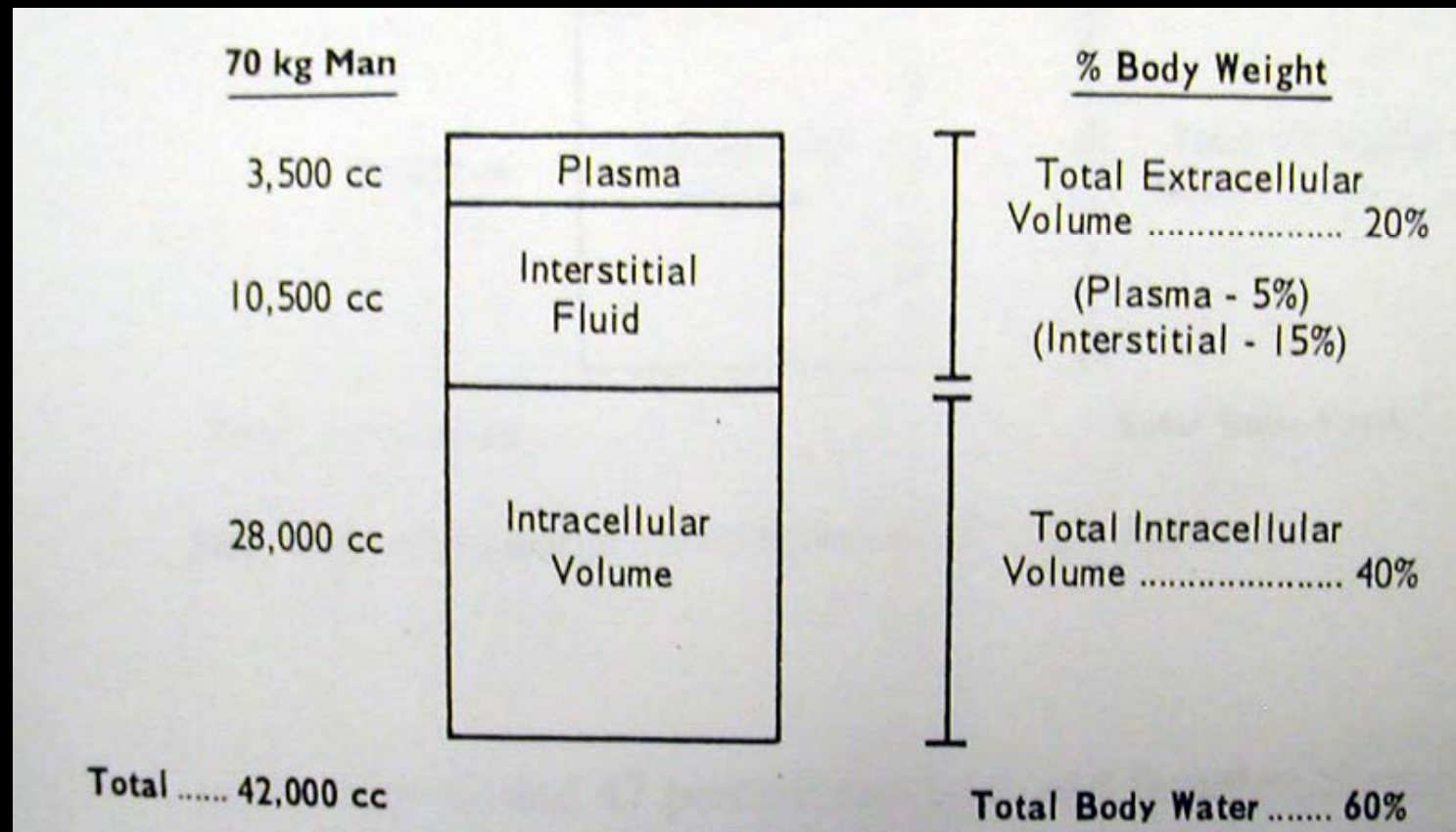


FLUIDS AND ELECTROLYTES

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NORMAL FLUID COMPARTMENTS



FLUID COMPARTMENT COMPOSITION

154 meq/L 154 meq/L

CATIONS		ANIONS	
Na ⁺	142	Cl ⁻	103
		HCO ₃ ⁻	27
		SO ₄ ⁻⁻⁻	3
		PO ₄ ⁻⁻⁻	
K ⁺	4	Organic Acids	5
Ca ⁺⁺	5	Protein	16
Mg ⁺⁺	3		

