MY GUT FEELING WHEN GUT IS FAILING

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GASTRO-INTESTINAL FAILURE (GIF) IN ICU

- Definition of GIT failure
  - **Food intolerance** - Vomiting (and/or nausea) - Increased volume of NG aspirate - Abdominal pain - Diarrhoea(related to EN, infection, medication)
  - **Gastro-intestinal haemorrhage** (hematemesis or hematochezia/malena)
  - **Ileus** - Abdominal distension – Constipation
  - **Liver/Gallbladder failure/Pancreatitis**

- Incidence Intolerance of feeding: Up to **60%** ICU admissions
- Regulation of gut function

  GI Motility = complex function

- Regulated **by Autonomic nervous system**
- Enteric nervous system Modulated by regulatory GI peptides , Neurotransmitters & Hormones
- **Disordered gut motility leads to: Altered gut-mucosa contact time → bacterial overgrowth**

AND **drug absorption also Can be AILTERED**

Regulation of GI function

Enteric (Intrinsic) nervous system:

interconnected

innervate smooth muscle of bowel

Secretory neurons regulate endocrine and exocrine secretion in mucosa AND sensory neurons that respond to stretch, tonicity, glucose and amino-acids can be seen as a 3d division of the autonomic nervous system

Substances secreted BY SCERETORY NEURONS:

NO, acetylcholine, serotonin, GABA, large number of polypeptides (synaptic transmitters , hormones and paracrine function)

NO is a major mediator of smooth muscle relaxation IN GIT

Regulation of GI function

Dual innervation from the autonomic nervous system

- ☐ Parasympathetic pathway increases activity
- ☐ Sympathetic system = relaxes bowel movement = cause sphincters to contract

- Blood vessels of the bowel

Extrinsic => sympathetic innervation => vasoconstriction

Intrinsic from the enteric nervous system => VIP and serotonin causes vasodilatation during digestion

- Peristalsis: = Reflex response to stretching
  (integrated activity of enteric nervous system)

Can be increased or decreased by autonomic input

Relaxation is mediated by NO

Dysmotility

- Can involve the entire GIT or can be limited to specific areas acting as a focus of obstruction
- Cause **of dysmotility in critically ill multifactorial**
  - reduced frequency and amplitude of antral contractions
  - Abnormal fundus motor activity in response to nutrient stimulus
  - failure to redistribute gastric content to rest of bowel

- Diagnosis of gut failure

- Clinical evaluation =Bowel sounds:

  ? Clinical significance & lack of evidence.

  Doesn't correlate with effective peristalses IN Abdominal distension/ constipation/ Diarrhoea/ Abdominal pain/Vomiting

- Gastric residual volume : Which volume indicates Gut failure **million dollar question!**

ASPEN and SCCM: GRV < 500ml for 4 HOURS
EN feeding should not be stopped in the absence of other signs of intolerance or GIT failure.

Poulard et al, 2010: Challenged assumption -- GRV is an accurate assessment of EN tolerance.

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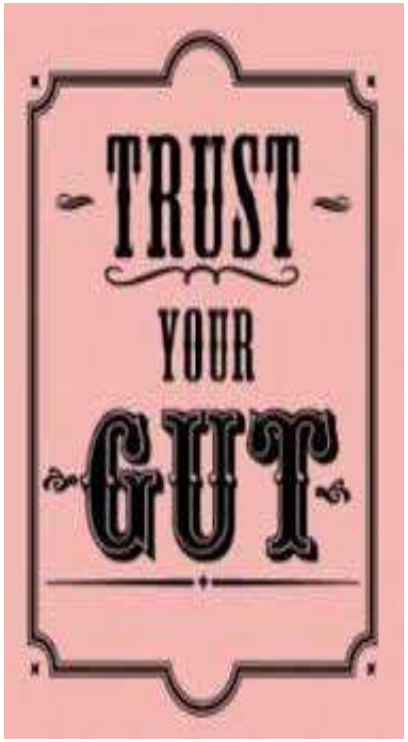
Paracetamol absorption test

Drug is absorbed from small bowel.

Good correlation between stomach emptying time and peak plasma concentrations.

Relevance of GI failure in critically ill

- GIF increased bacterial translocation
- Gut failure is a motor of MODS (multiple organ dysfunction syndrome)
- GIF is a common problem in ICU patients, yet it is **not given the same consideration** as other organ systems received when scoring and predicting outcome in ICU.



