

Not all turtles are slow.



Chrysemys picta marginata - the diving turtle

47-year-old man with shortness of breath and leg swelling for 2 weeks.

- Unemployed Berliner from a “garden colony”, (*Laubenpieper*), a term of endearment
- No meds, “denies” alcohol intake
- BP 115/60 mm Hg, HR 96/Min, RR 36/Min
- Rales bilateral
- Distant heart sounds, no murmurs, ?S3
- Bowel sounds diminished
- Edema to sacrum
- Suspect CHF

Portable chest roentgenogram.



Acute laboratory tests.

- Hb 13 g/dl, Hematocrit 38 vol%
- pH 7.30, PaCO₂ 20, PaO₂ 105 (mm Hg), HCO₃ 10 mmol/L
- Na 123, K 6.9, Cl 87 (mmol/L)
- Glucose 5.6 mmol/L
- Lactate 10 mmol/L
- Anything life threatening here?

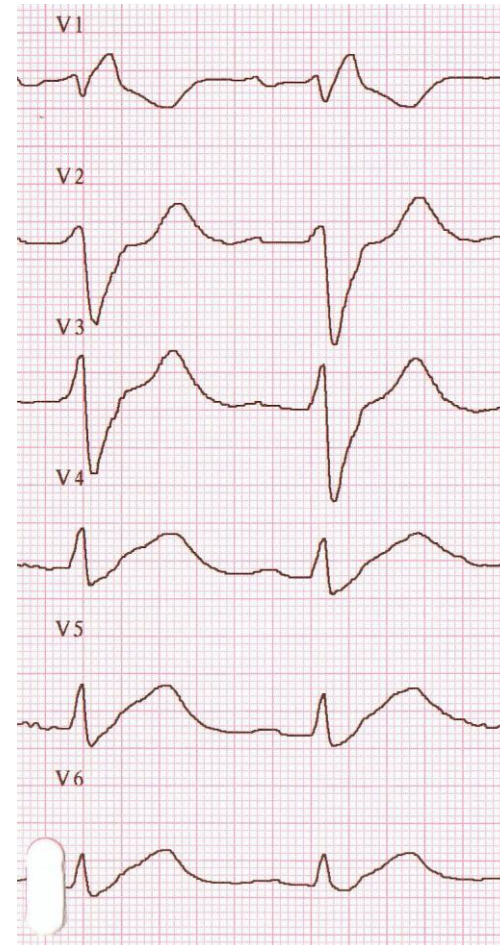
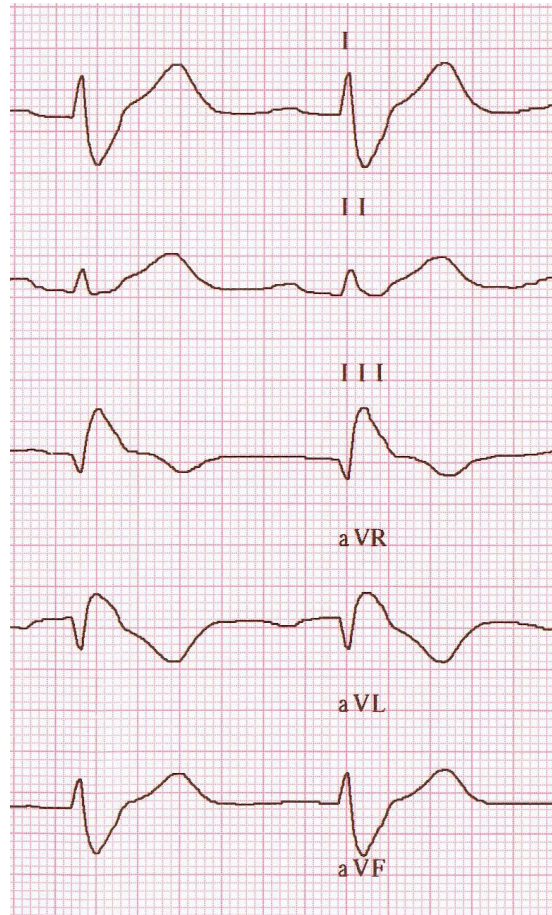
In this patient, which of the below is life threatening?