PLASMA

153 meq/L 153 meq/L

CATIONS		ANIONS	
Na ⁺	144	Cl ⁻	114
		HCO ₃ ⁻	30
		SO ₄ ⁻⁻⁻	3
		PO ₄ ⁻⁻⁻	
K ⁺	4	Organic Acids	5
Ca ⁺⁺	3	Proteins	1
Mg ⁺⁺	2		

INTERSTITIAL FLUID

200 meq/L 200 meq/L

CATIONS		ANIONS	
K ⁺	150	HPO ₄ [≡]	} 150
		SO ₄ ⁻⁻⁻	
		HCO ₃ ⁻	10
Mg ⁺⁺	40	Protein	40
Na ⁺	10		

INTRACELLULAR FLUID

DEFINITIONS

- Total body water
 - Varies from 50-70% of body weight
 - Young, healthy men = 60%
 - Young, healthy women = 50%
 - Variance of 15% within groups
 - TBW decreases with age

DEFINITIONS

- Intracellular fluid
 - 30-40% of body weight
 - Largest proportion of intracellular water is in skeletal muscle
- Extracellular fluid
 - Intravascular – plasma
 - Interstitial
 - Exchangeable
 - Non-exchangeable
 - Slowly exchangeable
 - Plasma proteins explain the higher cation concentration and lower anion concentration in plasma in keeping with the Gibbs-Donnan equilibrium.
- The difference in ionic concentrations in ICF vs ECF does not violate chemical laws governing osmolarity because the concentrations are expressed in milliequivalents and not milliosmoles. Some ions in ICF exist in undissociated state.

DEFINITIONS

- Osmotic pressure
 - Refers to pressure exerted by osmotically active particles or ions in solution
 - Expressed as a value per unit volume – mosm/L
 - Water is freely exchangeable across cell membranes and moves along osmotic gradients
- Equivalents
 - Refers to the chemical combining capacity of ions in solution
 - One equivalent is the atomic weight of an element in grams, milliequivalent is that number in milligrams
 - Chemicals move along electrical gradients and/or through specific membrane channels.

DEFINITIONS

- Slowly exchangeable spaces
 - Physiologic
 - Water chemically bound to extracellular matrix
 - Joint fluids
 - Cerebrospinal fluid
 - Ocular fluid
 - Pathologic
 - Ascites
 - “Third space”

OSMOTIC PRESSURE

- Effective osmotic pressure is determined, on each side of the cell membrane, by substances which do not cross the cell membrane.
 - Intracellular proteins
 - Plasma proteins (colloid oncotic pressure)
- An increase in osmotically active particles on either side of the cell membrane will be balanced by movement of water.

FLUID AND ELECTROLYTE BALANCE

- Water balance
 - Normal intake is 2-2.5 L/day
 - Minimal obligatory loss is 500 ml/day of urine plus 100-200 ml/day insensible losses
 - Insensible losses increased by fever, hyperventilation
 - Concentration capacity of the kidney decreases with age and with chronic renal disease

SODIUM BALANCE

<u>Sodium exchange</u>	<u>Average</u>	<u>Minimal</u>	<u>Maximal</u>
Sodium gain	50-90 mEq/day	0	75-100 mEq/day
Sodium loss: skin	10-80 mEq/day	0	300 mEq/hr
Sodium loss: urine	10-60 mEq/day	<1 mEq/day	110-200 mEq/L
Sodium loss: intestine	0-20 mEq/day	0	300 mEq/hr

GENERAL RULES

- Gains or losses of isotonic fluids result in no change of ionic composition.
- Losses of isotonic, sodium containing fluids over time (gastric outlet obstruction) will result in ECF volume constriction followed, in days, by hyponatremia as slowly equilibrating water moves out of cells into ECF
- Losses of other ions (K^+ , Cl^-) will result in compositional changes but no added effect on osmotic balance.

