Electrolyte and metabolic disorders in acute heart failure

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Disclosures

No disclosures/conflicts of interest

Outline

Glycaemic contol

Informed largely by ICU studies:

1. Minimise fluctuations
2. Avoid hypoglycaemia
3. Avoid hyperglycaemia
4. Trigger 10.0mmol/L (180mg/dL)
5. Relative trigger 8.3mmol/L (150mg/dL)
6. Target range?
7. Aiming <6.1mmol/L (110mg/dL) not recommended

11.9 Diabetes

Dysglycaemia and diabetes are very common in HF, and diabetes is associated with poorer functional status and worse prognosis.

Diabetes may be prevented by treatment with ARBs and possibly ACE inhibitors.[197] Beta-blockers are not contraindicated in diabetes and are as effective in improving outcome in diabetic patients as in non-diabetic individuals, although different beta-blockers may have different effects on glycaemic indices.[198] Thiazolidinediones (glitazones) cause sodium and water retention and increased risk of worsening HF and hospitalization, and should be avoided (see recommendations, Section 7.4).[131–133] Metformin is not recommended in patients with severe renal or hepatic impairment because of the risk of lactic acidosis, but is widely (and apparently safely) used in other patients with HF.[199] The safety of newer anti-diabetic drugs in HF is unknown.

Case study: diabetic patient with HF

- 63 year old female
- Known DM (Type II)
- Known hypertension
- Known HF
- Presents with
  - progressive SOB
- Previously normal renal function
- On arrival
  - Oliguric
  - Disoriented
  - Confused
- Observations:
  - RR 32 breaths/min
  - BP 76/46 mm Hg
  - HR 125 bpm (sinus)
  - Rectal temperature 36.8°C
- Cool, clammy extremities
- Reduced skin turgor

Case study

- Lab Results
- Blood glucose 9 mmol/l
- Urea 22 mmol/l
- Creatinine 779 µmol/l
- Potassium 6.8 mmol/l
- Clotting normal
- CRP <5
- Procalcitonin <0.5

- CXR Normal
- Anuric

- **Arterial blood gases**
  - pH 6.72
  - PCO_2 36 mm Hg
  - PO_2 106 mm Hg
  - HCO_3^- 12 mmol/l

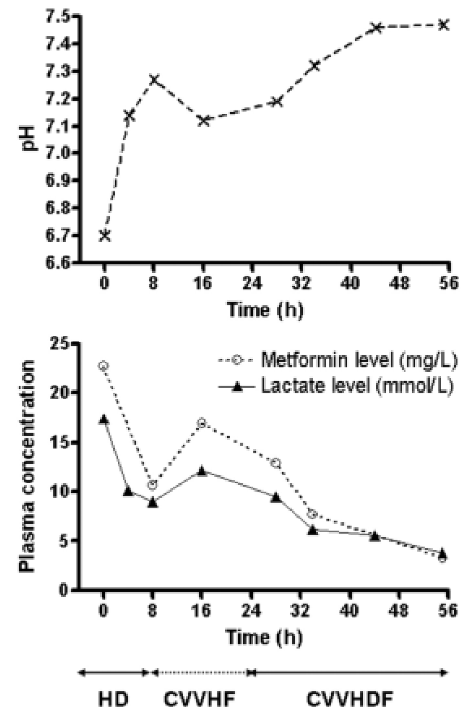
  - Anion Gap 20.3

  - Predicted bicarbonate: **Arterial $PCO_2 = 1.5 \times HCO_3 + 8 \pm 2$ (26$\underline{+}$2)**

  - **Additional information: lactate 17.4mmol/L**

Additional relevant information

- Aspirin, 75 mg
- Irbesartan 75 mg
- Furosemide
- Metformin 1g tds



Lemyze M et al. BMJ 2010;340:bmj.c857

Metformin & lactic acidosis

Protti *et al. Critical Care* 2012, **16**:R75
http://ccforum.com/content/16/3/R75

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Metformin overdose, but not lactic acidosis *per se*, inhibits oxygen consumption in pigs

Alessandro Protti[1*], Francesco Fortunato[2], Massimo Monti[1], Sarah Vecchio[3], Stefano Gatti[4], Giacomo P Comi[2], Rachele De Giuseppe[5] and Luciano Gattinoni[1]

Conclusions: Metformin intoxication induces lactic acidosis, inhibits global oxygen consumption and causes mitochondrial dysfunction in liver and other tissues. Lactic acidosis *per se* does not decrease whole-body respiration.

Mechanism Complex

- **Promotes the conversion of glucose to lactate in the splanchnic bed of the small intestine**
- **Inhibits hepatic gluconeogenesis from lactate, pyruvate, and alanine**
- **Results in increased substrate for lactate production**

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Hemodynamic consequences of severe lactic acidosis in shock states: from bench to bedside

Antoine Kimmoun[1 2 3], **Emmanuel Novy**[1 2], **Thomas Auchet**[1], **Nicolas Ducrocq**[1] and **Bruno Levy**[1 2 3 *]

- Metformin-associated lactic acidosis, even with pH values around 7.0 - observed mortality = 25%
- The same pH values during **shock-associated lactic acidosis**, regardless of origin, **no survival** was reported

"Consequently, severe lactic acidosis is much more of a precipitator than a direct causal factor of mortality. Lactic acidosis probably contributes to the decompensation of underlying comorbidities and, hence, to the mortality rate"

So what of lactic acidosis in HF?

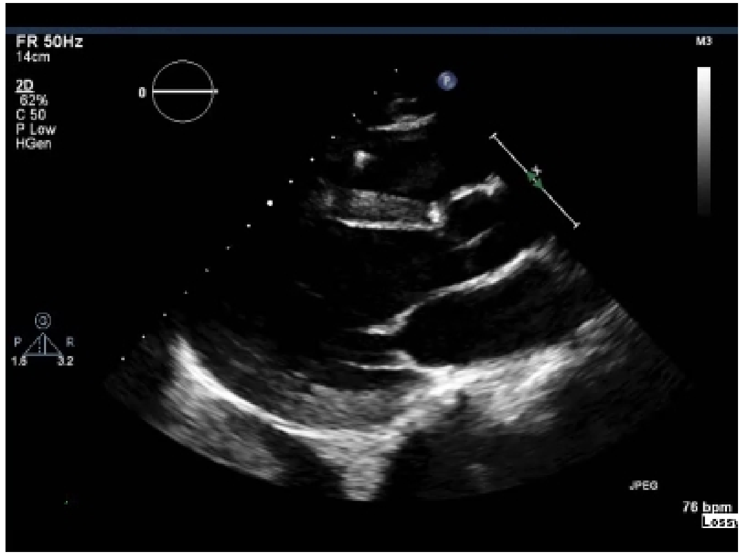


Table 2

Differences Between Groups: Mean Levels and Significance

Variable	Group	End of operation		2 hr postop		Morning of day 2	
Cardiac index (liters/min/m²)	Survivors	NS		2.18	$P < 0.05$	—	
	Nonsurvivors			1.22			
Osmolality (mOsm/kg H_2O)	Survivors	NS		NS		280	$P < 0.05$
	Nonsurvivors					301	
K (mEq/liter)	Survivors	3.64	$P < 0.05$	3.73	$P < 0.05$	3.80	$P < 0.05$
	Nonsurvivors	4.44		4.47		4.60	
Na (mEq/liter)	Survivors	138	$P < 0.02$	NS		NS	
	Nonsurvivors	133					
Glucose (mg/100 ml)	Survivors	NS		153	$P < 0.05$	NS	
	Nonsurvivors			243			
Insulin (μU/ml)		NS		NS		NS	
NEFA (mEq/liter)		NS		NS		NS	
Total ketones (mg/ml)	Survivors	14.7	$P < 0.05$	13.2	$P < 0.05$	40.8	$P < 0.05$
	Nonsurvivors	24.9		25.1		129.1	
Lactate (mmole/liter)	Survivors	NS		3.33	$P < 0.05$	1.47	$P < 0.05$
	Nonsurvivors			5.84		9.04	
Pcs_{O_2} (mm Hg)	Survivors	25.2	$P < 0.05$	25.2	$P < 0.01$	25.4	$P < 0.02$
	Nonsurvivors	18.4		17.5		17.7	
Growth hormone (ng/ml)	Survivors	NS		NS		7.7	$P < 0.05$
	Nonsurvivors					48.8	

Abbreviations: NEFA = nonesterified fatty acids; Pcs_{O_2} = tension of oxygen in coronary sinus; NS = not significant; $P < 0.05$.

