Management of Metabolic Acidosis

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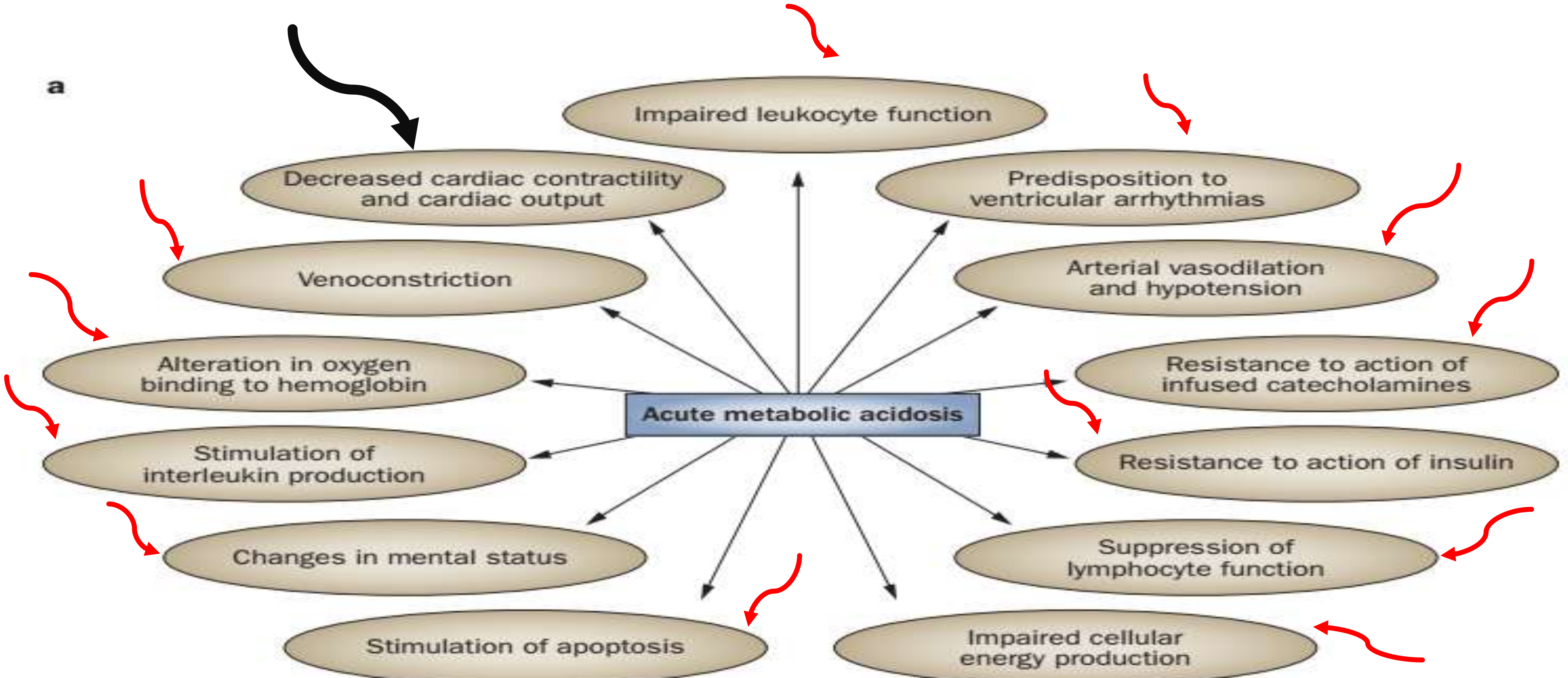
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Impaired leukocyte function
Decreased cardiac contractility and cardiac output
Predisposition to ventricular arrhythmias
Venoconstriction
Arterial vasodilation and hypotension
Alteration in oxygen binding to hemoglobin
Resistance to action of infused catecholamines
Acute metabolic acidosis
Stimulation of interleukin production
Resistance to action of insulin
Changes in mental status
Suppression of lymphocyte function
Stimulation of apoptosis
Impaired cellular energy production

Control of the respective cause

- Hemodynamic stabilization including volume therapy and vasopressors in cardiogenic, hypovolemic or septic shock
- Renal replacement therapy in certain types of intoxication if required

- Causal treatment often does not result in a rapid correction of serious pH abnormalities
- Therefore, ***alkali therapy*** is performed in hospitals all over the world
- Bicarbonate is metabolized into carbon dioxide (CO2) and water
- This process consumes protons

