BLOOD GAS ANALYSIS

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Learning Outcomes

- Indications for blood gas analysis
- Sampling arterial blood gases
- Information derived from blood gas reports
- Interpretation of blood gas data

Clinical Scenario - 1

- 70 year old male, heavy smoker, presented with confusion.

'House officer is to do an arterial blood gas'

Why do a blood gas?

How do I do an arterial blood gas?

How do I interpret the report?

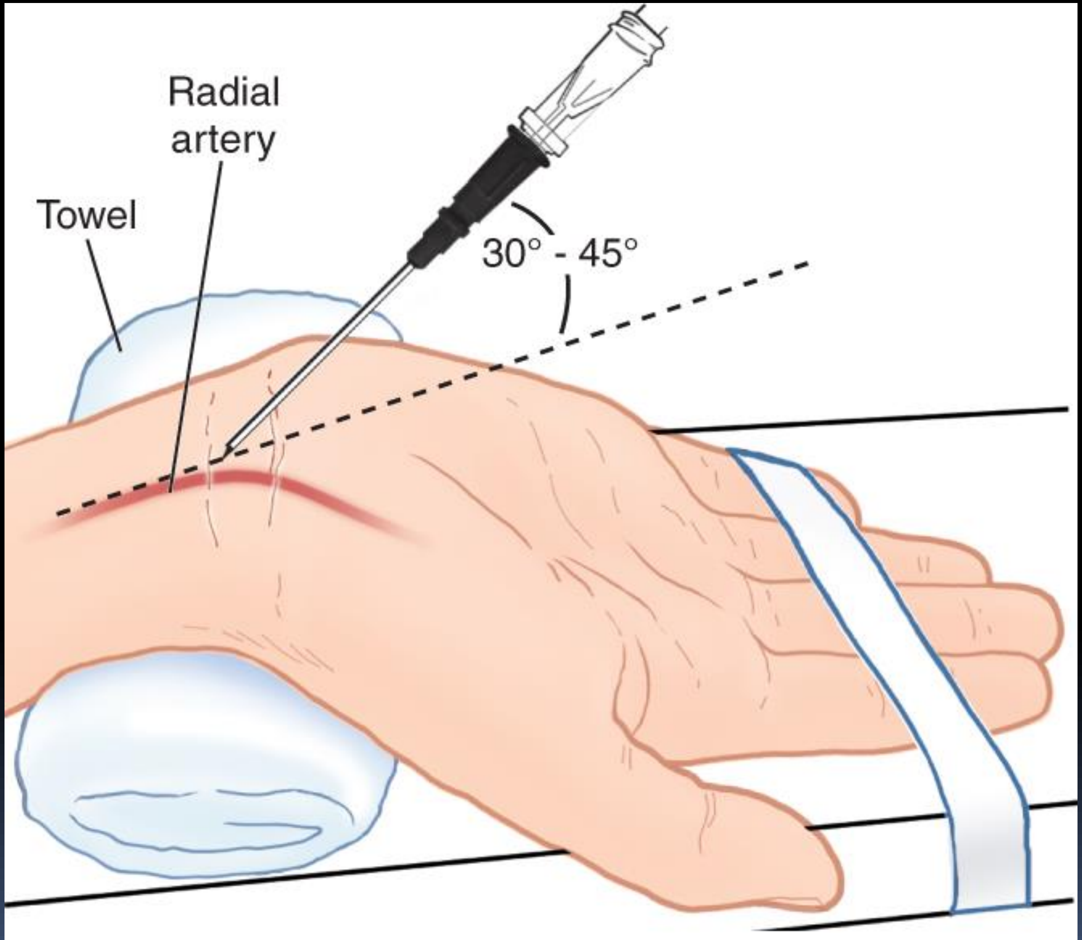
Indications for Blood Gas Analysis

- Assessment of
  Oxygenation
  CO_2 & Ventilation
  Acid-base
  Additional information
  eg.
  Electrolytes
  A-a gradient
  Oxygen supply-demand balance

How Do I Do a Blood Gas?

