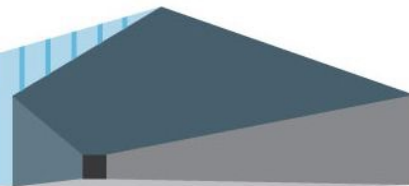


# pCO<sub>2</sub> gap

František Duška

Charles University, 3rd Fac Med, FNKV University Hospital in Prague,  
Czech Republic



# Objectives

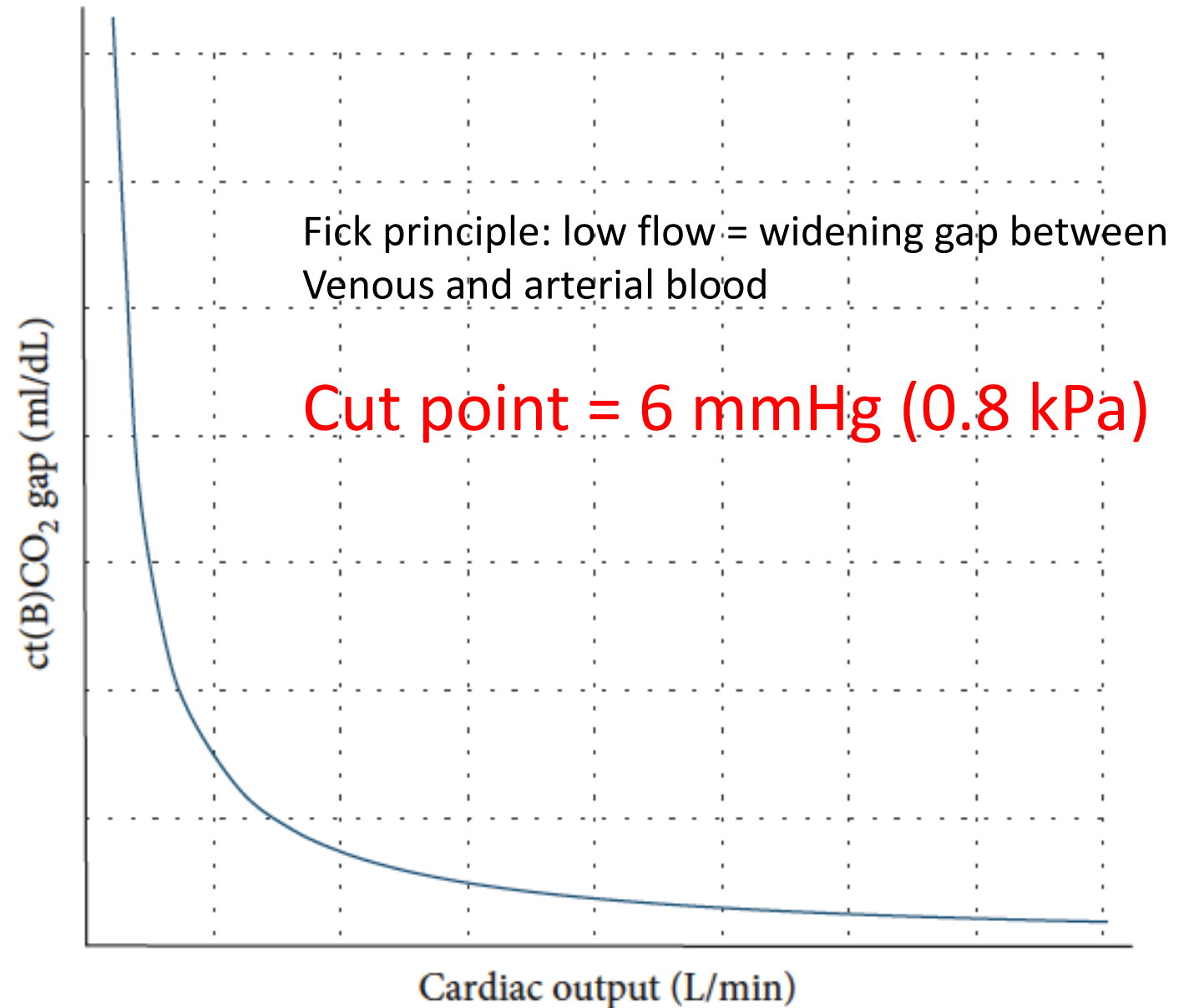
- Physiology
- Experimental data
- Clinical data
- Everyday use

# The Case

- Elderly man with good performance status
- Admitted after elective Whipples pro ampuloma
- Complicated procedure, 2L blood loss, multiple Pringles maneuvers
  
- O/E: A + B stable, major concern is lac 2.8...7.9mM and raising noradrenaline requirements (0.8 ug/ml), UO 60ml/min, echo: hypercontractile LV, PLRT negative, given 2xfluid challenges no effect
- CRT normal, ScvO<sub>2</sub> 71.3%, Hb 121 g/L

# Back to basics

- Clinical signs
- Waveforms
- Echo + monitors
- Lactate
- Fick-principle derived indices
  - ScvO<sub>2</sub>
  - $p\text{CO}_2$  gap =  $P_{\text{cvCO}_2} - P_{\text{aCO}_2}$