- Acid-base problem?
- Hyperkalemia?
- Poor oxygen delivery?
- Heart failure?
- Just being in our ICU?

The answer is most-likely hyperkalemia!

- Alveolar gas formula (Sea level)
- $PAO_2 = 150 - PaO_2 + 1.25(PaCO_2)$ [RQ]
- $= 150 - 105 + 25 = 20$ mm Hg
- A-a gradient modestly elevated. Thus, severe heart and lung disease probably not immediately lethal.
- How many people die because of hydrogen ion concentration, ie. a pH value?
- None!
- Is hyperkalemia lethal?

An electrocardiogram (50 mm/sec) was obtained.



Is this tracing a „hyperkalemia“ ECG?

And now for the acid-base problem!

- Is there an acid-base disturbance?
- Acidemia with a low HCO_3^- and low PaCO_2
- Metabolic Acidosis
- HCO_3^- fell by 15 mmol/L (25 to 10)
- Decrease in PaCO_2 was 20 mm Hg
- Metabolic Acidosis, *slightly* overcompensated
ie. respiratory Alkalosis

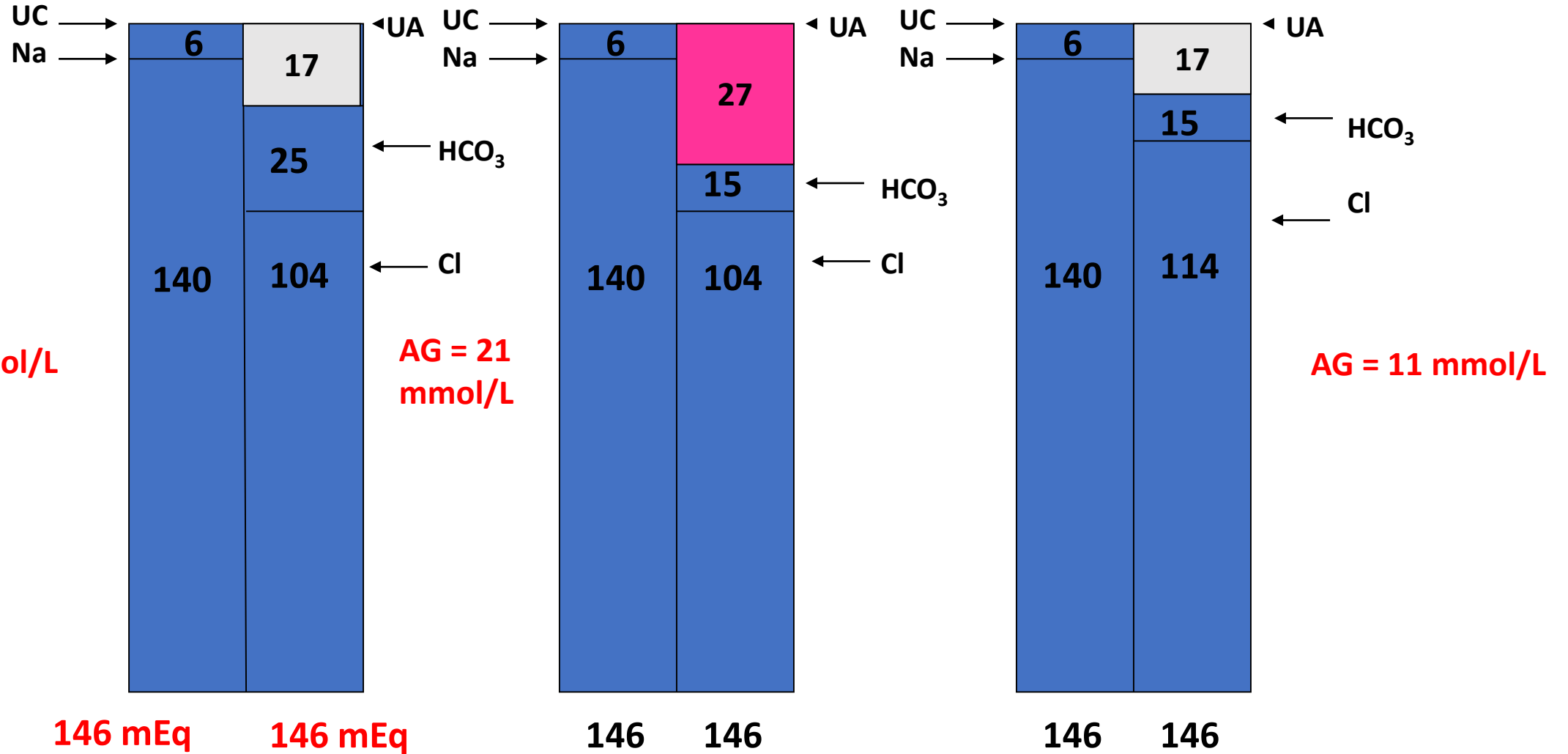
Which “Gamble”-gram fits our patient?