- Preparation – Contraindications, Blood gas analyser calibrated, consent, assistants, heparinized syringe/ ice bath, select site, aseptic technique
- Site – Radial, ulnar, femoral, brachial, dorsalis pedis
- Note FiO_2, PEEP, respiratory rate, tidal volume
- Sampling technique – Syringe free of gas bubbles, air tight
- Apply direct pressure, KUO for bleeding

Arterial Puncture



Source: Reichman EF: *Emergency Medicine Procedures, Second Edition:* www.accessemergencymedicine.com

Interpreting a Blood Gas Report - Normal values (arterial)

- PH 7.35-7.45

  $PaCO_2$ 35-40mmHg

  PaO_2 90-100mmHg

  SaO_2 95-100%

  Base excess -2 to +2 mEq/l

  Std HCO_3^- 24-28mmol/l

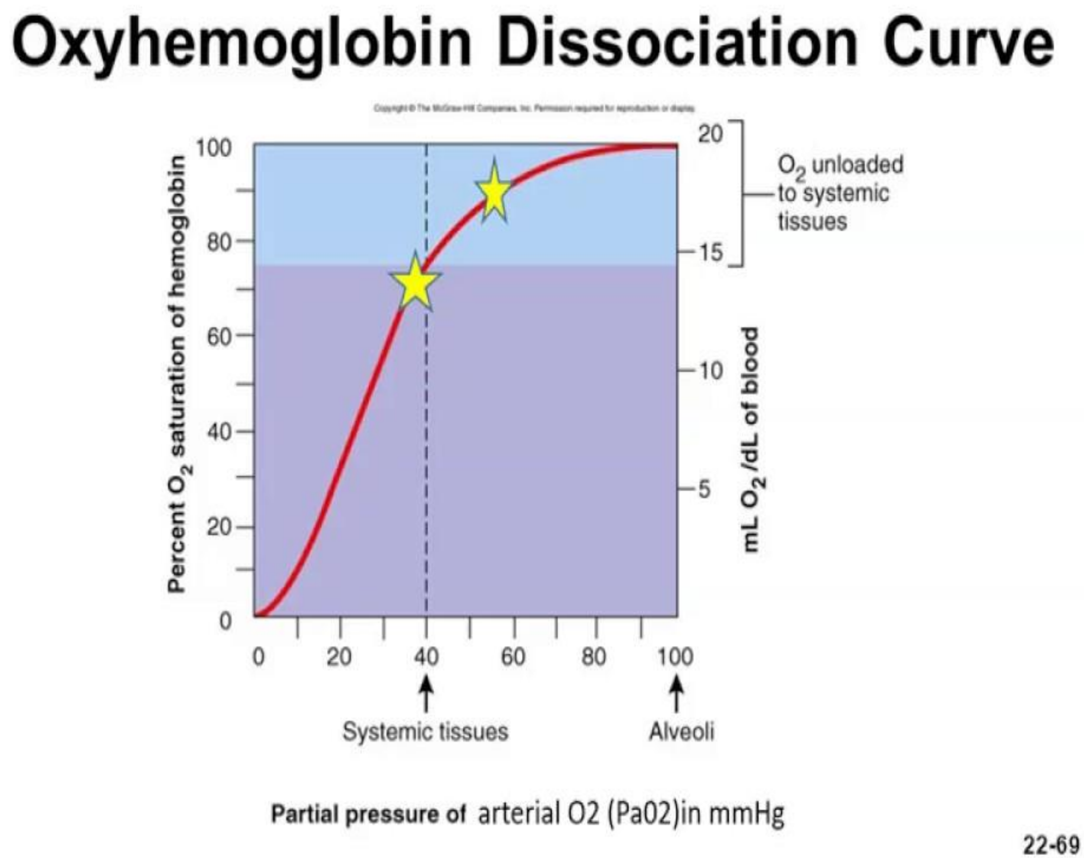
OXYGENATION

Oxygenation

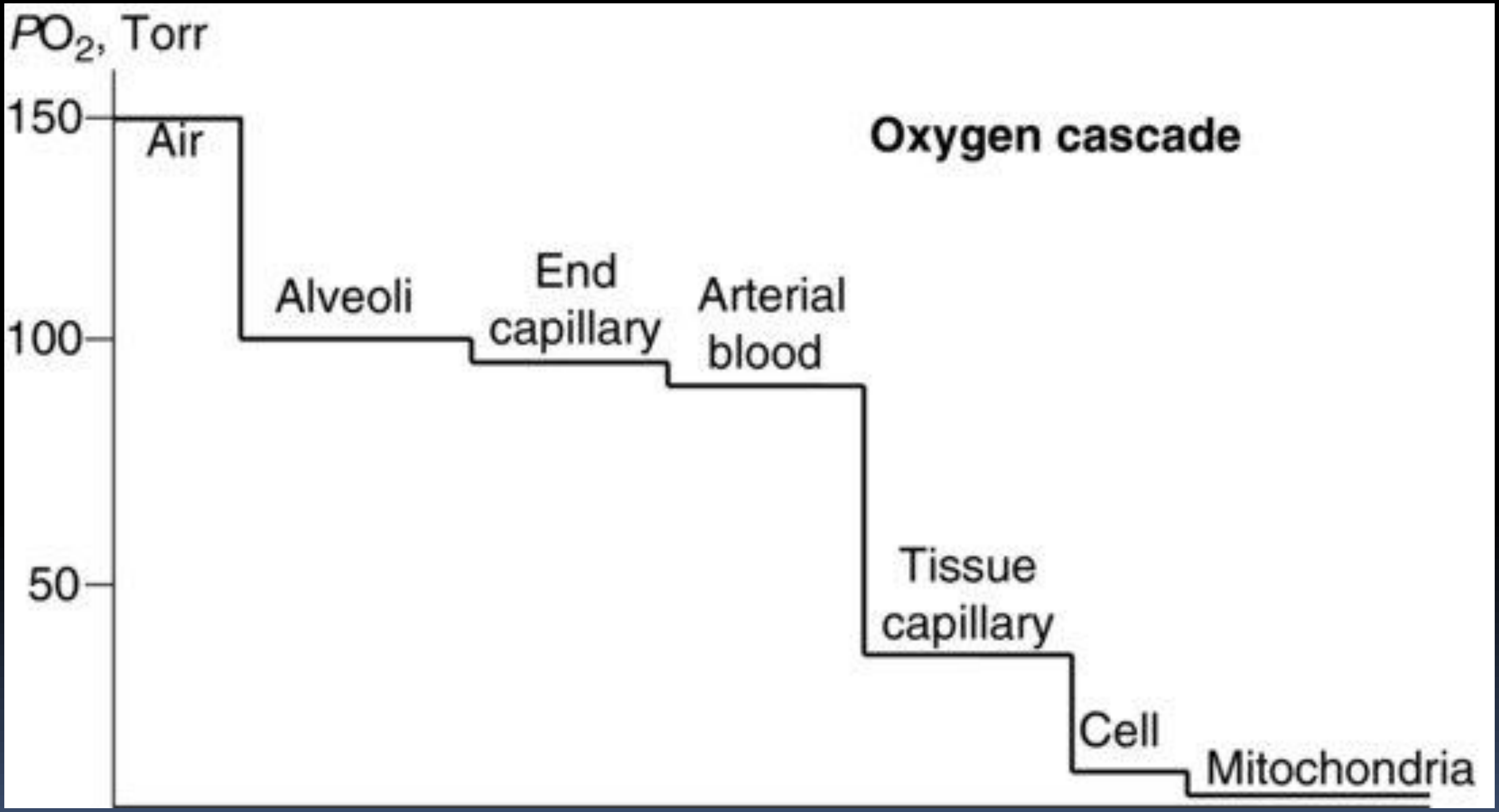
- $SpO_2 = HbO_2 / (Hb + HbO_2)$
- PaO_2 = Partial pressure of O_2 in arterial blood

Relationship: PaO_2 to SpO_2

- What is oxygen saturation?
- What is arterial PA02?
- What is the oxyhemoglobin dissociation curve?



