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Capnography Primer for Oral and Maxillofacial Surgery: Review and Technical Considerations

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Since the mid-19th century the histories of anesthesia and dentistry have been intertwined. However, office-based dental procedures which frequently involve analgesia, conscious sedation, and anesthesia vary from practice-to-practice with regard to respiratory monitoring. In modern oral and maxillofacial surgery, oxygenation is often emphasized over ventilation via usage of pulse oximetry (S_pO_2). A recent Pubmed search using multiple appropriate Medical Subject Headings Terms (MeSH Terms) and following up on related citation trails results in a limited number of topic specific citations dating back to 1987. In this review, we summarize the literature that compares oximetry (S_pO_2 and the less common $P_{TC}O_2$) to ventilatory measurements, specifically capnometry ($P_{ET}CO_2$) in the setting of oral and maxillofacial surgery and provide a comprehensive primer on the technological and respiratory considerations essential for a practitioner attempting to incorporate ventilatory monitoring.

Anderson et al. [1] studied the combination of a capnograph (expired carbon dioxide (CO_2) monitor) and a transcutaneous oxygen monitor ($P_{TC}O_2$) as a non-invasive system for monitoring of respiratory function in 10 ASA class I patients undergoing general anesthesia for removal of third molars. They concluded that the continuous display of the measured end tidal volume CO_2 ($P_{ET}CO_2$), which the author's state was measured via nasal prong sampling, proved to be a sensitive and accurate method for detecting apnea and airway obstruction. They further state that all episodes of apnea or obstruction were immediately detected as a dramatic decrease in the expired CO_2 level and that the $P_{ET}CO_2$ values served as useful indicators of hypoventilation. Interestingly, during steady-state conditions of respiration, $P_{TC}O_2$ correlated well with simultaneously measured partial pressure of oxygen in arterial blood (P_aO_2) measured by blood gas technology. However, during any period when oxygenation was rapidly changing (step increase in FIO_2, step decrease in FIO_2, or apnea) the $P_{TC}O_2$ lagged behind changes in P_aO_2 even after a five-minute equilibration period, thereby not accurately reflecting the true state of oxygenation. They concluded that transcutaneous oxygen monitoring does not appear to be optimal as a respiratory monitor in the setting of ultralight general anesthesia where rapid, critical changes in oxygenation must be detected without delay [1]. Based on a randomly enrolled blind study of fifty-five patients, Bennet et al. [2] stated that in patients with nasal ventilatory exchange rates maintained throughout anesthesia, sampling of nasal $P_{ET}CO_2$ was an effective way to monitor ventilation status or the respiratory system function. Respiratory depression or obstructive changes in ventilation were detected by capnography with a high sensitivity and

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low positive predictive value in detecting oxygen desaturation. They state that current technology did not show a clinically significant correlation between $P_{ET}CO_2$ and oxygen saturation as estimated by pulse oximetry (S_pO_2), however a combined increase in $P_{ET}CO_2$ and decrease in respiratory rate suggested a trend of decreasing oxygen saturation [2].

A clarification of terminology regarding both oxygen and carbon dioxide monitoring is appropriate and helpful at this point. Arterial oxygen tension (P_aO_2) is measured in a blood gas machine by an electrochemical cell (Clark polarographic electrode). Arterial carbon dioxide tension (P_aCO_2) is measured in a blood gas machine by a secondary sensor composed of a pH sensitive glass electrode in an electrolyte cell. Carbon dioxide diffuses through a membrane into the cell, reacts with water producing carbonic acid thereby changing the pH [3]. Transcutaneous oxygen partial pressure ($P_{TC}O_2$) was introduced in 1972 and is measured by a heated electrode applied to the skin surface. It must be understood that there is a difference between $P_{TC}O_2$ and pulse oximetry (S_pO_2) methodology which utilizes light absorbance based on the Beer Lambert Law. Transcutaneous carbon dioxide partial pressure ($P_{TC}CO_2$) was introduced shortly after $P_{TC}O_2$ in the early 1970s. Obtained $P_{TC}CO_2$ values are expected to be higher than P_aCO_2 because of the higher CO_2 concentration in the tissues and the heated sensor increases skin metabolism- raising CO_2 production. The heated sensor improves the correlation with P_aCO_2. Capnography, $P_{ET}CO_2$, on the other hand, utilizes the same Beer Lambert Law technology as is used in oximetry [3]. In the case of oximetry the technology is applied transcutaneously while in capnography it is applied to the expired gas flow.

In making a case for capnographic monitoring as a standard of care in patients undergoing sedation, Vascello and Bowe [4] concluded that it is difficult, if not impossible to justify not performing capnography on every patient undergoing sedation. The authors point out that in spite of many potential problems with capnography such as dislodged sampling catheters, diversion of exhaled gases by mouth breathing and obstruction of sampling ports by tissues or secretions, it is most useful in the very setting in which it is also the most accurate-the heavily sedated patient [4]. Bennett [5] supports the alternate argument by concluding that while there is no doubt that the use of capnography in the dental anesthesia model can yield additional information about the ventilatory status of the patient; the inappropriate use and interpretation of data from such technology may confuse the anesthetist. He states that it cannot be assumed that more expensive equipment than what is traditionally used will provide for simpler and safer anesthesia. Bennett concludes that although capnography "facilitates anesthetic monitoring, it has been demonstrated neither to decrease the incidence of hypoxic events nor to optimize anesthetic care". Bennett also states in his conclusion that "the data demonstrate that it is an acceptable technique to monitor ventilation, however, it cannot currently be recommended as a standard for ventilatory monitoring in the anesthetized dental patient" [5].

A recent editorial by Weaver [6] in Anesthesia Progress can be interpreted to send a rather mixed message to those who administer anesthesia in an outpatient setting. While he points out clearly that the American Association of Anesthesia (ASA) has amended its Standards for Basic Anesthesia Monitoring to include mandatory exhaled endtidal carbon dioxide ($P_{ET}CO_2$) monitoring during both moderate and deep sedation to its existing requirement for endotracheal and laryngeal mask airway general anesthesia, it stresses that the decision as to whether dentistry must follow this mandate is up to dentistry [6]. The 2010 House of Delegates of the ASA amended their standards to become effective as of July 2011 based on a consensus of the ASA Committee of Standards and Practice Parameters, rather than the preferable highest level of evidence-based scientific data (Class A, Level 1). In effect, however, the mandate by the ASA has extended the use of capnography to such settings as hospital OMFS clinics and by logical extension to offices in the private setting also. It must

not be lost in the "politics" regarding this issue that obviously the time has come to consider $P_{ET}CO_2$ use in OMFS anesthesia settings as standard of care. Pulse oximetry is currently considered mandatory for general anesthesia in OMFS and while it reflects the status of oxygenation of the patient directly, it does not help in establishing a differential diagnosis of hypoxia nor does it enable one to take measures in an expeditious enough fashion prior to hypoxia resulting in irreversible changes. The clinical indications for measurement of CO_2 concentration in the exhaled breath are multiple and varied. Beside routine use in the operating room for patient monitoring, Whitaker [7] suggests that capnography has a vital role in many other patient care settings. The critical care setting, resuscitation scenarios [8], critical patient transfer situations, the immediate postoperative recovery period, procedural sedation, neonatal resuscitation [9], emergency medicine and the treatment of respiratory diseases are examples he cites [7].he