Metabolic acidosis with elevated or non elevated AG

Normal

High AG

High Cl



Laubenpieper has an elevated AG.

- $AG = Na - Cl - HCO_3 = 26 \text{ mmol/L}$; normal (12 ± 2)
- AG is 14 mmol/L too high
- Lactate is elevated by 10 mmol/L
- Ahah! For about every mmol/L increase in lactate we observe one mmol/L increase in AG
- (Almost) „Delta“ for „Delta“
- So, we have a pure anion-gap metabolic acidosis!

Laubenpieper arrives at our ICU.

- Leukocytes 16 000/ μ^3
- D Dimer 0,48 mg/dL, Trop T 0,05 $\mu\text{g/L}$
- aPTT 18 sec, ALAT, ASAT, Bilirubin slightly elevated
- Crea 243 $\mu\text{mol/L}$, Urea 18 mmol/L
- Osmo 287 mosm/L
- Ethanol not detectable (at present)

Mneumonics – MUDPILES or German - KUSSMAUL

- Methanol, uremia, diabetic ketoacidosis (DKA), paraldehyde, phenformin, pyroglutamic acidosis (dicloxacillin), metformin, iron, isoniazid, lactic acidosis, ethylene glycol, salicylates (English)
- Kussmaul: Ketoazidose, Urämie, Salizyl-Säure, Metanol, Äthylenglykol, (mehr) Urämie, Laktat (German)

Could the urine give useful information?

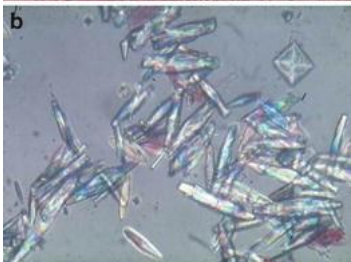
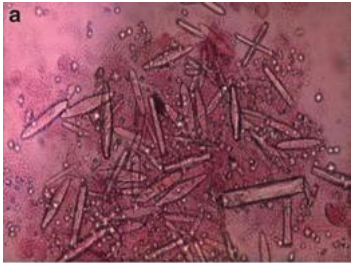


- Urine stinks and is seldom saved
 - Sediment for ethylene glycol
 - Ferric chloride for acetyl salicylic acid, ketonuria, urea
 - Lactate is 100% absorbed and does not appear in urine
 - Transtubular potassium gradient could help
 - All too true
- FEK is probably better



Can we dismiss methanol and ethylene glycol poisonings?

