RESPIRATORY MONITORING OF CARBON DIOXIDE AND OXYGEN: A TEN-YEAR PERSPECTIVE

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ABSTRACT. During the past 10 years, instrumentation has been developed that can continuously and noninvasively measure changes in carbon dioxide and oxygen. The information generated, which cannot be obtained through the human senses, provides vital clinical data regarding the effectiveness of intubation, ventilation, circulation, oxygenation, and the circuit. This instrumentation plays a major role in decision making both in the safe conduct of anesthesia and mechanical ventilation as well as in the detection and prevention of potentially catastrophic mishaps. For these reasons, a review of what has been learned regarding the instrumentation, collection, and interpretation of the clinical data, and the clinical value of the information is timely. The clinical significance of the carbon dioxide and oxygen waveforms, inspired to expired carbon dioxide and oxygen differences, alveolar-arterial gradients, and global supply-to-demand oxygen relationships measured by capnography, oxygraphy, and pulse oximetry are addressed in this essay.

KEY WORDS. Measurement techniques: capnography; oxygraphy; pulse oximetry.

During the past 10 years major advances have been made in our ability to monitor and quantify carbon dioxide (CO_2) and oxygen (O_2) exchange continuously and noninvasively. Since evidence is accumulating that monitoring improves outcome, it may be useful to give a perspective gained from 10 years of monitoring CO_2 and O_2 during anesthesia and in ventilated patients.

MEASUREMENT OF CO_2

The introduction of capnography by Smalhout and Kalenda [1] defined the relationship of exhaled CO_2 to metabolism, circulation, respiration, and the circuit. Under their direction, capnography survived a stormy gestational period as it reached maturity in The Netherlands. It was introduced in the United States at a small private meeting sponsored by a major instrument manufacturer held in conjunction with the World Congress on Intensive Care Medicine in Washington, DC, in May 1978. Five anesthesiologists attended the meeting, two of whom concluded that capnography would prove to be of very little value. Since then, capnography has come to be recognized as an extremely valuable method for the continuous monitoring of respiration and circulation in unconscious patients. Recognition of the value of capnography for the detection and prevention of mishaps has grown to the extent that some states (New York, New Jersey) have mandated its use on *all intubated patients*.

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Capnography refers to the graphic portrayal of the changing concentration of exhaled CO_2 during the entire respiratory cycle. It also may refer to the interpretation of the waveforms. It should not be confused with *capnometry,* which refers to only the digital presentation of the concentration without a waveform.

The basis of capnography is rooted in the fact that all mammalian cells, irrespective of their diversity of function, have one common denominator, namely, that they obtain the energy necessary to carry out their specific functions by continuously using O_2 to burn glucose to its products of combustion, CO_2 and water (H_2O). For CO_2 to be detected in the exhaled gases there must be the production of CO_2 in the cell (metabolism), the transport of CO_2 from the cell to the lung (circulation), and the elimination of CO_2 as it diffuses into the alveoli and through the airways (ventilation) [1]. Because the respiratory cycle alternates the CO_2-containing gases of exhalation with the fresh non–CO_2-containing gases of inspiration, a characteristic waveform of the changing concentration of CO_2 is produced. This waveform accurately reflects the above sequence of events, as well as the total integrity and efficiency of the breathing circuit, including the ventilator, if one is in use.

INSTRUMENTATION

Medical instrument manufacturers have provided a variety of CO_2 monitors that differ widely in response time, data display, and clinical usefulness [2]. There are two main types, mainstream and sidestream monitors [3].

Comparison of Mainstream and Sidestream Monitors

In mainstream monitors the detector is designed to be attached to an airway adapter that is connected to the endotracheal tube. The respiratory gases pass by windows in the airway adapter, so that the CO_2 concentration is measured by the detector without direct contact with the gases. In sidestream monitors, a small, lightweight T piece is attached to the end of the endotracheal tube, and gas is continuously aspirated back through the detector, which is housed within the monitor.

MONITORING INTUBATED VERSUS NONINTUBATED PATIENTS. With the mainstream technique, only those patients who are intubated can be monitored, since the detector is connected to the endotracheal tube. Sidestream sampling can be used on nonintubated as well as intubated patients. On nonintubated patients the sampling catheter can be placed inside a dental mouth hook, placed in one of the prongs of a nasal O_2 catheter, or shielded inside a catheter placed in the nose or pharynx.