AN APPROACH TO DIAGNOSIS

- Volume
 - Intravascular
 - Extracellular
 - Intracellular
- Concentration
 - Disorders of osmolarity
- Composition
 - Specific ion disorders
 - Acid-base disorders

DIAGNOSING VOLUME STATUS

- There is no laboratory test helpful in the diagnosis of volume status
 - An trend of increasing BUN and hematocrit infers ECF volume deficiency.
- Clinical signs of ECF volume deficiency
 - Fatigue, apathy, progressing to peripheral numbness and stupor. Nausea, tachycardia, orthostatic then overt hypotension, ↓ temperature.
- Clinical signs of ECF volume excess
 - Tissue edema, ascites, pulmonary vascular congestion, pulmonary edema in patients with heart disease or renal failure.
- What about tissue turgor, small tongue, etc.?

FLUIDS

- Maintenance fluids
 - Dextrose and $\frac{1}{4}$ or $\frac{1}{2}$ normal saline with added potassium
- Correction fluids
 - ECF correction: lactated Ringer's solution or normal saline
 - Hypertonic saline
- Specific ions: potassium, magnesium

SODIUM DISORDERS

■ Hyponatremia

- Clinical problems are due to central nervous system dysfunction; obtundation, seizures, coma, overt cerebral edema
- Correction: subtract measured from desired Na concentration and multiply by total body water. Because this will over correct, shoot for 60% correction. Rapidity of correction depends on symptoms.

SODIUM DISORDERS

■ Hyponatremia

- Common in critically ill patients receiving TPN
- Implies loss of water
- Symptoms: restlessness, weakness, tachycardia
- Correct over 48-72 hr depending on symptoms
- Percentage of Na increase over normal is the percentage by which total body water needs to increase for perfect correction.
- Problem did not occur rapidly so correction should not be rapid.

SPECIFIC ION DISORDERS

- Hyper and hypo kalemia: main danger is to cardiac function. Patients need to be on EKG monitor during treatment. Disorders of potassium balance very common in surgical patients. Replace, on emergency basis, with 20-40 mEq/hr infusion. Serum levels below 3.5 associated with deficits > 100 mEq.
- Emergent treatment of hyperkalemia with calcium, dextrose, and insulin. Definitive treatment is dialysis

SPECIFIC ION DISORDERS

- Magnesium deficiency is common in surgical patients. Diabetic patients, alcoholics, and burn patients are specifically at risk. Goes hand in hand with potassium deficiency. Serum level is linearly related to total loss. Effects are far reaching with cardiac abnormalities being most important. Signs of hypomagnesemia are similar to hypocalcemia (neuromuscular irritability, tetany). May give up to 20 mEq/hr for acute replacement with close ICU monitoring.

CASE STUDY

55 yo male with 10 year history of burning epigastric pain. 10 days ago, pain diminished and he began to have vomiting of large volumes of brown-black fluid sometimes containing previously ingested food. 36 hr ago he became dizzy and fainted at home. Complained of severe muscle weakness today and was brought to hospital. BP is 104/70 and HR is 110. Physical exam shows thin male with epigastric fullness. Hgb is 15.5, BUN 40, Cr 1.2, Na 129, K 2.4, Cl 84, CO₂ 32. Urine specific gravity is 1.034 and pH is 5. What is best approach?