Technology of $P_{ET}CO_2$ Measurement

The capability of measurement of CO_2 in the expired breath of a patient has been an important technological advance in medicine. The partial pressure of CO_2 in arterial blood is an indicator of the equilibrium of CO_2 production and elimination and its determination provides valuable data in regards to metabolism and cardiopulmonary pathophysiology [10]. Arterial blood CO_2 measurement requires an arterial sample, transport to a lab and a blood gas determination by chemical means, while monitoring CO_2 in the breath provides a point of care, continuous and non-invasive estimate of the arterial CO_2. An understanding of the basic methodology by which $P_{ET}CO_2$ in determined will assist in an understanding of this important monitoring modality. CO_2 is a polyatomic asymmetric gas molecule that avidly absorbs light in the Infrared (IR) part of the spectrum. The wavelength of IR rays exceeds 1.0 nanometer (nm) while the visible spectrum of light is between 0.4 and 0.8 nm. IR rays are absorbed by polyatomic gases (non-elementary gases such as nitrous oxide (N_2O), CO_2, and water vapor). CO_2 selectively absorbs IR light at 4.3 nm. The concentration of CO_2 in a gas sample can be measured by shining IR light on the sample and comparing the intensity of the light that passes through the sample with the original intensity. The light intensity is reduced as it passes through the sample in proportion to the CO_2 concentration present in the sample. The essentials of an IR analyzer are: (a) a source of IR radiation of with an emission spectrum that includes the absorption bands of the gas to be analyzed; (b) a sample cell with windows having suitable transmission properties; (c) an optical or gas filter to limit the range of wavelengths measured by the detector; (d) a physical or electronic means to modulate the IR radiation from the source; and (e) a detector (thermal or photonic) to convert the IR radiation into an electrical signal [10]. The terms capnography and capnometry are usually considered synonymous however, capnometry suggests measurement (i.e., analysis alone) without a continuous written record or waveform (capnography). Two terms associated with capnography which can benefit from clarification include side-stream and mainstream capnography. In side-stream capnography, the CO_2 sensor is located away from the airway and a pump aspirates samples from the patient's airway through a capillary tube which is transmitted to the main unit. The sampling tube is connected to a T-piece inserted at the endotracheal tube or anesthesia mask connector. A side-stream unit also allows for monitoring of non-intubated patients by sampling from the nasal cavity by nasal adaptors of many different varieties (Figure 1). Capnography units utilized in office and outpatient setting are sidestream devices. In main-stream capnography devices the sample cell is inserted directly between the breathing circuit and the endotracheal tube. An infrared sensor is attached to the adaptor which emits IR light through the adaptor to a photodetector on the opposite side of the adaptor. The light which reaches the photodecetor is used to measure the $P_{ET}CO2$. This technology eliminates the need for gas sampling and results in crisper waveforms reflecting real-time $P_{ET}CO_2$ in the patient's airway (Figure 2). It should be mentioned as a compliment to the information previously

presented here that a non-quantitative, colorimetric $P_{ET}CO_2$ detector may be useful to confirm ventilation during prehospital CPR using laryngeal mask airway or face mask, as well as for patients who have undergone tracheal intubation.

Physiology and Mechanics of Respiration and Ventilation

An understanding of the physiology and mechanics of both respiration and ventilation is an essential basis for those who practice anesthesia in any setting. What follows is a review of these topics to set the stage for a better comprehension of the necessity and particulars of S_pO_2 and $P_{ET}CO_2$ monitoring. Respiration is the exchange of gas and it occurs externally at the level of the lungs and internally at the level of the cells. Physiologic (external) respiration includes pulmonary ventilation (movement of air in and out of the lungs) as well as gas exchange in the alveoli, gas transport in the blood and gas exchanges between blood and tissues. Cellular (biochemical) respiration is the energy generation an organism produces by reacting oxygen with glucose to produce water, carbon dioxide and Adenosine Triphosphate (ATP). Physiologic respiration is essential to sustain cellular respiration but the processes must be considered as distinct with physiologic respiration concerned with the flow of metabolites to and from the organism while cellular respiration occurs in the individual cells of the organism.

About 98-99% of the total oxygen content of arterial blood is bound to the hemoglobin of the red cells and the remainder is dissolved in the plasma. It is the dissolved oxygen in the plasma that is responsible for the production of a gas pressure or arterial oxygen tension (P_aO_2). The oxygen in the plasma is unbound and it is this which is the substrate of cellular respiration. As the oxygen enters the cells from the plasma it is immediately replaced by the release of hemoglobin bound oxygen. There is a non-linear relationship between the degree of saturation of hemoglobin with oxygen (S_aO_2) and the P_aO_2 which is best illustrated by the classic oxygen-hemoglobin dissociation curve (Figure 3). The amount of oxygen bound to hemoglobin at any one time is related to the positive pressure of oxygen to which the hemoglobin is exposed. In the lungs, where alveoli and capillaries interface, the P_aO_2 in blood is high and oxygen readily combines with hemoglobin (S_aO_2 is also high). Under normal circumstances, hemoglobin in arterial blood is ~98% saturated and the P_aO_2 is about 95 mm Hg. As the blood circulates throughout the body and areas where the oxygen partial pressure is decreased are encountered the hemoglobin releases its oxygen. This occurs because hemoglobin cannot maintain its capacity for oxygen in the presence of lower P_aO_2 in the plasma (Haldane Effect) due to increased cellular respiration at the tissue level. At the tissue level, after oxygen is delivered from the arterial blood, the oxygen tension declines to ~40 mm Hg and the hemoglobin saturation drops to ~70-75%, the normal values for venous blood. Hemoglobin saturations of 95% and above sustain the P_aO_2 at or above 80 mm Hg which prevents hypoxia from occurring. Above 95% S_aO_2 the oxygen-hemoglobin dissociation curve becomes quite flat as the hemoglobin saturation and partial pressure of O_2 in the blood approach equilibrium. When the S_aO_2 falls to below 90% the dissociation of oxygen from hemoglobin occurs at lower P_aO_2 giving rise to the steep transition in curve. There are several variables which one should be aware of which will shift the oxygen-hemoglobin dissociation curve to the right or left. The P_{50} is the oxygen tension at which hemoglobin is 50% saturated (normal P_{50} is 26.7 mm Hg). A shift to the right increases the P_{50} and lowers the affinity of hemoglobin for oxygen making it more easily available to tissues (acidosis, exercise, sickle cell disease/trait, hemorrhage). A shift to the left (fetal hemoglobin) decreases the P_{50}, and increases the affinity of hemoglobin for oxygen making it less available to tissues (Table 1).

The effect of an increase or decrease of temperature on the oxygenhemoglobin dissociation curve is rarely significant in the range of 36-38°C. Extreme hyperthermia causes a rightward

shift, while hypothermia causes a leftward shift. 2,3-Diphosphoglycerate (2,3-DPG) is an organophosphate, which is an end-product of erythrocyte metabolism. The production of 2,3-DPG increases in the presence of decreased peripheral tissue O_2 availability, such as in chronic lung disease, anemia, hypoxemia and congestive heart failure. High levels of 2,3-DPG shift the curve to the right (children, hypoxia, chronic lung disease) assisting in the unloading of oxygen to the peripheral tissues. Low levels of 2,3-DPG (banked blood, septic shock, and hypophosphataemia) cause a leftward shift of the curve interfering with the unloading of oxygen [11]. Most of the effect of the P_aCO_2 on the oxygen-hemoglobin dissociation curve is due to its effect on the intracellular pH (the Bohr effect), but also the accumulation of CO_2 causes the generation of carbamino compounds which combine with the terminal amine groups in hemoglobin to form carbaminohemoglobin. Low levels of CO_2 decrease carbamino compound formation and have the effect of shifting the curve to the right while high levels are associated with a shift to the left. In fact little of the total CO_2 content of blood is transported as carbamino compounds and overriding the small effect of that which does is the fact that 80-90% of the CO_2 of blood is transported in the form of bicarbonate ions. Thus, increased CO_2 creates a respiratory acidosis by releasing a proton into the plasma shifting the oxygen-hemoglobin dissociation curve to the right overall [12].

