Nourishing the undernourished: a deep dive into complex clinical case studies from ANZ

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MEDICAL AND HEALTH SCIENCES
SURGICAL AND TRANSLATIONAL RESEARCH CENTRE

Disclosure and Acknowledgements

- Speaker Fee : Baxters HealthCare
- Dietitian Connection

Baxter

dietitianconnection

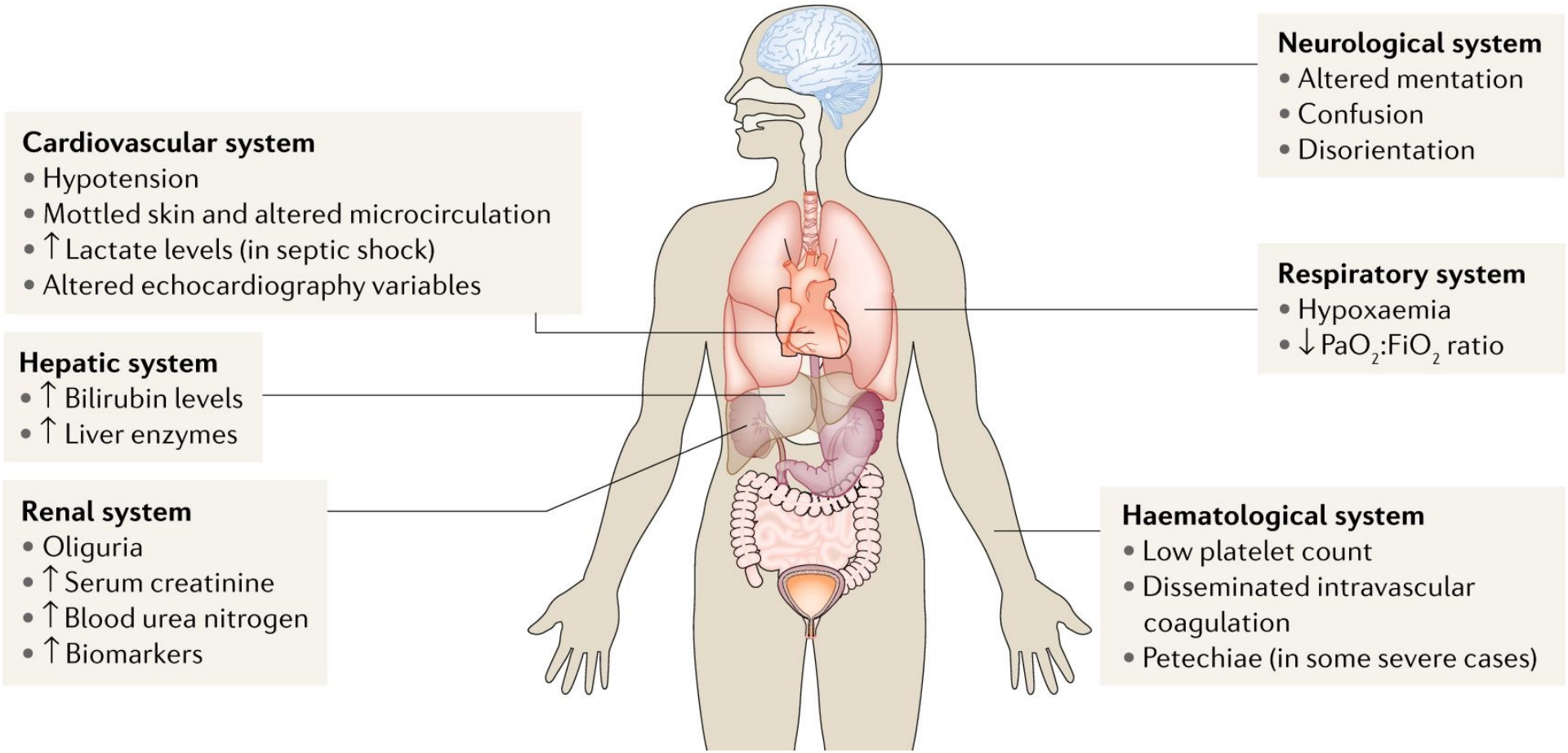
Overview

- Organ Failure In The ICU
- A Life-threatening Case Of Gut Failure
- Gut Dysfunction In Critical Illness : The State Of The Art
- Feeding Enterally – Good, Bad And Ugly!
- Development of a Novel Gut Scoring Tool : An Ambitious Goal
- Conclusion
- A Seat at the Table


8
MAQUET
DCCM BED 10
DCCM/82
AIRWAY BOX

Organ failure
is the leading cause
of mortality in ICU’s

Multiple Organ Failure



Lelubre, C., Vincent, JL. Mechanisms and treatment of organ failure in sepsis. Nat Rev Nephrol 14, 417–427 (2018).

Multiple Organ Failure scores

- APACHE II and III
- SOFA
- Marshall & Meakins
- Denver
- Logistic organ dysfunction score

None include gut failure

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A Life-threatening Case of Gut Failure

- 51-year-old female.
- Postop posterior fossa tumour excision.
- Extensive neurosurgery.
- Emergent extra-ventricular drain for decreased GCS, ⬆ ICP
- Enterally fed to target

Deterioration

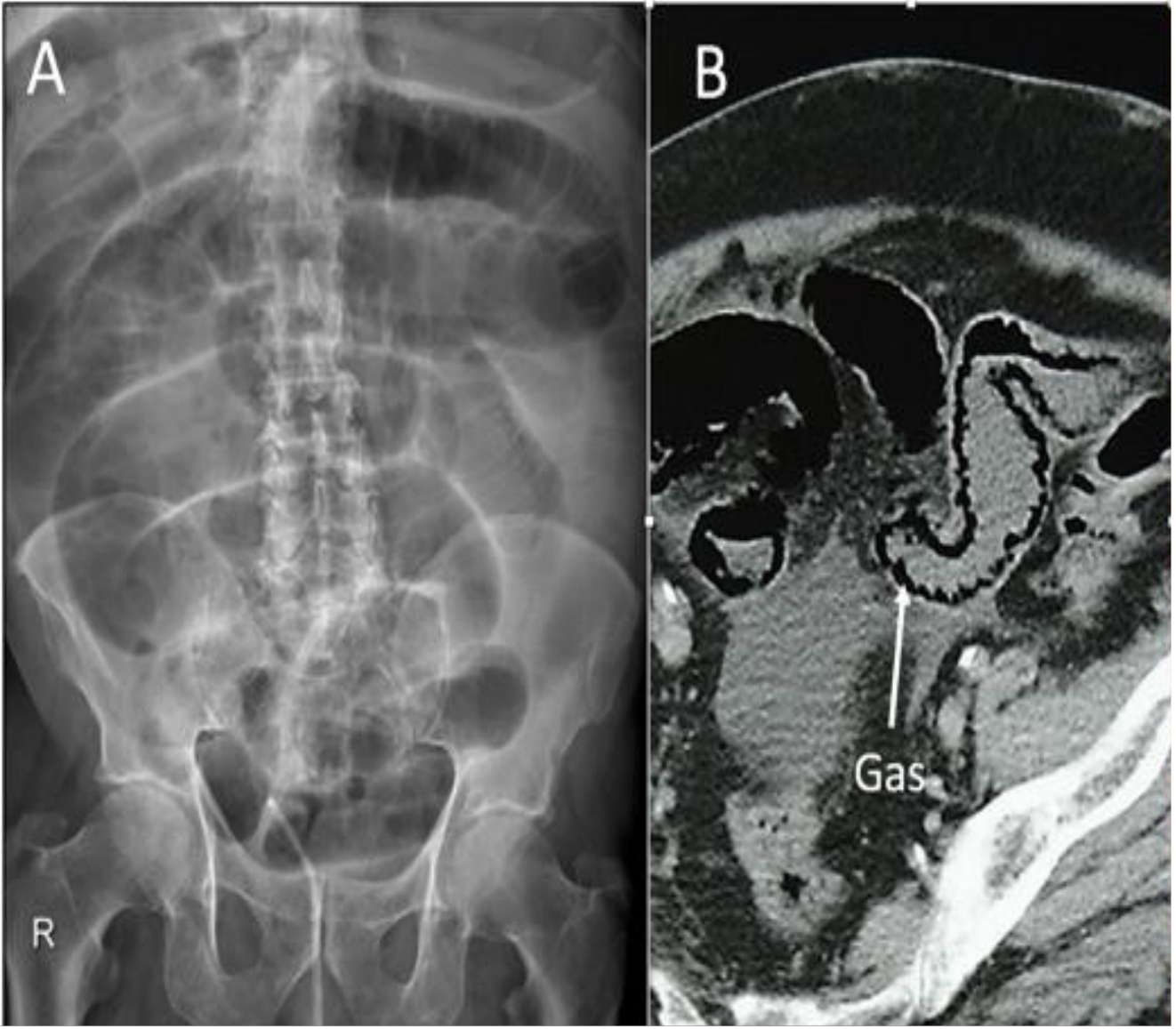
- SIRS response (tachycardia, fever and tachypnoea)
- Positive blood culture
- Hyperglycaemia requiring insulin
- Organ failure
  - Renal
  - Respiratory
- **Abdominal distension / hypertension**
- **Increased gastric residual volumes**

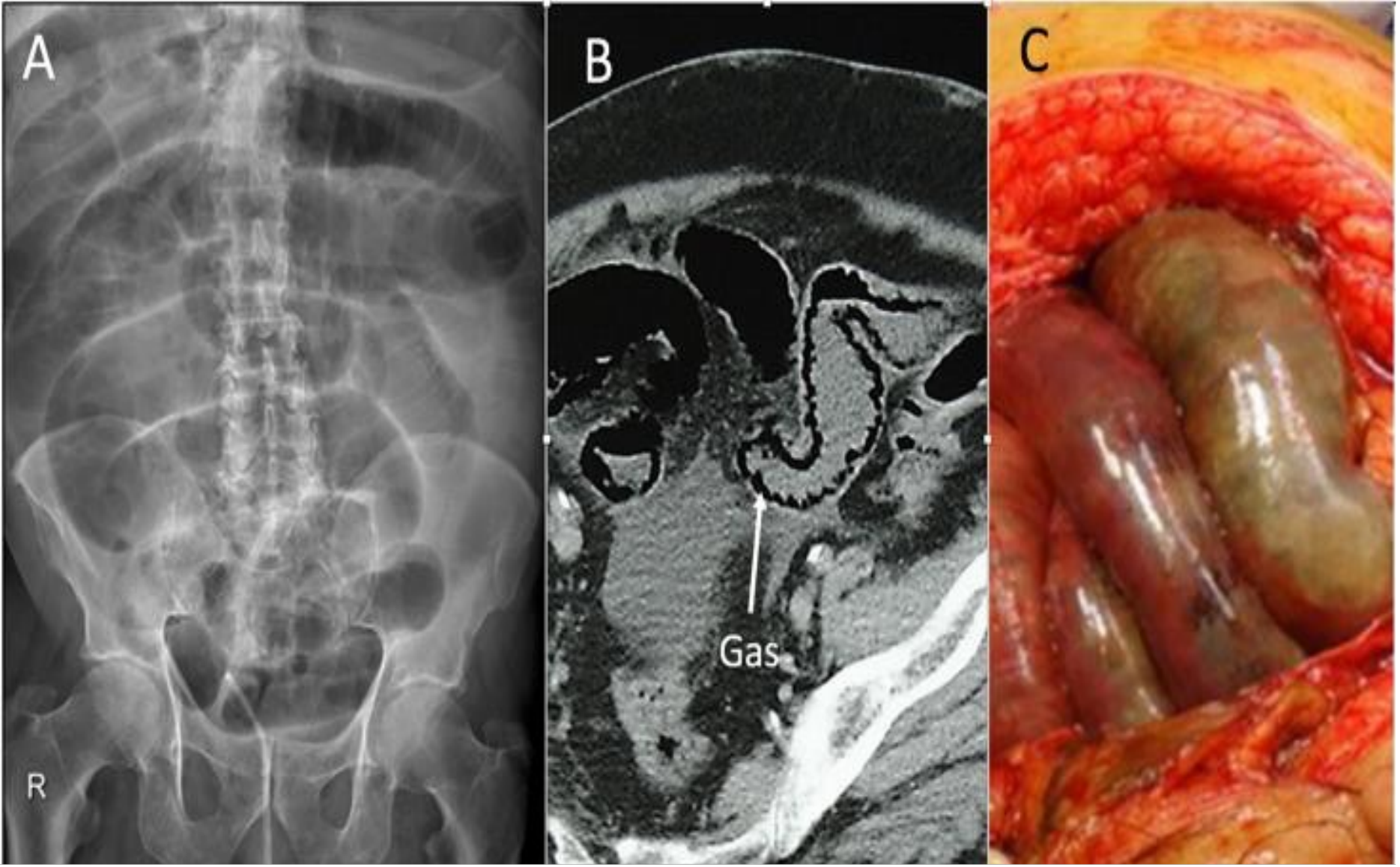
Poll Question 1

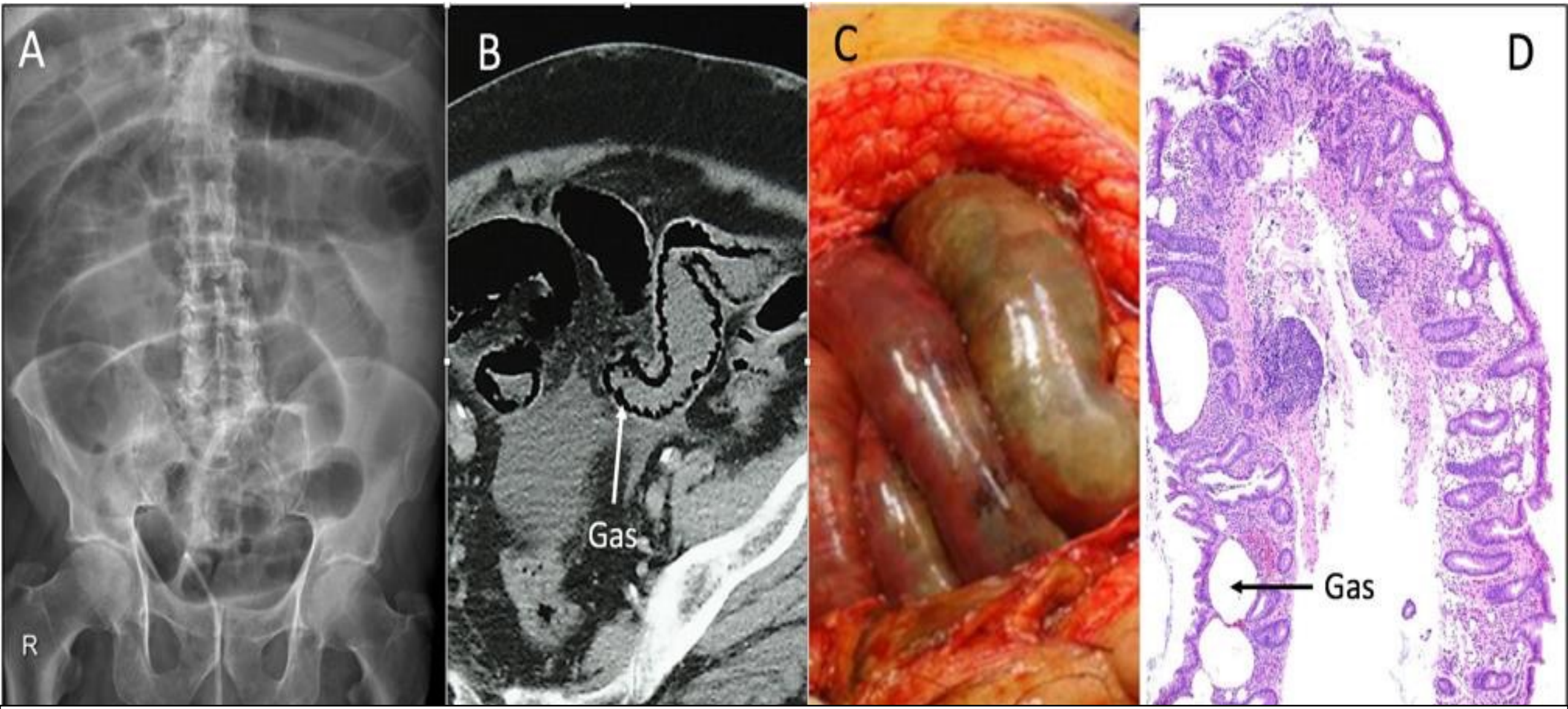
Name 1 typical sign/symptom of gut dysfunction that are least relevant when examining or assessing an ICU patient who is ventilated and sedated.

