ACID-BASE

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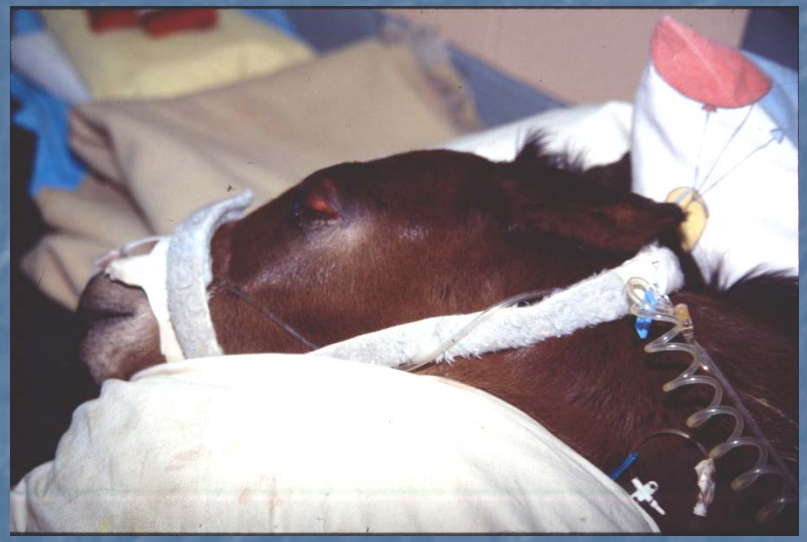
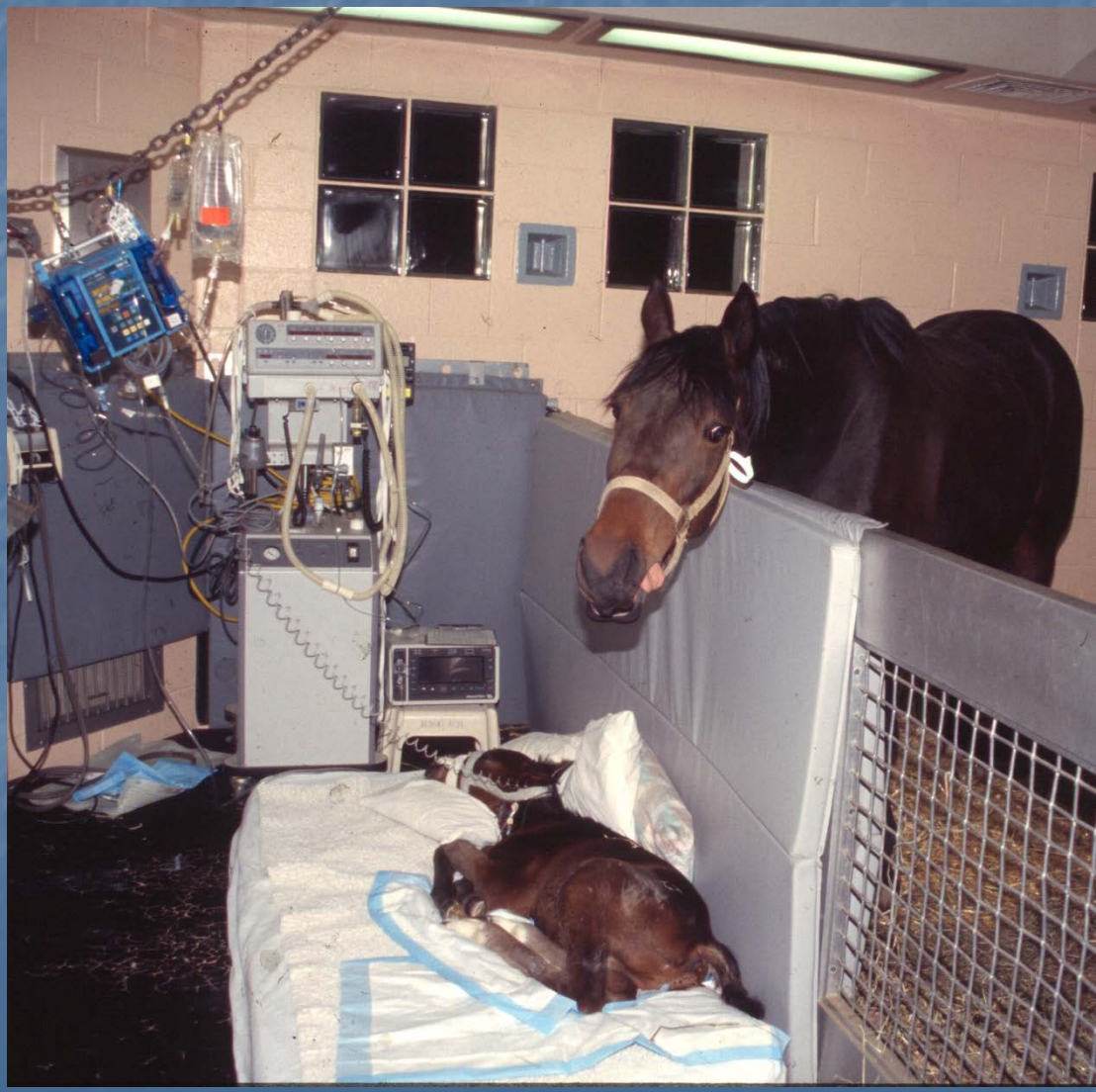
Acid-Base

- Introduction/ historic perspective
- Tools for acid-base analysis
  - Base Excess
  - Buffer base – weak acid buffers
  - Anion Gap
  - Strong ions – SID, SIG
- Metabolic acid-base abnormalities
  - Free water
    - Reflected in [Na]
  - Chloride – inorganic SID
  - Organic anions, Organic cations
  - Albumin level, phosphate level
- Differential diagnosis of metabolic disturbances
- http://ECEIM16.nicuvet.com

Acid-Base Disorders

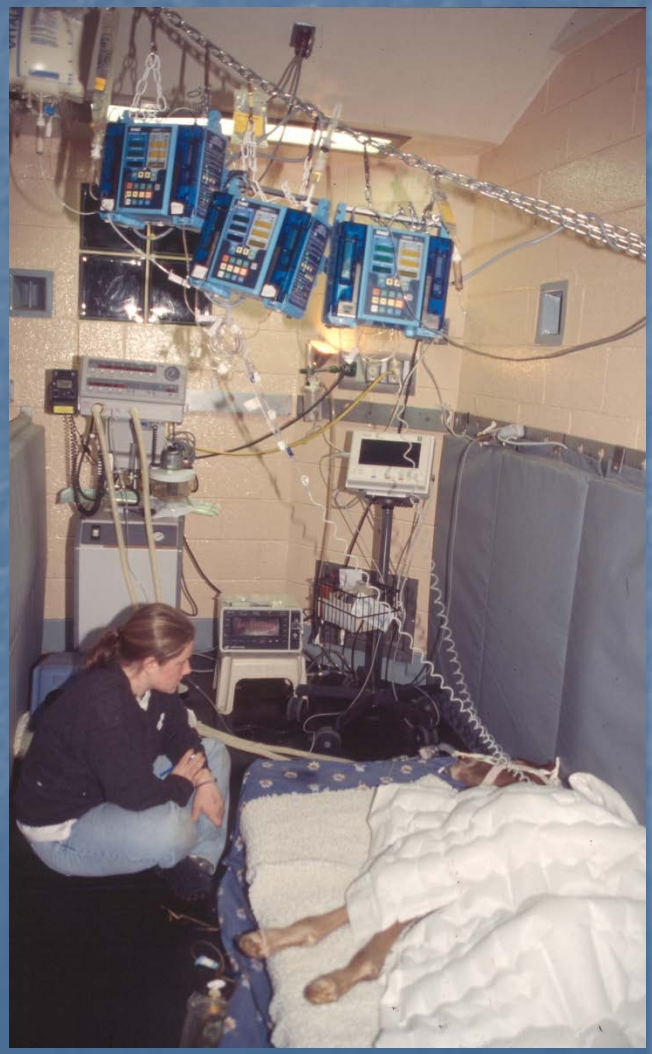




Acid-Base Abnormalities

Alterations in acid-base balance

Less important than the pathologic abnormalities causing them



Acid-Base Abnormalities

- Fatal disorders
  - Extreme (e.g., pH <7.0 or >7.7)
  - Develops quickly
  - Direct cause of organ dysfunction
- Harm because of the patient's response
  - Respiratory muscle fatigue
  - Diversion of blood flow from vital organs
  - Acidemia
    - Increases adrenergic tone
    - Increases myocardial oxygen demand

Acid Production

- Primarily CO_2
  - 150 to 250 mEq/kg/d of carbonic acid
  - Hemoglobin is major buffer
    - "Haldane" effect - H^+ bond, HCO_3 to plasma (Cl shift) – 65%
    - CO_2 bound to protein – 27%
    - Pco_2 – 8%
- Strong organic acids
  - 30 to 40 mEq/kg/d
  - Variety of acids
    - Lactic acid
    - Tricarboxylic acids
    - Keto acids
  - Produced/ metabolized to CO_2

Acid Production

- Inorganic acids
  - H_2SO_4
  - H_3PO_4
- Urinary excretion acid
  - 1 to 2 mEq/kg/d anions

History Acid-Base Analysis

- Henderson 1909

$$H^+ \propto \frac{HCO_3^-}{H_2CO_3}$$

- Hasselbalch 1916

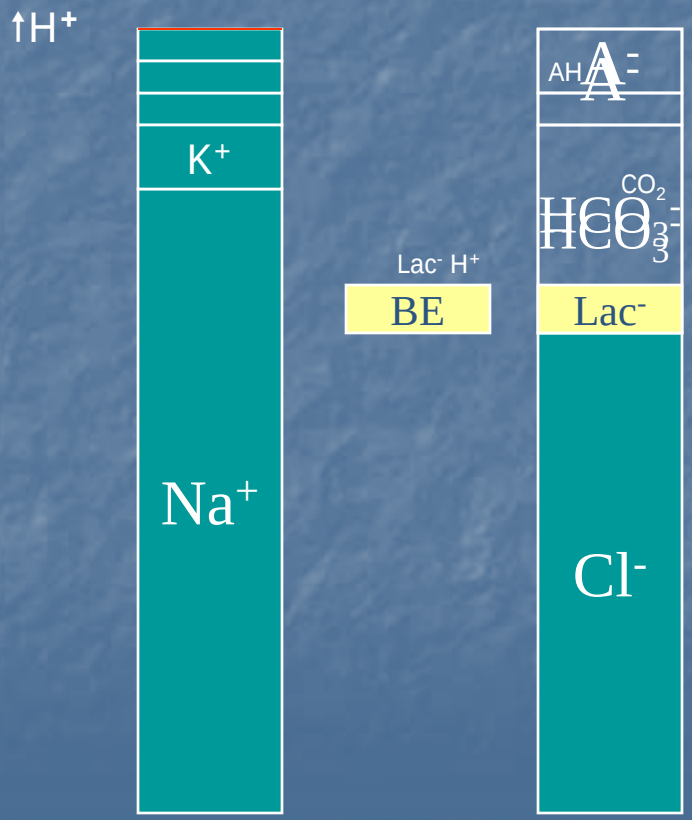
$$pH = 6.1 + \log\left[\frac{HCO_3^-}{P_{co_2} \times 0.03}\right]$$

- 1948 – Buffer Base
- 1957, 1958 – Standard Bicarbonate; Base Excess
- 1977 – Anion Gap
- 1981 – Stewart - Physical Chemistry

Base Excess

- Copenhagen Approach
  - Change in blood buffers
- Amount of acid/base added to whole blood
  - Return pH to 7.4
  - Assumptions
    - Pco_2 of 40 mm Hg
    - Temperature 37°C
    - Normal hemoglobin
    - Fully saturated blood
- Titration experiments
  - Nomograms
  - Formulas

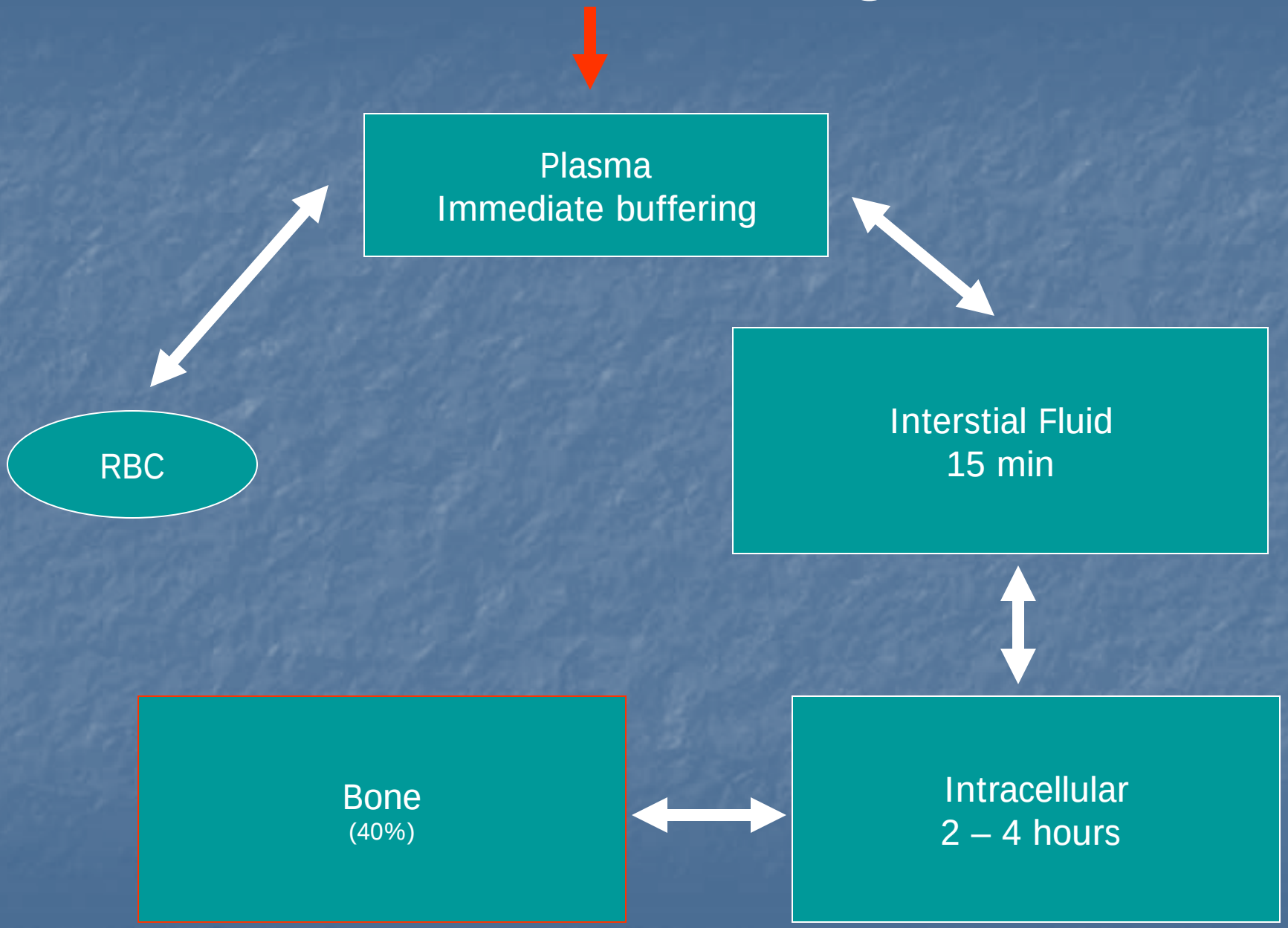
BE
Lactic Acidosis



Cations

Anions

Acid Buffering

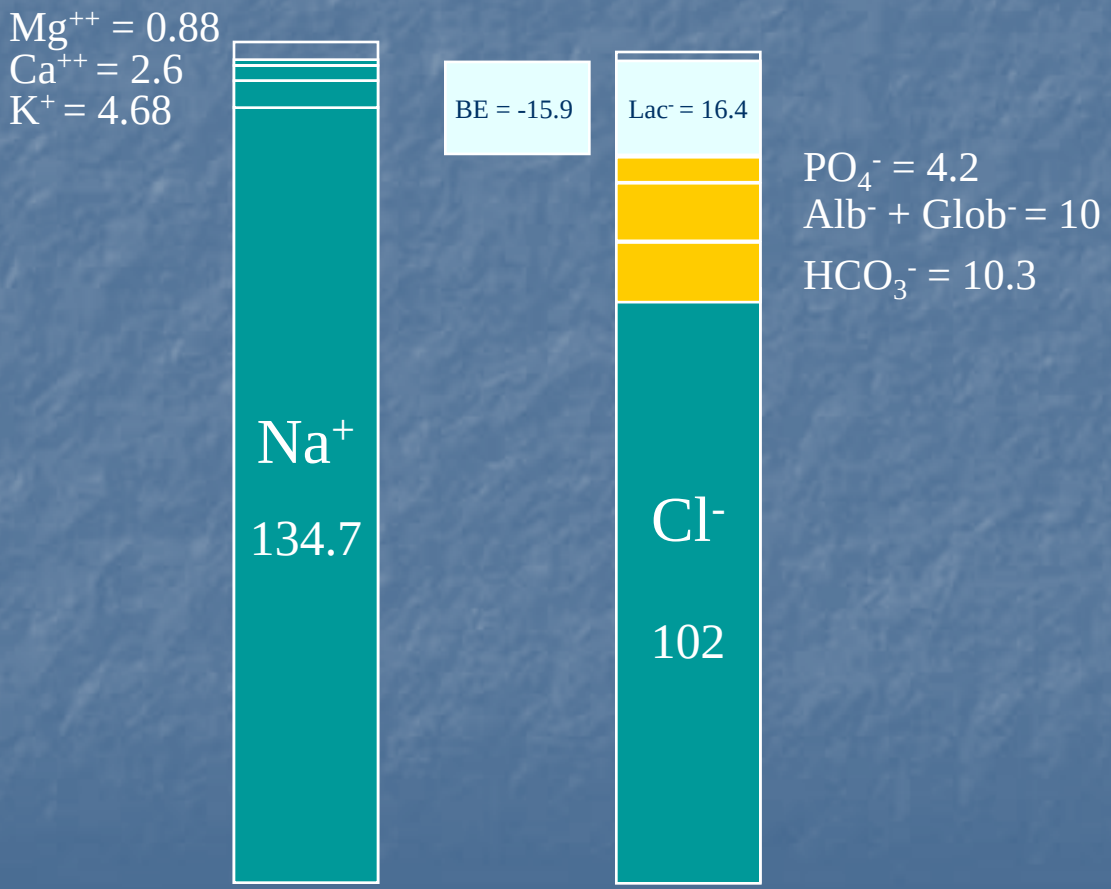


Standard Base Excess

- Buffer space
  - 1/3 normal hemoglobin
- Assumptions
  - Normal hemoglobin
  - Normal vascular/ECF ratio
  - Normal nonvolatile buffer
    - SBE_{corr} – Albumin, PO_4