GIF to be a **relevant independent clinical predictor of mortality in ICU.**

It significantly **associated with prolonged mechanical ventilation and ICU stay.**

GIF is a **syndrome** with a variable onset during ICU treatment (80% were identified after 1 week stay in ICU and 20% developed GIF later).

Gut failure score

PMC full text: Crit Care. 2008; 12(6): 436.
Published online 2008 Dec 4. doi: 10.1186/cc7120

Table 1

Lausanne Intestinal Failure Estimation (LIFE) based on the SOFA model including symptoms and continuous signs compared with the variables of Reintam and colleagues

Variable	Score 0	Score 1	Score 2	Score 3	Score 4
Reintam and colleagues [1]	Normal function	EN <50% of needs of no EN at day 3	EN intolerance (residues) or IAH	EN intolerance and IAH	Abdominal compartment syndrome
IAH (mmHg)	<12	12 to 15	15 to 20	20 to 25	>25
Lactate with pH <7.25 (mmol/l)	<2	2.0 to 3.0	3.0 to 4.0	4.0 to 5.0	>5.0
Gastric residue every 6 hours (ml)	<100	200 to 300	>300	>400 or vomiting regurgitation	-
Progression of feed by day of EN	Normal		<60% of needs by day 3		<60% of target by day 4
Constipation (stools over time)	One per 1 to 3 days	Zero in 4 days	Zero in 5 days, bloating	Abdominal distension	Ogilvie syndrome
Diarrhea[a] (number per day)	-	-	4 to 6	6 to 10	>10
Bowel sounds	Normal	Silence	-	Tympanic	-

The different items should be considered separately as relevant for the patient's condition, the highest (that is, worst) value being the gut score. EN, enteral nutrition; IAH, intra-abdominal pressure; SOFA = Sequential Organ Failure Assessment. aLiquid stool occurring at least four times per day.

Crit Care

Clinical trial …

Rientam et al 2008

- GIT failure is associated with a sharp increase of ICU mortality GIF
- **independent risk factor for death**
- Use of **catecholamines** to treat CVS failure is independent risk factor for GIF
- Food intolerance alone is not an independent predictor of outcome

Clinical trial..

Cheatham et al, 2007

- IAH (INTRA ADBOMINAL HYPERTENSION & and ACS (ACUTE COMPARTMENT SYNDROME) has a strong impact on mortality.
- **APP(abdominal perfusion pressure)* > 50mmHg better predictor of survival** than arterial pH, lactate, base deficit, hourly urine output.

*APP=MAP-IAP

MAP=MEAN ARTERIAL PRESSURE

IAP = INTRA ABDOMINAL PRESSURE

Contributing Factors for GIF

Inflammation (SIRS, aseptic pancreatitis)

Surgery (handling of bowel)

Medication - Opioid - Anticholinergic medication (Atropin) and Vasopressors

Electrolyte imbalances

Acidosis

- Hyperglycaemia
- Sepsis
- Increased intracranial pressure(cranial outflow of vagal nerve/hypothalamus and medulla influences the sympathetic activity)
- Mechanical ventilation (respiratory failure)
- Altered hemodynamics

Alterations of GI tract in critically ill

- Delayed gastric emptying/reduction in intestinal transit time
- Absence biliary and pancreatic secretions / abnormal glucose metabolism
- Mucosal ischemia/bowel oedema/pH and electrolyte abnormality/Excessive sympathetic tone/Pro-inflammatory mediators
- Altered carrier and nutrient transport proteins
- Villus atrophy/reduction mucosal surface area

Loss of barrier function(MALT and GALT)

(MALT=Mucosal associated lymphoid tissue &
GALT=Gastric associated lymphoid tissue)

- Splanchnic blood flow GI tract uses 30% of total O2 consumption at rest so tips of villi at highest risk Ischemia
- low PO2 → vasodilatation (metabolites)→ increase blood flow →oedema of bowel wall →dysmotility and altered absorption→ **bacterial overgrowth and toxin production / translocation of bacteria**

Mechanisms to maintain and improve gut function in critical illness

- Maintain visceral perfusion = Early resuscitation and maintenance of APP > 50mmHg
- Glycaemic control (STUDIES - Rayner et al, 2001 & Adibi et al, 2003)
- Correct acidosis and electrolyte abnormalities

- Adherence to **early ENTERAL feeding**
- Placement of feeding tubes
- Minimize medication that alter bowel function •
- Optimizing metabolic parameters improves bowel motility (removal of excess water, correction of acidosis & electrolytes imbalance)
- Add motility agents: Erythromycin/Rifaximine/Metoclopramide

CLINICAL GUIDELINES
FOR
ENTERAL NUTRITION

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Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.)

