Acid-Base Disorders
Diabetic Keto-acidosis
(sour and sweet)

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Objectives

- know
  - why acid-base relevant
  - how to diagnose acid base disorders
  - how to treat acid base associated conditions

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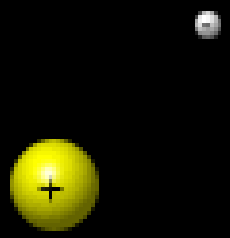
What are acids and bases?

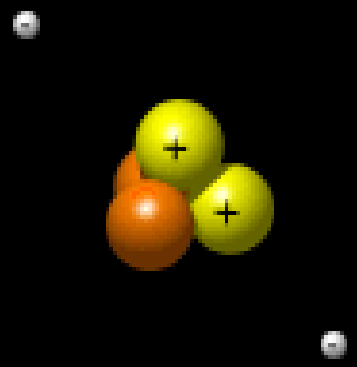
- Acid
  - can donate a proton
- Base (Alkali)
  - can accept a proton

Hydrogen



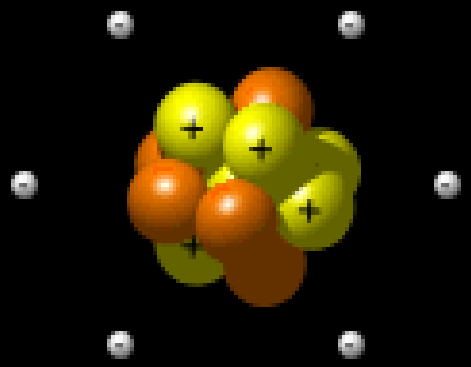
1 proton
1 electron
0 neutrons

Helium



2 protons
2 electrons
2 neutrons

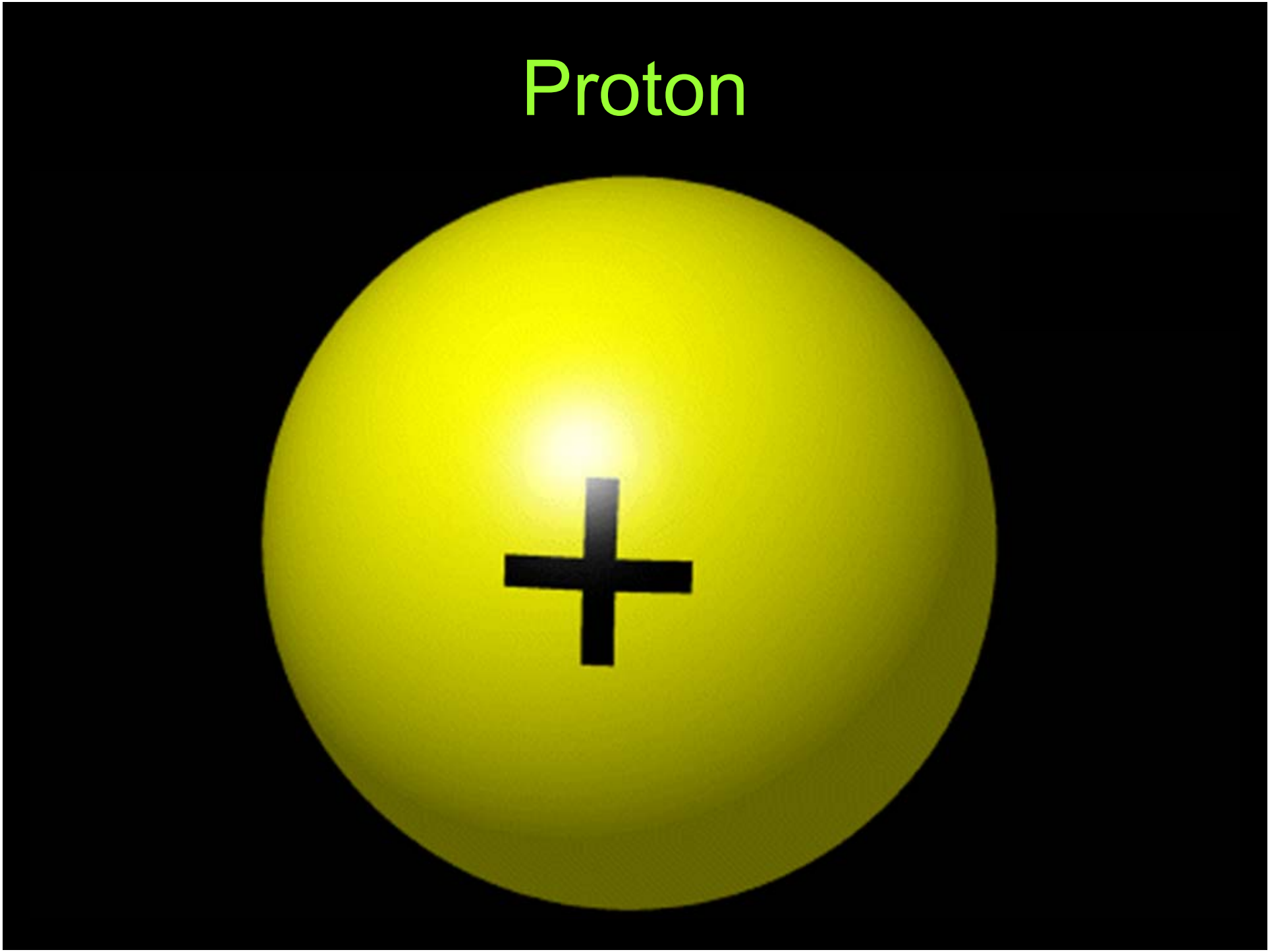
Carbon

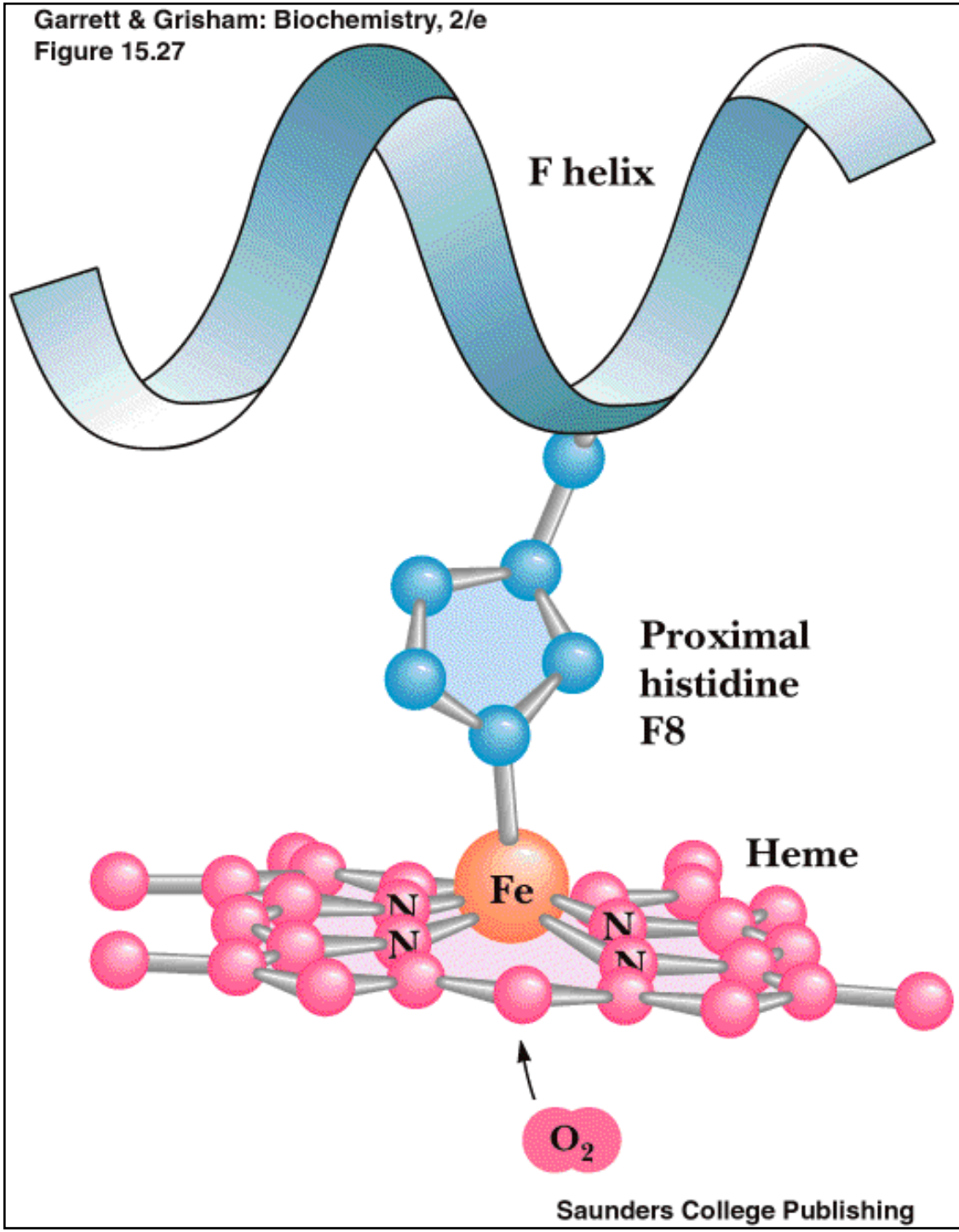


6 protons
6 electrons
6 neutrons