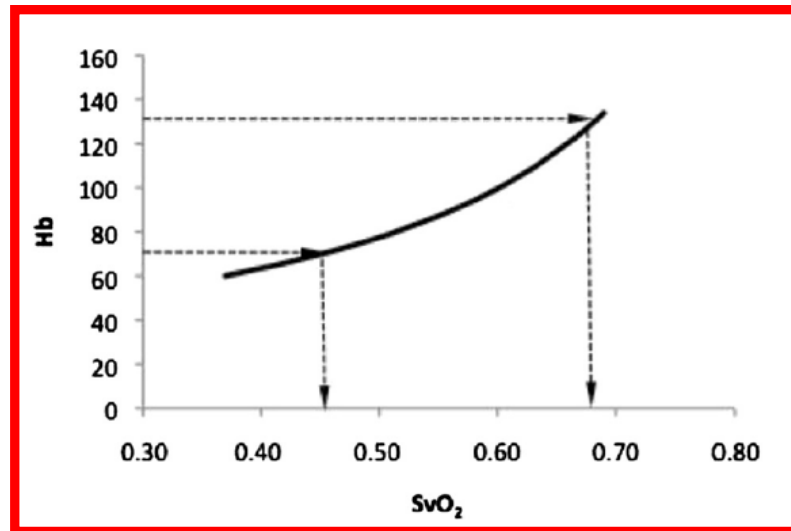
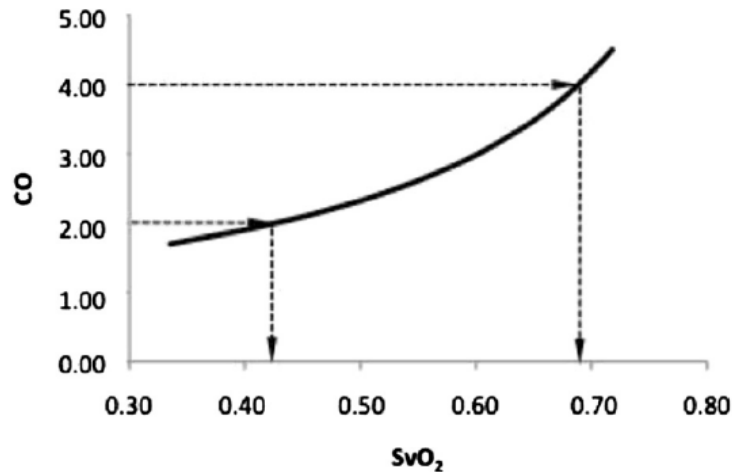
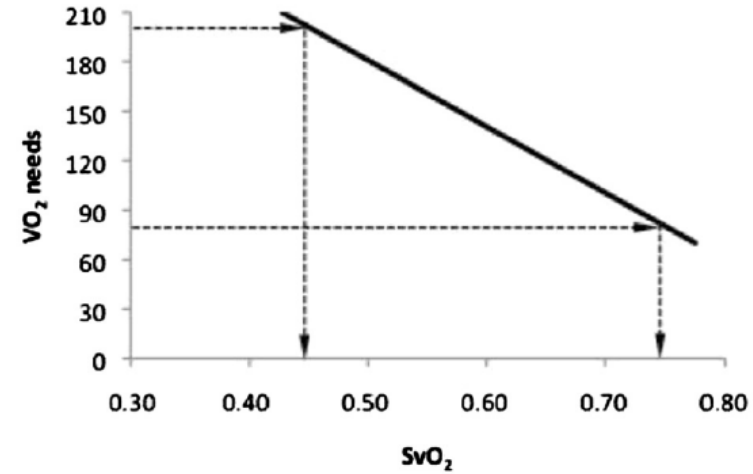
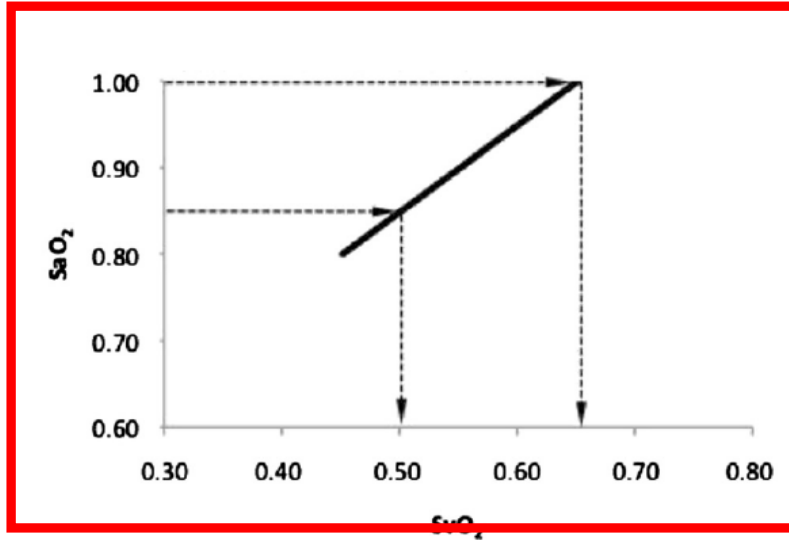


