Capnography: Principles and application in critical care medicine

Lokvendra Budania, Kush Goyal

Email:drbudania@gmail.com

Abstract

Capnography has become an essential monitoring tool in perioperative and critical care setting. It helps monitor dynamic physiological processes more precisely and promptly, compared to other monitoring modalities. Methods to measure and display carbon dioxide concentrations in respired gases include infrared spectrography, mass spectrography, Raman spectrography, photoacoustic analysers or colorimetry. It is used to confirm correct endotracheal tube placement, ensure effective and consistent chest compression, detect return of spontaneous circulation and diagnose alterations in haemodynamics and ventilation of critically ill patient. It helps to optimise fluid resuscitation and monitor shock progression by providing insight into adequacy of tissue perfusion. Thus, a detailed study and interpretation of capnographic waveform is essential.

Keywords: Capnogram, capnography, cardiac output, cardiopulmonary resuscitation, respiratory dead space.

Introduction

Capnography is a continuous, real time, noninvasive and rapid monitoring technique to measure and display carbon dioxide (CO_2) in the gases breathed in and out during respiration. This can be used reliably to monitor changes in ventilation, circulation and metabolism.[1-3] Since its application was first described in anaesthesiology over three decades ago, it has evolved as a mandatory monitoring equipment in the perioperative period and as an essential tool for monitoring ventilation and haemodynamics, in a critically ill mechanically ventilated patient.[4-6] It provides useful physiological information, helps to keep track of effects of physiological interventions and assess prognosis in a critically ill patient.[7,8] It prevents life-threatening events, by alerting health care providers allowing early institution of treatment.[1,4]

The CO_2 concentration can be plotted against time (when it is called timed capnography) or volume (when it is termed volume capnography).[1] The equipment used to generate the graph is called a capnograph. The waveform is called a capnogram.[1] Capnometry is the numerical measurement and display of maximum inspiratory and expiratory CO_2 concentration.[1] Capnometer is the equipment used to measure and display only the reading (no waveform).[1]

Principle

The capnographs usually used in the operation theatre use infrared absorption spectroscopy to measure carbon dioxide concentrations. Infrared light is passed through a sample of respired gas. Molecules having dissimilar atoms absorb infrared radiations at a specific wavelength producing absorption bands. According to Beer-Lambert law, the amount of light absorbed depends upon the

Lokvendra Budania, MD
Assistant Professor

Kush Goyal, DA, MD
Senior Resident, Department of Anaesthesiology, Kasturba Medical College, Manipal

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concentration of that substance. Energy generated by the absorption of infrared radiation causes molecules to vibrate, frequency of which depends upon molecular mass and atomic bonding within the molecule.[1,3,9,10]

Raman spectrography, photoacoustic (PAS) gas measurement and mass spectrography are other methods to measure carbon dioxide in the respired gases.

The colorimetric method consists of chemically treated foam indicator (Fenem indicator), in a plastic housing, which changes colour on exposure to CO_2.[1] It lacks waveform and gives false positive results when device is contaminated with epinephrine, surfactant or atropine.[11,12] It cannot be used in infants less than 1 kg. Increased resistance to airflow makes it unsuited for patients breathing spontaneously. Excessive humidity in expired gas will render it inoperative in 15-20 min. It can be damaged by mucous, gastric contents and intratracheal epinephrine.[11]

Methods of sampling

There are two ways of sampling of gas for analysis: mainstream and sidestream sampling.