Atoms are made from
Protons, Neutrons, Electrons

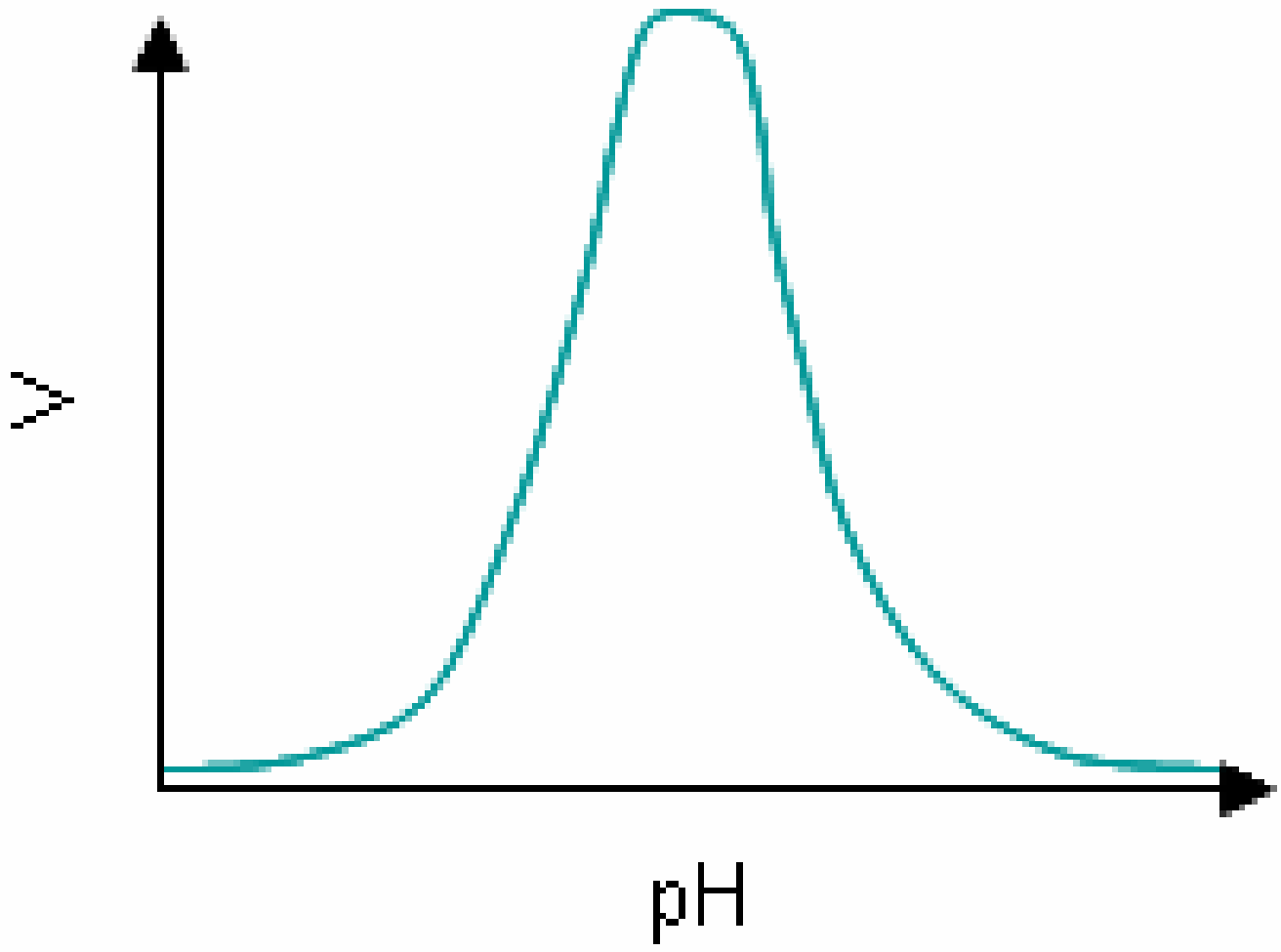
Proton




Garrett & Grisham: Biochemistry, 2/e
Figure 15.27
F helix
Proximal
histidine
F8
Heme
Fe
N
N
N
N
O_2
Saunders College Publishing

Myoglobin

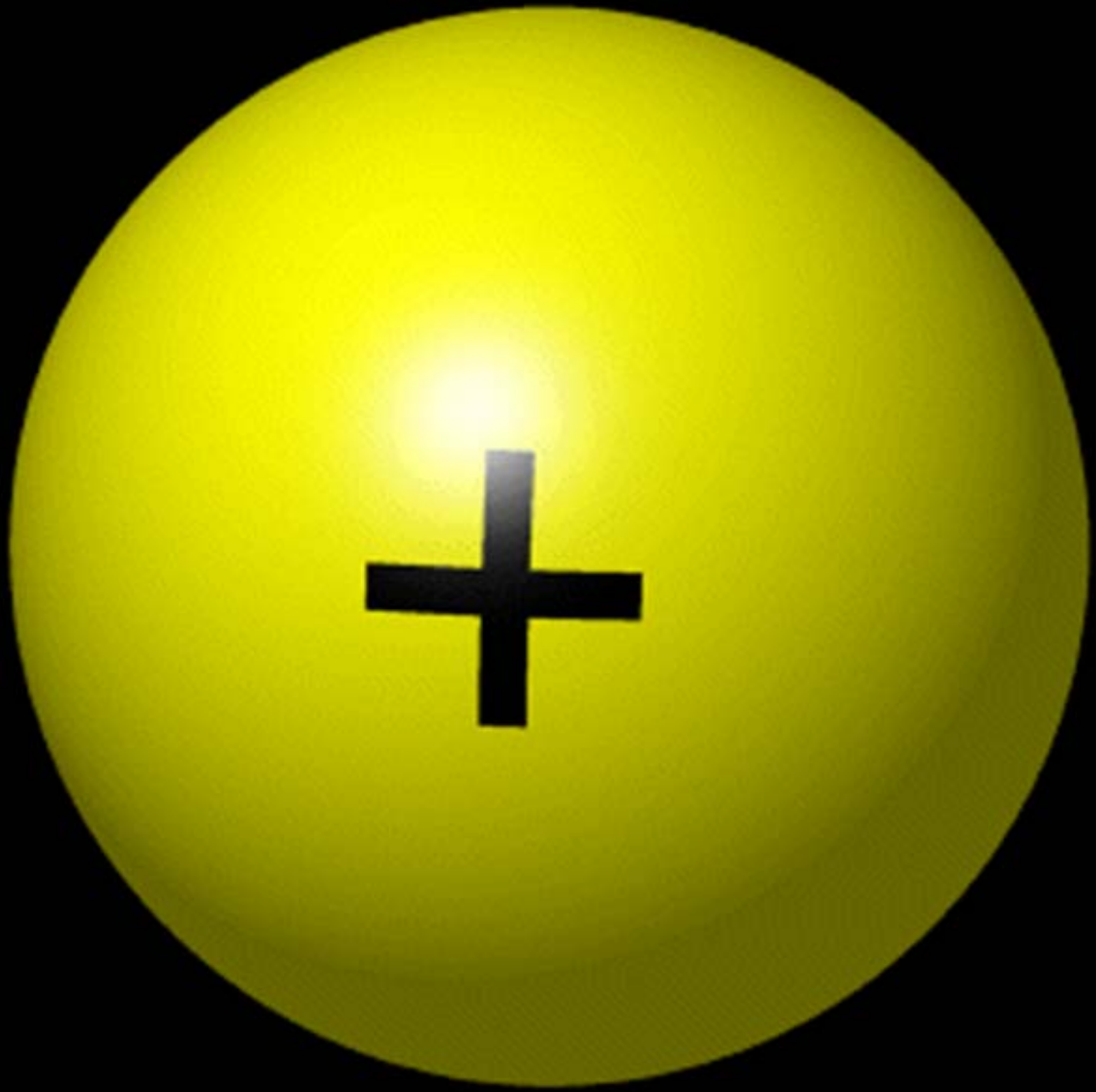
Acid effects enzyme function!



Protein!

TONG CHIN WEE ALAN TAY RANO ROYCE JR.