REVIEW

Central venous oxygenation: when physiology explains apparent discrepancies

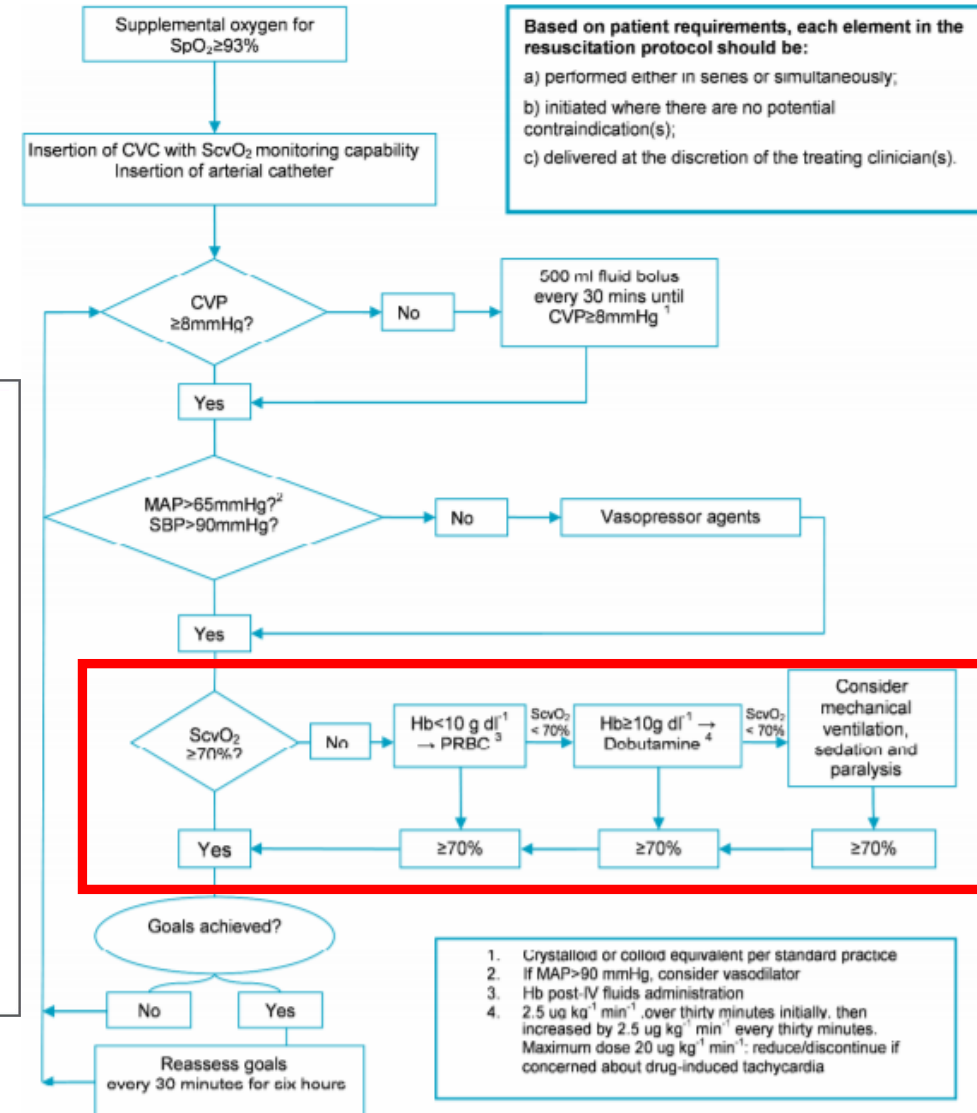
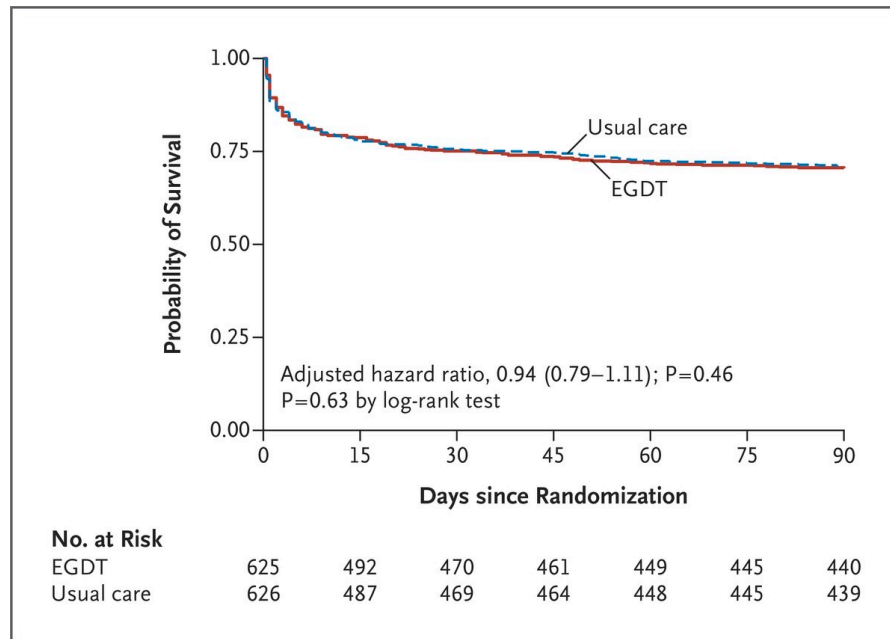
Pierre Squara

ScvO<sub>2</sub> will depend on SaO<sub>2</sub>, Hb and in situations where an O<sub>2</sub> diffusion barrier exists (e.g. non-functional and obliterated capillaries)



## Trial of Early, Goal-Directed Resuscitation for Septic Shock

Paul R. Mouncey, M.Sc., Tiffany M. Osborn, M.D., G. Sarah Power, M.Sc., David A. Harrison, Ph.D., M. Zia Sadique, Ph.D., Richard D. Grieve, Ph.D., Rahi Jahan, B.A., Sheila E. Harvey, Ph.D., Derek Bell, M.D., Julian F. Bion, M.D., Timothy J. Coats, M.D., Mervyn Singer, M.D., J. Duncan Young, D.M., and Kathryn M. Rowan, Ph.D., for the ProMISE Trial Investigators\*



In patients with septic shock who were identified early and received intravenous antibiotics and adequate fluid resuscitation, hemodynamic management according to a strict EGDT protocol did not lead to an improvement in outcome.