Hemoglobin binding of Carbon monoxide (CO) is 200-250 times greater than that with which it binds oxygen. CO bound hemoglobin (carboxyhemoglobin) impairs peripheral tissue oxygenation by directly reducing the amount of hemoglobin available for oxygen binding, causes the oxygen bound to the remaining normal hemoglobin to be more resistant to release and it lowers the P50 shifting the oxygen-hemoglobin dissociation curve to the left [12]. Changes in the hemoglobin affinity for oxygen with variations in intracellular hydrogen ion concentration are known as the Bohr Effect. Decreases in the pH shift the curve to the right; increases in pH shift the curve to the Left [12]. If one considers the changes in CO_2 and hydrogen ion levels in both the lung and peripheral tissues an understanding of the effect of pH on the oxygen-hemoglobin dissociation curve is facilitated. In the peripheral tissues where metabolism is occurring, the CO_2 produced diffuses into the circulation via the capillaries. As the P_aCO_2 increases in the tissue hydrogen concentration increases, tissue pH falls and hemoglobin's affinity for oxygen decreases, releasing oxygen to the tissues. In the lung elimination of CO_2 reduces P_aCO_2 resulting in a rise in pH increasing the affinity of hemoglobin for oxygen.

There are many genetic variations of the hemoglobin molecule, but most of them have unaltered affinity for oxygen. A notable exception to this is fetal Hemoglobin (HbF) which had two gamma (γ) chains instead of the β chains which characterize adult hemoglobin (HbA [P_{50}∼27 torr]). The oxygen-hemoglobin dissociation curve for HbF (P_{50}∼ 20 torr) is shifted to the left, possibly due to the fact that 2,3-DPG fails to bind to the γ chains. This causes the oxygen affinity of HbF to be high of setting the effect of low P_aO_2 associated with fetal life. Placental uptake of oxygen is enhanced by the increased avidity of HbF for oxygen [12]. In the case of sickle cell disease/trait the associated hemoglobin (HbS) has a P_{50} of about 30 torr and thus has a lower avidity for oxygen resulting in the ischemic sequel of this disease process.

Ventilation is the exchange of oxygen for CO_2 in the alveoli of the lungs. Pulmonary ventilation refers to the total exchange of gas while alveolar ventilation refers to ventilation within the alveoli. At rest, the lungs take in about 4 L/min of air (ventilation) while at the same time they are being perfused with about 5 L/min of blood (perfusion). With maximal exercise this flow can increase to 100 L/min ventilation and 25 L/min perfusion. At either end of this extreme the gas and blood are directed to within 0.2 μm of each other in the alveoli where oxygen is supplied for tissue metabolism and the major by-product of that metabolism, CO_2, is exchanged for removal. The lungs are able to ventilate largely without

conscious control while maintaining P_aO_2 within a 5% range [13]. The parenchyma of the lung is thin yet is able to maintain itself over a very large surface area. Collagen and elastin fibers make the lung a very elastic organ anatomically as well as physiologically.

During spontaneous ventilation, air enters the lung when the intrathoracic pressure is reduced below atmospheric pressure by chest wall expansion. The volume of air which enters the lung in a breath depends on the change in pressure in the lungs (pleural pressure) and compliance. Compliance is an intrinsic elasticity that relates a change in volume to a change in pressure. The chest wall and the lungs are both compliant and both contribute to the overall compliancy of the respiratory system. Within the physiologic range, the compliance of the chest wall does not change significantly with thoracic volume while the lung compliance varies in an inverse fashion to the lung volume. Functional Residual Capacity (FRC) is the volume of air present in the parenchymal tissue of the lungs at the end of passive expiration (resting lung volume). At FRC, the elastic recoil forces of the lungs (the tendency to collapse inward) and chest wall (the tendency to spring outward) are equal but opposite and there is no exertion either by the diaphragm or any of the accessory muscles of respiration. Total Lung Capacity (TLC) is the volume point at which the muscles of respiration cannot overcome the elastic recoil of the lungs and chest wall. Residual Volume (RV), the amount of gas remaining in the lung at the end of maximal expiration, is the point where the muscles responsible for expiration cannot overcome the elastic recoil of the chest wall. The difference between TLC and RV is the Vital Capacity (VC). Compliance is calculated by taking the slope of these pressure-volume relationships at a specific volume. It is evident by study of the interaction of the pressure-volume properties of the lungs and chest wall that compliance is greater at low lung volumes, and falls considerably above two-thirds of vital capacity. At FRC, the lungs are very compliant (about 200 ml/cm H_2O) so a pressure reduction of 5 cm/H_2O in the pleural space will draw a 1L breath or tidal volume (V_T). V_T (4-6 ml/kg of ideal body weight at rest) is defined as the volume of gas inhaled and exhaled in one respiratory cycle (Figure 4) [13].

The tendency of a structure which can be deformed to return to its baseline shape is termed its elastic recoil. The shape and structure of the thoracic cage determines the elastic recoil of the chest wall. The tissue elasticity of the lungs and the forces needed to change the shape of the air-liquid interface of the alveolus determine the elastic recoil of the lungs. The expansion of the lungs requires overcoming the surface tension at the air-liquid interface of the alveolus. Surface tension is created by the greater attraction between molecules of a liquid to each other than to molecules of that liquid to an adjacent gas. At the air-liquid interface of the lung alveolus molecules of water are more strongly attracted to each other than to the gas in the alveolus. This creates a net force which draws water molecules together in the plane of the interface. Since the force is stretched over a curve in the case of the alveolus those forces tend to collapse the curve. Laplace's Law states that the pressure needed to keep a curve open (a sphere in the case of the alveolus) is directly proportional to the surface tension at the interface and inversely proportional to the radius of the sphere (Figure 5) [13]. To reduce the surface tension which is tending to collapse the alveolus a hydrophibic molecule, surfactant, a mixture of phospholipids (predominately Dipalmitoyl Phosphatidycholine [DPPC]) and proteins, displaces water molecules from the air-liquid interface. This surface tension reduction diminishes the elastic recoil pressure in the lungs reducing the pressure required to inflate them thus reducing the work of breathing. It also allows surface forces to vary with alveolar surface area promoting alveolar stability and preventing atelectasis. Surfactant also limits the reduction of hydrostatic pressure in the capillary interstitium caused by surface tension and reduces the forces which would promote transudation of fluid from the circulation accumulating in the pulmonary interstitium (edema). Pathologic states can be caused by changes in lung compliance. An increase in compliance is seen in emphysema. A decrease in compliance is seen in pulmonary fibrosis

or a disruption of surfactant with increases in surface tension forces as is seen respiratory distress syndrome of the newborn [13].