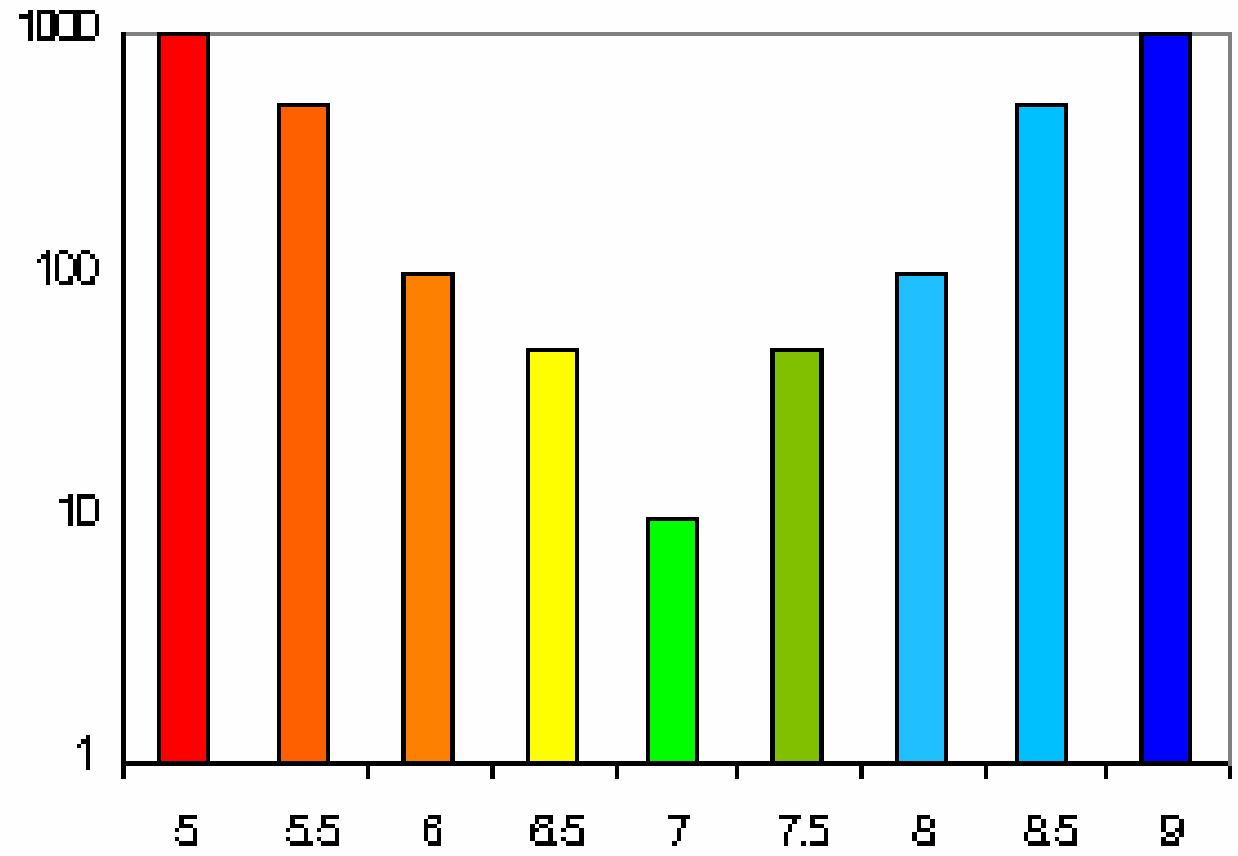
Protons



Acids (protons) are highly reactive

Alkalis (proton acceptors) are highly reactive

pH = — log concentration of protons



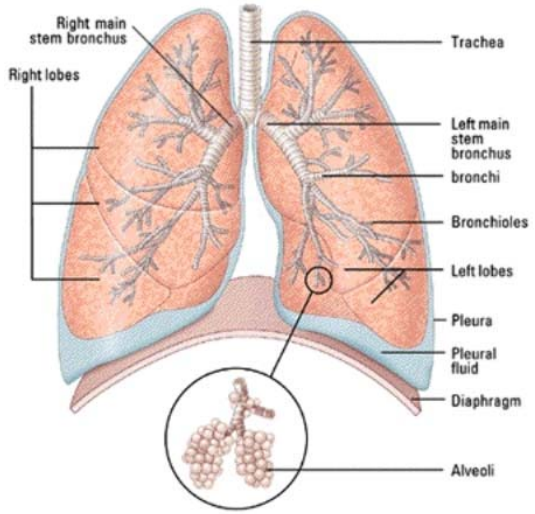
Buffers

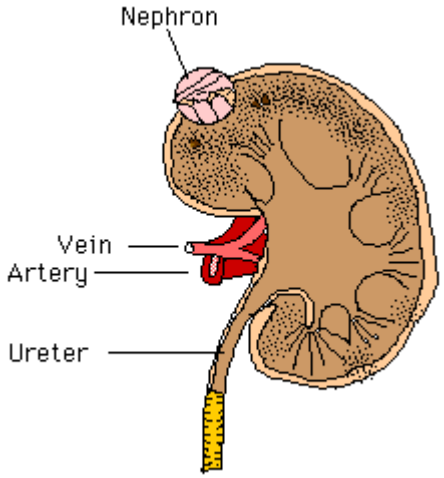
- Mammals
  - pH = 7.4
- Buffers
  - resist pH changes
  - weak acids and their salts
- Several buffers
  - carbon dioxide / bicarbonate most important

Carbonic acid system

$CO_2 + H_2O \longleftrightarrow HCO3^- + H^+$

carbon dioxide **bicarbonate** **proton**





13,000 mmol of CO2 produced per day

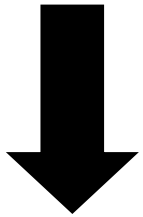
Carbonic acid system

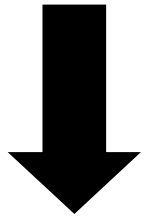
H^+

proton

$HCO3^-$

bicarbonate





Acidic

Basic

Objectives

- know
  - why acid-base relevant
  - how to diagnose acid base disorders
  - how to treat acid base associated conditions

Definitions

‘emia’ versus ‘osis’

- emia = blood level of something
- osis = process

Acidemia

- Acidemia
  - blood pH is lower than 7.4
  - acidosis must be present

Alkalemia

- Alakemia
  - blood pH is greater than 7.4
  - alkalosis must be present

Acidosis

- a disease process
  - that increases protons or decreases bicarbonate
  - puts downward pressure on pH

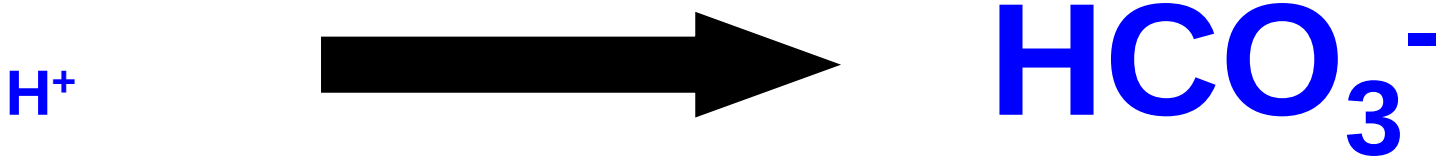
- an example: CO_2 retainer

$$CO_2 + H_2O \rightarrow HCO_3^- + \mathbf{H^+}$$

Alkalosis

- a disease process
  - that decreases protons or increases bicarbonate
  - puts upward pressure on pH

- an example: NG suction



Respiratory versus Metabolic

- Ventilation (one of lungs functions)
  - lungs blow off carbon dioxide (an acid)
    - too much ventilation decreases body acid
    - too little ventilation increases body acid
- Metabolic Potential Causes
  - Kidneys control bicarbonate or acid loss
  - GI tract is source of bicarbonate or acid loss
  - Metabolic disturbances
    - liver, diabetes, ischemia

Primary Disorders

- Respiratory Acidosis
  - acute or chronic
- Respiratory Alkalosis
  - acute or chronic
- Metabolic Acidosis
  - normal or high anion gap
- Metabolic Alkalosis
  - chloride responsive or resistant

Each primary process triggers compensation...