Can  $p\text{CO}_2$  gap outperform  $\text{ScvO}_2$   
in detecting low flow states?

# Physiology

- CO<sub>2</sub> production
  - Aerobic = decarboxylation Krebs cycle
  - Anaerobic = transient if ↓pH "Coca-cola effect"
- RQ =  $V_{CO_2}/V_{O_2}$ 
  - Normally depends on substrates oxidised
  - Oxygen limitation will also stop aerobic CO<sub>2</sub> production, but RQ will rise above one due to Coca Cola effect

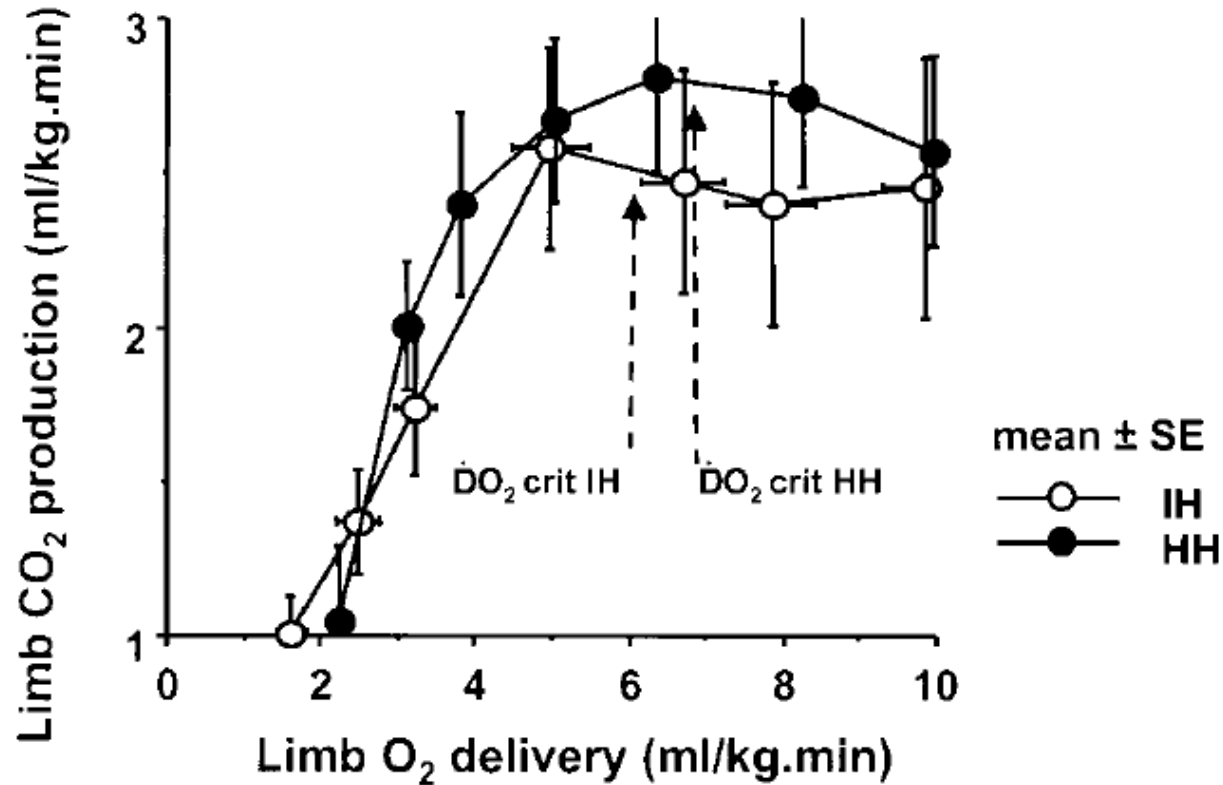




# Venoarterial CO<sub>2</sub> difference during regional ischemic or hypoxic hypoxia

*J Appl Physiol*  
89: 1317–1321, 2000.

BENOIT VALLET,<sup>1</sup> JEAN-LOUIS TEBOUL,<sup>2</sup> STEPHEN CAIN,<sup>3</sup> AND SCOTT CURTIS<sup>4</sup>

