ABG INTERPRETATION

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Outline of presentation

1. VBG vs ABG
2. Sources of error
3. Few concepts in Physiology
4. Terminology
5. Steps of acid-base analysis
6. Individual acid base disorders
7. Oxygenation and ventilation
8. Examples
9. Drawbacks of ABG

Venous pH can safely replace arterial pH in the initial evaluation of patients in the emergency department

A M Kelly [1], R McAlpine, E Kyle

- Shows close correlation in pCO_2 and pH
- **VBG pCO_2 > 45 mmHg** has **100 % sensitivity** and NPV for predicting arterial hypercarbia

(i.e. if VBG PaCO2 < 45 → rules out arterial hypercarbia)

- VBG pCO_2 can be used to screen for arterial hypercarbia
- VBG is less painful
- VBG is acceptable if only pH and pCO_2 are required

VBG	ABG
pH ↑ (by ~ 0.04)	pH ↓
pCO2 ↑ (by 4-5 mmHg)	pCO2 ↓

EMA Emergency Medicine Australasia

Review article: Can venous blood gas analysis replace arterial in emergency medical care

Anne-Maree Kelly

First published: 08 December 2010 | https://doi.org/10.1111/j.1742-6723.2010.01344.x | Citations: 59

Anne-Maree Kelly, MD FACEM, Director, JECEMR and Professorial Fellow.

Sources of error

Time delay

1. Escape of cellular contents – K^+ increases
2. Compartment shift – Na^+ decreases
3. Metabolism by RBCs – PaO_2 decreases and $PaCO_2$ increases. pH increases as cells use HCO_3^-

Hypothermia

1. pO_2 and pCO_2 decreases

Air bubbles

1. pO_2 increases and pCO_2 decreases
2. iCal decreases – because pCO_2 decreases, there is release of H^+ from albumin and in turn calcium is bound to albumin

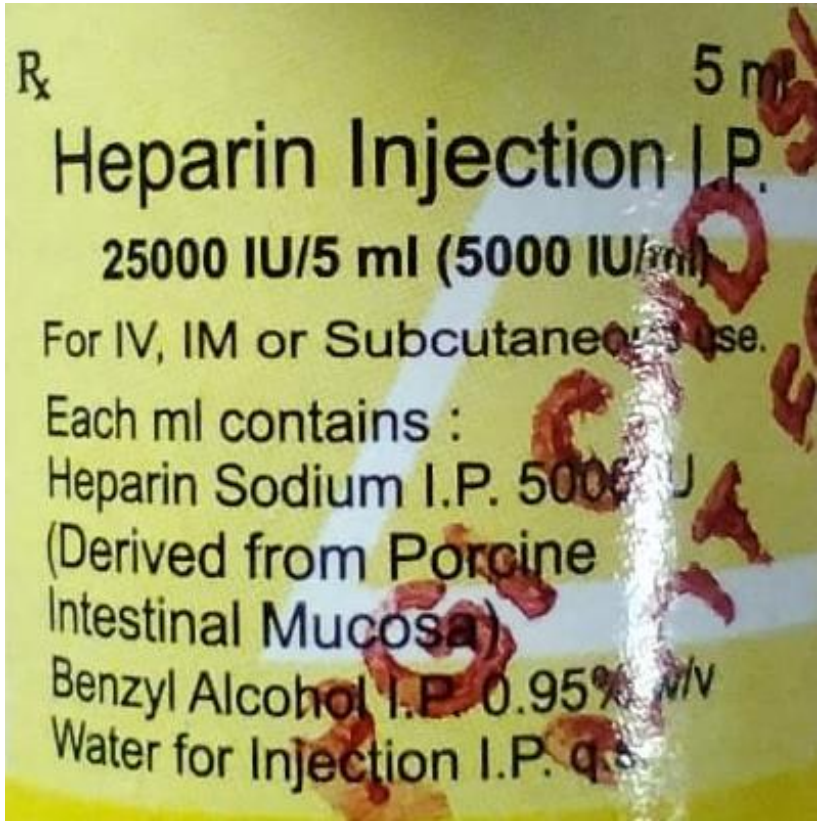
Leucocyte larceny

1. Spurious hypoxemia due to very high TLC

Sources of error

Excess heparin

1. Dilutional decrease in HCO_3^- and pCO_2
2. Effect is increased with increased size of needle (increased dead space) or decreased volume of blood
3. Recommended dose ~ 0.05 mL of low concentration (1000 IU/mL) heparin per mL of blood
4. A 2mL blood sample should have 0.1 mL of heparin which is roughly the dead space of the needle



A few concepts in Physiology

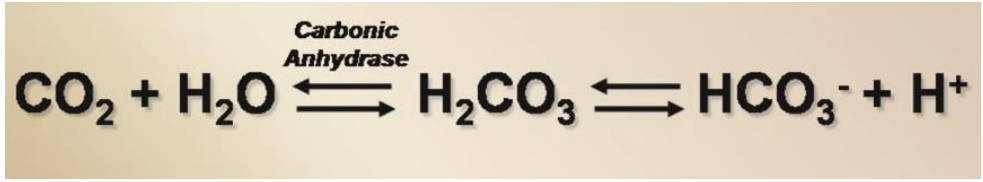
Q Why does pH matter? → Physiologic pH is essential to prevent enzyme inactivation and denaturation

Net acid production = Net acid elimination

Produced in normal metabolism → Intravascular transport → Eliminated via lungs + kidneys

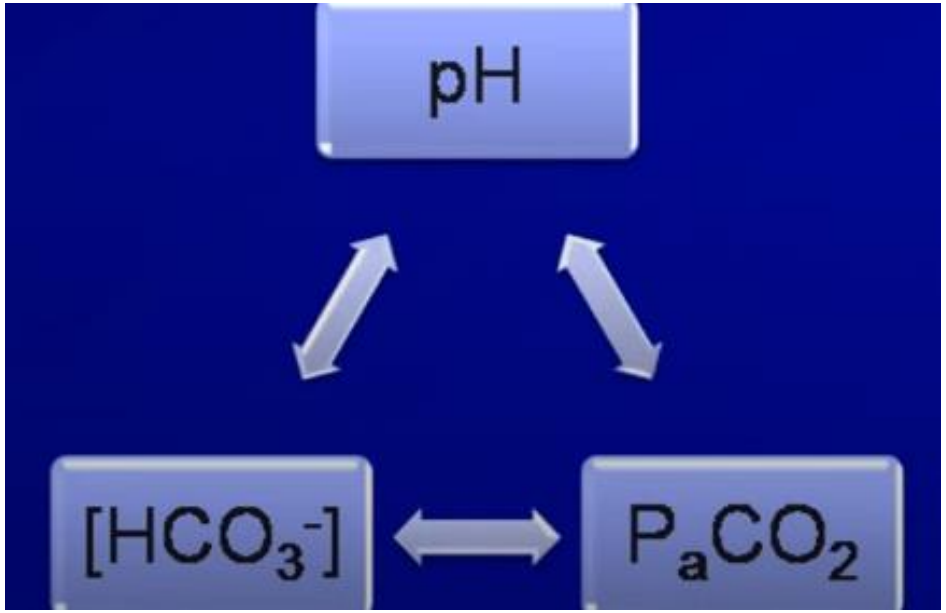
"buffers" to prevent large pH fluctuations (incompatible with life)