Base Excess

Septic shock, NE		mEq/l
pH	7.195	
Pco_2	26.4	
SBE	-15.9 mmol/L	-15.9
Na	134.7 mmol/L	134.7
K	4.68 mmol/L	4.68
Cl	102 mmol/L	102
Ca^{++}	1.3 mmol/L	2.6
Mg^{++}	0.44 mmolL	0.88
Lac	16.4 mmol/L	16.4
PO_4	2.38 mmolL	4.34
Alb	23 g/L	7.2
Glob	20 g/L	2.8
HCO_3	10.3 mmol/L	10.3



Buffer Base

- Weak Acid Buffer
- Volatile Weak Acid
  - $H_2CO_3 \Leftrightarrow H^+ + HCO_3^-$
- Nonvolatile Weak Acids, A_{TOT}
  - Hemoglobin
  - Albumin (& Globulin)
  - Inorganic phosphate
- Weak acids
  - pK_a act as buffers

Cations/Anions
Weak Ion Acid Buffer

Cations

PO_4^-
Alb^- Hb^-

Anions

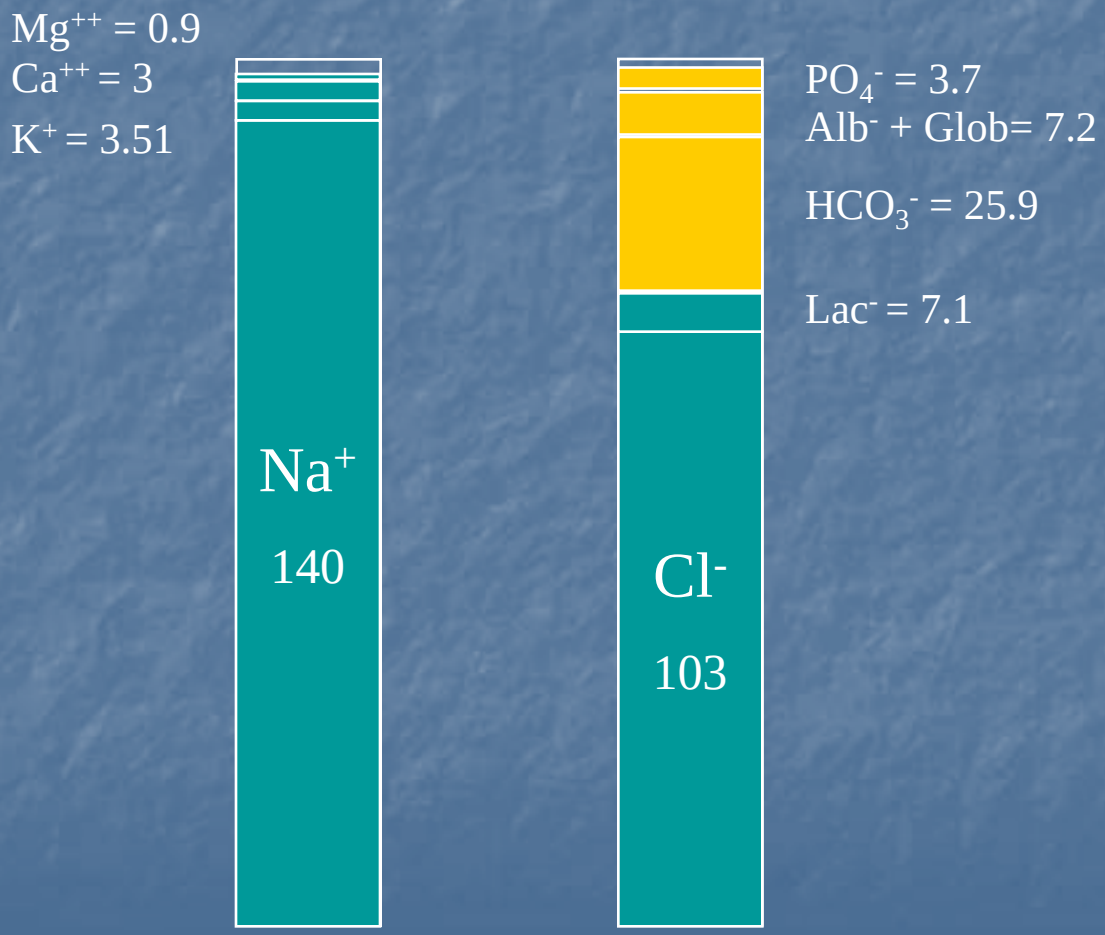
Calculating mEq/l

- Alb^- = (Alb) x ((0.123 x pH) – 0.631)
  - Alb^- = 0.28 x Alb
  - Horse: Alb^- = 0.225 x Alb [g/L]
  - Horse: $Glob^-$ = 0.14 x glob [g/L]
- PO_4^- = PO_4 x ((0.309 x pH) – 0.469)
  - Horse: $PO4^-$ = 1.83 x PO4 [mmol/L]

Acid-base calculator

Neonatal Encephalopathy		mEq/l
pH	7.295	
Pco_2	52.7	
SBE	1.2	1.2
Na	140 mmol/l	140
K	3.51 mmol/l	3.51
Cl	103 mmol/l	103
Ca^{++}	1.5 mmol/L	3
Mg^{++}	0.45 mmolL	0.9
Lac	7.1 mmol/l	7.1
PO_4	2.0 mmolL	3.7
Alb	21.8 g/L	4.9
Glob	16.2 g/L	2.3
HCO_3	25.9 mmol/l	25.9

Buffer Base



Anion Gap

Cations = Anions

$Na + K + Ca + Mg + UC = Cl + HCO_3 + Alb + PO_4 + UA$

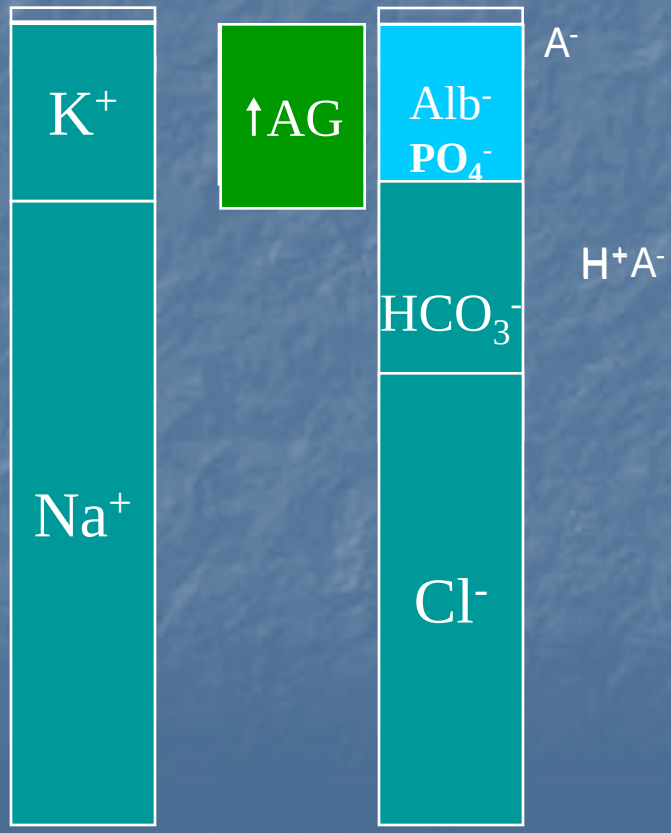
$(Na + K) – (Cl + HCO_3) = (Alb + PO_4 + UA) – (Ca + Mg + UC)$

$(Na + K) – (Cl + HCO_3) = UA - UC$

$(Na + K) – (Cl + HCO_3) = AG$

Cations/Anions
Anion Gap

$(Na + K) - (Cl + HCO_3) = AG$

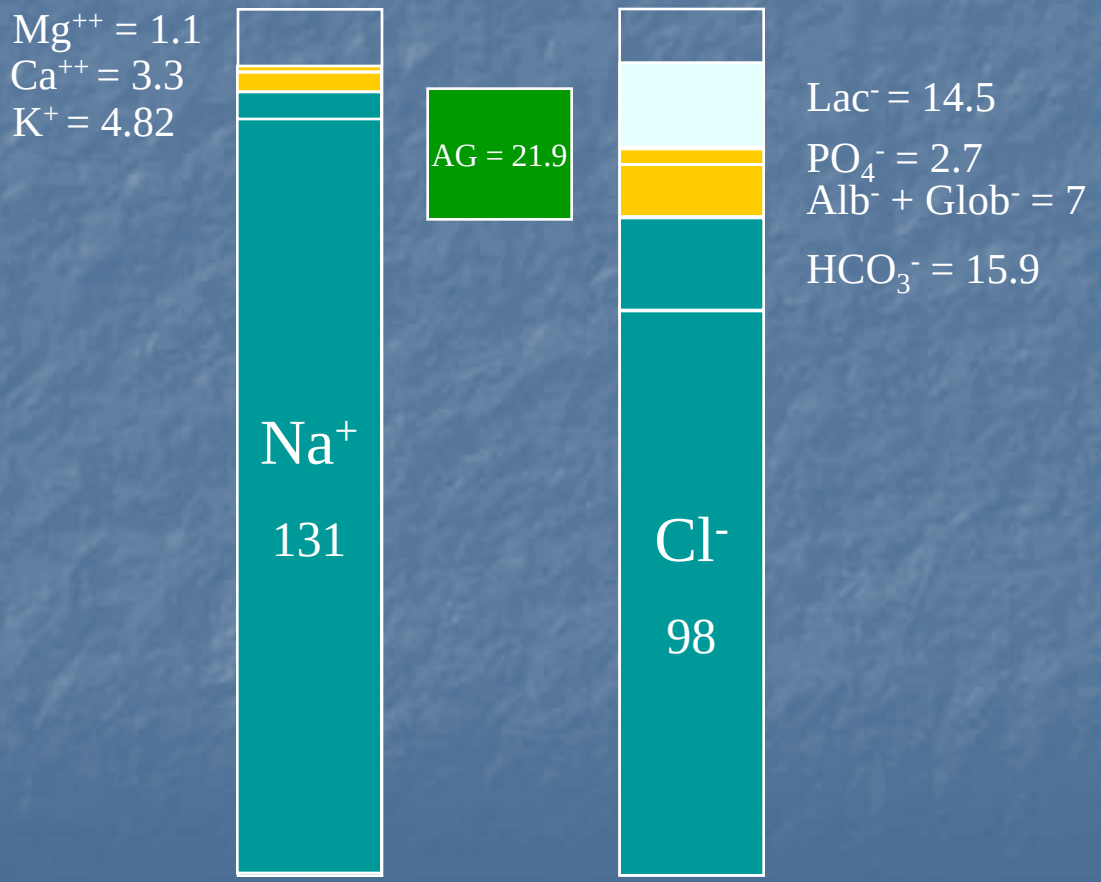


Cations

Anions

Anion Gap

Birth Asphyxia		mEq/l
pH	7.009	
Pco2	62.4	
AG	21.9 mmol/L	
Na	131 mmol/L	131
K	4.82 mmol/L	4.82
Cl	98 mmol/L	98
Ca^{++}	1.65 mmol/L	3.3
Mg^{++}	0.53 mmol/L	1.1
Lac	14.5 mmol/L	14.5
PO_4	1.61 mmol/L	2.9
Alb	27.8 g/L	6.3
Glob	19.2 g/L	2.7
HCO_3	15.9 mmol/L	15.9
SBE	-13.3	



Anion Gap

- Unidentified cations
  - Ca^{++}, Mg^{++}
  - Amines, many drugs
- Unidentified anions include Alb, PO_4
  - Low levels could mask presence of UA
  - High levels could mimic presence of UA
- Corrected AG
  - Corrected for Alb and Pi values
    - Acid pH

Stewart Approach

- Principles of physical chemistry
  - Electrical neutrality
  - Dissociation equilibriums
  - Conservation of mass
- Independent variables
  - SID
  - Weak acids (A_{TOT}) – buffer base
  - P_{CO2}

Strong Ions

- Inorganic
  - Na^{+}, Cl^{-}, K^{+}, $SO4^{--}$, Ca^{++}, and Mg^{++}
- Organic
  - Lactic acids
  - Tricarboxylic acids
  - Keto acids
- Strong organic anion
  - “footprint” or “ghost” of the strong acid

Cations/Anions

Cations

Anions

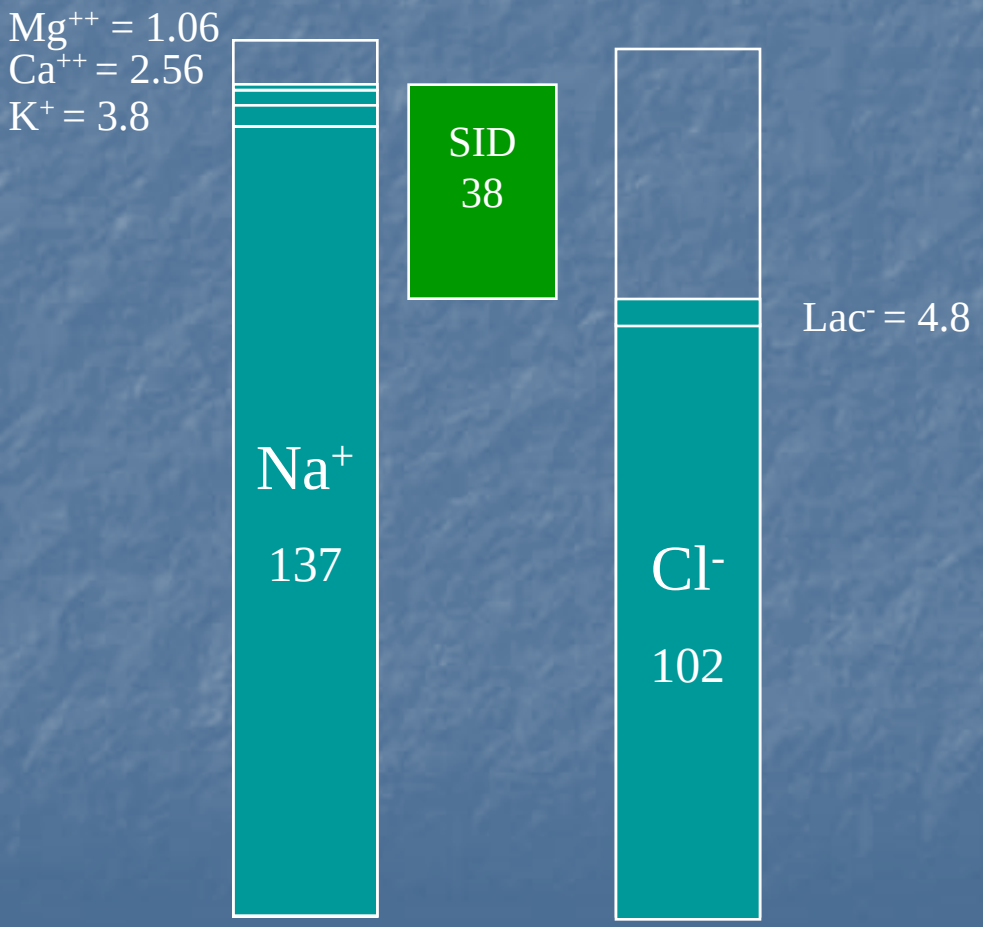
Strong Ions

Cations

Anions

Strong Ions

FIRS, Sepsis		mEq/l
pH	7.46	
Pco_2	39.8	
SID	38	
Na	137 mmol/L	137
K	3.8 mmol/L	3.8
Cl	102 mmol/L	102
Ca^{++}	1.28 mmol/L	2.56
Mg^{++}	0.53 mmol/L	1.06
Lac	4.8 mmol/L	4.8
PO_4	1.34 mmol/L	2.4
Alb	49 g/L	11
Glob	7.6 g/L	1.1
HCO_3	28.6 mmol/L	28.6
SBE	4.7	