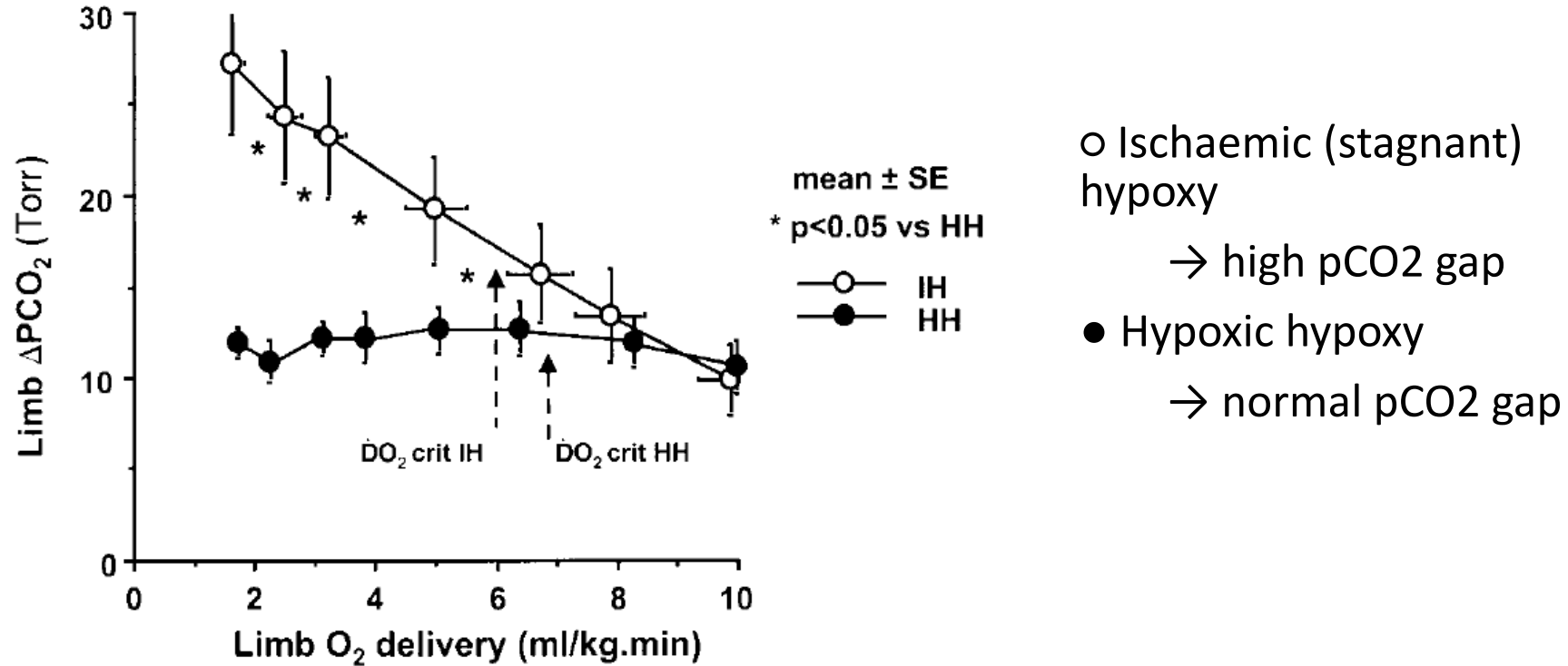


Any type of tissue hypoxia will reduce CO<sub>2</sub> production by the tissue (along with the reduction of oxygen use)

# Venoarterial CO<sub>2</sub> difference during regional ischemic or hypoxic hypoxia

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BENOIT VALLET,<sup>1</sup> JEAN-LOUIS TEBOUL,<sup>2</sup> STEPHEN CAIN,<sup>3</sup> AND SCOTT CURTIS<sup>4</sup>



... pCO<sub>2</sub> gap = PcvCO<sub>2</sub> – PaCO<sub>2</sub>

Is a marker of **tissue blood flow, but not tissue hypoxia!**

Signs of circulatory shock  
Lactic acidosis

$S(c)vO_2 < 70\%$

Pv-a  $CO_2$  gap

$> 6$  mmHg

$< 6$  mmHg

- Low cardiac output
- Stagnant dysoxia

- Anemic dysoxia
- Hypoxic dysoxia

$S(c)vO_2 > 70\%$

Pv-a  $CO_2$  gap

$> 6$ mmHg

$< 6$  mmHg

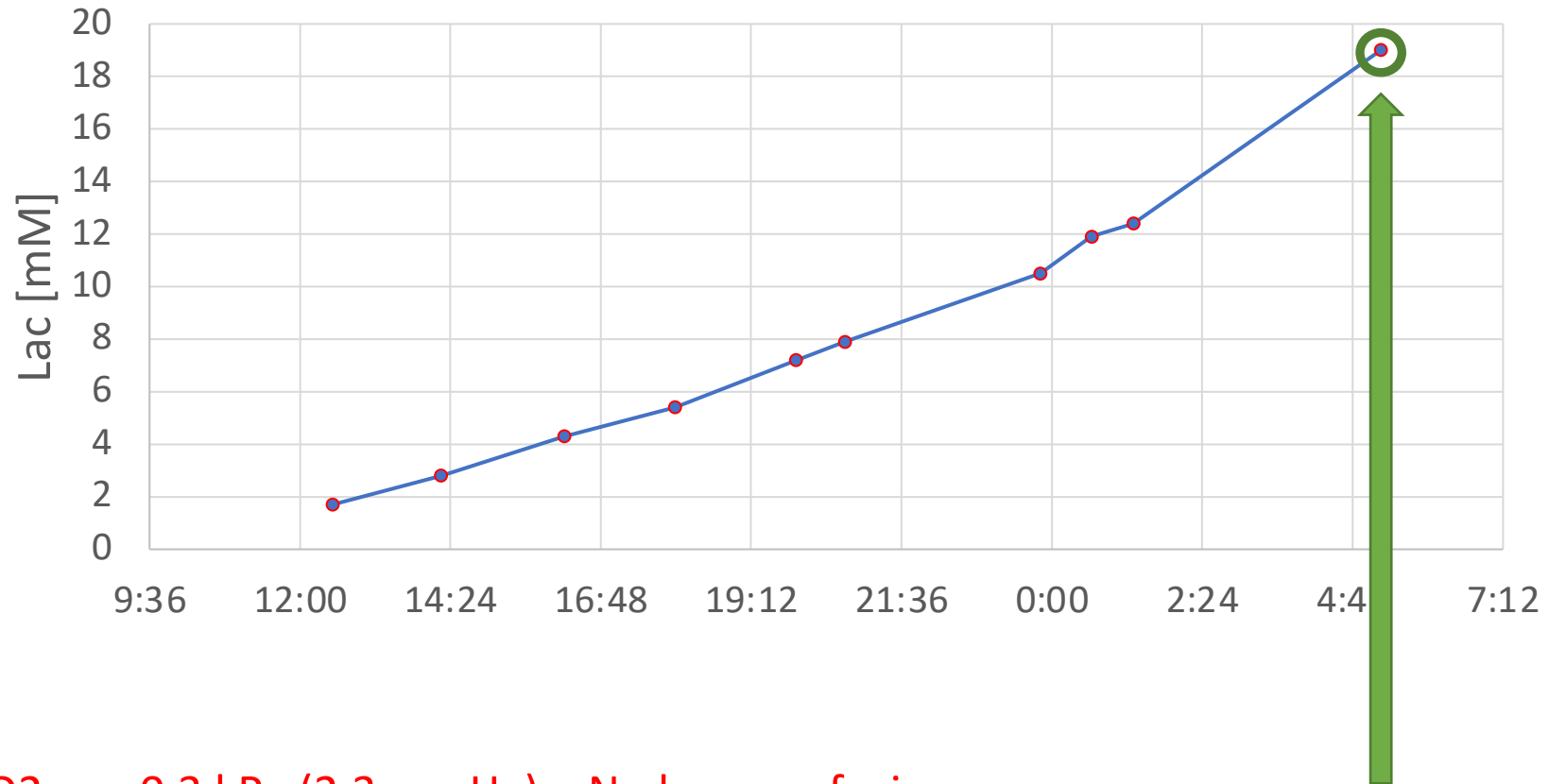
- Microcirculatory impairment
- Inadapted cardiac output
- Stagnant dysoxia