- Abdominal distension
- Enteral feeding intolerance
- Abdominal pain
- Vomiting


A
R


A
R
B
Gas

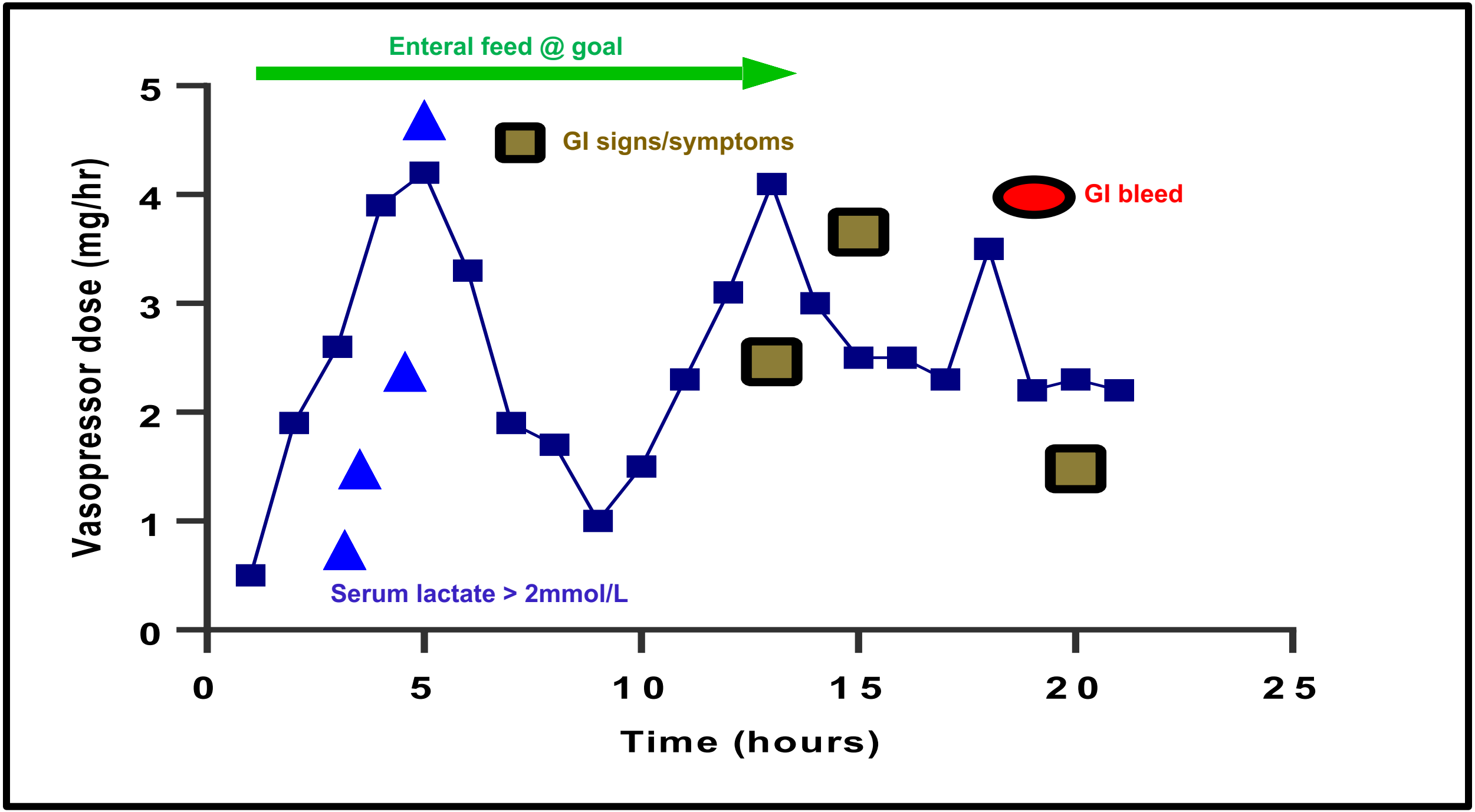

A
R
B
Gas
C



GUT dysfunction and injury is a feature of critical illness

Iatrogenic factors in gut dysfunction

Fluid therapy	hypoperfusion and ischaemia reperfusion injury /oedema
NBM / PN	atrophy and enterocyte loss
Aggressive EN	ischaemia (NOMI)
Inotropes	ischaemia
Narcotics	ileus
Antibiotics	dysbiosis


Enteral feed @ goal
GI signs/symptoms
GI bleed
Serum lactate > 2mmol/L
Vasopressor dose (mg/hr)
0
1
2
3
4
5
0
5
10
15
20
25
Time (hours)

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Spectrum of Gut Dysfunction/Failure

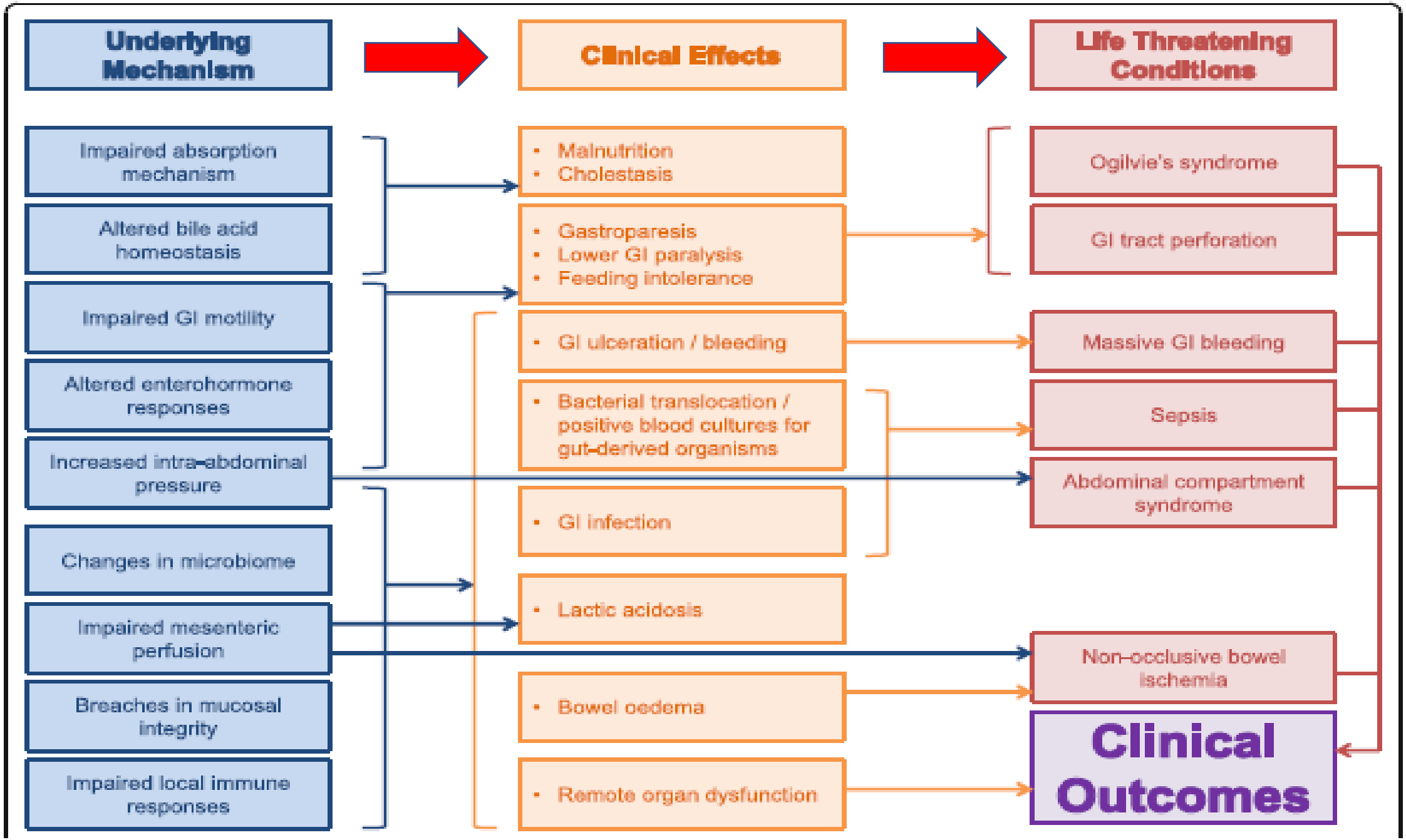
- Mild
  - Nausea
  - Reflux
  - Vomiting

  50-59%
- Moderate
  - Abdominal distension
  - Impaired gut motility/Ileus
  - Enteral feeding intolerance
  - Diarrhea

  30-32%
- Severe
  - Gut failure
  - Non-occlusive mesenteric ischemia
  - Perforation and peritonitis

  0.1-3.4%

*



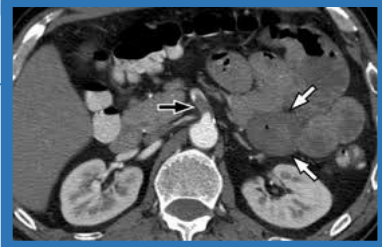
Reintam Blaser, A., Preiser, JC., Fruhwald, S. et al. Gastrointestinal dysfunction in the critically ill: a systematic scoping review and research agenda proposed by the Section of Metabolism, Endocrinology and Nutrition of the European Society of Intensive Care Medicine. Crit Care 24, 224 (2020). https://doi.org/10.1186/s13054-020-02889-4

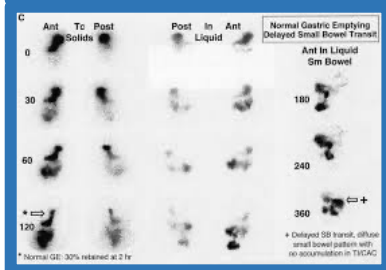
Gut dysfunction impacts clinical outcome in ICU patients from a range of mechanisms

*

Methods for scoring gut dysfunction

- **Clinical**
  - Signs/symptoms
  - Radiology
  - Laboratory tests
- **Research**
  - Scintigraphy (emptying)
  - Tonometry (PHi)
  - Breath Tests (H_2)
  - Absorption tests (Paracetomol)
  - Biomarkers (D-lactate, i-FABP, Citrulline)




Poll Question 2

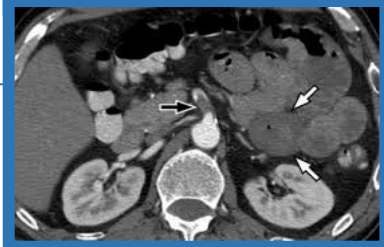
An elevated **serum lactate** indicates global/overall systemic hypoperfusion, but can be easily reversed by

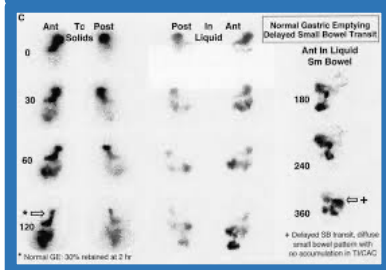
- Enteral feeding
- Fluid resuscitation
- Parenteral feeding
- Laparotomy

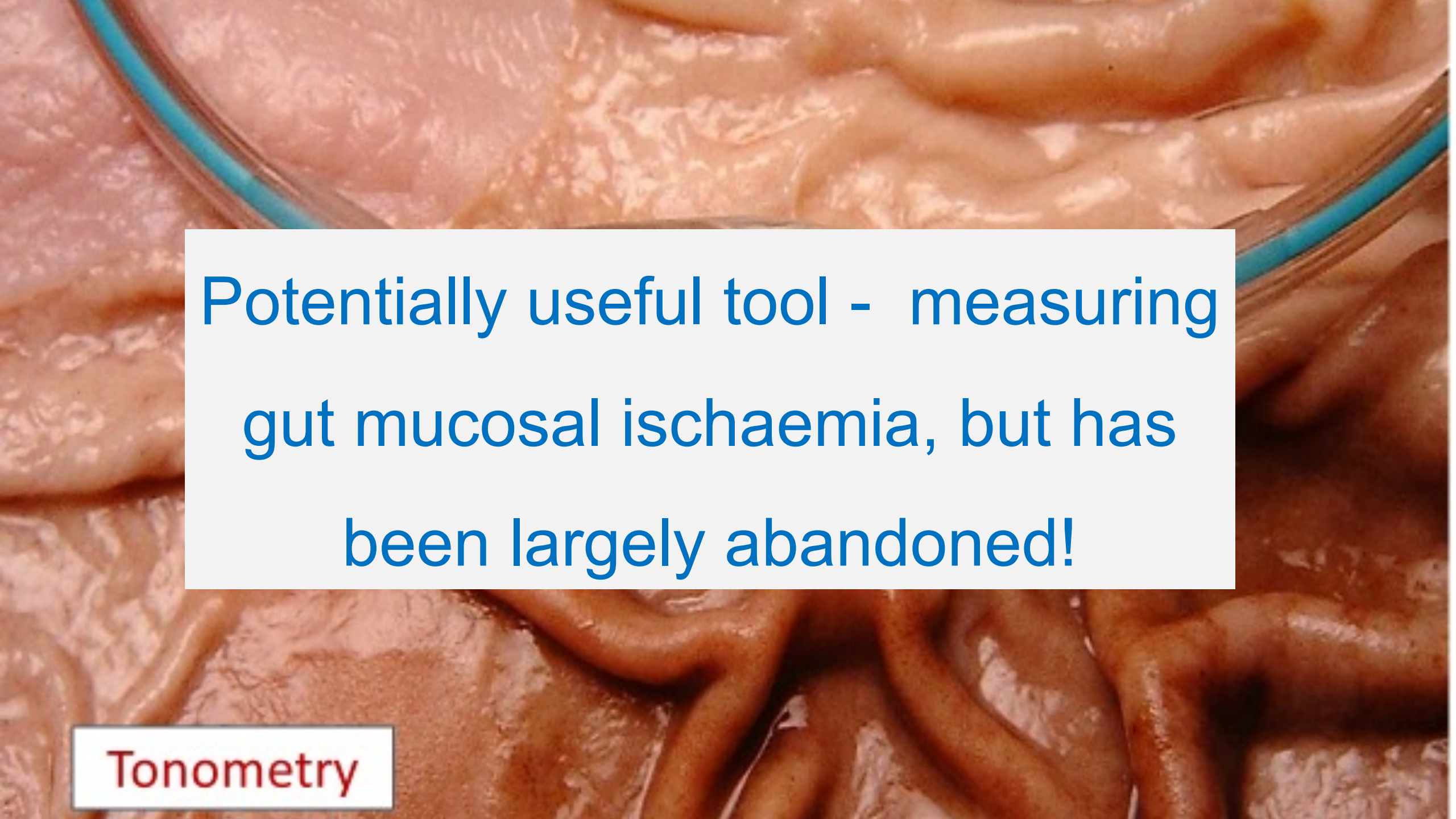
Methods for scoring gut dysfunction

- **Clinical**
  - Signs/symptoms
  - Radiology
  - Laborat
- **Resea**
  - cintigraphy (emptying)
  - Tonometry (PHi)
  - Breath Tests (H_2)
  - Absorption tests (Paracetomol)
  - Biomarkers (D-lactate, i-FABP, Citrulline)

None useful at the ICU bedside



Potentially useful tool - measuring gut mucosal ischaemia, but has been largely abandoned!