Oxygen Cascade



Alveolar O_2 - (P_AO_2)

- FiO_2 = Inspired O_2
- P_B = Barometric pressure
- PCO_2 = Partial pressure CO_2
- R = Respiratory quotient

Pulmonary Capillary PO_2

- Partial pressure of alveolar O_2 (P_AO_2)
- Permeability of alveolar-capillary membrane
- Mixed venous O_2 content

Arterial PO_2 – PaO_2

- Capillary PO_2
- Shunts

Arterial Hb-O_2 Saturation (SaO_2)

- PaO_2 (O_2-Hb dissociation curve)
- Factors affecting Hb-O_2 affinity
- Competitors for Hb eg Carbon monoxide
- Abnormal Hb (Sickle- HbS)

O_2 Delivery to Tissues

- O_2 Flux (DO_2) = Volume of O_2 delivered to all tissues in a minute

- DO_2 = Cardiac output x O_2 content
- O_2 Content = [(Hb x SaO_2 x 1.34)+ ~~(0.003* x PaO_2)~~]

Venous Oxygen – $SCVO_2$

- O_2 Supply – Demand Balance

- O_2 Supply

  DO_2 = Cardiac output x O_2 content

- O_2 Demand

  VO_2 = O_2 consumption by tissues

O_2 Supply-Demand Gap(O_2 Reserve)

- O_2 Consumed by tissues = X?

  O_2 content in arterial blood = DO_2 = 1000ml

  O_2 content in central venous blood = Hb x $ScvO_2$ x 1.34 = 750ml

- X = 1000ml/min – 750ml/min = 250ml/min

O_2 Supply-Demand Gap(O_2 Reserve)

