Treatment of acid-base disorders
Treatment of:

- Common causes of:
  - Metabolic Acidosis (Non-anion Gap)
  - Metabolic Acidosis (Anion Gap)
  - Metabolic Alkalosis
  - Respiratory Acidosis
  - Respiratory Alkalosis

- **FOCUS ON THE SURGICAL PATIENT.**
- **TREATMENT CONSISTS OF TREATING THE UNDERLYING CAUSE!!**
Metabolic acidosis (Non-AG)

• If caused by PRIMARY LOSS of bicarb, The loss is balanced by a GAIN in Chloride.

• A proportional increase in Cl- leaves the AG unchanged. Anion gap = (Na+ + K+) - [Cl- + HCO3-]

• Common causes of a normal AG metabolic acidosis:
  • Diarrhea (loss of HCO3- in the stool).
  • Early renal insufficiency (increased bicarb loss in the urine)
  • Infusion of isotonic saline.
  • Renal tubular acidosis.
Case 1

• 20YOM
• HPI: motocross racer fell on 100ft jump. Bottomed out suspension.
• PMHx: Nil
• Vitals HR 120, BP 90/50, RR 30, Afebrile.
• Physical: Talking, collared/boarded, Bruised right buttock/thigh, tender and deformed Right femur, unstable pelvis. No other injuries.
• X-ray: Fractured pelvis and Right femur.

• In trauma bay given 2L warmed N/S, BP 110/55 HR 120.

• Dx ????

• Next steps in fluid management???
Too Much Normal saline

• Dilution Acidosis:
  • Large volumes of fluid without bicarb or an anion that can be metabolized to bicarb such as lactate (Ringer’s) result in metabolic acidosis.

• Article Dec 2010 Chest:
  • HMA at 24 h was highly associated with infused NS ≥ 4 L.

  • Hyperchloremic metabolic acidosis following resuscitation of shock. Gheorghe C - Chest - 01-DEC-2010; 138(6): 1521-2

• TX: Use Ringers and appropriate use of colloid/blood products!!
Metabolic acidosis (AG)

- MUDPILES
  - M- Methanol
  - U- Uremia (impaired secretion of H+ into distal tubules)
  - D- Diabetic ketoacidosis/starvation ketoacidosis
  - P- Paraldehyde
  - I- Isoniazid, iron, ibuprofen
  - L- Lactate (ie. Seizures, shock)
  - E- Ethylene glycol, EtOH
  - S- Salicylates.

- TREAT UNDERLYING CAUSE!!!
Starvation/Alcoholic Ketoacidosis

- **Ketosis**
  - Insulin deficiency $\rightarrow$ Lipolysis $\rightarrow$ free fatty acid delivery to the liver
  - Glucagon + free fatty acids $\rightarrow$ ketoacids in the liver.

- **Starvation** *(3 days starvation increase ketones 10 FOLD)*
  - Decreased carbohydrate intake $\rightarrow$ Reduce insulin + increase glucagon

- **Alcoholic**
  - Malnourished
  - Alcohol inhibits gluconeogenesis and stimulates lipolysis
  - Metabolism of ethanol into acetic acid.

- **Treatment**
  - Volume resuscitation (Often dehydrated).
  - Dextrose (Stimulates insulin and inhibits glucagon)
  - **In ETOH Thiamine prior to dextrose to reduce risk of Wernicke’s.**
CASE 2

18YOM

• Presents to ER.
• HPI: POD13 ORIF of Open tibia fracture, complaining of feeling drowsy/unwell with vomiting, abdominal pain, been urinating alot.

• PMHX: Known T1DM gives himself his own injections but since not feeling well over last few day and not hungry hasn’t been giving it to himself.

• P/E: HR 110, BP 101/50, RR 20, Febrile 38.5C, Dry mucous membranes, Draining wound serosanguinous ?Pus.

• Labs: WBC 25, Urinalysis +Ketones, Serum ketones, ABG consistent with AG metabolic acidosis, BG=30, Na=135, K=5.4, Cl=97, BUN=12, Cr=150.

• DX and Initial treatment plan ????
Diabetic ketoacidosis (DKA)

- **Intro**
  - Often results of inappropriate Insulin dosing (T1DM)
  - Rule out other causes
  - 20% of patients with DKA are not known diabetics.

- **Medical Emergency**
  - ABCs status, mental status
  - Possible precipitating events (eg, source of infection, myocardial infarction, usually )
  - Volume status

- **Work up:**
  - Serum Glucose, *Lytes to calculate AG*, BUN/CR, CBC, Urine/Plasma ketones, Plasma osmolarity, ABG, ECG.
  - +/- CXR, cultures, Cardiac workup.
DKA Treatment

- **Volume** *(Loss = 3-8L)*
  - NS at a rate of 1L/hr for the first 2-3 hours,
  - decrease to 1/2 NS at 250-500cc/hr.
  - Add 5% dextrose to each liter once BGs in 12-14 range.