Monitoring of Oxygenation

Prior to the Second World War, a methodology for noninvasive continuous measurement of S_aO_2 was researched by military aviation without success [14]. The first successful quantitative S_aO_2 monitor was developed by Wood and Geraci in 1949 for use in experimental physiologic laboratories [14-16]. The principle of pulse spectrophotometry was conceived by Aoyagi in 1972 and applied to a pilot model of a pulse oximeter in 1974 [14]. Aoyagi realized serendipitously during an experiment to develop a dye-dilution technique to measure cardiac output that untoward changes in tissue light absorption caused by the pulsatile nature of arterial blood flow could be used to compute oxygen saturation. The “noise” of his primary experiment became the “signal” of a secondary application which lead to the development of the first “pulse’ oximeter in 1974 [14-17]. In 1984 Nellcor® (Covidien-Dublin, Ireland) put a pulse oximeter on the market and over the next several years the use of the instrument rapidly spread worldwide and has been adopted into standard anesthesia practice in many countries [14]. Over the years pulse oximetry has greatly improved due to advances in light emission and signal processing technologies leading to widespread application throughout medical practice [17,18]. Even with the aforementioned advances there remain many limitations which must be taken into consideration. These limitations include motion artifacts, poor perfusion at the measurement site, arrhythmia interference, ambient light or electromagnetic interferences, skin pigmentation variations, nail polish, probe positioning, time lag in detecting hypoxia, venous pulsations (tricuspid regurgitation and hyperdynamic circulation states), intravenous dyes, and abnormal hemoglobin species [17]. Pulse oximetry does not provide any information about the level of hemoglobin of the patient monitored nor does it indicate the efficiency of oxygen delivery to the tissues or the adequacy of ventilation. Although oxygen saturation has been proposed as the “fifth vital sign” [19-21] clinically relevant principles and inherent limitations of pulse oximetry are poorly understood by healthcare professionals [19,22]. Fouzas et al. [22], in a recent multiple choice questionnaire study looking at pediatric healthcare providers combined practical and theoretical knowledge of pulse oximetry found a mean test score of ∼62%, indicating a limited overall understanding of the method. When the test results were segregated into those which assessed theoretical issues a factor score of ∼44% was obtained while in those that assessed practical knowledge a factor score of ∼83% was obtained. Participants in the exam who worked in ICU settings and in higher level hospitals had a greater likelihood to score higher than the 75^{th} percentile of the total score and the 75^{th} percentile of the score for theoretical knowledge. Regarding practical knowledge no differences were noted in the likelihood of scoring better regardless of the care setting of the participant. Experience was positively related to the ability to score better on questions assessing practical knowledge, but negatively related to the ability to score better on those questions assessing theoretical knowledge [22]. The need for improved education techniques for healthcare providers who use pulse oximetry is evident as several studies show poor understanding and inadequate training is prevalent [19,22-27].

It must be understood that pulse oximeters measure only S_aO_2 and unless the relationship of S_aO_2 to P_aO_2 (oxygen-hemoglobin dissociation curve (Figure 3)) is known by the practitioner utilizing the monitor the reading is clinically meaningless. It is not important to know what the S_aO_2 is as a measure of the saturation of hemoglobin with oxygen, but rather what the P_aO_2 value it reflects is. Once the hemoglobin is fully saturated (P_aO_2 of about 120 mm Hg) no change in saturation reading will be noted. Significant changes in P_aO_2 are difficult to detect via S_aO_2 readings at high levels of oxygenation. A change in P_aO_2 from 100 mm Hg to 75 mm Hg will have minimal effect on the saturation values [28]. The

physiology of the oxygen-hemoglobin saturation curve and electronic delays in pulse oximeters mean that the events one wishes to monitor and prevent from occurring (e.g., obstruction, hypoventilation, apnea) can be going on for 2-3 minutes or longer while the S_aO_2 continues to register in the acceptable range. Only when hypoxia becomes severe and the S_aO_2 falls precipitously (reflecting a severe drop in P_aO_2) does the monitor reflect the gravity of the situation with its reading and low limit alarms. This problem is magnified if supplemental oxygen is administered to the patient (Figure 6) [29]. If one is dependent on a pulse oximeter to reflect adequate ventilatiory exchange of a patient, a misconception which has been shown to be quite prevalent, then valuable time is lost in recognition and correction of hypoxia. S_aO_2 monitoring is not actually “real time” nor does it provide clinically useful data to detect hypoxia in a manner timely enough to be beneficial to an anesthesia provider.

Monitoring of Carbon Dioxide

The non-invasive monitoring of carbon dioxide has become more prevalent in the last several years culminating in its inclusion as “Standard of Care” by the ASA in July 2011 for monitoring during both moderate and deep sedation in addition to its existing requirement for endotracheal and laryngeal mask airway general anesthesia. Capnography, as opposed to pulse oximetry which is a direct monitor to reflect the S_aO_2 only, is an indirect monitor which assists in the differential diagnosis of hypoxia by providing information about CO_2 production, pulmonary perfusion, alveolar ventilation, respiratory patterns, and the elimination of CO_2 from the anesthesia circuit. Capnography has been shown to be effective in the early detection of adverse respiratory events enabling remedial measures to be taken to reverse hypoxia before it results in irreversible brain damage. In a study of adults requiring procedural sedation in an emergency department setting Miner et al. [30] attempted to determine if capnography monitors can detect Respiratory Depression (RD) and the level of sedation as measured by the Observer's Assessment of Alertness/Sedation (OAA/S) scale. They found that no correlation between OAA/S and $P_{ET}CO_2$ could be determined making capnography an unreliable monitor of level of sedation. They did find that about 46% of their patients (thirty three) exhibited RD and eleven patients required assisted ventilation during their procedures. Pulse oximetry detected eleven of thirty three patients with RD. Post hoc study of their data showed that all patients with RD had a $P_{ET}CO_2$ >50 mm Hg, an absent waveform, or an absolute change from baseline in $P_{ET}CO_2$ of 10 mm Hg. The authors felt that using the three criteria above may detect subclinical RD not detected by pulse oximetry and add to the safety of parenteral sedation by quickly detecting RD not detected by pulse oximetry [30]. In the United Kingdom, utilizing an audit format, Cook et al. [31,32] found that in the years 2008-9 that there were 16 airway related deaths in the 3,000,000 patients undergoing general anesthesia monitored with continuous capnography, giving a death rate of 1:180,000. The similarly found that there were 18 deaths from a much smaller number of ICU patients (48,000) who were being artificially ventilated but not monitored with continuous capnography, giving a death rate of 1:2,700. The implication herein is that it is 66 times more likely to have airway catastrophes in the ICU where capnography was not used as compared to the operating room where continual capnography is the standard of care [6,31-33].

Primer on Capnography

The basics of capnographic waveform interpretation will be reviewed to acquaint the practitioner with the anatomy of the normal wave and clinically significant abnormal wave forms. The normal capnograph (Figure 7) shows that expiration occurs along the line from A to D as represented by: exhalation of the dead space A-B; exhalation of the lower airway B-C; exhalation of the alveoli C-D with end tidal CO_2 recorded at point D; inspiration occurs

from points D to E. The capnogram allows for the continuous assessment of the depth and frequency of each ventilation.

