Chapter 2
The Non-Invasive Monitoring of Blood Oxygen and Carbon Dioxide Levels

2

Contents

A. Hasan, *Handbook of Blood Gas/Acid-Base Interpretation*,
DOI 10.1007/978-1-4471-4315-4_2, © Springer-Verlag London 2013

2

2.1 The Structure and Function of Haemoglobin

The special ability of hemoglobin (Hb) to imbibe O_2 from the pulmonary capillaries and release it to the tissues derives from its unique quartenary structure.

Structure

Globin

The Hb molecule consists of four globin chains (two alpha chains, each of 141 amino acids; two beta chains each of 146 amino acids.

Heme

One heme group binds *each* globin chain. *Each heme group consists of:*

One ferrous ion (Fe++)

In order to carry O_2, it is necessary for heme's ferrous iron to remain in the ferrous state.

One protoporphyrin IX ring

This protoporphyrin ring is covalently bound to the ferrous ion.

FUNCTION: O_2 carriage is the most important function of Hb (Sect. 2.4), but Hb serves several other important functions as well:

CO_2 carriage

Although only about 5 % of all the CO_2 transported in the blood is in the form of carbamino compounds (viz, bound to Hb, the latter account for 30 % of the CO_2 that evolves in the lungs from the red blood cells circulating within the pulmonary capliiaries. Another 5 % of CO_2 is carried dissolved in plasma. The bulk of the CO_2, however, is carried in the form of bicarbonate.

Regulation of vasomotor tone

Nitric oxide (NO) is capable of reacting with a cysteine residue at position 93 of the ß chain of Hb. The resulting nitrosothiol, S-nitrosylated Hb is a vasodilator. The unique and recently recognized vasodilator property of Hb is dependent on its complex and ill-understood reactions with NO.

Bunn HF, Forget BG. Hemoglobin: molecular, genetic and clinical aspects. Philadelphia: WB Saunders; 1986.

McMahon TJ, Moon RE, Luschinger BP, Carraway MS, Stone AE, Stolp BW, Gow AJ, Pawloski JR, Watke P, Singel DJ, et al. Nitric oxide in the human respiratory cycle. Nat Med. 2002;8:711–7.

Perutz MF. Molecular anatomy, physiology, and pathology of hemoglobin. In: Stamatoyannopoulos G, Nienhuis AW, et al., editors. The molecular basis of blood disorders. Philadelphia: WB Saunders; 1987.

Stamler JS, Jia L, Eu JP, McMahon TJ, Demchenko IT, Bonaventura J, Gernert K, Piantadosi CA. Blood flow regulation by S-nitrosohemoglobin in the physiological oxygen gradient. Science. 1997;276:2034–7.

2

2.2 Co-operativity

Deoxygenated Hemoglobin

Deoxygenated hemoglobin exists in a tense (taut) configuration because of electrostatic bonds between its beta globin chains. The hemoglobin molecule has helical twists. In the non-helical sections the polypeptide chain folds upon itself, creating clefts within which the four heme groups lie at equidistant intervals.

The attachment of the first O_2 molecule

In its taut state, deoxygenated hemoglobin has little affinity for O_2. The attachment of the first O_2 molecule to one of the globin chains generates chemical and mechanical stresses resulting in the severing of electrostatic bonds.This allows the hemoglobin molecule to unfold slightly.

The attachment of the second O_2 molecule

As the hemoglobin molecule relaxes and unfolds it exposes the other O_2 binding sites within its clefts; this facilitates the addition of another molecule of O_2 to the hemoglobin, more rapidly than the first.

The attachment of the third and fourth O_2 molecules

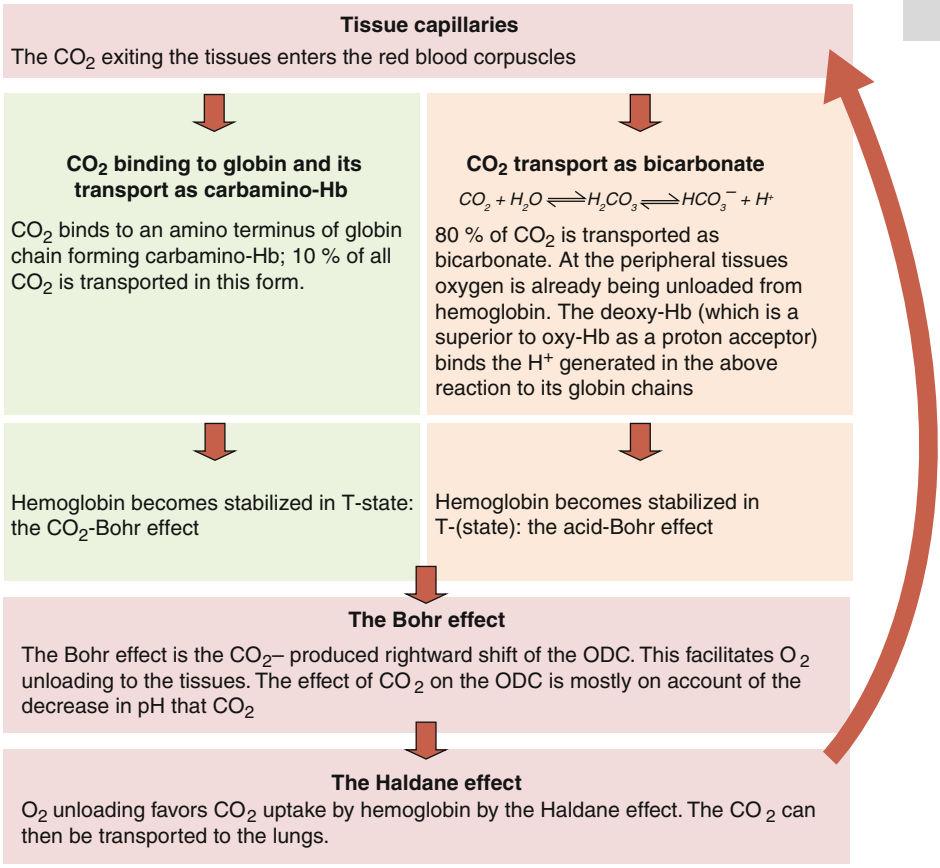
The binding of the second molecule of O_2 results in further relaxation of the coils of the hemoglobin molecule, accelerating the uptake of the third and the fourth O_2 molecules.

The co-operativity among its binding sites that results in the accelerated uptake of O_2 gives the oxy-hemoglobin dissociation curve its characteristic sigmoid shape.

Bunn HF, Forget BG. Hemoglobin: molecular, genetic and clinical aspects. Philadelphia: WB Saunders; 1986.

Perutz MF. Molecular anatomy, physiology, and pathology of hemoglobin. In: Stamatoyannopoulos G, Nienhuis AW, et al. editors. The molecular basis of blood disorders. Philadelphia: WB Saunders; 1987.

2.3 The Bohr Effect and the Haldane Effect



Bohr C, Hasselbalch K, Krogh A. Ueber einen in biologischer Beziehung wichtigen Einfluss. den die Kohlen- sauerespannung des Blutes auf dessen Sauerstoffbinding ubt. Skand Arch Physiol. 1904;16:402.

Klocke RA. Mechanism and kinetics of the Haldane effect in human erythrocytes. J Appl Physiol. 1973;35:673–81.

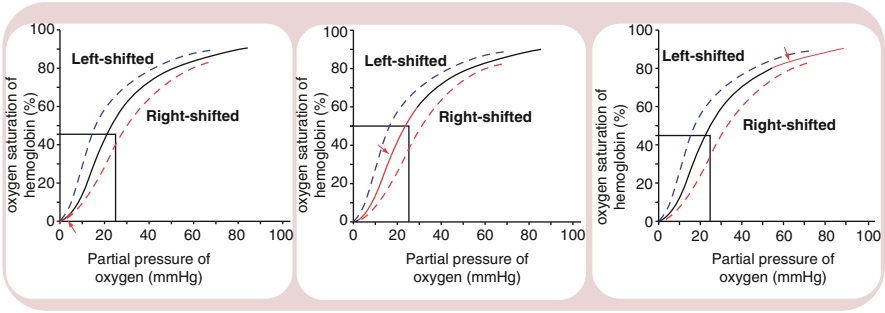
2

2.4 Oxygenated and Non-oxygenated Hemoglobin

Oxygenated Hb (syn: OxyHb)	Each Hb molecule has four heme sites to each of which an O_2 molecule can bind. *The percentage of O_2 binding heme sites that bind to O_2* is the O_2 saturation (SpO_2) of the blood. In other words SpO_2 is the number of heme sites occupied by O_2 of every 100 heme sites.	The SpO_2 (as read out on the pulse oximeter (represents) the Oxy-Hb
Non oxygenated Hb	The percentage of heme sites that are not bound to O_2 molecules. Non-oxygenated Hb includes:	**Dexoxy-Hb (syn:reduced) Hb** The percentage of heme groups that are not bound to O_2. Reduced Hb % = 100 % – [SPO_2 + MetHb + COHb] %* **Carboxy-Hb** The percentage of heme groups in the form of Carboxy-Hb **Met-Hb** The percentage of heme groups in the form of Met-Hb

*See Co-oximetry (Sect. 2.10)

2.5 PaO_2 and the Oxy-hemoglobin Dissociation Curve



PaO_2 below ~ 20 mmHg

Below a PaO_2 of 20 mmHg, the Oxy-hemoglobin dissociation curve is almost flat; until the PaO_2 reaches 20 mmHg, any increase in PaO_2 that occurs on this segment does not translate into an increase in SpO_2.

Between ~ 20 and 60 mmHg

Once the PaO_2 rises to about 20 mmHg (at a PaO_2 of between20 mmHg and 60 mmHg), a relatively small increase in PaO_2 results in a dramatic rise in SpO_2.

Above ~ 60 mmHg

Above a PaO_2 of 60 mmHg, the oxy-hemoglobin dissociation curve is again flat, and the SpO_2 is in the high 90s; from here on, any further rise in SpO_2 will result in little further rise in SpO_2.