SID

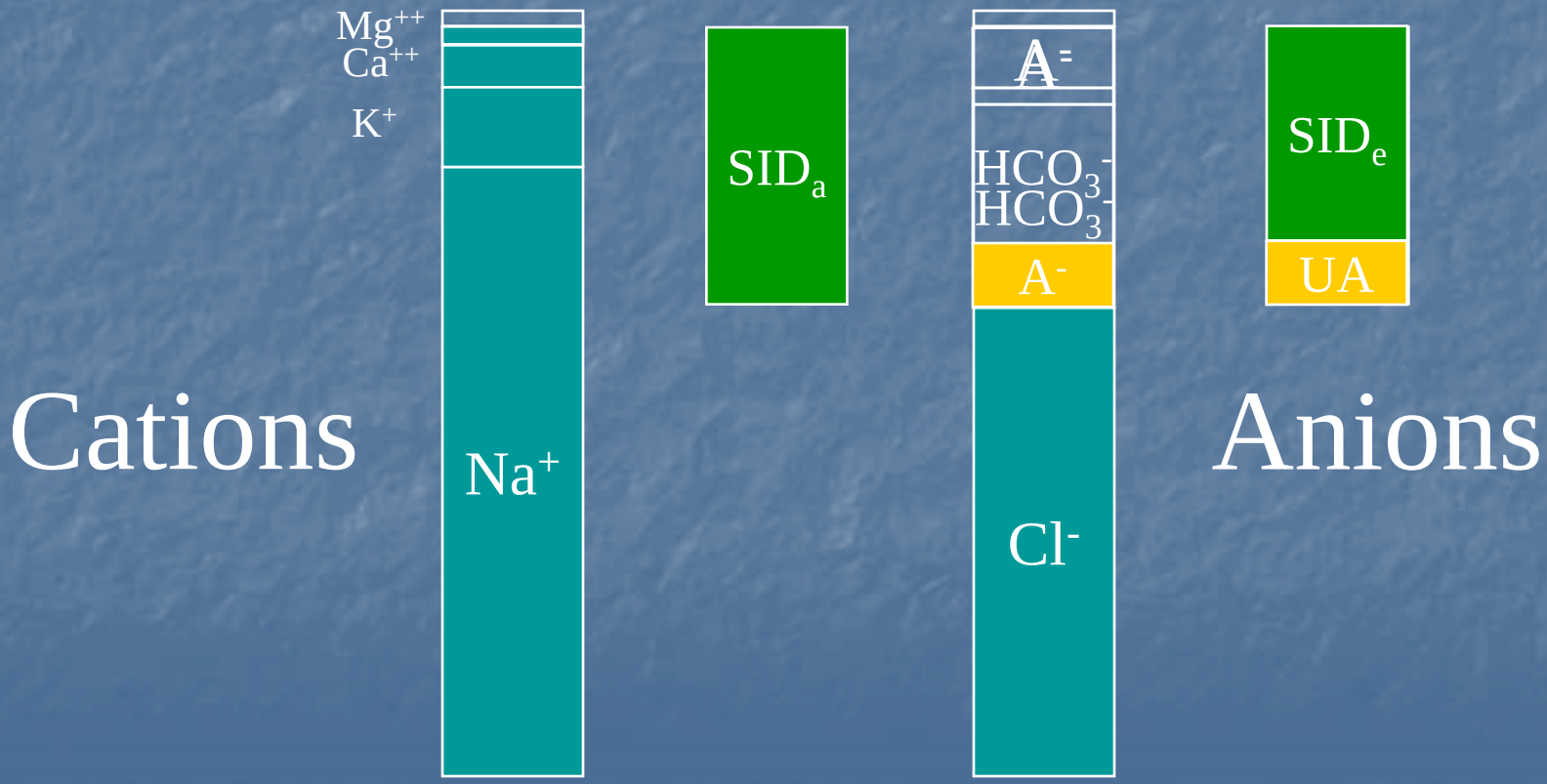
- Approximately 40±2
- Strong ion balance
  - SID > 40 - alkalizing
  - SID < 40 - acidifying
- Quantitate
- Hyper/hypochloremia - relative
  - Decrease Cl < decrease Na – acidosis
  - Decrease Cl > decrease Na – alkalosis

SIG

$SID_a = (Na + K + Ca + Mg) - (Cl + Lac)$

$SID_e = Alb^- + PO_4^- + HCO_3^-$

$SIG = SID_a - SID_e = UA - UC = 0$



SIG

- SIG = SIDa – SIDe
- SIG > 0 – unmeasured anions
  - Sepsis
  - Liver disease
  - If lactate is not part of SIDa, D-Lac
    - Most common cause of SIG > 0
    - Lactate mmol/l = SIG
- SIG < 0 – increased unidentified cations
- Can have mixed picture but UC very rare
- SIG does not change with
  - pH, Pco_2 changes
  - Changes in albumin, phosphate

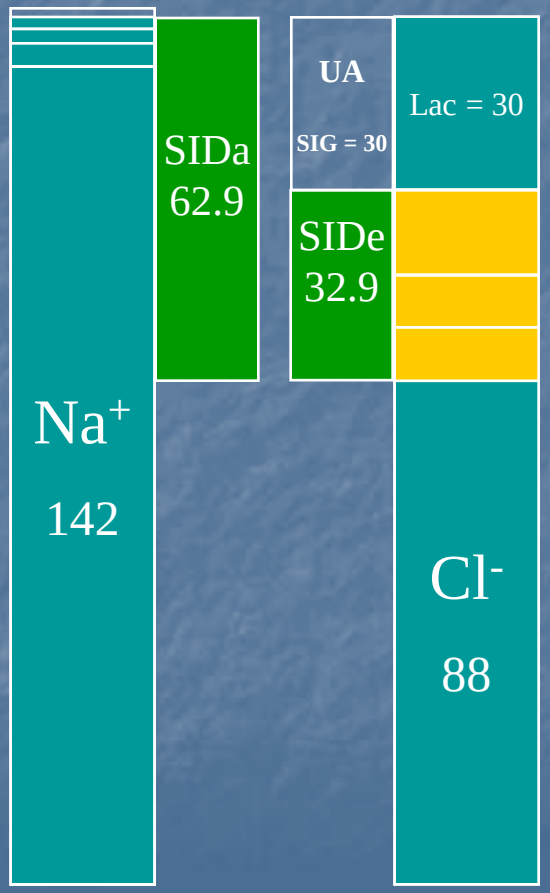
Intrauterine distress Birth asphyxia		mEq/l
pH	6.791	
Pco_2	59.6	
SIDa	62.9	
SIDe	32.9	
SIG	30	
Na	142 mmol/L	142
K	4.13 mmol/L	4.13
Cl	88 mmol/L	88
Ca^{++}	1.37 mmol/L	2.74
Mg^{++}	1.02 mmol/L	2.04
Lac	?? mmol/L	??
PO_4	8.98 mmol/L	16.4
Alb	29.7 g/L	6.7
Glob	17.3 g/L	2.4
HCO_3	9.2 mmol/L	9.2
SBE	-22.5 mEq/L	-22.5

SIG – UA

Mg^{++} = 2.04

Ca^{++} = 2.74

K^{+} = 4.13



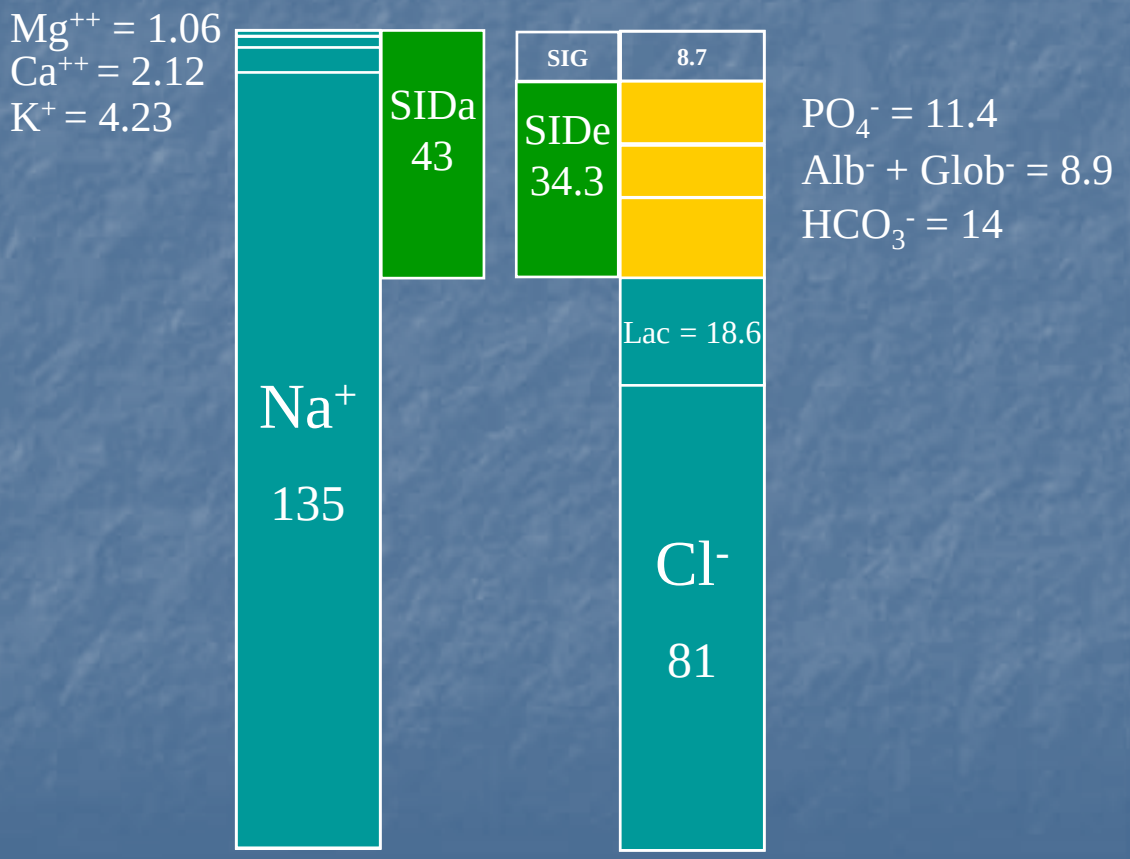
PO_4^{-} = 14.6

Alb^{-} + $Glob^{-}$ = 9.1

HCO_3^{-} = 9.2

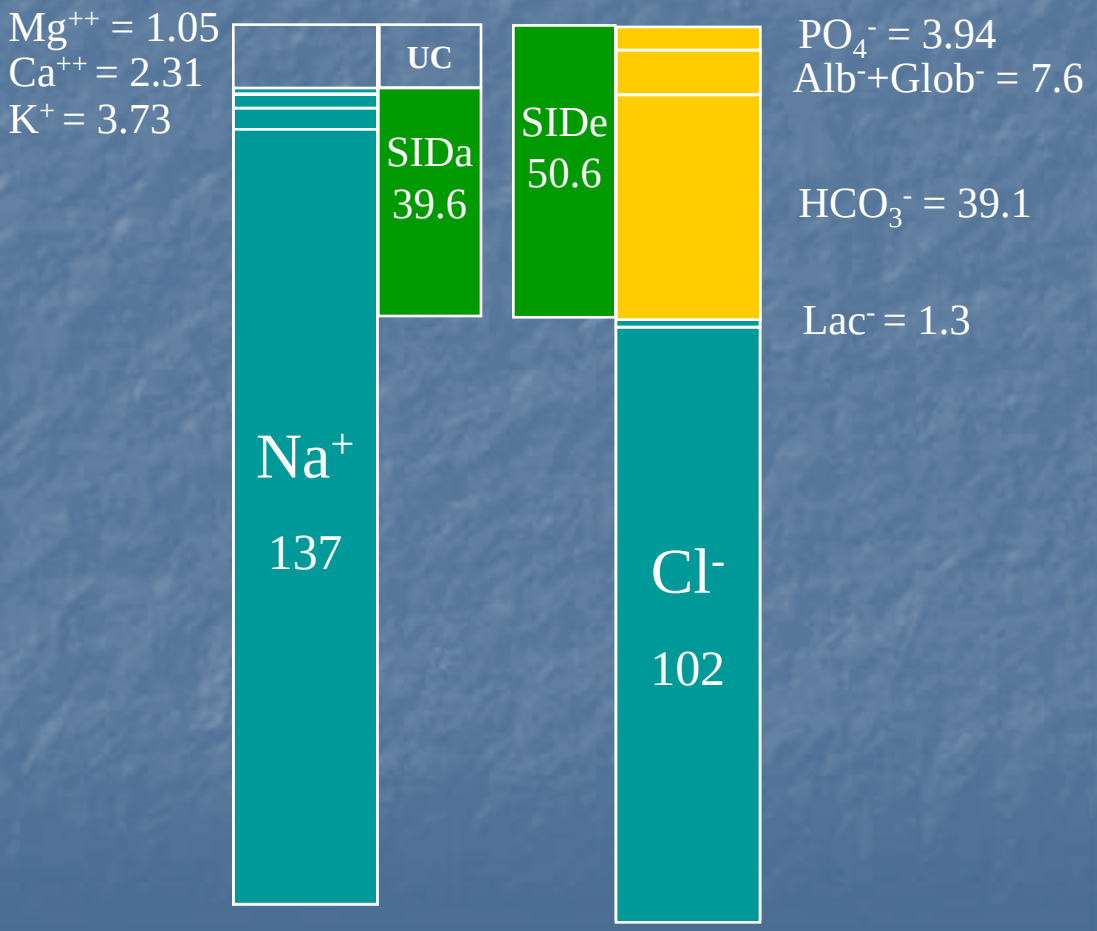
Case 91645		mEq/l
pH	7.088	
Pco_2	45.9	
SIDa	43	
SIDe	34.3	
SIG	8.7	
Na	135 mmol/L	135
K	4.23 mmol/L	4.23
Cl	81 mmol/L	81
Ca^{++}	1.06 mmol/L	2.12
Mg^{++}	0.53 mmol/L	1.06
ssLac	18.6 mmol/L	18.6
PO_4	6.63 mmol/L	12.1
Alb	28.9 g/L	6.5
Glob	17.1 g/L	2.4
HCO_3	14 mmol/L	14
SBE	-15.2 mEq/L	-15.2