Sidestream sampling

A tiny aspirator samples gases from the patient's end of breathing system through a sampling tube and the gas is delivered in to a sample chamber *(Figure 1)*. The sampling tube is made up of Teflon, impermeable to CO_2 and does not react with anaesthetic agents. The optimal gas flow rate is 50-200 mL/min, which ensures reliable capnograms in both adults and children. If sampling flow exceeds the expired gas flow, fresh gas will be entrained into sampling chamber. The sampling gas pump, tubing, connector to breathing circuit and water trap, all constitutes the site for gas leak. Depending upon the size and length of sampling tube, there is a delay in gas detection (CO_2 flight time), which significantly increases when sampling gas flow is low and sampling dead space (length of tubing) is more. Gas from sampling chamber is diverted towards gas scavengers or reinjected in breathing circuit. It can be used for spontaneously breathing patients, along with O_2 supplementation. Additionally, there is no problem with sterilisation, ease of connection and ease of use when patient is in unusual position.[1,9,10]

Mainstream sampling

In mainstream sampling analysers, sample chamber will be positioned within the patient's breathing circuit *(Figure 2)*. The infrared rays traverse the gas within the sample chamber to an infrared detector within the chamber. It does not remove gas from breathing circuit. Hence, it has a faster response with no delay, minimal leakage and no sampling suction pumps are required. To prevent water condensation from humidity of expired gas, sensors are heated above body temperature. Hence, it must be kept away from patient's skin as it can cause burns. The device is relatively heavy and can cause endotracheal tube kinking or accidental extubation.[1,3,9-11] Lighter mainstream capnographs are now available that do not add weight to the connections.

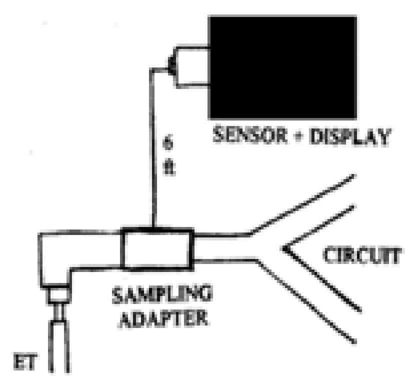

Figure 1: Sidestream analyser

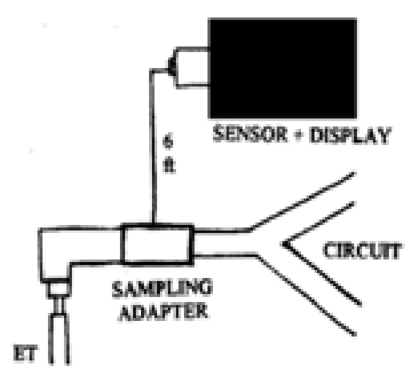

Figure 2: Mainstream analyser

Basic physiology

Carbon dioxide (CO_2) generated as a result of cellular metabolism is transported to the heart by systemic circulation, from where it is pumped to pulmonary circulation. CO_2 diffuses into alveoli and equilibrates with end-capillary blood ($PACO_2 = PcCO_2$).[1,13] The actual concentration of CO_2 in the alveoli depends upon ventilation and perfusion into the alveoli (V/Q ratio). The alveoli with higher V/Q ratio have lower CO_2 compared to alveoli with low V/Q ratio that would have higher CO_2.

As expiration begins, exhaled gas from breathing circuit and anatomical dead space (no perfusion, no gas exchange) containing no CO_2 is exhaled (Phase I) *(Figure 3)*. As expiration continues, there is a rapid rise in carbon dioxide concentration suggesting mixing of dead space and alveolar gas producing a S-shaped upstroke on capnograph (Phase II). This is followed by a plateau phase (Phase III) represents the CO_2 gas from the alveoli. Although expiratory flow is higher initially and it tapers off at end of expiration, alveolar plateau has a positive slope. Positive slope indicates that the alveolar gas sampled at the end of exhalation is having higher CO_2 concentration, as the diffusion of CO_2 from pulmonary capillaries continues at same rate, but the dilution of CO_2 into lung volumes reduces progressively as exhalation continues.[1] The under ventilated alveoli (low V/Q, high PCO_2) empty slowly and contribute to rising slope of phase III. Slope and height of phase III depends upon alveolar CO_2 concentration and their emptying pattern. In turn, alveolar concentration depends upon ventilation and perfusion.