The following co-ordination points should be expected for an ODC that lies in its normal position:

PaO_2 40 mmHg*	PaO_2 of 70 mmHg	PaO_2 of 100 mmHg
Corresponds to SpO_2 of 75 %	Corresponds to SpO_2 of 92 %	Corresponds to SpO_2 of 97 %

(*As in mixed venous blood)

Canham EM, Beuther DA. Interpreting arterial blood gases. Chest. 2007. PCCSU Article. 2 Jan 2007.

2

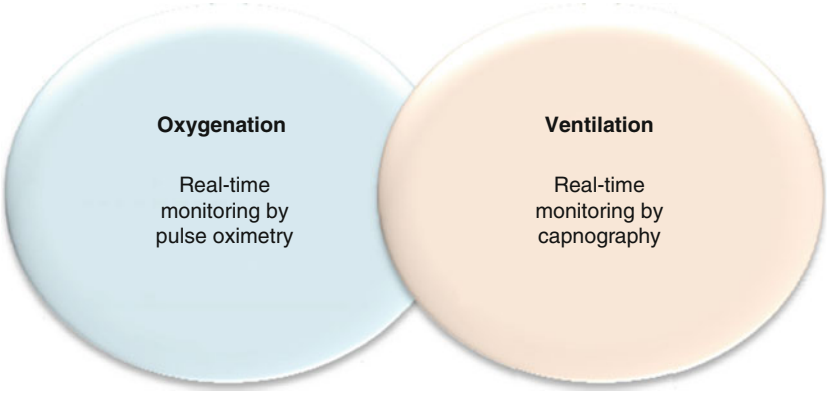
2.6 Monitoring of Blood Gases

2.6.1 *Invasive O_2 Monitoring*

Although direct measurement of arterial O_2 tension by arterial blood gas (ABG) sampling is a very accurate way of assessing oxygenation, it has its disadvantages.

Intermittent ABG sampling	Continous ABG sampling
Inconvenient Painful Blending Infection Arterial thrombosis Rarely, gangrene of extremity	Obviates the need for frequent arterial Punctures Generally used unstable clinical Situations where real time monitoring is required. *Can also produce the complications* that intermittent sampling is associated with.

2.6.2 *The Non-invasive Monitoring of Blood Gases*



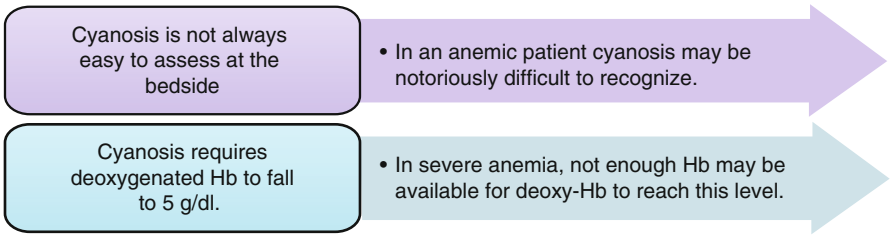
Though it offers a convenient way of monitoring oxygenation in real time, pulse oximetry has its own disadvantages (Sect. 2.21).

Bongard F, Sue D. Pulse oximetry and capnography in intensive and transitional care units. West J Med. 1992;156:57.

Pierson DJ. Pulse oximetry versus arterial blood gas specimens in long-term oxygen therapy. Lung. 1990;168 Suppl:782.

2.7 Principles of Pulse Oximetry

Cyanosis is the *clinical* hallmark of hemoglobin desaturation.

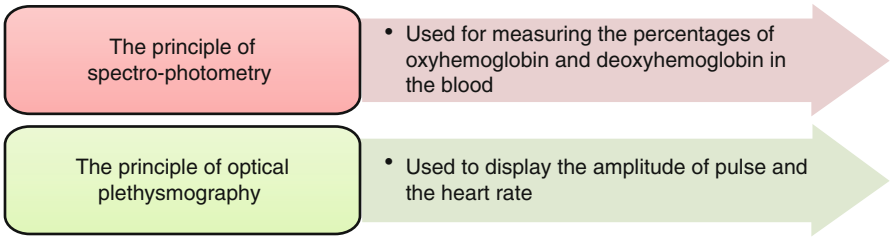


Severe hypoxia can therefore manifest without apparent cyanosis.

Pulse oximetry, which does not suffer from these limitations, has been described as "…the greatest advance in patient monitoring since electrography" and is today regarded as the "fifth vital sign".

Older oximeters calculated the SpO_2 from the values of PaO_2 and pH, deriving values through nomograms and the Severinghaus slide rule.

Modern pulse oximetry is based upon two fundamental principles:



Comroe JH Jr, Botelho S. The unreliability of cyanosis in the recognition of arterial hpoxemia. Am J Med Sci. 1947;214:1.

Hanning CD, Alexander-Williams JM. Fortnightly review: pulse oximetry: a practical review. BMJ. 1995;311:367–70.

Neff TA. Routine oximetry: a fifth vital sign? Chest. 1988;94:227.

2.8 Spectrophotometry

The principle of spectrophotometry is based upon on the *Beer-Lambert law* which states that "*…the concentration of light-absorbing species within a sample is a logarithmic function of the amount of light absorbed by that sample*."

In respect of blood, the light-absorbing species are oxy-hemoglobin and deoxy-hemoglobin.

Two photodiodes emit light phasically at several hundred times per second, one at 660 nm (in the red band of the spectrum) and the other at 940 nm (in the infra-red band of the spectrum).

Oxyhemoglobin

Light emitted at 660 nm is better absorbed by saturated (oxygenated) haemoglobin. *Oxygenated (red blood) absorbs light maximally in the infrared band.*

Deoxyhemoglobin

Light emitted at 940 nm is better absorbed by reduced (deoxygenated) haemoglobin. *Deoxygenated (blue blood) absorbs light maximally in the red band.*

By making the diodes blink rapidly, it is possible to make about 600 measurements each second. The relative amount of light transmitted through the interposed tissue at these two wavelengths is compared to an algorithm of oxygen saturation derived from healthy human volunteers, and a microprocessor computes the patient's SpO_2 based on this.

The phasic emission of light differentiates the light absorbance of the arterial blood from that of the light absorbance of venous blood and the surrounding tissue.

Hanning CD, Alexander-Williams JM. Fortnightly review: pulse oximetry: a practical review. BMJ. 1995;311:367–70.

Jubran A. Pulse oximetry. Intensive Care Med. 2004;30:2017–20.

Mendelson Y. Pulse oximetry: theory and applications for noninvasive monitoring. Clin Chem. 1992;38:1601.

2.9 Optical Plethysmography

Light transmitted through tissue is absorbed by *static elements* (muscle, bone, venous blood, and the static components of arterial blood), as well as the *pulse added volume* of arterial blood. The pulsatile arterial signal typically comprises 0.5–5 % of the total transmitted light.

The principle of optical plethysmography is made use of to display the amplitude of pulse and the heart rate. Each peak of the arterial waveform corresponds to one cardiac cycle. Occasionally a smaller secondary peak due to the venous pressure pulse can be distinguished. The phasic signal presented to the sensor, calculates the pulse amplitude according to the relative light absorbencies during systole and diastole.

Ventricular systole	Ventricular diastole
Phasic increase of blood volume in perfused organs.	Phasic decrease of blood volume in perfused organs.
Light has to travel a longer distance through the *distended* subcutaneous tissue (of the finger or ear lobe).	Light has to travel a shorter distance through the contracted subcutaneous tissue (of the finger or ear lobe).
Light transmission through the sampling site decreases.	Light transmission through the sampling site increases.

This difference is made use of to generate a waveform which is displayed on the monitor.

Mendelson Y. Pulse oximetry: theory and applications for noninvasive monitoring. Clin Chem. 1992;38:1601.

2

2.10 Types of Pulse Oximeters

Transmission pulse oximeters	Reflectance pulse oximeters
The conventional oximeters. A pair of light emitting diodes (LEDs) emit light through 5 to 10 mm of interposed tissue (typically a finger,toe or an earlobe,but also the bridge of nose, nares, cheek, tongue).	Photowaves from LEDs are bounced off an appropriate surface (e.g. the skull bone). This promising technology, once improved, should address several of the drawbacks in current oximeters
The change in light frequency is read out by a photodetector placed on the *opposite side* of the interposed tissue.	The reflected beam of light passes back through the tissue, (e.g. the skin of the forehead), to reach a photodetector placed *adjacent* to the LEDs.

With advancements in technology, pulse oximeters have become less expensive, smaller, lighter, and more robust. Improved algorithms now ensure fewer artefacts in measurement.

CO-oximetry

Standard pulse oximetry cannot differentiate carboxyhemoglobin from oxyhemoglobin. CO-oximeters measure absorption at several wavelengths and are primarily used to monitor SpO_2 in carboxy-hemoglobinemia (CO poisoning), and methemoglobinemia. CO-oximeters require blood sampling, though newer oximeters that pulse light at eight wavelengths are now available that reliably measure carboxyhemoglobin and methemoglobin.

Barker SJ, Curry J, Redford D, et al. Measurement of carboxyhemoglobin and methemoglobin by pulse oximetry: a human volunteer study. Anesthesiology. 2006;105:892–7.

Marr J, Abramo TJ. Monitoring in critically ill children. In: Baren JM, Rothrock SG, Brennan JA, Brown L, editors. Pediatric emergency medicine. Philadelphia: Saunders Elsevier; 2008. p. 50–2.

Tallon RW. Oximetry: state-of-the-art. Nurs Manage. 1996;27:43.

2.11 Pulse Oximetry and PaO_2

One of the main disadvantages of oximetry is that it monitors oxygen saturation (SpO_2) and not the PaO_2.

SpO_2 can miss a drop in PaO_2.

Major changes in PaO_2 on the flat upper segment of the ODC can occur without appreciable changes in SpO_2.

SpO_2 is unreliable in severe hypoxemia.