Most important buffer is **HCO_3^- /CO_2 buffer system**



A few concepts in Physiology

Henderson-Hasselbalch equation



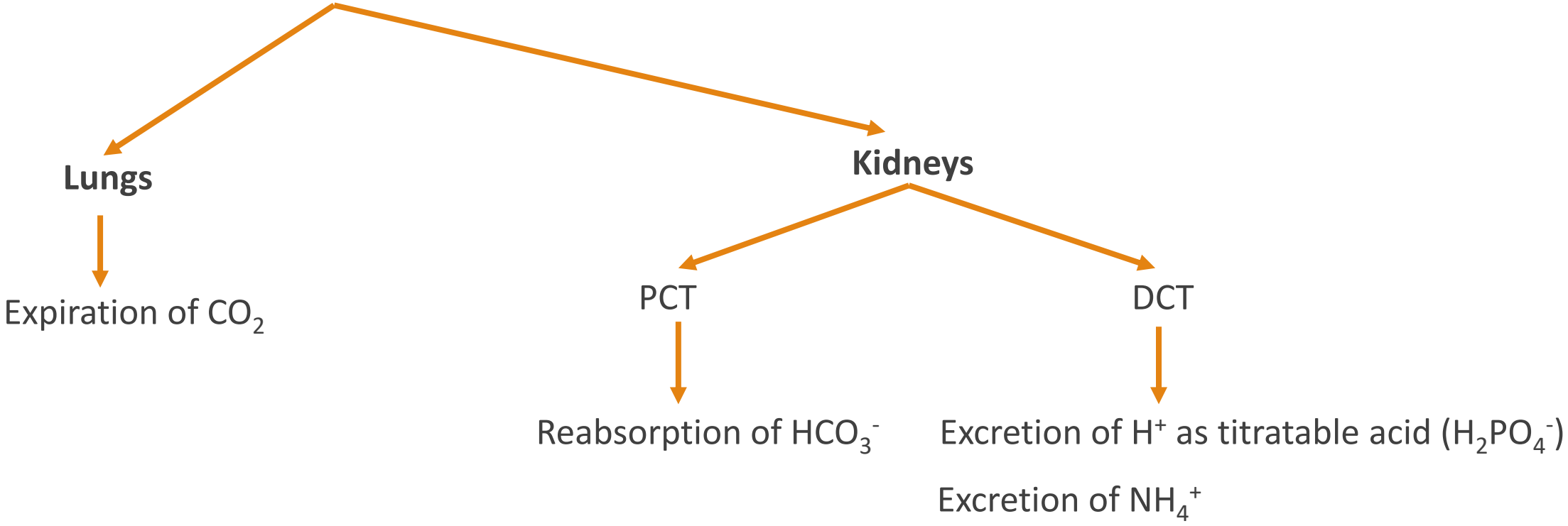
$$pH = pK_a + \log\left(\frac{[A^-]}{[HA]}\right)$$

$$pH = pK_a + \log\left(\frac{[HCO_3^-]}{[H_2CO_3]}\right)$$

$$pH = 6.1 + \log\left(\frac{[HCO_3^-]}{0.03 \times P_aCO_2}\right)$$

A few concepts in Physiology

How is acid eliminated?

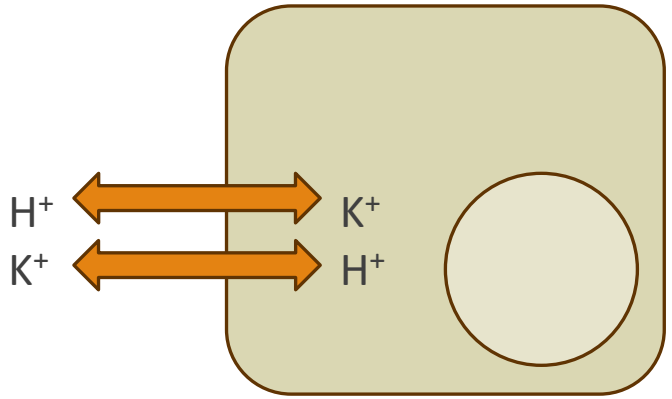


A few concepts in Physiology

- **K^+/H^+ exchange** (transcellular shifts)

Acidemia → Hyperkalemia

Alkalemia → Hypokalemia



- **All acid-base disorders can coexist** except respiratory acidosis and respiratory alkalosis

Terminology

TERMINOLOGY

1. Acidosis vs Acidemia
2. Alkalosis vs Alkalemia
3. Respiratory vs Metabolic
4. Acute vs Chronic

"**-emia**" → Actual pH of blood
"**-osis**" → process

A patient can be Acidemic or Alkalemic but not both

A patient can have one or more acidosis, or one or more alkalosis or both.

What is **normal**?

- pH 7.35 – 7.45
- pO_2 80 – 100 mmHg
- pCO_2 35 – 45 mmHg
- HCO_3^- 22 – 26 mmol/L

Terminology

1. **Respiratory disorders** – Pathologies which disrupt acid-base balance due to their effect on lung

Respiratory acidosis → ↑ pCO_2 Respiratory Alkalosis → ↓pCO_2

2. **Metabolic disorders** – Pathologies which disrupt acid-base balance due to their effect on anything other than the lung (like kidney and GIT)

Metabolic acidosis →↓ HCO_3^- Metabolic alkalosis → ↑ HCO_3^-

STEPS OF ACID-BASE ANALYSIS

Step 0 – Check internal validity

$$[H^+] = \frac{24\ (pCO_2)}{[HCO_3^-]}$$

Compare this value against the pH table

pH	7.099	(-)
PO_2	49.5	mmHg (-)
PCO_2	31.3	mmHg (-)
$cHCO_3^-$	9.5	mmol/L

Example:

- Calculated $[H^+]$ = [24][31.3]/[9.5]

  = 79.07

- Corresponding pH from table is 7.10 which is comparable to pH calculated from ABG.
- The ABG is valid

pH (from ABG report)	$[H^+]$ (nmol/L) (check from formula)
7.00	100
7.05	89
7.10	79
7.15	71
7.20	63
7.25	56
7.30	50
7.35	45
7.40	40
7.45	35
7.50	32
7.55	28
7.60	25
7.65	22

Step 1 – Check pH

pH < 7.35 → Acidemia

(and at least 1 acidosis is present)

pH > 7.45 → Alkalemia

(and at least 1 alkalosis is present)

Normal pH ≠ No acid-base disorder

Step 2 – Check pCO_2 **ROME**

pH and pCO_2 in **O**pposite directions

Respiratory pathology

pH and pCO_2 in same (**E**qual) directions

Metabolic pathology

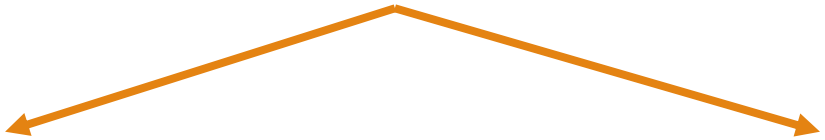
Step 3 – Compensation

1. Aim is to bring pH back to baseline (but generally does not return pH back to normal)
2. Respi pathology → Compensation is by kidneys (takes time – **12 hours - 5 days**)
3. Metab pathology → Compensation is by lungs (relatively rapid – **1-24 hours**)
4. If compensation is not adequate → it is likely that another acid-base abnormality is present [Mixed disorder]