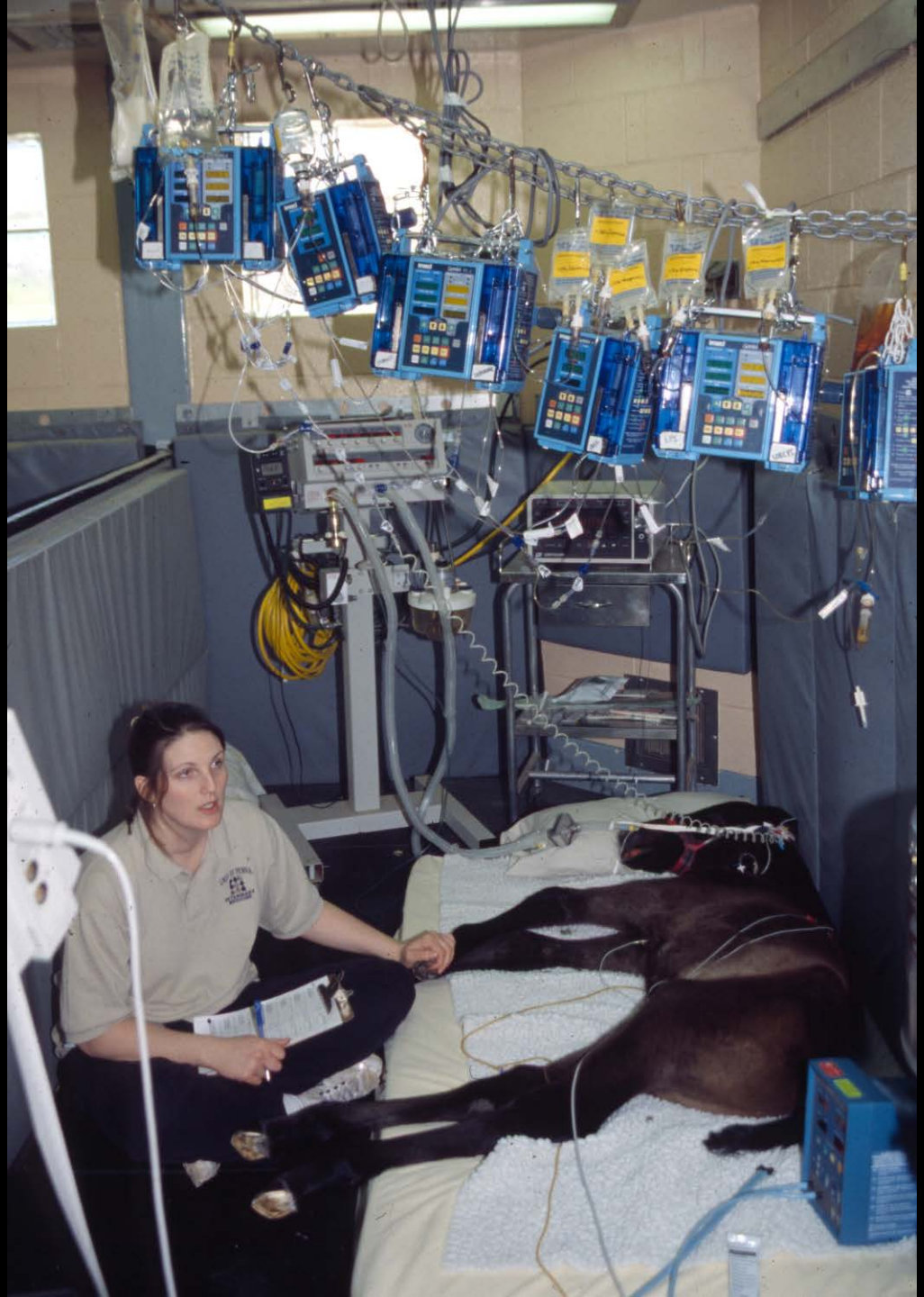
SIG – UA

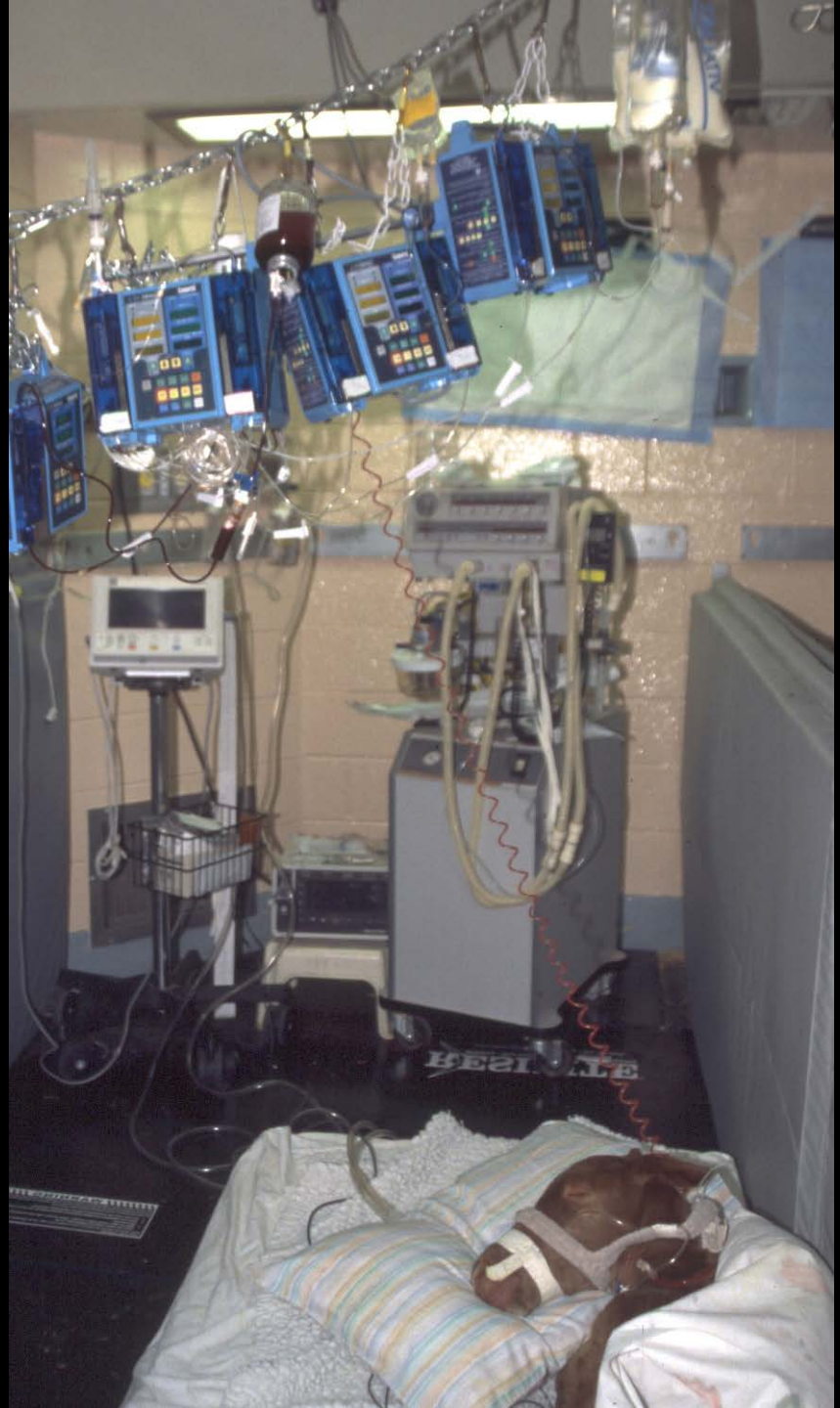


FIRS, Sepsis		mEq/l
pH	7.361	
Pco_2	68.3	
SIDa	39.6	
SIDe	50.6	
SIG	-11	
Na	137 mmol/L	137
K	3.73 mmol/L	3.73
Cl	102 mmol/L	102
Ca^{++}	1.16 mmol/L	2.31
Mg^{++}	0.42 mmol/L	0.84
Lac	1.3 mmol/L	1.3
PO_4	2.18 mmol/L	3.98
Alb	18.2 g/L	4.1
Glob	24.8 g/L	3.5
HCO_3	39.1 mmol/L	39.1
SBE	13.1	

SIG - UC





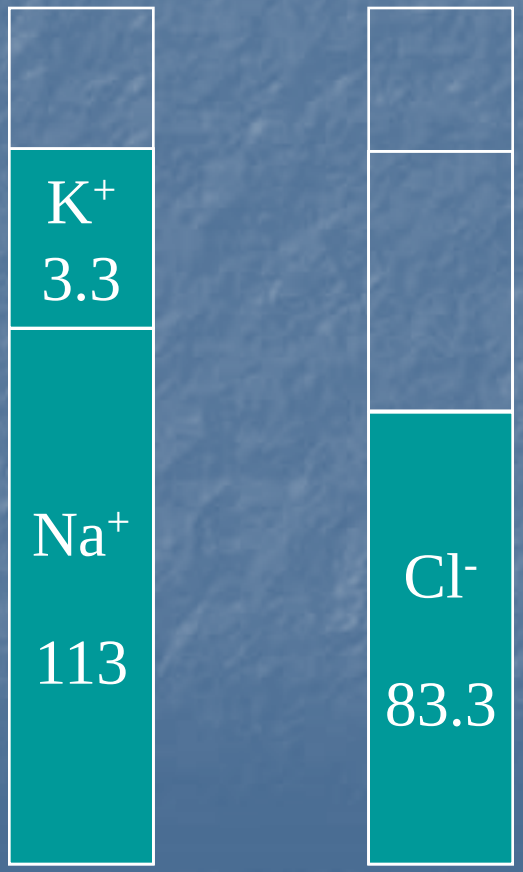


Metabolic Acid-Base Abnormalities

- Free water
  - Dilutional Acidosis
  - Contraction Alkalosis
- Hypochloremia/ Hyperchloremia
- Unidentified Anions/ Unidentified Cations
- Albumin/Phosphate concentrations

Dilutional Acidosis
Free Water

Na = 136
K = 4
Cl = 100
SID = 40



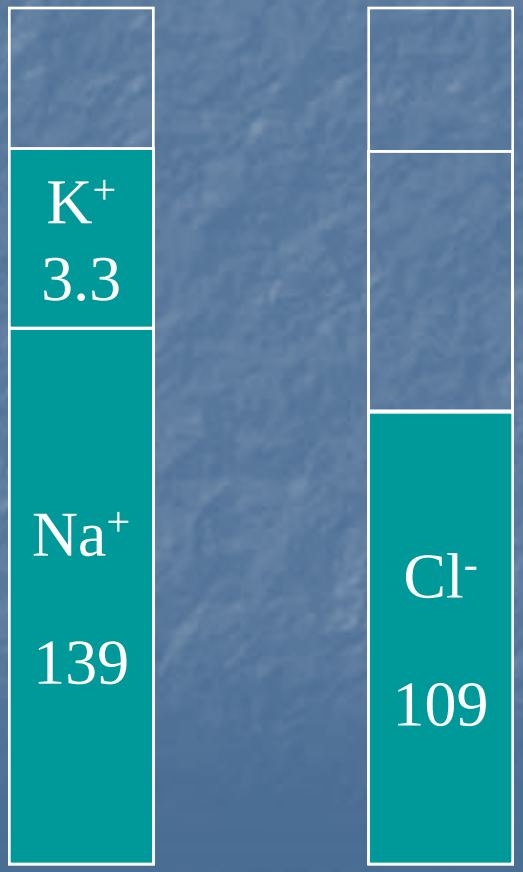
Add 20% water
Na = 113
K = 3.3
Cl = 83.3
SID = 33

Dilutional Acidosis

- Addition of free water (hyponatremia)
  - Will cause a decrease SID
  - Dilutional acidosis
    - Any osmotically active particle
      - Increase volume of ECF, no change in charge
      - Mannitol (before the diuresis)
      - Hyperglycemia
      - Ethylene glycol or methanol poisoning

Dilutional Acidosis
Saline

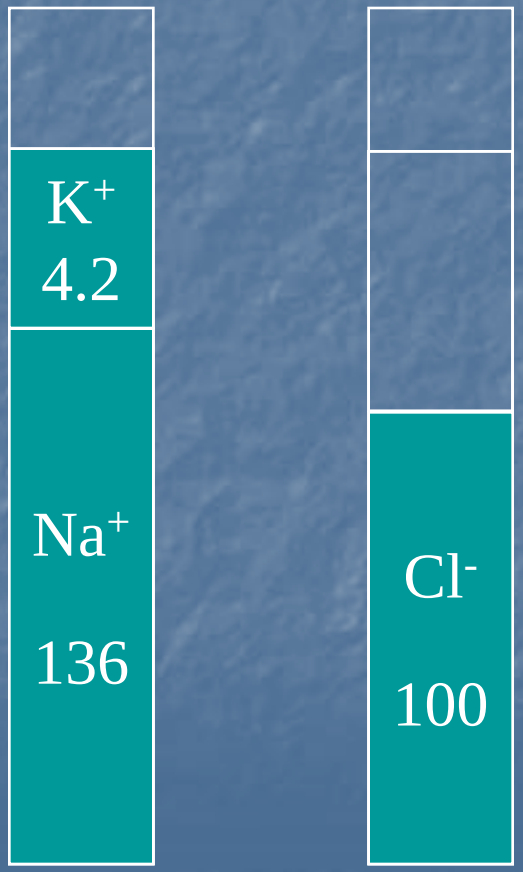
Na = 136
K = 4
Cl = 100
SID = 40



Add 20% saline
Na = 139
K = 3.3
Cl = 109
SID = 33.3

Dilutional Acidosis
Add SID balance fluid

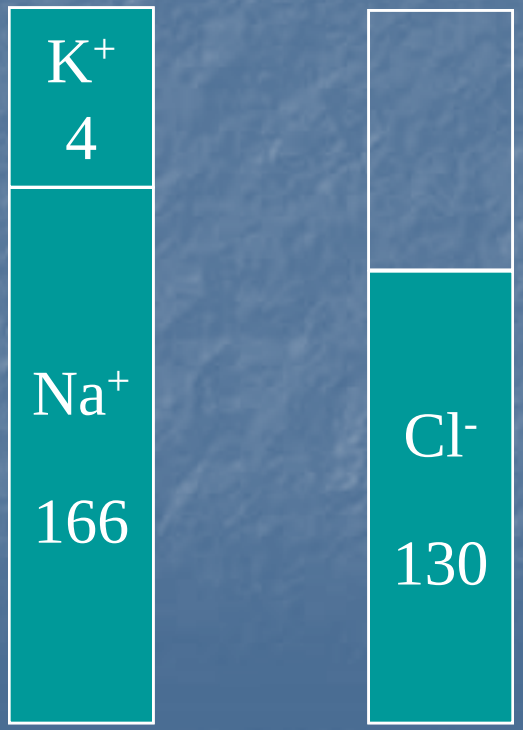
Na = 136
K = 4
Cl = 100
SID = 40



Add 20% Normisol R
Na = 137
K = 4.2
Cl = 100
SID = 41

Dilutional Acidosis
Add NaCl – no volume

Na = 136
K = 4
Cl = 100
SID = 40



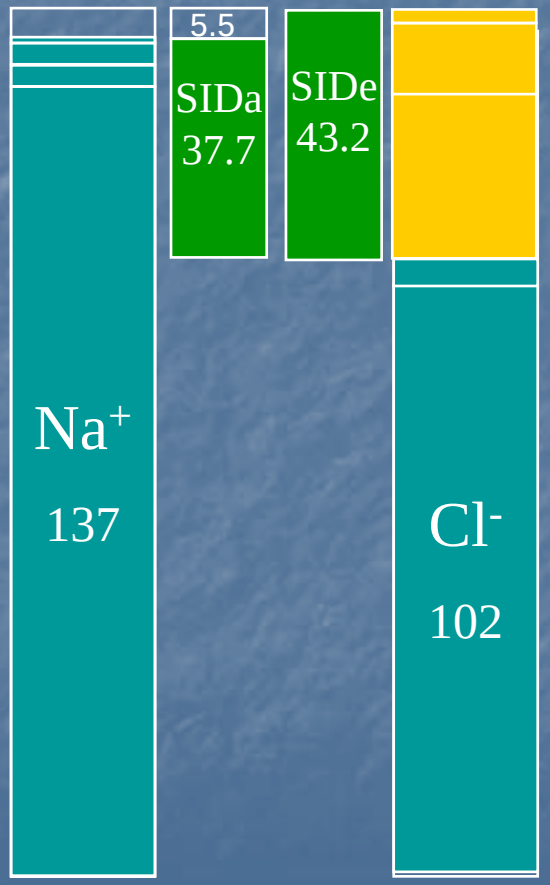
Add 30 mEq
Na = 166
K = 4
Cl = 130
SID = 40

Dilutional Acidosis

- Dilution effect
  - Depends on the SID of added fluid
  - Amount of fluid added
- How much of the SID is from free water?
- To correct for the free water effect
  - $Na_{ref}/Na_{measured}$
  - $Cl_{Corr} = (Na_{ref} /Na_{measured}) \times Cl^-_{measured}$
- Not that simple – in real life
  - Dilute Alb, PO_4
  - Alkalizing effect

Free Water

FIRS, Sepsis		mEq/l
SIDa		37.7
SIDe		43.2
SIG		-5.5
Na	137 mmol/L	137
K	3.8 mmol/L	3.8
Cl	102 mmol/L	102
Ca^{++}	1.28 mmol/L	2.56
Mg^{++}	0.53 mmol/L	1.05
Lac	4.8 mmol/L	4.8
PO_4	1.34 mmol/L	2.44
Alb	49 g/L	11
Glob	7.6 g/L	1.1
HCO_3	28.6 mmol/L	28.6
SBE	4.7	



PO_4^- = 2.4

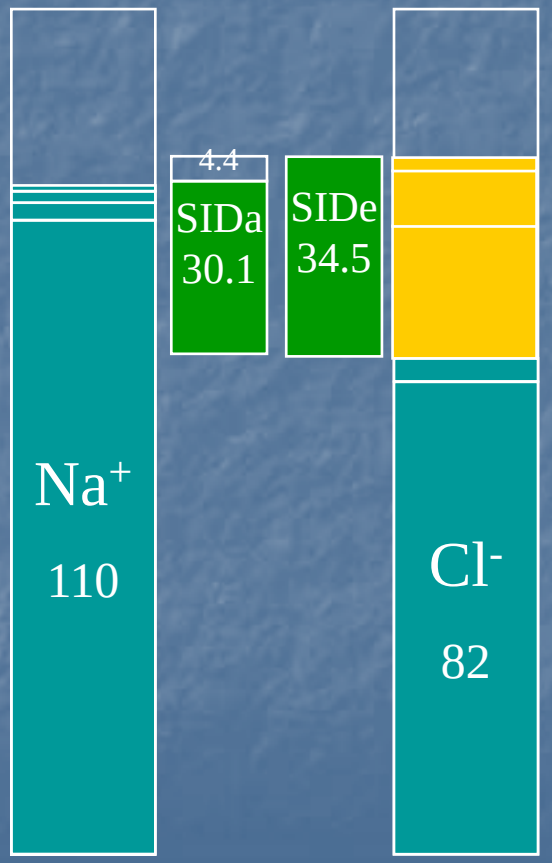
Alb^- + $Glob^-$ = 12.2

HCO_3^- = 28.6

Lac^- = 4.8

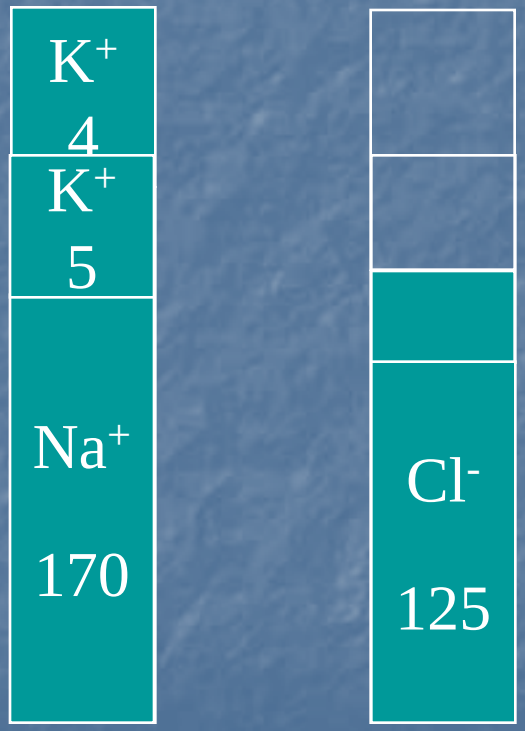
FIRS, Sepsis		mEq/L	mEq/L
SIDa		37.7	30.1
SIDe		43.2	34.5
SIG		-5.5	-4.4
Na	137 mmol/L	137	110
K	3.8 mmol/L	3.8	3.04
Cl	102 mmol/L	102	82
Ca^{++}	1.28 mmol/L	2.56	2.04
Mg^{++}	0.53 mmol/L	1.05	0.88
Lac	4.8 mmol/L	4.8	3.84
PO_4	1.34 mmol/L	2.44	1.95
Alb	49 mg/L	11	8.8
Glob	7.6 g/L	1.1	0.88
HCO_3	28.6 mmol/l	28.6	22.9
SBE	4.7		

Free Water +20% water



Contraction Alkalosis

Na = 136
K = 4
Cl = 100
SID = 40



Contract 20%
Na = 170
K = 5
Cl = 125
SID = 50

Hypochloremia
Hyperchloremia

- Normal renal handling of Cl
  - Renal acid-base control
  - Adjust SID by excreting Cl without Na
  - Diet – equal Na and Cl
- Abnormal renal handling of Cl
  - Renal Tubular Acidosis
  - Renal tubular disease

Hypochloremia
Hyperchloremia

- Hyperchloremic acidosis
  - Non-renal
    - GI losses Na
    - Excessive saline therapy
  - Renal
    - Renal compensation
    - RTA
- Hypochloremic alkalosis
  - Renal compensation
  - Chloriuresis (furosemide)
  - GI loss Cl
  - Contraction alkalosis (loss of free water)
    - Glucose diuresis