Hypoventilation produces a reduction in the number of waves as the rate slows. There are increasing end tidal CO_2 levels and the waveform still has a normal look (Figure 8). This pattern is seen in patients with decreased respiratory drive due to narcotics, CNS depression, or heavy sedation. Hypoventilating patients are not breathing at a rate fast or deep enough to completely remove CO_2 from the lungs and increasing end tidal CO_2 levels may occur. Assisting ventilations with a Bag Valve Mask (BVM) device and administering a reversal agent, such as naloxone or flumazenil will help improve ventilation and reduce CO_2 levels.

In hyperventilation, the capnograph starts as normal but when the rate increases waveforms become closer to each other and the level of end tidal CO_2 decreases (Figure 9). The most common cause of hyperventilation in the intubated patient is over zealous manual ventilation. When decreasing end tidal CO_2 levels are noted, simply slowing the rate in which the patient is being ventilated until end tidal CO_2 levels return to normal is often the treatment of choice. In the spontaneously breathing patient, increasing respiratory rate and decreasing end tidal CO_2 levels can be a sign of hyperventilation syndrome in which case the treatment would involve increased sedation levels or perhaps additional local anesthesia. Hyperventilation accompanied by a prominent S wave in lead I, Q waves, and inverted T waves in lead III on the ECG (“S1Q3T3”) (Figure 10) should raise the suspicion of acute pulmonary embolism.

The disappearance of a normal alveolar plateau replaced by a more upward sloping line is an indication of incomplete or obstructed exhalation. This waveform often will resemble a “shark fn” showing that exhalation is being slowed (Figure 11). Such a pattern is noted in states of bronchoconstricion. Common causes include asthma, COPD, or an airway obstruction. In extremely severe bronchospasm no exchange takes place and $P_{ET}CO_2$ may not be detectable at all. Aggressive tracheal suctioning and bronchodilators are utilized in such situations. Bronchospasm can be quite difficult to recognize and treat and frequently patients with this entity will require placement of an advanced airway for adequate management.

The capnogram of apnea is indicated by the sudden loss of a waveform indicating that no CO_2 is present (Figure 12). If an intubated patient demonstrates such a waveform there is a problem with the airway (displaced or obstructed). Treatment is a quick assessment of the airway and if doubt about the location or patency of the airway is discovered, the airway should be removed, the patient ventilated by BVM ventilation and the airway subsequently reestablished. In the spontaneously breathing patient such a capnogram indicates that the patient is most likely in respiratory arrest. If the patient is still breathing with the apnea pattern on the capnogram the equipment must be rapidly assessed. Treatment for apnea is establishment of a patent airway immediately followed by BMV ventilation and rapid assessment of the cause of the cessation of breathing.

Elevation of the baseline of the capnogram is an indication that incomplete inhalation and/or exhalation is occurring (Figure 13). If CO_2 is not being completely washed out during inhalation air trapping in patients with a history of asthma or COPD should be suspected. Elevation of the base line can also occur when there is a malfunction in the exhalation valve of the BVM or ventilator. Increasing expiratory time will help remove excess CO_2 in patients who are experiencing air trapping.

Summary

The purpose of this review is to point out the paucity of information in the current Oral and Maxillofacial Surgery concerning end tidal CO_2 monitoring. A very brief review of the basic technology of the modality is presented followed by a look at the physiology and mechanics of respiration and ventilation which stresses the importance of an understanding of this topic for those who practice anesthesia. The monitoring of oxygen saturation and its serious limitations are presented to serve as a point of comparison to the much more accurate and clinically applicable monitoring of ends tidal CO_2. The roles of capnography in other fields of medicine are presented and the emergence of $PETCO_2$ as the "standard of care" according to the ASA is offered for serious consideration by OMFS practitioners. A very basic primer on capnography completes the review in the hopes that it will serve as the foundation on which the prudent practitioner will build his knowledge basis of this life saving technology. The current dependence on pulse oximetry as a monitoring modality must be replaced with the much more effective $P_{ET}CO_2$ devices in a timely manner. $P_{ET}CO_2$ is the monitor of choice in the outpatient anesthesia setting and the delay in adaptation of this modality in the OMFS practice setting will compromise safe anesthesia delivery as long as it persists.

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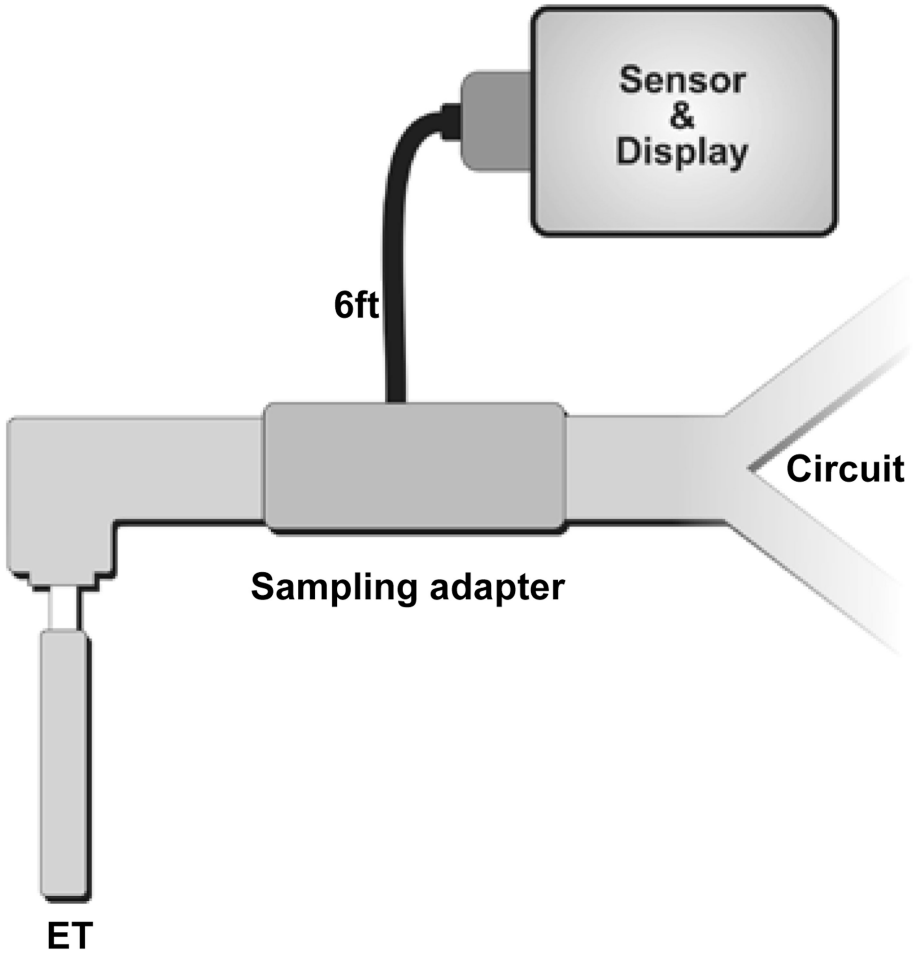

Figure 1.
Side-stream capnography with the sensor connected by tubing to the breathing circuit.