Tonometry

No agreed or established method

to score gut dysfunction

in critical illness.

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How to feed the undernourished ICU patient

When Enteral Feeding is Good!

- Post-operatively
- Critical illness
- Patients unable to eat orally
- Trauma patients
- Patients with an intact GI tract
- Moderate to severe weight loss
- Catabolism
- Head injuries

Mild Gut Dysfunction

- Nausea
- Vomiting

Enteral feeding should be the first line of intervention

- Constipation
- Mild diarrheoa

When Enteral Feeding could be Bad!

- Multiple abdominal surgeries
- Severe ileus
- Obstruction (mechanical)
- Active GI bleed
- NG output > 1.5 L /day
- Abdominal compartment syndrome
- Penetrating trauma
- Worsening anastomotic leak

Moderate Gut Dysfunction

- High NG output
- Projectile vomiting

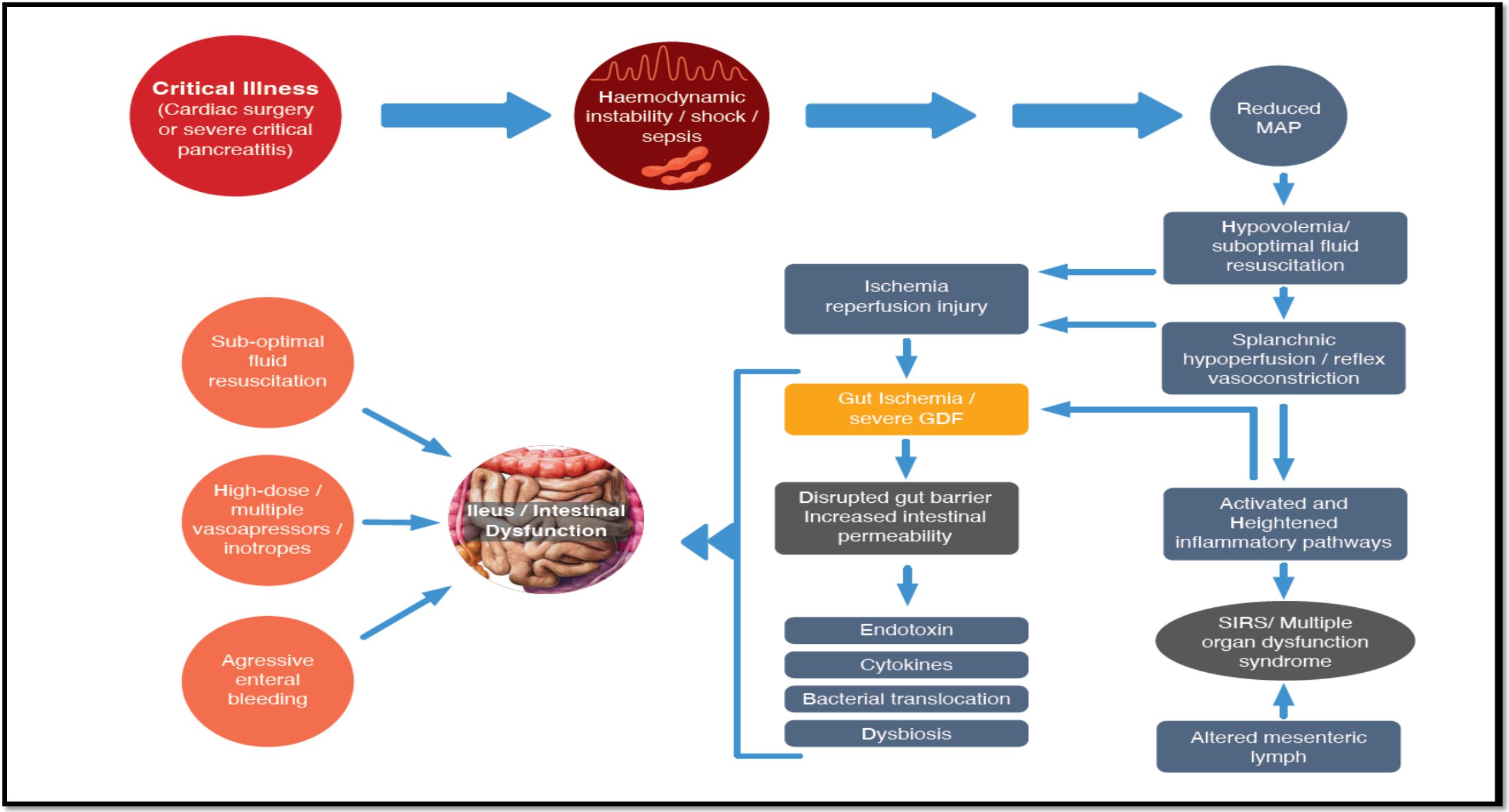
Trophic feed with gradual increase to goal +/-
Consider supplemental PN
Assess for EN eligibility

When is Enteral Feeding Ugly!

- Haemodynamically unstable patients
- Unstable core body temperature
- Shock and severe haemorrhage
- Inadequate resuscitated
- High catecholamines use
- Significant risk of bowel ischemia (NOMI)
- Hypotensive with a labile mean arterial pressure

Severe Gut Dysfunction / Life-threatening Gut Failure

Stop enteral feeding and start PN as indicated



Enteral versus parenteral early nutrition in ventilated adults with shock: a randomised, controlled, multicentre, open-label, parallel-group study (NUTRIREA-2)

- Classic Study
- Target feeding Day 1 ICU
- 2.2mg/hr (0.53ug/kg/min) high dose vasopressor
- Severe complications – NOMI/ACPO (3% on EN)
- 74% Mortality

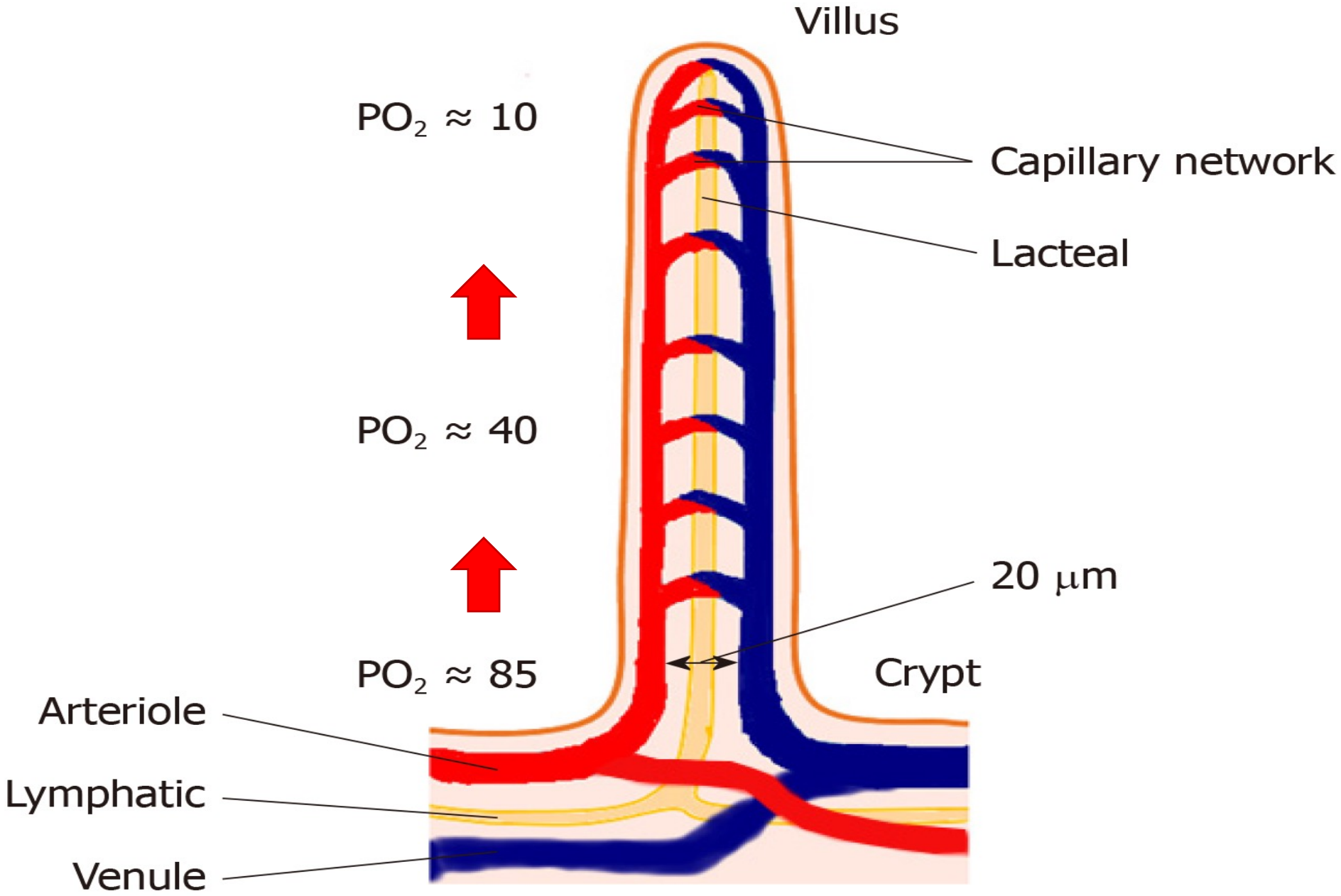
Reignier et al, 2018

Poll Question 3

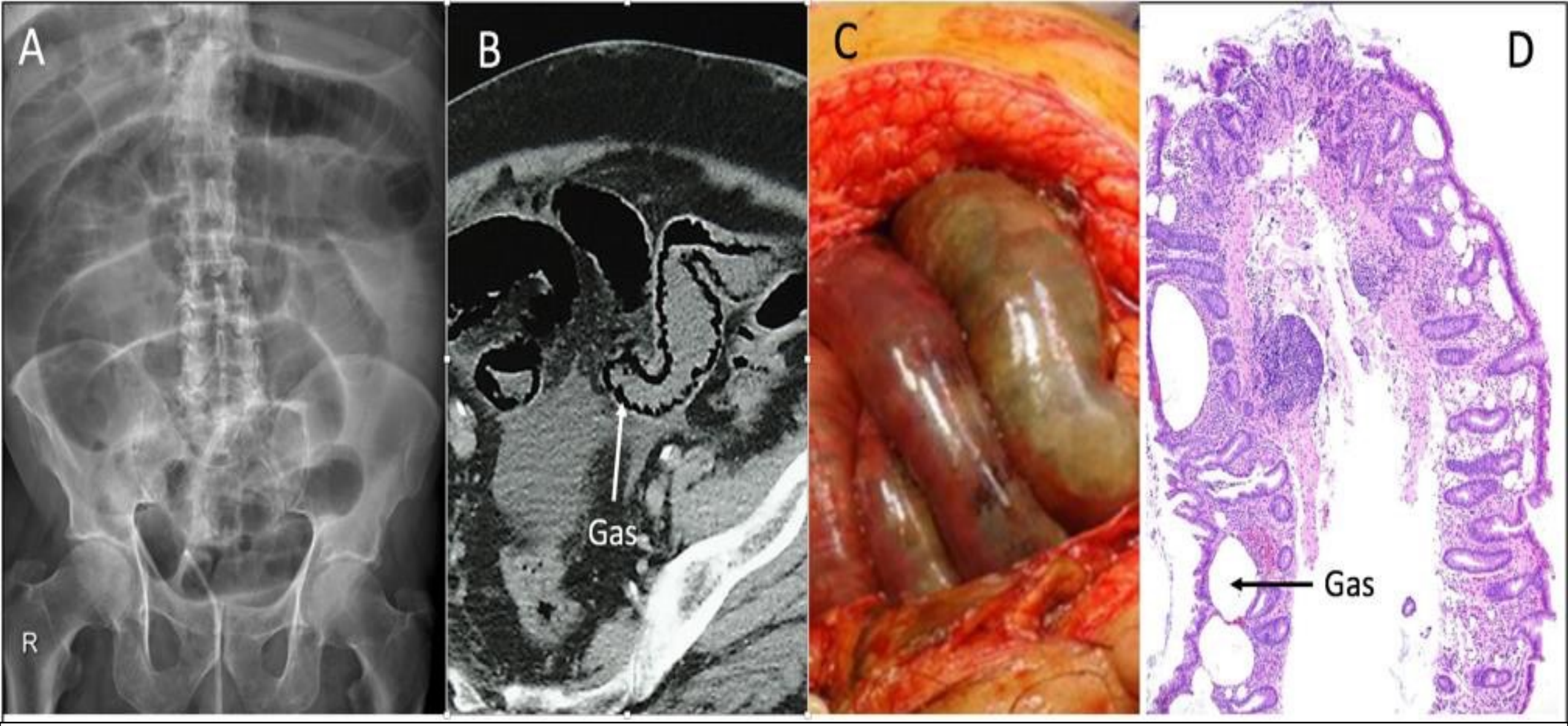
Oxygen extraction beyond __% of the intestine triggers bowel infarction and ischaemia

- 20%
- 50%
- 75%
- 80%

The oxygen battle in the gut



Hewawasam, S. P. (2020). Hypoxia and oxidative stress: The role of the anaerobic gut, the hepatic arterial buffer response and other defence mechanisms of the liver. World Journal of Meta-Analysis, 8(2), 78-88.