- Cytopathic hypoxia

# The Case

- Elderly man with good performance status
- Admitted after elective Whipples pro ampuloma
- Complicated procedure, 2L blood loss, multiple Pringles maneuvers
- O/E: A + B stable, major concern is lac 2.8...7.9mM and raising noradrenaline requirements (0.8 ug/ml), UO 60ml/min, echo: hypercontractile LV, PLRT negative, given 2xfluid challenges no effect
- CRT normal, ScvO<sub>2</sub> 71.3%, Hb 121 g/L
- pCO<sub>2</sub> gap 0.3 kPa (2.3 mmHg) = no signs of tissue hypo

	OR		ICU							
Time	12:31	14:15	16:13	17:59	19:55	20:42	23:49	0:38	1:18	5:15
Laktát	1.7	2.8	4.3	5.4	7.2	7.9	10.5	11.9	12.4	19



pCO<sub>2</sub> gap 0.3 kPa (2.3 mmHg) = No hypoperfusion

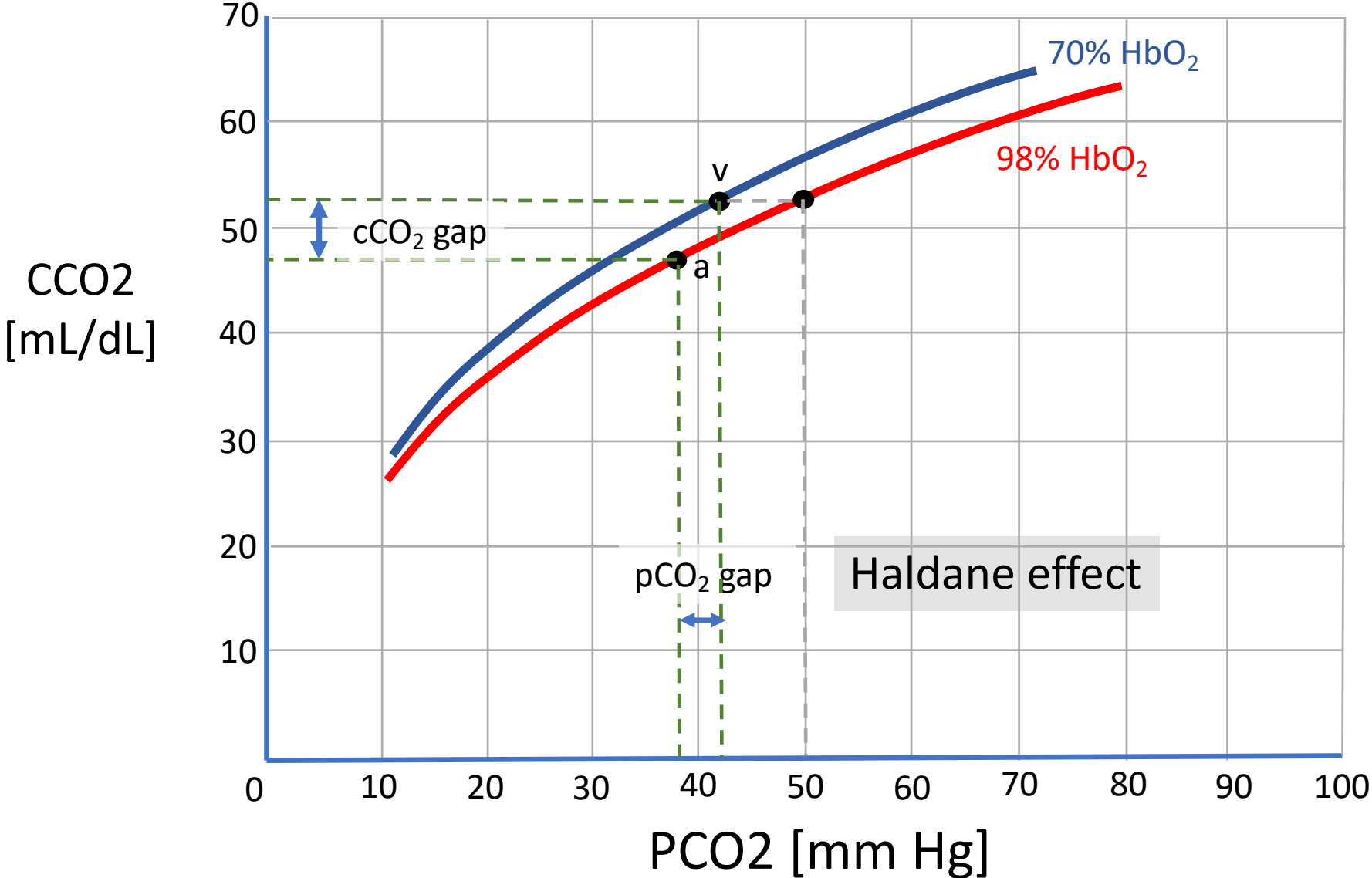
Cause was fulminant liver failure with reduced lactate clearance