Primary Process		Compensation
Resp acidosis	CO_2 ↑	HCO_3 ↑
Resp alkalosis	CO_2 ↓	HCO_3 ↓
Met acidosis	HCO_3 ↓	CO_2 ↓
Met alkalosis	HCO_3 ↑	CO_2 ↑

Meaure bicarb in mmol/l and carbon dioxide in mmHg

Respiratory Acidosis
acute or chronic

- Hypoventilation (CO_2 build up)
  - Central nervous system
    - trauma, stroke, drugs…
  - Phrenic outflow
    - trauma, tumor, iatrogensis
  - Motor End Plate
    - Eaton Lambert, myasthesthenia gravis, anesthetics
  - Muscular disease
    - ALS, polio
  - Airway and Lung Disease
    - COPD, obstruction, destruction, infiltration

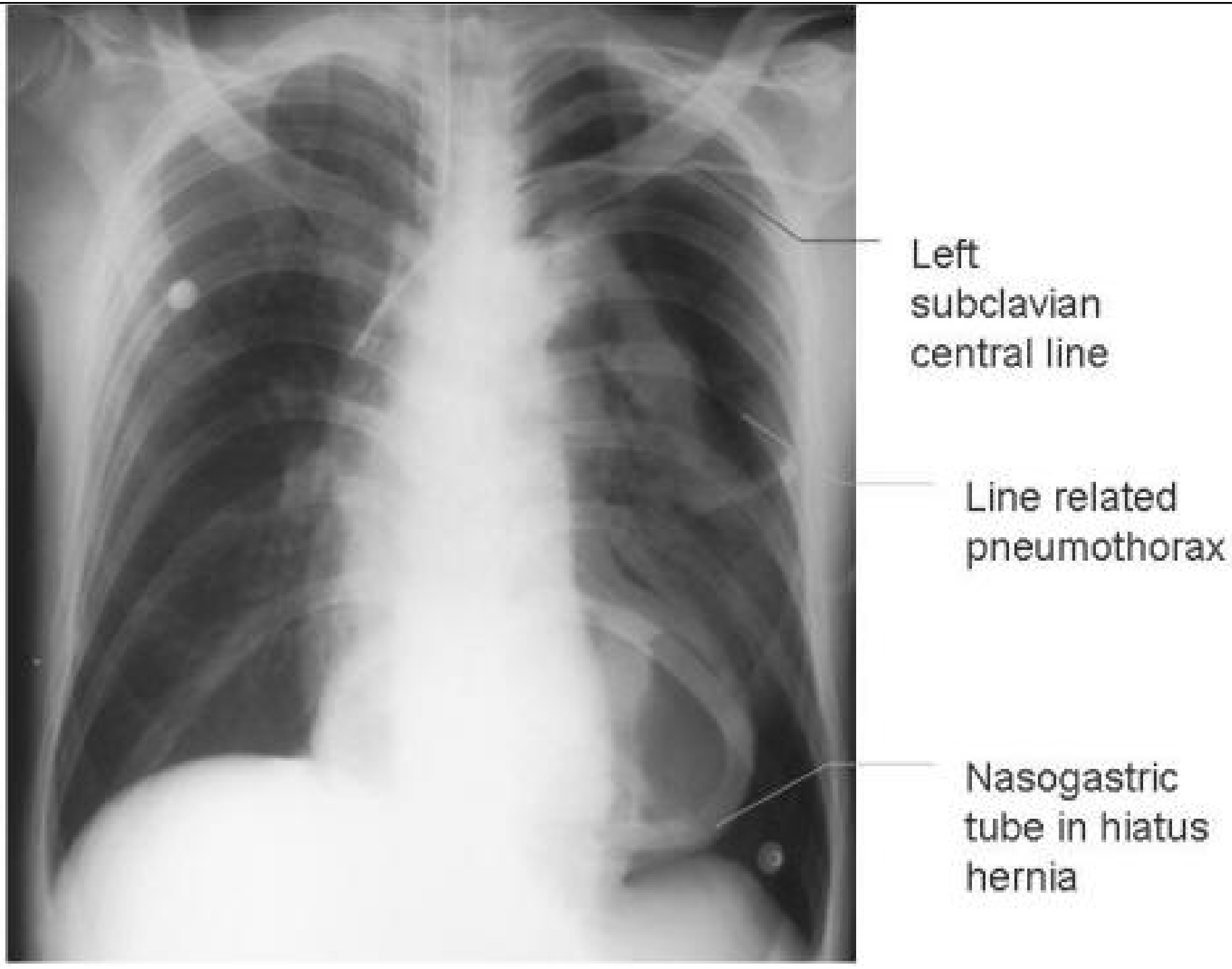
Respiratory Acidosis, what happens?