A disbalance in O_2 demand and supply leads to NOMI

Vasopressor Choice	Vasopressor Dose	Resuscitation Markers and Suggestions for EN Delivery Safety	Feeding Strategy	Signs of Intolerance
Norepinephrine, norepinephrine/ dobutamine and phenylephrine > epinephrine > vasopressin/ dopamine	Keep norepinephrine doses (equivalents) lower:	1. Lactate normalized or falling rapidly	1. Start with gastric delivered trophic feeding (10–20 cc/ hr) (no post-pyloric feeding)	1. Increased gastric residual (> 500 cc's)
(Observational data and animal data supporting rec- ommendation)	< 0.1 μg/kg/min—more optimal	2. Vasopressor dose decreasing or stable	2. Advance EN slowly and watch for signs of intolerance	2. Abdominal distension
	0.1–0.3 μg/kg/min— may be acceptable	3. Mixed venous O_2—within normal limits or elevated	3. Consider elemental or peptide formula to min- imize gut O_2 consump- tion for absorption	3. Nausea/vomiting
	> 0.3– 0.5 μg/kg/min— significant risk—should not be done	4. Fluid requirements stabi- lizing, no ongoing active bleeding		4. New abdominal pain
		5. Limit crystalloid fluid over- resuscitation to reduce bowel edema (especially in septic shock—with more pronounced vas- cular leak)		5. Unexplained elevation in lactate with feeding initiation or escalation
				6. Intra-abdominal hypertension or abdominal compartment syndrome

EN = enteral nutrition.

Wischmeyer, 2019

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Clinical signs and symptoms

- Bedside assessment (twice daily)
- Subjective assessment by nursing or junior ICU team
- Did not use a validated score
- Did not use a standardized protocol

Acute Gastrointestinal Injury (AGI)

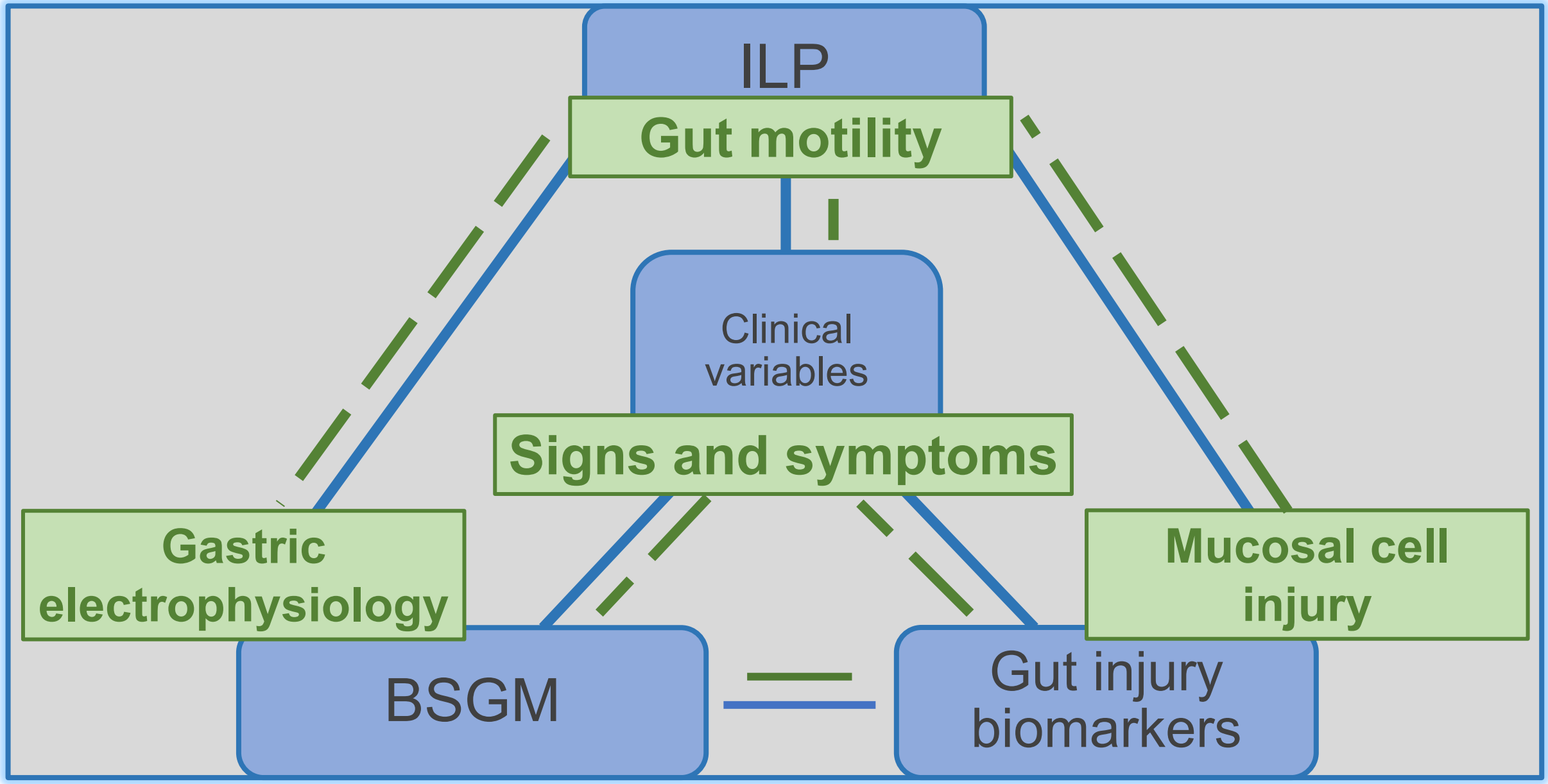
I	**Increased risk** of developing gut dysfunction (self-limiting)
II	**Gut dysfunction** (requires intervention)
III	**Gut failure** (cannot be restored with interventions)
IV	**Dramatic gut failure** (immediately life threatening)

Reintam et al. 2012

Gastrointestinal Dysfunction Score (GIDS)

0	**No risk** of developing gut dysfunction
1	**Increased risk** of developing gut dysfunction
2	**Gut dysfunction**
3	**Gut failure**
4	**Life threatening**

Reintam et al. 2021


ILP
Gut motility
Clinical variables
Signs and symptoms
Gastric electrophysiology
Mucosal cell injury
BSGM
Gut injury biomarkers

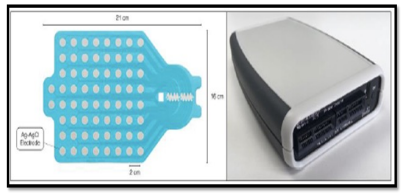
Admission

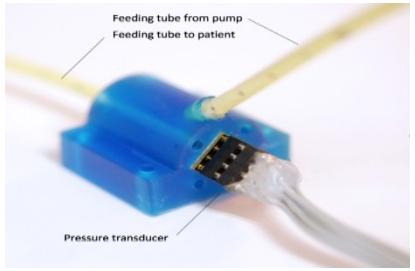
Screening and Eligibility

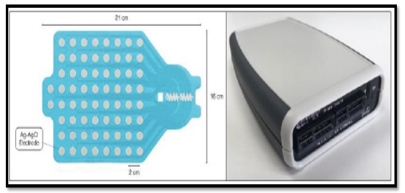
72hrs

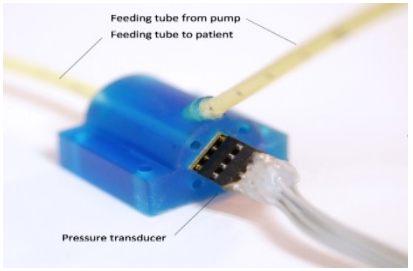
Day 5-7

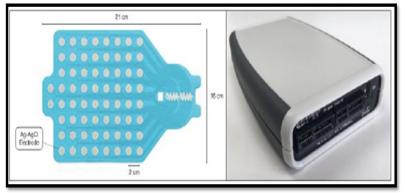
Day 7-10

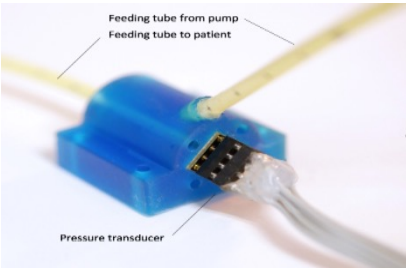




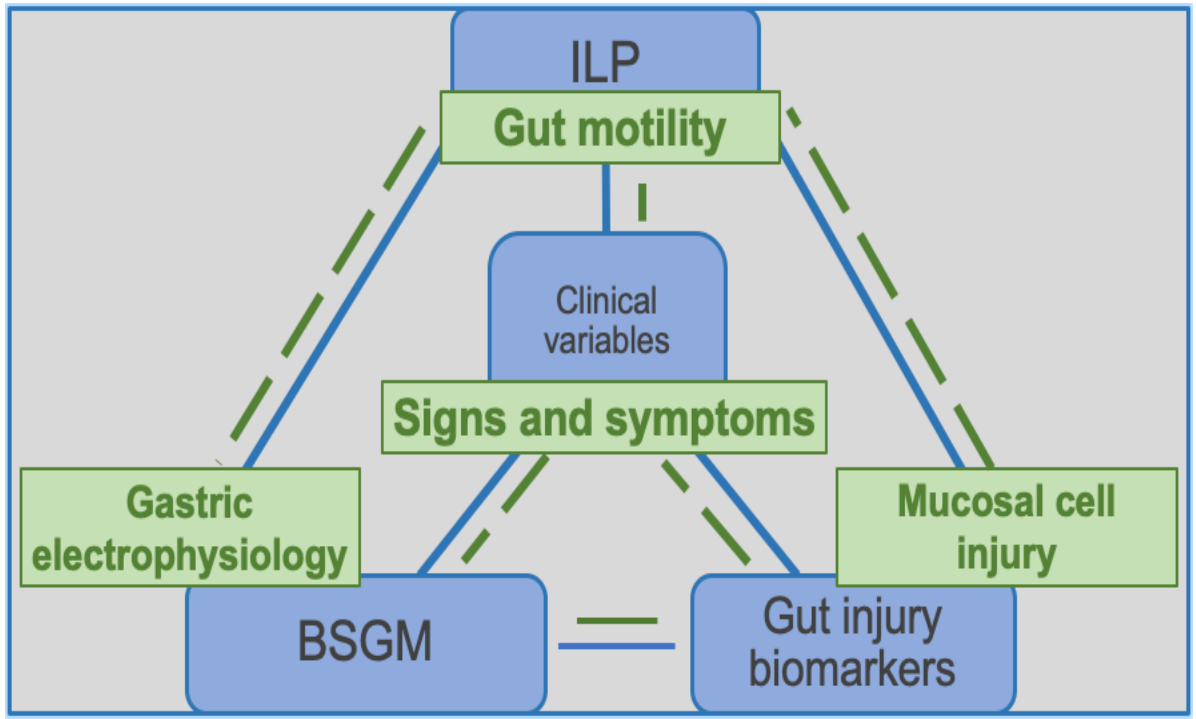








Developing and validating a novel gut dysfunction scoring tool in critical illness



Future steps

- Validating the new scoring tool in a prospective study to assess the severity of gut dysfunction and predict outcomes
- Determining whether a simplified metric is possible and can be incorporated into multiorgan SOFA score or equivalent

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Conclusion

- Gut dysfunction is common in ICU patients and contributes to disease severity.
- Feeding early but with caution is a priority in sicker patients with high risk of life-threatening gut failure.
- ICU dietitians need to be proactive in assessing the risk of ‘gut failure’ and in particular with ‘vulnerable critically ill’ patients.
- Early trophic and Supplemental PN may be the answer for severe cases of gut failure.
- Gut dysfunction assessment and early diagnosis is an urgent and important priority.

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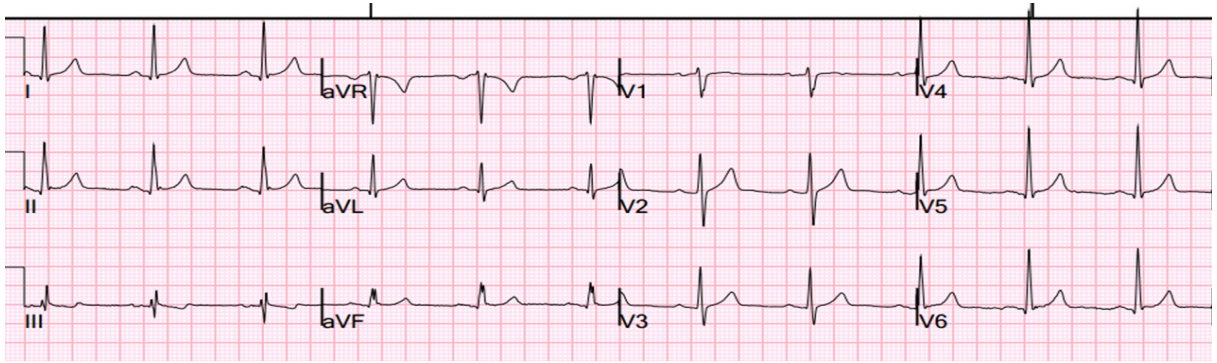
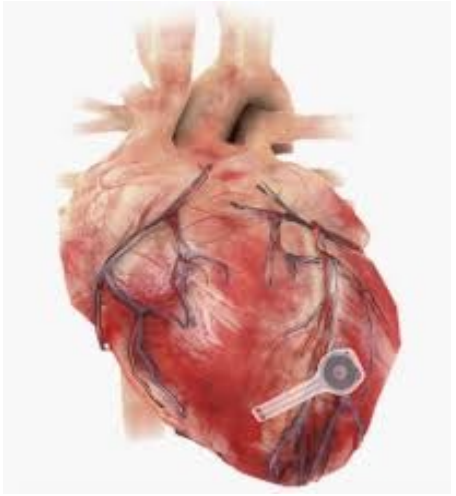