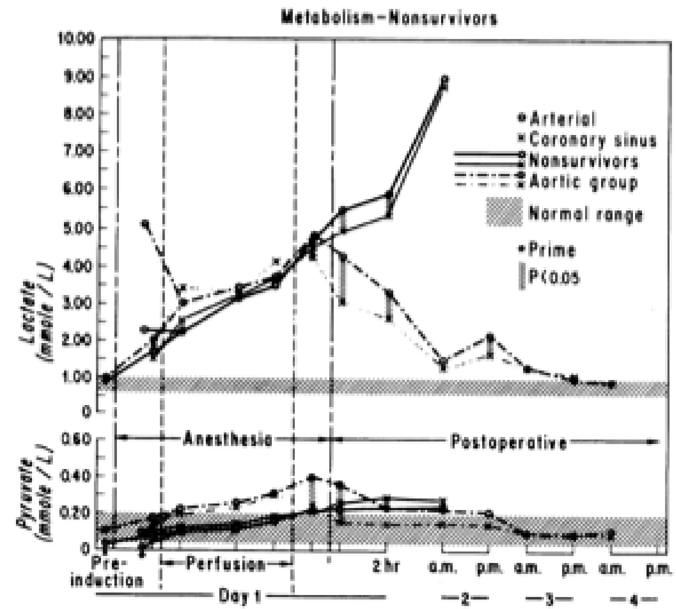


Figure 5

Lactate rose steadily postoperatively in nonsurvivors, while it fell in survivors.

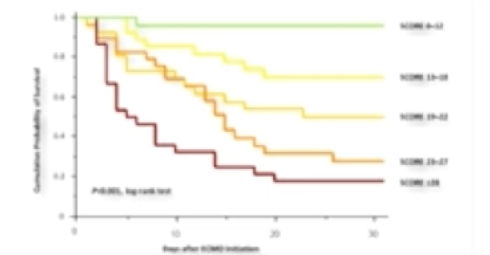
Lactic acidosis and mortality in AHF and shock

- **pH ≤7.35 and lactatemia >2.0 mmol.l^{-1} with a $PaCO_2$ ≤ 42 mmHg define lactic acidosis**

- Not extensively studied
- Independent predictor of mortality in variety of shock states
- Requiring ECMO: useful parameter to predict mortality
- Post STEMI: those with ineffective lactate clearance – lower survival rate

- Levels and associated mortality depend upon cause (same cutoff – lactate>4mmol/L, mortality zero in uncomplicated DKA, >75% post-cardiac arrest)

Parameter	OR (95% CI)	P	Component score
Age >60 years	2.63 (1.03–6.85)	0.048	5
Female	4.35 (1.29–14.72)	0.018	7
Body mass index >25 kg/m^2	3.10 (1.21–7.92)	0.018	6
Glasgow Coma Score <6	3.09 (1.19–8.05)	0.021	6
Creatininaemia >150 mmol/L	2.60 (1.05–6.45)	0.04	5
Serum lactate			
<2 mmol/L	1		0
2–8 mmol/L	4.71 (1.31–17.01)	0.02	8
>8 mmol/L	8.71 (1.76–43.10)	0.004	11
Prothrombin activity <50%	2.80 (1.01–7.77)	0.049	5



Muller G et al, submitted to Eur Heart J
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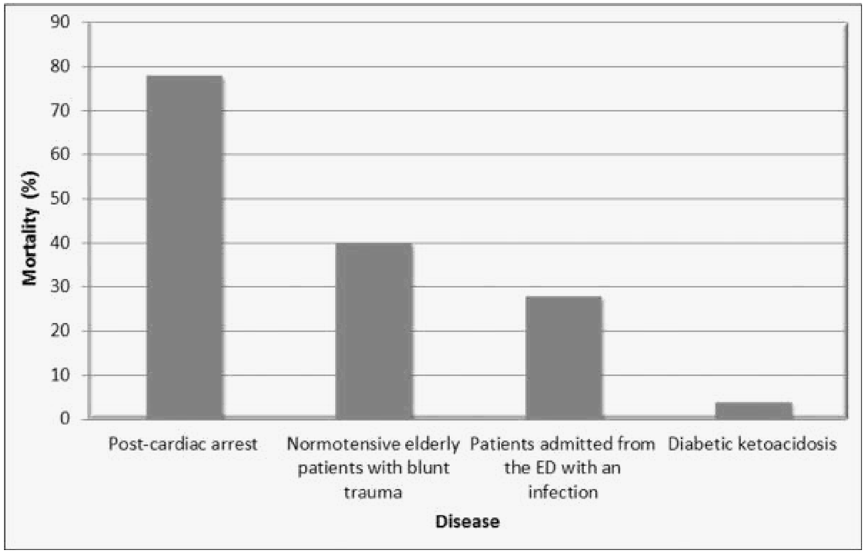
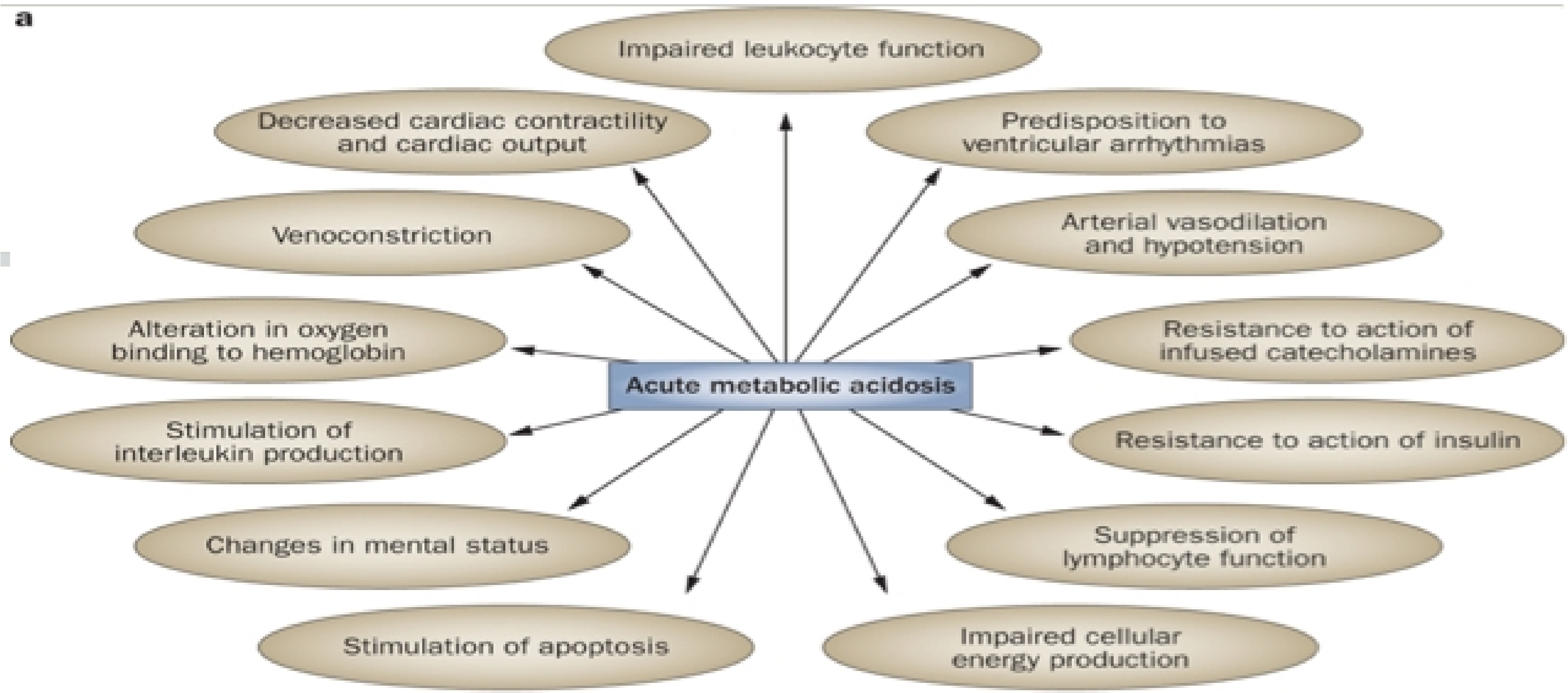


