Approach to Blood Gasses

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Objectives

- By the end of this session attendees should be familiar with:
  1) an approach to arterial blood gasses including assessment of oxygenation and simple/mixed acid base disorders
  2) An approach to anion gap metabolic acidosis
  3) an approach to respiratory acidosis
  4) an approach to non anion gap metabolic acidosis

Disclosures

- I have received honoraria/speaking fees for CME sessions funded by AstraZeneca, Valeo and GSK, none of which are related to today's content
- The university will provide an honoraria for this talk

Disclosures

Blood Gas Normal Values

	Arterial	Venous (peripheral)
pH	7.35-7.45	~7.31-7.42
PaO2	>80	N/A
PCO2	35-45	38-53
HCO3	21-27	23-30
SaO2	>95%	
CO-Hgb	Non smokers: ≤3% Smokers: ≤10-15%	
Met-Hgb	~1%	

Approach to Interpretation

- Step 0 – Ensure the ABG is correct
- Step 1 – Assess oxygenation and consider the A-a gradient, oxygen carrying capacity and oxygen delivery
- Step 2 - Determine if the pH is acidemic, alkalemic, or normal
- Step 3 - Determine which of the $PaCO_2$ or HCO_3^- is causing the above change
- Step 4 - Assess for appropriate compensation
- Step 5 - Measure the anion gap
- Step 6 - If anion gap present, measure the delta-delta

0 – Check for Internal Validity

- Using the Henderson-Hasselbalch equation
- [H+] = 24 x (PCO_2 / HCO_3)

pH	[H+]
7.00	100
7.10	79
7.20	63
7.30	50
7.40	40
7.50	32
7.60	25

1 - Oxygen

- Look at the PaO2
  -What should it be?
- Look at the SaO2
- If Met-Hgb / CO-Hgb available, look at those too

A-a Gradient

- A lot is made of the A-a gradient
- Not overly useful clinically in a lot of scenarios, except if you want to know if hypoxemia is purely due to hypercapnia/hypoventilation

A-a Gradient

- $PAO_2 – PaO_2$
- The gradient between alveolar PO_2 and arterial PO_2
- Reflects how easily oxygen moves from the alveoli to the arterial blood
- If hypoxemia is present with a normal A-a gradient, the hypoxemia is due to hypoventilation (high CO_2) or low inspired O_2
- $PAO_2 = FiO_2(Patm – PH_2O) – PCO_2/R$
  $= 0.21(760 – 47) –$
  $= \sim 150 – PCO_2/0.8 = 150 – 50 = 100$