INFECTION CONTROL. Sidestream sampling is simpler because the water trap, connectors, and sampling catheter are all disposable. With mainstream detectors the cuvette can be sterilized.

WEIGHT. Sidestream sampling adds very little weight and bulk to the endotracheal tube connector. Mainstream detectors add weight and bulk, which may increase the chances of inadvertently pushing the endotracheal tube into the right bronchus if care is not taken.

SAMPLING PROBLEMS. Moisture, blood, pus, and mucus may present a problem with both the mainstream and the sidestream techniques. In mainstream monitors, this material may cover the cuvette window; in sidestream monitors it may be aspirated directly into the detector. Line plugging is possible, but these problems are minimized in the better designed monitors of both techniques. Also, in sidestream monitors the aspiration of air from leaks or fresh inflow gases can dilute the exhaled CO_2 sample and slur the waveform. This occurrence is less likely in mainstream monitors because there is no negative pressure in the circuit.

RESPONSE TIME. With mainstream monitors the response time is less than half a second; in sidestream monitors the response time is 2 to 3 seconds because of the time required for the sample to be aspirated through the sampling catheter to the detector. This difference in response time can be significant because of the need to furnish as early a warning as possible. With sidestream monitors the waveform can be influenced by the rate of aspiration, the length and diameter of the sampling catheter, the flow rate of the incoming gas, and sample catheter turbulence that may distort the proximal and distal end of the expired CO_2 bolus.

IMPORTANCE OF THE CO_2 WAVEFORM

There is only one normal capnogram (Fig 1), and all variations from the normal pattern indicate some abnormality that must be recognized and corrected if possible [2].

Any factor that impairs the free exhalation of gas, such as a kinked, displaced, or otherwise obstructed endotracheal tube; asthma; bronchospasm; or chronic obstructive pulmonary disease, will produce a change in the angle of rise of the ascending limb from about 90 to 160°, depending on the severity of the expiratory obstruction. An ascending limb with a long rise time and

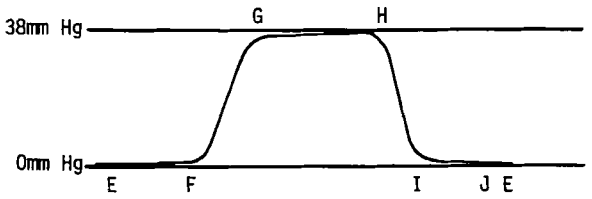

Fig 1. The normal carbon dioxide (CO_2) waveform. E *represents the beginning of expiration;* EF *represents exhalation of upper airway dead-space gases, which do not contain* CO_2. FG, *the ascending limb, rises almost at a right angle from the baseline and represents the increasing concentrations of* CO_2*-containing gases coming from the more distal airways.* GH, *the plateau, makes an almost right angle turn and parallels the baseline, gently rising a few mm, representing the product of the mixed expired* CO_2*-containing gases.* H *represents the end-tidal* CO_2*, normally between 35 and 40 mm Hg. This measurement is valid as a trending relationship with arterial* CO_2 *tension* only *if a normal plateau has been present.* HI, *the descending limb, makes an almost right angle turn and rapidly descends to the base line. It represents the inspiratory phase during which fresh non–*CO_2*-containing gases are inhaled and the* CO_2 *value falls to 0.* IE *represents the phase of inspiration during which the upper airway dead space is filled with fresh non–*CO_2*-containing gases. Thus,* IEF, *the baseline, represents both the fresh gas that filled the upper airway during inspiration and that which is exhaled in early expiration.*

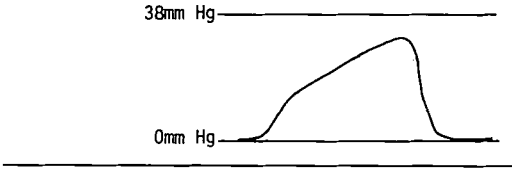

Fig 2. Capnogram indicating expiratory obstruction.