# What $p\text{CO}_2$ gap adds to $\text{ScvO}_2$ measurement?

- Helps to detect patients who might benefit from increasing cardiac output despite having  $\text{ScvO}_2 > 70\%$  (eg. microcirculatory shunts)
- Help to diagnose "nonstagnant hyperlactatemia" and avoid futile fluid boluses

Pitfalls of pCO<sub>2</sub> use

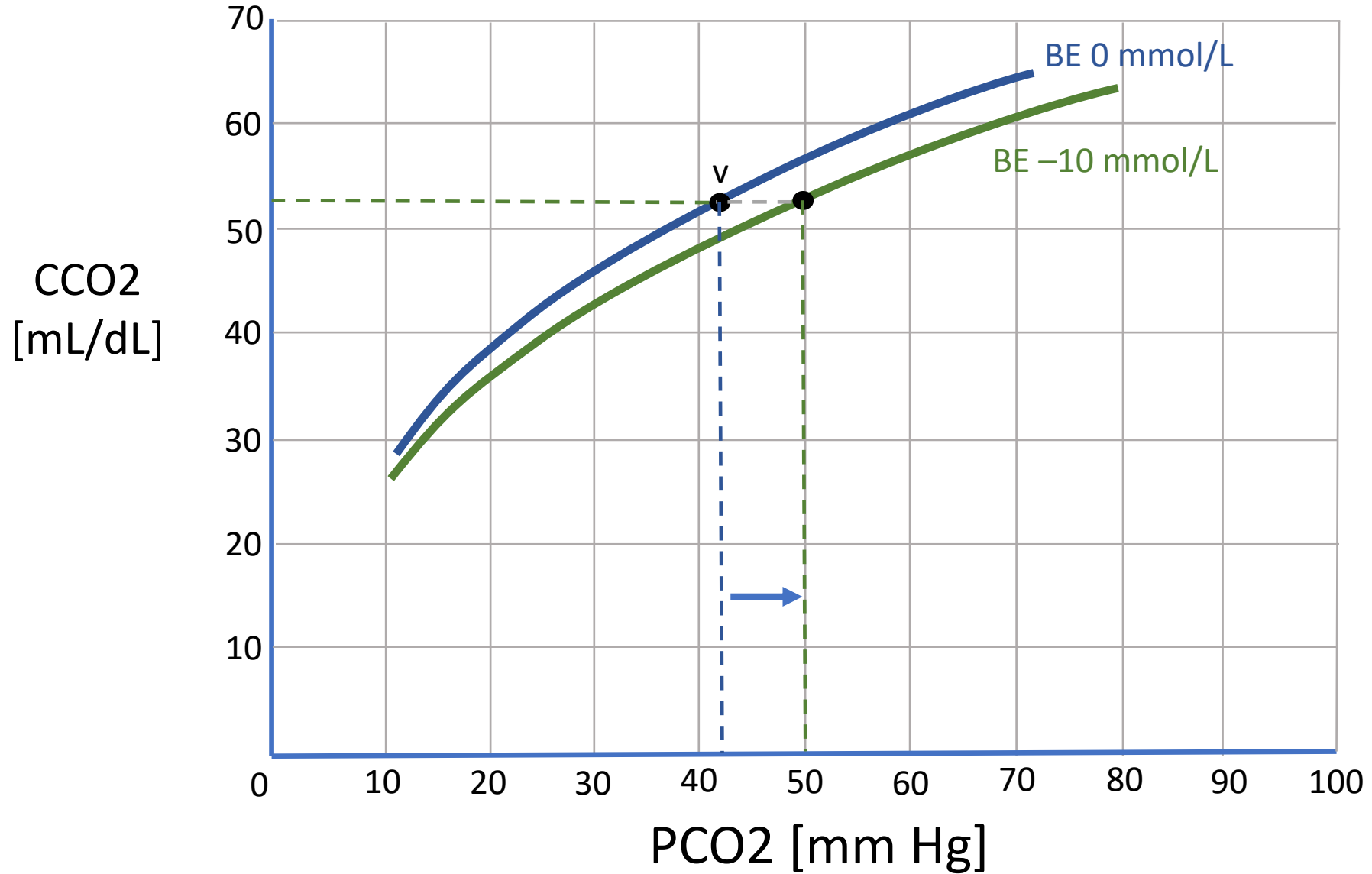
# Haldane effect: oxygenation of Hb enhances CO2 release