Overcompensation never occurs

Primary acid-base abnormality	Compensation
Metabolic acidosis	Hyperventilation to decrease pCO_2
Metabolic alkalosis	Hypoventilation to increase pCO_2
Compensatory mechanism for Metab alkalosis is relatively poor as compared to others (Hypoventilation → Dec P_AO2 → stimulus to breathe more)	
Respiratory acidosis	Increased reabsorption of HCO_3^- and excretion of H^+ by kidneys
Respiratory alkalosis	Decreased reabsorption of HCO_3^- and excretion of H^+ by kidneys

Different approaches are there



Boston approach

- Bicarbonate based
- Commonly used
- Contains the 6 bicarbonate based “bedside rules” for compensation

Copenhagen approach

Standard base excess (SBE) based

Formulae – Bicarbonate based **Boston** interpretation

1. All are approximations
2. Multiple formulae may exist for the same disorder

Metab acidosis:

Winter's formula $pCO_2 = 1.5(HCO_3^-) + 8 \pm 2$

Metab alkalosis:

$pCO_2 = 0.7(HCO_3^- - 24) + 40 \pm 2$

	Acute	Chronic
Respiratory acidosis	HCO_3^- increases by **1** mEq/L for each 10 mmHg pCO_2 above 40 mmHg	HCO_3^- increases by **4** mEq/L for each 10 mmHg pCO_2 above 40 mmHg
Respiratory alkalosis	HCO_3^- decreases by **2** mEq/L for each 10 mmHg pCO_2 below 40 mmHg	HCO_3^- decreases by **5** mEq/L for each 10 mmHg pCO_2 below 40 mmHg

"1-4-2-5"

Step 4 – Anion gap

Q Why do we have to calculate anion gap?

A To narrow the differential

[HAGMA vs NAGMA]

Total cations = Total anions

Electroneutrality is always maintained in the body

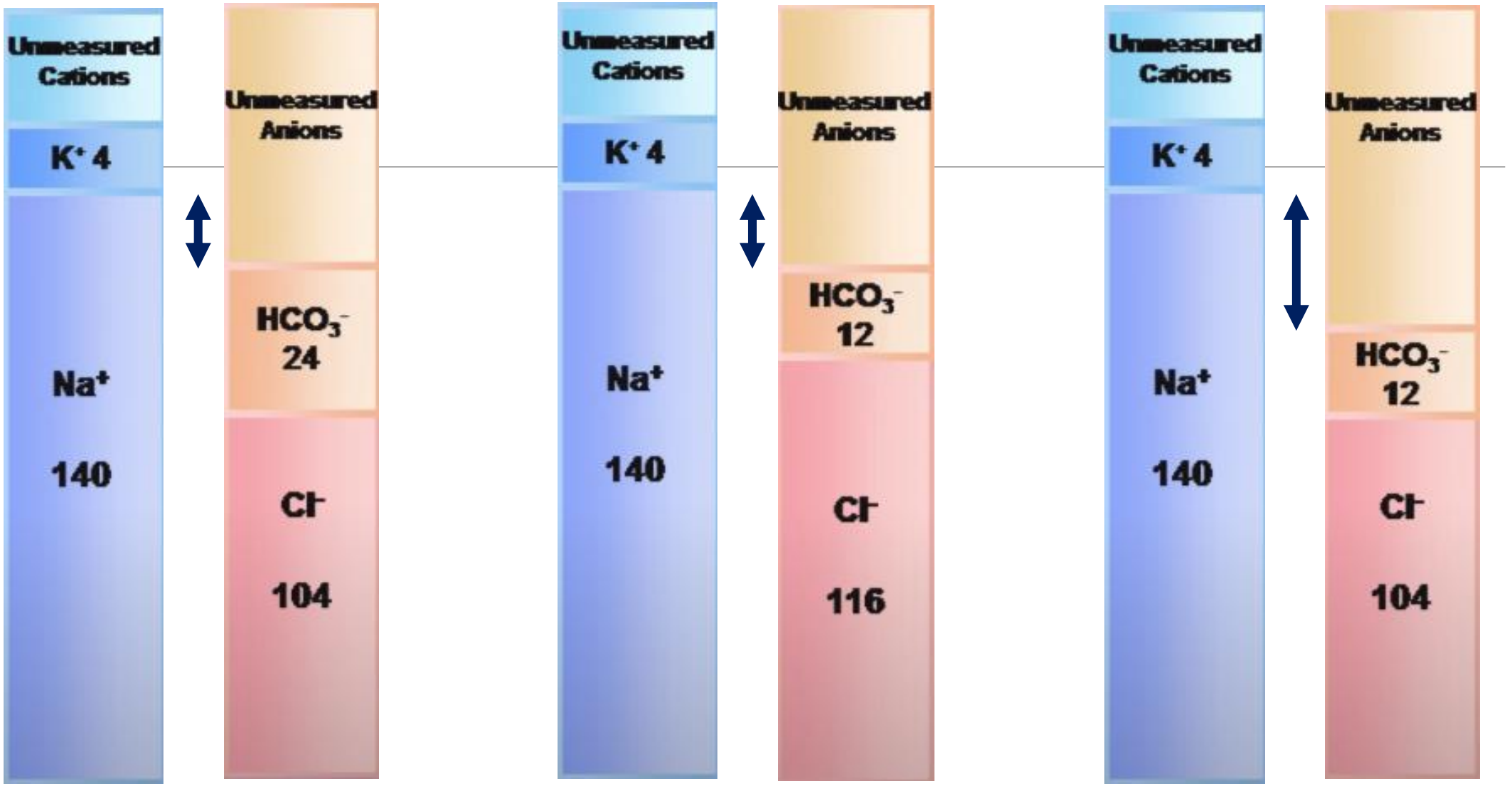
Cations (Measured + Unmeasured) = **Anions** (Measured + Unmeasured)

Unmeasured anions – Unmeasured cations = Measured cations – Measured anions

Anion gap = $Na^+ – [HCO_3^- + Cl^-]$

Why is potassium not a part of this?

Maybe because the absolute contribution to changes in potassium is so small that it can be neglected
Inclusion of K^+ is variable in the literature and it does not affect the interpretation in most cases



Normal

NAGMA

HAGMA

High AG	Low AG	Negative AG
HAGMA	Lab error (underestimate Na, Overestimate Cl, HCO_3^-)	Lab error
Hyperalbuminemia	Hypoalbuminemia	Multiple myeloma → pseudohyponatremia
Hyperphosphatemia	Monoclonal IgG/Polyclonal gammopathy	Bromide/Iodide intoxication
Anionic paraprotein (IgA)	HyperCa/HyperMg	Lithium chloride intoxication
Metabolic alkalosis	Bromide (Myestin, Grilinctus)/Iodide intoxication	
	Lithium chloride intoxication	

Dec in unmeasured anions

Inc in unmeasured cations

Interfere with chloride analysis and produce "**pseudohyperchloremia**"

Albumin correction of AG

1. Albumin is the main contributor of AG → if there is reduction in albumin then AG baseline has to be adjusted appropriately
2. Low albumin → Falsely low AG
3. $\mathbf{AG_{adjusted} = AG_{measured} + 2.5\ [4 - Albumin]}$