- Primary
  - blood carbonic acid?
  - pH?
- Acute Compensation (< 1 day)
  - bicarbonate increases 3 - 4 mmol in total
- Chronic Compensation (> 2 - 4 days)
  - bicarbonate increases **0.3** mmol / 1 mmHg pCO_2

- 74 yr man back from DI after central line
- confused and diaphoretic -> ABG

	patient	normals	change
pH	7.15	7.40	-
pCO_2	85 mmHg	40	+ 45
HCO_3	27 mmol	24	+ 3

- Alkalemia or Acidemia?
  - pH low, therefore acidemia and acidosis must be present
- Respiratory or Metabolic Acidosis?
  - CO_2 is elevated, therefore hypoventilation
- Acute or Chronic?
  - bicarbonate changed 3 mmol, therefore acute

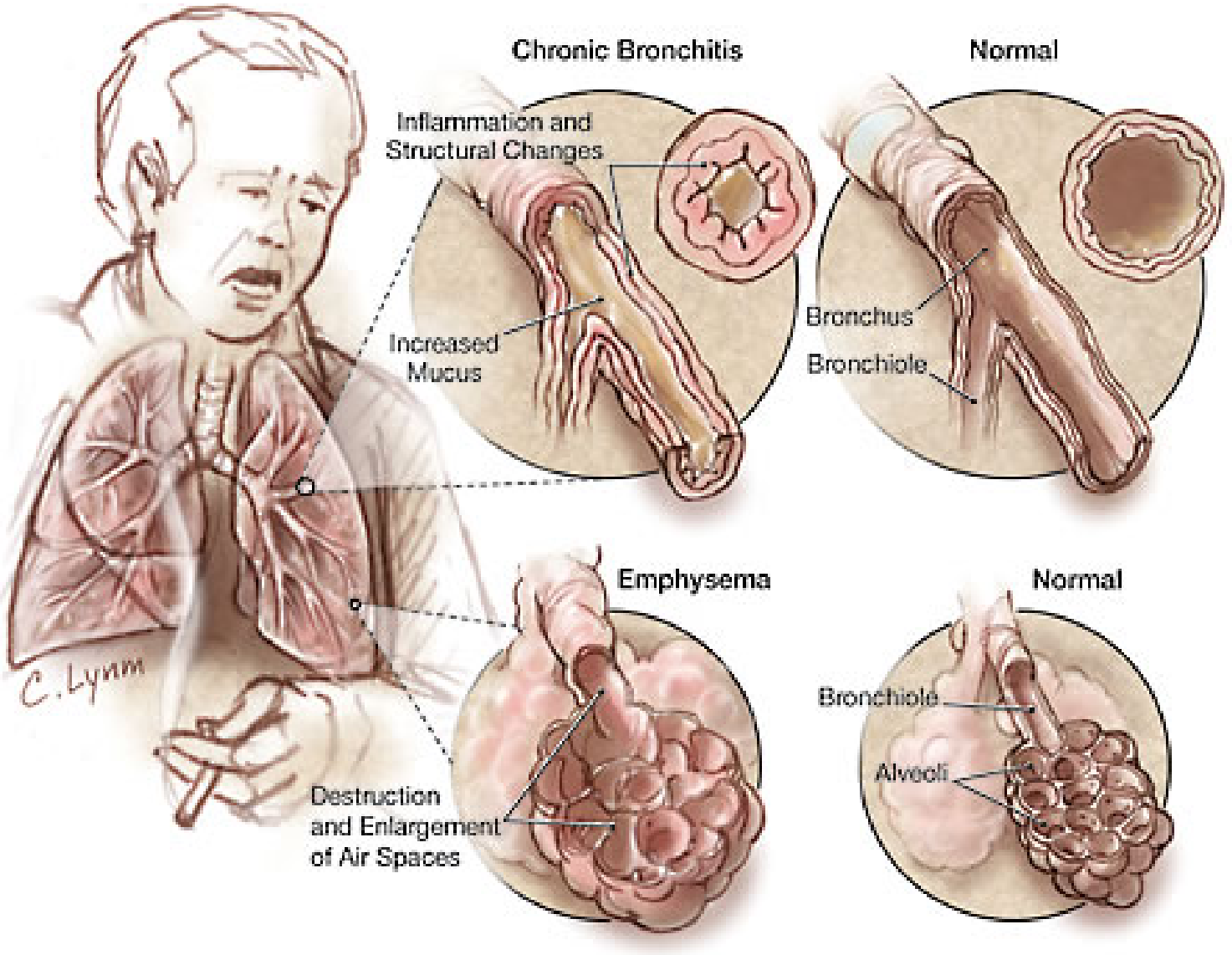

Left
subclavian
central line
Line related
pneumothorax
Nasogastric
tube in hiatus
hernia

- 74 yr man back presents to ED with SOB
- confused and diaphoretic -> ABG

	patient	normals	change
pH	7.30	7.40	-
pCO_2	85 mmHg	40	+ 45
HCO_3	39 mmol	24	+ 15

- Alkalemia or Acidemia?
  - pH low, therefore acidemia and acidosis must be present
- Respiratory or Metabolic Acidosis?
  - CO2 is elevated, therefore hypoventilation
- Acute or Chronic?
  - HCO_3 increased 1/3 that of increase in pCO_2

COPD exacerbation



Respiratory Alkalosis
acute or chronic

- Hyperventilation (excess CO_2 blown off)
  - hypoxemia without hypoventilation
    - interstitial pulmonary disease
    - asthma, pneumonia
  - drugs
    - salicylates, progesterone (pregnancy)
  - fever
  - hyperthyroidism
  - chronic liver disease
  - anxiety neurosis

Respiratory Alkalosis, what happens?

- Primary
  - blood carbonic acid?
  - pH?
- Acute Compensation (< 1 day)
  - bicarbonate decreases 2 - 3 mmol in total
- Chronic Compensation (> 2 - 4 days)
  - bicarbonate decreases **0.5** mmol / 1 mmHg pCO_2

- 34 yr old medical student presents with perioral numbness during exam season...

	patient	normals	change
pH	7.48	7.40	+
pCO_2	30 mmHg	40	- 10
HCO_3	21 mmol	24	- 3

- Alkalemia or Acidemia?
  - pH high, therefore alkalemia and alkalosis is present
- Respiratory or Metabolic Acidosis?
  - CO2 is decreased, therefore hyperventilation
- Acute or Chronic?
  - HCO_3 decreased 3 mmol, acute

anxiety

- 55 yr old high school dropout presents with jaundice and ….

	patient	normals	change
pH	7.44	7.40	+
pCO_2	24 mmHg	40	- 16
HCO_3	16 mmol	24	- 8

- Alkalemia or Acidemia?
  - pH high, therefore alkalemia and alkalosis is present
- Respiratory or Metabolic Acidosis?
  - CO2 is decreased, therefore hyperventilation
- Acute or Chronic?
  - HCO_3 decreased ½ have change in $PC0_2$, chronic

Cirrhosis of the liver

Metabolic Alkalosis
chloride responsive or resistant

- What happens?
  - GI tract, Renal or other Metabolic disturbances cause retention of bicarb or loss of acid