ECG

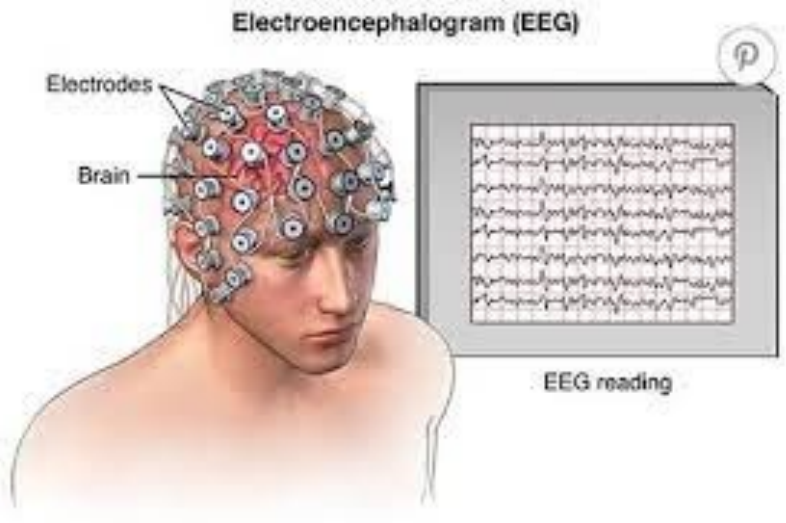
Heart rate



Troponin

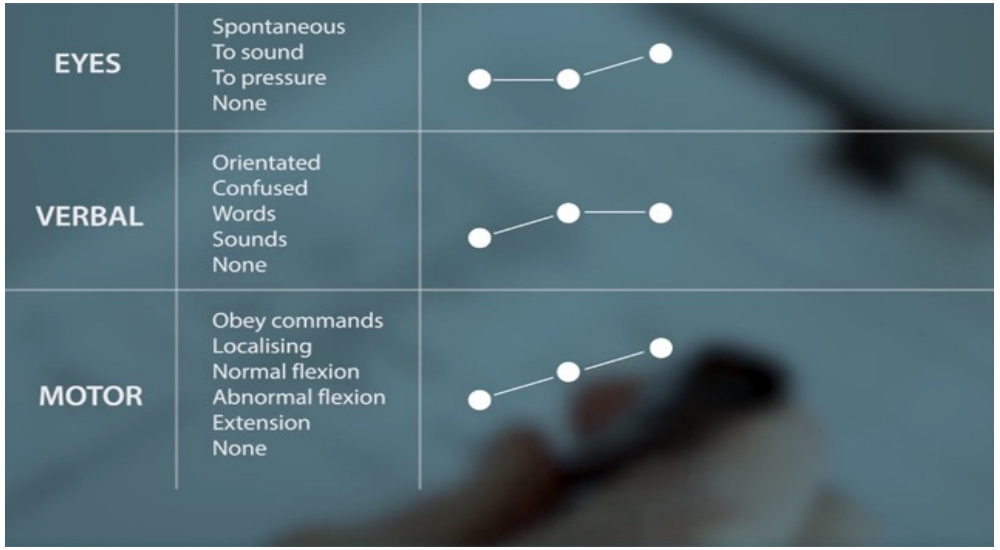
Blood Pressure

Serum Sodium

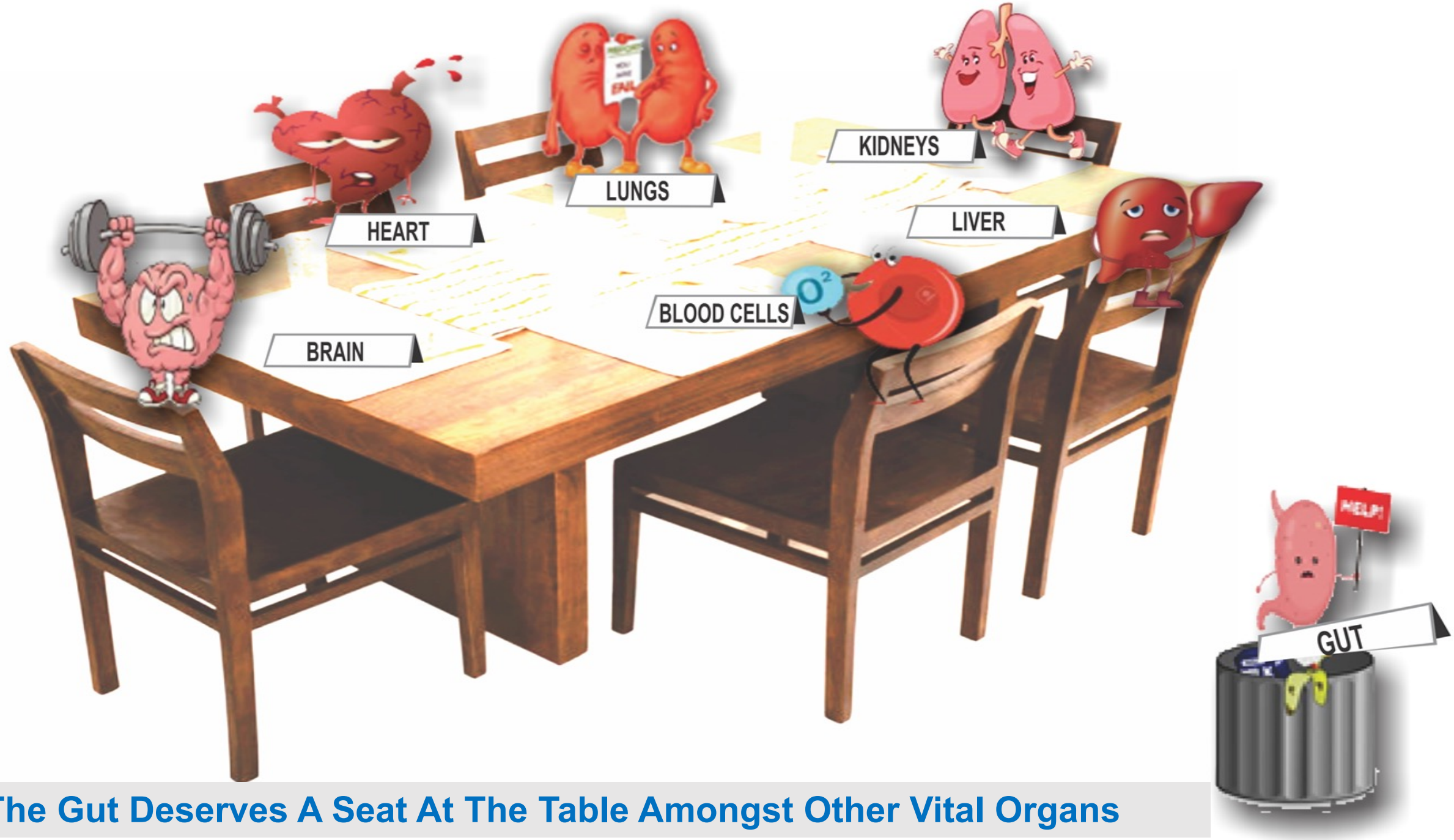


EEG

ICP monitor

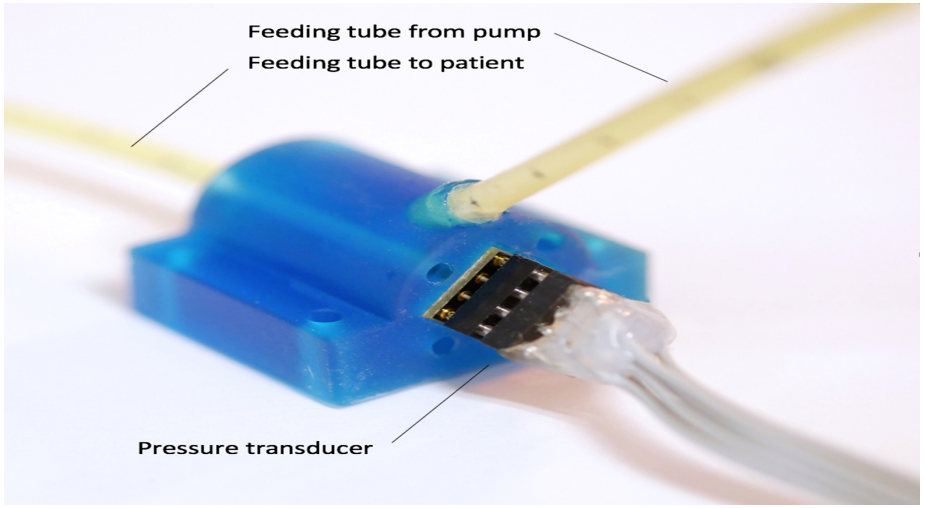


GCS score



The Gut Deserves A Seat At The Table Amongst Other Vital Organs

Body Surface Gastric Mapping



Intraluminal pressure

Gut Biomarker

Gut Scoring System

STaR Centre Directors and Collaborators

Prof John Windsor

Prof Ian Bissett

Prof Anthony Phillips

Prof Greg O'Grady

Dr Colin McArthur

Dr Jiwon Hong

Dr Shay McGuiness

Prashanna Khwaounjoo

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Desktop Indirect Calorimetry in the Burns Unit

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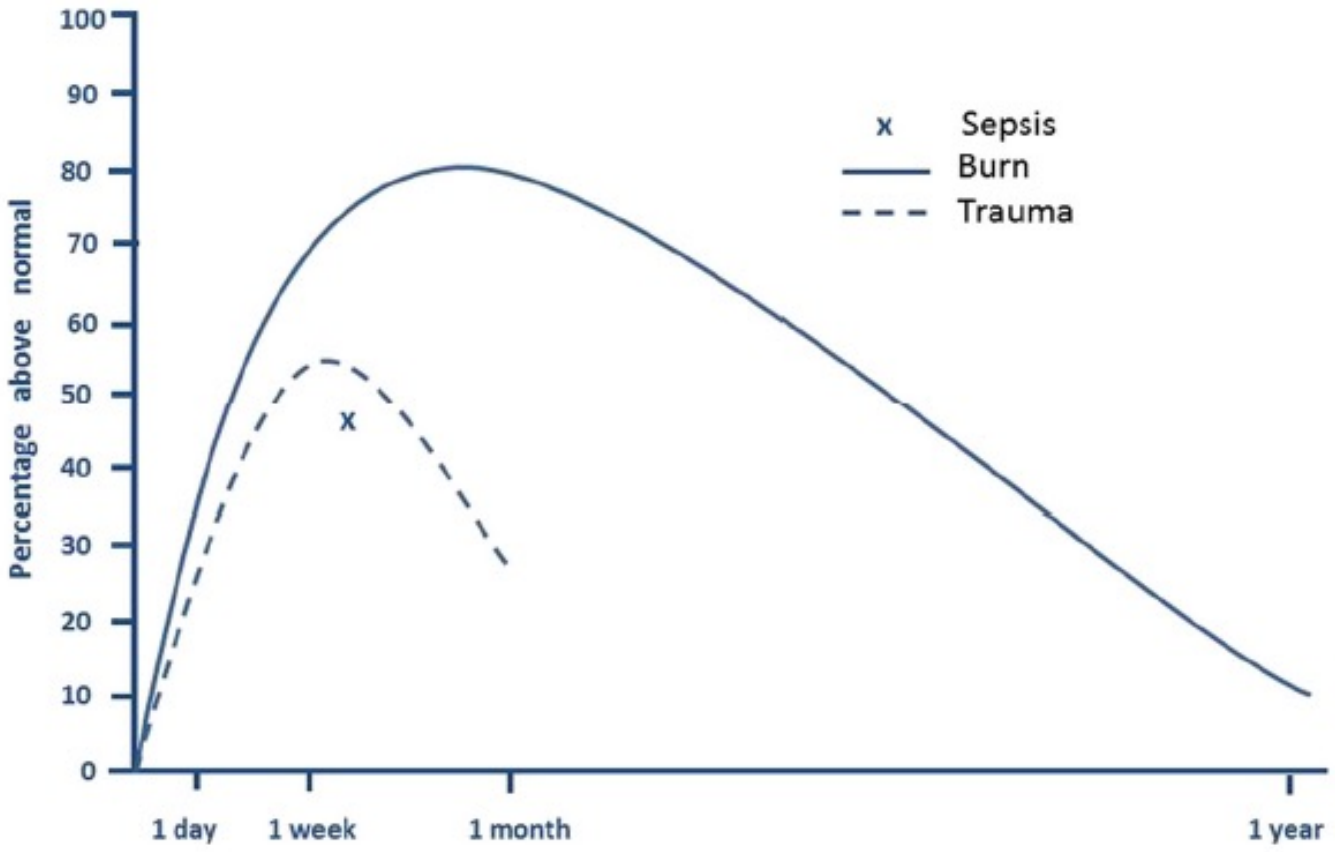
Disclosures

- Speaker Fee - Baxter

Hypermetabolism following Burn Injury



Hypermetabolism in Burn Injury

- Consequences:
  - Abnormal metabolism of nutrients
  - Increased nutrition requirements
  - If untreated:
    - Loss of muscle mass
    - Immune compromise
    - Delayed wound healing

Hypermetabolism in Burn Injury

- Management:

Thermoneutral environment

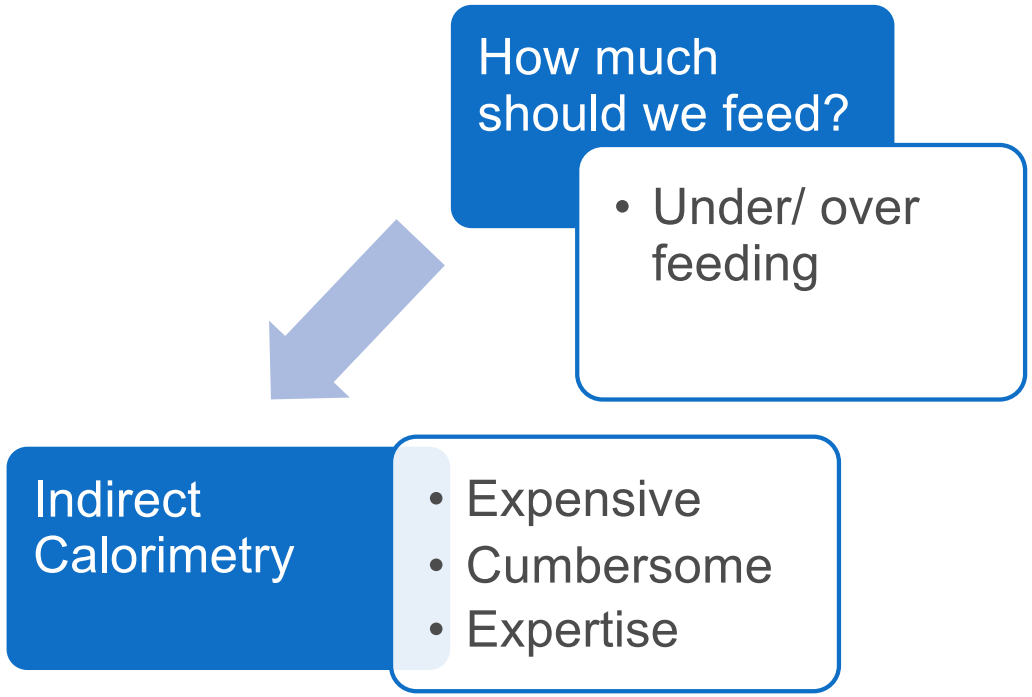
Early wound excision and grafting

Early nutrition support

Occlusive dressing

Analgesia

How much should we feed?