For comparison

$Ca_{adjusted} = Ca_{measured} + 0.8\ [4 - Albumin]$

Osmolal gap

- Difference between measured serum osmolality and calculated serum osmolality
- Calculated serum osmolality (mOsm/kg) = $2\,[Na^+] + \frac{BUN}{2.8} + \frac{Glucose}{18}$
- Normal = – 10 to + 10 mOsm/kg
- Major causes:

1. Ethanol
2. Toxic alcohols and glycols – Methanol and Ethylene glycol
3. Advanced CKD
4. Pseudohyponatremia (due to hyperlipidemia or hypertriglyceridemia)

Step 5 – Delta ratio

$$\Delta \text{ ratio} = \frac{\Delta \text{ AG}}{\Delta \text{ HCO}_3^-} = \frac{\text{AG}_{measured} - \text{AG}_{normal}}{\text{HCO}_3^-{}_{normal} - \text{HCO}_3^-{}_{measured}} = \frac{\text{AG}_{measured} - 12}{24 - \text{HCO}_3^-{}_{measured}}$$

Delta ratio in HAGMA

In a pure HAGMA, the increase in AG should be equal to the decrease in HCO_3^-

i.e. Delta ratio = 1

Delta ratio	Interpretation
<0.4	Pure NAGMA
0.4 – 0.8	HAGMA + NAGMA
0.8 – 2	Pure HAGMA
>2	HAGMA + Metabolic alkalosis

One would expect the ratio to be 1 for pure HAGMA but in reality it is a range of values because anions are not only buffered by HCO_3^- but by other buffers also

One should not expect this method to yield an accurate stoichiometric information- at best, it may point one to the existence of another acid-base disorder, which may cause one to reconsider that extra bottle of bicarbonate, or bag of saline.

Normal pH ≠ No acid base disorder

Follow the sequence of steps

1. Check pH → Normal
2. Check pCO_2
3. Check compensation → Since pH is normal, evaluating compensation is not appropriate, rather the aim is to identify the balancing disorder. This disorder will have to be the complete opposite of the first disorder to bring the pH to normal.
4. Calculate AG
5. Calculate Delta ratio if AG high

Normal pH acid-base disorders

PCO_2	HCO_3^-	Anion Gap	Mixed Disorder
High	High	Normal	Respiratory acidosis + metabolic alkalosis
High	High	High	Respiratory acidosis + metabolic alkalosis + elevated gap metabolic acidosis
Low	Low	Normal	Respiratory alkalosis + normal gap metabolic acidosis
Low	Low	High	Respiratory alkalosis + elevated gap metabolic acidosis +/- another metabolic disorder
Normal	Normal	High	Elevated gap metabolic acidosis + metabolic alkalosis

Individual acid-base disorders

Metabolic acidosis

Causes of NAGMA

GI Bicarb loss	Renal loss
Diarrhea	RTA – 1, 2, 4
Ureterosigmoidostomy	Early CKD
Volume expansion with NS	

Causes of HAGMA

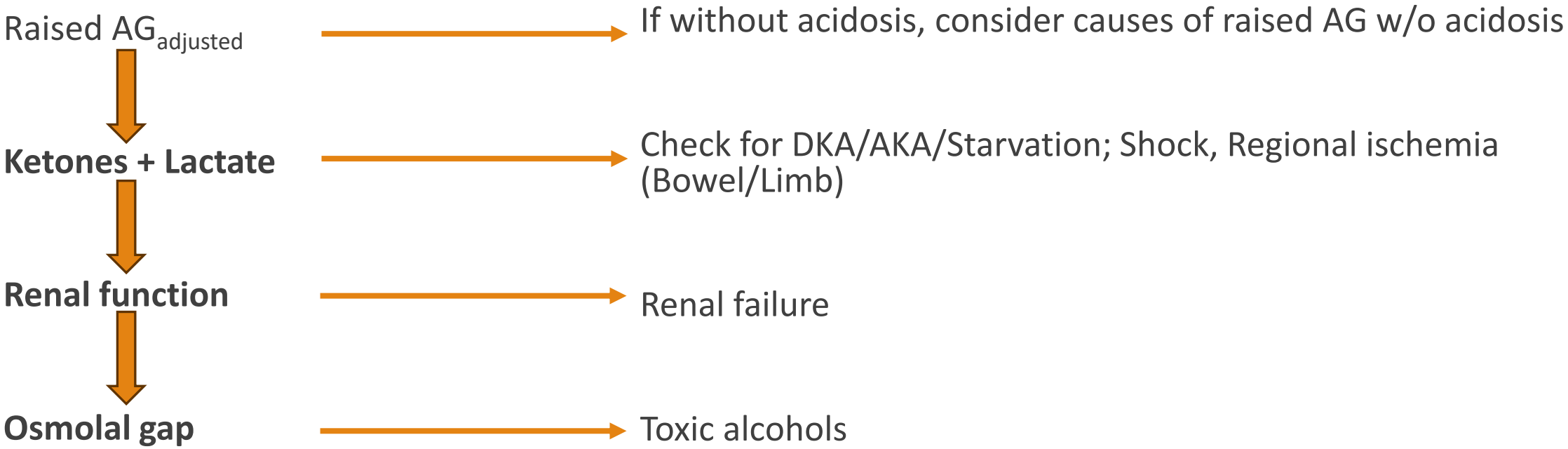
Lactic acidosis	Toxins
Ketoacidosis	Ethylene glycol
Diabetic	Methanol
Alcoholic	Salicylates
Starvation	Propylene glycol
	Pyroglutamic acid (5-oxoproline)
	Renal failure

CKD can cause **NAGMA** in early phases → **HAGMA** in late severe phase

Tubular dysfunction resulting in decreased HCO_3^- reabsorption and impaired H^+ excretion → retained H^+ buffered by HCO_3^- in ECF

Decline in GFR contributes to raising AG by retention of phosphate, sulfate, urate and hippurate

Approach to HAGMA



NAGMA/Hyperchloremic

Concept of **Urine Anion Gap**

Urine Anion gap = [$Na^+ + K^+$] – [Cl^-]

The major unmeasured cation in urine is NH_4^+

(which usually can't be measured directly)

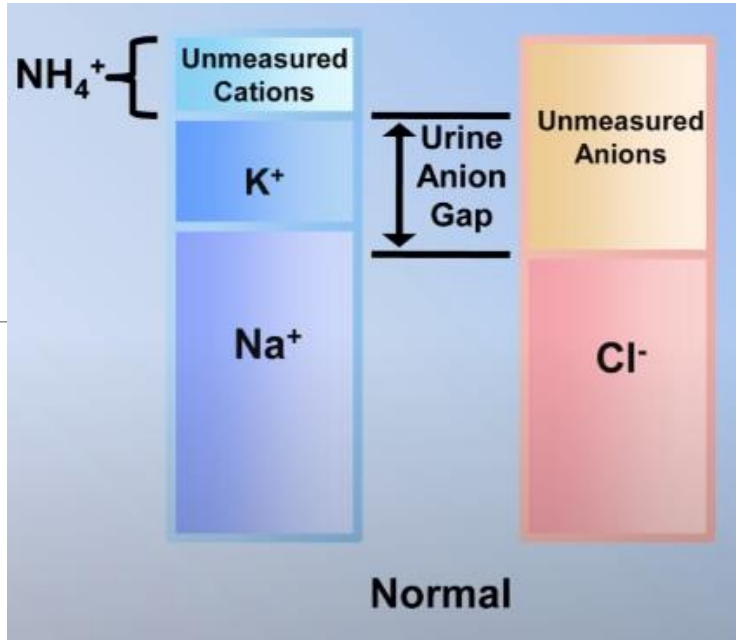
The higher the urine NH_4^+, lower is the UAG

