LACTIC ACIDOSIS by Mark Ramzy DO & Nick Mark MD

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DEFINITIONS:

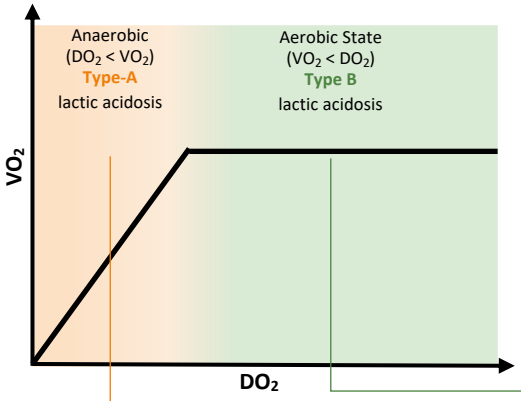
· **Lactic acid** is an endogenous substrate for gluconeogenesis, that is constantly produced by muscle and other tissues and is increased with exercise/activity. Lactic acid is non-toxic, though it can cause a metabolic acidosis and importantly can be a marker for severity of underlying disease.

· **Lactate** is the conjugate base of lactic acid (this is why LR does not cause acidosis)

· **Lactic Acidosis** is defined as an arterial lactate level ≥ 2 mmol/L PLUS a pH < 7.35

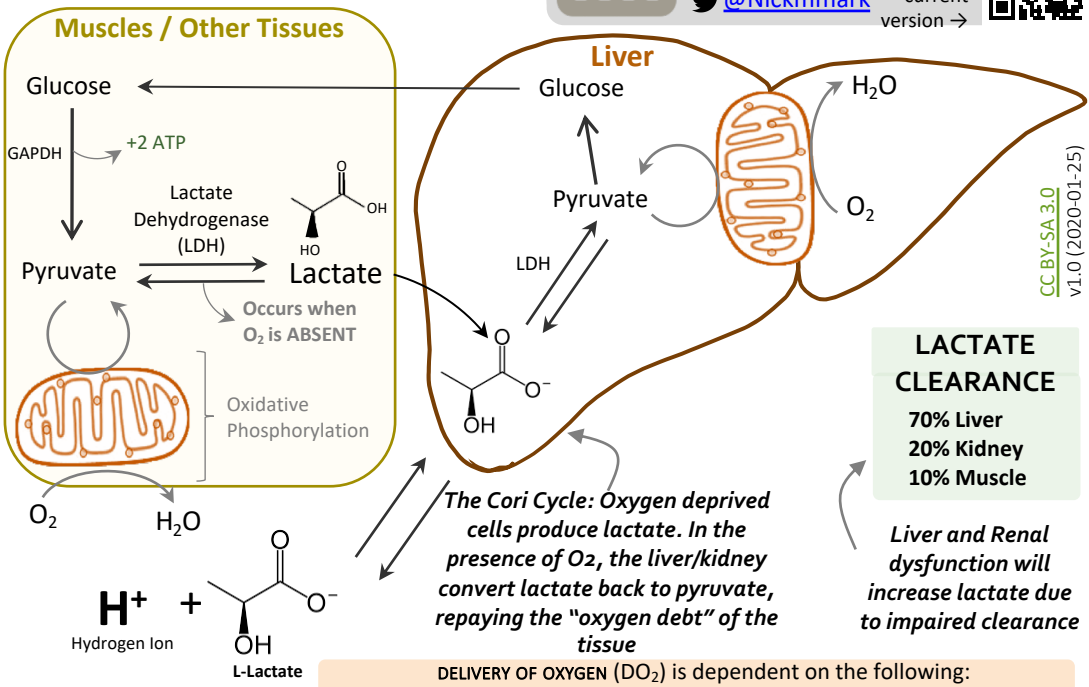
ROLE IN DISEASE:

Lactatic acid may be elevated in critical illness due to ***impaired O2 delivery*** (Type A lactic acidosis) ***or impaired O2 utilization by cells*** (Type B Lactic Acidosis). Rarely, an enantiomer of lactate (D-lactate) may be produced by gut bacteria in patients with bacterial overgrowth, causing another type of lactic acidosis. Elevation in serum lactic acid is associated with severe sepsis, ***but*** lactic acid clearance is unreliable as a resuscitation endpoint.



· In sepsis & septic shock, lactate elevations may be due to increased β^2 adrenergic tone rather than end-organ hypoperfusion

· Septic patients with elevated lactate typically have hyperdynamic circulation & O2 delivery Importantly, in severe sepsis increasing oxygen delivery (DO2) may not increase O2 consumption (VO2) nor does it affect lactate clearance.



LACTATE CLEARANCE

70% Liver
20% Kidney
10% Muscle

Liver and Renal dysfunction will increase lactate due to impaired clearance

DELIVERY OF OXYGEN (DO_2) is dependent on the following:

CARDIAC OUTPUT (CO) & BLOOD O_2 CONTENT (CaO_2)

$$DO_2 = (HR \times SV) \times [(1.34 \times Hb \times SaO_2) + (PaO_2 \times 0.003)]$$

TYPE A: IMPAIRED O_2 DELIVERY (DO_2)

DECREASED O2 *DELIVERY*

Consider etiologies that impair adequate perfusion

- **Hypotension & Hypovolemia**
- **Trauma & burns**
- **Cardiogenic & Septic Shock**
- **Severe Anemia**
- **Cardiac Arrest**
- **Severe Hypoxemia**
- **Regional Ischemia**
- **Compartment Syndrome**

INCREASED O2 *DEMAND*

Consider etiologies that increase O2 consumption

- **Stress / Pain / Exercise**
- **Fever**
- **Hypothermia & Shivering**
- **Seizures**
- **βeta-Agonists**
- **↑ work of breathing**
- **Localized soft-tissue infection**
- **Mesenteric Ischemia**
- **Microcirculatory dysfunction**

TYPE B: IMPAIRED O_2 UTILIZATION (VO_2)

DRUGS / TOXINS

- Propofol (PRIS),
- Valproic Acid)
- Biguanides (Metformin)
- Linezolid, Lactulose
- HIV Antiretrovirals (esp. NRTIs)
- Acetaminophen
- Ethanol, Methanol & Other toxic alcohols
- Sodium Nitroprusside
- Others (ie. Ricin, Strychnine, Niacin, Salicylates, Isoniazid)

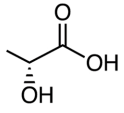
IMPAIRED CLEARANCE

- Systemic liver Failure
- Renal failure
- Mitochondrial dysfxn
- Inborn Errors of Metabolism

OTHER

- Infections (ie. HIV, Malaria, Late Sepsis)
- Malignancy (Leukemia/Lymphoma)
- Diabetes Mellitus +/- DKA
- Alcoholic lactic acidosis
- Deficiencies (Thiamine & Biotin)

TYPE D: BACTERIAL OVERGROWTH



D-Lactic Acid

- Presents as AG acidosis with negative lactate. Difficult to diagnosis as it requires separate D-lactate testing
- Seen in Short Bowel Syndrome, where decreased carbohydrate digestion leads to presence of additional sugars in the colon.
- Bacteria ferment and convert these sugars into D-Lactate
- Diabetic Ketoacidosis and Propylene Glycol administration have also been associated with D-Lactate accumulation