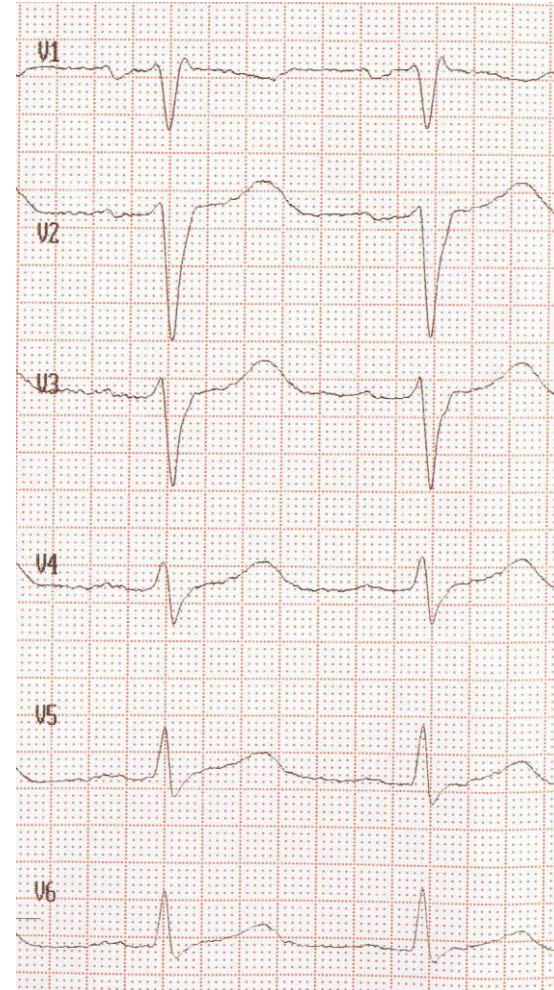
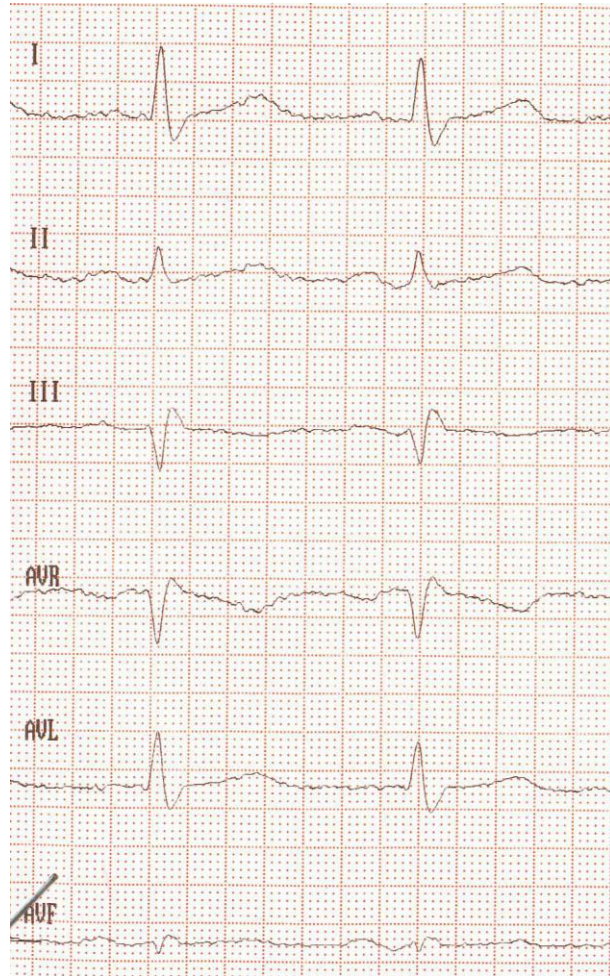
- Calculated Osmo = $2(\text{Na}+\text{K}) + \text{Glu} + \text{Urea}$
- = $2(129) + 5.6 + 19 = 283 \text{ mosm/L}$
- Difference is only 4 mosm/L
- Most common cause of increased AG metabolic acidosis today?



The urine gave us further information.

- UNa 10 mmol/L
- UCl 15 mmol/L
- UK 65 mmol/L
- Sp.Grav. 1.030 (What is Uosm??)
- Prot 2+
- Leucocytes und granulated cylinders
- Ketone not present
- Mneumonics lead us to lactate
- Prerenal acute renal failure?

Glucose and insulin did not help, but dialysis did.