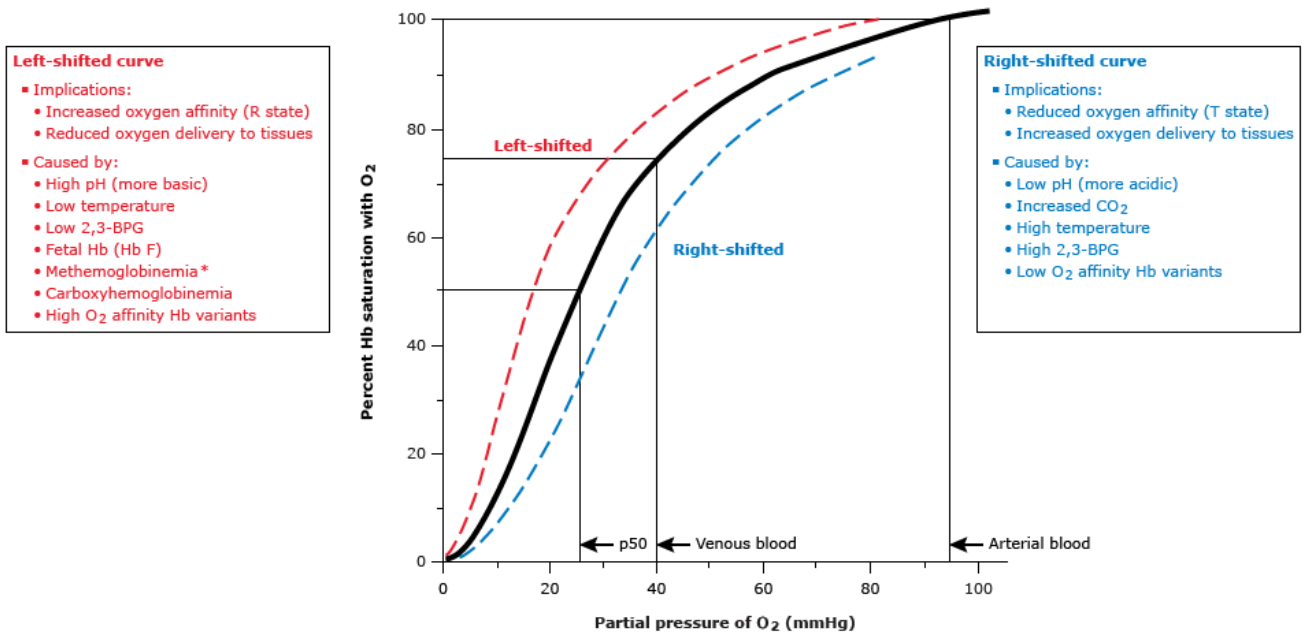
A-a Gradient

- Normal A-a gradient is roughly Age/4 + 4
- On Room air at sea level, if the PCO2 is roughly 60, the PO2 will be roughly 60, assuming a roughly normal A-a gradient

Oxygen is for the tissues

- Arterial oxygen content is highly dependent on hemoglobin concentration
  -$CaO_2 = Hgb \times 1.39 \times SpO_2 + 0.003 \times PaO_2$
- Oxygen delivery depends on CaO_2 and cardiac output
  -$DO_2 = CaO_2 \times CO$
- Once O_2 is delivered, then the tissues need to extract it

Oxyhemoglobin dissociation curve



The oxygen-hemoglobin dissociation curve correlates the oxygen saturation of hemoglobin across a range of oxygen pressures. The solid black line shows the curve for normal adult hemoglobin (Hb A). Notable points on the curve include:

- **p50** — The p50 is the pressure at which hemoglobin is 50% saturated (27 mmHg on the X-axis).
- **Arterial blood** — Hemoglobin is approximately 100% saturated at an oxygen pressure of 100 mmHg.
- **Venous blood** — Hemoglobin is approximately 75% saturated.

Conditions that shift the curve may affect oxygen delivery to the tissues; these effects are most pronounced at low partial pressures of oxygen:

- **Left shift** — Conditions that shift the curve to the left (dashed red line) increase the oxygen affinity; hemoglobin holds more tightly onto oxygen and delivers less oxygen to the tissues at a given arterial oxygen pressure. The left-shifted curve for Hb F is what allows transfer of oxygen from the maternal to the fetal circulation.
- **Right shift** — Conditions that shift the curve to the right (dashed blue line) decrease oxygen affinity; hemoglobin holds less tightly onto oxygen and delivers more oxygen to the tissues at a given arterial oxygen pressure.

Hb: hemoglobin; O_2: oxygen gas; Hb F: fetal hemoglobin; R state: relaxed state of hemoglobin; T state: tense state of hemoglobin.

* The ferric hemes of methemoglobin do not bind oxygen, but they increase the oxygen affinity of the normal ferrous heme in the hemoglobin tetramer, shifting the curve left. With high methemoglobin levels, oxygen saturation will be low for a physiologic PaO_2 due to inability of ferric heme to bind oxygen, shifting the curve right.

2 - pH

- Assess whether the pH is academic, normal, or alkalemic

3 - Metabolic or Respiratory

- Assess whether PCO_2 or HCO_3 is causing the pH abnormality
- If they're both contributing, see which one is more abnormal (relative to their normal/baseline)
- Reminder:
  High HCO_3, low $PaCO_2$ = Alkalosis
  Low HCO_3, high $PaCO_2$ = Acidosis

4 - Assess Compensation

- There are a variety of formulas used. They often work out to roughly the same numbers.
- Metabolic Acidosis – Every 1 HCO_3 = 1.2 PCO_2
- Metabolic Alkalosis – Every 1 HCO_3 = 0.6 PCO_2
- Respiratory Acidosis/Alkalosis – Every 1 PCO_2 = 0.4 HCO_3
  -Takes time, acute will be less. ≤0.1.
- If the compensation is inappropriate, there is an additional acid base abnormality

5 - Calculate AG

- Do it every time, regardless of HCO3 or pH
- Na – Cl – HCO3
- Normal is 12, but needs to be corrected for albumin concentration
  - Every decrease in [alb] by 10, reduce "normal" AG by 3 (Eg. If Alb = 30, N AG = 9)
  - If there is less negatively charged albumin around, the normal "gap" should be less

6 - delta delta

- Change in AG / Change in HCO3
- Calculated AG – N AG / N HCO3 – measured HCO3
  -Eg. AG 20 and HCO3 16 -> 20-12/24-16 = 1
- <1 = AGMA + NAGMA
  1-2 = AGMA
  >2 = AGMA + met. alk
- Lactic acidosis ~1.6, Ketoacidosis closer to 1