Nevin et al. Feasibility and acceptability of implementing indirect calorimetry into routine clinical care of patients with a spinal cord injury. *Top Spinal Cord Inj Rehabil* 2016; 22 (4):269-276
Rattanachaiwong S, Singer P. Indirect calorimetry as point of care testing. *Clinical Nutrition*. 2019 Dec;38(6):2531-2544
Wise AK et al. Energy expenditure and protein requirements following burn Injury. *Nutr Clin Pract*. 2019;34:673-680

Poll

- Are you currently using IC?
  - Yes
  - No

- If the measured energy expenditure was higher than 40 kCal/ Kg, would you feed to this?
  - Yes
  - No
  - Depends on the patient

Poll

- Can IC be used in unventilated patients?
  - Yes
  - No

- Would you add any injury or activity factors?
  - a. Injury factor only
  - b. Activity factor only
  - c. Both injury and activity factor
  - d. Neither

Indirect Calorimetry

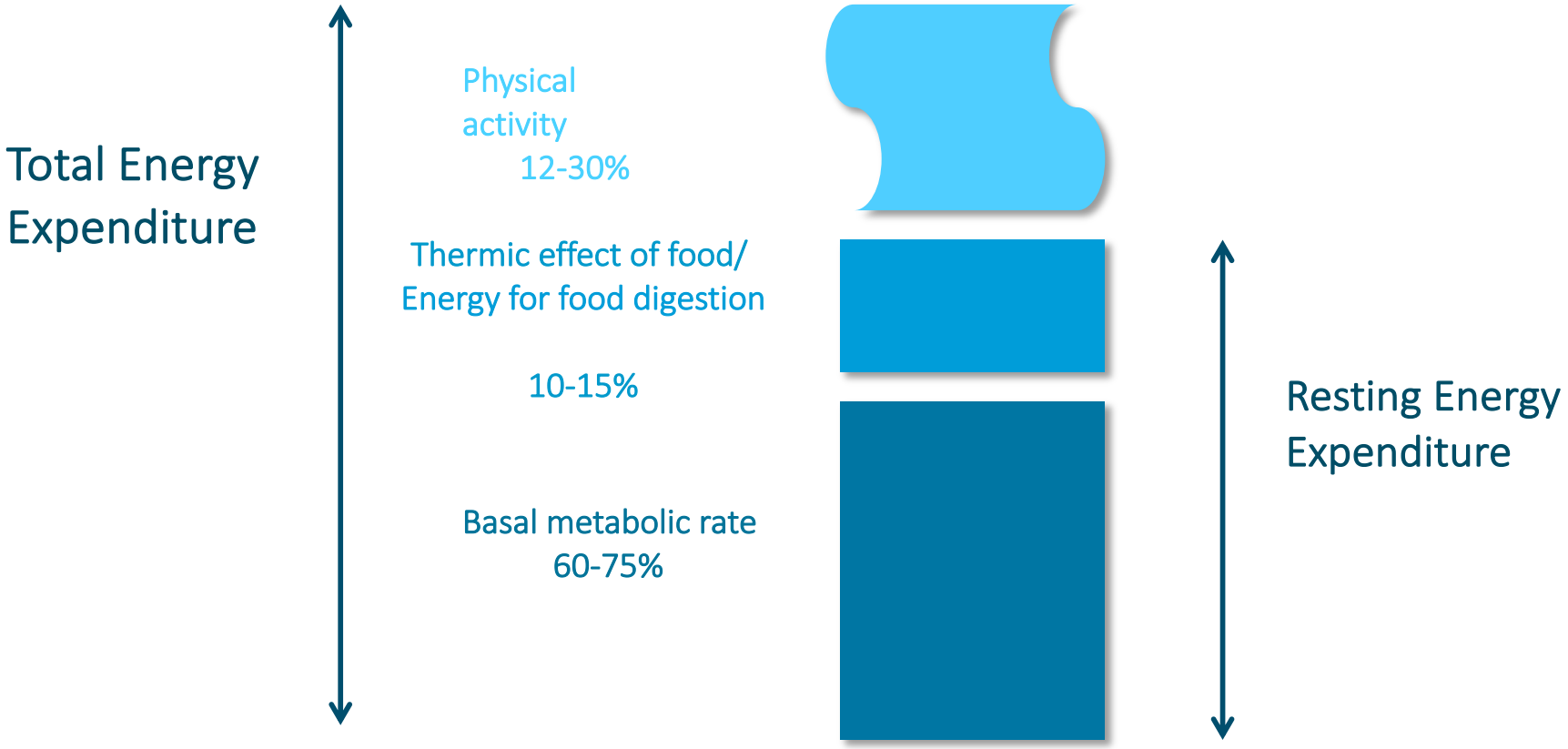
- Machine which measures the volume (V) of expired gas and the inhaled and exhaled concentrations of O_2 and CO_2

- Resting energy expenditure (REE) determined by
  - O_2 consumption
  - CO_2 production

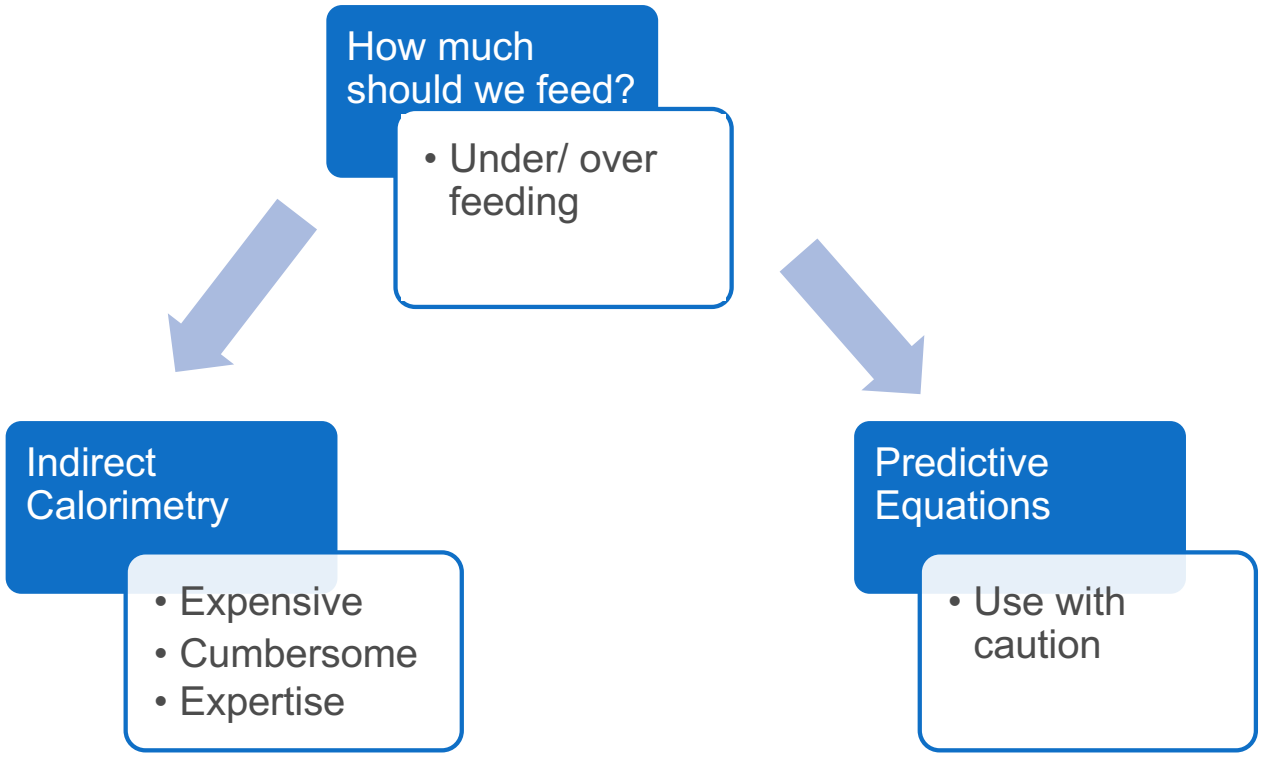
  - Via the Weir Equation:
- kcal per day = 1440 (3.94 VO2 + 1.11 VCO2)

Haugen HA et al. Indirect Calorimetry: A practical guide for clinicians. *Nutrition in Clinical Practice*. 2007;22:377-388.

Total energy expenditure



How much should we feed?



Rousseau et al. ESPEN endorsed recommendation: Nutrition therpay in major burns. *Clin Nutr* 2013;32;497-502
Leung J et al. Predictive energy equations are inaccurate for determining energy expenditure in adult burn injury: a retrospective observational study. *ANZ J Surg* 2019;89:578-583

Predictive equations in burn injured patients

- Studies comparing the predictive equations in burns injured patients with indirect calorimetry all recommend different equations

Accuracy of Predictive Methods to Estimate Resting Energy Expenditure of Thermally-Injured Patients*

Roland N. Dickerson, PharmD†; Jane M. Gervasio, PharmD†; Marti L. Riley, MS, RD‡; James E. Murrell, CRT §; William L. Hickerson, MD¶; Kenneth A. Kudsk, MD¶; and Rex O. Brown, PharmD†

Reliability of resting energy expenditure in major burns: Comparison between measured and predictive equations

Jinwoo Jeon [1], Dohern Kym [1], Yong Suk Cho*, Youngmin Kim, Jaechul Yoon, Haejun Yim, Jun Hur, Wook Chun

Determination of Resting Energy Expenditure After Severe Burn

Beth A. Shields, MS, RD,* Kevin A. Doty, MS,* Kevin K. Chung, MD,* Charles E. Wade, PhD,*† James K. Aden, PhD,* Steven E. Wolf, MD*‡

Dickerson et al JPEN 2002; 26(1): 17-29
Jeon et al Clinical Nutrition 2019; 38(6): 2763-2769
Shields et al J Burn Care Res 2013; 34: e22-e28

Indirect Calorimetry - Today

Case Study

19 M with 80% TBSA flame burn

- Delayed transfer to CRGH (10 weeks post injury)
- Ventilated via tracheostomy
- Unspecified mental health Hx

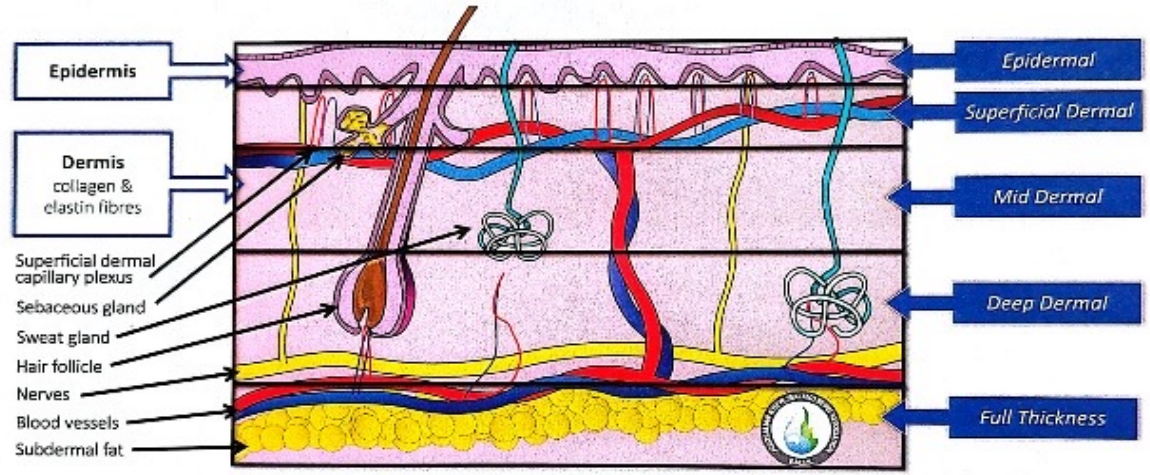


FIGURE 5.2 – Depth of burn injury

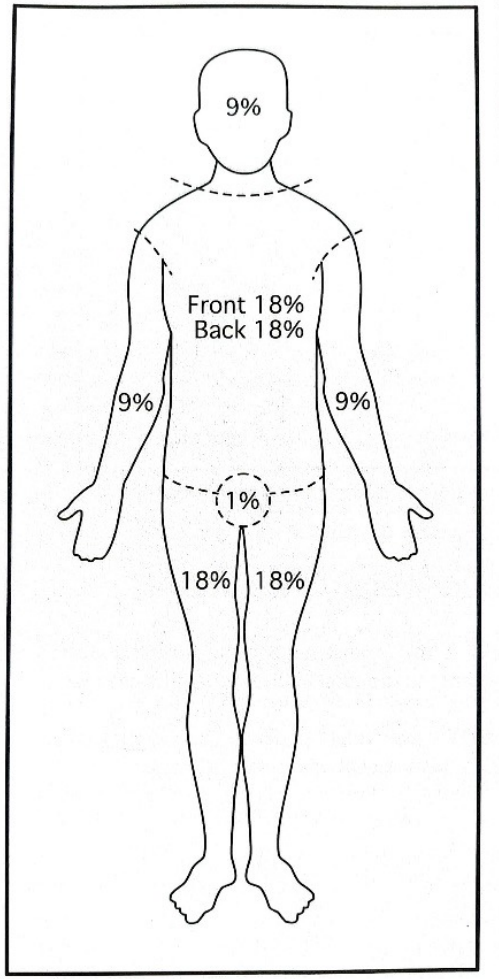


FIGURE 5.3 – Adult Rule of Nines

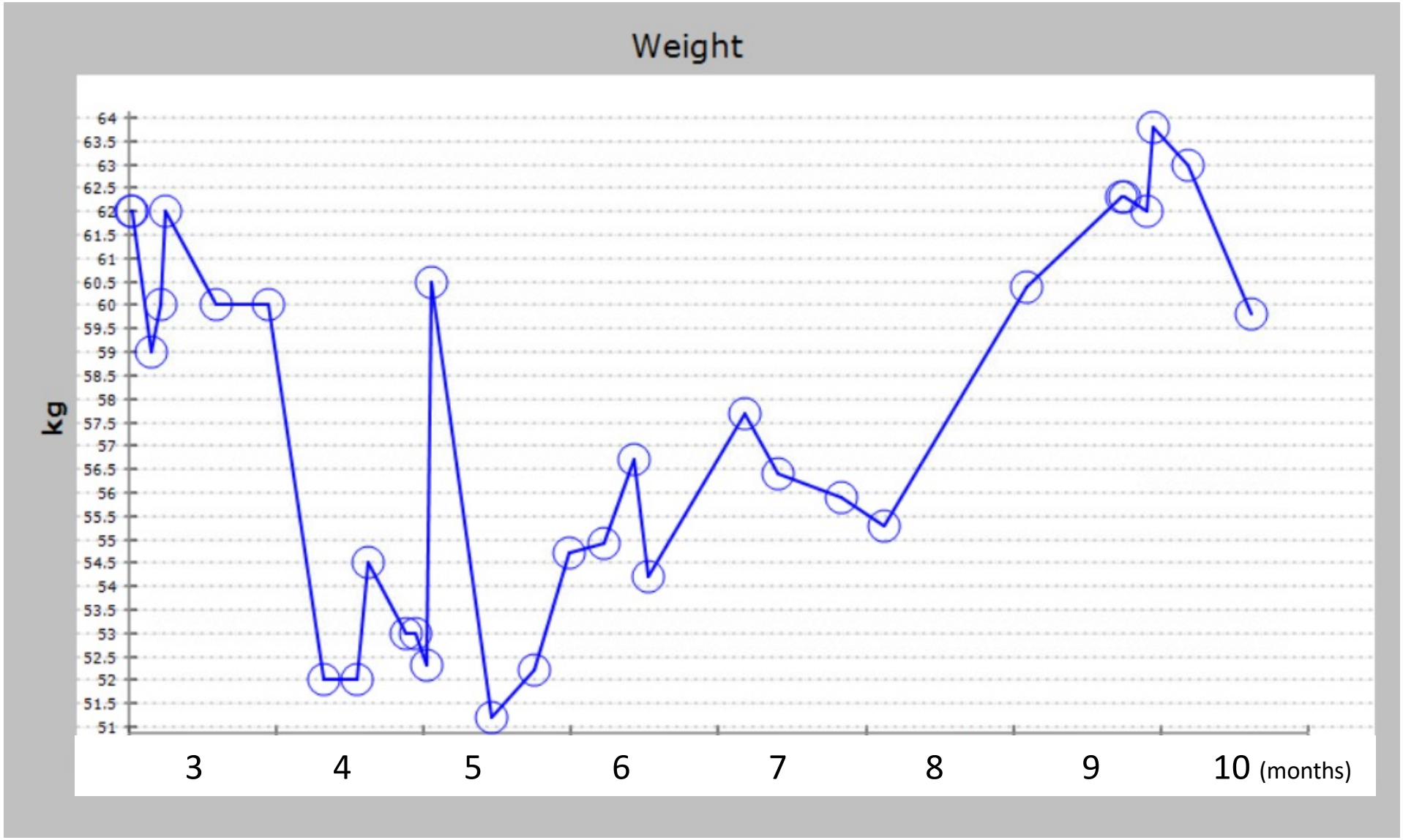
Australian and New Zealand Burn Association Ltd (ANZBA) (2021/2). Emergency Management of Severe Burns course manual (version 19).