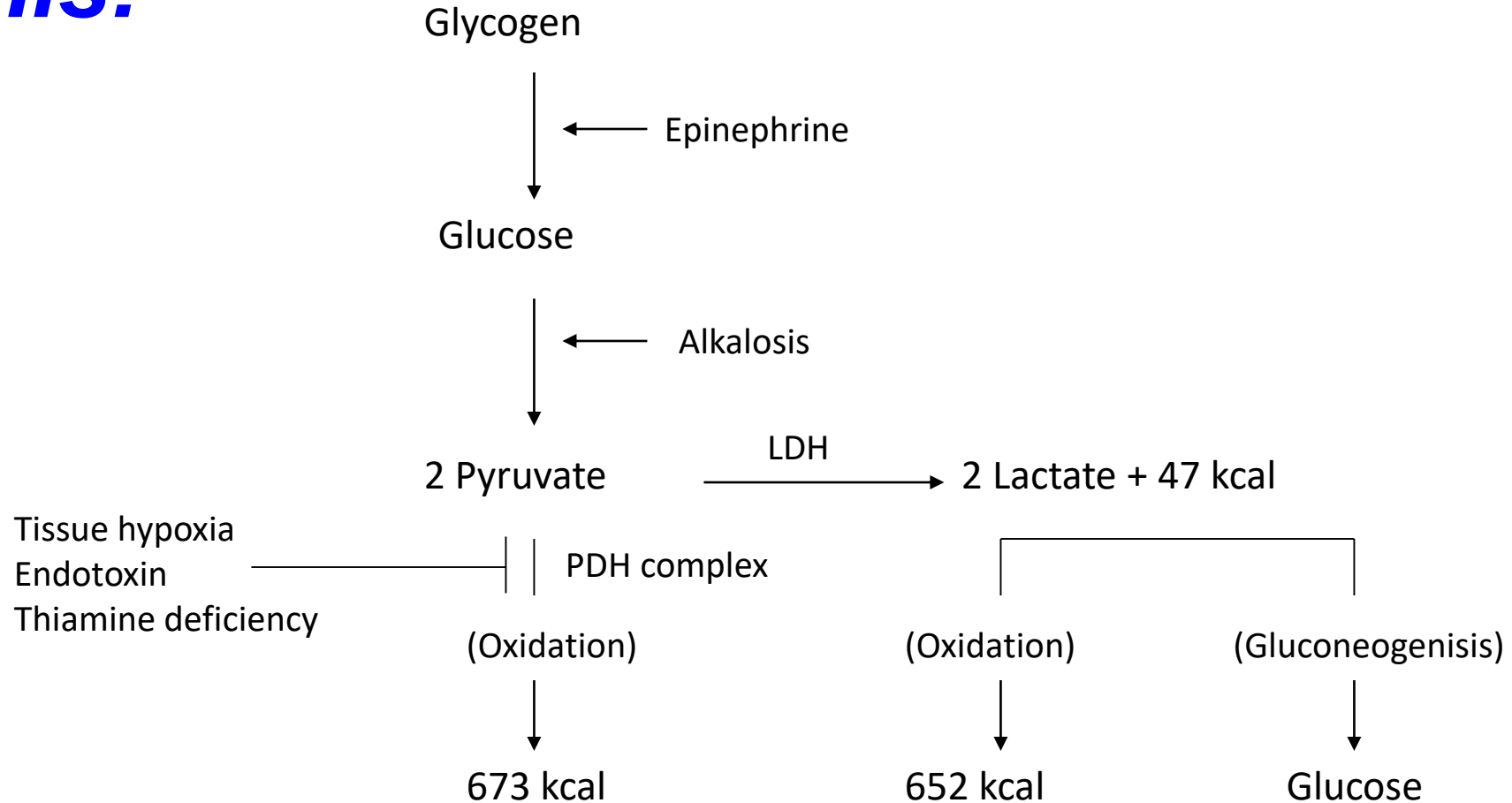


Why did insulin and glucose fail in this patient?

