ACID-BASE BALANCE
BLOOD GASES

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Blood gases

- pH and pCO_2 may be determined accurately on venous blood.
- Lithium heparin anticoagulant permits use of plasma for other testing.
- SaO_2 may be determined non-invasively.
- Pulse oximetry is not used to assess the adequacy of ventilation, however.
- pO_2 determination is reserved for evaluation of need for O_2 therapy (e.g., mechanical ventilation) as determination requires arterial puncture.

BLOOD GASES

- $pO_2 = FiO_2$ x (760 – 47) mmHg at sea level.
- pO_2 values directly related to O_2 content of inspired gas.
- For each 1000 feet elevation in altitude, pO_2 falls 3-4 mmHg.
- SaO_2 is linearly related with pO_2 and holds to an altitude of 10,000 feet.
- An SaO_2 of 90% implies a pO_2 of >60 mmHg.
- Visible cyanosis in a patient who is not anemic suggests an SaO_2 of <85%. (3g/dl desaturated hemoglobin).

Altitude and Oxygen

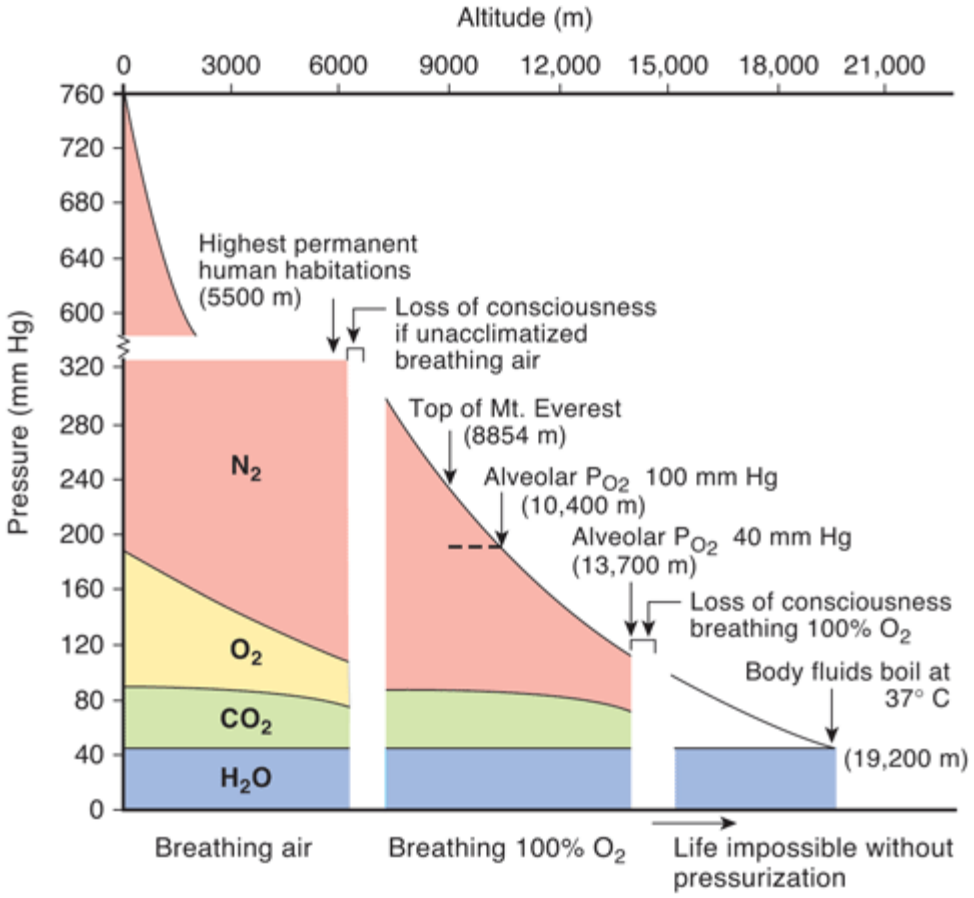

Fig. 36-12 Accessed 02/01/2010

The minimal alveolar PO_2 that an unacclimatized subject can tolerate without loss of consciousness is about 35–40 mm Hg.
Note that with increasing altitude, the alveolar pCO_2 drops because of the hyperventilation due to hypoxic stimulation of the carotid and aortic chemoreceptors.
The fall in barometric pressure with increasing altitude is not linear, because air is compressible.