Below an SpO_2 of 80 %, oximetry is not dependable

SpO_2 can be influenced by a shift of the oxy-hemoglobin dissociation curve.	*Leftward shift of the* oxy-hemoglobin dissociation curve (as in alkalemia or hypothermia).	Hemoglobin is more saturated relative to the PaO_2. SpO_2 can overestimate the PaO_2.
	Rightward shift of the oxy-hemoglobin dissociation curve (as in acidemia or fever).	Hemoglobin is less saturated relative to the *PaO_2. SpO_2 can underestimate the PaO_2.*

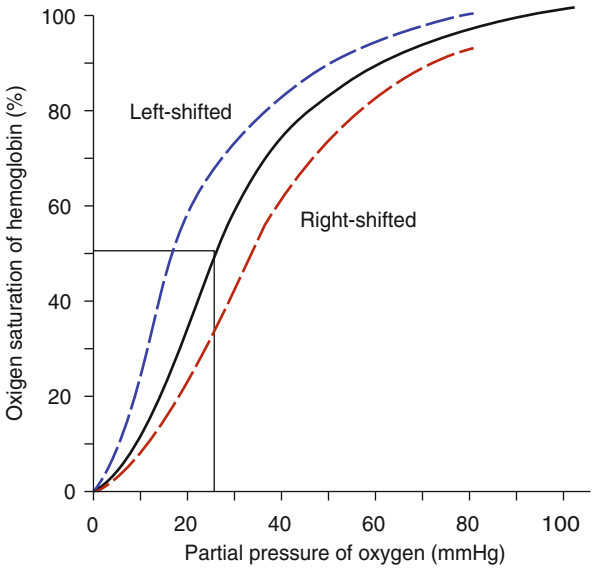
Lastly, stating the obvious, oximetry measures oxygenation but gives no information about *ventilation*; for the latter, capnography or $PaCO_2$ measurements (by arterial blood gas sampling) are required.

Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry. III: Effects of interference, dyes, dyshaemoglobins and other pigments. Anaesthesia 1991;46:291–295.

Stoneham MD. Uses and limitations of pulse oximetry. Br J Hosp Med. 1995;54:35.

2

2.12 P_{50}



P_{50}

The position of the oxy-Hb dissociation curve (ODC) can be assessed from the P_{50}, which is the PaO_2 at which the Hb is 50 % saturated.

The normal P_{50} is 26.6 mmHg

P_{50} < 26.6 mmHg

A lower than normal P_{50} means a leftward shifted ODC.

P_{50} > 26.6 mmHg

A higher than normal P_{50} means a rightward shifted ODC.

2.13 Shifts in the Oxy-hemoglobin Dissociation Curve

Leftward shift of the ODC occurs in the following conditions:	**Rightward shift of the ODC occurs in the following conditions:**
Alkalemia Hypothermia Abnormal hemoglobins, e.g: *Carboxy-hemoglobin* *Met-hemoglobin* *Fetal hemoglobin* Myxedema Low inorganic phosphates Acute pancreatitis*	Acidemia Fever Abnormal hemoglobins, e.g.: *Hb Kansas* Thyrotoxicosis Raised inorganic phosphate Anemia Steroid therapy
Implications of a leftward shifted ODC:	**Implications of a rightward shifted ODC:**
Within the blood: Tighter binding of O_2 to Hb. *At the peripheral tissues:* With a left shifted ODC, SpO_2 is higher, but less of the O_2 (which is tightly bound to the Hb) is released to the tissues.	*Within the blood:* "Looser" binding of O_2 to Hb. *At the peripheral tissues:* With a right shifted ODC, although the SpO_2 is lower, more of the O_2 (which is relatively loosely bound to the Hb) is released to the tissues.
The PaO_2 is low relative to the SpO_2.	**The PaO_2 is high relative to the SpO_2.**
With a left-shifted ODC the SpO_2 may be falsely reassuring, and the PaO_2 may be lower than expected.	*The right shifted ODC facilitates oxygen delivery to the peripheraltissues.*
SpO_2 overestimates the oxygenation (i.e. PaO_2).	***SpO_2 underestimates the PaO_2.***

*Linolenic acid, Linoleic acid and Oleic acid, the fatty acids released into the circulation as a result of pancreatic cell destruction, bind to hemoglobin and increase its affinity for O_2.

Greenberg AG, Terlizzi L, Peskin G. Oxyhemoglobin affinity in acute pancreatitis. J Surg Res. 1977;22:561–5.

2

2.14 Oxygen Saturation (SpO_2) in Anemia and Skin Pigmentation

Anemia unless it is very severe (Hb < 5 g/dl) does not influence the SpO_2.

Since SpO_2 is expressed as a percentage of the available binding sites for O_2, anemia can critically affect the O_2 content of the blood (CaO_2), but has virtually no impact on SpO_2.

The amount of Hb in the blood determines the O_2 content of the blood, not the SpO_2.

The colour of the interposed tissue can influence the SpO_2.

Skin pigmentation	Hyper-bilirubin-emia	Nail polish	
Minor and inconsistent effect on SpO_2. However some studies have shown that racial pigmentation may cause as much as 4 % difference in measured SpO_2.	Minimal effect on SpO_2.	**Red nail polish** May have a trivial effect on SpO_2.	**Other shades of nail polish** May produce a spurious fall in SpO_2 of as much as 3–6 %.

Schnapp LM, Cohen NH. Pulse oximetry: uses and abuses. Chest. 1990;98:1244.

2.15 Oxygen Saturation (SpO_2) in Abnormal Forms of Hemoglobin

Abnormal forms of hemoglobin can have very different absorption spectra, and oximetric readings (SpO_2) can overestimate the true oxygen saturation of Hb (see Sect. 2.20)

CO-Hb* *SpO_2 overestimates SaO_2*	CO-Hb has an almost identical absorption spectrum (660 ηm) to oxy-Hb.	Because the oximeter interprets CO-Hb as normal Hb, normal SpO_2 can be displayed even in severe hypoxia. Diagnosis is by CO-oximetry (Sect. 2.19).
Met-Hb** *SpO_2 overestimates SaO_2*	Met-Hb absorbs light at both wavelengths 660 ηm and 940 ηm) that standard oximeters emit. Because of this property, Met-Hb has a complex effect on SpO_2.	At low levels of Met-Hb, SaO_2 overestimates the SpO_2. When Met-Hb levels increase to over 30 %, SpO_2 tends to drift towards 85 %, which is a gross overestimation of SaO_2. Presumptive diagnosis is by CO-oximetry; it is confirmed by the Evelyn-Malloy method (Sect. 2.26 and 2.27).
Hb-S*** *Variably affects SpO_2*	Hb-S has a similar absorption spectrum to oxy-Hb.	Hb-S can lead to spuriously high or low SpO_2 values.
Fetal Hb	Has no special impact on SpO_2	

*Carboxy-hemoglobin **Met-hemoglobin *** in Sickle-cell disease

Barker SJ, Curry J, Redford D, et al. Measurement of carboxyhemoglobin and methemoglobin by pulse oximetry: a human volunteer study. Anesthesiology. 2006;105:892–7.

Eisenkraft JI, Pulse oximeter desaturation due to methemoglobinemia. Anesthesiology. 1988;68:279.

Ernst A, Zibrak JD. Carbon monoxide poisoning. New Engl J Med. 1998;339:1603–8.

Evelyn K, Malloy H. Microdetermination of oxyhemoglobin, methemoglobin, and sulfhemoglobin in a single sample of blood. J Biol Chem. 1938;126:655.

Ortiz FO, Aldrich TK, Nagel RL, Benjamin LJ. Accuracy of pulse oximetry in sickle cell disease. Am J Respir Crit Care Med. 1999;159:447.

2.16 Mechanisms of Hypoxemia in Methemoglobinemia

Normal hemoglobin	Methemoglobin
Normal hemoglobin carries its iron as ferrous ions. Hb is capable of binding O_2 provided the ferrous iron remains in its reduced state. The special configuration of the hemoglobin chains appears to protect the ferrous ions from oxidation to the ferric state. **Pulmonary capillaries** In the pulmonary capillaries each ferrous iron moiety binds an O_2 atom, in the process briefly donating an electron to the latter. **Tissue capillaries** At the tissue capillary level the O_2 atom cleaves away from the Hb molecule, in the process reacquiring its electron. The reduction of the iron back to its ferrous form makes it free to bind and transport O_2 again.	**Met-Hb carries its iron as ferric ions.** Met-Hb, as opposed to deoxy-Hb carries its iron in the ferric form, in which state it is unable to bind O_2. That amount of Hb that exists as Met-Hb cannot participate in O_2 transport. Also, the ferrous iron that is present in the adjacent hemoglobin chains binds more strongly to O_2 than usual. The oxygen dissociation curve is shifted to the left leaving little O_2 for the tissues.

Methemoglobin has peak absorbance at 631 ηm. CO-oximeters use a fixed wavelength to screen for methemoglobin: all readings in the 630 ηm range are reported as methemoglobin. Several pigments (including sulfhemoglobin and methylene blue) can evoke false positive results.

Curry S. Methemoglobinemia. Ann Emerg Med. 1982;11:214–21.

Wright RO, Lewander WJ, Woolf AD. Methemoglobinemia: etiology, pharmacology, and clinical management. Ann Emerg Med. 1999;34:646–56.

2.17 Methemoglobinemias: Classification

Methemoglobinemias can be classified into the hereditary methemoglobinemias and the acquired methemoglobinemias.