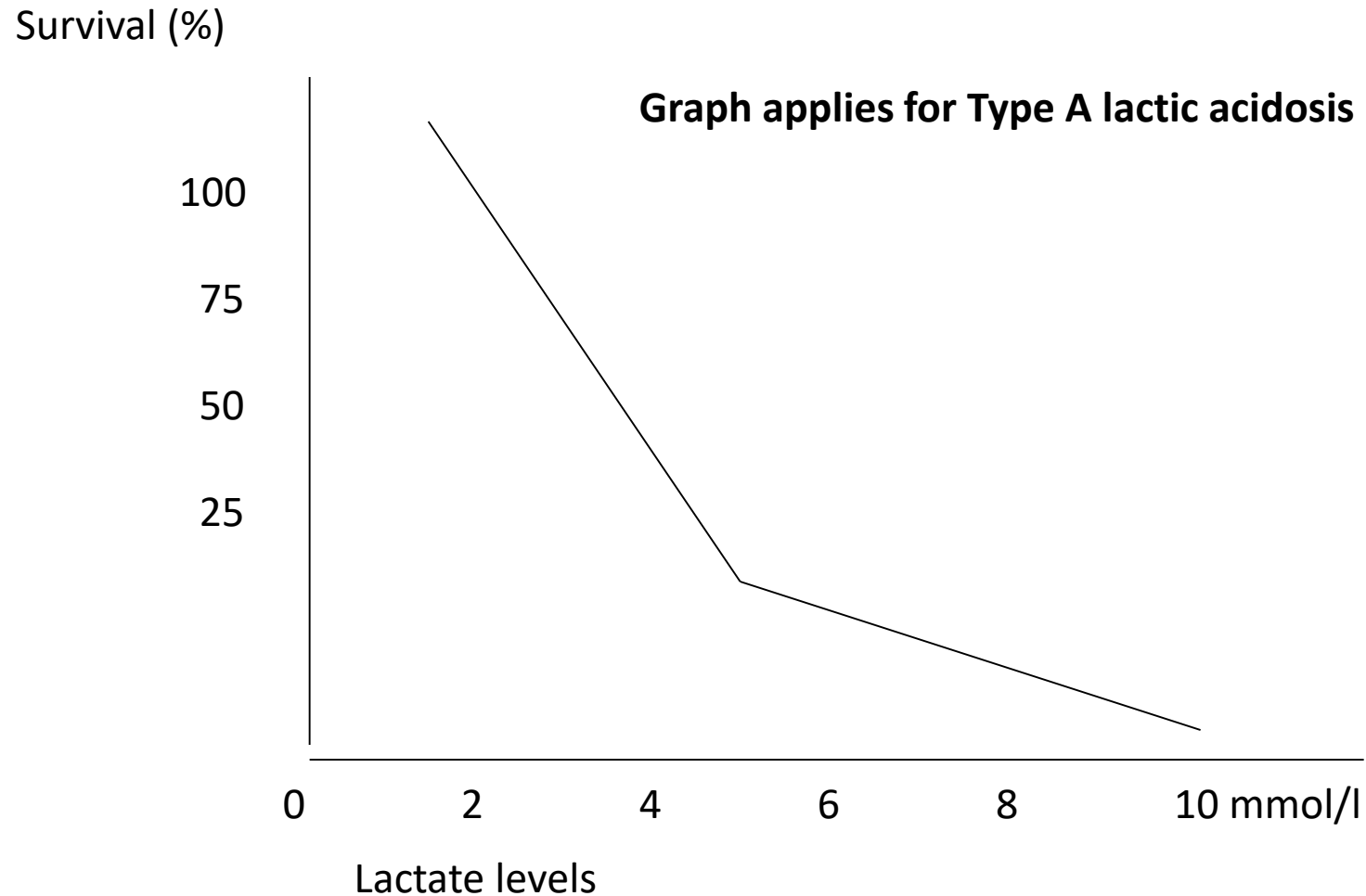


- Lactate is the end product of anaerobic glycolysis
- Production is 1 mmol/kg/h, circa 2000 mmol/day for our 80 kg Laubenpieper
- Production in skeletal muscle, GI tract, brain and erythrocytes
- The liver uses it (for gluconeogenesis) or for fuel

