FJ

PARENTERAL NUTRITION

00:40:07

- Providing direct calories through IV
  1. TOTAL/CENTRAL
     - Through central vein
     - Hyperosmolar
     - More calories(1200kcals)
     - qty-1.4 L
  2. PERIPHERAL
     - Through peripheral veins
     - Less osmolar
     - Less calories (750-800 kcals)
     - qty - <1L

Forumulations:

- Carbs-45-60%
- Proteins-20%
- Lipids-15-20%
- Lipid emulsion with glucose, essential and non-essential amino acids, electrolytes, trace elements and vitamins Phosphate daily added
- Folic acid weekly
- Vit B12 if planned long term

PICC lines

- Most commonly used - basilic vein, cephalic, medial antecubital vein
- The parenteral nutrition bag should be covered at all times, including during infusion, with an opaque protective bag to prevent the vitamins from degradation. If the parenteral nutrition infusion is disconnected from the line for any reason during administration the bag will need to be discarded

Complications of parenteral feeding

- Insertion complications
  - Pneumothorax
  - Misplacement
- Line complications
  - Sepsis
  - Thrombosis
- Metabolic complications
  - Electrolyte disorders, including refeeding syndrome
  - Blood sugar derangement
  - Liver dysfunction-25%
  - Metabolic bone disease
  - Vitamin deficiencies
- Pneumothorax in 0.5% to 1%-subclavian line
- Line Sepsis in 15%-stop using; take cultures from line& peripheral veins
- Line blockage and thrombosis

Refeeding syndrome 00:44:24

- Accentuation of nutrient deficiency secondary to immediate nutrition in malnourished patients
- Risk factors- Prolonged fasting
  Anorexia
  Alcohol dependence
  Severe malnutrition
- Fat metabolism→ carbohydrate metabolism
- Requires ATP
- All phosphates are required
- Major intra to extracellular electrolyte shifts happen
  - Hypophosphatemia
  - Hypoglycemia
  - Hypomagnesemia
  - Hypokalemia
  - Hypocalcemia

Cardiac failure

- Arrhythmias
- Respiratory failure
- Renal liver dysfunction
- Seizures Coma
- Tetany and Death
- Start at rate of 10kcal/kg/day→ escalate every 4 days

Refeeding syndrome
Patient is considered to be at risk of developing refeeding syndrome with EITHER One or more of the following: • BMI<16 kg/m² • Unintentional weight loss >15% within the last 3-6 months • Little or no nutritional intake for more than 10 days • Low potassium, phosphate or magnesium levels prior to feeding OR Two of more of the following: • BMI <18.5 kg/m² • Unintentional weight loss >10% within the last 3-6 months • Little or no nutritional intake for more than 5 days • History of alcohol abuse or on medication, including insulin, chemotherapy, antacids or diuretics

Q&A discussion

Q. What is the major differentiating feature between metabolic response to starvation and Injury or Surgery?

a. Lipid oxidation
b. Adaptive ketogenesis
c. Protein breakdown
d. Glucagon levels

Q. Which of the following is not a metabolic response to Injury?

a. Increased energy expenditure
b. Preferential oxidation of lipids
c. Adaptive ketogenesis
d. Decreased peripheral glucose utilisation

Q. What is the most reliable indicator of nutritional status?

a. Albumin
b. BMI
c. Skin fold thickness
d. Psoas muscle quantity and quality at L3 level on CT

Q. Among the tools for nutritional risk assessment, which of the following tool takes current severity of disease status into account?

a. MUST tool
b. Mini Nutritional assessment tool
c. Preoperative Nutrition screen tool
d. NRS 2002

Q. Which of the following nutritional risk assessment tool uses Albumin as a parameter?

a. NRS 2002 tool
b. Preoperative Nutritional screening tool
c. Malnutritional Universal Screening tool
d. NUTRIC Score

Q. Gold standard test to calculate energy requirements?

a. Indirect calorimetry
b. Nitrogen balance
c. Harris benedict equations
d. Korth equation

Q. Which is true regarding Harris benedict equation?

a. Age and weight only
b. Age, height and weight
c. Age, sex, height and weight
d. Weight, height, age and different for men and women

Q. What are the advantages of Enteral Nutrition EXCEPT?

a. Gut integrity
b. Gut Immunity
c. Use in Malabsorption syndromes
d. Prevents infection

Q. What is not a feature of Refeeding syndrome?

a. Hyperphosphatemia
b. Hypomagnesemia
c. Hypokalemia
d. Hypocalcemia

Q. 55 male Patient diagnosed with carcinoma hypopharynx and absolute dysphagia. Patient is receiving definitive chemoradiation for locally advanced carcinoma hypopharynx. What is the ideal and best route to provide prolonged nutritional support?

a. Percutaneous Gastrostomy
b. Feeding jejunostomy
c. Nasogastric feeding
d. Total parenteral nutrition

Q. Which of the following statement is FALSE?

a. Main advantage of TPN is caloric needs are met rapidly
b. Early TPN within 48 hours has decreased infectious complications and mortality benefit
c. EN has decreased infectious complications and ICU stay compared to TPN
d. EN has no mortality benefit over TPN in critically ill patients

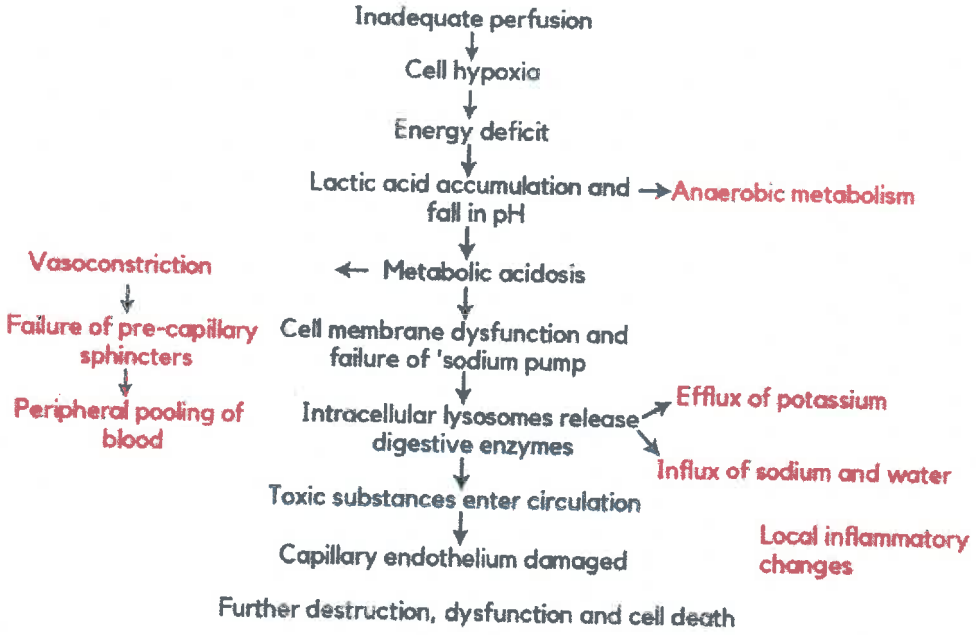
SHOCK

Definition, Pathophysiology, Classification

00:01:05

- Shock is defined as-" A systemic state of low tissue perfusion that is inadequate for normal cellular respiration"
- Most common cause of death in surgical patients

Cellular level



Systemic level