- Compensation is the same for resist / respons
  - Hypoventilation
  - Lungs retain CO_2 (acid)
  - BUT hypoxemia not well tolerated so compensation is limited.

$$pCO_2 = 0.9\ (HCO_3^-) + 15.6$$

Metabolic Alkalosis
‘chloride sensitive’

GI losses

- vomiting
- gastric suction

- Diuretics
  - furosemide

urine chloride is < 10 - 20 mmol/l

Metabolic Alkalosis
chloride resistant

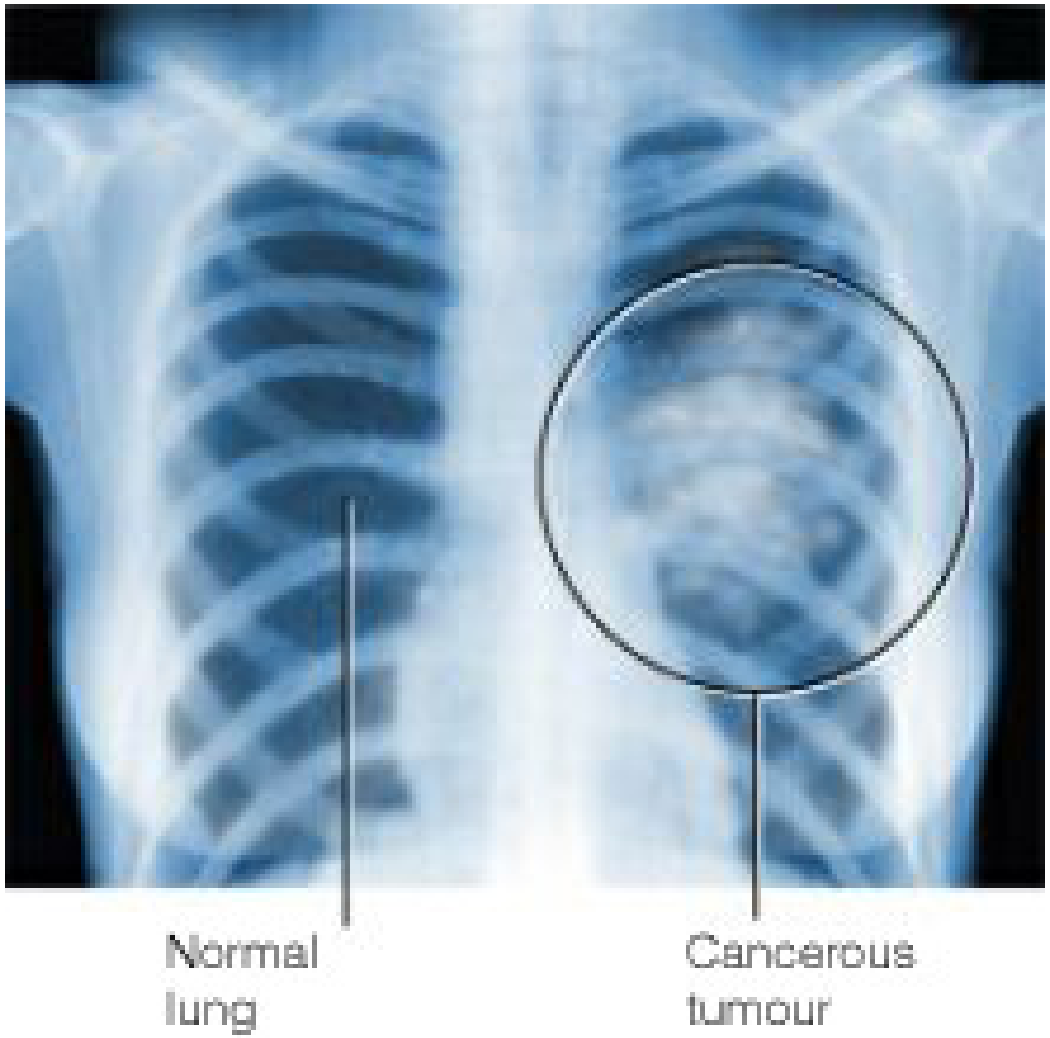
- Endocrine or Renal Causes
  - Hyperaldosteronism
  - Adrenal hyperplasia
  - Barter's syndrome
  - Licorice ingestion
  - Severe potassium depletion

urine chloride is > 10 - 20 mmol/l

- 66 yr old female heavy smoker presents with hemoptysis

	patient	normal	change
pH	7.52	7.40	+
pCO_2	53 mmHg	40	+ 13
HCO_3	42 mmol	24	+ 18
$_UCl$	35 mmol		

- Alkalemia or Acidemia?
  - pH high, therefore alkalemia and alkalosis is present
- Respiratory or Metabolic Alkalosis?
  - bicarbonate is increased, therefore metabolic cause
- Chloride Responsive or Resistant?
  - uCl is high, therefore endocrine / renal problem



lung cancer produces ACTH

ACTH stimulates cortisol production

cortisol causes kidney to dump K+ and retain bicarb

Metabolic Acidosis
normal and high anion gap

- What happens?
  - kidney, GI tract, metabolism
  - either acid builds up or bicarbonate is loss

- Compensation for normal or high AG is the same
  - Ventilation increases
  - Lungs blow off more CO2 (acid)

pCO_2 decreases 1.1 mmHg / 1 mmol HCO_3^-

Positive and Negative Charges

Anions (-)	Cations (+)
• Proteins 15	• Calcium 5
• Organic acids 5	• Magnesium 1.5
• Phosphates 2	• Potassium 4.5
• Bicarbonate 24	• Sodium 140
• Sulfates 1	
• Chloride 104	
• **TOTAL 151**	• TOTAL 151

These fluctuate most

Anions (-)	Cations (+)
• Bicarbonate 24	• Sodium 140
• Chloride 104	
• **TOTAL 128**	• TOTAL 140

Anion Gap

140 - (104 + 24) = 12

If > 12, there are unmeasured anions in the blood

NO NEED FOR ABG TO CALCULATE

Normal Anion Gap
relative bicarbonate loss

- Diarrhea
- Small bowel losses (surgical drainage, fistula)
- Carbonic anhydrase inhibitor
- Proximal RTA
- Expansion acidosis (normal saline IV)