Is metabolic acidosis bad for you?

- Yes it is
- Also bad for our patients
- Why?

Potential impact?



Cardiovascular effects

- **Myocardial depression**
  - Decrease in pHi of myocytes
  - Inhibition of most steps of excitation-contraction coupling
  - Alterations in intracellular calcium
  - Changes in calcium binding to troponin-myosin complex
  - Impairment of actin-myosin cross-bridge cycling by monovalent phosphate

  - Decrease in pHe
  - Association demonstrated with contractility (*in vitro, in vivo*, worse when combined repiratory & metabolic acidosis)
  - Small positive inotropic effect (pH decreased from 7.4 to 7.2) ?mediated by rise in circulating catecholamines

Cardiovascular effects

- **Dysrhythmias**
  - **Conduction abnormalities caused by extracellular acidosis (with or without myocardial ischaemia)**
  - **MA: small reductions in blood pH (to 7.3) decrease VF threshold**
  - **Respiratory acidosis: repolarisation abnormalities – no effect on VF threshold**
  - **MA-induced arrhythmias -?in part due to rise in diastolic depolarisation state**
  - **Other MA associated changes that are relevant: changes in blood and intracellular potassium, calcium and magnesium concentrations, plus increase in sympathetic discharge**

Cardiovascular effects

- **Hypotension**
  - **Direct vasodilatory effect – reduction in SVR (may be counterbalanced by increase in sympathetic discharge)**

  [Beta-blockade - more profound decrease in BP (loss in compensatory mechanisms)]

- **Vasopressor resistance**
  - **Reduction in vascular response to alpha- and beta-adrenergic stimulation – most evident with combination of low pH and high lactate**

- **Not all vascular beds identical:**
  - **Cerebral circulation – decrease in cerebral vascular resistance**
  - **Renal vascular resistance – variable response**
  - **Myocardial blood flow**
    - **complicated by direct effect of MA and indirect effect that causes rise in myocardial O2 consumption**
    - **Decrease and increase in myocardial blood flow have been demonstrated**

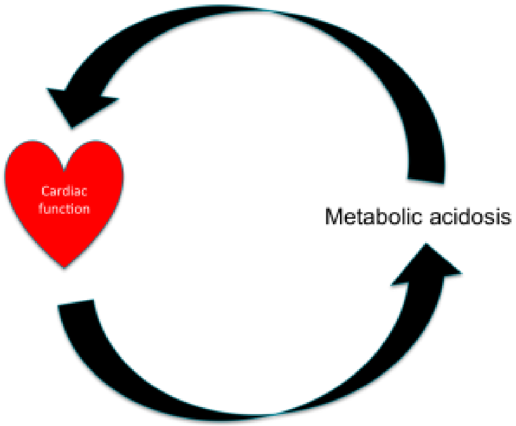
- **Venoconstriction**
  - **Associated with MA – increased sympathetic discharge, with rise in pulmonary vascular volume and pressure**

Net effects in AHF and shock

- Profound and widespread reduction in effective tissue perfusion
- Leads to cellular dysfunction and organ failure

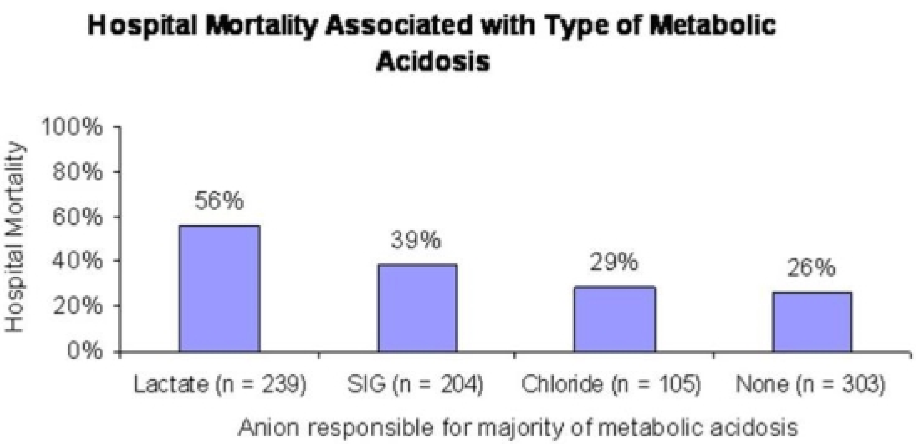
- Multifactorial in cause and effects:
  - **Inadequate cardiac performance**
  - **Maldistribution of cardiac output**
  - **Alterations in microcirculatory flow**
  - **Abnormalities in cellular bioenergetic function**

- **Lactic acidosis: cardinal manifestation of circulatory and cardiac shock**
- **Vicious cycle established…**



