Impaired splanchnic perfusion – detected (?) neglected (?)

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Does anybody really care?
The gut in shock - „motor“ of multiple organ failure?

• **Architecture of the villi** *(O Lundgren, Life Sci, 1978)*
  - countercurrent exchange
  - "plasma skimming"

  - disproportionally higher compared to other beds

• **higher “critical” DO₂** *(DP Nelson, J Appl Physiol, 1988)*
  - particularly in sepsis

• **Translocation of bacteria and toxins** *(EA Deitch, Arch Surg, 1990)*
  - stimulation of innate immune cells
  - release of inflammatory mediators
Hepatosplanchnic perfusion
serial and parallel resistors..... Reilly et al, Shock 2001
Physiology of hepatosplanchnic perfusion

- $Q_{spl}$ 20-25% of CO
- $O_{2spl}$ extr. 25-30%
- ShvO$_2$ lower than SvO$_2$
Hepatosplanchnic perfusion

serial and parallel resistors..... Reilly et al, Shock 2001
Perfusion of the villi

Ischemia of villus tip via:

• “Countercurrent exchange”
• arteriolar vasomotion
• plasma-”skimming” (Fahraeus effect)

potential loss of barriere function
translocation of PAMPs, bacteria, fungi
The gut in shock - „motor“ of multiple organ failure?

• **Architecture of the villi** (*O Lundgren, Life Sci, 1978*)
  - countercurrent exchange
  "plasma skimming"

• **Vasoconstriction in the hepato-splanchnic bed**
  - disproportionally higher compared to other beds

• **higher “critical” DO₂** (*DP Nelson, J Appl Physiol, 1988*)
  - particularly in sepsis

• **Translocation of bacteria and toxins**
  (*EA Deitch, Arch Surg, 1990*)
  - stimulation of innate immune cells
  - release of inflammatory mediators
Hypovolemia in healthy volunteers
specific vasoregulatory effects in different vascular beds

Persisting vasoconstriction despite volume replacement!

AR Edouard et al.,
Intensive Care Med, 1994
The gut in shock - „motor“ of multiple organ failure?

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Critical $\text{DO}_2$ of the splanchnic bed

*Perry et al. Am J Physiol 1982*

Thus, high extraction rate, but also high $O_2\text{crit.}$ !
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Splanchnic ischemia – a classical vicious circle

Chieveley-Williams S and Hamilton-Davies C, IAC 1999; 37:81
Fungaemia and Funguria After Oral Administration Of Candida albicans

* „a self experience“:
Oral uptake of $10^{12}$ Candida albicans
after 2 hours: felt sick
after 3, 7, 9 hours fever, chills
after 3 and 6 hours positive blood culture
after 3 hours urine positive for Candida albicans

W. Krause*, H. Matheis, K. Wulf
Lancet 1969, 1: 598-599
Bacterial Translocation
a historic perspective

• **1891 A. Fränkel:** Über peritoneale Infektion.
  Wien Klin Wochenschr: 4, 265-285

• **1928 L. Arnold:** Passage of bacteria through the intestinal mucosa

• **1950 F.B. Scheinburg:** Transmural migration of intestinal bacteria.

• **1954 F.B. Scheinburg:** The bacterial factor in experimental hemorrhagic shock.
  Am J Physiol: 179, 532-540

• **1960 H.A. Ravin:** On the absorption of bacterial endotoxin from the gastro-intestinal tract of the normal and shocked animal.

• **1979 R.A. Berg:** Translocation of certain indigenous bacteria from the gastro-intestinal tract to the mesenteric lymph nodes and other organs in a gnotobiotic mouse model.
  Infect Immun: 23, 403-411
"Translocation": Myth or reality?

\[ \beta\text{-glucan [pg/ml]} \]

CABG PO day 1

Sept. shock
day 1 day 2

Control

„Translocation“: Myth or reality?

LPS log [pg/ml]

Peptidoglukan log [pg/ml]

CABG day 1

Sept. Shock day 1  day 3

control

Does anybody really care?
How to assess (and target) perfusion of the gut?

Hesselvik JF, et al., Crit Care Med, 1989
Meadows D, et al., Crit Care Med, 1988
Desjars Ph, et al., Crit Care Med, 1987

Martin C, et al., Crit Care Med, 1990
Desjars Ph, et al., Crit Care Med, 1989
The splanchnic circulation
Options for monitoring

- Intraabdominal pressure
- Gut tonometry
  - NaCl, buffer solutions
  - Air (Tonocap), CO₂-sensor (Paratrend 7)
- Blood flow, oxygen availability
  - ICG (Fick’s principle, ICG extraction)
  - S_vspO₂, regionae DO₂ and VO₂
- Ultra sound-Doppler-flowmetry
  - blood flow in hepatic veins (TEE)
- Laser-Doppler-Flowmetry
  - Mukosa e.g. of the gut
- Remission-spectrophotometry (EMPHO)
- Global liver excretory/metabolic function
  - PDR ICG
  - Lidocain metabolism (MEGX-test)
The splanchnic circulation
therapeutic concepts and options

• **Optimization of blood flow and oxygenation**
  - Optimizing volume status (PiCCO)
  - Inotropics (catecholamines)
  - Epidural anesthesia

• **Nutritional support**
  - early enteral feeding
  - Glutamine, ω-3 fatty acids, trace elements

• **Antioxidants**
  - vitamines
  - N-acetylcysteine

• **Other vasoactive agents**
  - Prostacyclin and stabile analogues

Why bother?
Why bother?


** p < 0.05

n=50 pts.

RCT
Why bother?

NAC – beyond paracetamol intoxication…..

Practical Monitoring: \( \text{PDR}_{\text{ICG}} \)
Monitoring of \( P_{\text{ICG}} \)

Options and limitations

Indocyanine green (ICG): anionic dye
Absorption maximum: 805 nm
strictly hepatic elimination (no entero-hepatic recirc.)
Elimination depends on:
- \( \text{CO} \)
  - sinusoidal perfusion
  - excretory function

Normal value: 18-25%/min
How to interpret PDR$_{ICG}$
Perfusion versus hepatobiliary transport
Peridural anesthesia to improve Splanchnic blood flow

Kortgen et al. EJA Feb 2009
The splanchnic circulation
Confounders and impact on monitoring

**Correlation IAP - PDR**

\[ y = -0.8813x + 13.34 \]

\[ R^2 = 0.6131 \]

Manu Malbrain, Antwerp
Why measure PDR-ICG?

Liver blood flow = \( \frac{\text{Extraction}}{\text{Clearance}} \) of ICG

Hepatosplanchnicus - „The canary of the body“
Why measure PDR-ICG?

PDR$_{ICG}$ (AUC 0.81, p=0.006)

Kortgen et al. Shock 2009
Impaired cardiovascular function?

Hypodynamic shock?

IAP?

Impaired hepatosplanchnic perfusion?

Response to EGDT?

Option to target splanchnic perfusion selectively?