Unidentified Anions
Unidentified Cations

- Unidentified anions
  - L-lactate
  - D-lactate
  - Endogenous unidentified anions
    - Ketoacids
    - VFA
    - Sulfates
  - Exogenous organic unidentified anions
    - Salicylates
    - Methanol
    - Ethylene glycol

Unidentified Anions
Unidentified Cations

- Unidentified cations
  - Endogenous organic cations
    - Amines
  - Exogenous organic cations
    - Toxins
    - Drugs
- Detect unidentified anions/cations
  - Numbers don't "add up"
  - "Gap"
    - AG
    - SIG
  - Occurrence of unidentified cations
    - Can mask the presence of unidentified anions

Albumin/Phosphate Concentrations

- A_{TOT}, Buffer Base, weak acids
- Metabolic acidosis
  - Hyperphosphatemia
    - Renal failure, catabolism
  - Hyperalbuminemia
    - Hemoconcentration
    - Plasma/albumin therapy
- Metabolic alkalosis
  - Hypoalbuminemia
- Neonates
  - Hypoalbuminemia
  - Hyperphosphatemia

Differential Diagnosis Metabolic Acid-Base Disturbances

- Free water
  - Reflected in [Na]
- Chloride – inorganic SID
- Organic anions
- Organic cations
- Albumin level
- Phosphate level

Changes SIDa

- SID acidosis
  - Renal tubular acidosis
  - GIt - Diarrhea
  - Iatrogenic
- SID alkalosis
  - GIt
  - Diuretics/diuresis
  - Compensation for respiratory acidosis
  - Pathologic renal loses
  - Na loading – iatrogenic

SIG Acidosis

- Multiple sources
  - D-lactate
  - Intermediary metabolites
    - Ketones
    - Sulfates
  - Exogenous administered
    - Gelatins
    - Acetate, gluconate*, citrate
  - Acute phase proteins
  - Other inflammatory proteins
    - Cytokines
    - Chemokines
    - Other mediators

SIG Acidosis

- Accumulate - renal and liver dysfunction
  - Magnitude of the inflammatory response
  - Presence of organ dysfunction
- Prognostic significance
  - Lactic acidosis
  - SIG acidosis
  - Hyperchloremia
  - Respiratory acidosis

Mixed Acid Base Disorders

- One obvious disturbance
  - Inappropriate compensation
  - Separate primary disorder
- pH can be normal
  - Disorders cancel other's effects
- Compensation
  - P_{aCO2} and HCO_3 change in the same direction
  - Could be a mixed disorder
  - Excessive, insufficient, or appropriate

Mixed Acid Base Disorders

- Common in critically ill patients
  - Can lead to dangerous extremes of pH
- Four factors that determine pH
  - SIDa excluding lactate
  - Lactate plus and other organic anions (UA)
  - Abnormalities in the buffer base
  - Respiratory component

Mixed Acid Base Disorders

- Abnormalities of the SIDa (not lactate)
  - Chronic – time to develop and correct
  - Appropriate renal compensation
  - Primary abnormality
  - Na+K and Cl concentrations
    - Renal
    - Placental
    - Gastrointestinal

Mixed Acid Base Disorders

- Lactate, other organic anions (UA)
  - Abnormal intermediary metabolism
  - Can develop rapidly and resolve rapidly
  - Imply underlying pathophysiologic forces
- Abnormalities in the buffer base
  - Levels of plasma proteins and phosphate
  - Reflect underlying pathophysiology

Mixed Acid Base Disorders

- Respiratory component
  - Normal respiratory compensation
  - Underlying neuro-respiratory abnormalities
- Examining each part of the puzzle
  - Why pH is normal or abnormal
  - Understand underlying pathophysiology

Acid Base Disorders

- Prognosis
  - Underlying cause more important than degree
  - Not all acidosis equal
    - Dilution
    - Poisoning
    - Hyperchloremia
    - Saline infusions
    - Dysox - lactate production
    - Sepsis - lactate production

Metabolic Acid-Base Disturbances

Abnormality	Acidosis	Alkalosis
Abnormal SIDa		
Free water excess/deficit	Water excess = dilutional ↓ SID +↓[Na^+]	Water deficit = contraction ↑ SID ↑[Na^+]
Chloride	↓ SID ↑[Cl^-]	↑ SID +↓[Cl^-]
UA (e.g. D-lactate, keto acids)	↓ SID ↑[UA^-]	—
UC (e.g. organic cations)	—	↑ SID ↑[UC^+]
Abnormal Buffer Base, SIDe		
Albumin [Alb]	↑[Alb]	↓[Alb]
Phosphate [Pi]	↑[Pi]	↓[Pi]

Acid Base Case Problems

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5 yr QH Mare

- Presenting problem
  - Anorexia
  - Not responsive to omeprazole
- Physical Exam
  - Mild fever
  - Lethargic, anorexic
  - Gut sounds decreased
- Gastric distension – not resolve with fasting
- Periods excitement and compulsive walking

5 yr QH Mare

Cr	168 umol/L
BUN	10.57 mmol/L
Na	131.7 mmol/L; 132 mmol/L
K	3.16 mmol/L; 2.4 mmol/L
Cl	115.2 mmol/L; 109 mmol/L
HCO_3	10.3 mmol/L
Ca^{++}	1.883 mmol/L
Mg^{++}	?? mmol/L
Lac	<1 mmol/L
PO_4	0.95 mmol/L
Alb	33.4 g/L
Glob	31.0 g/L

pH	7.077
Pco2	34.9
HCO_3	10.3 mmol/L

U_{Na}	12 mmol/L
U_{Cl}	23 mmol/L
U_K	16 mmol/L
FX_{Na}	0.07 (0-0.7)
FX_{Cl}	0.11 (0.7-2.1)
FX_K	7 (15-200)
$(U_{Na} + U_K) - U_{Cl} = 28$	

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Case 1

- 30 hr old Friesian filly
- Septic Shock – *E. coli* bacteremia
- Other problems:
  - Mild pharyngeal collapse, Neonatal Encephalopathy, Neonatal Gastroenteropathy (reflux, dysmotility), hypothermia, premature ventricular contractions ...
- 12 day hospital stay

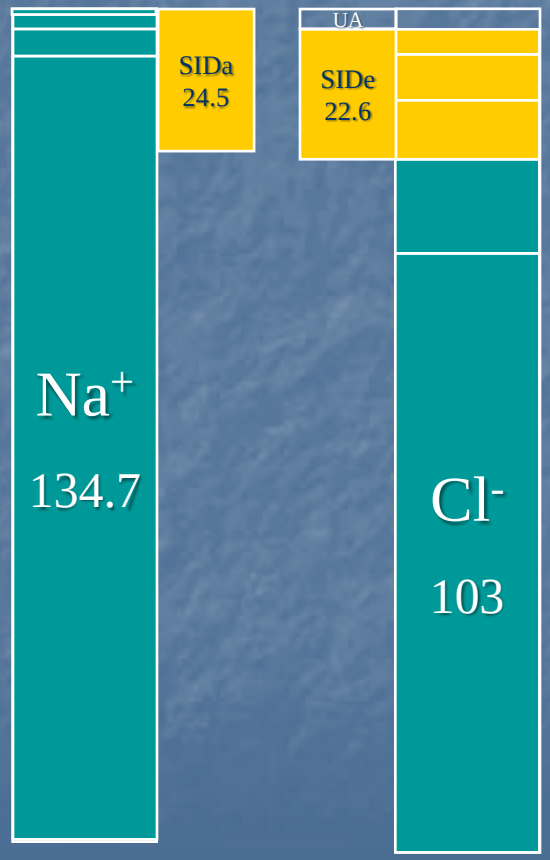
Case 1

Cr	398 umol/L
BUN	10 mmol/L
HCO_3	10.3 mmol/L
Na	134.7 mmol/L
K	4.68 mmol/L
Cl	102 mmol/L
Ca^{++}	1.33 mmol/L
Mg^{++}	0.44 mmol/L
Lac	16.4 mmol/L
PO_4	2.38 mmol/L
Alb	23 g/L
Glob	20 g/L

Case 1
Septic Shock

Septic Shock		mEq/L
pH	7.195	
Pco_2	26.4	
SBE	-15.9	
HCO_3	10.3 mmol/L	10.3
Na	134.7 mmol/L	134.7
K	4.68 mmol/L	4.68
Cl	102 mmol/L	102
Ca^{++}	1.33 mmol/L	2.66
Mg^{++}	0.44 mmol/L	0.88
Lac	16.4 mmol/L	16.4
PO_4	2.38 mmol/L	4.34
Alb	23 g/L	5.18
Glob	20 g/L	2.8

Mg^{++} = 0.88
Ca^{++} = 2.66
K^+ = 4.68



PO_4^- = 4.34
Alb^- + Glob= 7.98
HCO_3^- = 10.3

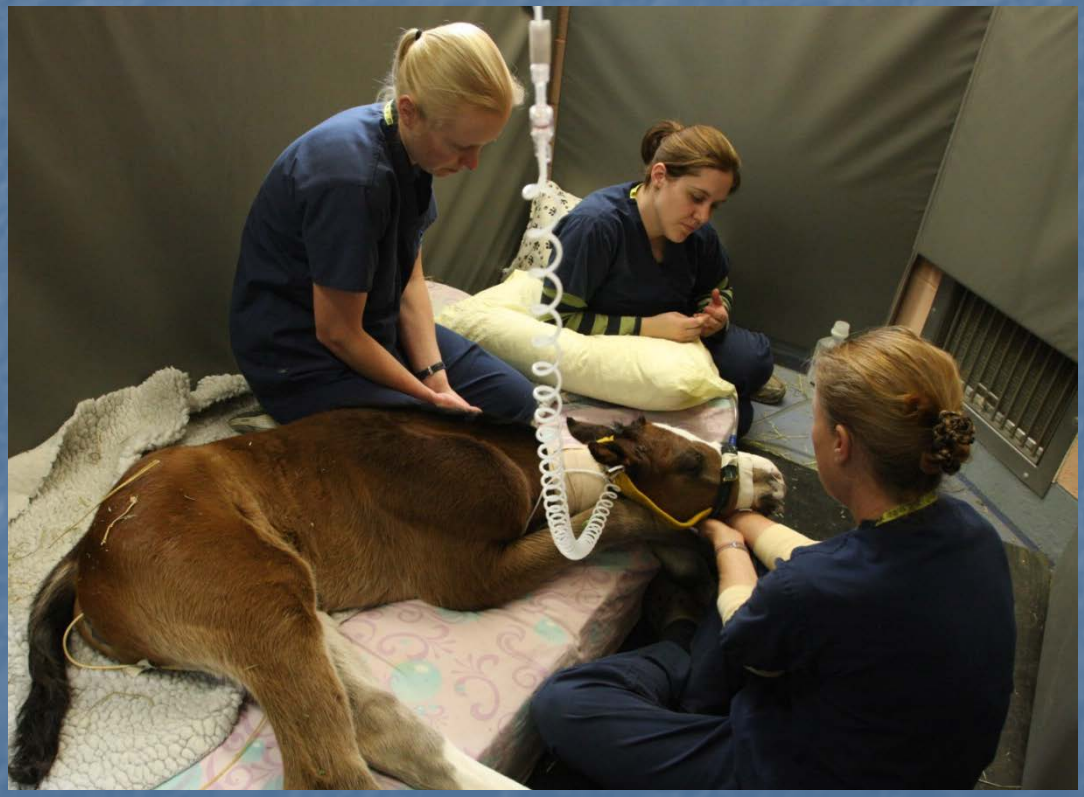
Lac^- = 16.4

Case 2

- 3 hr old STD filly
- Born 2:30 pm – attended
  - Premature placental separation
  - Birth arrhythmia
- Problems
  - Fetal distress
  - Neonatal Encephalopathy, hypothermia (29.4C), early urination (4 hr), lactic acidosis, hypochloremic alkalosis

Case 2

Cr	2378 umol/L
BUN	6.43 mmol/L
HCO_3	9.2 mmol/L
Na	142 mmol/L
K	4.13 mmol/L
Cl	88 mmol/L
Ca^{++}	1.37 mmol/L
Mg^{++}	1.02 mmol/L
Lac	?? mmol/L
PO_4	8.98 mmol/L
Alb	29.7 g/L
Glob	17.3 g/L



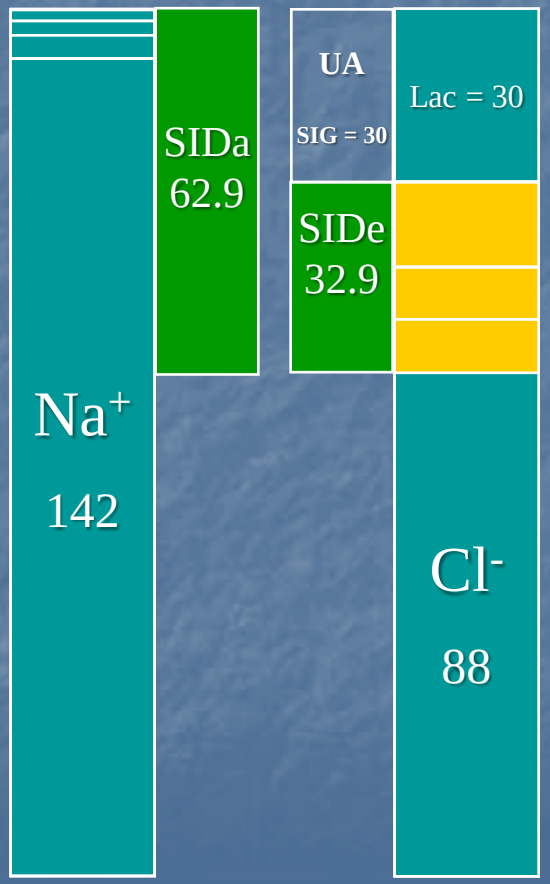
Case 2
UA

		mEq/L
pH	6.791	
Pco_2	59.6	
HCO_3	9.2 mmol/L	9.2
SBE	-22.5 mEq/L	-22.5
Na	142 mmol/L	142
K	4.13 mmol/L	4.13
Cl	88 mmol/L	88
Ca^{++}	1.37 mmol/L	2.74
Mg^{++}	1.02 mmol/L	2.04
Lac	??	??
PO_4	8.98 mmol/L	16.4
Alb	29.7 g/L	6.7
Glob	17.3 g/L	2.4

Mg^{++} = 2.04

Ca^{++} = 2.74

K^{+} = 4.13



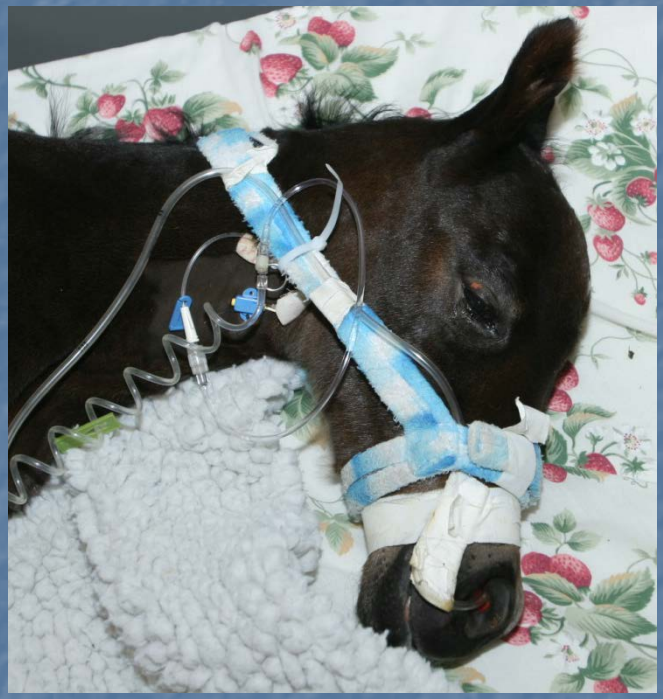
PO_4^{-} = 16.4

Alb^{-} + $Glob^{-}$ = 9.1

HCO_3^{-} = 9.2

Case 3

- 16 hr old STD filly
- History
  - Foaling unexpected
    - Born in a field
  - Never stood
  - Nursed from bottle
- Problems
  - FIRS/IUGR/prematurity (4 weeks)
  - NE
  - NG (distension, fetal diarrhea)
  - Sepsis, coagulopathy, hypoperfusion
  - Developed respiratory failure
    - $Paco_2$ > 100
    - Refused ventilation



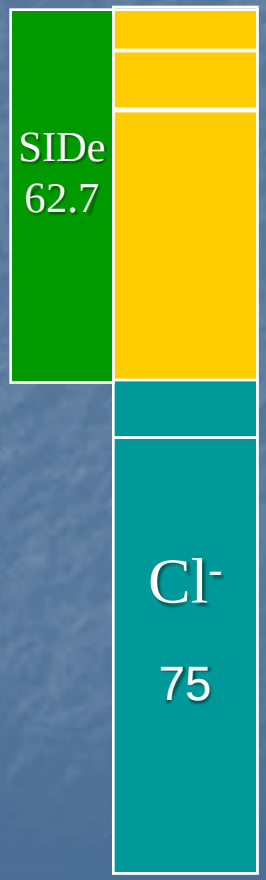
Case 3

Cr	488.9 umol/L
BUN	10 mmol/L
HCO_3	46.1 mmol/L
Na	142 mmol/L
K	3.4 mmol/L
Cl	75 mmol/L
Ca^{++}	1.17 mmol/L
Mg^{++}	0.32 mmol/L
Lac	9.8 mmol/L
PO_4	3.71 mmolL
Alb	24.5 g/L
Glob	30.5 g/L

Case 3

		mEq/L
pH	7.424	
Pco_2	69.8	
HCO_3	46.1 mmol/L	46.1
SBE	20.5	20.5
Na	142 mmol/L	142
K	3.4 mmol/L	3.4
Cl	75 mmol/L	75
Ca^{++}	1.17 mmol/L	2.35
Mg^{++}	0.32 mmol/L	0.65
Lac	9.8 mmol/L	9.8
PO_4	3.71 mmolL	6.79
Alb	24.5 g/L	5.51
Glob	30.5 g/L	4.3