Critically ill: importance of metabolic acidosis

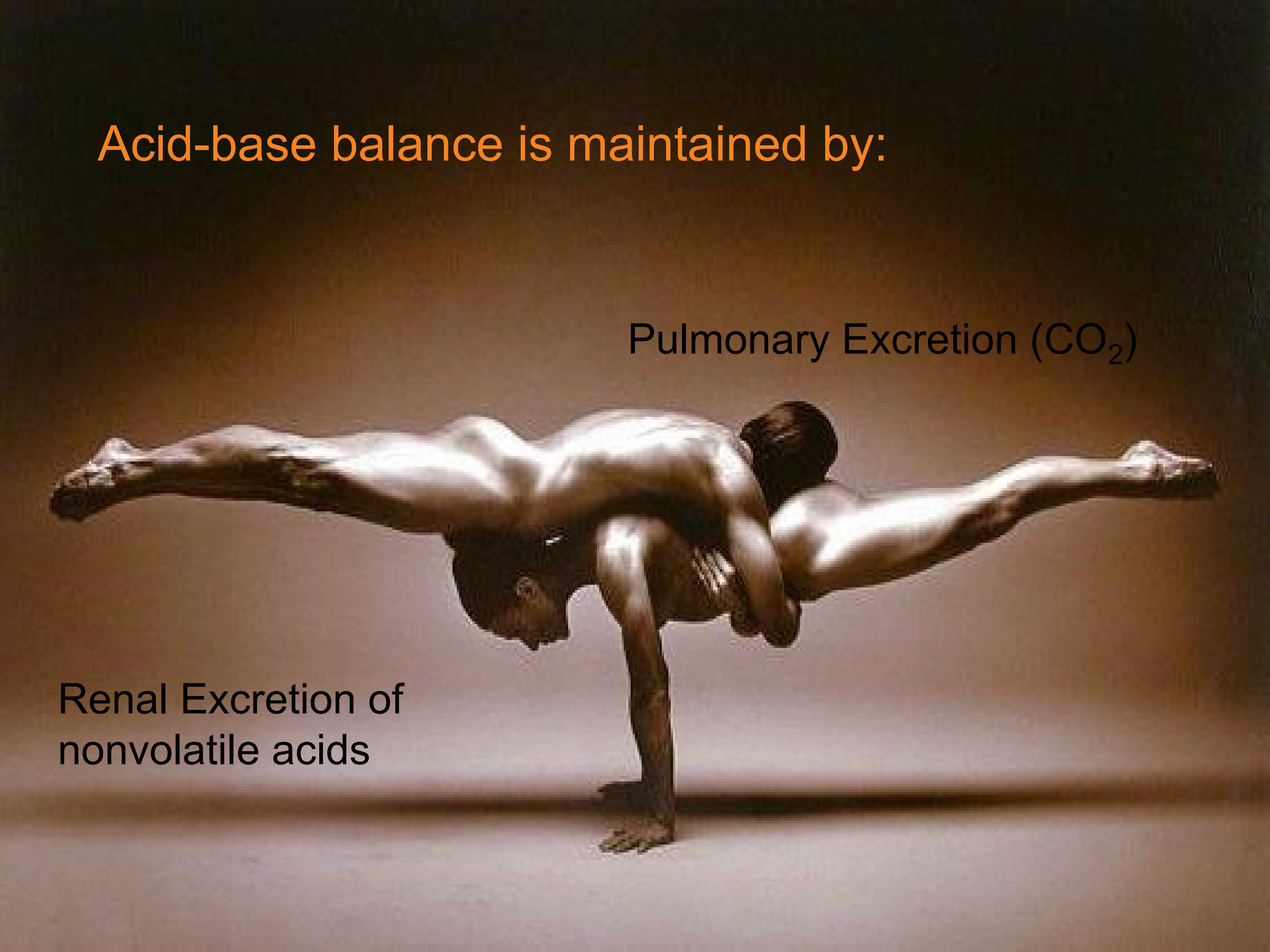
- **Associated with poor prognosis**
  - Marker of critical illness
  - Central mediator in matrix of critical illness
  - BE significant variable in predicting mortality, independent of APACHE II
- **Differences in outcome between respiratory vs metabolic acidosis in similar pH ranges**
- **Arises from variety of organic/inorganic fixed acids**
- **The "metabolic" aspect of metabolic acidosis may be more significant than perturbation of [H⁺]**



Gunnerson et al Critical Care 2006, **10:**R22

Metabolic acidosis: revision of some basics

Acid-base balance is maintained by:



Pulmonary Excretion (CO_2)

Renal Excretion of nonvolatile acids

Causes of metabolic acidosis

3 major mechanisms:

- **Increased Acid Generation:**
  - **Complicates a variety of clinical settings: lactic acidosis, ketoacidosis (uncontrolled DM, excess EtOH, fasting) or ingestion (methanol, ethylene glycol, aspirin, toluene)**
- **Loss of HCO_3^-**
  - **Mainly diarrhoea/ureteral diversion (rarely Type 2 RTA)**
- **Diminished Renal Acid Excretion**
  - **Type 1 RTA or renal failure**

Defining metabolic acidosis

1. Low serum pH
2. Low serum bicarbonate

3. Increased or normal serum anion gap:

- Serum AG = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$
  - **Normal range increases by 4**
- In critically ill, don't forget hypoproteinaemia
  - **AG $\Downarrow$ by 2.3 to 2.5 mmol/L for every 10 g/L fall in [albumin]**

Anion Gap can also be viewed as:

- Unmeasured Anions - Unmeasured Cations
- **Increase in AG can be induced by a fall in :**
  - Calcium, Magesium
- **More commonly by a rise in unmeasured anions**
  - accumulation of lactate in lactic acidosis
  - ketoacid anions in ketoacidosis
  - hyperalbuminemia due to volume contraction

Mechanism of acidosis	Increased AG	Normal AG
Increased acid production	Lactic acidosis	
	Ketoacidosis	
	Diabetes mellitus	
	Starvation	
	Alcohol-associated	
	Ingestions	
	Methanol	
	Ethylene glycol	
	Aspirin	
	Toluene (if early)	Toluene ingestion (if late due to urinary excretion of hippurate)
	Pyroglutamic acid (5-oxoproline)	
Loss of bicarbonate or bicarbonate precursors		Diarrhea or other intestinal losses (eg, tube drainage)
		Type 2 (proximal) renal tubular acidosis (RTA)
		Posttreatment of ketoacidosis
		Carbonic anhydrase inhibitors
		Ureteral diversion (eg, ileal loop)
Decreased renal acid excretion	Chronic kidney disease	Some cases of chronic kidney disease
		Type 1 (distal) RTA
		Type 4 RTA (hypoaldosteronism)

Pathophysiological classification of lactic acidosis

HYPOXIC	NON-HYPOXIC
Ischemia	**Delayed Clearance**
Shock, severe anemia, cardiac arrest	Renal or hepatic dysfunction
Global Hypoxia	**Pyruvate Dehydrogenase Dysfunction**
Carbon monoxide poisoning	Sepsis, thiamine deficiency, catecholamine excess, alcoholic and diabetic ketoacidosis
Respiratory Failure	**Uncoupling of Oxidative Phosphorylation**
Severe asthma, COPD, asphyxia	Cyanide, salicylates, methanol & ethylene glycol metabolites, anti-retroviral drugs, valproic acid, biguanides, INH
Regional Hypoperfusion	**Accelerated Aerobic Glycolysis**
Limb or mesenteric ischemia	Increased effort, sepsis, seizures, large fructose loads, malignancies

What do these "unmeasured" anions indicate?