The acquired methemoglobinemias are due to extrinsic agents, *which result in increased formation of Met-Hb.*	In the **hereditary methemoglobinemias,** faulty pathways result in *decreased reduction of Met-Hb, which consequently accumulates.*	
e.g.: • p-Amino salicylic acid • Aniline dyes • Benzene derivatives • Clofazimine • Chlorates • Chloroquine • Dapsone • Local anesthetic agents • Metoclopramide • Nitrites (eg Amyl nitrite, Nitroglycerin) • Nitric oxide • Phenacetin • Primaquine • Sulfonamides	**Cytochrome b5 reductase deficiency:** Normally about 0.5–3 % of Hb is converted to Met-Hb daily by auto-oxidation. Some of this Met-Hb gets reduced back Hb (by a NADH dependent, cytochrome b5 reductase catalysed reaction 0. As a result, Met-Hb comprises about 1 % of total Hb in the blood. Cytochrome b5 reductase deficiency can result in increased methemoglobin levels.	**Hemoglobin-M disease:** As a result of a mutation in the alpha or beta globin chain, tyrosine replaces one of the histidine residues. Ferric phenolate complex formed: Fe^{+++} cannot be effectively reduced to the ferrous state. Persistent lifelong methemoglobinemia occurs.

Curry S. Methemoglobinemia. Ann Emerg Med. 1982;11:214–21.

Jaffe ER. Enzymopenic hereditary methemoglobinemia: a clinical/biochemical classification. Blood Cells. 1986;12:81–90.

Prchal JT. Clinical features, diagnosis and treatment of methemoglobinemia. In: Basow DS, editors. UpToDate. Waltham: UpToDate; 2012. Last updated 22 Mar 2012. Last accessed 13 May 2012.

2.18 Sulfhemoglobinemia

STEP 1: Oxidation of Hb to methemoglobin

First, the oxidation of the ferrous to ferric iron results in the formation of methemoglobin.

STEP 2: Formation of sulfhemoglobin

Next, the exposure to specific agents results in covalent binding of the sulfur atom to heme, resulting in the formation of sulfhemoglobin.

Similarities with methemoglobinemia

Sulfhemoglobin, like methemoglobin, can neither transport O_2 nor CO_2.

Differences with methemoglobinemia

Right shift of ODC

Unlike Met-Hb, Sulf-Hb causes a right shift of ODC and so relatively more oxygen is released to the tissues.

Severity of hypoxia

Hypoxia in sulfhemoglobinemia is not as severe as that in methemoglobinemia.

Sulfhemoglobinemia is irreversible

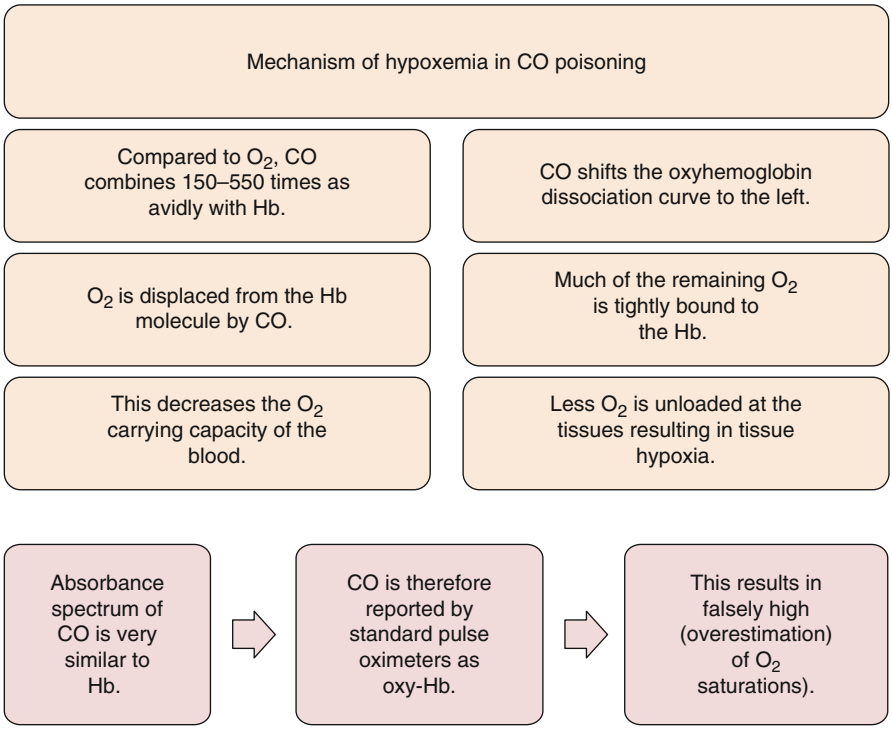
Unlike methemoglobinemia, sulfhemoglobinemia is irreversible.

Oximeters that measure Met-Hb can erroneously read Sulf-Hb as Met-Hb

Park CM, Nagel RL. Sulfhemoglobinemia. N Engl J Med. 1984;310:1579–84.

2.19 Carbon Monoxide (CO) Poisoning

The incomplete combustion of hydrocarbons leads to the formation of CO, a colourless, odourless gas. Normally, the levels of carboxy-hemoglobin (CO-Hb) are <3 % of total Hb in the urban population; smokers have a CO-Hb level of 5–10 % of total Hb in their blood. At levels above 50 %, CO-Hb is capable of causing death.



CO-oximetry (multi-wavelength spectrophotometry that separately measures CO-Hb, Oxy-Hb, and reduced Hb) reliably measures CO levels and should be used when CO poisoning is suspected (Sect. 2.10).

Caughey WS. Carbon monoxide bonding in hemeproteins. Ann N Y Acad Sci. 1970;174:148.
Weaver LK. Carbon monoxide poisoning. Crit Care Clin. 1999;15:297.

2.20 Saturation Gap

The O_2 analyses by the pulse oximeter and the ABG are based upon the premise that only two forms of Hb are possible: Oxyhemoglobin and deoxyhemoglobin; and that no abnormal forms of hemoglobin are present.

SpO_2 *(low)*	***SaO_2*** *(normal)*
The Hb saturation of O_2 as measured by pulse oximetry.	The Hb saturation of O_2 as calculated by the ABG machine.
The pulse oximeter measures light absorbance at two wavelengths (2.08). With significant levels of methemoglobin in the blood, the SpO_2 drifts towards 85 % (2.16).	The ABG machine first measures the PaO_2 and then calculates the expected SaO_2 from this, based on the position of the oxyhemoglobin dissociation curve. In the absence of cardiopulmonary disease, the SaO_2 (not being dependent on oxy-Hb concentration) will be normal even though abnormal hemoglobins be present.

The saturation gap

When the difference in SaO_2 and SpO_2 is >5 %, a saturation gap is said to exist.
A saturation gap is a clue to significant levels of certain abnormal hemoglobins in the blood (Sect. 2.15, 2.16, 2.17, 2.18 and 2.19).

Eisenkraft JI. Pulse oximeter desaturation due to methemoglobinemia. Anesthesiology. 1988;68:279.

Haymond S, Cariappa R, Eby CS, Scott MG. Laboratory assessment of oxygenation in methemoglobinemia. Clin Chem. 2005;51(2):434–44.

Mokhlesi B, Leiken JB, Murray P, Corbridge TC. Adult toxicology in critical care, part I: general approach to the intoxicated patient. Chest. 2003;123:577–92.

Oesenberg B. Pulse oximetry in methaemoglobinemia. Anaesthesia. 1990;45:56.

2.21 Sources of Error While Measuring SpO_2

Time lag	*Output stabilization:* There is often a time lag between a change in O_2 saturation and its detection by the oximeter. The signal averaging by the oximeter may take several seconds. This can be disadvantageous in a rapidly changing clinical situation. Modern pulse oximeters take less than a minute for output stabilisation to occur. Subsequent SpO_2 changes usually take less than ten seconds to register. Response time: toe>finger>earlobe.
Weak signal	*Hypoperfusion of the interposed part: SpO_2 falsely low* Vasoconstriction BP < 80 mmHg Inflation of a BP cuff Edema of an extremity
	Noise amplification: When the pulse is weak, the pulse oximeter boosts its amplitude. In doing so it may amplify the background noise and lead to errors. Most current devices warn of weak pulse strength may simply not display the saturation.
Proximity to instruments	MRI scanners Cell-phones Electrical interference Power outlets and cords, cardiac monitors, cautery devices etc.
Motion artifact	Shivering Convulsions Movement
Arrythmias	Irregular rhythms such as atrial fibrillation can unpredictably affect displayed values.
Optical issues	*Optical shunt: underestimation of the SpO_2* Light from the photodiode reaches the photodetector without passing through the interposed part (penumbra effect). A calculated SpO_2 (usually in the low eighties) will result in underestimation of the actual SpO_2. *Light interference: underestimation of the SpO_2* Light interference may occur by extraneous light directly impinging on the photodetector especially if the probe is too large or improperly placed. Ambient light, direct sunlight, fluorescent, infrared, and xenon lamps may cause interference. The calculated SpO_2 tends towards 85 % and is therefore underestimated. Execptionally (strong ambient light, completely displaced probe), the SpO_2 may be falsely high.

To avoid errors, the amplitude of the pulse waveform should be routinely checked. In the presence of a satisfactory waveform with an observable dicrotic notch, the SpO_2 readings are likely to be correct. A close agreement between the displayed pulse rate on the oximeter and the manually counted pulse rate suggests that the SpO_2 reading is likely to be correct. When the pulse signal is strong, pulse oximeters are accurate provided saturations range above 80 %. At lower saturations however, they lose some of their reliability. When the stroke output fluctuates synchronously with the respiratory cycle (such as in a ventilated patient who develops auto-PEEP), the tracing will oscillate noticeably about the baseline.

Rarely, pulse oximetry has been associated with complications. Prolonged use on hypoperfused digits can potentially cause digital injury. Metal components of oximeter probes will heat up in strong electromagnetic fields, and the use of non-MRI compatible oximeter probes during MRI scanning has been associated with thermal injury.

Cannesson M, Attof Y, Rosamel P, et al. Respiratory variations in pulse oximetry plethysmographic waveform amplitude to predict fluid responsiveness in the operating room. Anesthesiology. 2007;44(4):273–9.

Costarino AT, Davis DA, Keon TP. Falsely normal saturation reading with the pulse oximeter. Anesthesiology. 1987;67:830–1.

Dempsey MF, Condon B. Thermal injuries associated with MRI. Clin Radiol 2001;56:457–65.