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nutrition; critical care; intensive care unit; enteral; parenteral; evidence-based medicine; Grading of Recommendations, Assessment, Development, and Evaluation criteria; guidelines

Entral nutrition(EN) : Guidelines

Initiation of Feeding

- **Ventilated patients** should receive an orogastric tube (OGT), nasogastric tube (NGT).
- Enteral feeding should be **initiated within 12-24 hours of admission to ICU**, unless the patient is not hemodynamically stable, adequately resuscitated, or the gastrointestinal (GI) tract is believed to be non-functioning.

Why early enteral feeding

Early means initiation of enteral feeding **within 12-24 hours of admission to ICU**

- Safer, better patient outcome, shorter ICU stay
- Prevents villi atrophy
- food absorption(food supply to mucosal cells itself)
- Helps maintain GALT (=Gastric associated lymphoid tissue)
- Maintains gut mucosal barrier → Less bacterial translocation due to improved MALT (=Mucosal associated lymphoid tissue) function SO **reduction of systemic sepsis incidence**

Why early enteral feeding (continue)

- ❑Better glucose control
- ❑Delivery of food not into systemic circulation, but into portal system to liver.
- ❑Protects against LBM(LEAN BODY MASS) loss
- ❑Helps maintain visceral blood flow
- ❑IMPROVES Visceral protein synthesis/glutathione production
- ❑Delivers calories and nitrogen

Initiation of Feeding

Patients receiving enteral feedings should be placed in the **semi-recumbent position with 30- 45** degree unless otherwise indicated

Patients receiving therapeutic hypothermia for 24 hours can begin **enteral nutrition (EN) during the rewarming process.**

Various methods for EN (ENTERAL NUTRITION)

- **In continuous feeding**, an hourly rate of EN is administered using a feeding pump over 24 h(LIKE INFUSION PUMP)

- **In intermittent feeding**, EN is administered over 20-60 min every 4-6 h with or without a feeding pump.

- **In bolus feeding**, EN is administered via a syringe or gravity drip over a 4-10-min period EVERY 2/3 HOURLY.

Intermittent and Bolus Methods of Feeding in Critical Care

Chapter · January 2014

In book: Diet and Nutrition in Critical Care.Publisher: Springer New York.

Various methods for EN (ENTERAL NUTRITION)

- However, **too little data are available at present to make a strong recommendation for one particular method of enteral feeding over others.**
- In practice, it is generally **considered acceptable for pump-assisted continuous feeding in critically ill patients to be initiated at a rate of 10-20 ml/h** and then gradually increased to the target rate

Various methods for EN (ENTERAL NUTRITION)

- For **medically stable patients, intermittent and bolus feeding methods are preferred** due to practical issues, such as patient mobility, convenience, and cost.
- At present, **no evidence is available regarding the optimum feeding modality** for not only an ordinary clinical setting but **also critical care setting.**

How much we feed ?

Estimated energy intake should be adjusted according to the severity and type of illness. a. Energy requirements may be calculated either

- through simplistic formulas (25-30 kcal/kg/d), published predictive equations, or
- the use of Harris-Benedict Equation

1. Men: 66 + (13.7 x weight (kg)) + (% x height (cm)) – (6.8 x age)
2. Women: 66.5 + (9.6 x weight (kg)) + (1.8 x height (cm)) – (4.7 x age)

Energy calculation as per clinical condition

ii. Based on disease condition

Clinical Condition	Energy (Kcal/Kg/day)
Maintenance	25
Stressed/MICU	25-30
Trauma/General Surgery	30
Trauma/ICU	30-35
Burns	Curreri Formula: 25 *(weight (kg)) + 40 *(%TBSA burned)
Cancer	Inactive, nonambulatory 25-30 Wt gain, nutritional repletion 30-35 Hypermetabolic, stressed 35 *Use Actual BW unless BMI >29.9, then use Ideal
Obesity BMI >29.9	Mifflin St. Jeor Equation: Men: (10 x kg) + (6.25 x cm) – (5 x age) + 5 Women: (10 x kg) + (6.25 x cm) – (5 x age) - 161

Protein requirement

- Estimated protein needs should be adjusted according to the severity and type of illness. Protein provision will be included in total calorie intake in critically ill patients while they are in ICU.