Markers of deranged cellular energetics

- **Lactate/pyruvate ratio**
- **Acetoacetate/3hydroxybutyrate ratio**

Markers of mitochondrial redox state

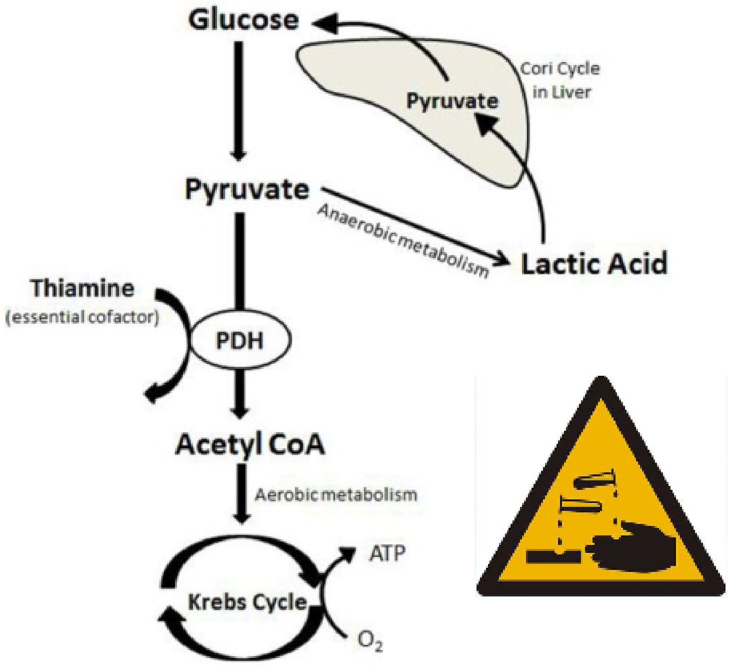
ratio of NADH to NAD+

Markers of poor prognosis
in pts with haemodynamic instability
(Levy et al)

The anaerobic threshold: lactic acidosis & low CO state

- O_2 delivery limited
- Switch to anaerobic metabolism

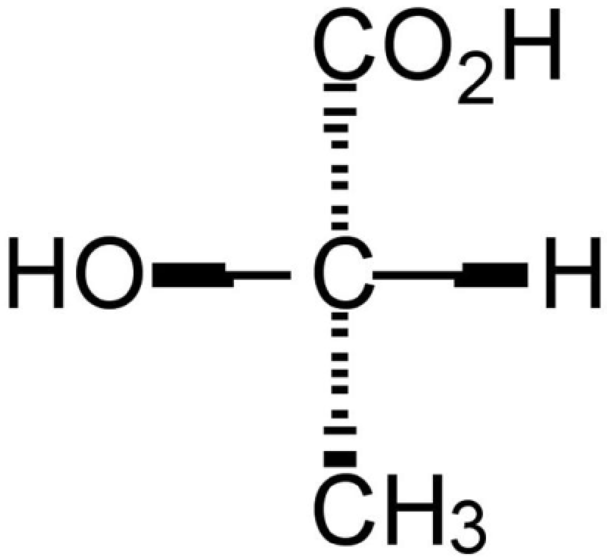
- Lactate generated
- Become acidotic



Lactic Acidosis

- Carboxylic acid moeity has a low pKa (pH = 3.87)
- Immediate and near total ionization of lactic acid across the range of cellular pH