Mg^{++} = 0.68
Ca^{++} = 2.35
K^+ = 3.4



PO_4^- = 6.79
Alb^- + Glob= 9.81
HCO_3^- = 46.1
Lac^- = 9.8

Case 4

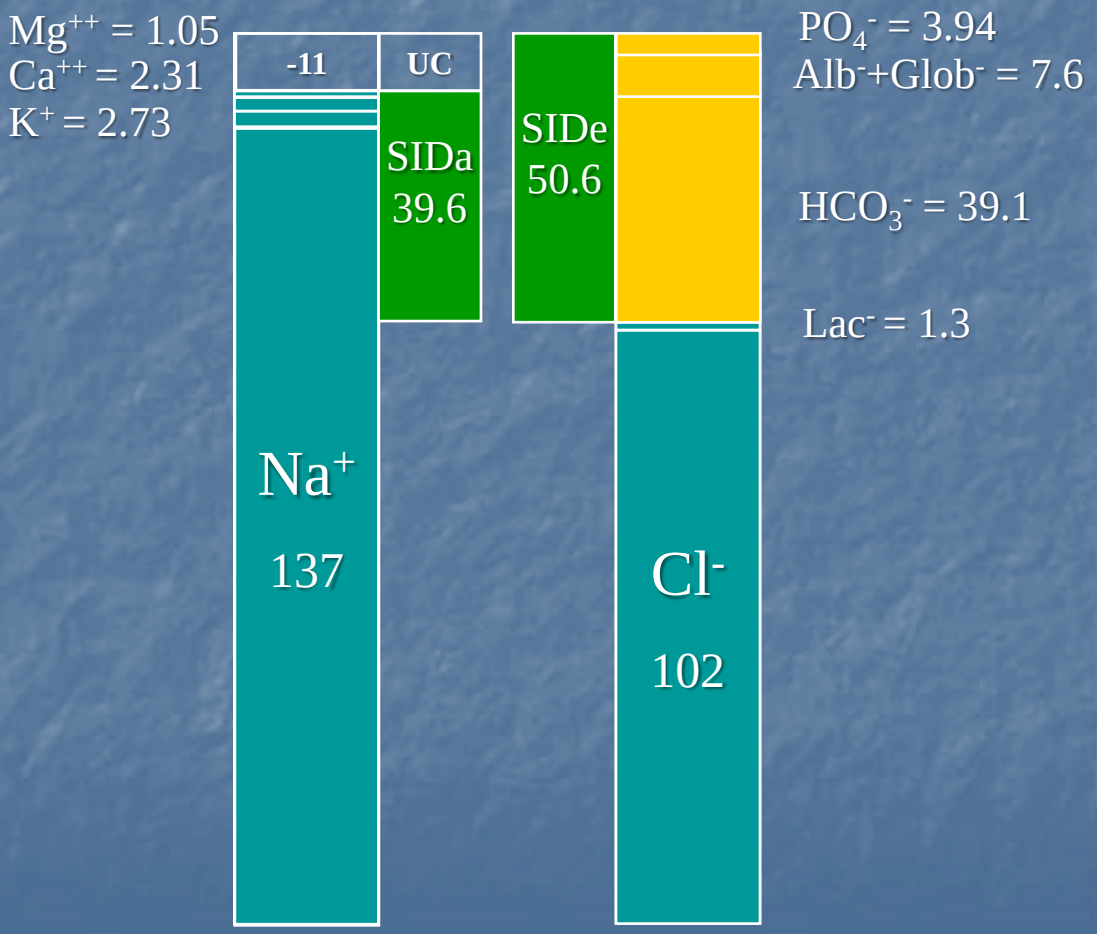
- 4.5 hr STD filly
- History
  - Mare dripping fluids in field
  - Foaled uneventfully
  - Passed membranes 20 minutes
- Problems
  - Fetal distress – Cr 9.6, fetal diarrhea
  - NE (weakness), NN (slow Cr drop), NG (dsymotility)
- 24 day hospital stay

Case 4

Cr	831 umol/L
BUN	8.6 mmol/L
HCO3	39.1 mmol/L
Na	137 mmol/L
K	2.73 mmol/L
Cl	102 mmol/L
Ca^{++}	1.16 mmol/L
Mg^{++}	0.42 mmolL
Lac	1.3 mmol/L
PO_4	2.18 mmolL
Alb	18.2 g/L
Glob	24.8 g/L

Case 4
UC

		mEq/L
pH	7.361	
Pco_2	68.3	
HCO_3	39.1 mmol/L	39.1
SBE	11.6	11.6
Na	137 mmol/L	137
K	2.73 mmol/L	2.73
Cl	102 mmol/L	102
Ca^{++}	1.16 mmol/L	2.31
Mg^{++}	0.42 mmolL	0.84
Lac	1.3 mmol/L	1.3
PO_4	2.18 mmolL	3.98
Alb	18.2 g/L	4.1
Glob	24.8 g/L	3.5



Case 5

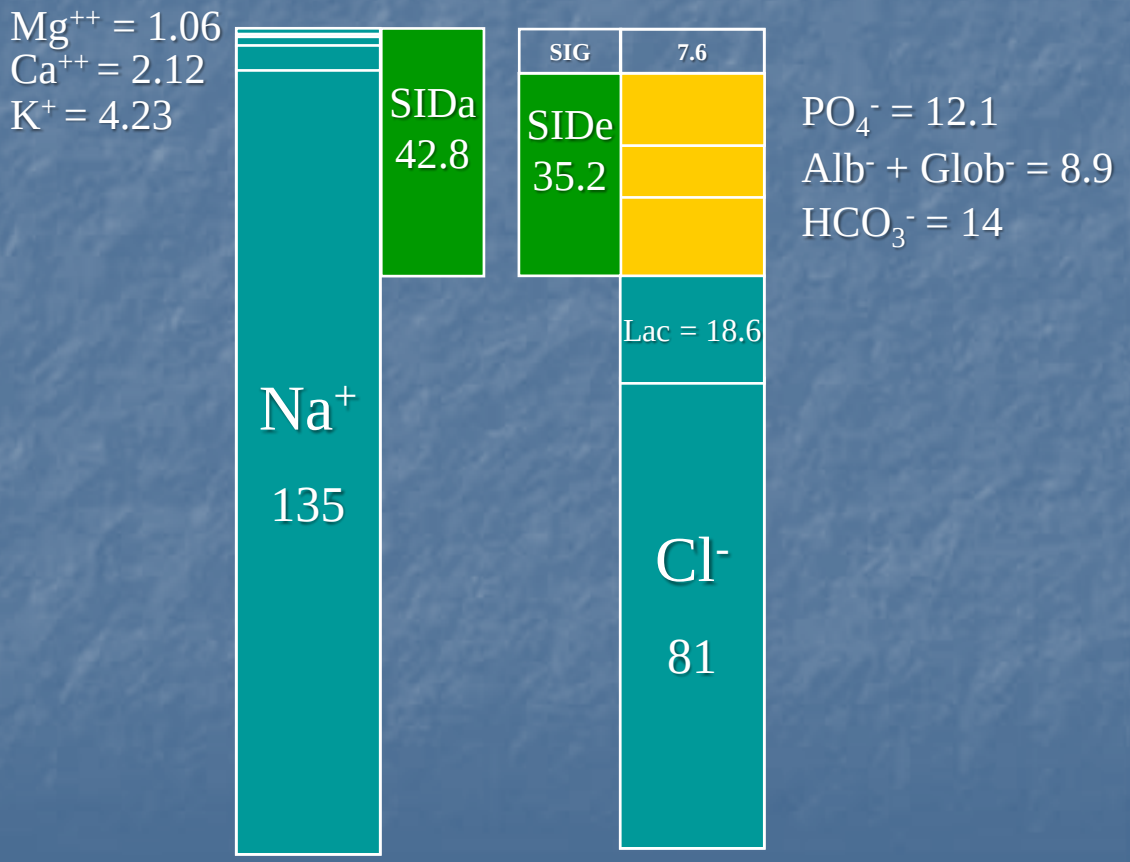
- 45 min TB colt
- Histroy
  - Mare found passing fetal fluids
  - Eventration of intestines through vagina
  - Dystocia
  - Foal not breathing
    - 3-4 min ventilation
    - Spontaneous breathing, became active
- Problems
  - Intrauterine challenge
  - Fractured ribs
  - Neonatal Encephalopathy
  - Neonatal Vasogenic Nephropathy
  - Necrotizing enterocolitis
- Outcome – euthanized at 48 hr
  - Fractured ribs (8 lt side)
  - NEC (reflux, feeding intolerance)
  - Renal disease

Case 5

Cr	1485 umol/L
BUN	7.8 mmol/L
HCO_3	14 mmol/L
Na	135 mmol/L
K	4.23 mmol/L
Cl	81 mmol/L
Ca^{++}	1.06 mmol/L
Mg^{++}	0.51 mmol/L
Lac	18.6 mmol/L
PO_4	6.62 nnol/L
Alb	28.9 g/L
Glob	17.1 g/L

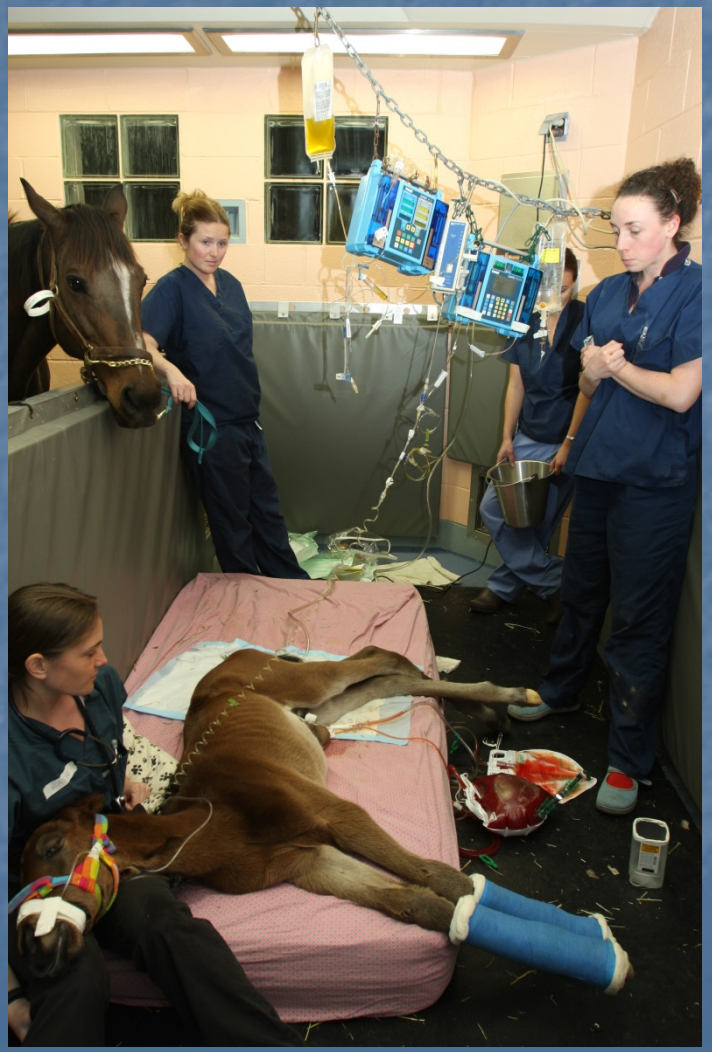
Case 5
UA

		mEq/L
pH	7.088	
Pco_2	45.9	
HCO_3	14 mmol/L	14
SBE	-15.2	-15.2
Na	135 mmol/L	135
K	4.23 mmol/L	4.23
Cl	81 mmol/L	81
Ca^{++}	1.06 mmol/L	2.12
Mg^{++}	0.51 mmol/L	1.06
Lac	18.6 mmol/L	18.6
PO_4	6.62 nnol/L	12.1
Alb	28.9 g/L	6.5
Glob	17.1 g/L	2.4



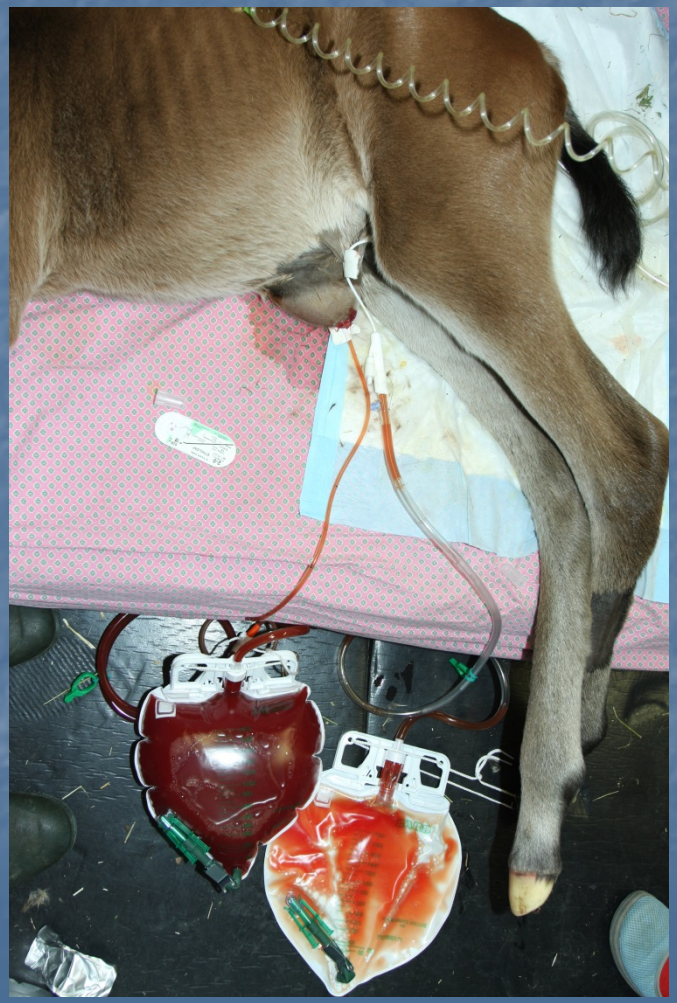
Case 6

- 5.5 hr TB colt
- History
  - Unremarkable birth
  - Weakness from birth
    - Unable to rise unassisted
  - Large umbilical hernia
- Problems
  - Fractured ribs
  - Neonatal Encephalopathy, FIRS
  - Urinary
    - NN
    - Patent urachus
    - Urachal tear – uroperitoneum
    - Megacystis
  - Other
    - Entropion, pastern contracture, colic
- 8 day hospital stay



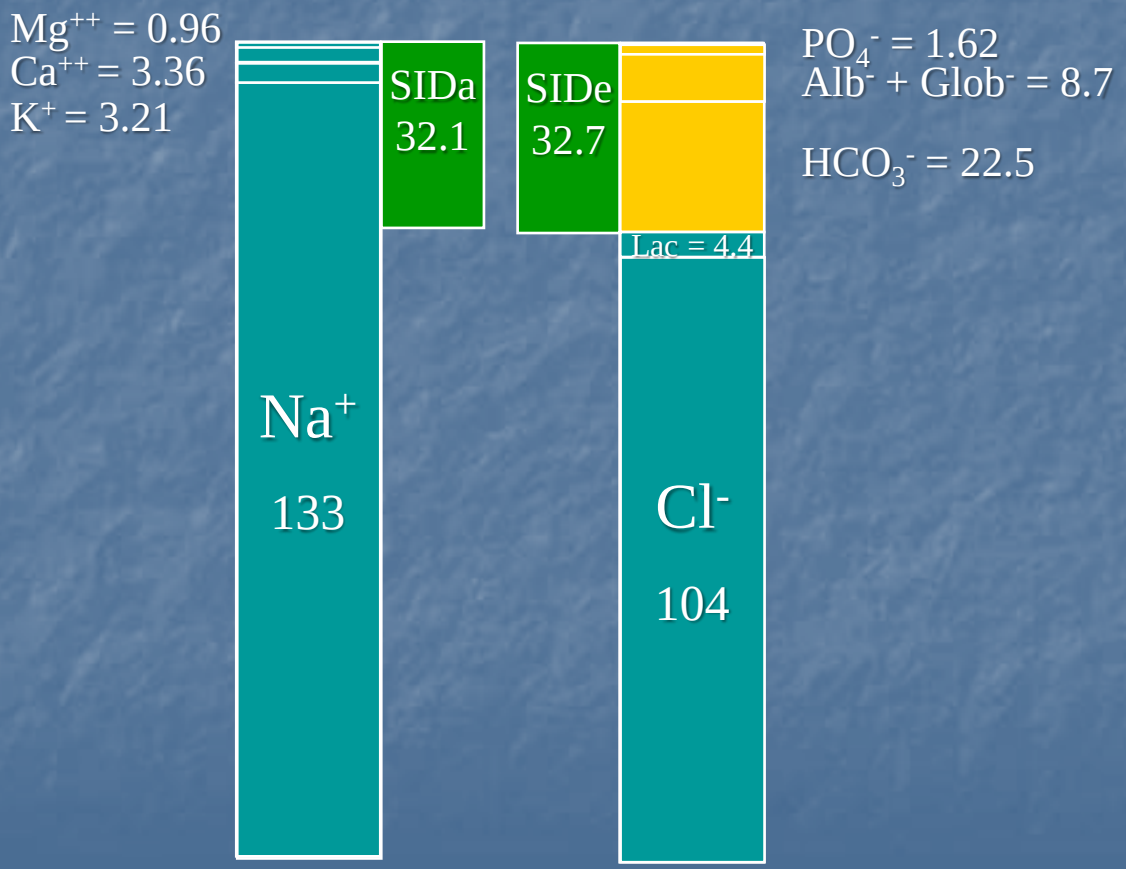
Case 6

Cr	1087 umol/L
BUN	10.0 mmol/L
HCO_3	22.5 mmol/L
Na	133 mmol/L
K	3.21 mmol/L
Cl	104 mmol/L
Ca^{++}	1.68 mmol/L
Mg^{++}	0.48 mmol/L
Lac	4.4 mmol/L
PO_4	0.885 mmol/L
Alb	24.1 g/L
Glob	22.9 g/L