METABOLIC ACIDOSIS

- Normal blood pH is 7.38-7.42
- Corresponds to $[H^+]$ 42-38 nanomoles/liter
- Decrease in pH triggers compensatory hyperventilation
 - if ventilation normal, compensation for mild-moderate acidosis occurs within hours
 - In mechanically ventilated patients, especially under general anesthesia, compensation may not occur
- Acidosis is either respiratory or metabolic as predicted by Henderson-Hasselbalch equation
- Diagnosis can be made with ABG and electrolyte panel

DIAGNOSING ACIDOSIS

- ABG allows examination of pH, PCO_2
 - Base deficit is equal to mEq/liter of acid or base required to move pH to normal
- Electrolyte panel allows measurement bicarbonate and calculation of anion gap
- Anion gap = $\text{Na} - (\text{Cl}^- + \text{HCO}_3^-)$
 - Normal anion gap is 10 - 4 mEq/liter
 - Maintained by serum albumin
 - Elevated anion gap means metabolic or exogenous acid has been added to ECF

CLASSIFICATION OF ACIDOSIS

- Anion gap
 - Lactic acidosis
 - Diabetic ketoacidosis
 - Alcoholic ketoacidosis
 - Uremia
 - Toxins
- Non-anion gap
 - RTA
 - TPN
 - Arginine, lysine, chloride
 - Drugs
 - Carbonic anhydrase inhibitors, spironolactone
 - ATN
 - Dilutional
 - Diarrhea

USING THE ANION GAP

- Three step approach of Haber
 - Measure the pH
 - Remember that compensation occurs but is never complete and the body never over-compensates
 - Determine the anion gap
 - If anion gap > 14 mmol/liter, anion gap acidosis exists
 - If anion gap is normal but SBE is negative, a non-anion gap acidosis exists
 - If anion gap is > 20 , primary metabolic acidosis exists

HAZARDS OF THE ANION GAP

- Anion gap may be falsely normal in:
 - Hypoalbuminemia
 - Normovolemic ketoacidosis
 - Many instances of lactic acidosis
 - One study showed 50% of patients with lactate levels between 4 and 7 had normal anion gap
 - When kidney excretes ketoacids in exchange for chloride
 - Efficiency of ketoacid excretion is a function of GFR

ALTERNATIVE TO THE ANION GAP

■ Stewart method

- Basically an explanation of acid-base physiology founded on principles of physical chemistry
 - Acid base balance determined by degree of dissociation of water into hydrogen and hydroxyl ions
 - Dissociation determined by PCO_2 , the concentration of weak acids (proteins), and the strong ion difference

STRONG ION DIFFERENCE

- Strong ions
 - Sodium, potassium, calcium, magnesium, phosphate, chloride, lactate
- Strong ion difference is $\text{Na} + \text{K} + \text{Ca} + \text{Mg} + \text{PO}_4 - \text{Cl} - \text{lactate}$
 - Normally 40-42 mEq/liter
- SID explains hyperchloremic “dilutional” acidosis
 - Waters et al studied 12 surgical patients given normal saline all of whom developed acidosis
 - Plasma volume remained unchanged, so no dilutional effect
 - Degree of acidosis related to chloride load administered

CASE STUDY

- 19 y/o male motorcycle crash victim sustains open-book pelvic fracture, bilateral open femur fracture and open left tibia-fibula fracture. Femurs and tib-fib treated with traction and pelvic arteries embolized. Patient receives 10 units packed rbc and 12 L NaCl. Twelve hours after injury, the patient is hemodynamically stable with good urine output. The pH is 7.36, Cl is 114, SBE is -8. What should be done?

EFFECTS OF ACIDOSIS

- Cardiovascular depression
- Obtundation and coma
- Increased minute ventilation
- Hyperkalemia
- Total scope of adverse effects largely unknown

CARDIAC EFFECTS

- Acidosis with pH of 7.2 or greater associated with increased myocardial contractility
- Below pH of 7.2, heart is less responsive to catecholamines but no intrinsic metabolic defect has been identified
 - Infusion of bicarbonate does not restore catecholamine responsiveness
 - Cardiac function may transiently improve after bicarbonate given
 - May be due to transfer of calcium into cells
 - May explain temporary positive inotropy associated with calcium infusion in shock

MANAGEMENT OF ACIDOSIS

- Most important form of acidosis in surgical patients is lactic acidosis due to shock, tissue hypoxia and anaerobic metabolism
 - Frustration abounds because:
 - Restoration of oxygen delivery does not always reverse lactic acidosis
 - Heterogeneity of tissue perfusion
 - Different varieties of lactate
 - Different stimuli for lactate production

WHERE DOES LACTATE COME FROM?