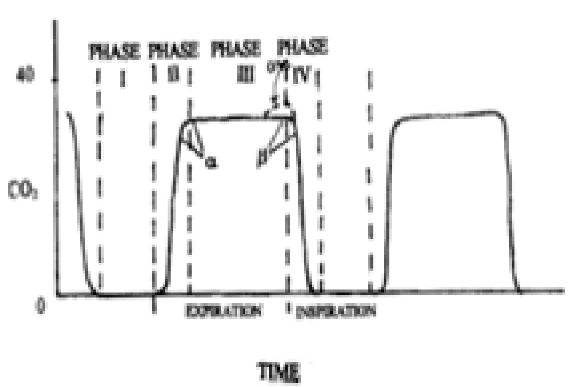

Figure 3: Normal time capnogram

The height of phase III depends upon cardiac output and the slope of phase II and III is dependent on emptying patterns of alveoli as well as V/Q ratio.[7,8,14] At the end of exhalation, phase III reaches a peak, and is called endtidal partial pressure of CO_2 ($PETCO_2$). Hence, slow exhalation, as in acute asthmatic attack or COPD, will produce a steeper alveolar plateau and a higher $PETCO_2$ *(Figure 4)*.[1] At the end of phase III, with the beginning of inspiration, capnograph takes an almost right angle turn and descends to baseline (Phase IV).

Concentration of CO_2 in the inspiratory gas depends upon breathing circuit, inspiratory flow and fresh gas flow rate *(Figure 5)*.[1] Cardiac oscillations appear as small, regular, saw tooth like positive waves in Phase IV at the end of the expiratory phase. These are due to contraction and relaxation of the heart and intrathoracic great vessels on the lungs, forcing air in and out *(Figure 6)*.[2]

The angle between phase II and III is referred to as the alpha angle *(Figure 3)*. Normally, it is 100-110°. It increases with increase in slope of phase III and indirectly indicates V/Q status of lung.[1] The angle between phase III and IV is nearly 90°, referred to as the beta angle *(Figure 3)*. It increases during rebreathing and prolonged response time of the capnograph.[1]

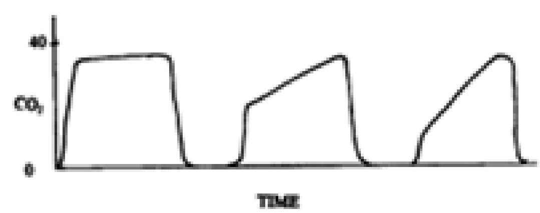

Figure 4: Steeper alveolar plateau due to obstruction to expiratory flow

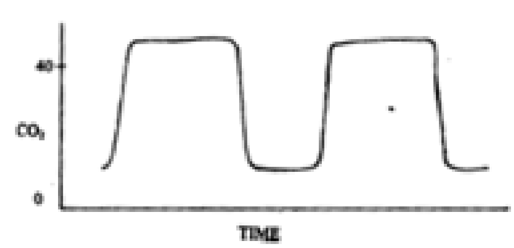

Figure 5: Rebreathing (elevated baseline) due to incompetent expiratory valve, low fresh gas flow rate, exhausted CO_2 absorbent

Figure 6: Cardiac oscillations

Volume capnograph

CO_2 concentration when plotted against expired volume is termed as 'volume capnograph' *(Figure 7)*. It can be used in mainstream capnographs, where cuvette containing CO_2 sensor is inserted between endotracheal tube and breathing circuit. Unlike timed capnography, inspiration is not monitored, and presence of endotracheal tube is mandatory for use of volume capnography.[1,7,8]

Phase I – Dead space gas (no CO_2)

Phase II – Progressive alveolar emptying (midportion of phase II marks the mean airway-alveolar interface that separate the airway from alveoli)[7,8]

Phase III - Alveolar plateau (small positive slope reflects the different time constants of emptying alveoli and continuous influx of CO_2 from pulmonary capillaries)