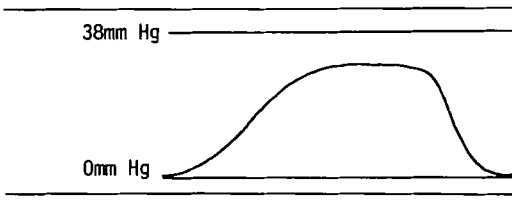

Fig 3. Capnogram indicating diluted inspired gases.

no plateau indicates that exhalation has not been completed before inhalation occurs. In this situation the arterial-alveolar CO_2 [(a − A)Co_2] gradient will increase significantly (Fig 2). It is important to recognize that the value of the end-tidal CO_2 is clinically significant only if a normal plateau is present.

The character and height of the plateau indicate whether exhalation has proceeded normally or abnormally, if both lungs have emptied evenly or unevenly, if the effect of relaxants or other respiratory depressants is still present, if inspiratory efforts are evident, or if hypo- or hyperventilation may be present. In addition, the height and character of the plateau serve as a valuable guide for fine tuning the ventilator settings. Rote calculation or nomogram determination of required tidal and minute volume may suffice for many patients, but be totally inadequate for an obese patient or a patient with multiple respiratory or circulatory problems.

Increased metabolism will raise the height of the plateau, as occurs in fever, excitement, hyperthyroidism, pheochromocytoma, shivering, convulsions, and malignant hyperthermia. Decreased metabolism will result in a fall in the height of the plateau, as occurs in hypothermia.

Any significant failure in circulation, such as decreased effective circulating blood volume, decreased cardiac output, cardiac arrest, or any significant hypotension, will decrease the height of the plateau. In a circulatory crisis the plateau will fall in proportion to the severity of the event and slowly rise in height as pulmonary circulation improves, because the height of the plateau is affected by pulmonary perfusion. In fact, of all available monitors in use during cardiac arrest, capnography furnishes the best real-time, continuous information regarding the effectiveness of resuscitative efforts [4].

The descending limb reflects the dynamics of inspiration. If inspiration is prolonged or if there is a leak around the endotracheal tube causing CO_2-containing gases from the trachea to be entrained, the angle of the descending limb and the plateau will be greater than 90° and the descending limb will take a longer time to reach baseline, as shown in Figure 3. The baseline may not reach zero if CO_2-containing gases are rebreathed, a phenomenon that may occur if the circuit valves malfunction, if the soda lime becomes exhausted, or if the inspiratory flow rate is inadequate, as may occur with a Mapleson D or Bain circuit.

The exact cause of cardiogenic oscillations is not fully understood. They may result from expansion of the pulmonary vascular bed during cardiac systole that displaces 25 to 35 ml of alveolar gas and causes a slight increase in CO_2 concentration, which appears as a small blip on the capnogram synchronous with the QRS of the electrocardiogram. They may also be produced by changes in heart size associated with systole and diastole. In the clinical setting their presence may indicate that the respiratory rate is too slow.

For optimal waveform interpretation, the capnogram should be displayed *continuously* and not intermittently and two recording speeds, fast and slow, should be

available. The fast speed should furnish detailed diagnostic features of the capnogram, while the slow speed should display a compressed, but complete, waveform. Continuous slow-speed waveforms yield the most diagnostic information, because changes in waveform patterns and trends are more readily recognizable. Trend displays that are presented as a single horizontal line of the highest CO_2 values obtained from each respiration are not as clinically useful as information obtained from continuous slow-speed capnography, in which the ascending limb, plateau, and descending limb are clearly discernible.

SIGNIFICANCE OF THE (a − A)CO_2 GRADIENT

If a capnogram is obtained from a normal, conscious, spontaneously breathing person, the end-tidal CO_2 will read between 35 and 40 mm Hg, and if simultaneous arterial blood gases are measured, the arterial CO_2 tension ($PaCO_2$) will be between 1 and 4 mm Hg higher [5]. The arterial to end-tidal CO_2 gradient, which approximates the (a − A)CO_2 gradient in a normal person, is less than 4 mm Hg. However, if mechanical ventilation is instituted or if an anesthetic is administered, the resultant disturbance to normal physiology will generally produce an (a − A)CO_2 gradient of 5 to 10 mm Hg.

With this disturbance, some alveoli are overventilated and others are underventilated, some alveoli are overperfused and others underperfused, micro- and macroatelectasis increases, and both ventilation and perfusion imbalances develop. These changes produce a preponderance of dead-space ventilation, which dilutes the CO_2 coming from ventilated perfused alveoli and results in an increase in the (a − A)CO_2 gradient. The influence of shunting plays a lesser role in the contribution to the increase in the (a − A)CO_2 gradient.