Gehring H, Hornberger C, Matz H, et al. The effects of motion artifact and low perfusion on the performance of a new generation of pulse oximeters in volunteers undergoing hypoxemia. Respir Care. 2002;47:48.

Hinkelbein J, Genzwuerker HV, Fielder F. Detection of a systolic pressure threshold for reliable readings in pulse oximetry. Resuscitation. 2005;64:315.

Kelleher JF, Ruff RH. The penumbra effect: vasomotion–dependent pulse oximeter artifact due to probe malposition. Anesthesiology. 1989;71:787–91.

Lee WW, Mayberry K, Crapo R, Jensen RL. The accuracy of pulse oximetry in the emergency department. Am J Emerg Med. 2000;18:427.

Poets CF, Seidenberg J, von der Hardt H. Failure of a pulse oximeter to detect sensor displacement. Lancet. 1993;341:244.

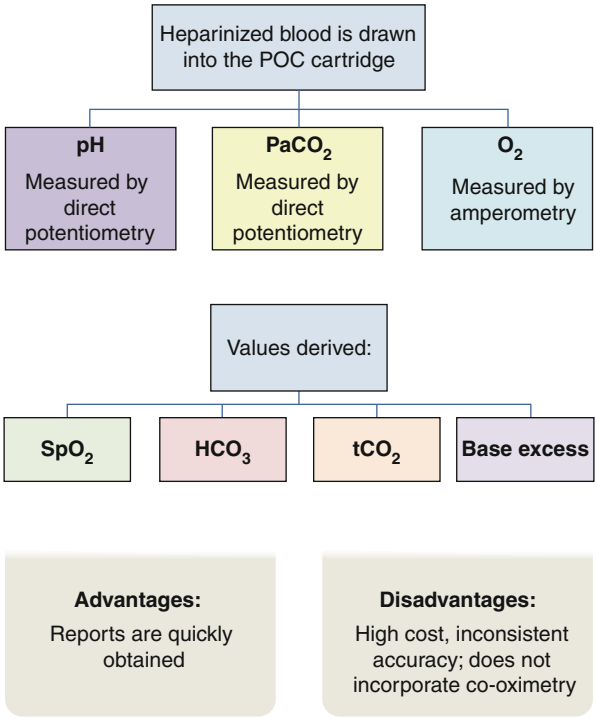
Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry. III: Effects of interference, dyes, dyshaemoglobins and other pigments. Anaesthesia 1991;46:291–295.

Van de Louw A, Cracco C, Cerf C, et al. Accuracy of pulse oximetry in the intensive care unit. Intensive Care Med. 2001;27:1606.

Wille J, Braams R, van Haren WH, et al. Pulse oximeter–induced digital injury: frequency rate and possible causative factors. Crit Care Med. 2000;28:3555–7.

2.22 Point of Care (POC) Cartridges

Point of Care (POC) cartridges are now in use for the bedside measurement of the pH, $PaCO_2$ and PaO_2.



Canham EM. Interpretation of arterial blood gases. In: Parsons PE, Weiner-Kronish JP, editors. Critical care secrets. 3rd ed. Philadelphia: Hanley and Belfus, Inc, 2003; p. 21–4.

2.23 Capnography and Capnometry

Capnography		Capnometry
Capnography is the real time monitoring of the exhaled CO_2 over time (or sometimes, over volume): it is displayed as a waveform.		Capnometry is the non-invasive measurement of exhaled CO_2, which is displayed as an end expiratory (end-tidal) value.The inspiratory and expiratory levels of CO_2 are shown as a partial pressure or percentage on a digital or analog display. However, the terms capnography and capnometry are often used interchangeably.
Time Capnography: The CO_2 levels displayed against *time* on the x axis.	**Volume Capnography:** The CO_2 levels displayed against *expired volume* on the x axis.	

Waveform analysis can provide valuable information regarding the adequacy of gas sampling, and leaks in tubing, and can identify certain prevailing disorders.

Capnograph waveform analysis provides information on CO_2 production, alveolar ventilation, perfusion, breathing pattern, status of the ventilator circuit and endotracheal tube position.

Height (E_tCO_2)	Frequency	Shape	Height
E_tCO_2 is the maximum partial pressure of CO_2 achieved at end-exhalation (Sect. 2.24).	Frequency represents the respiratory rate.	Can provide information about specific abnormalities (Sects. 2.35, 2.36 and 2.37).	See (Sect. 2.35)

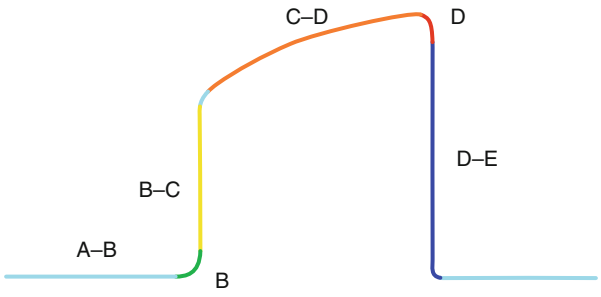
*CO_2 measurements techniques use Raman spectrography, mass spectrography, photoacoustic spectrography and chemical colorimetric analysis and infrared spectrography. The last is the most widely used. Single-use qualitative colorimetric end-tidal CO_2 detectors use indicator discs that change color when the CO_2 concentration of exhaled gas exceeds 2 %: from purple ($EtCO_2 < 3$ mmHg) to yellow ($EtCO_2 > 15$ mmHg).

Sullivan KJ, Kissoon N, Goodwin SR. End–tidal carbon dioxide monitoring in pediatric emergencies. Pediatr Emerg Care. 2005;21(5):327–32.

2.24 The Capnographic Waveform

The capnographic wave form is divided into six distinct parts.

A–B: dead-space exhalation	The first part of exhalation contains air from the proximal airway (the conductive zone of the lung).	This air is devoid of CO_2, (provided there is no rebreathing) and so the CO_2 waveform is a flat line that hugs the baseline.
B: the onset of alveolar exhalation	Alveolar air contains CO_2.	As alveolar air begins to arrive at the sampling site, the capnograph shows a sudden upturn.
B–C: the continuance of alveolar exhalation	The CO_2 rises rapidly as alveolar gas mixed with dead-space gas arrives at the sensor.	The capnograph shows a steep upslope.
C–D: the alveolar plateau	Most of the gas received at the sensor is now alveolar gas.	The gradually upsloping plateau represents the constant emptying of viable alveoli.
D: End-tidal CO_2 (E_tCO_2)	The peak at the end of the plateau represents the averaging of alveolar CO_2 levels.	The peak represents the end-tidal CO_2.
D–E: inspiratory washout	The graph then falls rapidly to the baseline.	The nadir represents the negligible CO_2 (0.003 % or 0.02 mmHg) that reaches the alveoli from the ambient air.



Stock MC. Capnography for adults. Crit Care Clinics. 1995;11:219.

2.25 Main-Stream and Side-Stream Capnometers

Mainstream capnometers	Side-stream capnometers
A CO_2 sensor (infrared detector) mounted on a cuvette (T- adapter) is interposed between the ET and the patient-circuit. CO_2 analysis is performed within the airway, obviating the need for need for gas sampling	A relatively long sampling tube connected to the piece draws away the gas sample to a CO_2 sensor located in a central unit. The sampling flow rate can be as high as 150 ml/min. This can result in substantial deformation of the waveform when low tidal volumes are used as in neonates and infants.
There is no sampling tube. Sensor windows are prone to clogging by secretions, aerosols or water droplets.	Sampling tube prone to becoming obstructed as secretion can be sucked in by the rapid aspiration rate. Leakages from the circuit are possible (Sect. 2.27)
No time lag owing to the centrally located processor.	Time lag in display (CO_2 flight time) owing to the distance of the sensor from the airway.
Unaffected by changes in water vapour pressure. The temperature within is maintained at around 39°C to prevent condensation (which can spuriously elevate E_tCO_2).	Affected by changes in water vapour pressure. By slightly modifying the standard nasal cannulae, it is possible to make fairly precise measurements even in patients breathing supplemental oxygen through nasal cannulae.
Cannot be used in the absence of an artificial airway.	Since exhaled gas is sampled from the nasal cavity using nasal adaptors, measurement is possible in the absence of an artificial airway.
Difficult to use in patients undergoing prone ventilation.	Relatively easy to connect in unusual positions (such as prone position).
Sterilization is difficult.	Easy to sterilize.
Can increase circuit dead-space & so elevate $PaCO_2$	Side stream capnometers using micro-stream technology have been developed. Using sampling flow rates of as low as 50 ml/min**. The emitted wavelength is within a narrower IR band (4.2–4.35 μm) which more closely matches the absorption spectrum for CO_2.

*Burns may occur if the heated sensing head lies in contact with the patient's skin.
**The rate of gas sampling ranges from 50 to 2,000 ml/min (usually 50–200 ml/min). When the sampling flow rate exceeds the expired gas flow, contamination from the base gas flow source is inevitable.

Kalenda Z. Mastering infrared capnography. Utrecht, The Netherlands: Kerckebosch-Zeist, 1989, p101.

Moon RE, Camporesi EM. Respiratory monitoring. In: Miller RD, editor. Miller's anesthesia. 6th ed. Philadelphia: Elsevier/Churchill Livingstone; 2005.

2.26 $P_{Et}CO_2$ (E_tCO_2): A Surrogate for $PaCO_2$

CO_2 diffuses rapidly across all biological membranes including the alveolo-capillary membrane. Arterial CO_2 ($PaCO_2$) equilibrates rapidly with alveolar CO_2 ($PACO_2$), and is effectively identical with it. $P_{Et}CO_2$ offers a non-invasive means of monitoring $PaCO_2$, given that:

In health:	In disease:
The value of $P_{Et}CO_2$ is close to the value of $PACO_2$; and therefore to that of $PaCO_2$. $PaCO_2$ and $PACO_2$ differ by such a small amount (generally < 5 mm) such as usually makes no clinical difference. The trends in $P_{Et}CO_2$ closely match the trends in $PaCO_2$.	In disease, physiological dead-space is often increased because of patent but under-perfused alveoli. Due to the lack of an effective pulmonary circulation CO_2 cannot effectively diffuse into alveoli. Under such circumstances, $PaCO_2$ can substantially exceed $P_{Et}CO_2$. In spite of this, in the absence of major *changes* in dead-space ventilation, the $P_{Et}CO_2$ trends still match those of $PaCO_2$.