- Therefore generates H^+

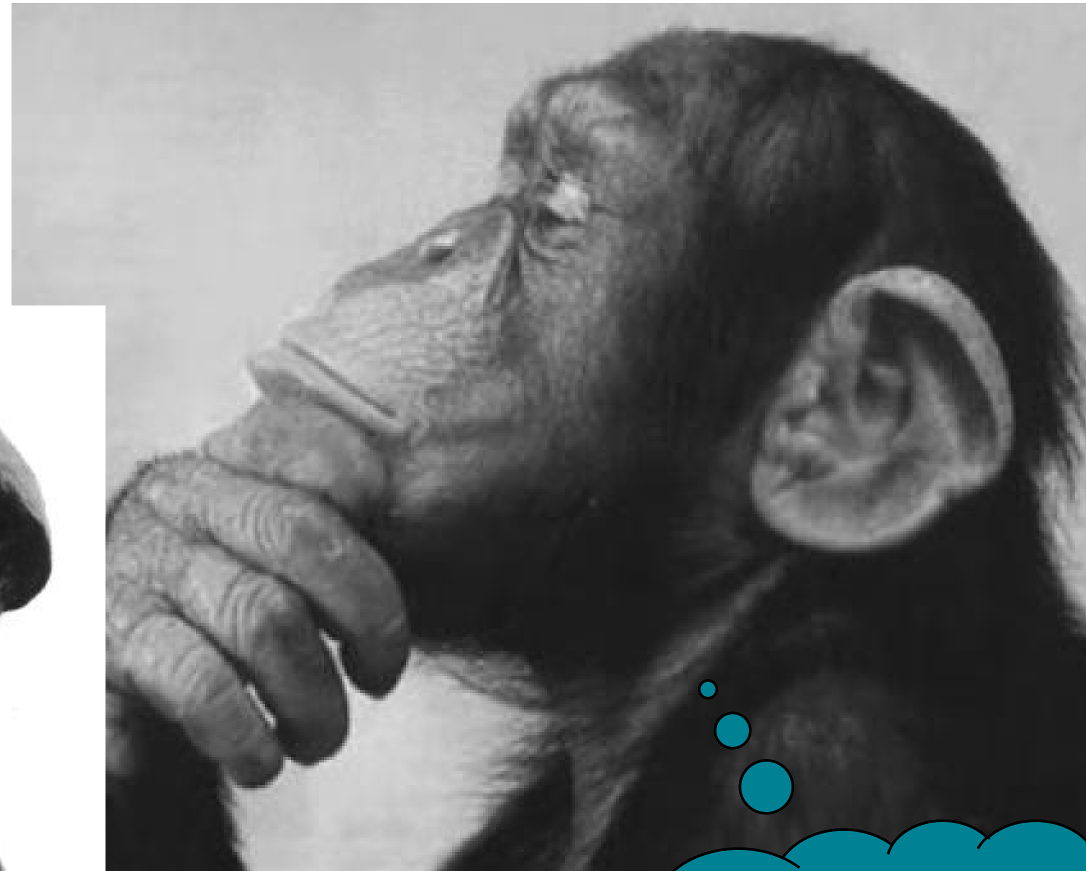


Lactic Acidosis: in simple terms

- Acidosis explained by the production of lactate

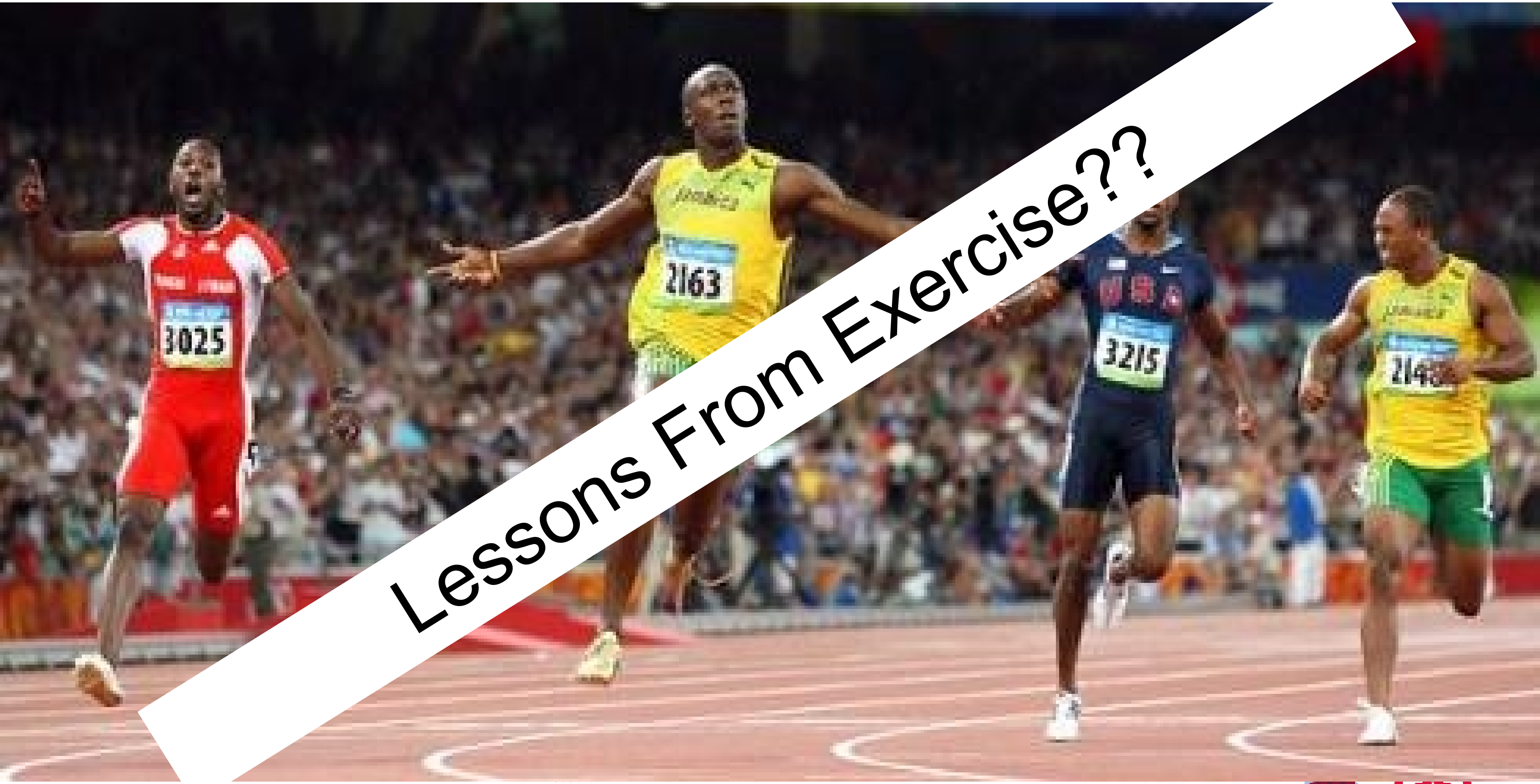
- Causes release of a proton
- Final product being the acid salt
- Termed Lactic Acidosis

That makes sense….



No it doesn’t…

Lessons From Exercise??



What do exercise physiologists say?

- The lactic acidosis explanation of metabolic acidosis:
  - Not supported by fundamental biochemistry
  - No research base of support
  - Remains a negative trait of all clinical, basic, and applied science fields and professions that still accept this construct……..

Table 2. *The reactions of glycolysis balanced for charge, protons, and water*

#	Reaction	Enzyme	H^+ Source	
			Glu	Gly
	G6P from glycogen			
	$\text{Glycogen-}_n + Pi^{2-} \rightarrow \text{Glycogen-}_{n-1} + \text{Glucose 1-phosphate}$	Phosphorylase		
	Glucose 1-phosphate → Glucose 6-phosphate	Phosphoglucomutase		
	G6P from glucose			
	$\text{Glucose} + MgATP^{2-} \rightarrow \text{Glucose 6-phosphate}^{2-} + MgADP^{-} + H^+$	Hexokinase	1	
	Glycolysis			
1	$\text{Glucose 6-phosphate}^{2-} \rightarrow \text{fructose 6-phosphate}^{2-}$	Glucose-6-phosphate isomerase		
2	$\text{Fructose 6-phosphate}^{2-} + MgATP^{2-} \rightarrow \text{fructose 1,6-bisphosphate}^{4-} + MgADP^{-} + H^+$	6-Phosphofructokinase	1	1
3	$\text{Fructose 1,6-bisphosphate}^{4-} \rightarrow \text{Dihydroxyacetone phosphate} + \text{Glyceraldehyde 3-phosphate}^{2-}$	Aldolase		
4	$\text{Dihydroxyacetone phosphate} \rightarrow \text{Glyceraldehyde 3-phosphate}^{2-}$	Triose Phosphate Isomerase		
5	$2\ \text{Glyceraldehyde 3-phosphate}^{2-} + 2NAD^+ + 2Pi^{2-} \rightarrow 2\ \text{1,3-bisphosphoglyerate}^{4-} + 2\ NADH + 2\ H^+$	Glyceraldehyde-3-Phosphate dehydrogenase	2	2
6	$2\ \text{1,3-bisphosphoglyerate}^{4-} + 2\ MgADP^{-} \rightarrow 2\ \text{3-phosphoglycerate}^{3-} + 2\ MgATP^{2-}$	Phosphoglycerate kinase		
7	$2\ \text{3-phosphoglycerate}^{4-} \rightarrow 2\ \text{2-phosphoglycerate}^{4-}$	Phosphoglycerate mutase		
8	$2\ \text{2-phosphoglycerate}^{3-} \rightarrow 2\ \text{phosphoenolpyruvate}^{3-} + 2H_2O$	Phosphopyruvate hydratase		
9	$2\ \text{phosphoenolpyruvate}^{3-} + 2\ MgADP^{-} + 2\ H^+ \rightarrow 2\ \text{pyruvate}^{-} + 2\ MgATP^{2-}$	Pyruvate kinase	−2	−2
		Net protons per 2 pyruvate	2	1

Proton source refers to the number of protons released (positive numbers) or consumed (negative numbers). Either glucose (Glu) or glycogen (Gly) fuel glycolysis. Adapted from Stryer (54).

- **For production of 2 Pyruvate there is :**
  - **2 H^+ when source is glucose**
  - **1 H^+ when source is glycogen**

Where Do The Protons Come From?

Proton generation comes from 3 steps:

- Formation G-6-P (ATP hydrolysis)
- Formation F-1,6-BiP (ATP Hydrolysis)
- Oxidation Glyceraldehyde 3-P

- **So**
- **What Happens On Exercise?**

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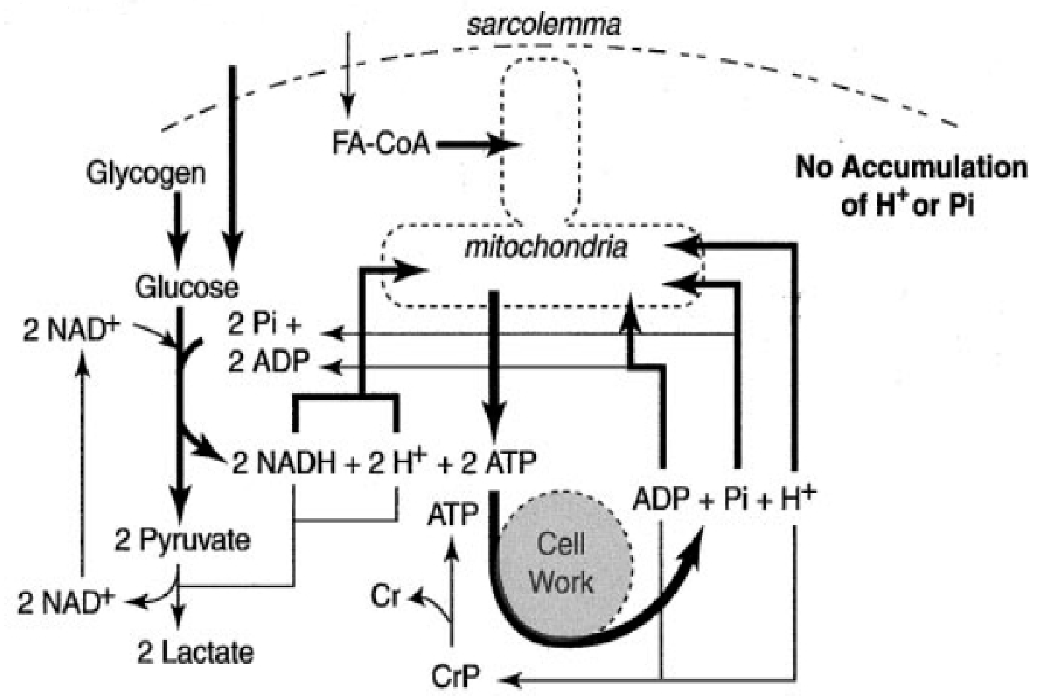
Exercise: Low Workloads