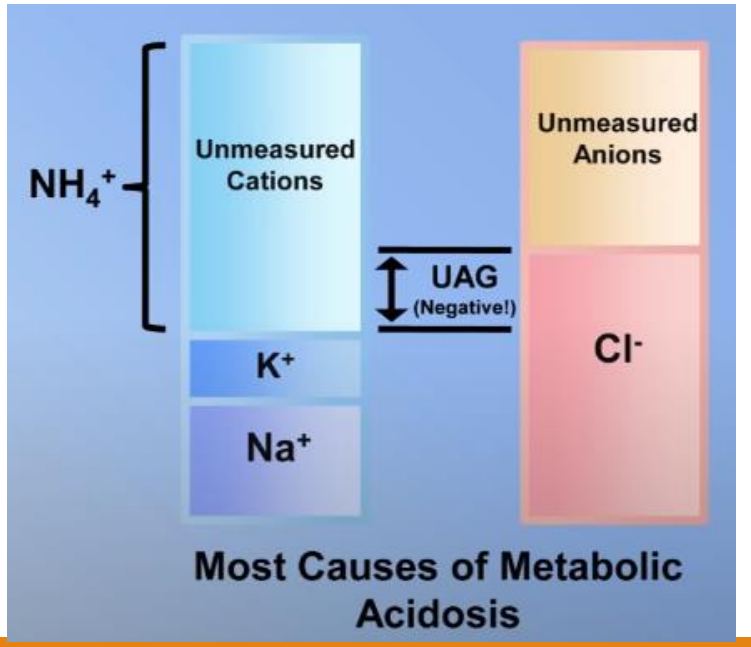
Q Why do we need UAG?

A To indirectly estimate the Urine NH_4^+ excretion

Q Why do we need an estimate of Urine NH_4^+ excretion?

A To narrow down differential of NAGMA





Renal acid secretion

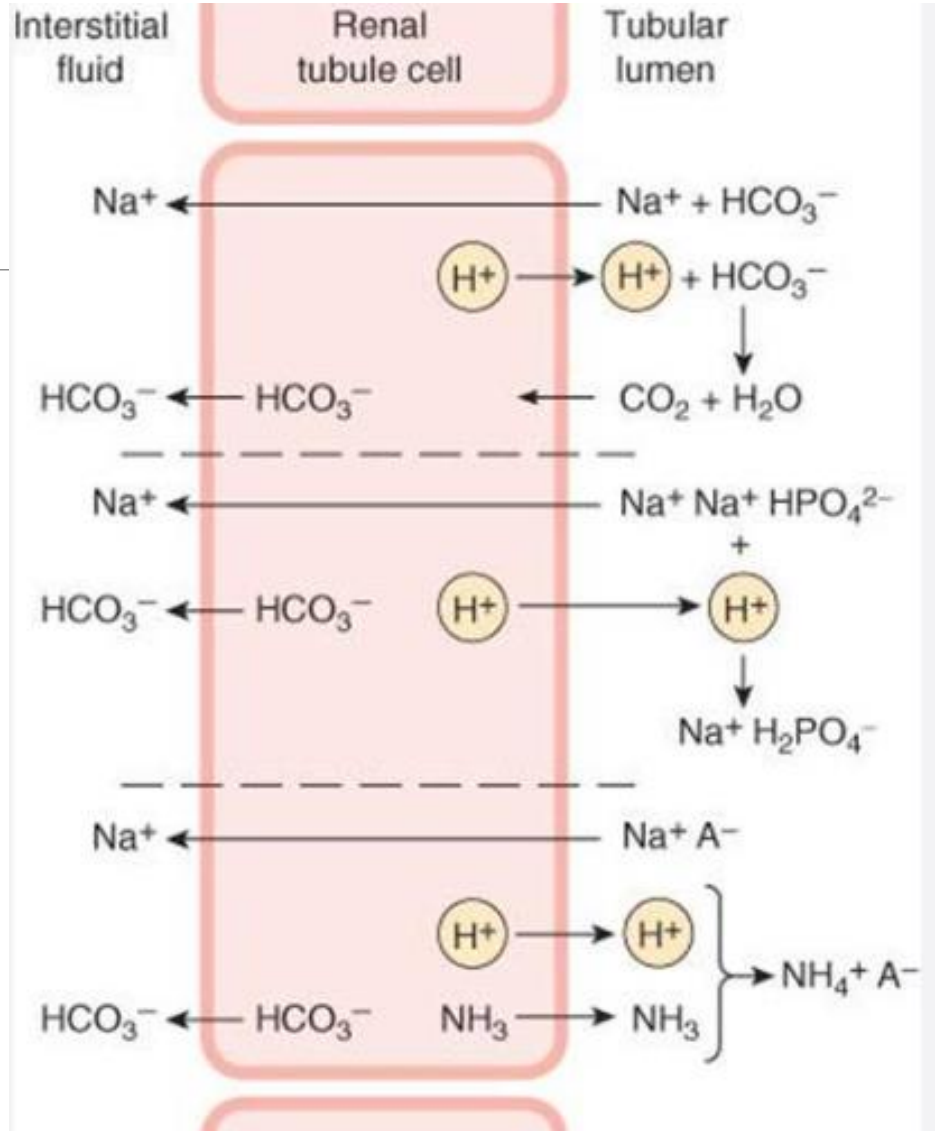
Absorption of HCO_3^-

Secretion of H^+

The 2 main urinary buffers

- Phosphate → $H_2PO_4^-$
- Ammonium → NH_4^+

(stimulated by intracellular acidosis)



Main adaptive response to chronic metabolic acidosis is to increase H+ secretion in the form of **NH_4^+**

Excretion of acid load in urine requires urinary buffers to bind H^+ (if H^+ were free and buffers were not present in urine, the urine pH would fall to <2.5 and result in a high pH gradient between the cell interior and tubular lumen, which cannot be achieved)

NAGMA/Hyperchloremic

Q How does Urine NH_4^+ narrow down differential of NAGMA?

Diarrhea causing NAGMA + hypokalemia

↓

Increased renal acid secretion

↓

Increased urine NH_4^+

↓

Low/Negative UAG

Indicates appropriate acidification of urine

CKD; RTA 1, 4

↓

Impaired distal NH_4^+ excretion

↓

Decreased urine NH_4^+

↓

High UAG

Indicates renal acidification defect

Metabolic alkalosis

Generation – Any factor causing loss of acid or gain of HCO_3^- will generate a Metab alkalosis

This can normally be corrected quickly by renal HCO_3^- excretion → To sustain metabolic alkalosis, other factors are required

Maintenance – When there is failure to eliminate excess HCO_3^- from kidneys from ECF.