CO_2 values cannot be used as an absolute surrogate for $PaCO_2$. However the E_tCO_2 may be expected to *parallel* the changes in $PaCO_2$ (i.e, the $[A\text{-}a]CO_2$ gradient remains constant) provided that:

- Stable cardiac condition
- Stable pulmonary condition
- Stable body temperature

Fletcher R, Jonson B. Deadspace and the single breath test for carbon dioxide during anaesthesia and artificial ventilation. Br J Anaeasth. 1984;56:109–19.

Nunn JF, Hill DW. Respiratory dead space and arterial to end-tidal CO_2 tension difference in anesthetized man. J Appl Physiol. 1960;15:383–9.

Shankar KB, Moseley H, Kumar Y, Vemula V. Arterial to end-tidal carbon dioxide tension difference during cesarean section anaesthesia. Anaesthesia. 1986;41:698–702.

2.27 Factors Affecting $P_{Et}CO_2$

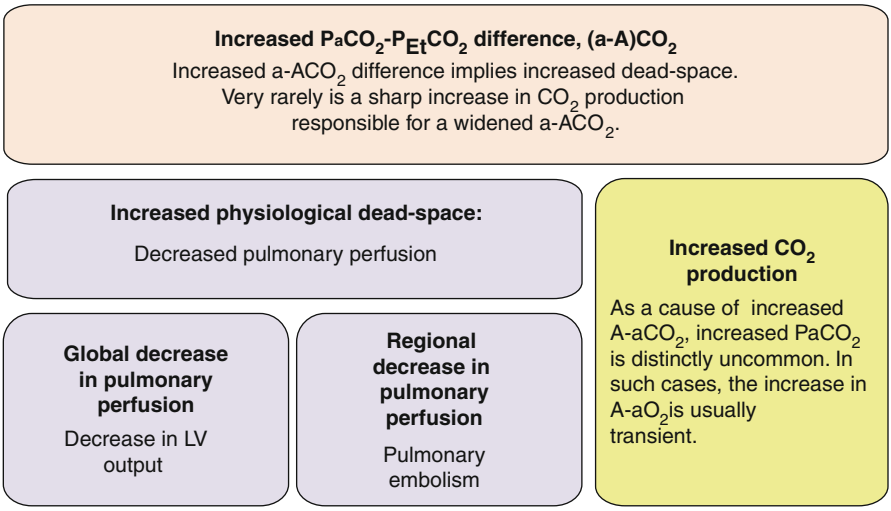
Factors that increase $P_{Et}CO_2$	Factors that decrease $P_{Et}CO_2$
Increase in CO_2 production*: Fever, shivering, convulsions; infusion of $NaHCO_3$, blood, glucose or parenteral nutrients Release of a tourniquet CO_2 insufflation or embolism	**Decreased CO_2 production:** Hypothermia
Increase in pulmonary perfusion: Increase in cardiac output Increase in blood pressure	**Decrease in pulmonary perfusion:** Decreased cardiac output Fall in BP, hypovolemia Pulmonary embolism Wedged PA catheter
Decrease in alveolar ventilation: Hypoventilation (see Sect. 1.35)	**Increase in alveolar ventilation** Hyperventilation
Airway related problems Bronchial intubation Partial airway obstruction	**Airway related problems** Accidental extubation Partial or complete airway obstruction Apnea
Machine-related factors: CO_2 scrubber used up Insufficient inflow of fresh gas Leaks in circuit Malfunctioning ventilator valves	**Machine-related factors:** Circuit disconnection Leakage of gas during sampling: gas pump, flow regulator, sampling system (connector to the sampling port, water trap) Malfunction of ventilator

*Unlike in paralyzed mechanically ventilated patients, an increase in CO_2 production will not lead to a rise in $P_{Et}CO_2$ in spontaneously breathing individuals (owing to the reflex hyperventilation that a high CO_2 level evokes in these persons).

(Modified from: Kodali BS. Factors influencing $P_{Et}CO_2$. 2007. Welcome to capnography.com. Last accessed 6 June 2012.)

Shankar KB, Moseley H, Kumar AY, Delph Y. Capnometry and anaesthesia. Review article. Can J Anaesth. 1992;39(6):617–32.

2.28 Causes of Increased $PaCO_2$-$P_{Et}CO_2$ Difference



Phan CQ, Tremper KK, Lee SE, Barker SJ. Noninvasive monitoring of carbon dioxide: a comparison of the partial pressure of transcutaneous and end-tidal carbon dioxide with the partial pressure of arterial carbon dioxide. J Clin Monit. 1987;3:149–54.

2

2.29 Bohr's Equation

It is possible to estimate the dead space, utilizing Bohr's equation.

All the exhaled CO_2 comes from the alveolar gas. None of the exhaled CO_2 comes from the dead-space air.
Therefore,

$$\mathbf{VT = VA + VD}$$

Or, tidal volume (VT) = Alveolar gas volume (VA) + dead-space gas (VD)
Rearranging,

$$\mathbf{VA = VT–VD \ldots(Eq\ 2.1)}$$

$$\mathbf{VT \times FECO_2 = VA \times FACO_2 \ldots(Eq\ 2.2)}$$

Where,
VT = tidal volume
$FECO_2$ = Fractional concentration of CO_2 in exhaled gas
VA = Alveolar gas volume
$FACO_2$ = Fractional concentration of CO_2 in alveolar gas

Substituting the value of VA (Eq 2.1) within Eq 2.2

$$\mathbf{VT \times FECO_2 = (VT–VD) \times FACO_2}$$

Therefore,

$$\mathbf{VD/VT = (FACO_2–FECO_2) / FACO_2}$$

Since the partial pressure of a gas is proportional to its concentration, the equation can be rewritten as "Bohr's equation":

$$\mathbf{VD/VT = (PACO_2 – PECO_2)/PACO_2}$$

And since the PCO_2 of alveolar gas ($PACO_2$) very nearly equals the PCO_2 of arterial gas ($PaCO_2$),

$$\mathbf{VD/VT = (PaCO_2 – PECO_2)/PaCO_2}$$

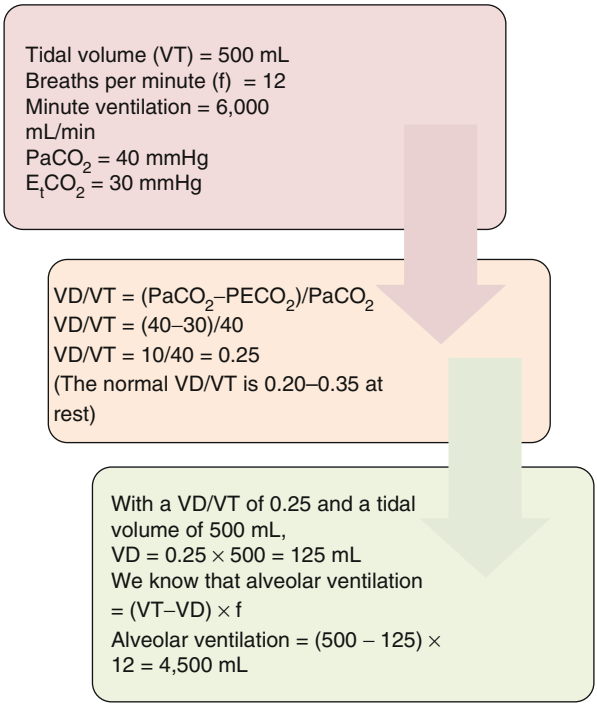
Thus, by simultaneously measuring the end-expiratory CO_2 ($P_{Et}CO_2$) and the $PaCO_2$, the dead-space to tidal volume ratio can be calculated (see Sect. 2.30).

Criner GJ, D'Alonzo G, editors. Pulmonary pathophysiology. Lyndell: Fence Creek Publishing Co.; 1998.

Shankar KB, Moseley H, Kumar AY, Delph Y. Capnometry and anaesthesia. Review article. Can J Anaesth. 1992;39(6):617-32.

2.30 Application of Bohr's Equation

Consider the following data in a patient:



Criner GJ, D'Alonzo G, editors. Pulmonary pathophysiology. Lyndell: Fence Creek Publishing Co.; 1998.

2.31 Variations in E_tCO_2

Discrepancy between the $PaCO_2$ and the $P_{Et}CO_2$ can occur when there is an increase in the dead-space, or if a significant V/Q mismatch occurs. The impact of pulmonary disease on $P_{Et}CO_2$ is unpredictable and widening of the gradient often occurs.

On rare occasions, when large tidal volumes are used to inflate lungs with low-V/Q ratios, the $P_{Et}CO_2$ may actually exceed the $PaCO_2$

Since CO_2 is an easily diffusible gas with respect to biological membranes, the drop in the end-tidal CO_2 tension relative to arterial CO_2 is only about 2–5 mmHg. However, this is at best a rough approximation and in disease the end-tidal CO_2 may be prone to substantial variation.

High concentrations of either oxygen or nitrous oxide may cause variations in the capnogram as both these gases have similar infrared spectra to CO_2 and correction factors should be applied when mixtures of these gases are breathed.

Thus, in health, trends in arterial CO_2 are matched by the end-tidal CO_2. With unstable or evolving lung pathology, the end-tidal CO_2 may neither reflect nor parallel changes in $PaCO_2$.

Moorthy SS, Losasso AM, Wilcox J. End-tidal PCO_2 greater than $PaCO_2$. Chest. 1984;12:534.