Volumetric capnography simultaneously measures expired CO_2 and tidal volume (V_T). Analysis of expired CO_2 as function of exhaled volumes along with $PaCO_2$ will give a precise idea about ratio of V_d (dead space) and V_T.[7,8]

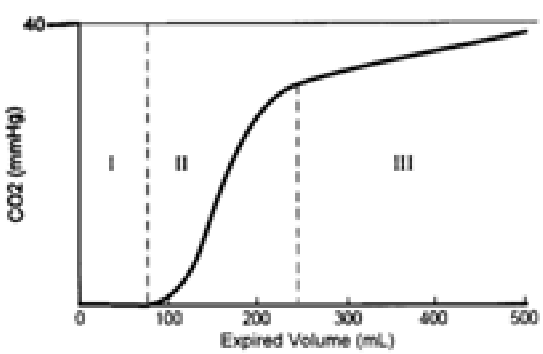

Figure 7: Volume capnogram

Clinical applications of capnography

The CO_2 value as measured at the mouth reflects the following: tissue production of CO_2, its transport from venous system to pulmonary artery (pulmonary perfusion), its elimination by lungs (alveolar ventilation) and gas delivery apparatus.[15,16] Five characteristics should be inspected: height, frequency, rhythm, baseline and shape.[1] Any leak or malfunction in gas sampling system from the airway to the sampling chamber, will result in an abnormal capnograph.

Metabolic causes of increased $PETCO_2$ includes increased body temperature, shivering, convulsions, excessive production of catecholamines, administration of sodium bicarbonate, release of an arterial tourniquet with reperfusion of ischemic areas, intravenous glucose therapy, parenteral hyperalimentation and CO_2 absorbed from peritoneal cavity after capnoperitoneum, pleural cavity after thoracoscopy or a joint after arthroscopy *(Figure 8)*.[1,7,8]

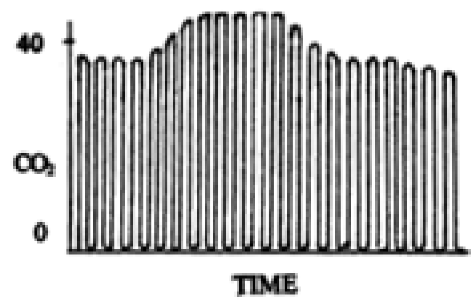

Figure 8: Abrupt increase in $PETCO_2$, unclamping arterial occlusion or release of arterial tourniquet

Utility of capnography in various clinical conditions

The actual value of end-tidal carbon dioxide, whether it is zero, low, or high, abruptly changing or gradually changing and the shape of the capnogram can provide valuable information about the clinical condition.

Verification of correct placement of endotracheal tube: The single most important utility of end-tidal carbon dioxide in the exhaled gas is the immediate and definitive evidence of correct endotracheal tube placement.[1,3,4,10,11,17-20] Other devices such as colorimetric CO_2 detectors, self-inflating oesophageal detectors, and no-waveform capnography do not match up the accuracy of waveform capnography for confirmation of endotracheal tube placement. Effective ventilation through supraglottic airway device should result into waveform capnography during cardiopulmonary resuscitation (CPR) and after return of spontaneous circulation (ROSC).[14,33] With oesophageal intubation, small waveform capnogram may be visible transiently, as a result of gas that has entered stomach during bag and mask ventilation, bystander mouth-to-mouth ventilation, or if patient consumed carbonated beverages or medication. However, this diminishes rapidly in concentration, after six to eight complexes, and abnormal waveform will usually differentiate oesophageal from tracheal intubation *(Figure 9)*.[10,18,19,21,22]

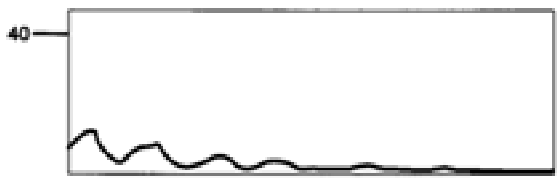

Figure 9: Oesophgeal intubation

Capnography can aid in performing blind oral or nasal intubation, to identify correct needle placement during transtracheal cricothyrotomy, and to verify tracheal intubation during awake fibreoptic bronchoscopy, especially when fibreoptic vision is obscured.[10,22]

Detection of apnoea, accidental extubation or ventilator disconnection: In a spontaneously breathing patient, it is used to detect apnoea whereas it may indicate ventilator disconnection in a patient on controlled ventilation.