High Anion Gap
acids added or produced

- Ketoacidosis
  - diabetic, starvation, alcoholic
- Lactic Acidosis
- Ingestion
  - ASA, methanol, paraldehyde, ethylene glycol
- Uremia

LIFE THREATENING DIAGNOSES

- 20 yr old female presents with abdominal pain

	patient	normals	change
pH	7.15	7.40	-
pCO_2	22 mmHg	40	- 18
HCO3	8 mmol	24	- 16
Na	130	140	
Cl	95	104	

- Alkalemia or Acidemia?
  - pH low, therefore acidemia and acidosis is present
- Respiratory or Metabolic Acidosis?
  - bicarbonate is down, therefore metabolic
- Normal or High Anion Gap?
  - 130 – (95+8) = 27 mmol (high)
  - therefore 15 mmol (27 – 12) of new acid needs to be accounted for

DDx High AG met acidosis

- glucose is elevated
- ASA is negative
- no history of antifreeze ingestion

- Dx: New Type 1 diabetes mellitus

Mixed Disorders

- If compensation is unexpected, there is a co-existing process!

- 70 yr old female presents with abdominal pain

	patient	normal	change	predict
pH	7.03	7.40	-	
pCO2	32	40	- 8	22
HCO3	8	24	- 16	
Na	130	-		
Cl	94	-		

- Acid Base Protocol
  - ph is low -> Acidemia
  - bicarbonate is low -> metabolic acidosis
  - anion gap is 28 mmol -> high anion gap metabolic acidosis
  - expected pCO_2 compensation is 1.1 x -16 = - 18 (or 40-18 = **22**)
  - actual pCO2 is 32 mmHg which is higher than expected
  - elevated pCO2 implies co-existing respiratory acidosis

Ischemic Gut with Lactic acidosis in COPD patient (CO_2 retainer)

Objectives

- know
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  - how to diagnose acid base disorders
  - how to treat acid base associated conditions

Acid Base

- Treatment
  - underlying cause
  - may add bicarbonate in selected situations
    - eg renal failure or profound acidemia (pH < 7.0)

- Underlying causes
  - acid base disorders points the way
  - history, clinical examination, other labs usually required to confirm the diagnosis
  - e.g. ASA level

DKA

DKA

- Relative Insulin deficiency
  - Type 1 diabetes
  - stressed Type 2 diabetes

DKA

- glucose can’t enter cells
- cells metabolize free fatty acids
- liver produces gluocose
- potassium can’t enter cells
  - no insulin
  - acidosis

DKA

- High glucose
- Polyuria
- Volume Contraction
- Renal Failure
- High adrenaline levels
  - risk of heart attack
  - dumps potassium
- Ketones and B-hydroxybutyric acid
  - abdominal pain

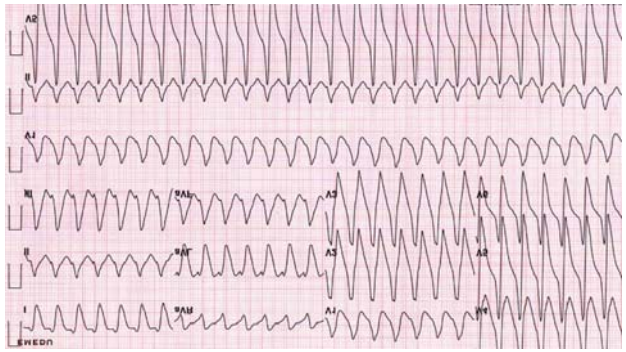
DKA & POTASSIUM

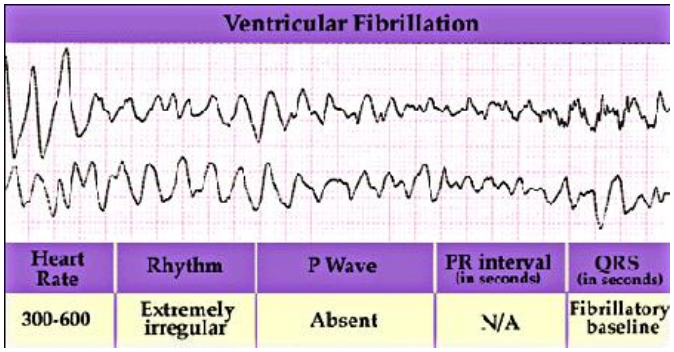
- Potassium: vast majority is IN cells
- DKA causes large potassium losses
  - vomiting and / or diarrhea
  - stress -> cortisol -> kidney dump potassium
- Even with Body Loss, blood levels are ‘high’
  - potassium can’t enter cells without insulin
  - potassium shifted outside cells by acidemia

DKA & POTASSIUM

- Treatment
  - insulin and correction of acidosis shifts potassium into cells

- POTASSIUM WILL FALL QUICKLY!!!
- LOW POTASSIUM → FATAL ARRYTHMIA





DKA Treatment

- treat the acidosis above all!
- restore volume
- restore potassium
- treat precipitants and complications

(treating glucose is of secondary importance!)

DKA Treatment

- 22 yr old female with DKA from ER
- NS at 150 / hour and insulin drip at 2 U/hr
  - Na^+ 150
  - K^+ 3.8
  - Cl^- 110
  - HCO_3 12
  - glucose 4.0
  - AG 135 – (100 + 10) = 28
- What are your concerns?

DKA Case Concerns

- DKA not resolved
- needs insulin to correct acidosis
- needs glucose IV to allow insulin without causing hypoglycemia
- needs potassium
- does not need repeat ABG if AG is improving
- hypernatremia owing to NS

- IV D5½NS + KCL 40 mmol / l at 150 ml/hr

Objectives

- know
  - why acid-base relevant
  - how to diagnose acid base disorders
  - how to treat acid base associated conditions

Diagnoses and treatment

- Lactic acidosis
  - correct or resect ischemic tissue
- ASA intoxication
  - alkalinize the urine with IV bicarb drip
- COPD attack
  - bronchodilators, steroids, BiPAP, Ventilate
- Corticosteroid effect
  - reduce steroids or resect tumor
- Anxiety
  - psychotherapy, SSRI


See my friend behind me? I secretly ate his share of his fish just now! Hahaha....
HTTP://WWW.CHEIRY.COM/
Canon PowerShot G1, 2001-12-05

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