7 – Assess for underlying cause

Example

- 55 F with a BMI of 45 and 20 PY ex smoking history is brought in somnolent and wheezy with peripheral edema.
- 100/70 HR 105 RR 10 SpO2 70% RA 95% on 3L
- RA ABG: pH 7.15 PO2 35 PCO2 79 HCO3 27
- Na 132 Cl 96 HCO3 27 Alb 30

Example

- 0 – internal validity checks out
- 1 – hypoxemia is present and the A-a gradient is normal
- 2 – pH is acidemic
- 3 – PCO2 is high and bicarb is high. Therefore primary issue is acute respiratory acidosis

Example

- 4 – PCO2 is up by 39. Expect acutely the HCO3 to be up by ~2-4. compensation is appropriate for an acute respiratory acidosis
- 5 – AG is 9. Because Alb is down by 10, a normal AG is 9.
- 6 – not necessary since AG normal

Respiratory Acidosis

- $PCO2 \propto VCO2 / V_A$
- $V_A \propto V_E$ – dead space
- V_A is alveolar ventilation. V_E is minute ventilation.
- Dead space is “wasted ventilation” ie the amount of ventilation that doesn’t take part in gas exchange

Respiratory Acidosis

- It follows, that the approach to hypercapnia can be derived from the above equations
- Practically, most causes of hypercapnia are things that reduce minute ventilation and alveolar ventilation
- “won’t breath, can’t breath, can’t breath enough”

Respiratory Acidosis

Etiologies and mechanism of hypercapnia

Respiratory pathway affecting carbon dioxide elimination	
Central nervous system ↓	**"Won't breathe"**
Peripheral nervous system ↓	**"Can't breathe"**
Respiratory muscles ↓	
Chest wall and pleura ↓	
Upper airway ↓	
Lungs	**Abnormal gas exchange: "Can't breathe enough"**

Respiratory Acidosis

- Initial management of acute respiratory acidosis involves increasing alveolar ventilation
- If there is a rapidly reversible cause (ie. Recently given opioids), then attempts to reverse would be reasonable
- Often there isn't an easily/quickly reversible cause and we need to help increase ventilation while we treat underlying causes

Respiratory Acidosis

- For this case potential contributors to the respiratory acidosis include volume overload and obstructive lung disease likely on a background of obesity hypoventilation syndrome
- So initial treatment would include a trial of BPAP while treating volume overload and possible AECOPD

Example

- 30 M with DM1 presents with 3 days of N/V and SOB. Looks tired, hypovolemic, accucheck is High
- HR 120 BP 100/70 RR 26 SpO2 99% RA.
- RA ABG: 7.05 PO2 114 PCO2 19 HCO3 5
- Na 130 K 4.6 Cl 93 tCO2 7 Alb 41

Example

- 0 – internal validity checks out
- 1 – SpO2 is normal. PaO2 is high. A-a gradient is normal
- 2 - pH is acidemic
- 3 – HCO3 is low, PCO2 is low, therefore primary issue is metabolic acidosis
- 4 – HCO3 is down by 19, PCO2 expected to be down by ~22. Compensation is appropriate.

Example

- 5 – AG is 30. A normal AG is 12.
- 6 – AG is up by 18, HCO3 is down by 17. Delta / Delta is 18/17 = ~1.1. Therefore only AGMA is present.

Example

- Certainly suggestive of DKA
- Still important to consider alternative causes depending on beta hydroxybutyrate concentration
- Management – DKA management… Isotonic IV fluids, potassium replacement, insulin, looking for underlying cause

Causes of AGMA

- MUDPILES CAT
  -Methanol
  - “uremia” (azotemia)
  -(diabetic) ketoacidosis, d lactate
  -propylene glycol, pyroglutamic acid, paraldehyde, PRIS
  -Iron, INH
  -Lactate
  -Ethylene Glycol
  -Salicylates
  -Cyanide
  -Alcohol (/starvation) ketosis
  -Toluene

Example

- 60 F with solitary kidney, DM2, HTN, CKD BL 300 in with 7 days abdominal cramping and diarrhea
- HR 110 BP 115/75 RR 16 SpO2 98% RA
- RA ABG: 7.16 PO2 90 PCO2 36 HCO3 13
- Na 143 K 2.6 Cl 116 tCO2 13 Cr 294 Mg 0.55 Alb 18 Ca 2.07