- Pyruvate, NADH and H^+ increase
- Produced by glycolysis
- Consumed by mitochondria
- Products of ATP hydrolysis consumed by mitochondria

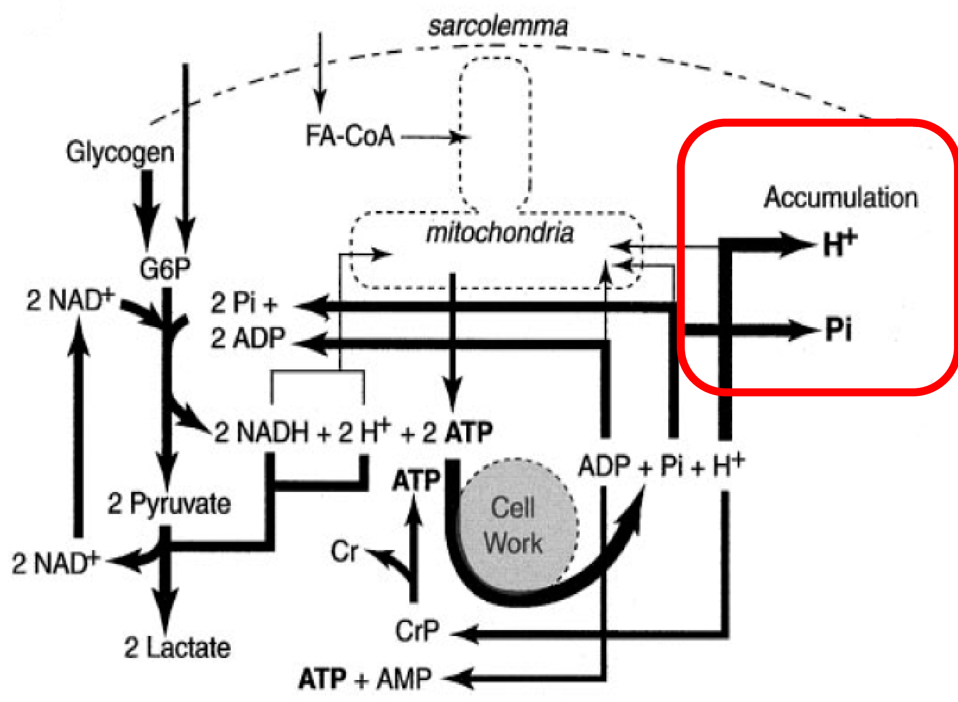
- pH neutral



Exercise: High Workloads

- ATP hydrolysis outstrips mitochondrial respiration
- Increased reliance on cellular ATP

- Each ATP generates a H^+ (and Pi)
- Therefore Acidifying….

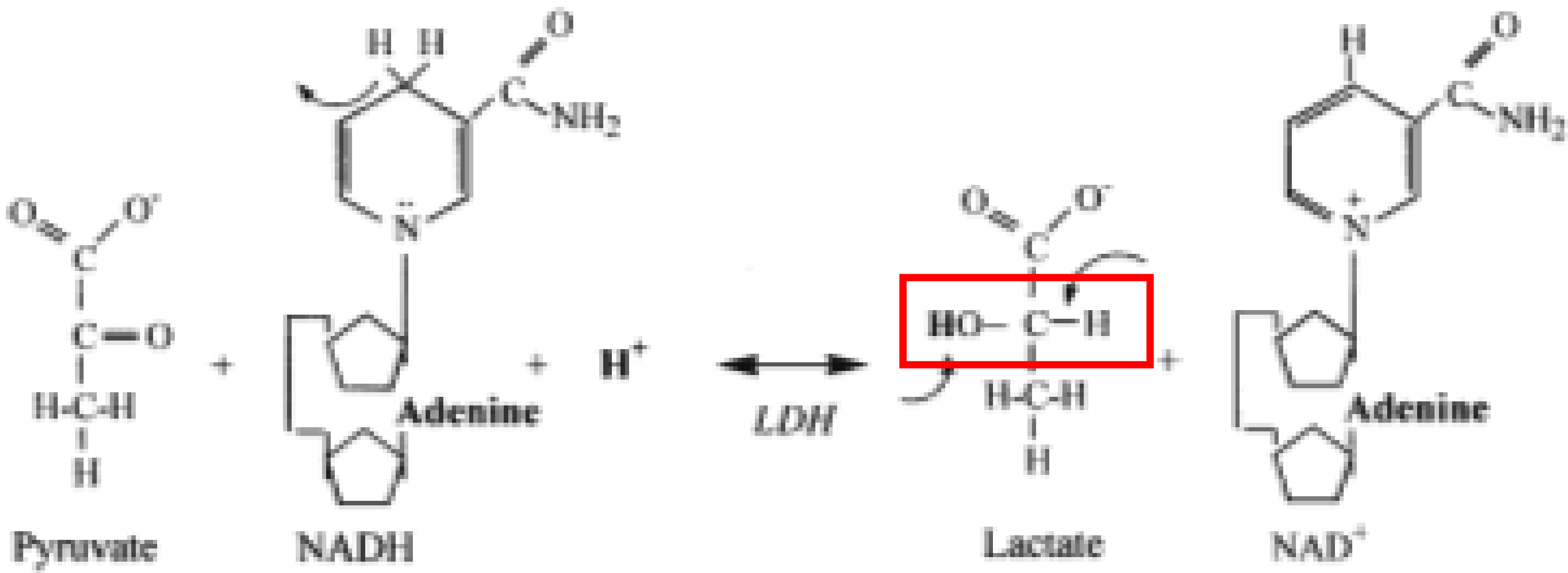


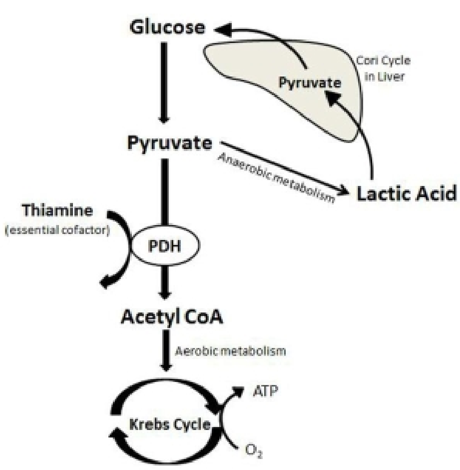
In context of inadequate oxygen supply

- Lactate is formed to:
  - **Produce cytosolic NAD^+**
  - **Support continued ATP regeneration from glycolysis**

- **So lactate : Good or Bad guy?**

Lactate : The Good Guy....