***Rebreathing*:** If fresh gas flow (FGF) is inadequate, rebreathing of exhaled gas occurs and can be detected by rising baseline and higher inspired CO_2 concentration (*Figure 5)*. FGF rate can be increased till there is no rebreathing of gas available to the patient.[1]

***Other uses*:** Gradually rising or decreasing end-tidal carbon dioxide will indicate hypo and hyperventilation respectively. Other uses of capnography include monitoring of patients during transport, to ensure integrity of airway, to confirm and monitor endotracheal tube placement, and to monitor ventilation and circulation by ensuring continuous waveform capnography. Life-threatening airway disasters related to misplacement or displacement of endotracheal tube and ventilator disconnection can be averted by early detection.[18,21]

Capnography can detect significant acute respiratory events, even before changes in oxygen saturation or observed apnoea or hypoventilation, during emergency department procedural sedation and analgesia.[21,23,24] It can detect inadvertent placement of nasogastric tube into trachea.[18,21]

In healthy individuals, there is no alveolar dead space which means there are no nonperfused alveoli. In them, $P(a\text{-}A)CO_2$ equals to $P(a\text{-}ET)CO_2$, which makes end-tidal carbon dioxide concentration ($PETCO_2$) a good estimate of arterial carbon dioxide ($PaCO_2$). In normal individuals, $P(a\text{-}ET)CO_2$ may vary from 2-5 mm Hg, which is an index of gas exchange reflecting resistance to diffusion across alveolar-capillary membrane of CO_2.[1,7,8,25] It increases with age, pulmonary disorders, pulmonary embolism, decreasing cardiac output (CO), hypovolaemia and anaesthesia.

Changes in $PETCO_2$ is usually indicative of change in $PaCO_2$, and thus measuring $ETCO_2$ reduces the need for repeated, difficult and painful, arterial blood gas analysis.[2,3,16,26] This correlation is insignificant in case of abnormal cardiac output and ventilation perfusion ratio.[11]

Lung heterogeneity creates regional differences in CO_2 concentration and sequential emptying causes rise in alveolar plateau. Alveolar dead space represents alveoli with high V/Q ratio, ventilated but not perfused. In pulmonary thromboembolism, sudden vascular occlusion leads to increase in dead space indicated by drop in $PETCO_2$.[7,8,11,18,25] Similarly, in acute lung injury, there is increase in alveolar dead space. PEEP and recruitment open up some of these collapsed alveoli improving ventilation. This is indicated by an increase in $PETCO_2$.[7,8,19] However, if high PEEP causes overdistention of alveoli, alveolar dead space increases, leading to drop in $PETCO_2$.[7,8,19] $PaCO_2$ represents mean alveolar concentration of CO_2, whereas, $PETCO_2$ represents peak alveolar concentration of CO_2 eliminated from lung areas with low V/Q ratio. Difference between $PaCO_2$ and $PETCO_2$ is a reflection of total dead space of lungs. This helps to assess severity of pulmonary disease and evaluate the response to therapy, especially therapies intended to improve dead space to tidal volume ratio.[3,18,19,21]

Bronchospasm and kinking of ETT (at least 50% occlusion of the ETT) causing partial airway obstruction (expiratory obstruction) leads to prolonged phase II, steeper alveolar plateau (phase lll), increased alpha angle (angle between Phase II and Phase III) and increased height of CO_2 waveform (higher $PETCO_2$) *(Figure 4)*.[7,8] Severe bronchospasm causing total airway obstruction, can result in failure to detect $PETCO_2$.[7,8,10,18,21] Therefore, breath-by-breath analysis of exhaled CO_2 concentration monitors alveolar ventilation, pulmonary perfusion and gas exchange, in critically ill mechanically ventilated patient.