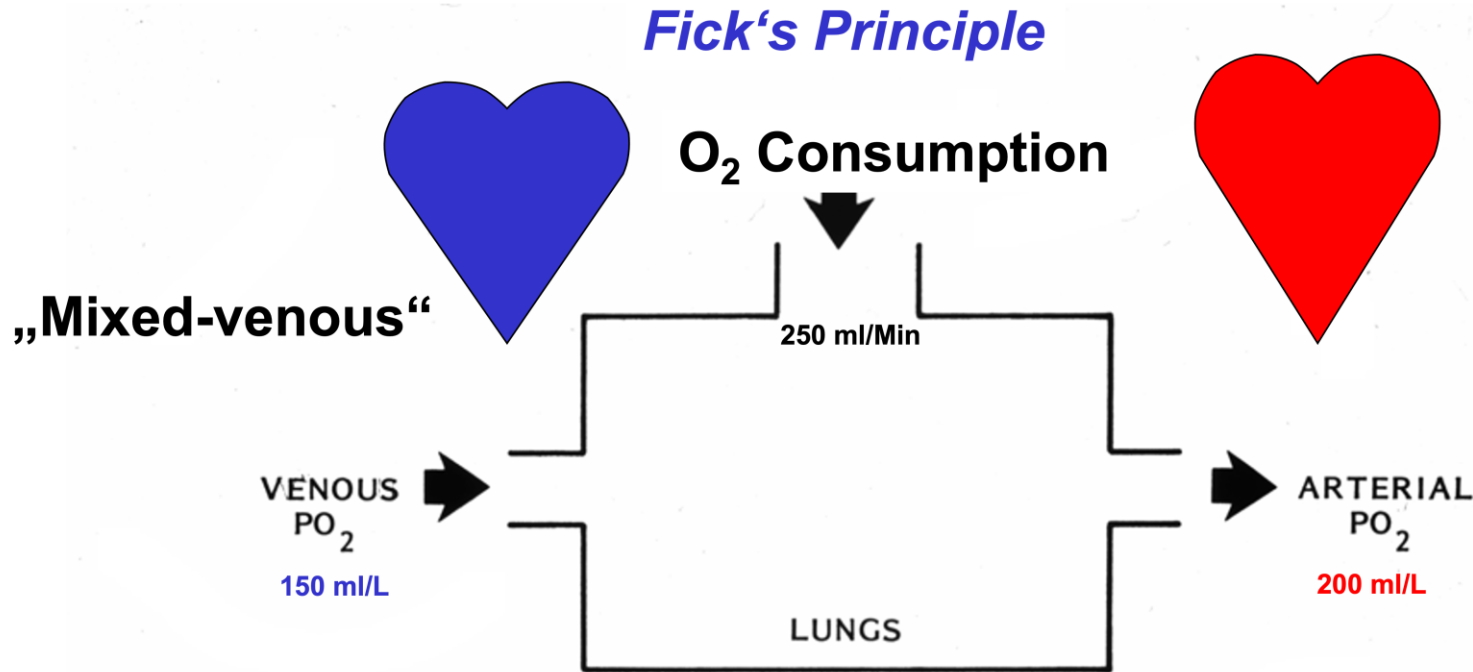
Lactate metabolism should look something like this:



Is tissue hypoxia responsible for lactic acidosis in the Laubenpieper?



In ICU, how do we measure C.O. and O₂ consumption?



$$\text{C.O. (5 L/min)} = \frac{250 \text{ ml/min}}{\text{CaO}_2 (200 \text{ ml/l}) - \text{CvO}_2 (150 \text{ ml/l})}$$

Laubenpieper was not in shock!

$$SVR = \left(\frac{MAP - CVP}{CO} \right) \times 80$$

- BP 118/46 mm Hg
- CVP 28 mm Hg
- PCWP 22 mm Hg
- CO 9 L/min
- SVR 337 dyne/cm⁻⁵

What were DO_2 , VO_2 and O_2 extraction?

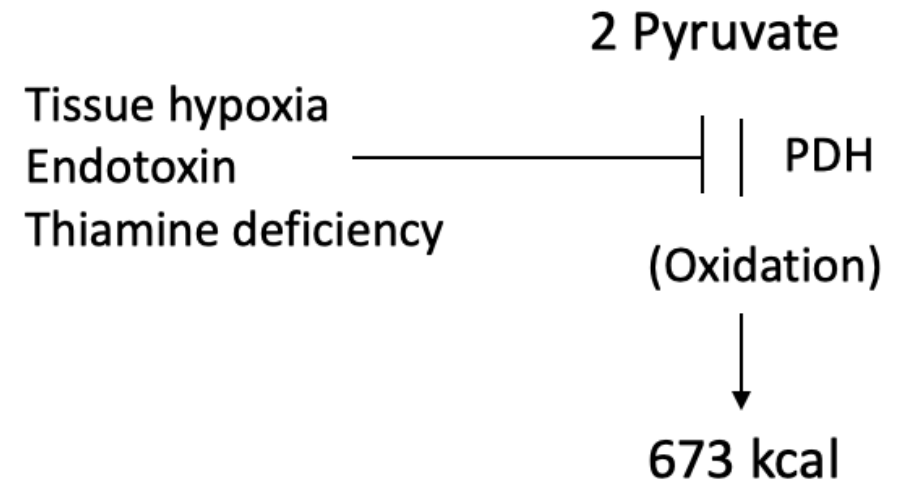
The most important reason to place a Swan-Ganz catheter is to determine oxygen balance.