Blood gases

- Low pO_2 associated with inadequate ventilation, poor pulmonary gas diffusion, airway obstruction, and diaphragm failure.
- High pCO_2 associated with poor pulmonary gas exchange. May aggravate clinical situation if concomitant consumption of >450g carbohydrate/day.
- Low pCO_2 associated with hyperventilation.
- $pCO_2 = FICO_2$ (0.5%) X (760-47) mmHg at sea level
- End tidal pCO_2 is best measure of alveolar concentration.
- Expected Pa_{CO_2} is 1.5 [HCO3-] + 8

Oxygen dissociation curves

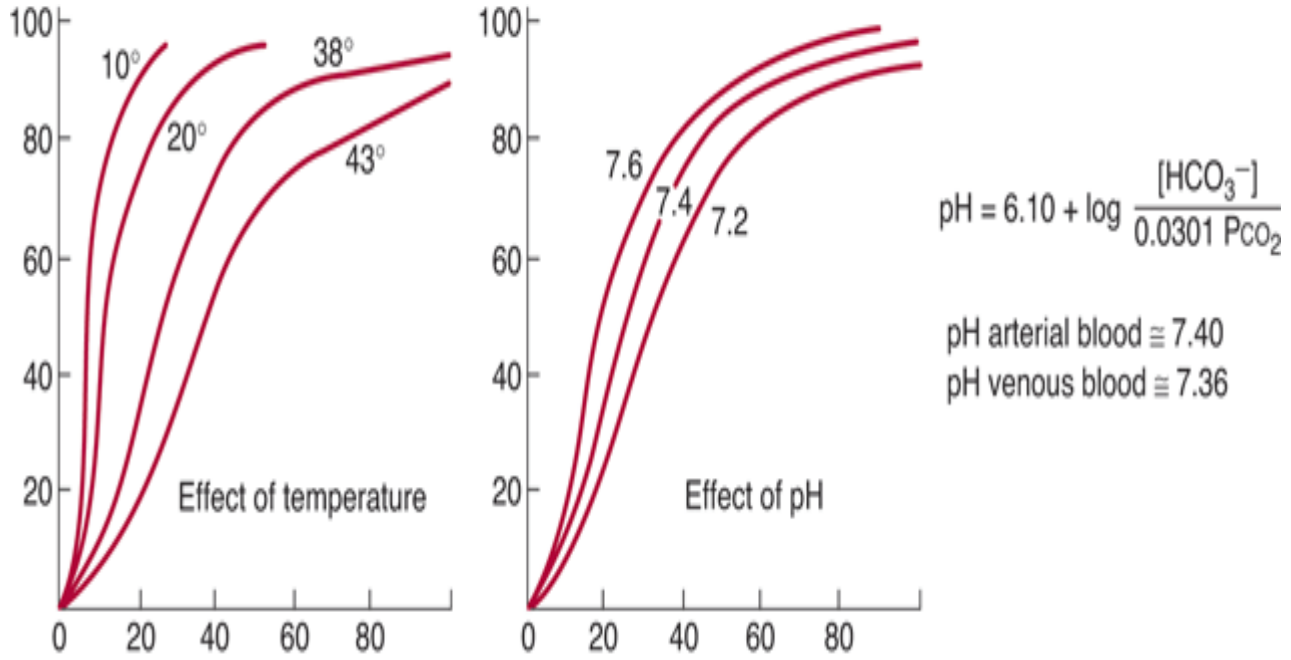

Fig. 36-3 Accessed 02/01/2010

Carbon dioxide dissociation curve

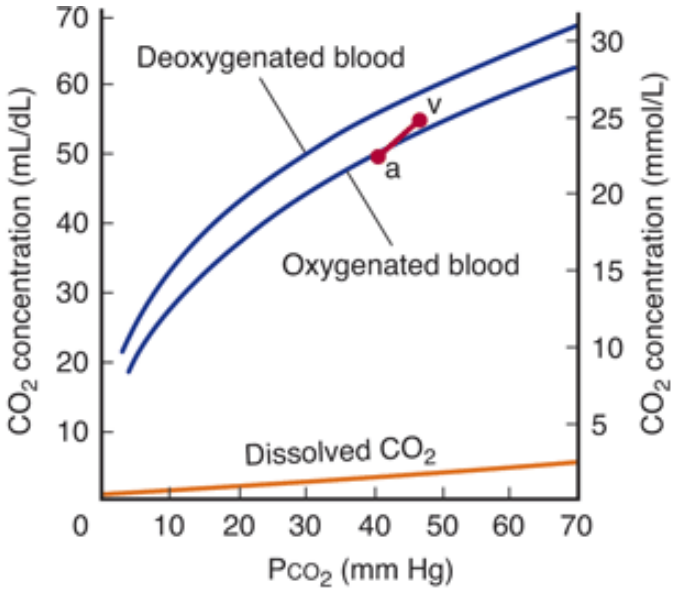

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Fig. 36-7 Accessed 02/01/2010

The arterial point (a) and the venous point (v) indicate the total CO_2 content found in arterial blood and venous blood of normal resting humans. Note the low amount of CO_2 that is dissolved (orange trace) compared to that which can be carried by other means.