- $DO_2 - X$ = Venous O_2 content

- With Normal O_2 delivery & consumption

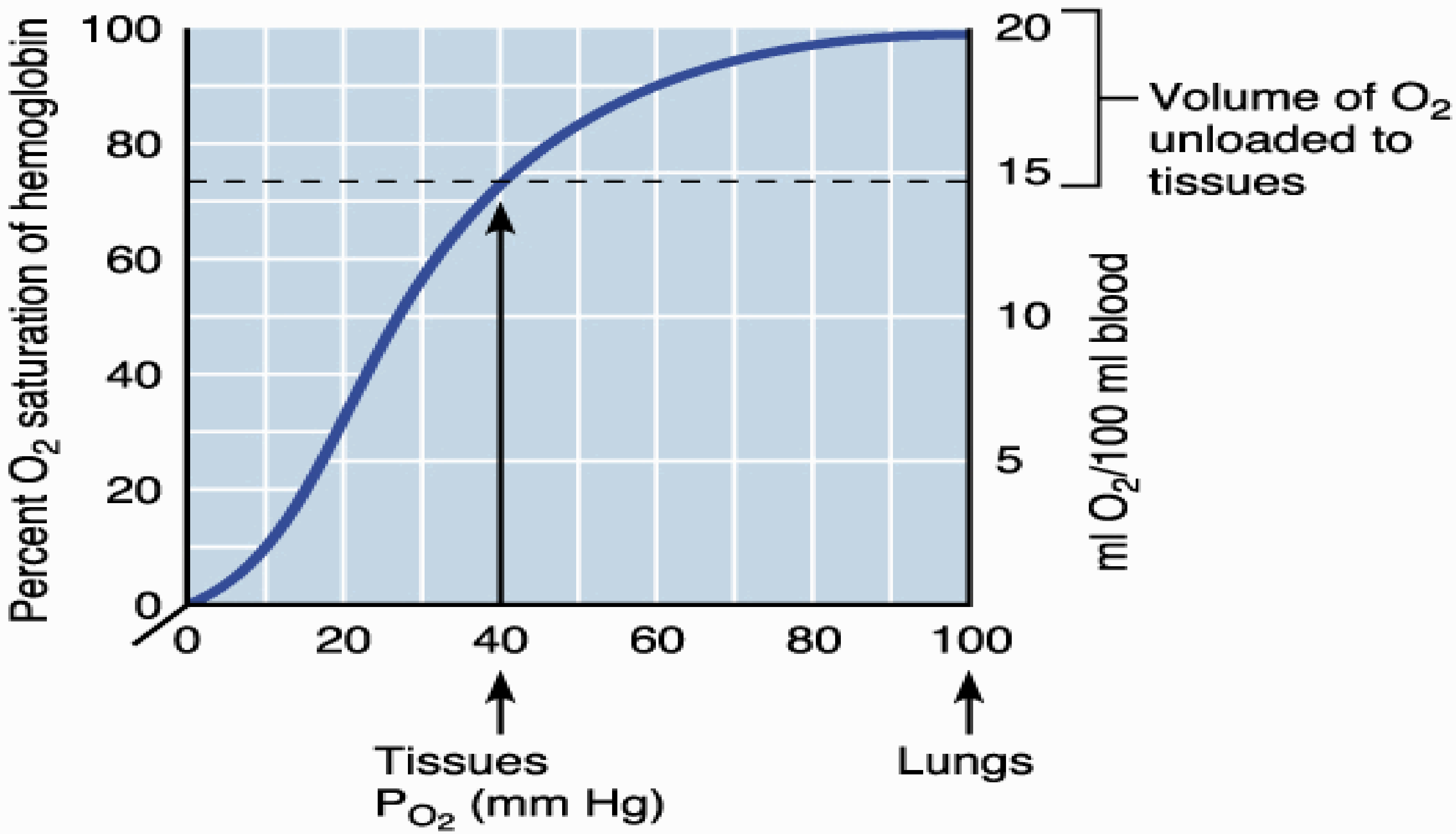
  Central venous O_2 content = 750ml

  Central venous O_2 saturation ($SCVO_2$) = 70%

- If Supply decreases or demand increases

  $SCVO_2 < 70\%$

O_2 Supply – Demand Balance



CARBON DIOXIDE

$PaCO_2$ – Arterial PCO_2

Effects on,

- Ventilatory function
- Cardiovascular effects

    Tachycardia, BP, peripheral resistance

- Cerebral circulation

  Cerebral vasodilation/ constriction

  Intracranial pressure

Factors influencing PaCO2

- Normal $PaCO_2$ - 35-40mHg
- CO_2 production (VCO_2) - metabolism
- CO_2 excretion (VA) - alveolar minute ventilation

$$PaCO_2 = VCO_2 / VA$$

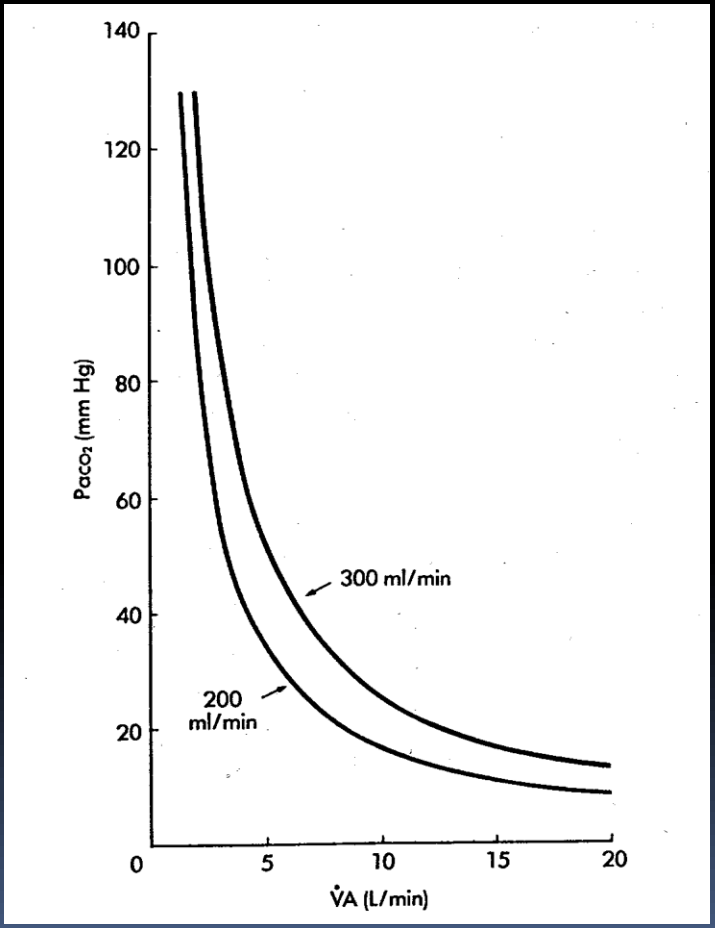
Hypercapnia

$PaCO_2$ 50mmHg (35-40mmHg)

Possible causes?