Laubenpieper had high-output heart failure.

- $\text{Hb}(13 \text{ g/dL}) \times 1.34 = 17.4 \text{ ml O}_2/\text{dL}$ or 174 ml/L
- $\text{CO} = 9 \text{ L/min}$
- $\text{DO}_2 = 174 \times 9 = 1566 \text{ ml/min}$ (O_2 offered per min.)
- $\text{VO}_2 = \text{DO}_2 \times (\text{SaO}_2 - \text{SvO}_2)$
- $\text{SaO}_2 = 100\%$
- PvO_2 was 60 mm Hg; $\text{SvO}_2 = 90\%$ (40 mm Hg; 75%)
- $\text{VO}_2 = 156.6 \text{ ml/min}$
- $\text{O}_2\text{ER} = 10\%$ (normal O_2ER is 25%)
- Why does the Laubenpieper not use O_2 ?

What leads to O_2 uncoupling?

- Poisoning cytochrome C oxidase
 - Cyanide, CO
- Dinitrophenol
 - Atractyloside (a plant glycoside)
 - Bongkrekic
- Metformin
- Disturbances of PDH
- Endotoxin
- Thiamine deficiency



Who first described “Beri-beri”?



Nicholas Tulp from Leyden, 1739,
Rembrandt in the
“Anatomy demonstration”

***We gave the Laubenpieper thiamine.
(He had “wet” Beri-beri and not “dry” Beri-beri)***

- Within 12 h Lactate fell to 1 mmol/L
- pH rose to 7.4
- HCO_3 increased to 24 mmol/L
- O_2 extraction was 20%
- Cardiac function improved within 1 week
- Liver and renal function normalized
- Signed out against medical advice

Laboratory values (mmol/L) of our turtle.

Prior to hibernation

Na	117
Cl	73
HCO ₃ ⁻	39
AG	5

Lactate 4

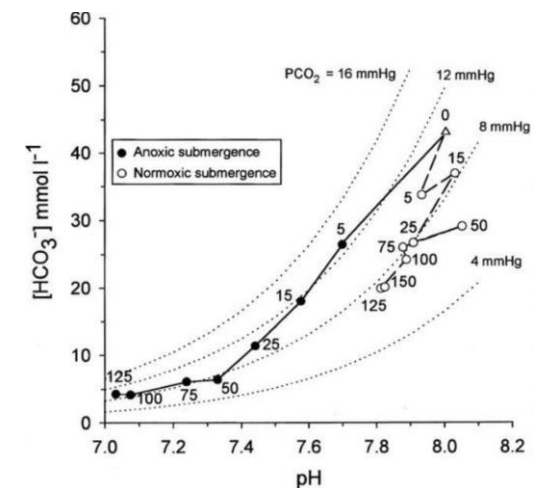
K	2
Mg	2,6
Ca	3,7

Stuck under water for 5 months

- Na⁺ 99
- Cl⁻ 44
- HCO₃⁻ 5 Δ 34
- AG⁻ 50 Δ 45

- Lactate⁻ 185 Δ 181
- K⁺ 10
- Mg²⁺ 12
- Ca²⁺ 59

Reese SA et al. The physiology of hibernation among painted turtles: the midland painted turtle (*Chrysemys picta marginata*). Respir Physiol 2001;124(1):43-50.



Teaching points:

- Acid-base problems are not acutely lethal.
- Hyperkalemia (when treated) needs insulin (and glucose) – dialysis can help.
- AG (like mosm/L) is a clinical tool; AG is not a law of nature
- Swan-Ganz allows metabolic assessments: CO, DO₂, and VO₂.
- Type B lactic acidosis.