On Admission

Anthropometry

Usual Weight and Height	On admission	Nutritional State
• 69kg • 180cm • BMI = 21kg/m^2	• 62kg • significant lean tissue and adipose wasting	• Malnourished

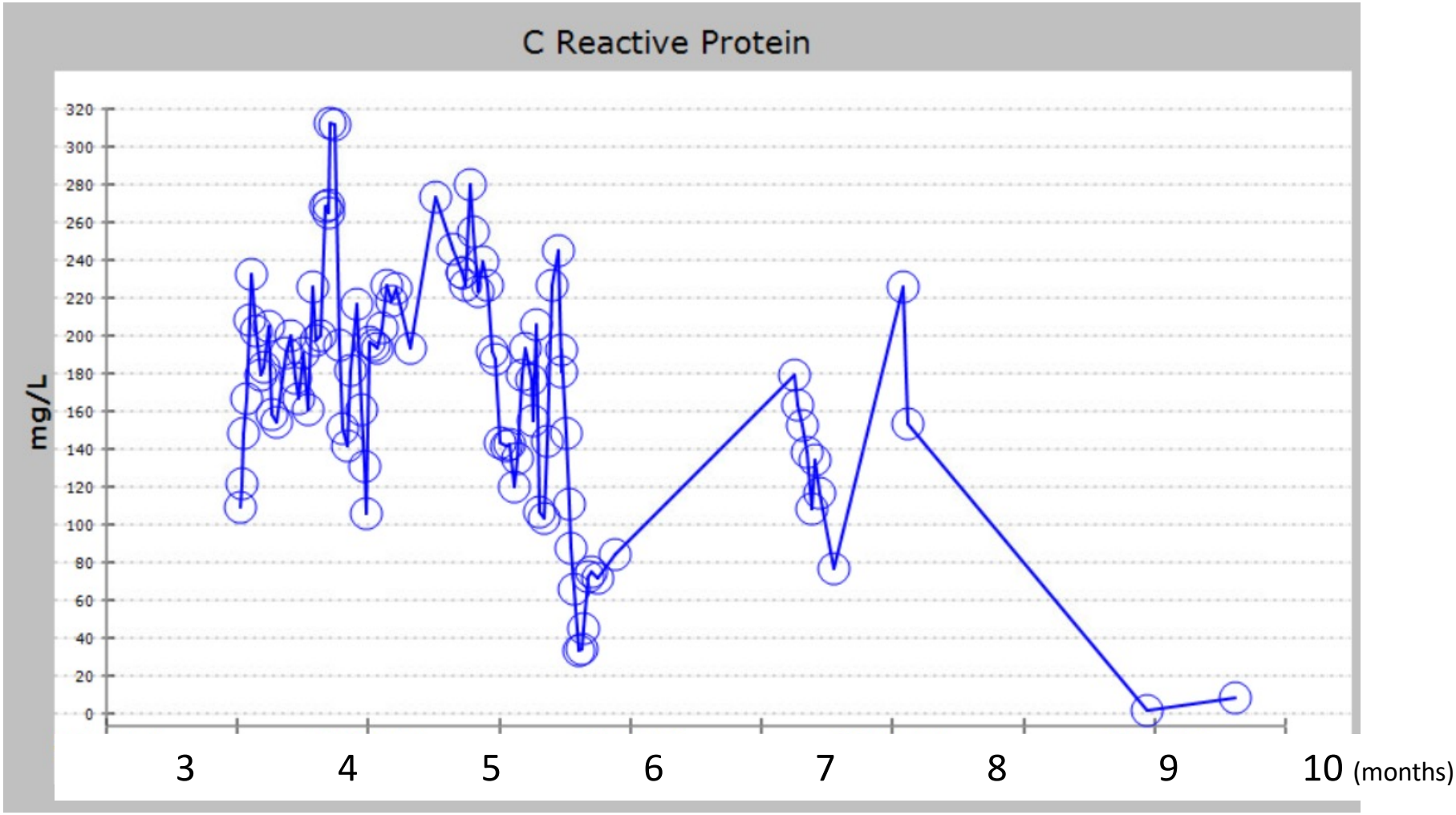
Weight over admission



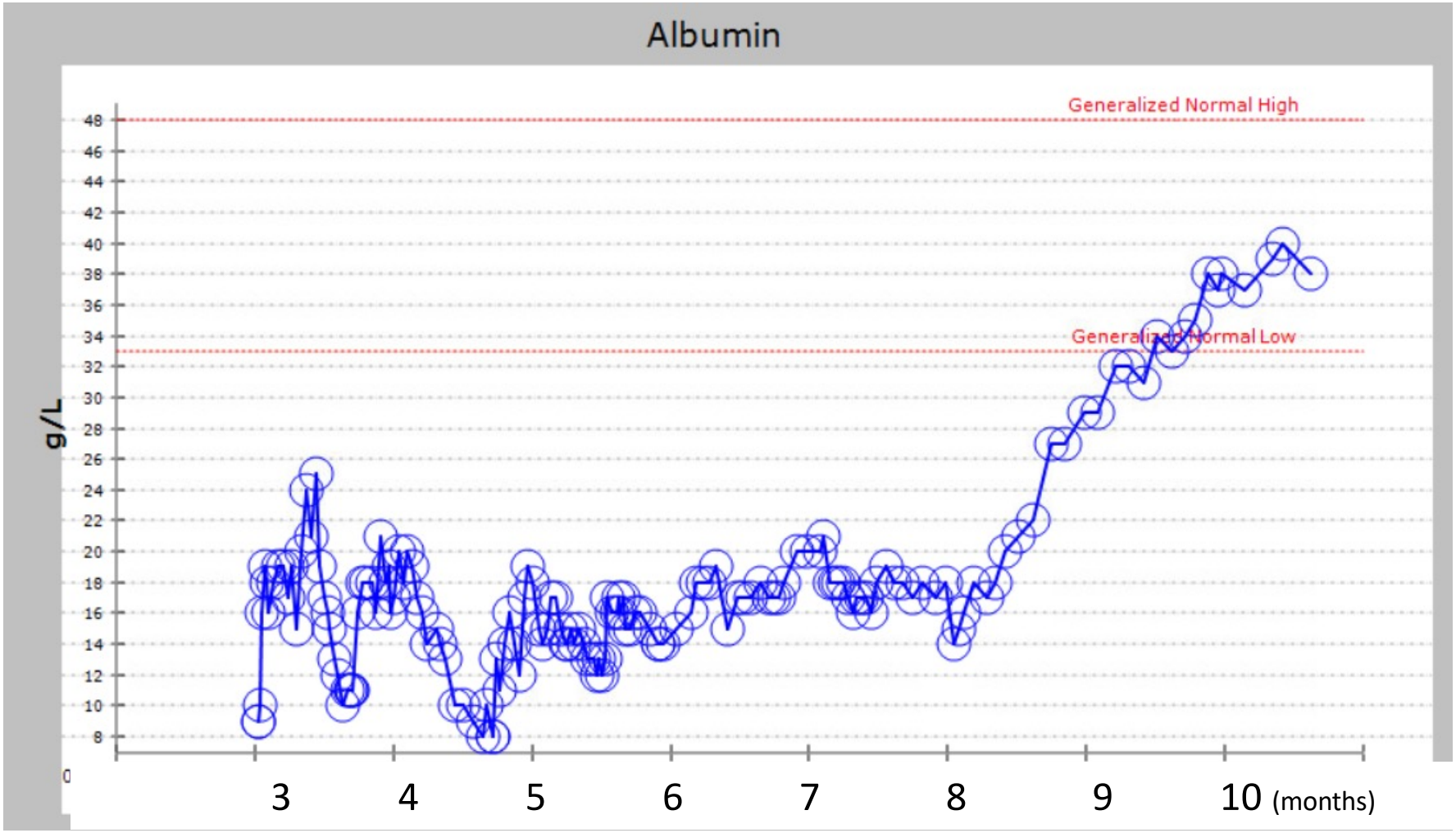
Biochemistry

- Biochemistry on day 1 (10 weeks post injury)
  - Na 144 K 4.0, Ur, 5.1, Cr 26 (L)
  - Mg 0.76, PO4 1.17
  - Alb 10 (L), CRP 135.1 (H)
  - PreAlb 0.12 (L)
  - Cu 6.9 (L), Se 0.67 (L), Zn 9 (L)
  - glu 6.0