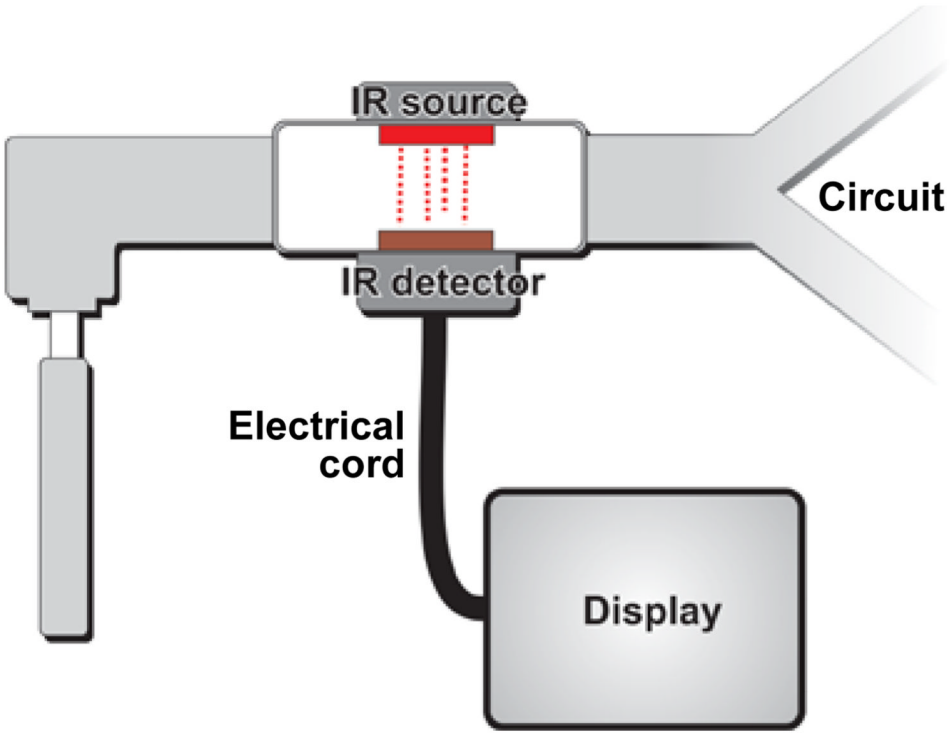

Figure 2.
Main–stream capnography with the sensor inserted directly into breathing circuit.

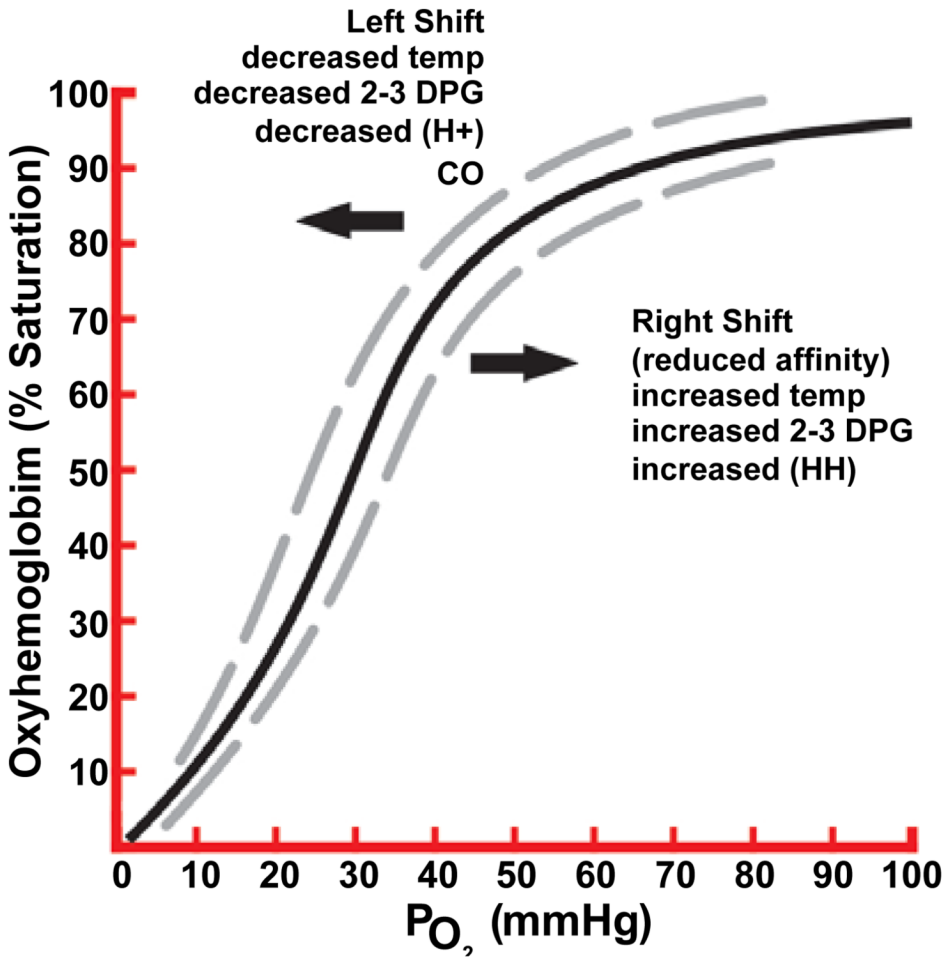

Figure 3.
Oxygen-hemaglobin dissociation curve.

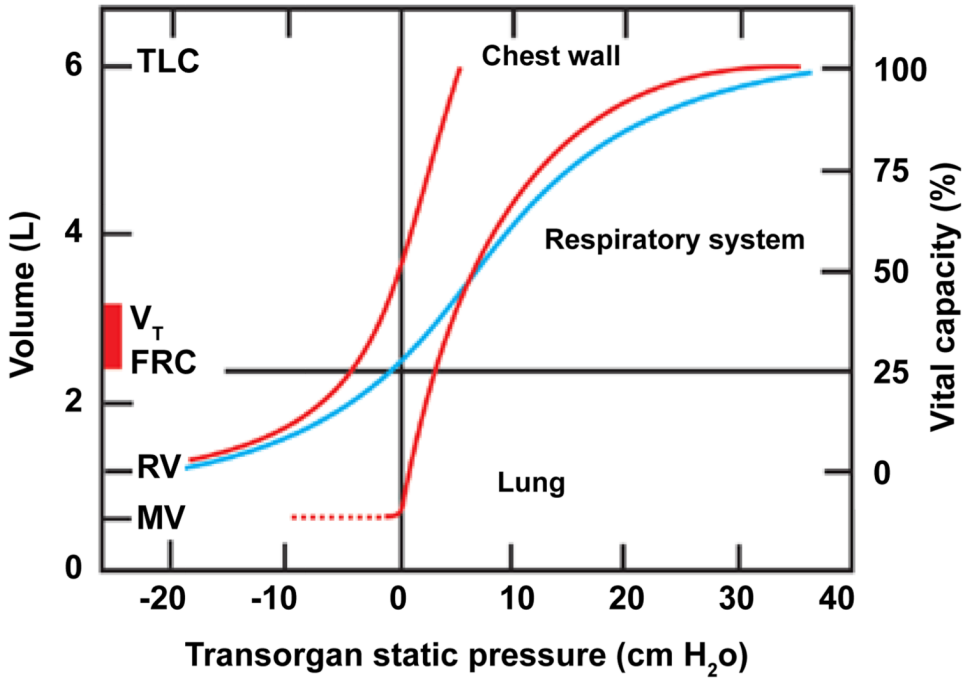

Figure 4.
Pressure-volume curves of the lungs and chest wall.