In obese patients, capnography demonstrates biphasic emptying and higher $PETCO_2$ than $PaCO_2$. Increases in expiratory flow resistance may reflect a slower expiratory flow with slow accumulation of alveolar CO_2 and alveoli that empty last may have more time for CO_2 diffusion *(Figure 10)*.[13]

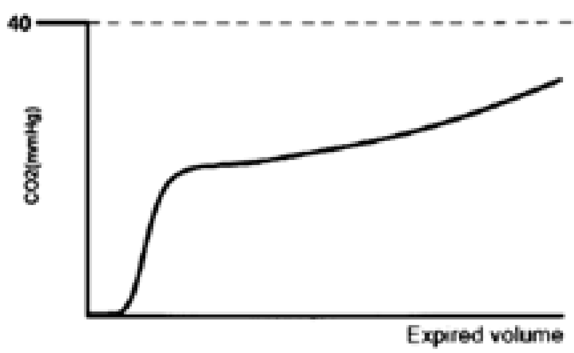

Figure 10: Concave volume capnograph associated with obstructive expiratory flow

In patients with constant ventilation, there is a significant drop in $PETCO_2$ along with decrease in cardiac output. Reduction in CO leads to decreased delivery of CO_2 to the lungs and, consequently, increase in alveolar dead space, resulting in high V/Q ratio.[10,11] Reduced blood flow to lungs can also result from surgical manipulation of heart or thoracic vessels, wedging of pulmonary artery catheter and pulmonary thromboembolism (amniotic fluid, thrombus, fat, gas, or tumour) *(Figure 11)*.[1]A rapid decrease in $PETCO_2$ in absence of changes in blood pressure or central venous pressure of heart rate indicates an air embolism without hemodynamic fluctuations, which can further enlarge in size and lead to further decrease in $PETCO_2$.[1]

$PETCO_2$ is a good indicator of early stages of haemorrhage due to early detection of changes in cardiac output. During early phase of haemorrhage blood pressure can still be normal with substantial amount of blood loss. Capnometry alerts health care provider about tissue hypoperfusion, pulmonary hypoperfusion causing an increase in physiological dead space (alveolar ventilation is same but perfusion reduced drastically) and provides a very useful warning of impending haemorrhagic shock.[25,27,28] Capnometry guided fluid resuscitation helps enhance tissue perfusion without detrimental effects of aggressive resuscitation.

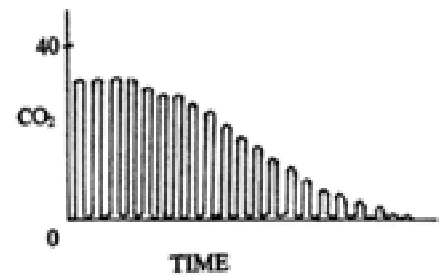

Figure 11: Abrupt decrease in $PETCO_{2,}$ sudden hypotension, pulmonary thromboembolism or circulatory arrest

Sublingual capnometry shows great promise for early recognition of patient at risk of tissue hypoperfusion, hypoxia and shock progression because it avoids disturbance of measurement caused by hyperventilation.[25,29] High gradient between $PslCO_2$ and $PaCO_2$ is associated with impaired microcirculation and a worse prognosis in critically ill patient.[29] Transcutaneous capnography differentiates between hypoxaemia related to V/Q mismatch and hypoventilation, documents correction of nocturnal hypoventilation, and may detect ventilator induced hyperventilation, a possible cause for central apnoea/hypopnoea and glottis closure.[30]