► ***Although the pH and serum bicarbonate level subsequently increase, several problems are unavoidable:***

- Firstly, increased CO_2 production requires ventilatory compensation
- Secondly, bicarbonate administration lowers the intracellular pH (most likely as a result of increased CO_2 transfer into the intracellular compartment)
- Finally, bicarbonate infusion has been shown to even elevate systemic lactate levels

- The use of buffers in the critically ill is common and largely lacks consensus on indications and possible benefits

- A substantial clinical benefit from bicarbonate for the management of aMA, particularly in AKI patients

Injudicious administration of (hypertonic) sodium bicarbonate may be associated with :

- Overshoot metabolic alkalosis
- Volume overload and Cardiac failure
- Hypernatraemia ($NaHCO_3$ solution contains 900 meq/lit Na)
- Extracellular cation depletion
- Cerebral edema
- exacerbation of intracellular acidosis caused by generation of the permeable gas CO_2 in the process of buffering

- Metabolic acidosis may be in part protective during ischemia by minimizing hypoperfusion induced tissue injury.

- Organic anion salts which act as an endogenous source of bicarbonate :

- lactate, citrate or acetate as their sodium salts

- Tromethamine (THAM) raise extracellular pH without reducing intracellular pH and might even increase it (rare cases of ***liver toxicity*** have been reported in newborn babies, ***hyperkalemia*** , ***hypoglycemia*** and ***pulmonary dysfunction***)

- requires ***sufficient renal function*** to ensure its urinary excretion and thus, its effectiveness

- $THAM - NH_2 + H^+ = THAM - [NH_3^+]$
- $THAM - NH_2 + H_2O + CO_2 = THAM - [NH_3^+] + [HCO_3^-]$

* **Bicarbonate is administered in a variety of :**

- Fixed doses
- Plasma bicarbonate level or pH
- Titrated to the base deficit multiplied by ‘bicarbonate space’

- If ***sodium bicarbonate*** is given, administer it slowly as an isotonic solution, with the initial dose limited to ≤1–2mEq/kg body weight

- The ***'bicarbonate space'*** concept suggests equivalence with 60% of the body weight in mild acidosis, but also that this space is heavily influenced by the initial [HCO_3^-] and thus the effects of therapy influenced by metabolic/ respiratory interactions

- The space of distribution of administered bicarbonate can vary from ***60%*** body weight when serum HCO_3^- concentration is >10 to as high as ***100%*** body weight or more when serum HCO_3^- concentration is ≤5

- Bicarbonate space = [0.4+ (2.6/ HCO_3^-])] × body weight

- Bicarbonate requirement = desired [HCO_3^-] – measured [HCO_3^-] × HCO_3^- space

- In intubated patients, a mild increase in ventilation to raise pH by reducing $PaCO_2$ might be an effective treatment, but its benefits should be weighed against the risk of barotrauma.

- In patients with ketoacidosis , consider administration of base :
  - if acidemia is severe (pH <7.1)
  - there is evidence of cardiovascular compromise
  - insulin and fluids fail to rapidly improve academia
- aim to maintain blood pH at ~7.2 and monitor patient carefully

- There is general consensus that ***pH > 7.20–7.25*** is desirable

- $[H+] = 24 \times \frac{(Pco2)}{[Hco3]}$
- PH= 7.2
- $63 \quad = 24 \times \frac{(Pco2)}{[Hco3]}$

* ***EXAMPLE :***

PH=7.1 Pco2=20 mmHg [Hco3]= 6meq/L

B.W=10 kg

- $63 = 24 \times \frac{20}{[Hco3]}$
- [Hco3] =8 meq/L
- $63 = 24 \times \frac{25}{[Hco3]}$
- [Hco3] =10 meq/L

- Bicarbonate space = [0.4+ (2.6/ HCO 3 –])] × body weight
- Bicarbonate space = [0.4+ (2.6/ 6)]× body weight
- Space = 0.8

- Bicarbonate requirement = desired [HCO3–] – measured [HCO 3 –] × [HCO 3] space

- Bicarbonate requirement = (10 – 6) × 0.8 ×10= ***<u>32</u>***

- Base required = (base deficit) × (B.W in kg) × 0.4- - 0.5

Dialysis

TAKE HOME MESSAGE