- **Insulin** *(R/O hypokalemia)*
  - IV bolus of 0.1 units/kg,
  - infusion at 0.1 U/kg/hr.
  - Check BG levels every hour.
  - Once BG 12-14 cut infusion by 1/2

- **Potassium**
  - First 3-5 hours critical.
  - Start replacing once K+ <5
  - Add 20-30 mmol KCL/L.
  - If K<3.5 hold insulin and replace K.

- **Bicarb** *(Controversial)*
  - Can Worsen hypoK and acedemia.
  - Replace only if PH<7.1
  - Not effective buffer below this.

Serum Glucose < 11.0 mmol/L  pH > 7.3
HCO3 ≥ 18 mmol/L  AG < 12 mmol/L
Lactic Acidosis

- Most common cause of metabolic acidosis in hospitalized patients.

- Plasma lactate concentration above 4 meq/L.

- Caused by Impaired tissue oxygenation (Hypoperfusion) >> increased anaerobic metabolism (Lactate end product of glycolysis).
  - MI, heart failure, hypovolemic/septic shock, VTE, Cardiopumonary arrest)

- Metformin toxicity (Glucose >> lactate in small intestine)
Back To Case

60 YOM – Alcoholic with UGIB

Vitals: RR 30, HR 120, BP 98/50, Afebrile

BW: HGB 62, Lactate 20, ABG (consistent with metabolic acidosis), PH=7.1

Next Steps????????
Lactic Acidosis (Treatment)

• **ABCs**
• Treat the underlying problem *(ANEMIA, Hypovolemia, Stop the bleeding)*
• **Buffering Agents (controversial)**
  • Bicarb (Standard Buffer)
  • Carbbicarb
  • Tris-[hydroxymethyl] aminomethane (THAM)
  • Dichloroacetate

• **Is treating the Acidosis helpful?**
Is treating the acidosis helpful?

• **Maybe**
  • (+) Severe acidemia may contribute to tissue hypoperfusion by decreasing cardiac contractility via a reduction in myocardial cell pH.
  • (-) Bicarb not effective buffer.
    • PK for carbonic acid is 6.1, which means 50% of acid is dissociated at Ph of 6.1.
    • Useful only at 1 PH around the PK (5.1<<6.1>>7.1)
  • (-) Bicarb may be harmful.
    • Worsens acidosis by increasing C02 (Needs lung perfusion and ventilation)
    • Postrecovery metabolic alkalosis (as the excess lactate is converted back to bicarbonate.
  • (-) Acidosis decreases apoptosis of energy deplete cells.
Alternative Buffers

- **Carbicarb**
  - Equimolar mixture of sodium carbonate (Na$_2$CO$_3$) and sodium bicarbonate.
  - Carbonate component of carbicarb will diminish the tendency toward venous hypercapnia.
  - Not studied in humans yet.

- **Tris-[hydroxymethyl] aminomethane (THAM)**
  - Buffers acids and CO2 by virtue of its amine (-NH$_2$) moiety.
  - No increase in CO2 but also no change in hemodynamics or serum bicarb levels.

- **Dichloroacetate**
  - Increase pyruvate dehydrogenase activity to oxidize pyruvate instead of converting to lactate.
  - No change in hemodynamics or bicarb concentrations.
Metabolic alkalosis

• Major causes surrounding the surgical patient.
  • Gastric depletion of H+ (NG tubes, vomiting, villous adenoma).
  • Contraction alkalosis (Loop/thiazide diuretics).

• Treatment (3 General principles initial management)
  • Correct volume depletion.
  • Correct potassium depletion.
  • Correct chloride depletion.

TREAT THE UNDERLYING ISSUE!!!!!
Respiratory Acidosis

• Hypoventilation
  • Acute (abrupt decrease in ventilation)
    • COPD exacerbation
    • Asthma exacerbation
    • Airway obstruction
    • Central depression of respiratory center.
    • Drugs (Narcotics)

• Chronic
  • COPD
  • Obesity hypoventilation syndrome.
  • Obstructive sleep apnea (positive airway pressure).

TREAT THE UNDERLYING DISORDER!!!
Respiratory Alkalosis

- Hyperventilation
- Causes
  - Asthma
  - Head trauma
  - Hyperthyroidism
  - Meningitis
  - MI
  - Pneumonia
  - Pneumothorax
  - Pulmonary edema
  - Pulmonary Embolism
  - Pulmonary fibrosis
  - Sepsis

TREAT UNDERLYING PATHOLOGY!!!