- Increased production (eg. Sepsis)
- Reduced excretion (eg. hypoventialtion due to narcotic overdose)

Ventilation & PaCO2



pH

pH

- Indicator of H^+ activity/ concentration
- Affects enzyme activity

Regulated by Ventilation

$$CO_2 + H_2O \leftrightarrow$$

$$H_2CO_3 \leftrightarrow$$

$$H^+ + HCO_3^-$$

Regulated by Buffers & Kidneys

Regulated by Kidneys

Acid-Base Homeostasis

- Henderson- Hassellbach equation (simplified)

$$PH \rightarrow \frac{[HCO_3^-]}{[PaCO_2]}$$

Metabolic acidosis- 1

- Due to H+ excess,

  Keto acidosis

  Impaired excretion eg. Acute renal failure
- $H^+ + HCO_3^-$ -----→ H_2CO_3
- $\underline{[HCO_3^-]}$ ↓ →→→ pH↓

  $[PaCO_2]$
- HCO_3- less than 25mmol/l

Arterial Blood Gases in Metabolic Acidosis

pH < 7.35

Base Excess (BE) – More negative than -2

Bicarbonate - Less than 24mmol/l

Compensation in Metabolic acidosis

- $H_2CO_3 \rightarrow CO_2 \uparrow + H_2O$
- CO_2 Stimulates respiratory center
- $\frac{[HCO_3^-]}{[PaCO_2]\downarrow} \rightarrow\rightarrow\rightarrow pH \uparrow$

Arterial Blood Gases in Compensated Metabolic Acidosis

- pH < 7.35 (but closer to 7.4)
- Base Excess (BE) – less negative than before
- Bicarbonate - Lesser than before

Metabolic Alkalosis

- PH > 7.45
- Std HCO_3^- > 28mEq/l

eg. Loss of acid eg. Vomiting & loss of HCl

Addition of alkali eg iv $NaHCO_3$

Respiratory Acidosis

PH < 7.35

$PaCO_2$ > 45mmHg

Std HCO_3^- Normal

Eg. Morphine overdose & respiratory depression

Respiratory Alkalosis

- PH > 7.45
- PCO_2 < 35mmHg
- Std HCO_3^- Normal

eg. Hysterical hyperventilation

Acid-Base Disturbance

- Primary disturbance eg. Metabolic acidosis DKA

1. $[H^+]$ ↑

2. $\dfrac{HCO_3^-}{PCO_2}$ ↓

Compensation for Metabolic Acidosis

- Compensation aims to correct PH
- Respiratory compensation by hyperventilation in DKA

1. $\dfrac{HCO_3^-}{PCO_2} \downarrow$

2. pH $\uparrow$

Case Scenario-1

- ABG report

PH 7.51

$PaCO_2$ 25mmHg

PaO_2 65mmHg on FiO_2 0.6

SpO_2 92%

BE -1

SBC 26

What does the ABG indicate?

Case Scenario-1

- Always relate to the clinical picture

  Age,

  Medical & drug history,

  Progression of present illness,

  Level of conciousness, temperature,

  Respiratory rate, Tidal volume

  Heart rate, BP, CRFT

Case Scenario-2

- 25 year old female, drowsy, febrile
  ABG
  PH 7.15
  PCO_2 18mmHg
  PO_2 98mmHg
  SpO_2 99%
  BE -15
  SBC 14
  RBS 445mg/dl
- What does the ABG indicate?

Summary

- To provide additional information
- Analysis of acid-base, oxygenation, ventilation status
- Correlate clinical findings to blood gas report