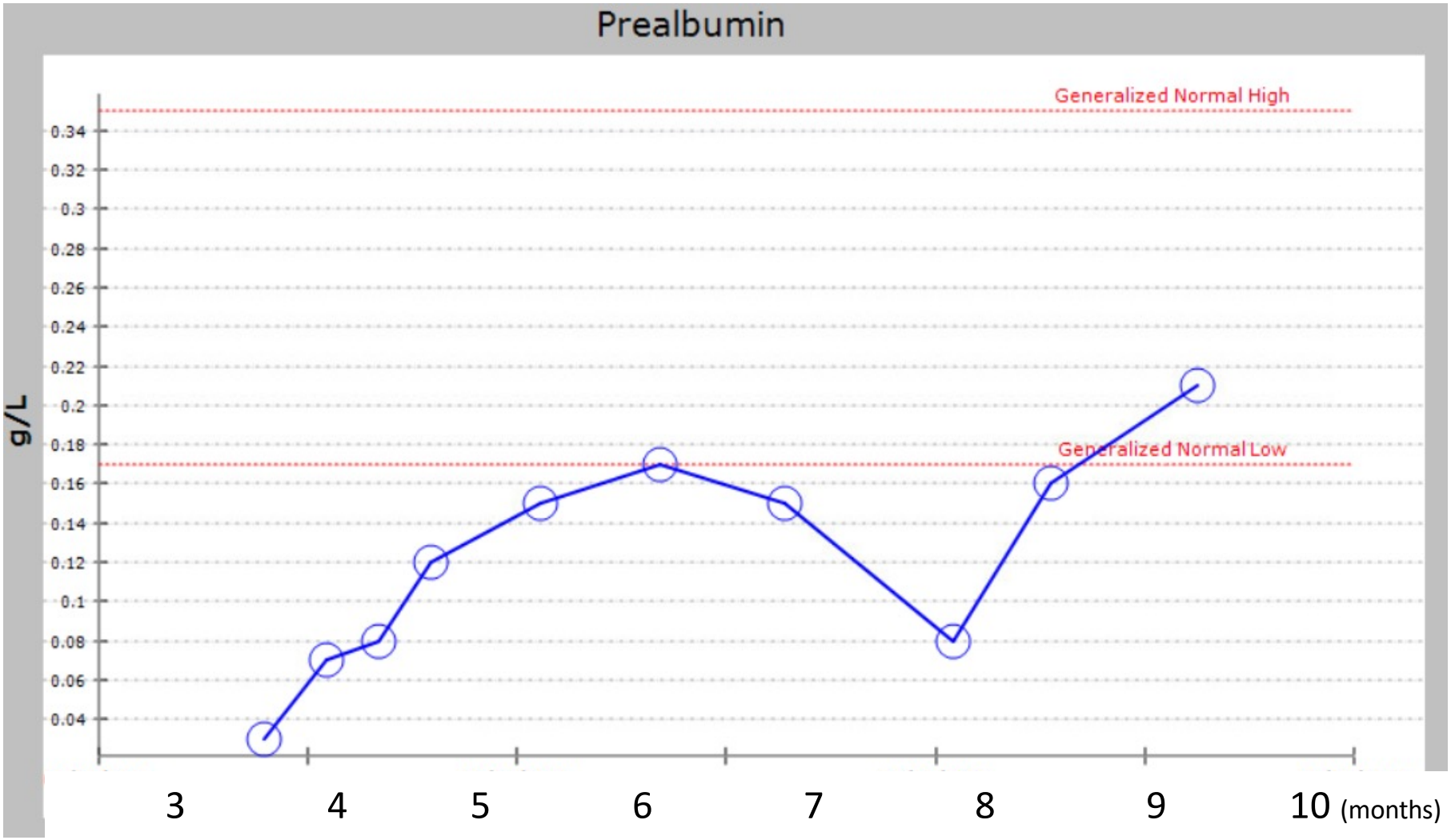
C Reactive Protein



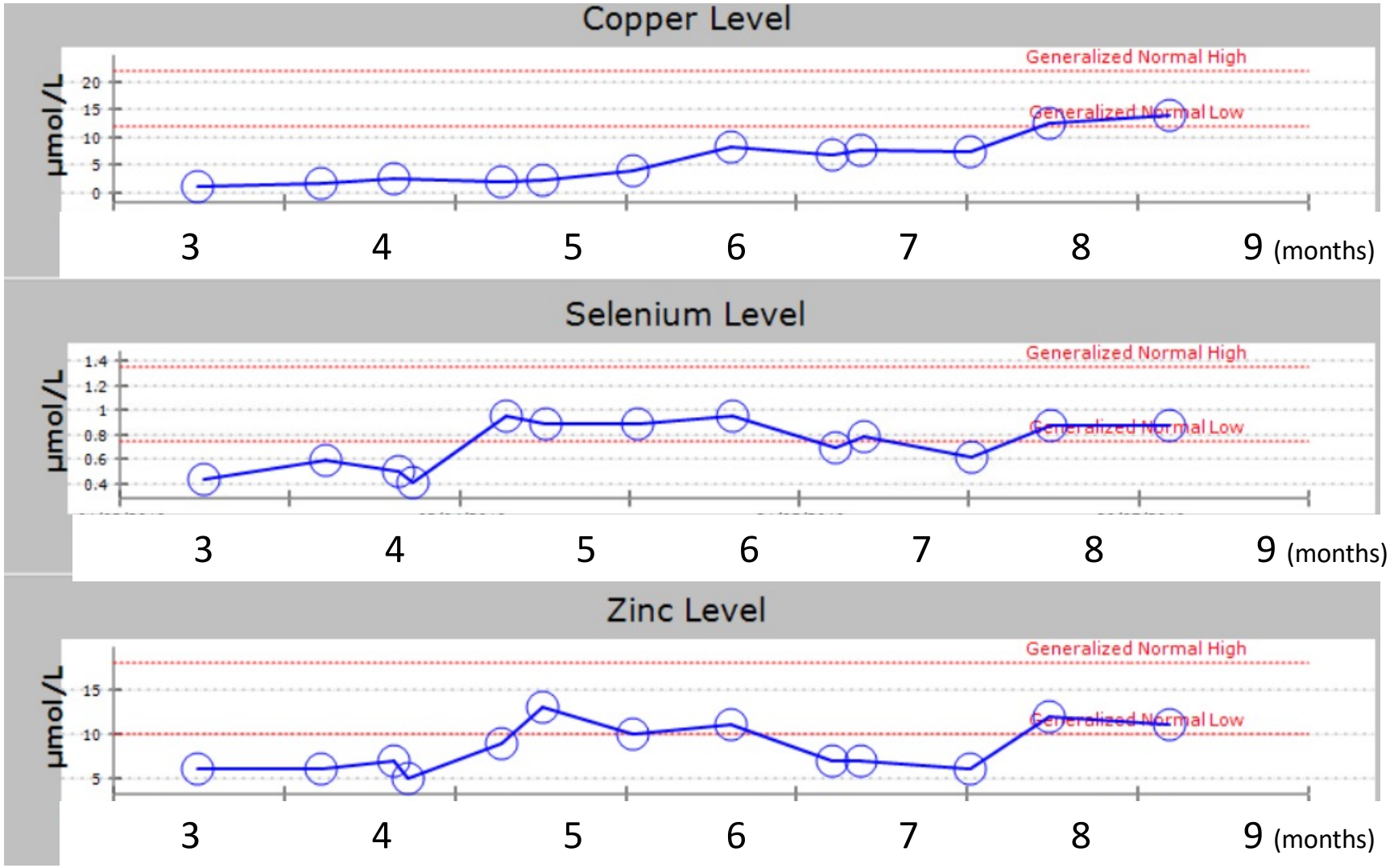
Albumin


Albumin
Generalized Normal High
Generalized Normal Low
g/L
48
46
44
42
40
38
36
34
32
30
28
26
24
22
20
18
16
14
12
10
8
3
4
5
6
7
8
9
10 (months)

Prealbumin


Prealbumin
Generalized Normal High
Generalized Normal Low
g/L
0.34
0.32
0.3
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
3
4
5
6
7
8
9
10 (months)

Trace Elements

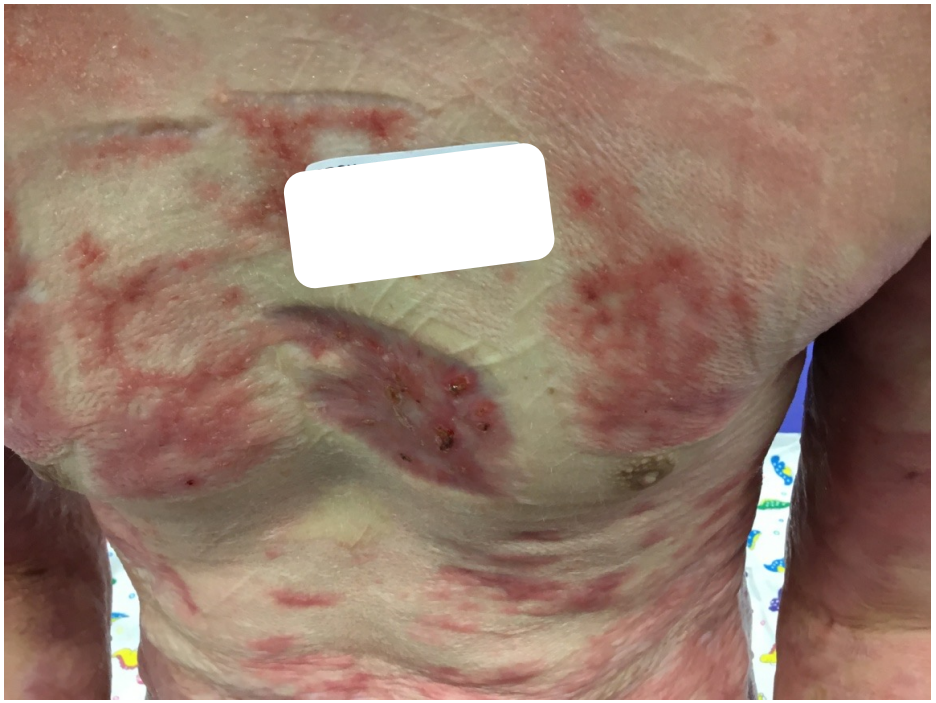


Admission

Surgical Interventions	Medications	Complications
• Debridement + Allograft + Autograft (x1) • Debridement + Autograft (x3) • Debridement + BTM (x2) • Trache revision (x1)	• Propanalol • Multivitamin/ mineral supplements • Pain Mx	• Sepsis/ SIRS • HAP • Deconditioning • Pressure injuries

Admission

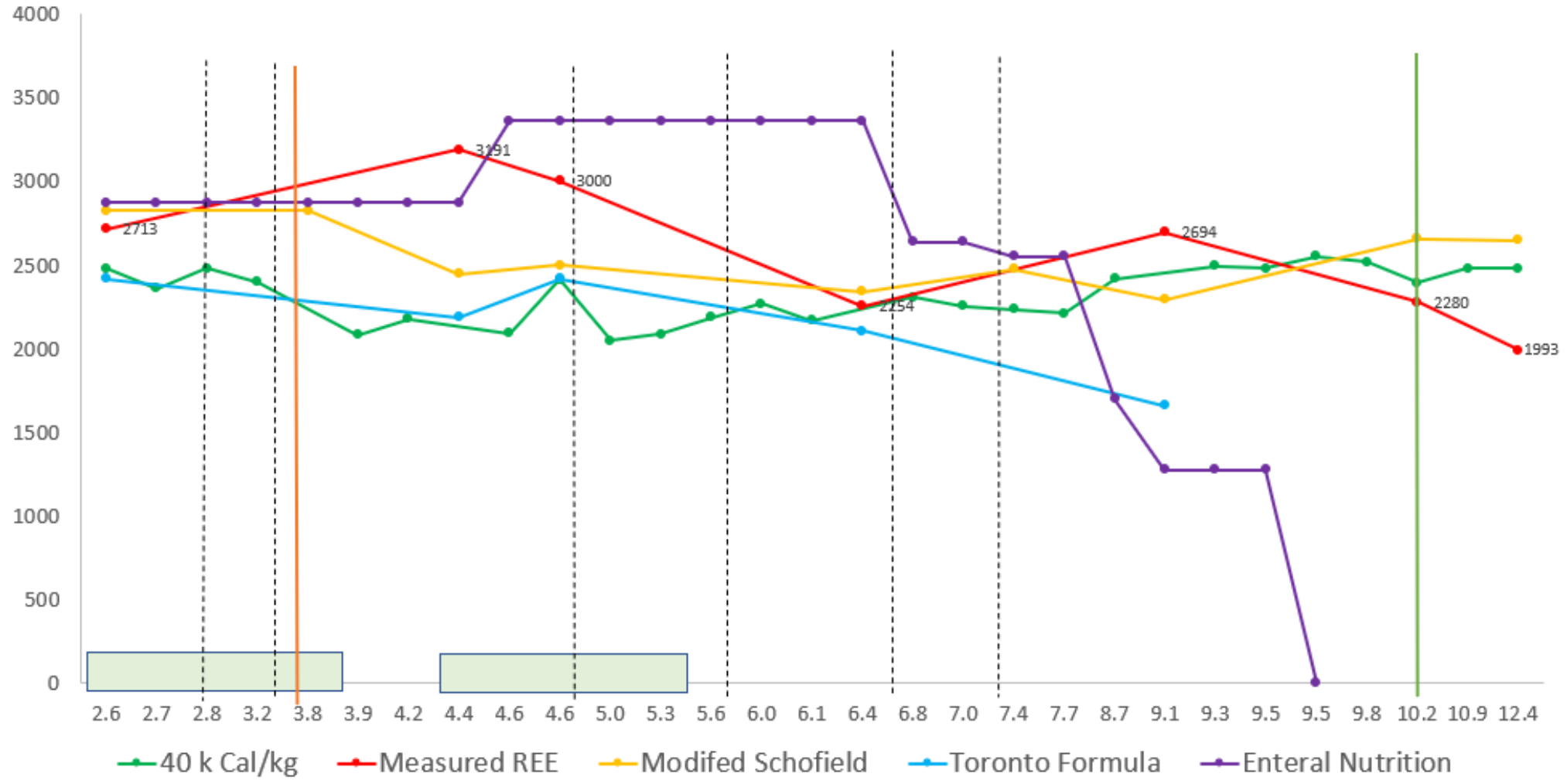
ICU	Ward	Discharge
• 5-week LOS • Readmitted for 4 weeks with HAP	• NG feeds 7 months • Trache out 7 weeks	• Home with OP rehab • 8 month total LOS



Indirect Calorimetry

- FitMate®
- Ideal conditions
  - 10-15 mins rest
  - Fasting for 5 hours
  - 4 hours post activity
  - 4 hours post caffeine
  - 1 hour post nicotine
- Steady State 5-10 minutes

Indirect Calorimetry



Practical Limitations

- Dietitian time
- Clinical setting
  - fasted/ therapy/ dressings

  = likely increases to measured energy expenditure
- Alternative equipment (hood)
- Infection control

Conclusions

- IC provides the opportunity to optimise energy assessment in patients with burn injuries to improve nutrition care

- Standard practice in severe burn patients

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- Australian and New Zealand Burn Association Ltd (ANZBA) (2021/2). Emergency Management of Severe Burns course manual (version 19).

Case Discussion

Dr Callum Pearce

Lead Clinician

Intestinal Failure Unit, Fiona Stanley Hospital

Female Patient Born 1990

- 2013: Seeing rheumatology, cardiology, orthopedics, frequent admissions to RPH & Fremantle Hospital

Past History:

- Hypermobile type-Ehlers Danlos Syndrome (h-EDS): Multiple dislocations, POTS, mast-cell activation syndrome (MCAS), neuralgia, dysphagia, reflux, N and V
- Multiple reported allergies, previous eating disorder including increasingly restrictive food intolerances. Asthma
- PTSD; patient receives regular counselling at sexual assault referral centre (SARC)

Progress Prior to TOC by FSH

- *2013: "This young lady returned for review and certainly has had a lot of medical attention in the last 4 months or so. She has been to EDs on several occasions with a combinations of chest pain, tachycardia and generalised symptoms."*
- *"We need to be careful not to over-interpret physical symptoms. She has a high level of sensitivity to many things including drugs, food…"*
- 2014: No admissions
- 2015: 7 admissions. PUJ obstruction and infection x2, viral illness, gastroenteritis
- 2016-2018: 6 admissions

Presentation to FSH - IF

- 2019: 9 admissions

- Jan 2019: NGT inserted after BMI <14
- Mar 2019: *“functional disorder GI tract. Not tolerating NG feeds”*
- Lost confidence in RPH so referred to FSH – IF team

- Apr 2019: BMI 16. NGT in for 8 weeks. PEG inserted
- Sep 2019: Started iv fluid weekly for POTS
- Dec 2019: BMI 19

2020 Further Medicalisation

- 2020: 6 admissions

- Barium swallow: Tertiary contractions mid to distal oesophagus…oesophageal dysmotility
- Feb 2020: BMI 21
- Jul 2020: Struggling to keep up with fluid requirements via PEG. Changed to PEGJ

- Infusaport arranged privately Sep 2020 to help with iv fluid for POTS

2021 Deterioration

- 2021: 8 admissions

- Seeing private dietitian
- Mar 2021: BMI 18
- Jul 2021: BMI 17

- Aug-Sep 2021: admissions to SCGH, RPH, FSH with infusaport infections and problems with PEGJ
- Sep 2021: BMI 14. Increasingly intolerant to feed. Admitted to FSH

Long Admission to FSH Sept-Oct 2021

- Patient intolerant to all types of feed including elemental feed
- Blind trial of feeds at different rates unsuccessful
- Started HPN; trained and discharged

- TPN 7/7 2L bag (1L olimel N9 + K to 90mmol, TE + MV + 1000ml 5% glucose) - provides 5257kJ, 56.9g amino acids, 158.2g glucose, 36.5mmol Na, 90mmol K, 15mmol Po4
- Emphasise that main aim is to reduce reliance of TPN and work towards oral and jej feeding

Osteoporosis

- Feb 2021 Bone density normal fem neck and hip. Osteopenia spine
- Used prednisolone extensively on and off during 2021. Reason not clear; related to Mast Cell Activation Syndrome, Allergies, asthma-like symptoms

- Jan 2022: Fine on TPN; BMI 17
- Feb 2022: Compression fractures with severe osteoporosis; L1, L2, L3
- Vit D and Calcium levels monitored. All been normal until Dec 2021

Hypermobile-type EDS (hEDS)

- EDS a clinically and genetically heterogeneous group of disorders resulting from gene mutations causing collagen abnormalities
- 13 types. hEDS most common. Diagnosis is clinical; no genetic test
- Prevalence increasing:
  - 1:5000 Belgium 2012: The Ehlers–Danlos syndrome, a disorder with many faces. Clinical Genetics
  - 1:500 Wales 2019: Diagnosed prevalence of Ehlers-Danlos syndrome and hypermobility spectrum disorder in Wales, UK: a national electronic cohort study and case-control comparison. BMJ Open

Gastrointestinal Issues in hEDS

Cleveland study n=218*

- 135 at least one GI symptom. 34 confirmed dysmotility
- 18 gastroparesis
- 10 tube feeding (56%)
- 5 TPN (27%)

FSH IF Group hEDS with gastroparesis:

- n=30
- 12 tube feeding (40%)
- 5 TPN/PS (16%)

*Alomari M, et al. Prevalence and Predictors of Gastrointestinal Dysmotility in Patients with hEDS: A Tertiary Care Center Experience. Cureus. 2020

hEDS & Mental Health

- Increased prevalence of GI symptoms and coexisting psychiatric disorders among patients with hEDS, reaching 62.2%
- POTS was found to be an independent predictive factor for gut dysmotility
- hEDS patients are more likely to have eating disorders and more likely to be unemployed*

*Gastrointestinal and eating problems in women with EDS. Eat Weight Disord. 2021

Discussion

- Transition from oral EN (Jan 2019) to PN (Sep 2021)
  - Timing
  - Indication
- Osteoporosis
  - Longstanding malnutrition
  - Almost normal DEXA
- hEDS
  - Consider more aggressive nutritional intervention