Case 6

		mEq/L
pH	7.329	
Pco_2	42.5	
HCO_3	22.5 mmol/L	
SBE	-2.9	
Na	133 mmol/L	133
K	3.21 mmol/L	3.21
Cl	104 mmol/L	104
Ca^{++}	1.68 mmol/L	3.36
Mg^{++}	0.48 mmol/L	0.96
Lac	4.4 mmol/L	4.4
PO_4	0.885 mmol/L	1.62
Alb	24.1 g/L	5.42
Glob	22.9 g/L	3.3



Case 7

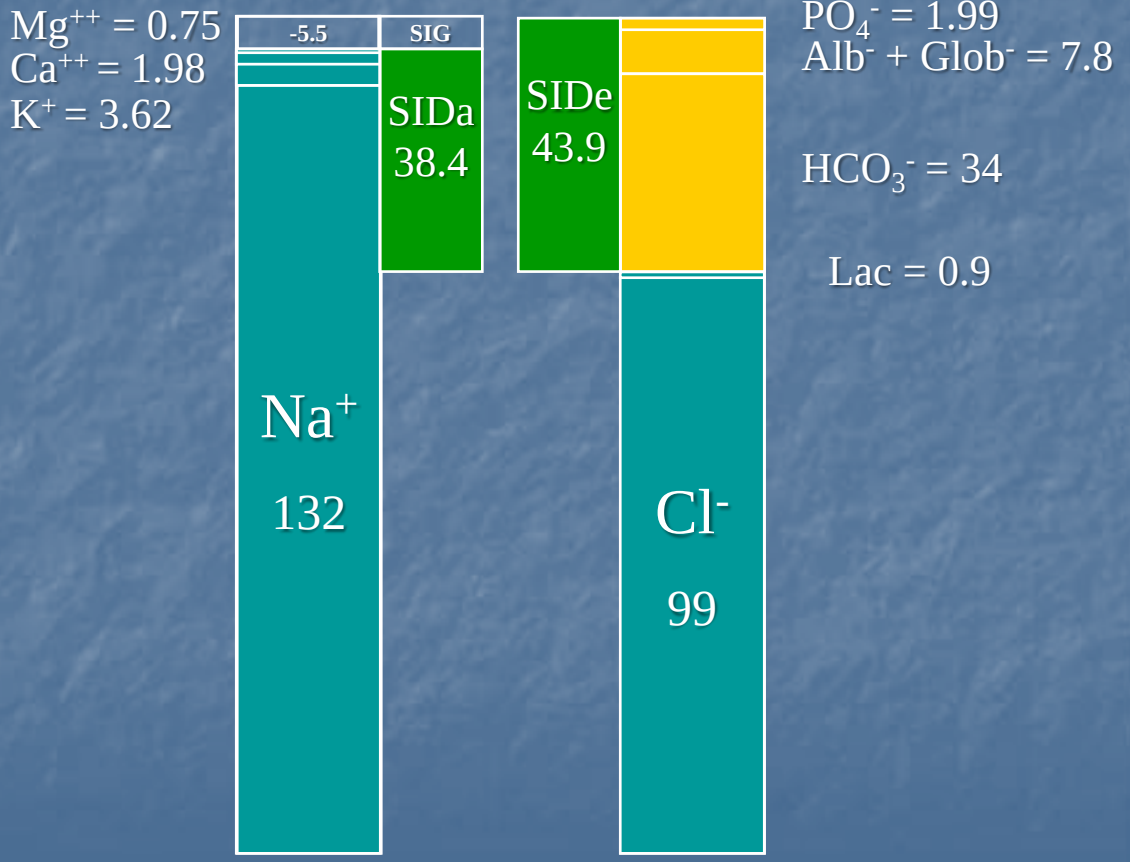
- 20 hr old TB filly
- History
  - Evidence of significant umbilical bleeding
  - Down, cold, lethargic, pale
    - HR 80
    - Rx 500 ml whole blood from mare, fluids, dextrose
  - 'Perked up‘
    - But some bleeding – unsuccessful suture/clamp artery
    - Nursing?
  - Fade again
  - Referred for blood transfusion
- Problems
  - Umbilical remnant hematoma
  - NE, anemia (20%), sepsis, NN, hepatopathy, NG, contracture
- 10 day hospital course

Case 7

Cr	150.3 umol/L
BUN	3.93 mmol/L
HCO_3	34 mmol/L
Na	132 mmol/L
K	3.62 mmol/L
Cl	99 mmol/L
Ca^{++}	0.95 mmol/L
Mg^{++}	0.375 mmol/L
Lac	0.9 mmol/L
PO_4	1.09 mmol/L
Alb	20.1 g/L
Glob	23.9 g/L

Case 7
UC

		mEq/L
pH	7.455	
Pco_2	48.6	
HCO_3	34 mmol/L	34
SBE	10.3	10.3
Na	132 mmol/L	132
K	3.62 mmol/L	3.62
Cl	99 mmol/L	99
Ca^{++}	0.95 mmol/L	1.89
Mg^{++}	0.375 mmol/L	0.75
Lac	0.9 mmol/L	0.9
PO_4	1.09 mmol/L	1.99
Alb	20.1 g/L	4.52
Glob	23.9 g/L	3.3



Case 7

Case 8

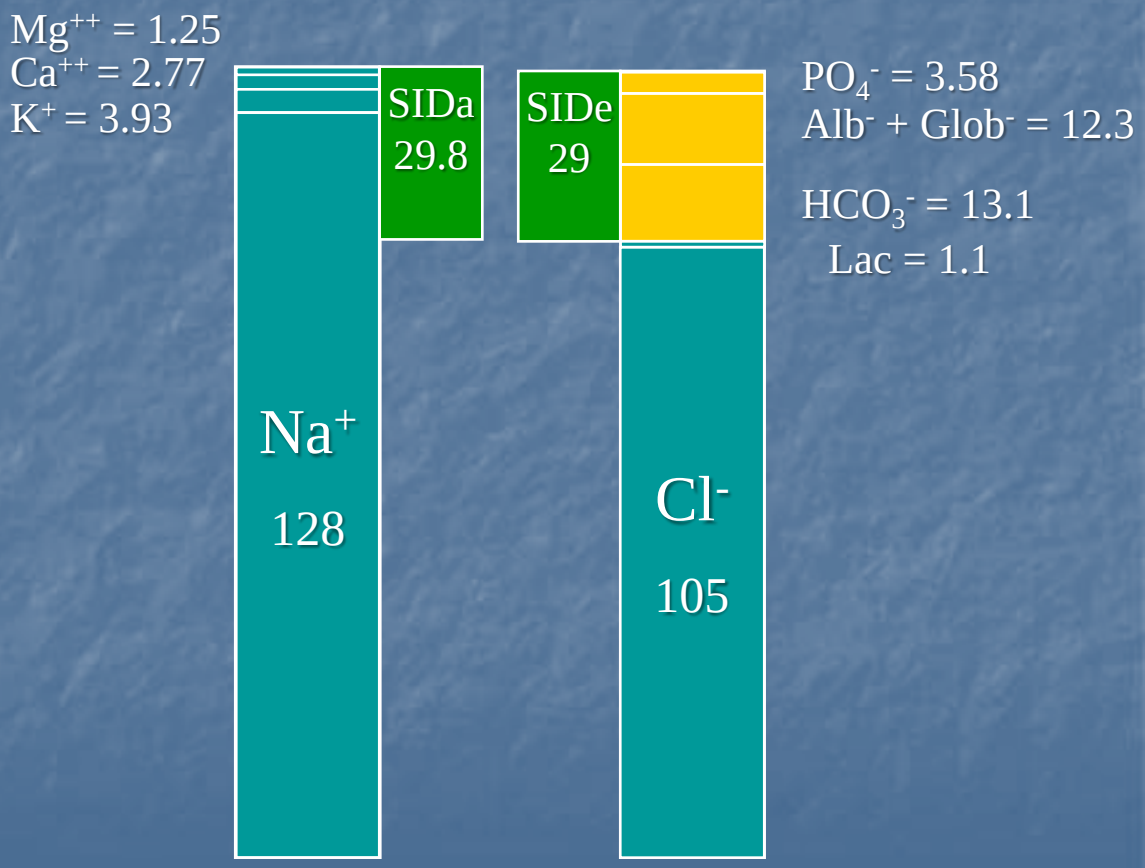
- 3 day old TB filly
- History
  - Never vigorous
  - Contracted left hind
  - Diarrhea 12 hr
  - Colicy, abdominal distention
  - Free fluid in abdomen - ruptured bladder?
- Problem: enteritis

Case 8

Cr	133 umol/L
BUN	5.4 mmol/L
HCO_3	13.1 mmol/L
Na	128 mmol/L
K	3.93 mmol/L
Cl	105 mmol/L
Ca^{++}	1.4 mmol/L
Mg^{++}	0.62 mmol/L
Lac	1.1 mmol/L
PO_4	1.96 mmol/L
Alb	27.6 g/L
Glob	43.4 g/L

Case 8

		mEq/L
pH	7.239	
Pco_2	31.2	
HCO_3	13.1 mmol/L	13.1
SBE	-12.2	-12.2
Na	128 mmol/L	128
K	3.93 mmol/L	3.93
Cl	105 mmol/L	105
Ca^{++}	1.4 mmol/L	2.77
Mg^{++}	0.62 mmol/L	1.25
Lac	1.1 mmol/L	1.1
PO_4	1.96 mmol/L	3.58
Alb	27.6 g/L	6.21
Glob	43.4 g/L	6.1

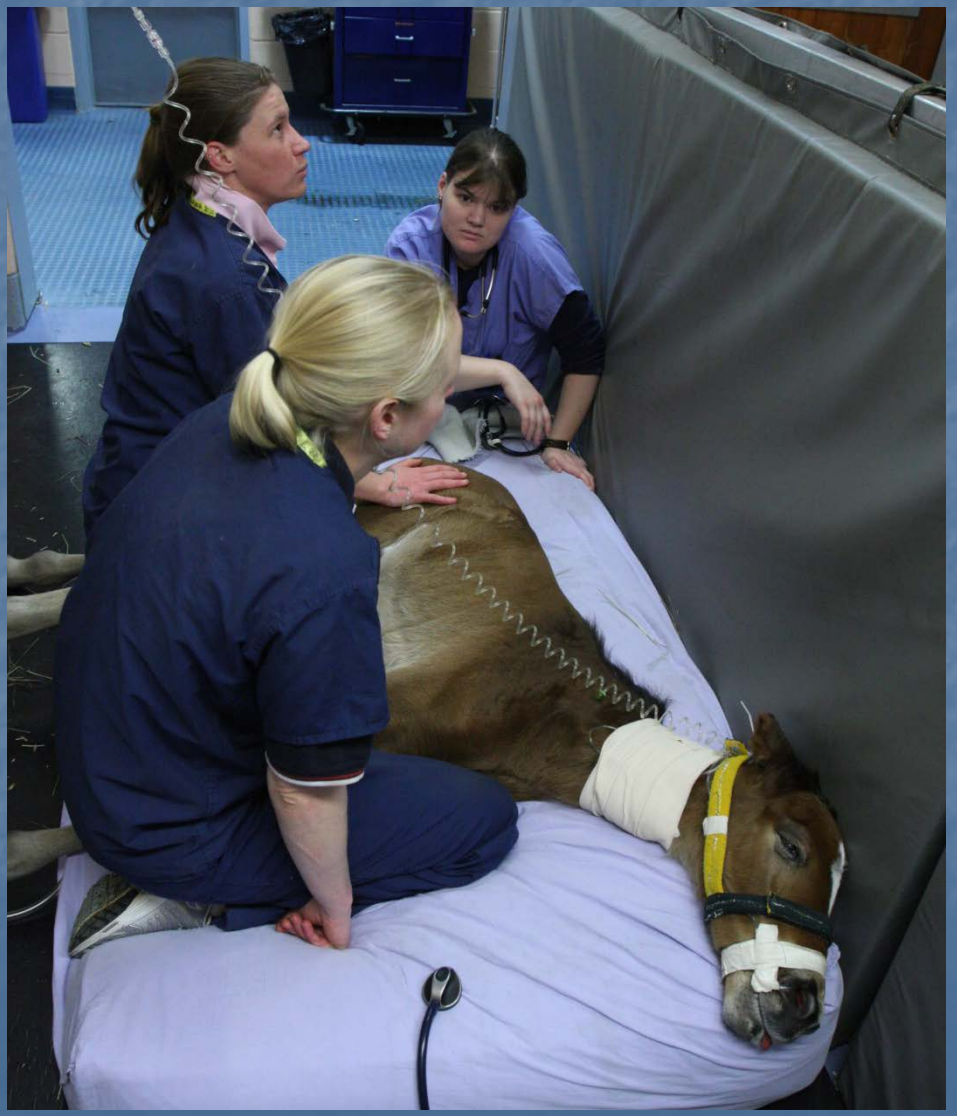


Case 9

- 30 min old TB colt
- History
  - Premature placental separation
  - Upper airway obstruction
- Problems
  - Respiratory distress
    - Pharyngeal collapse
    - Central Tachypnea
  - NE, NG, NN, Shock
  - Respiratory failure
  - Anuria
- Euthanized 48 hr

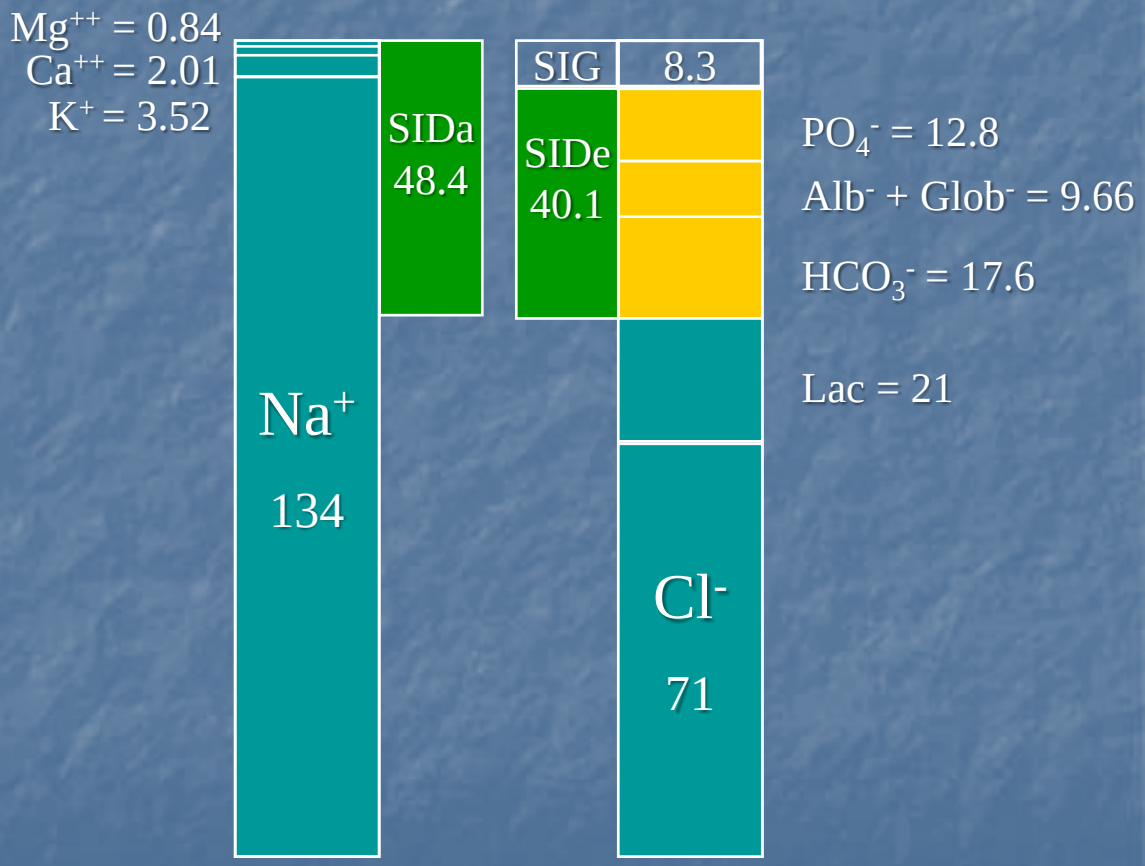
Case 9

Cr	2431 umol/L
BUN	5.7 mmol/L
HCO_3	17.6 mmol/L
Na	134 mmol/L
K	3.52 mmol/L
Cl	71 mmol/L
Ca^{++}	1.0 mmol/L
Mg^{++}	0.42 mmol/L
Lac	21 mmol/L
PO_4	7.01 mmo/L
Alb	31.8 g/L
Glob	18.2 g/L



Case 9

		mEq/L
pH	7.084	
Pco_2	58	
HCO_3	17.6 mmol/L	17.6
SBE	-12.5	-12.4
Na	134 mmol/L	134
K	3.52 mmol/L	3.52
Cl	71 mmol/L	71
Ca^{++}	1.0 mmol/L	2.01
Mg^{++}	0.42 mmol/L	0.84
Lac	21 mmol/L	21
PO_4	7.01 mmo/L	12.8
Alb	31.8 g/L	7.16
Glob	18.2 g/L	2.5



Acid-Base Workshop

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Critically ill patients rarely have a single acid–base disorder. These patients typically manifest mixed acid–base physiology with multiple, often conflicting metabolic derangements superimposed on respiratory disease or compensation. There are a number of approaches that are used clinically to diagnose and understand the origin of a patient's acid–base status. No matter which approach is used by a clinician to formulate a therapeutic plan, usually the result is the same making the choice of approach more a matter of style than one approach being superior to another. When trying to understand the underlying pathophysiology leading to a patient's acid-base status, no matter what approach is used, because of the complex interactions which often cancel each other's effects, no method will completely explain all derangements. This workshop will focus on the Stewart-Fencl approach to acid base abnormalities and I hope to demonstrate how that approach may help in the understanding of the underlying genesis of acidbase abnormalities in critically ill foals and also how this understanding may help direct therapy.