With cardiac arrest, CO_2 produced at tissue level is no longer transported to lungs, where elimination of CO_2 takes place *(Figure 11)*.[11,18,25] During cardiopulmonary resuscitation (CPR), presence of exhaled CO_2 is a better guide to presence of circulation as compared to electrocardiogram, blood pressure or pulse. Consistency of CPR is an important determinant of successful resuscitation, and a continuous waveform capnography helps maintaining consistent chest compressions. If $PETCO_2$ is greater than 10 mm Hg during chest compression, chances of survival and return of spontaneous circulation (ROSC) increases significantly. [4,10,11,14,18,20,31] A sudden increase in $PETCO_2$ during CPR may indicate ROSC *(Figure*

12).[4,10,11,18,20,31] Capnography can thus predict and improve outcome following CPR.

In several cases of cardiac arrest with ongoing CPR, absence of detectable capnography during ventilation was misinterpreted as being due to cardiac arrest, rather than due to oesophageal intubation or ETT obstruction.[32] Capnography is not susceptible to mechanical artefacts associated with chest compression. However, false high values of $PETCO_2$ were noted when epinephrine or sodium bicarbonate was used.[11,18,25]

Any leak in breathing circuit will decrease the minute ventilation, as detected by increase in $PETCO_2$ *(Figure 13)*. Low fresh gas flow, exhausted soda lime and ventilator malfunction may also lead to increase in $PETCO_2$ *(Figure 13)*. However, a leak in sample tubing, circuit disconnection and ventilator malfunction can lead to decrease in $PETCO_2$ *(Figure 14)*.[1,7,8] Contamination of sample expired gas with fresh gas or too high a sampling rate results in loss of alveolar plateau (phase ll) *(Figure 15)*. Leak in sampling tube, during positive pressure ventilation, can lead to peaking of alveolar plateau *(Figure 16)*. Spontaneous breathing attempt in a mechanically ventilated patient, due to wearing off of neuromuscular blockade, produces clefts in capnograph, also known as 'curare cleft' *(Figure 17)*.

The shape of normal neonatal capnograph is different because the dead space is smaller and respiratory rate is higher. Thus, the baseline is shorter, there is a sharper rise in CO_2 concentration and little if any, alveolar plateau.[13] Capnography can also be used in paediatrics to detect leak around an uncuffed ETT and thereby aid appropriate ETT size selection, blockage or kinking of ETT, and endobronchial intubation.[4]