pH

- Acid-base balance maintained by
- (1) pulmonary excretion of CO_2
- (2) renal excretion of HCO_3^- and non volatile acids
- Strong Nonvolatile acids are principally sulfuric acid resulting from metabolism of sulfur containing amino acids
- This is an open system.
- H^+ is combined with ammonia or phosphates in the kidney for excretion.
- Ammonia is the principal adaptive response.
- Ammonia production from glutamine metabolism can be increased in the presence of an acid load.

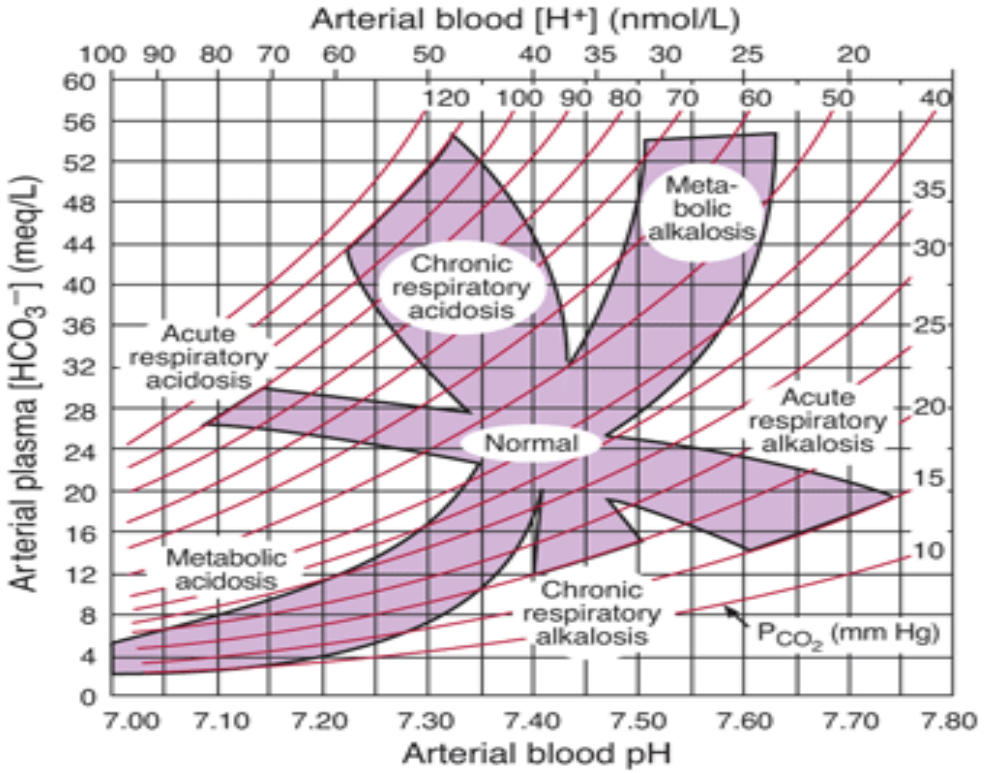
pH

- H^+ (and K^+ under aldosterone control) is excreted into the urine in the distal tubule in exchange for Na^+.
- HCO_3^- formed in the tubule cell combines with Na^+ and enters the circulation.
- Whatever increases intracellular H^+ or K^+ also increases their secretion into the distal tubular lumen and increases Na^+ reabsorption.
- Weak nonvolatile acids are largely protein, principally albumin.