Example

- 0 – Checks out
- 1 – SpO2, PaO2, A-a gradient are normal
- 2 – pH is acidemic
- 3 – HCO3 is low, CO2 is low. Primary issues is metabolic acidosis.
- 4 – HCO3 is down by 11, Expect PCO2 to be down by ~13 (~27). PCO2 is 36, inappropriately high, so additional resp acidosis

Example

- 5 – AG is 14. Alb is 18, so a normal AG is 6. Therefore, there's also an anion gap acidosis.
- 6 – AG is up by 8. HCO3 is down by 11. Delta / Delta = 8/11. <1. Therefore there's an AGMA and NAGMA

Example

- AGMA – work through your usual ddx. Practically important to check ketones, lactate, and consider the longer ddx
- Resp acidosis – In this case the CO2 is mildly higher than it should be, this is a minor component of the case. Think through your usual approach to insufficient ventilation especially looking for quickly treatable causes

NAGMA

- H+ Gain: Saline
- HCO_3 – loss:
  Gut: Diarrhea, high output ostomy
  Renal: Type 2 RTA. Acetazolamide
- Reduced H+ Excretion: Type 1 RTA, Type 4 RTA

NAGMA

- Approach to NAGMA is based on the clinical context as well as the renal response to acidemia.
- The renal response is assessed by calculating the urinary anion gap
- The urinary anion gap gives us information on if the kidneys are appropriately acidifying the urine

NAGMA

- If the kidneys are not appropriately acidifying the urine, then that suggests they are at least part of the problem causing the NAGMA
- If the kidneys are appropriately acidifying the urine then that suggests the problem causing the NAGMA is elsewhere (exogenous H+ gain or GI HCO_3 losses)

NAGMA

- UAG = (UNa + UK) – UCl
- UAG should be negative (Neg-GUT-tive) if the kidneys are appropriately excreting the acid load (ie. The GUT is the problem… (diarrhea))

NAGMA

- In reality the UAG is not perfect and when calculated values are close to 0 (-20 to +20) it is difficult to interpret
- Also less reliable in setting of AKI, CKD, unmeasured anions in urine (ketones, HCO3, etc)

Example

- NAGMA – mostly diarrhea +/- baseline RTA from DN
- AGMA – azotemia
- Respiratory acidosis – likely her elevated BMI, possible R hemidiaphragmatic paralysis and weakness from profound hypoK impaired her ability to hyperventilate appropriately

In Conclusion

- Don’t ignore the oxygen even if you order an ABG to assess the acid base status
- Practice doing this stuff in real life
- Always calculate the anion gap
- In reality these calculations don’t always work out perfectly and the clinical context needs to be considered when interpreting blood gases

References

- Williams AJ. ABC of oxygen: assessing and interpreting arterial blood gases and acid-base balance. BMJ 1998; 317:1213.
- Hansen JE. Arterial blood gases. Clin Chest Med 1989; 10:227.
- Adrogué HJ, Rashad MN, Gorin AB, et al. Assessing acid-base status in circulatory failure. Differences between arterial and central venous blood. N Engl J Med 1989; 320:1312.
- Mellemgaard K. The alveolar-arterial oxygen difference: its size and components in normal man. Acta Physiol Scand 1966; 67:10.
- Malatesha G, Singh NK, Bharija A, et al. Comparison of arterial and venous pH, bicarbonate, PCO2 and PO2 in initial emergency department assessment. Emerg Med J 2007; 24:569.
- Adrogué HJ, Madias NE. Secondary responses to altered acid-base status: the rules of engagement. J Am Soc Nephrol 2010; 21:920.
- Rose BD, Post TW. Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed, McGraw-Hill, New York City 2001.

References

- Batlle DC, Hizon M, Cohen E, et al. The use of the urinary anion gap in the diagnosis of hyperchloremic metabolic acidosis. N Engl J Med 1988; 318:594.
- Oh M, Carroll HJ. Value and determinants of urine anion gap. Nephron 2002; 90:252.
- Weinberger SE, Schwartzstein RM, Weiss JW. Hypercapnia. N Engl J Med 1989; 321:1223.
- Williams MH Jr, Shim CS. Ventilatory failure. Etiology and clinical forms. Am J Med 1970; 48:477.
- Hasan, A. THE pH. In: Handbook of Blood Gas/Acid–Base Interpretation. Springer, London. 2009 https://doi.org/10.1007/978-1-84800-334-7_6
- Bernstein K. University of Manitoba Pre Clerkship Nephrology Notes. 2011