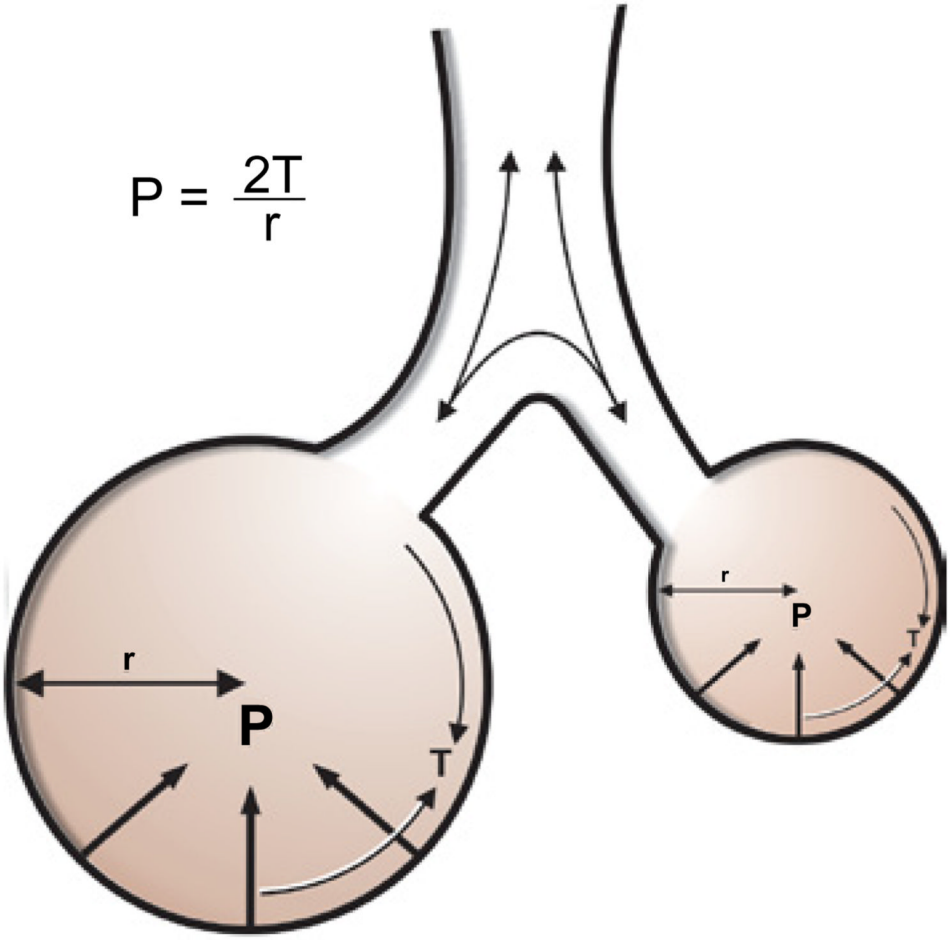

Figure 5.
Law of Laplace and alveolar collapse.

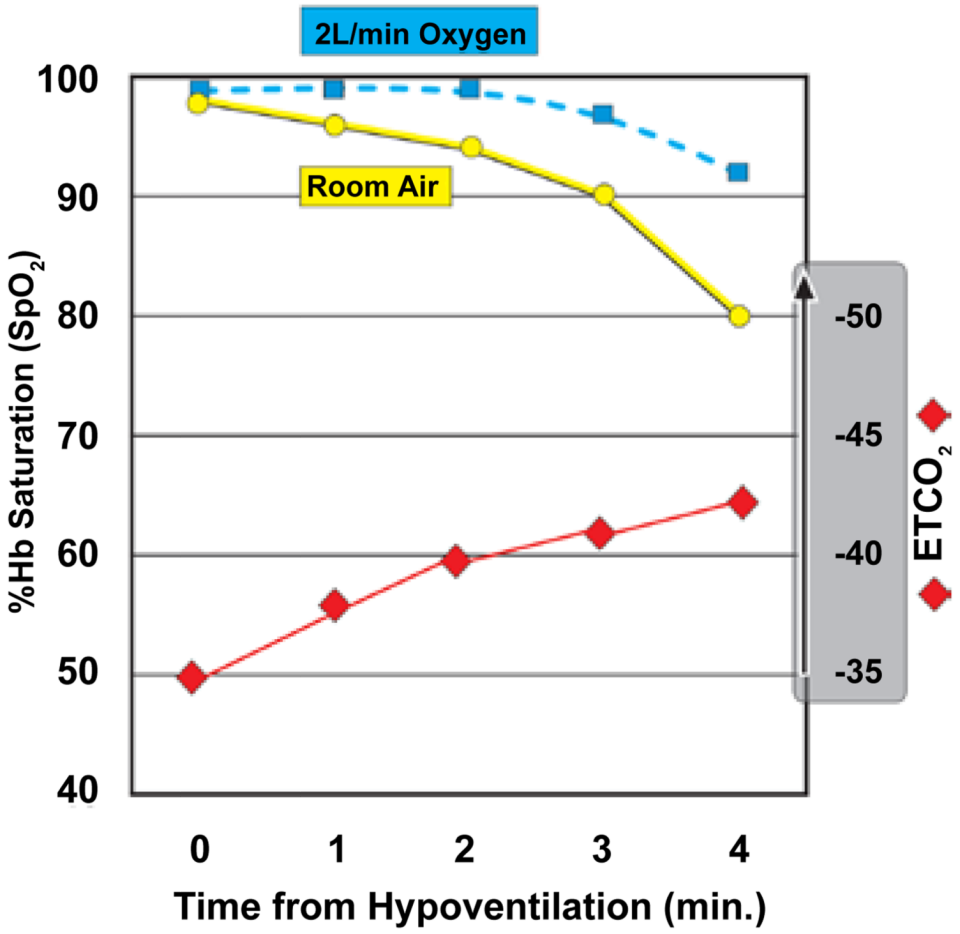

Figure 6.
Illustration of capnography as the purest form of the measure of hypoventilation. The top two tracings demonstrate pulse oximetry readings for patients on room air and supplemental oxygen at 2L/min. Note that the top tracing (supplemental oxygen) gives no warning of hypoventilation while the tracing for patients breathing room air declines much more in coordination with the capnography reading.

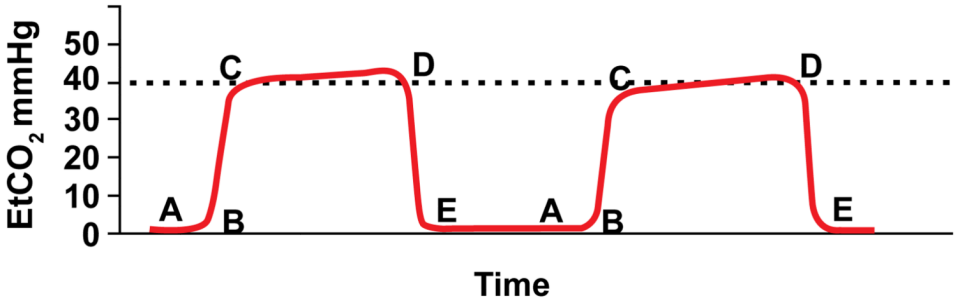

Figure 7.
The normal capnography tracing.