pH

- Hemoglobin is the buffering agent against CO_2 in the blood.
- Carbonic anhydrase in the red cell facilitates movement of CO_2 in and out of the cell.
- O_2 binds to hemoglobin in the alveolar capillaries.
- This leads to H^+ release.
- H^+ combines with HCO_3^- in the plasma, permitting CO_2 to be released into alveoli.
- HCO_3^- is the moment-to-moment buffer that maintains pH in a narrow range.
- Phosphate and protein are the chief intracellular buffers.
- pH rises with hypoproteinemia.

Acid-base nomogram



Changes in the PCO_2 (curved lines), plasma HCO_3^-, and pH (or $[H^+]$) of arterial blood in respiratory and metabolic acidosis are shown.

(Reproduced with permission from Cogan MG, Rector FC Jr.: Acid–base disorders. In: *The Kidney,* 4th ed. Brenner BM, Rector FC Jr. [editors]. Saunders, 1991.)

Fig. 36-10 Accessed 02/01/2010

pH and the anion gap

- Henderson-Hasselbach equation:
  $pH = 6.1 + \log (HCO_3^-)/.03pCO_2$
- 1meq HCO_3^- is equivalent to 1.2 mmHg pCO_2
- Low pH likely a result of metabolic abnormality.
- High pH likely due to decreased renal perfusion.
- Hypokalemia also promotes HCO_3^- absorption.
- Vomiting, nasogastric suction, diuretic use as other causes.

PLASMA ANION GAP = Na – (Cl + HCO3).

URINE ANION GAP = (Na + K) – (Cl + 13).

pH

- pH< 7.20
- Diminished contractility
- Increased arrhythmia risk
- Vasodilation
- Hyperventilation
- K^+ and insulin increase

pH

- pH> 7.60
- Arteriolar vasoconstriction
- Decreased coronary blood flow
- Increased arrhythmia risk
- Hypoventilation
- Decreased K^{+}
- Increased Ca^{2+} and Mg^{2+}

Diminished plasma anion gap

- Hypoalbuminemia
- Hypercalcemia
- Cationic immunoglobulins (as in myeloma)
- Lithium drug use
- Dilution (hypertriglyceridemia)

Diminished urine anion gap

- The urine anion gap is a rough measure of ammonium production.
- In the hyperchloremic acidotic patient,
  if (Na + K) < Cl , interstitial renal disease is likely.

Normal plasma anion gap

- A normal anion gap does not exclude lactic acidosis.
- Renal tubular acidosis (type IV)
- Characterized by diminished renin and aldosterone
- Hyperkalemia
- Acidosis
- Impaired ammonia production

Elevated plasma anion gap

- Ethylene glycol usual cause of large anion gap
- Other causes:
- Elevated albumin (1g/dL accounts for 2.5meq/L anion gap)
- Low Calcium, low Magnesium
- Aspirin
- Methyl alcohol
- Increased acid generation:
- Lactate, ketoacids.
- Serum lactate level more sensitive indicator of lactic acidosis than is an increase in the anion gap.

Elevated plasma anion gap

- HCO_3^- loss:
- Diarrhea
- mEq for mEq exchange of Cl^- for HCO_3^-
- Na^+ and Cl^- are retained by the kidney to maintain circulating volume
- Proximal renal tubular acidosis (Type I)
- Carbonic anhydrase inhibitor
- Ethylene glycol usual cause of large anion gap

Lactic acidosis

- Endogenous lactate is L-lactic acid.
- That is what the assay measures.
- Serum lactate level more sensitive indicator than is an increase in the anion gap.
- A normal anion gap does not exclude lactic acidosis.
- D-lactic acidosis may be seen with carbohydrate malabsorption where it is metabolized to D-lactic acid and is absorbed.
- Serum lactate levels are normal as only the L-form is measured.

Lactic acidosis

- Endogenous lactate is L-lactic acid.
- That is what the assay measures.
- Cuases:
- Hypoxemia
- Including CO interference with O_2 binding
- Including HCN- (cyanide) binding to hemoglobin Fe^{2+}
- Shock
- Tissue necrosis
- Infarcted bowel or muscle