2.32 False-Positive and False-Negative Capnography

FALSE NEGATIVE (a flat wave form in spite of a properly sited endotracheal tube)	
Cardiac arrest	The sluggish pulmonary blood flow delivers little CO_2 to the alveoli for excretion.
Large air leak (e.g. ruptured ET cuff)	A large amount of atmospheric air dilutes the exhaled air the CO_2-concentration of which resultantly falls.
An obstructed ET tube	CO_2 from the exhaled air has no access to the capnograph sensor.
FALSE POSITIVE (CO_2 detected on the capnograph in spite of a improperly sited endotracheal)	
Endotracheal tip resides with in the pharynx	In spite of this effective (or partially compromised) ventilation may still be possible.
Aggressive 'bagging'	Aggressive bag-and-mask ventilation has resulted in gastric distension with CO_2 containing air.
Carbonated bevereges	In animal studies, ingestion of carbonated beverages has also resulted in false positive capnographic measurements.

When capnography is false positive, the E_tCO_2 values will inevitably decline over successive breaths. It has therefore been suggested that E_tCO_2 levels be closely monitored for a minimum of six successive breaths.

The shape of the capnograph remains remarkably similar in all healthy humans. This of course means that any deviation from the typical shape must be inquired into (see following sections).

Hasan A. Esophageal intubation. In: Understanding Mechanical Ventilation: a Practical Handbook. London: Springer; 2010. p.183, 309–10.

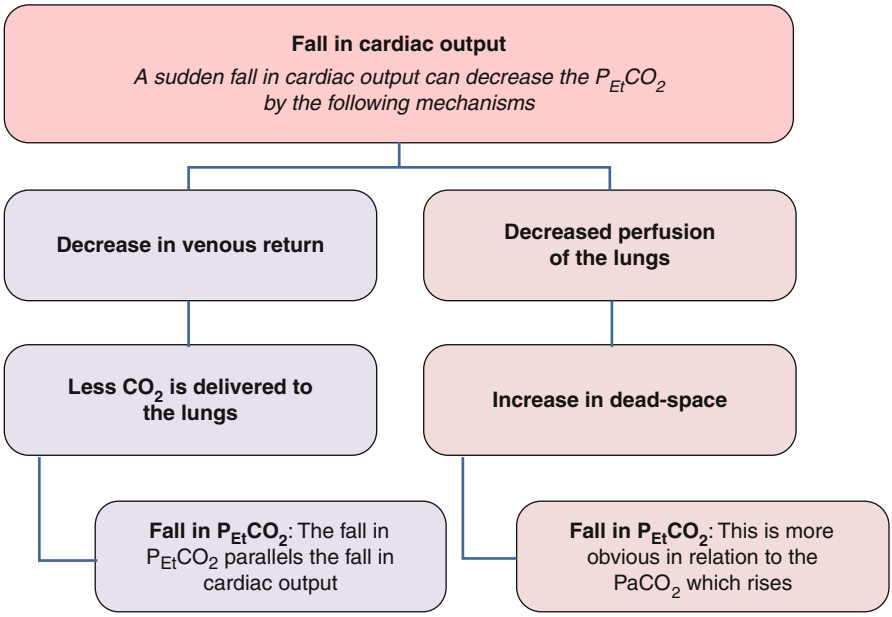
Puntervoll SA, Soreide E, Jacewicz W, et al. Rapid detection of oesophageal intubation: take care when using colorimetric capnometry. Acta Anaesthesiol Scand. 2002;46(4):455–7.

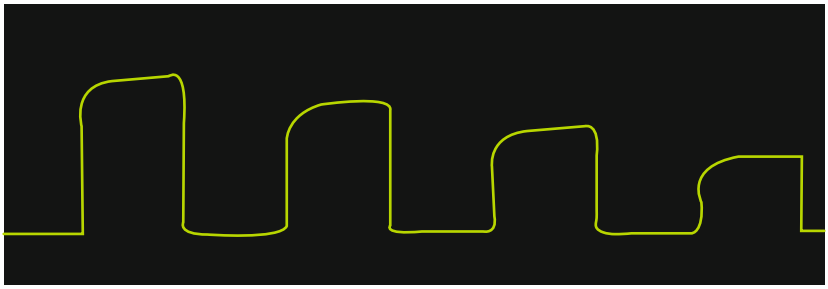
Qureshi S, Park K, Sturmann K, et al. The effect of carbonated beverages on colorimetric end–tidal CO(2) determination. Acad Emerg Med. 2000;7(10):1169.

2

2.33 Capnography and Cardiac Output

When alveolar ventilation is constant, the $P_{Et}CO_2$ reflects pulmonary perfusion, which itself is dependent upon the cardiac output.





Real-time capnograph showing fall in cardiac output due to cardiac arrest

Isserles S, Breen PH. Can changes in end-tidal PCO_2 measure changes in cardiac output? Anesth Analg. 1991;73:808.

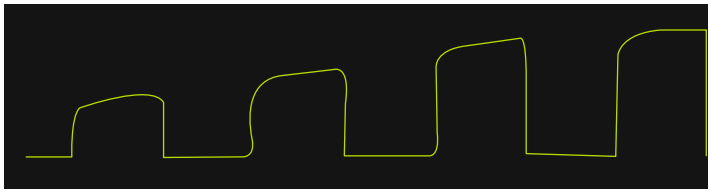
Shibutani K, Shirasaki S, Braaz T, et al. Changes in cardiac output affect $P_{Et}CO_2$, CO_2 transport, and O2 uptake during unsteady state in humans. J Clin Monit. 1992;8:175–6.

2.34 Capnography as a Guide to Successful Resuscitation

Using capnography, it is possible to differentiate *asphyxic cardiac arrest* (very high $P_{Et}CO_2$) from *primary cardiac arrest* (increase in $P_{Et}CO_2$ is not as high). $P_{Et}CO_2$ can provide a valuable guide to CPR.

Successful CPR	CPR resumption	CPR termination	
A sudden rise in $P_{Et}CO_2$ is often the earliest indicator of the revival of the hemodynamics. It is more sensitive than the ECG, pulse, or blood pressure, and is unaffected by the artefacts produced by chest compression. The transient rise in $P_{Et}CO_2$ reflects the elimination of the CO_2 built up within tissues.	Conversely, a drop in $P_{Et}CO_2$ in apatient who has just been successfully revived may indicate the need for resumption of CPR.	In a patient with pulseless electrical activity, the $P_{Et}CO_2$ measured at 20 min after the commencement of CPR can provide a valuable guide of outcome.	
		$P_{Et}CO_2$ after 20 min CPR: <10 mmHg Further continuation of CPR is unlikely to be fruitful*.	**$P_{Et}CO_2$ after 20 min CPR: >18 mmHg** Heralds a successful out come to the CPR*.

*No specific number can assigned as a cut off value in distinguishing survivors from non-survivors: it is believed that the chances for survival increase by 16 % for every 1 mmHg that the $P_{Et}CO_2$ rises.



The return of spontaneous circulation following successful CPR

Callaham M, Barton C. Prediction of outcome of cardiopulmonary resuscitation from end-tidal carbon dioxide concentration. Crit Care Med. 1990;18:358.

Falk JL, Rackow ED, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. N Engl J Med. 1988;318(10):607–11.

Grmec S, Klemen P. Does the end-tidal carbon dioxide ($ETCO_2$) concentration have prognostic value during out-of-hospital cardiac arrest? J Emerg Med. 2001;8:263–9.

Sanders AB, Kern KB, Otto CW, et al. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation: a prognostic indicator for survival. JAMA. 1989;262:1347–51.

2.35 Capnography in Respiratory Disease

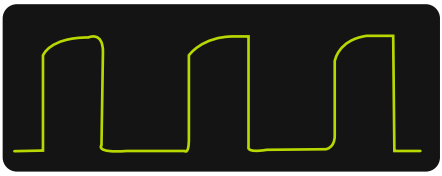
Pulse oximetry which measures *oxygenation* cannot serve as a replacement for capnography, which monitors *ventilation*. Capnography will diagnose hypoventilation long before the latter results in hypoxia, and this is especially the case in patients on supplemental oxygen. In *hypoventilation,* tall (high $P_{Et}CO_2$) low-frequency waves are manifest with a well-defined alveolar plateau (a similar waveform can occur when the dead space is increased). In *hyperventilation*, short (low $P_{Et}CO_2$) high-frequency waves with a well-defined alveolar plateau are seen.

Hypoventilation

The capnograph shows:

slow respiratory rate (low frequency)

High CO_2 levels (tall waves)



Hypoventilation

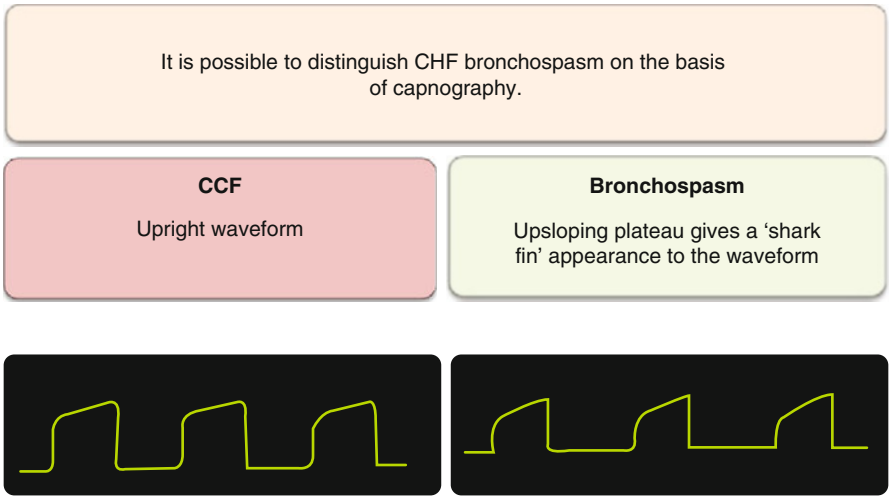
The capnograph shows:

High respiratory rate (low frequency)

Low CO_2 levels (relatively short waves)

Simple pneumothorax (doesn't affect cardiac output: $P_{Et}CO_2$ rises) can be differentiated from tension pneumothorax (cardiac output falls: $P_{Et}CO_2$ falls).