- “Shock lactate”
 - Anaerobic metabolism
 - Stimulation of Na-K ATPase by epinephrine
- “Stress lactate”
 - Decreased lactate uptake in liver
 - Liver takes up L-lactate
 - Excess endogenous lactate production
 - Phagocytes, wounds, lung, intestine

TREATMENT OF ACIDOSIS

- Treat the underlying cause
- Acidosis probably does not need to be specifically treated until bicarbonate level is less than 5 mEq/liter
- Buffers
 - Bicarbonate
 - THAM
 - Carbicarb
 - Dichloroacetate

SHOULD BICARBONATE BE USED?

- For bicarbonate wasting states, bicarbonate is indicated
- For lactic acidosis:
 - Low pH well tolerated in critically ill patients treated with permissive hypercapnia
 - Rapid, intentional hypoventilation reducing pH from 7.40-7.26 associated with increased cardiac output and lowered SVR
 - Bicarbonate can raise the blood pH but intracellular pH remains the same or is lowered
 - In patients with septic shock and acidemia, bicarbonate increased pH but hemodynamics and catecholamine responsiveness were not improved

WHAT IS THE BOTTOM LINE?

- Bicarbonate should only be used to temporarily augment pH in patients with pH < 7.1 with hyperkalemia or documented dangerous cardiac arrhythmia

CASE STUDY

61 yo woman with ESRD on 2x/week hemodialysis is injured in a automobile crash. She is brought to hospital with bilateral flail chest and pulmonary contusion. Left chest tube is placed for hemo-pneumothorax. She is intubated because of labored breathing. ABG on admission shows PaO₂ 61, PaCO₂ 33, pH 7.35, HCO₃ 20. BUN is 43 and Cr is 4.2. Electrolytes are normal. She is admitted to ICU and placed on SIMV with TV of 600 and rate of 10. Four hours later, ABG shows pH of 7.19, PCO₂ of 47 and PaO₂ of 90. ABG 12 hours post admit shows pH of 7.10, PCO₂ of 50. What is the problem?

METABOLIC ALKALOSIS

- More than half of the cases of acid-base imbalance in hospitalized patients are metabolic alkalosis
 - Diuretics
 - Vomiting
 - Nasogastric suction
- Mortality is 45% with $\text{pH} > 7.55$ and 80% when pH is > 7.66

CAUSES OF ALKALOSIS

- Accumulation of base or loss of acid?
 - With normal renal function, significant accumulation of base is rare
 - Hospitalized patients get alkalosis because of:
 - Unrecognized losses
 - Especially with malnutrition and hypoalbuminemia
 - Inappropriate fluid management
 - Aggressive diuresis
 - “You get it from your doctor!”

COMPONENTS OF ALKALOSIS

- Generation
- Maintenance
- Correction

WHY DOES ALKALOSIS PERSIST?

- Fluid losses cause hypovolemia and increased secretion of aldosterone
 - Aldosterone causes further renal losses of potassium and chloride
 - Magnesium deficiency may be additive to problem
- Effective therapy requires correction of volume deficits, replacement of sodium, potassium, and magnesium

CORRECTION STRATEGY

- Assess ECF volume status
 - Restore volume with normal saline
- Assess renal function
 - Restore potassium and magnesium
- How rapidly does correction need to be accomplished?
 - CHF
 - Dig toxicity
 - Arrhythmias
- If renal function good – acetazolamide 250-500 mg/day
- If urgent correction needed – 0.1 N HCl via central vein
 - Replacement formula: $0.5 \times \text{wt (kg)} \times \text{desired decrement in bicarbonate}$
- Monitor patient carefully in ICU

SUMMARY

- Acidosis and alkalosis important in surgical patients and equally dangerous
- Detect and correct underlying cause
 - Control the cause
 - Manage continuing losses
 - Establish correction strategy