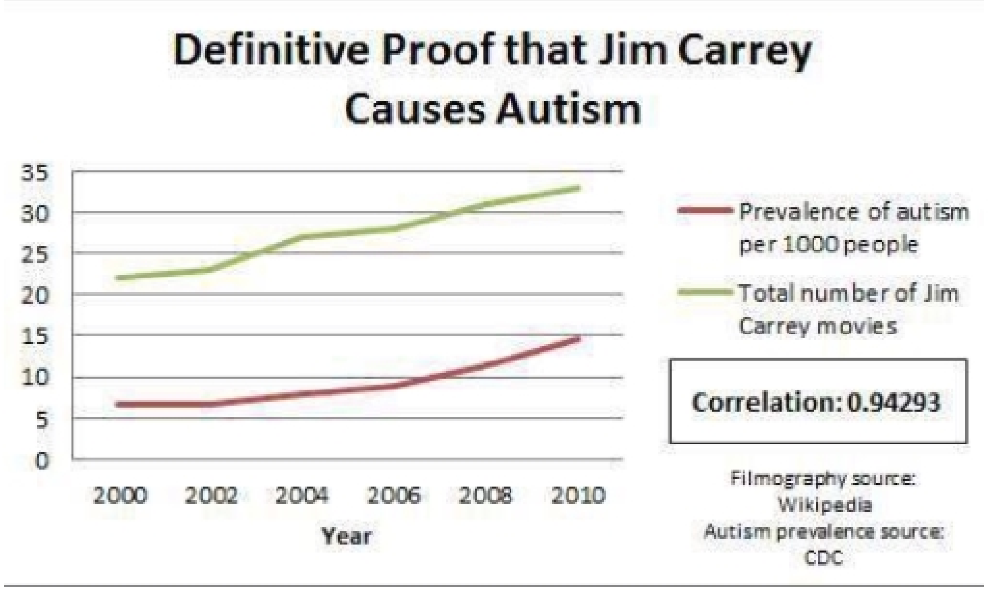
- Lactate production consumes two protons
- Therefore retards acidosis

Three key messages

Lactic acid is not formed –
should term this lactate anion acidosis

Inadequate oxygen supply & lactate anion acidosis

- Lactate facilitates H^+ removal from muscle via co-symport
- Lactate levels are good indirect indicators of increased proton release
-
- Such relationships should not be interpreted as cause and effect



Inadequate oxygen supply & lactate anion acidosis

Don't forget:

- Profound activation of HPA axis – increased sympathetic outflow from brain – epinephrine from adrenals
- ?hyperlactemia from B_2 adrenergic effects on glycolytic flux in skeletal muscle

J Appl Physiol (1985). 1995 Oct;79(4):1206-11.

Effect of graded epinephrine infusion on blood lactate response to exercise.

Turner MJ[1], Howley ET, Tanaka H, Ashraf M, Bassett DR Jr, Keefer DJ.

⊕ Author information

Abstract

In an attempt to determine whether the lactate threshold (LT) is the result of a sudden increase in plasma epinephrine (Epi), eight healthy college-aged males (22.4 +/- 0.4 yr) were recruited to perform three cycle ergometer exercise tests. Each subject performed a graded exercise test (GXT) to determine LT, Epi threshold, and norepinephrine threshold (64.6 +/- 2.4, 62.5 +/- 2.4, and 60.8 +/- 4.3% peak oxygen uptake, respectively). Each subject also completed, in random order, two 30-min submaximal (20% peak oxygen uptake below LT) exercise tests. During one test, graded Epi infusions were carried out at rates of 0.02-0.12 micrograms.kg-1.min-1; the other served as a control test. Infusion resulted in plasma Epi concentrations similar to those observed during GXT. The increase in blood lactate with Epi infusion was significantly greater than that during the control test (3.0 +/- 0.3 vs. 1.4 +/- 0.1 mmol/l at minute 30) but did not approach levels exhibited during GXT. We suggest an interaction of the increasing plasma Epi with other factors may be responsible for the sudden increase in blood lactate during graded exercise.

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Half-molar sodium lactate infusion improves cardiac performance in acute heart failure: a pilot randomised controlled clinical trial

Marek Nalos[1]*, Xavier Maurice Leverve[2], Stephen Joseph Huang[1], Leonie Weisbrodt[1], Ray Parkin[1], Ian Mark Seppelt[1], Iris Ting[1] and **Anthony Stuart Mclean[1]**

Theoretical background:

Normal energy supply for the heart: fatty acid oxidation, 10-40% energy derived from pyruvate (from glycolysis or conversion of lactate)

FAs have higher yields of ATP/molecule, ATP yield/O2 molecule is 5%-10% better with lactate and glucose

Exercise, inotropes and fast pacing: lactate uptake by myocardium and use as fuel increase

Lactate may exceed glucose as oxidative substrate

A number of studies support the role of lactate as a preferred oxidative substrate in stressed myocardium [13-16].

Half-molar lactate well-tolerated, & increases CO post-bypass surgery

Lactate not harmful – but potentially helpful

Introduction

Acute heart failure (AHF) is characterized by inadequate cardiac output (CO), congestive symptoms, poor peripheral perfusion and end-organ dysfunction. Treatment often includes a combination of diuretics, oxygen, positive pressure ventilation, inotropes and vasodilators or vasopressors. Lactate is a marker of illness severity but is also an important metabolic substrate for the myocardium at rest and during stress. We tested the effects of half-molar sodium lactate infusion on cardiac performance in AHF.

Methods

We conducted a prospective, randomised, controlled, open-label, pilot clinical trial in 40 patients fulfilling two of the following three criteria for AHF: (1) left ventricular ejection fraction <40%, (2) acute pulmonary oedema or respiratory failure of predominantly cardiac origin requiring mechanical ventilation and (3) currently receiving vasopressor and/or inotropic support. Patients in the intervention group received a 3 ml/kg bolus of half-molar sodium lactate over the course of 15 minutes followed by 1 ml/kg/h continuous infusion for 24 hours. The control group received only a 3 ml/kg bolus of Hartmann's solution without continuous infusion. The primary outcome was CO assessed by transthoracic echocardiography 24 hours after randomisation. Secondary outcomes included a measure of right ventricular systolic function (tricuspid annular plane systolic excursion (TAPSE)), acid-base balance, electrolyte and organ function parameters, along with length of stay and mortality.

Results

The infusion of half-molar sodium lactate increased (mean ± SD) CO from 4.05 ± 1.37 L/min to 5.49 ± 1.9 L/min ($P < 0.01$) and TAPSE from 14.7 ± 5.5 mm to 18.3 ± 7 mm ($P = 0.02$). Plasma sodium and pH increased (136 ± 4 to 146 ± 6 and 7.40 ± 0.06 to 7.53 ± 0.03, respectively; both $P < 0.01$), but potassium, chloride and phosphate levels decreased. There were no significant differences in the need for vasoactive therapy, respiratory support, renal or liver function tests, duration of ICU and hospital stay or 28- and 90-day mortality.

Conclusions

Infusion of half-molar sodium lactate improved cardiac performance and led to metabolic alkalosis in AHF patients without any detrimental effects on organ function.

Trial registration

Clinicaltrials.gov NCT01981655. Registered 13 August 2013.

Conclusions: lactate anion acidosis

- Please do not think a high lactate is a good thing
- Lactate production– a teleological response to stress?
- Metabolic derangements in AHF are complex (despite seeming superficially simple)

Thank you

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