c. Daily protein intake based on disease condition

Clinical Condition	Protein needs (g/Kg IBW/day)
Normal (nonstressed)	0.8
Mild stress	1-1.2
Critical Illness/injury/moderate stress	1-1.5
Acute Renal Failure (undialyzed)	0.8-1
Acute Renal Failure (dialyzed)	1.2-1.4
Peritoneal Dialysis	1.3-1.5
Infection, major surgery, cancer	1.4-1.6
Burn/Sepsis/ multiple trauma/CHI	1.5-2
CRRT/CVVHD	1.7-2.5
Lower protein requirements may be necessary in hepatic encephalopathy	

EN AND RESPIRATORY DISEASES

- **High-lipid, low-carbohydrate** specialty formulas designed to manipulate the respiratory quotient may be utilized in the **SEVERE RESPIRATORY DISEASES WITH CO2 retaining patients** who are difficult to wean from mechanical ventilation, but should not be routinely used.

EN AND RENAL FAILURE

- ICU patients with **acute renal failure or acute kidney injury** should be placed on standard enteral formulations

  If significant electrolyte abnormalities exist or develop, that are not being corrected by usual ICU care and renal replacement therapy, a **special formulation designed for renal failure** (with appropriate electrolyte profile (LIKE LOW POTTASIUM ,LOW PHOSPHATE) may be considered.

EN AND LIVER DISEASE

- **EN is the preferred route** of nutrition therapy in ICU patients with acute and/or chronic liver disease.
- **Branched chain amino acid formulations (BCAA) should be reserved** for the rare encephalopathic patient who is NOT ABLE TO manage with standard treatment (antibiotics and Lactulose)
- **BCAA supplementation*** may enhance detoxification of ammonia in skeletal muscle and may be a possible therapeutic strategy for Hepatic encephalopathy.

* **Effects of oral branched-chain amino acids on hepatic encephalopathy and outcome in patients with liver cirrhosis.** 2013 Oct;28(5):580-8. doi: 10.1177/0884533613496432. Epub 2013 Aug 14.

EN and Acute Pancreatitis

- Patients with **severe acute pancreatitis may be fed enterally by the jejunal route**. Tolerance to EN may be enhanced by **early initiation of EN, displacing the level of infusion more distally BY PUTTING NASO-JEJUNAL (NJ) FEEDING TUBE or changing the EN nearly fat-free elemental formulation.**

EN AND DIABETES

- For patients with a history of diabetes, The American Diabetes Association suggests either a **standard (50% carbohydrate) or a lower carbohydrate content (33-40%) formula** should be used.
- It is appropriate to start with a standard formula with close monitoring of blood glucose, however if glycemic control is difficult to achieve then it is reasonable to **switch to a diabetic or low carbohydrate formula.**

EN TOLERANCE

- EN tolerance should be monitored by multiple markers (pain and/or distention, physical exam, passage of flatus and stool, abdominal radiographs).

Gastric residual Volume (GRV) should be checked 4^{th} hourly.

- 1. If GRV 200-500 ml : continue formula at previous rate AND AGAIN IF GRV=200-500 ml AFTER 4 HOUR then consider adding gastric motility agent

- 2. If GRV >500 mL: **Clinically examine** for signs of intolerance: abdominal distention, fullness, discomfort, or presence of emesis and hold EN x 2 hours.

  Recheck residuals after 2 hours, if **GRV remains >200 mL: Continue to hold EN and**

  check for…patient position for head up, Consider to rule out ileus/obstruction, Consider gastric motility agent, small bowel feeding, changing to volume restricted formula or decreasing goal rate, and all above fails then consider total parenteral nutrition (TPN).

Reasons why TPN resulted in poor outcomes because …

- Systemic immune suppression SO CHANCES OF SEPSIS
- Hyperglycaemic control
- Imbalance of specific nutrients
- Lack of luminal nutrient delivery - Systemic vs portal delivery of nutrients

Conclusion…

- Incidence Intolerance of feeding: Up to **60%** ICU admissions (more in ventilated patient)
- **GIVE the same consideration FOR GI FAILURE** as other organ systems received when scoring and predicting outcome in ICU.
- **GI FAILURE is relevant independent clinical predictor of mortality in ICU.**
- **EARLY enteral feeding** within 12-24 hours of admission to ICU is recommendation. It helps to maintain gut mucosal barrier → Less bacterial translocation SO **reduction of systemic sepsis incidence**


THANK
YOU

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