It is also possible to differentiate congestive cardiac failure (CCF) from bronchospasm on the basis of the shape of the capnographic waveform: in CCF, the waveform is relatively upright.



In airways obstruction (severe bronchoconstriction in an acute attack of asthma, or airway narrowing due to loss of elastic recoil in COPD), slow exhalation leads to slow CO_2-elimination results in a steeply upsloping alveolar plateau, giving a 'shark fin' appearance to the waveform. This slope correlates closely with spirometric indices of airway obstruction, making it possible to monitor bronchodilator therapy—and potentially, to estimate bronchospasm in those who cannot perform spirometry (such as patients at the extremes of age). The length of the alveolar plateau divided by the respiratory rate (the 'E_tCO_2 ratio'), closely correlates with the airway resistance.

Soto RG, Fu ES, Vila H Jr, et al. Capnography accurately detects apnea during monitored anesthesia care. Anesth Analg 2004;99(2):379–82.

Grmec S, Lah K, Tusek-Bunc K. Difference in end-tidal CO_2 between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. Crit Care. 2003;7:R139–44.

Kodali BS. Factors influencing $P_{Et}CO_2$. 2007. Welcome to capnography.com. Last accessed 6 June 2012.

Krauss B, Deykin A, Lam A, et al. Capnogram shape in obstructive lung disease. Anesth Analg 2005;100(3):884–8.

Kunkov S, Pinedo V, Silver EJ, et al. Predicting the need for hospitalization in acute childhood asthma using end–tidal capnography. Pediatr Emerg Care 2005;21(9):574–7.

Yaron M, Padyk P, Hutsinpiller M, et al. Utility of the expiratory capnogram in the assessment of bronchospasm. Ann Emerg Med 1996;28(4):403–7.

2.36 Esophageal Intubation

Unrecognized placement of the endotracheal tube (ETT) into the esophagus can prove catastrophic. None of the checks that are routinely performed to ascertain correct placement of the tube are infallible. For example, conduction of breath sounds to the chest wall is possible even though the tube may reside in the esophagus (in fact, the chest wall can still move on bagging the patient because gastric inflation can transmit some movement to the chest wall; gas exchange may even be sustained for a while because of the diaphragmatic movement so produced; a fall in the SpO_2 is often a late sign).

Conversely, the absence of breath sounds over the epigastrium does not rule out esophageal intubation.

Capnograph in esophageal intubation

Carbon dioxide has its origins in the lungs. Measurable CO_2 in the ETT can only mean that the ETT resides in the tracheobronchial tree. Capnometry may be the most reliable of the available indices—short of bronchoscopic confirmation of tracheal intubation—that distinguish tracheal from esophageal intubation (Note that if the patient had initially been manually ventilated with bag and mask, some of the exhaled gas that was forced into the esophagus during bagging can be measured during the first few exhalations. On the other hand, occlusion of the tip of the ETT by cricoid pressure, applied PEEP and bronchospasm can result in failure to detect CO_2).

E_tCO_2 can help guide the ETT during a blind oral (or nasal) intubation. In a spontaneously breathing patient, a capnometer hooked to the ETT will register increase in amplitude of the $EtCO_2$ as the tube approaches the larynx, displaying the classical capnographic waveform as the ETT passes between the vocal cords.

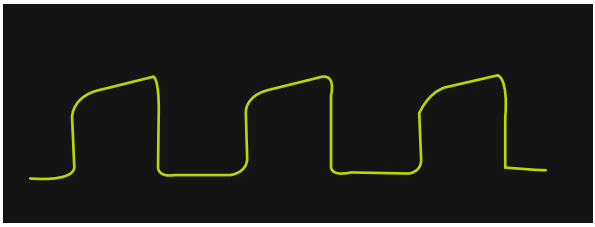
Hasan A. Esophageal intubation. In: Understanding Mechanical Ventilation: a Practical Handbook. London: Springer; 2010. p.183, 309–10.

Ionescu T. Signs of endotracheal intubation. Anaesthesia. 1981;36:422.

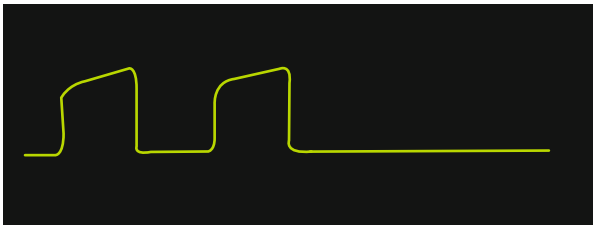
Linko K, Paloheimo M and Tammisto T: Capnography for detection of accidental oesophageal intubation. Acta Anaesthesiol Scand. 1983;27:199–202.

Murry IP, Modell JH. Early detection of endotracheal tube accidents by monitoring carbon dioxide concentration in respiratory gas. Anesthesiology. 1986;59:344–6.

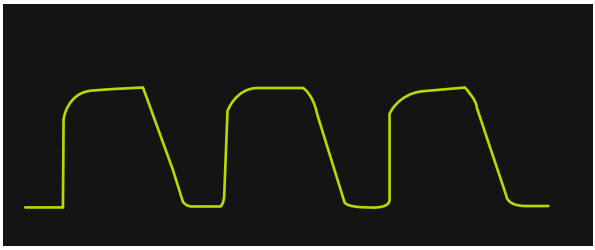
2.37 Capnography in Tube Disconnection and Cuff Rupture



Normal capnograph



Capnograph in self-extubation or disconnection

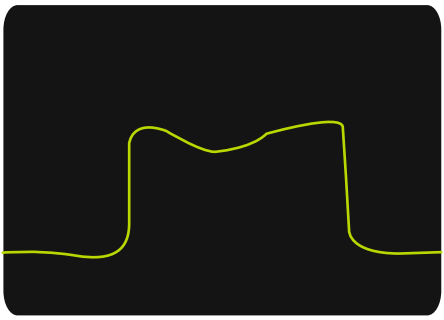


Ruptured ET cuff showing a gradual descent

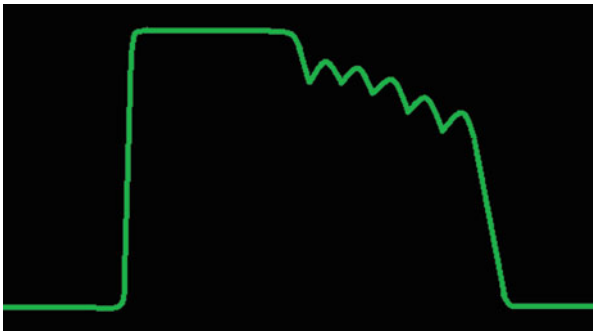
2.37.1 Biphasic Capnograph

A biphasic pattern on the capnogram may be obtained under some circumstances, eg when there are *air leaks* in the sampling system.

In *severe kyphoscoliosis*, lung volumes and lung mechanics may considerably differ on both sides. The phases of lung emptying can therefore be out of synchrony on the two sides. Consequently, the plateau of the capnogram shows a late hump (see fig below).



The pulsation of the heart and great vessels, by gently compressing the lungs, can produce minor changes in airflow. These *cardiac oscillations* can sometimes be recognized on the waveform, especially when the respiratory rate is low. They appear as diminutive serrations towards the end of expiration, and their frequency correlates with the heart rate (see fig below).



Kodali BS. Factors influencing $P_{Et}CO_2$. 2007. Welcome to capnography.com. Last accessed 6 June 2012.

References

Birmingham PK, Cheney FW, Ward RJ. Esophageal intubation: a review of detection techniques. Anesth Analg. 1986;65:886–91.

Brand TM, Brand ME, Jay GD. Enamel nail polish does not interfere with pulse oximetry. J Clin Monit Comput. 2002;17:93.

Busch MR, Mace JE, Ho NT, Ho C. Roles of the beta 146 histidine residue in the molecular basis of the Bohr effect of hemoglobin: a proton nuclear magnetic resonance study. Biochemistry. 1991;30:1865.

ECRI Health Devices Program. Carbon dioxide monitors. Health Devices. 1986;15:255–85.

Fluck Jr RR, Schroeder C, Frani G, et al. Does ambient light affect the accuracy of pulse oximetry? Respir Care. 2003;48:677.

Greene GE, Hassel KT, Mahutte CK. Comparison of arterial blood gas with continuous intraarterial and transcutaneous PO2 sensor in adult critically ill patients. Crit Care Med. 1987;15:491.

Inman KJ, Sibbald WJ, Rutledge FS. Does implementing pulse oximetry in a critical care unit result in substantial arterial blood gas savings? Chest. 1993;104:543.

Kalenda Z. Mastering infrared capnography. Utrecht, The Netherlands: Kerckebosch-Zeist, 1989:p101.

Linlo K, Paloheimo M, Tammisto T. Capnography for detection of accidental oesophageal intubation. Acta Anaesthesiol Scand. 1983;27:199–202.

Martin L, Khalil H. How much reduced hemoglobin is necessary to generate central cyanosis? Chest. 1990;97:182.

O'Flaherty D, Adams AP. The end-tidal carbon dioxide detector. Assessment of new method to distinguish oesophageal from tracheal intubation. Anaesthesia. 1990;45:653–5.

Sanders AB, Kern KB, Otto CW, et al. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation: a prognostic indicator for survival. JAMA. 1989;262:1347.

Veyckemans F, Baele P, Guillaume JE, et al. Hyperbilirubinemia does not interfere with hemoglobin saturation measured by pulse oximetry. Anesthesiology. 1989;70:118.

Zeballos RJ, Weisman IM. Reliability of noninvasive oximetry in black subjects during exercise and hypoxia. Am Rev Respir Dis. 1991;144:1240.

http://www.springer.com/978-1-4471-4314-7

Handbook of Blood Gas/Acid-Base Interpretation

Hasan, A.

2013, XVIII, 332 p. 361 illus., 359 illus. in color.,
Softcover
ISBN: 978-1-4471-4314-7