Occurs in these situations:

1. Chloride deficiency
2. Hypokalemia
3. Volume deficiency

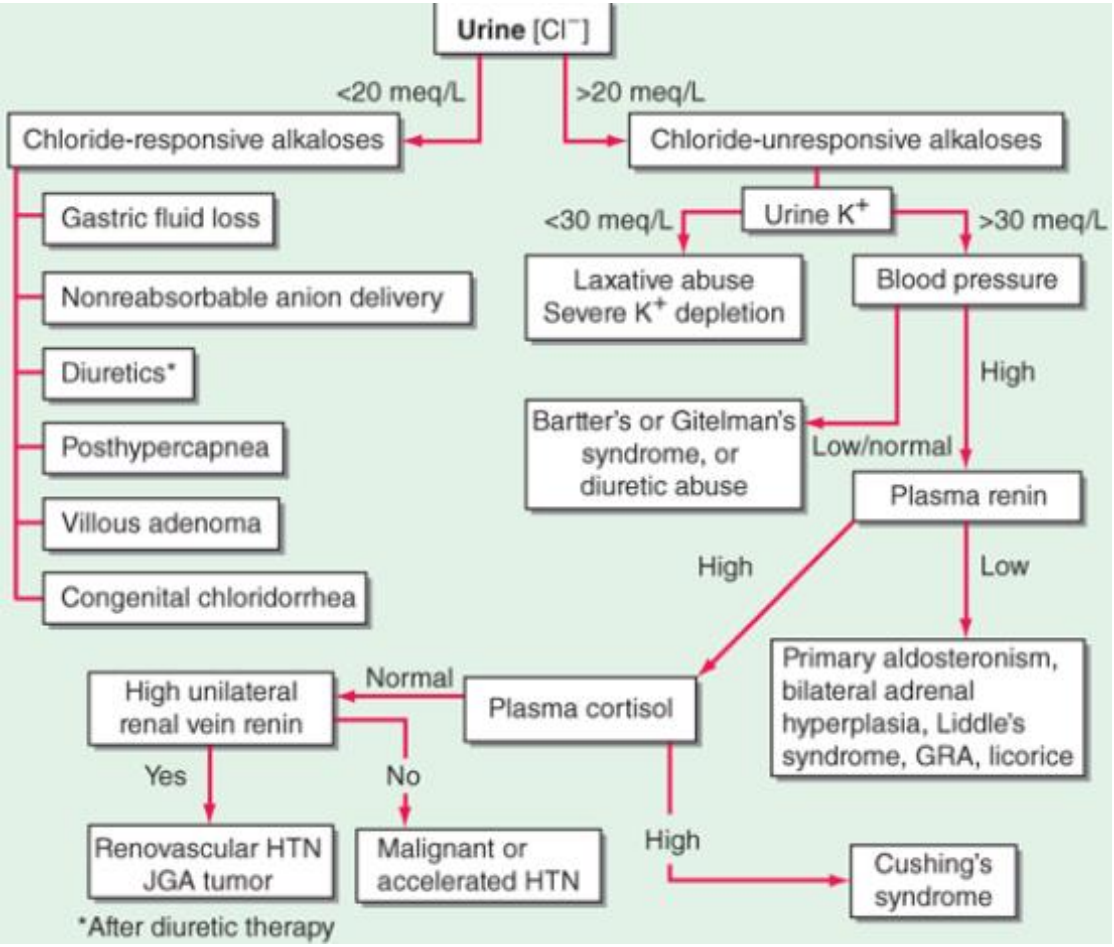
} In combination with reduced eGFR

4. Hypokalemia due to autonomous hyperaldosteronism

Causes of metabolic alkalosis

Primary issue	GI	Renal
Loss of H^+	Vomiting	Loop/Thiazide diuretics
	NG suction	Mineralocorticoid excess
	Congenital chloride diarrhea	Contraction alkalosis
		Post hypercapneic
		Bartter/Gitelman syndromes
Gain of HCO_3^-	Milk-alkali syndrome	Contraction alkalosis
	Ingestion of $NaHCO_3^-$	

Approach to metabolic alkalosis



Differential diagnosis requires assessment of volume status, BP, Serum K^+ and assessment of RAAS.

Can be divided into

1. **Saline responsive** (Urine Cl^- < 20 mEq/L)
2. **Saline unresponsive** (Urine Cl^- > 20 mEq/L)

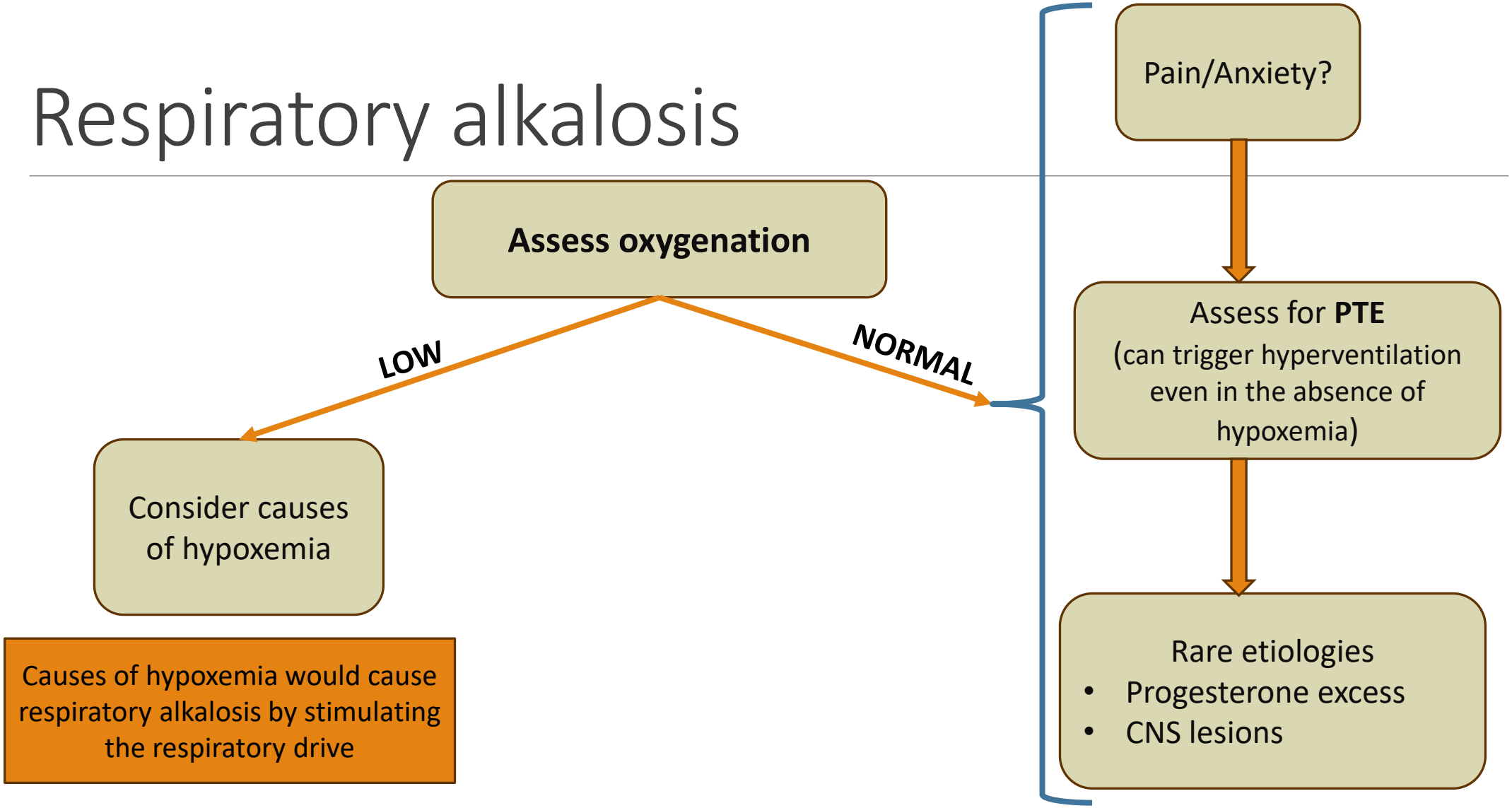
T/t

1. Underlying disorder
2. Treat the factors responsible for maintenance (ECFV deficiency, Hypokalemia, Chloride deficiency) → i.e. Normal saline