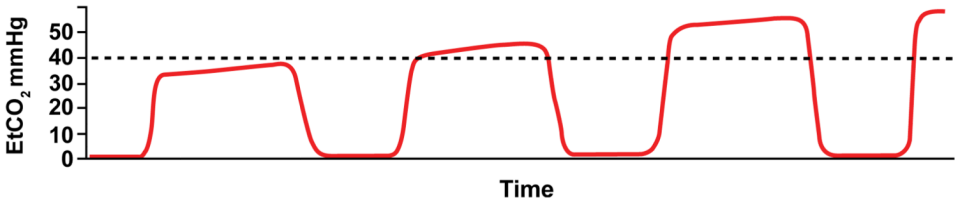

Figure 8.
Capnogram illustrating hypoventilation.

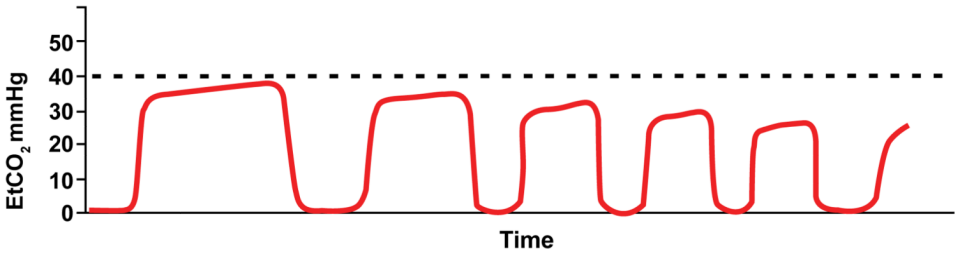

Figure 9.
Capnogram illustrating hyperventilation.

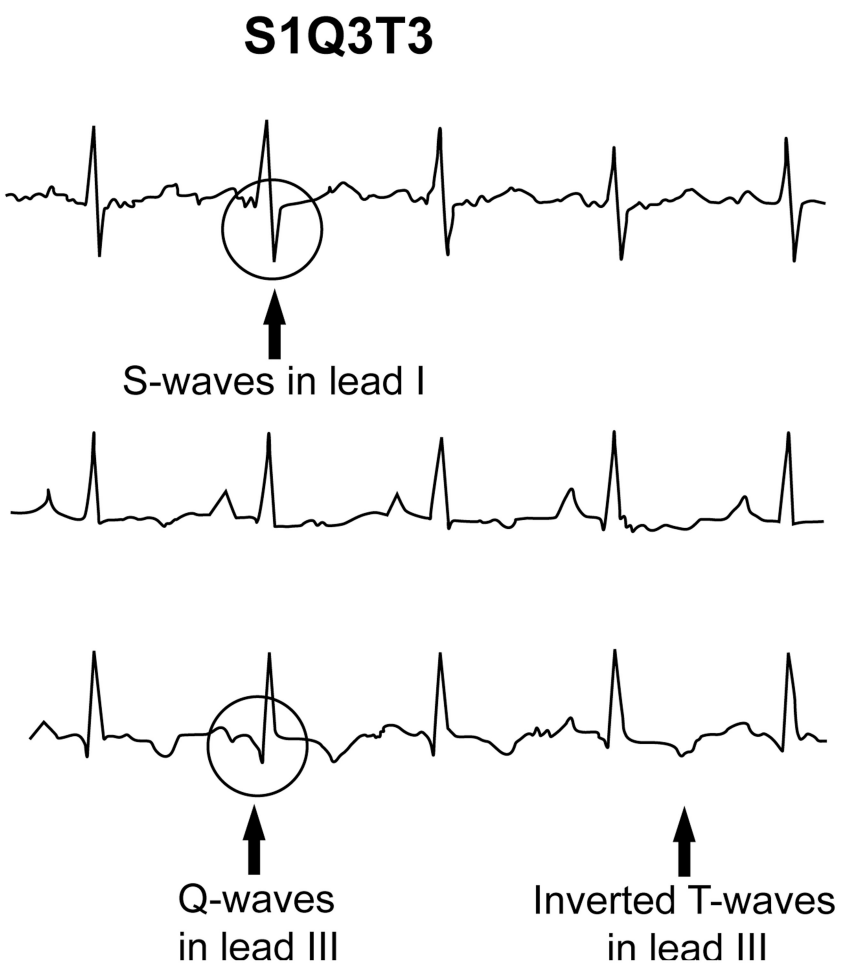

Figure 10.
Electrocardiographic changes classically described for acute pulmonary embolism.

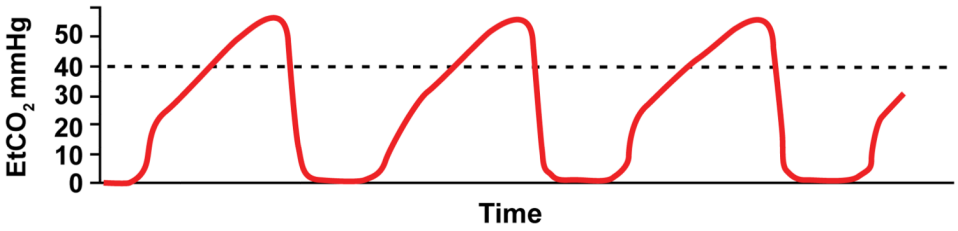

Figure 11.
"Shark-fn" capnogram seen in obstructed exhalation (asthma, COPD, obstruction).

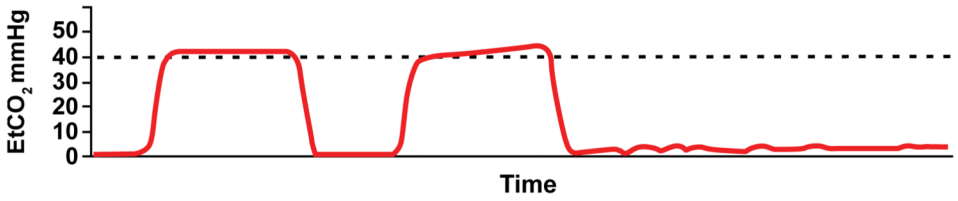

Figure 12.
Capnogram illustrating the sudden disappearance of the wave form indicating apnea.

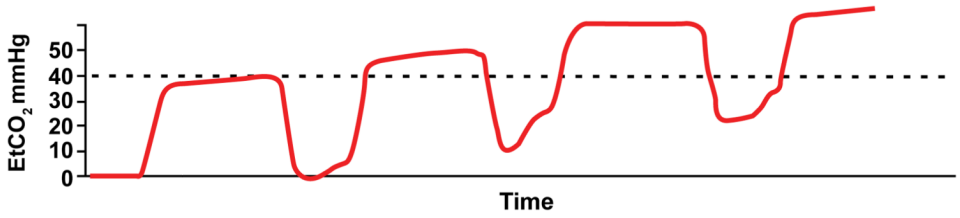

Figure 13.
Capnogram illustrating incomplete inhalation and/or exhalation.

Table 1

Factors that affect the oxygen-hemoglobin dissociation curve.

	Left shift (>affinity for O_2)	**Right shift (<affinity for O_2)**
Temperature	Decrease	Increase
2,3-diphosphoglycerate(2.3-GPD)	Decrease	Increase
p(CO_2)	Decrease	Increase
p(CO)	Increase	Decrease
pH (Bohr effect)	Increase (alkalosis)	Decrease (acidosis)
Type of hemoglobin	Fetal hemoglobin	Adult hemoglobin