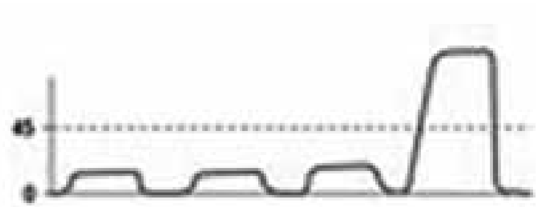

Figure 12: Abrupt increase in $PETCO_2$, suggestive of ROSC

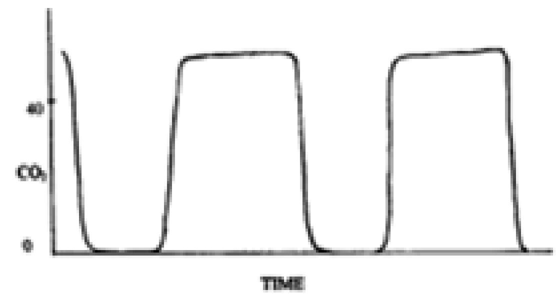

Figure 13: High $PETCO_2$

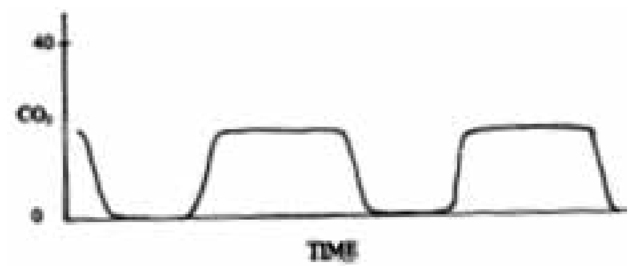

Figure 14: Low $PETCO_2$

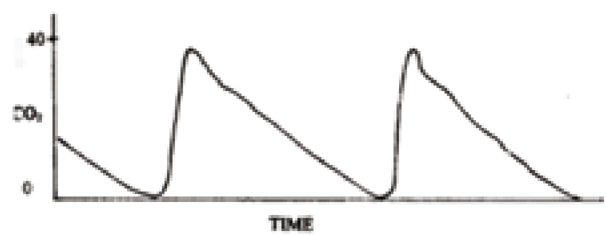

Figure 15: Contamination of sample expired gas with fresh gas or too high a sampling rate

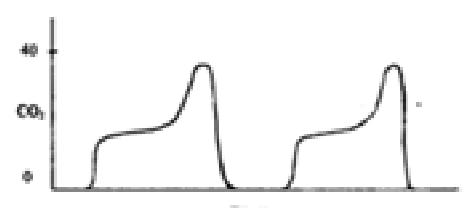

Figure 16: Leak in sampling line during positive pressure ventilation

Figure 17: Spontaneous breathing attempts to breath in mechanically ventilated patient

Table 1: $PETCO_2$ increases

CO_2 production	**Alveolar ventilation**
Fever	Hypoventilation
Sodium bicarbonate	Endobronchial intubation
Venous embolism	Rebreathing
Tourniquet release	Partial airway obstruction
Hyperthermia	**Apparatus malfunction**
Exercise	Leak in breathing circuit
Malignant hyperthermia	Exhausted soda lime
Pulmonary perfusion	Less FGF
Increased cardiac output	Ventilator malfunction
Increased BP	

Table 2: $PETCO_2$ decreases

CO_2 production	Alveolar ventilation
Hypothermia	Hyperventilation
	Extubation
Pulmonary perfusion	Total airway obstruction
Decreased cardiac output	Apnoea/hypopneat
Hypotension	Apparatus malfunction
Hypovolemia	Leak in sample tubing
Pulmonary embolism	Circuit disconnection
	Ventilator malfunction

Table 3:

Sudden increase in $PETCO_2$	**Gradual increase in $PETCO_2$**
Sudden increase in cardiac output (ROSC)	Hypoventilation
Sodium bicarbonate	Increased CO_2 production
Sudden decrease in $PETCO_2$	**Gradual decrease in $PETCO_2$**
Sudden hypotension	Decreased CO_2 production
Sudden hyperventilation	Hyperventilationt
Massive pulmonary embolism	
Occlusion of ETT	
Disconnection or leakage in artificial ventilator system	

Drawbacks

Capnographs, especially the mainstream type add dead space in breathing circuit.[18] They also add to the weight of breathing circuit and can lead to accidental extubation or kinking of ETT. In sidestream capnography, gas sampling rate may sometimes be high enough to trigger a mechanical breath when flow-triggering is used in mechanically ventilated patient.[18]

Infrared spectrum of CO_2 has some similarities to spectra of oxygen and nitrous oxide, hence, a correction factor has to be incorporated into the calibration of capnograph when nitrous oxide is being used.[18] Contamination of the monitor by secretions, high sampling rate, too long a sampling tube, or obstruction of sampling chamber can lead to unreliable results.[18] Reusable mainstream sensors should be subjected to high-level disinfection as per manufacturer's recommendations.[18]

Conclusion

Capnography is a safe, reliable, cost-effective, accurate and noninvasive modality of monitoring, which enhances the patient safety and may help to optimise metabolism, haemodynamics and ventilation in a mechanically ventilated patient. It displays information about CO_2 production at tissue level, its transport to pulmonary circulation, diffusion into lungs and finally, its elimination by alveolar ventilation, in a single graphical representation.[7] A better understanding of various CO_2 curves and dead space is of utmost importance.

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