Extracellular hydrogen ion concentration is tightly controlled by the body. This control takes the form of control of volatile and metabolic acids. Various intracellular and extracellular weak acid buffering systems have evolved to prevent rapid changes in the extracellular electrochemical balance that may interfere with transcellular ion pumps. Normal metabolism is the major source of volatile acid. CO_2 production results in the generation of 12,500 mEq of H^+ daily. By contrast, only 20 to 70 mEq of anions are excreted through the kidney each day. Hemoglobin is the major buffer of volatile acid. Deoxyhemoglobin is an active base. Within the erythrocyte, CO_2 combines with H_2O, under the influence of carbonic anhydrase, to form H_2CO_3. This ionizes to hydrogen and bicarbonate. Hydrogen ions bind to histidine residues on deoxyhemoglobin (the "Haldane" effect), and bicarbonate is actively pumped from cells. Chloride moves inward to maintain electroneutrality (chloride shift) and to ensure the continued production of carbonic acid. CO_2 also is buffered directly by hemoglobin (carbaminohemoglobin) and by plasma proteins (carbamino proteins). The CO_2 added to venous blood is usually distributed as follows: 65% as HCO_3^- and H^+ bound to hemoglobin, 27% as carbaminohemoglobin (CO_2 bound to hemoglobin), and 8% dissolved. When respiratory failure occurs, the principal CO_2 buffering system, hemoglobin, becomes overwhelmed. This leads to the rapid development of acidosis. The major effect of the kidney on acid-base balance relates to renal handling of sodium and chloride ions. Because dietary intake of sodium and chloride is roughly equal, the kidney excretes a net Cl^- load using NH_4^+, a weak cation, to accompany Cl^- in the urine. This is the essence of metabolic compensation (acid-base balancing).

Strong Ions

The degree of dissociation of substances in water determines whether they are strong acids or strong bases. Lactic acid, which has an ion dissociation constant (pK_a) of 3.4, is completely dissociated at physiologic pH and is a strong acid. Conversely, carbonic acid, which has a pK_a of 6.4, is incompletely dissociated and is a weak acid. Similarly, ions such as Na^+, K^+ and Cl^- that do not easily bind other molecules are considered strong ions as they exist free in solution. Strong ions in normal ECF include Na^+, Cl^-, K^+, $SO4^{--}$, Ca^{++}, and Mg^{++}.

Electric neutrality must always hold. Consequently, the accumulation of strong anions (Cl^-, LA^-, ketones, sulfate and formate, etc.) is the "footprint" or "ghost" left by a strong acid. When a strong acid such as lactate (LA^- H^+) is produced, much of the H^+ is buffered (combines with a weak base) thus the electrical neutrally is preserved as anionic buffering sites are neutralized by combining with H^+ allowing the accumulation of LA^- without changing the electrical neutrality. A small amount of unbuffered H+ lowers the pH. The accumulation of strong anions is a reflection of the amount acid added to the system.

Buffer Base, Weak Acid Buffer, Nonvolatile Weak Acids, A_{TOT}

There are a number of substances in the body that because of their pKa act as buffers. But in plasma, where pH is measured, there are only a few substances that act as nonvolatile weak acids and have concentrations great enough so that changes in them can produce significant acid–base disturbances: inorganic phosphate (PO_4) and

serum albumin (Alb). Albumin, because of its negative charge at the physiologic (and pathophysiologic) pH range can act as a nonvolatile weak acid. Serum globulins in the horse, unlike in man, also play a role as they can carry a significant net electric charge at pH values prevailing in plasma.

For Alb (g/L) Alb^- (mEq/l) = (Alb) x ((0.123 x pH) – 0.631) *
Or shortcut without pH (assumes pH = 7.40): Alb^- = 0.28 x Alb *
* These formulas are for human albumin; in the horse Alb^- = 0.225 x Alb and $Glob^-$ (mEq/L) = 0.14 x Glob

For Pi (mmol/L) Pi^- (mEq/l) = (Pi) x ((0.309 x pH) – 0.469)
Or shortcut without pH (assumes pH = 7.40): Pi^- = 1.8 x Pi

Anion Gap (AG)

Anion Gap was developed (1977) to estimate the accumulation of unmeasured anions as strong acids are produced. The H^+ decreases the buffer base and leaves behind an unmeasured anion producing the anion gap. These strong organic acids may accumulate because of increased production such as with lactate or ketoacids, toxic ingestion, decreased renal excretion, or errors of metabolism.

$AG = (Na + K) – (Cl + HCO_3)$

In reality AG = UA – UC. Unidentified anions (UA) are primarily albumin, PO_4 and organic anions of interest e.g. lactate plus minor amounts OH^-, SO_4^{2-}, CO_3^{2-}. Unidentified cations (UC) are Mg^{2+}, Ca^{2+}, and a large number of organic cations such as amines (epinephrine, dopamine, etc.), many drugs (40% of all conventional drugs) plus minor amounts of H^+. Usually UC are a minor contributor (but not always) and are ignored.

Errors in AG can come from variations in UC and UA which are not of interest. It is unusual for UC to change much. On the other hand UA, especially low albumin and low PO_4 can have a large effect which could mask the appearance of an organic acid. High levels of PO_4, as we often find in neonates and can occur in renal failure, will add to the AG falsely suggesting the presence of organic acids. The almost universal occurrence of hypoalbuminemia in critical patients has lead to the development of a corrected AG: $AG_{Corr} = AG + (25 \times (Alb_{ref} - Alb_{measured}))$. Another problem with AG is its reliance on HCO_3^- as it does not account for changes associated with changes of P_{CO2}.

AG method does not identify acid–base abnormalities that are due to alterations in plasma free water. Additionally, the AG method does not account for the correction of chloride concentration in the setting of altered plasma free water. As a result, a hyperchloremic acidosis in the setting of a dilutional alkalosis would not be identified with an analysis using the AG method. In general AG analysis is well suited to detect the rapid increase of UA in a patient that was previously normal but is suffering from an emergent problem such as hypovolemic shock with resulting lactic acidosis but can fail in more complex, chronic situations.

Stewart-Fencl Approach

Peter Stewart (1981) based his approach on principles of physical chemistry keeping true to electrical neutrality, dissociation equilibriums and mass conservation. Although his original analysis is too cumbersome for routine use, his ideas have lead to the derivation of clinically useful tools helpful in understanding the underlying cause of acid-base disturbances.

This approach recognizes only three independent variables: SID (strong ion difference), concentration of weak acids (A_{TOT}), and P_{CO2}. All acid-base disturbances are a result of changes of these variables which are then reflected in changes in the dependent variables H^+ and HCO_3^-. Changes in P_{CO2} are called respiratory acid-base disturbances and changes in SID and/or A_{TOT} are metabolic acid-base disturbances. A_{TOT} is defined as the total amount of weak acid species in both the dissociated and the non-dissociated form, which is an independent variable whereas the amount dissociated is dependent on the pH (number of H^+ that need buffering) and the amount of weak acid present.

SID can be calculated two different ways. Apparent SID (SID_A) is the difference between the identified strong cations and the identified strong anions: $SID_A = (Na^+ + K^+ + Ca^{2+} + Mg^{2+}) - (Cl^- + Lac^-)$. Effective SID essentially includes the UC and UA in the formula but practically is calculated as the sum of the nonvolatile and

volatile weak acid buffer: $SID_E = (Na^+ + K^+ + Ca^{2+} + Mg^{2+} + UC) - (Cl^- + Lac^- + UA) = HCO_3^- + Alb^- + HPO_4^- + PO_4^{2-}$. The presence of UA or UC will cause a strong ion gap (SIG): $SIG = SID_A - SID_E = UC - UA$. UA contributing to the gap may include D-lactate, formate, ketoacids, salicylates, and sulfates. Unlike AG, a normal SIG should be 0 no matter what the albumin and phosphate levels and is not confounded by respiratory influences on HCO_3^-. Also, unlike AG, assuming a normal of SIG = 0 and no confounding UC, the SIG value will equal the amount of UA present in mEq/l.

$SID_A = (Na^+ + K^+ + Ca^{2+} + Mg^{2+}) - (Cl^- + Lac^-)$
$SID_E = HCO_3^- + (0.225 \times Alb) + (0.14 \times Glob) + (1.83 \times PO_4)$

Standard Base Excess

Siggard-Andersen (1948) first introduced the base deficit/excess as defined as the amount of strong acid or base required to return 1 liter of whole blood to pH to 7.4, assuming a PCO_2 of 40 mm Hg and temperature of 37°C. Although this worked well *in vitro* it failed *in vivo* because of the combination of the important role of hemoglobin as a buffer and the buffer space being the ECF and not confined to the vascular space. As a result, changes in patient Pco_2.can result in changes of BE. In the 1960s this approach was modified to estimate total ECF buffering by development of the standard base excess (SBE). Since the vascular space is usually about 1/3 of the ECF, SBE used 1/3 the usual hemoglobin concentration (or 5 g/dl) in its calculation. As a result it will become less accurate as Hb levels deviate from 15 g/dl and as the ratio of vascular volume to ECF changes. Also this algorithm was developed using human hemoglobin. As this formula assumes a normal A_{TOT} which is frequently absent in critical patients, Wooten (2003) developed a corrected formula (SBE_{corr}) which overcomes this latter problem.

METABOLIC ACID BASE ABNORMALITIES

Dilutional Acidosis/ Contraction Alkalosis

Change in free water content will change SID. As free water dilutes the strong ion concentrations, the SID will also decrease resulting in a metabolic acidosis. Conversely, free water deficit causes a metabolic alkalosis by increasing the SID through a relative increase in concentration of all strong ions. Changes in free water are reflected by changes in sodium concentration. Any process that leads to dilution of the total number of ions will cause an acidosis including infusion of mannitol (before the diuresis), hyperglycemia or other osmotic agents. The increase of any osmotically active particle may increase in the volume of extracellular water without changing the net charge.

Because of this free water effect, it may be difficult to tell how much the abnormal SID is from changes in free water and how much from changes in the relative concentrations of Na and Cl. To address this problem a Cl value corrected for free water can be used: $Cl_{Corr} = (Na_{ref} / Na_{measured}) \times Cl^-_{measured}$. The same correction can be used on all strong ions. The difference between SID calculated with all measured values and the SID calculated with all corrected values (and the reference Na) will indicate how much of the alkalosis or acidosis is for free water. It should be noted that the effect is from free water excess or deficit and will not be seen with fluid overload or loss when consisting of strong ion balanced fluids.

Saline/ Water Infusion

Large volumes of normal saline infusion will produce an acidosis. Take for example the extreme example of a patient with a 1liter ECF volume (Na 140, K 5, Cl 100, SID 45) receiving 1liter saline (Na 154, K 0, Cl 154, SID 0). The new 2 l ECF will have a Na = 147, K 2.5, Cl 127 and SID = 22.5. The same change will occur if the fluids given were 1 l of water (Na 0, K 0, Cl 0, SID 0) which will result in Na = 70, K 2.5, Cl 50 and SID = 22.5. So it is the volume and the SID of the fluid which results in the change. To illustrate this, giving 0.5 l of 1.8% NaCl (Na 308, K 0, Cl 308, SID 0) will result in a 1.5 l ECF with Na 196, K 3.3, Cl 169.3, SID 30. So hypertonic saline will result in less acidosis than normal saline if less volume is given but the administered Na and Cl remain constant. Of course after therapy with hypertonic saline, fluid transfer from the intercellular space will dampen the effect.

Hypochloremia/ Hyperchloremia

Although the kidneys excrete many fixed acids, control of Cl reabsorption/ excretion is the major renal acid base balancing mechanism. In metabolic acidosis, Cl is preferentially excreted by the kidney. In metabolic alkalosis, Cl is retained, while sodium and potassium are excreted. Change in Cl concentration relative to Na leading to change in SID is the major renal compensatory mechanism. As the normal diet usually consists of a balance of Na and Cl the kidneys can retain or excrete the surplus Cl depending on acid-base needs.

Abnormalities in the renal handling of chloride may be responsible for several inherited acid-base disturbances. In renal tubular acidosis, there is inability to excrete Cl^- in proportion to Na^+. The diagnosis can be made by observing a hyperchloremic metabolic acidosis with inappropriately low levels of Cl^- in the urine: The urine SID is positive. If the urine SID is negative, the process is nonrenal. Similarly, pseudohypoaldosteronism seems to be due to high reabsorption of Cl. The other causes of hyperchloremic metabolic acidosis are gastrointestinal losses of Na (diarrhea, small bowel or pancreatic drainage), parenteral nutrition, excessive administration of saline, and the use of carbonic anhydrase inhibitors.

Any process that removes chloride without sodium such as reflux with pyloric obstruction or a chloriuresis such as with furosemide can lead to a hypochloremic alkalosis. Anything leading to a net loss of free water over sodium and chloride such as with a diuresis induced by hyperglycemia or use of diuretics may cause a contraction alkalosis. Any process where Na is lost without Cl such as severe diarrhea, will reduce the SID leading to a hyperchloremic acidosis. A hyperchloremic acidosis can also be induced by increased chloride salt administration in the form of saline or PPN infusions.

Unidentified Anions/ Unidentified Cations

Traditionally L-lactate has been the main unidentified anion. In fact the AG analysis was developed to identify the presence of lactate. Today, with routine availability of lactate assays, L-lactate is accounted for except in cases with laboratory failures. At least in herbivores, D-lactate continues to be a major UA. Other major endogenous UA include ketoacids, VFAs and sulfates. The other main source of organic UA are ingested organic acids such as salicylates, methanol, ethylene glycol and paraldehyde. The presence of UC is much less common than UA. They include endogenous organic cations such as amines and exogenous organic cations such as ingested toxins and toxic levels of drugs (almost half of the organic drugs we use are cations). It is very rare for UC to be found in significant concentrations to confound acid-base analysis in adults; however, in neonatology we do occasionally find endogenous UC in significant levels. For the most part they remain unidentified but can contribute to an alkalosis.

The discovery of the presence of UA/UC is by the realization that the numbers don't "add up" and there is a "gap" (AG or SIG). Unaccounted for changes in other major players, especially hypoalbuminemia or occurrence of UC, can mask the presence of UA by making it appear that the numbers do add up as they exert an opposite effect. This is the reason to use AG_{corr} or SIG and part of the reason a significant proportion of acid-base disturbances in our patients defy our efforts to fully explain them.

Albumin/ Phosphate concentrations

A_{TOT} is defined as the total amount of weak acid species in both the dissociated and the non-dissociated form. The major contributors to A_{TOT} in plasma are albumin, globulin and phosphate. Increase in weak acid concentration such as occurs with hyperphosphatemia in renal failure and hyperalbuminemia/hyperglobulinemia of hemoconcentration results in a metabolic acidosis. Decrease in weak acid concentration, such as hypoalbuminemia and hypoglobulinemia as is often present in critical patients, results in a metabolic alkalosis. The neonates that I often deal with frequently are born with low albumin and globulin concentrations but concurrently high PO_4 concentrations, both which appear to be normal. The two balance each other well until they become catabolic because of neonatal problems.

DIFFERENTIAL DIAGNOSIS

In the complex disturbances of critically ill patients, alkalinizing and acidifying disturbances may both be present and may escape detection because of their offsetting effects. There are significant differences between the mechanisms causing acid-base imbalances. There are likewise significant differences in outcomes for patients developing acidosis from dilution, poisoning, hyperchloremia, excessive use of normal saline infusions, dysoxia and other causes of increased lactate production. The acid-base abnormalities themselves may be of less clinical significance than previously thought.

This has been a very short review of the underlying causes of metabolic acid-base disturbances and the strengths and weakness of current analysis tools designed help us understand the origin of the disturbance in our cases. I have completely ignored respiratory acid-base problems because of space and in realization that the respiratory problems are more easily understood by most intensivists. Acute metabolic acidosis can be a complex problem and may be caused by an alteration in the SID or A_{TOT}. An altered SID reflects a change in the relative ratio

of strong anions to strong cations. This change can be caused by anion gain, as occurs with lactic acidosis, renal acidosis, ketoacidosis, or hyperchloremia. Alternatively, cations may be lost, as occurs with severe diarrhea or renal tubular acidosis. Acute acidosis also may reflect increased free water relative to strong ions (dilutional acidosis) that may accompany excessive hypotonic fluid intake or the presence of excessive osmoles such as with hyperglycemia or alcohol poisoning (ethanol, methanol, isopropyl alcohol, ethylene glycol). The plasma concentration of albumin and phosphate also can have a influence over acid-base balance by resulting an abnormal amounts of A_{TOT} which in turn is reflected by the amount of the nonvolatile weak acid present.

Table: Useful formulas (not correcting for pH introduces very small error until pH reaches extremes)

Alb^- (mEq/L) = 0.225 x Alb (horse)
$Glob^-$ (mEq/L) = 0.14 x Glob (horse)
Pi^- (mEq/l) = 1.83 x Pi (horse)
$SID_A = (Na^+ + K^+ + Ca^{2+} + Mg^{2+}) - (Cl^- + Lac^-)$
$SID_E = HCO_3^- + (0.225 \times Alb) + (0.14 \times Glob) + (1.83 \times PO_4)$
$SIG = SID_A - SID_E$
For above Pi in mmol/L, Alb in g/L, Glob g/L, Pi in mmol/L

Further Reading

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