Acid-Base Disorders Algorithm

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Acid-Base Physiology by Kerry Brandis http://www.gldanaesthesia.com

- PaCO2 increases by 0.7mmHg for each 1mEq/L increase in HCO3

- TTKG = u + K u - CI u

- AG = (Na + K + Mg2+) - (CI + HCO3)

- AG = 10 - 14

- PaCO2, NI HCO3 Non-compensated

- PaCO2 ± 3

- Predicted PaCO2 decreases 0.7-0.9 mmHg for each 10mmHg increase in HCO3

- Predicted PaCO2 = 1.5 (HCO3) + 8 ± 2

- No acid-base disorder

- Predicted = Measured

- Non-compensated

- Predicted > Measured

- Chronic Respiratory Alkalosis

- Predicted < Measured

- Chronic Respiratory Acidosis

- No additional acid-base disorder

- Predicted > Measured

- Acute Respiratory Acidosis

- Acute Respiratory Alkalosis

- No AG Metabolic Acidosis

- AG Metabolic Acidosis

- Renal HCO3 oversimplifies complex acid-base relationships, and with minor deviation (0.7-1.5) the gap is as likely to misidentify a mixed acid-base disorder; however, with greater deviations (i.e. > 2) the presence of a mixed disorder is likely.

Notes:

- Usually compensation does not produce a normal PH except in chronic Respiratory Alkalosis, where compensatory Metabolic Acidosis can convert PH. Sufficient time must elapse for compensation to reach steady state, approximately 24 hrs.

- There is no identifiable cause of an elevated AG in 1/3 of patients.

- Response to bicarbonate (0.5-1mEq/Kg/hr) infusion, urine PH and FeHCO3" could be "in RTA II.

- Trans-tubular K+ gradient <5, valid only if Uosm > Posm.

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