# Coca-cola effect

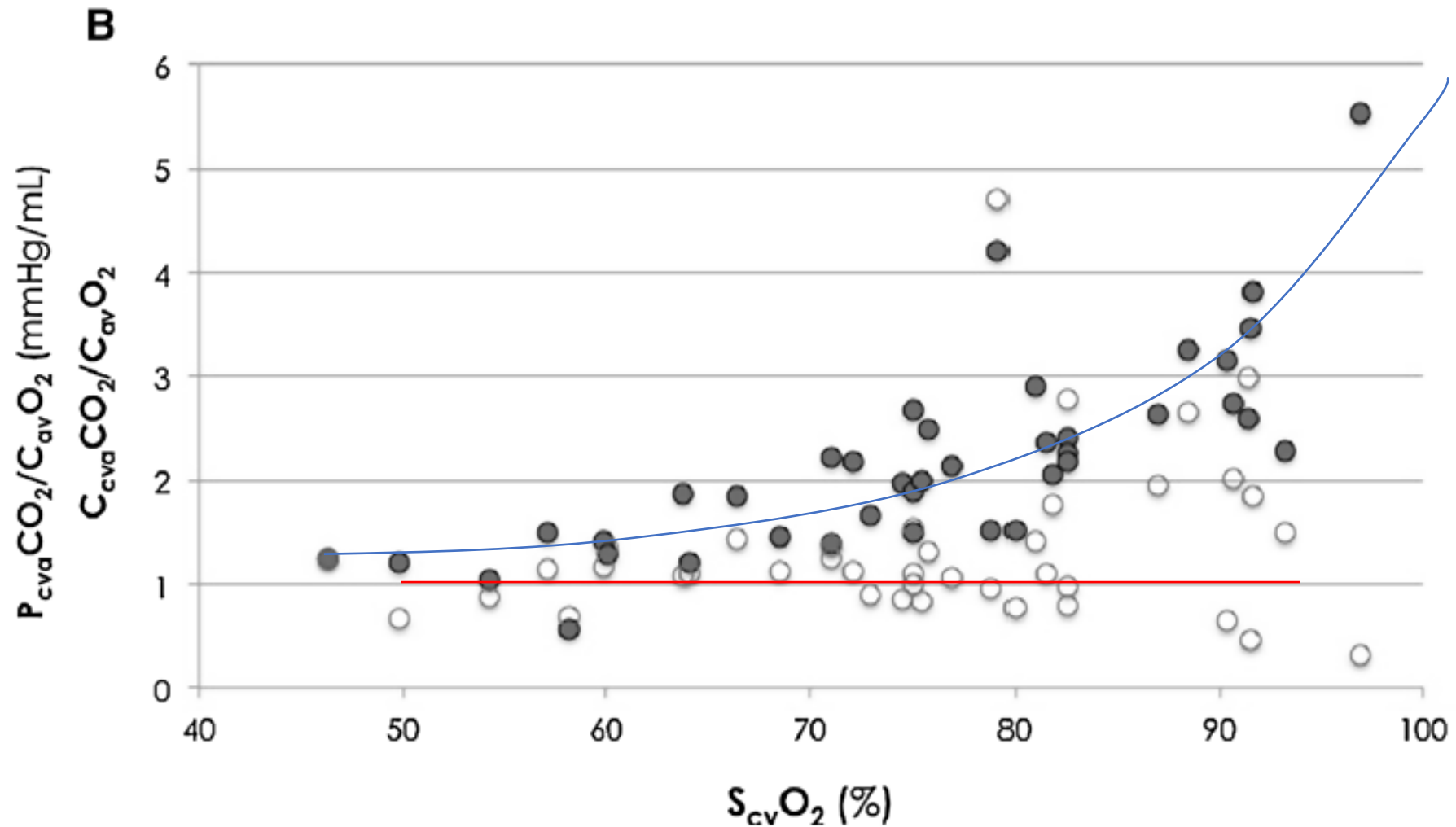
Tissue acidosis enhances CO<sub>2</sub> release



# Pitfalls

- $p\text{CO}_2$  gap is a surrogate for  $\text{CCO}_2$  gap and relation of  $p\text{CO}_2$  and  $\text{CCO}_2$  is not strictly linear
- Normally deoxygenation and mild acidification of the blood in tissues cancel out each other and  $p\text{CO}_2$  vs.  $\text{CCO}_2$  relation remains linear, but hypoxemia (e.g.  $\text{FiO}_2$ ) or respiratory alkalosis might cause false  $p\text{CO}_2$  elevation

For constant flow, central venous hyperoxemia increases CO<sub>2</sub> gap



(Saludes et al. 2016)

# Conclusions

- pCO<sub>2</sub> gap can help in the differential diagnosis of shock and/or hyperlactatemia
- Mind the gap before giving fluid for elevated lactate
  - pCO<sub>2</sub> gap >6 mmHg (0.8 kPa) seems to be a good marker of tissue hypoperfusion (fluids may help)
  - pCO<sub>2</sub> gap remains normal in anemic, hypoxic and cytopatic hypoxia (fluids will not help)
- Interpretation should be cautious in hyperoxemia or respiratory alkalosis, which widen pCO<sub>2</sub> gap artificially
- Now we need clinical trials and look at patient-centred outcomes

Thank you



**13<sup>th</sup> ifad** International Fluid Academy Days  
Antwerp – Belgium  
#IFAD2023

**SAVE THE DATE**  
ANTWERP – 23-25 NOV 2023

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