Respiratory acidosis

MECHANISM		ETIOLOGY
Decreased minute ventilation	Central hypoventilation	Sedatives CVA/Brainstem disease/Hypothyroidism
	Neuromuscular	Spinal cord + Thoracic cage + Metabolic disorders
Increased dead space		Short shallow breathing COPD
Increased CO_2 production		Fever Thyrotoxicosis Sepsis

Respiratory alkalosis



Assessment of oxygenation and ventilation

Hypoxia and O_2 delivery

Hypoxia

Decreased tissue oxygenation

Hypoxemia

Decreased arterial oxygen concentration

- Hypoxic hypoxia
- Anemic hypoxia
- Stagnant hypoxia
- Histotoxic hypoxia

Hypoxemia is just one of the causes of hypoxia

O_2 delivery		Measured as	Formula
	Dissolved in blood (1.5%)	PaO_2	PaO_2 x 0.03
	Bound to hemoglobin (98.5%)	SpO_2/SaO_2 (Pulse-ox / ABG)	1.34 x Hb x SaO_2

→ Not a good measure of blood oxygenation as it represents only ~1.5% of O_2
Better for assessing "ventilation"

→ **Better measure of arterial oxygenation** as it represents of 98.5% of O_2 in blood

A-a gradient

$$P_AO_2 - P_aO_2$$

Measured directly via ABG

Alveolar gas equation

$$\left(F_IO_2\,(P_I - PH_2O)\right) - \left(\frac{P_aCO_2}{RQ}\right)$$

$$\left(\quad 150\ mmHg \quad\right) - \left(\frac{P_aCO_2}{0.8}\right)$$

Room air and sea level

$P_I = 760$

$P_{H2O} = 47$

$F_IO_2 = 0.21$

Normal diet, RQ = 0.8

1. Check A – a gradient
2. Compare with predicted

   A-a gradient for age =

   (Age/4) + 4
3. Check saturation gap

Increased A – a gradient		Normal A – a gradient
V/Q mismatch (eg Pulmonary vascular disease, embolic disease)		**Hypoventilation** (a/w ↑pCO_2)
Right to Left shunt (severe form of V/Q mismatch in which V/Q is 0)		**Low P_I** (High altitude)
Anatomic shunt AVM Hepatopulmonary syndrome	**Physiologic shunt** Atelectasis Pneumonia	
Diffusion defect (eg ILD)		

The only real utility of A-a gradient in most real-world scenarios is to catch **hypoventilation** as an etiology

Drawbacks of A-a gradient

1. **Variability with age** – Increases with age due to increasing V/Q mismatch. This needs to be accounted for.

2. **Variability with FiO_2** – With increasing FiO_2 (i.e. the patient is on supplemental oxygen) both PAO_2 and PaO_2 will increase but **PAO_2 increases disproportionately** and there is a widening of the A-a gradient.

- **Limited usefulness in critically ill patients in ICUs** because of this, as almost all patients on supplemental oxygen will have a widened A-a gradient.

- It also requires an exact value of FiO_2 which we can only roughly estimate in most ICU situations

Saturation gap = $|SaO_2 - SpO_2|$

- Gap between SaO_2 (on ABG) and SpO_2 (on pulse oximetry)
- Should be <5%
- If **>5%** → significant difference
- Suggests that there is an abnormal hemoglobin which is detected by ABG but not by pulse oximetry

1. Carboxyhemoglobin
2. Methemoglobin
3. Sulfhemoglobin (H_2S)

PaO_2/FiO_2 ratio

- Measure of oxygenation
- Normal – 300 to 500 mmHg
- Can be used as an estimate of A-a gradient if the pCO_2 is normal and no shunt is suspected
- Used in **Berlin definition** for ARDS severity
- *Disadvantage* → It tells the relationship between FiO_2 and arterial O_2 but does nothing to discriminate among the potential causes
- Markedly dependent on FiO_2
- Rule of thumb: $PaO_2 = 500 \times FiO_2$

Example: $FiO_2 = 0.21$

$PaO_2 = 500 \times 0.21 = 105$

P/F ratio		Mortality
200 – 300	Mild ARDS	27%
100 – 200	Moderate ARDS	32%
<100	Severe ARDS	45%

Examples

Example 1

pH 7.34
pCO_2 55
HCO_3^- 29

COPD since 5 years

Step 1 – Validity: 24 x 55/29 = 45.5 → corresponds to pH of 7.35 → Valid

Step 2 – pH → Acidemia

Step 3 – pCO_2 → Respiratory acidosis

Step 4 – Compensation: as it is chronic, HCO_3^- should be 24 + 4[(55-40)/10] = 30

Final Dx – Chronic compensated respiratory acidosis due to COPD

Example 2

pH 7.25
pCO_2 28
HCO_3^- 12

Post cardiac arrest

Step 1 – Validity: 24 x 28/12 = 56 → corresponds to pH of 7.25 → Valid

Step 2 – pH – 7.25 → Acidemia

Step 3 – pCO_2 → Metabolic acidosis

Step 4 – Compensation: pCO_2 = 1.5(12) + 8 ± 2 = 24 to 28 (appropriate)

Final Dx - Compensated metabolic acidosis

?lactic acidosis

Example 3

pH 7.47	K 3.2
pCO_2 25	Cl 110
HCO_3^- 17	Albumin 2.7
Na 148	

75/F

Fever and SOB

RR 35/min
HR 122/min
BP 95/40 mmHg
SpO_2 86% on RA

CXR – Pneumonia

Example 3

Step 1 – Validity: 24 x 25/17 = 35.3 → Corresponds to pH 7.45 → Valid

Step 2 – pH: Alkalemia

Step 3 – pCO_2: Respiratory alkalosis

Step 4 – Expected HCO_3^- is 21 → Metabolic acidosis is present

Step 5 – AG = 148 – (110+17) = 21 → Corrected AG = 24 → HAGMA

Step 6 – Delta ratio = 12/7 = 1.7 → Pure HAGMA

Final Dx – Respiratory alkalosis + HAGMA

Differential – Alkalosis due to hypoxemia and acidosis due to lactic acidosis in sepsis

Example 4

pH 7.41	Na 133
pCO_2 55	K 3.4
HCO_3^- 34	Cl 92

61/M

Morbid obesity
COPD and CHF

Routine ABG during pulmonary testing

Example 4

Step 1 – Validity: 24 x 55/34 = 38.8 → Corresponds to pH 7.40 → Valid

Step 2 – pH: Normal

Step 3 – pCO_2 Elevated → Respiratory acidosis

Step 4 – Compensation: Opposite disorder must be present → Metabolic alkalosis

Step 5 – AG = 133 – (92+34) = 7 → Normal

Final Dx – Respiratory acidosis + Metabolic alkalosis

Differential – Acidosis due to COPD and alkalosis due to diuretics

Example 5

pH 7.41	Na 146
pCO_2 42	K 3.2
HCO_3^- 26	Cl 92

32/F

Vomiting since 4 days

HR 145
BP 78/42

Example 5

Elevated anion gap with **negative delta ratio** may be seen in → **HAGMA + Metabolic alkalosis**

Step 1 – Validity: 24 x 42/26 = 38.7 → Corresponds to pH 7.40 → Valid

Step 2 – pH: Normal

Step 3 – pCO_2 Normal

Step 4 – Compensation: none

Step 5 – AG = 146 – (92+26) = 28 → HAGMA

Step 6 – Delta ratio = $\frac{28-12}{24-26} = \frac{16}{-2} = -8$ → Metabolic alkalosis

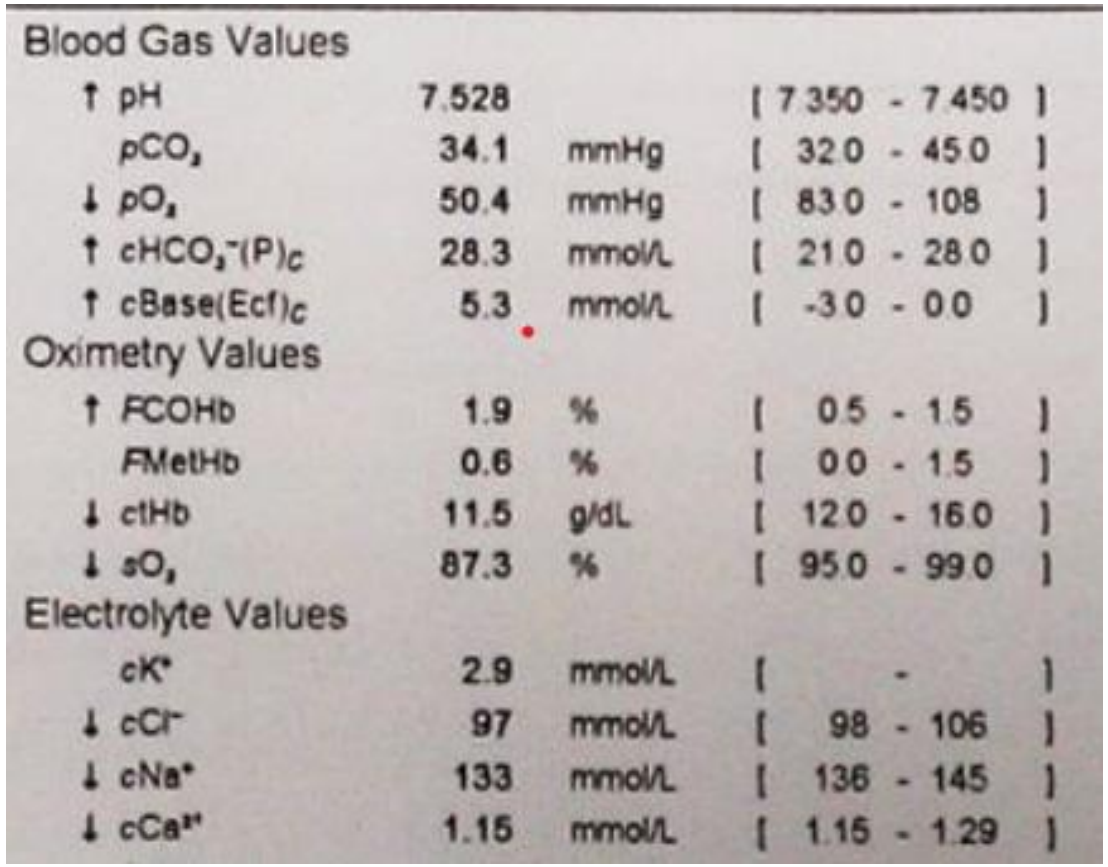
Final Dx – HAGMA + Metabolic alkalosis

Differential – Lactic acidosis due to shock and alkalosis due to vomiting

Example 6

59/M, CAD s/p LAD PCI→ SOB with crepts and RR 30

Given diuretics as CXR was s/o pulm edema



Blood Gas Values			
↑ pH	7.528		[7.350 - 7.450]
pCO_2	34.1	mmHg	[32.0 - 45.0]
↓ pO_2	50.4	mmHg	[83.0 - 108]
↑ $cHCO_3^-(P)_c$	28.3	mmol/L	[21.0 - 28.0]
↑ cBase(Ecf)$_c$	5.3	mmol/L	[-3.0 - 0.0]
Oximetry Values			
↑ FCOHb	1.9	%	[0.5 - 1.5]
FMetHb	0.6	%	[0.0 - 1.5]
↓ ctHb	11.5	g/dL	[12.0 - 16.0]
↓ sO_2	87.3	%	[95.0 - 99.0]
Electrolyte Values			
cK^+	2.9	mmol/L	[-]
↓ cCl^-	97	mmol/L	[98 - 106]
↓ cNa^+	133	mmol/L	[136 - 145]
↓ cCa^{2+}	1.15	mmol/L	[1.15 - 1.29]

1. pH – Alkalemic
2. pCO_2 – Low → Respiratory alkalosis
3. Compensation – Expected HCO_3^- is 22.8 Actual bicarb is 28.3 → Metabolic alkalosis
4. AG – 8
5. Delta ratio – x
6. Final Dx – Respiratory alkalosis + Metabolic alkalosis
7. Differential – Metabolic component due to diuretics and respiratory component due to hypoxia

Drawbacks of ABG

Chest. 1983 Jul;84(1):14-8. doi: 10.1378/chest.84.1.14.

Variability of arterial blood gas values in stable patients in the ICU

S H Thorson, J J Marini, D J Pierson, L D Hudson

1. Pulse oximetry is a better measure of tissue oxygenation than ABG (as SpO_2 is better than PaO_2 for oxygenation)
2. Checking A-a gradient may be misleading in ICU. A normal A-a gradient does not rule out PTE
3. Large **random variability** in PaO_2. This means that a 2nd sample showing a slightly lower PaO_2 may just be random variation and does not necessarily indicate that oxygenation is worsening.
4. Captures only a "snapshot" of the patient status.
5. Painful and invasive

Repeatability of blood gas parameters, PCO2 gap, and PCO2 gap to arterial-to-venous oxygen content difference in critically ill adult patients

Jihad Mallat [1], Ali Lazkani, Malcolm Lemyze, Florent Pepy, Mehdi Meddour, Gaëlle Gasan, Johanna Temime, Nicolas Vangrunderbeeck, Laurent Tronchon, Didier Thevenin

Summary

ACID BASE

- Step 0 – Validity
- Step 1 – pH
- Step 2 – pCO_2 to see primary disorder
- Step 3 – Compensation
- Step 4 – Anion gap
- Step 5 – Delta ratio
- Step 6 – Formulate a differential diagnosis

OXYGENATION

- Step 1 – A-a gradient
- Step 2 – Compare with age corrected
- Step 3 – Saturation gap (to not miss out occult poisoning)

Sources used

1. Harrison's Textbook of Internal Medicine
2. UpToDate – Acid Base disorders
3. Strong medicine – YouTube
4. The ICU book – Paul Marino
5. FOAMed– LITFL, EMCrit, PulmCrit, DerangedPhysiology

Thank you