High tidal volumes, positive end-expiratory pressure, and continuous positive airway pressure often may not correct these changes and may, under some circumstances, aggravate them [6]. In addition, the excessive minute volumes that are recommended to ensure the adequacy of oxygenation may produce a ventilation-perfusion ratio greater than 1, further increasing the (a − A)CO_2 gradient by diluting the exhaled CO_2-containing gases [5].

The physiologic changes produced by anesthetic and ancillary agents also contribute to the gradient. The administration of such agents as halothane, enflurane, isoflurane, or nitroprusside results in a loss of the normal pulmonary compensatory mechanisms and also attenuates reflex hypoxic pulmonary vasoconstriction [7]. These changes further imbalance the ventilation-perfusion ratio and result in an increase in dead space and shunt.

Anesthetic agents, positive-pressure ventilation, inadequate effective circulatory blood volume, and hypocapnia [8] all can reduce cardiac output. If the fall in cardiac output is of sufficient magnitude to result in pulmonary hypoperfusion, ventilation will exceed perfusion, further increasing the (a − A)CO_2 gradient.

Factors related to the instrumentation and the technique used may also contribute to the (a − A)CO_2 gradient. For example, aspiration of room air through a loose connection or break in the circuit or sampling tube, a leak around the cuff, or aspiration of fresh gases will dilute the exhaled CO_2 and result in an increase in the (a − A)CO_2 gradient and an altered waveform.

When the genesis of the (a − A)CO_2 gradient is considered, it becomes apparent that the gradient is usually a reflection of changes produced by anesthesia and mechanical ventilation. Under these circumstances the gradient varies between 5 and 10 mm Hg. Gradients that are greater than 15 mm Hg indicate significant pathophysiologic changes, providing they are not caused by sampling errors. The gradient therefore is important because it furnishes an approximation of dead-space ventilation, and its size can serve as a gauge of physiologic aberration. Over time it serves as an indication of whether the patient is improving or deteriorating.

In view of all of the factors discussed above, it is unrealistic to expect the end-tidal CO_2 be the same as the arterial CO_2, and we should not lose confidence in capnography because a gradient exists, but rather take advantage of the information that the gradient provides.

MEASUREMENT OF O_2

It is recognized that the most common cause of anesthesia- and ventilator-related mortality and morbidity is an inadequate delivery of O_2 to the tissues. The resultant tissue hypoxia may be caused by patient, anesthesia, ventilator, or pharmacologic factors, and it generally must be diagnosed and corrected within a matter of minutes if hypoxic tissue damage is to be avoided. Because time under these circumstances plays a pivotal role, O_2 availability must be continuously monitored both centrally and peripherally to ensure adequate oxygenation. Ideally the level of O_2 at every stage of delivery from the circuit, to the airway, to the alveoli, to the blood, to the tissues, and to the mixed venous blood should be known.

During the past 10 years great advances have been made in our capability to noninvasively and continuously monitor some of these critical stages in the cascade of oxygenation. The adoption of the inspired oxygen

fraction (F_IO_2) analyzer and the pulse oximeter have allowed us to rapidly identify a low F_IO_2 in the circuit and to recognize hypoxic hypoxemia in the central arterial circulation. We have also gained a better understanding of preoxygenation, denitrogenation, O_2 delivery, O_2 uptake, and O_2 availability. Currently, anemic, toxic, ischemic, and mixed venous hypoxia cannot be identified noninvasively. However, if the monitoring of oxygenation is to be complete, all the factors involved in the equation at each stage of oxygenation will eventually have to be monitored continuously.

O_2 Analyzer

The role of the O_2 analyzer in ensuring that the circuit concentration of O_2 is satisfactory is accepted as an essential measure of the first stage of O_2 delivery. Its importance has been acknowledged by its inclusion into the Harvard and American Society of Anesthesiologists standards for patient safety.

Pulse Oximetry

The great contribution of pulse oximetry in the measurement of O_2 delivery has become well established, because it verifies that O_2 has been delivered from the circuit, through the airways, and into the alveoli, and that it has diffused into the blood [9].

The pulse oximeter measures the redness of the oxyhemoglobin and in the great majority of cases provides an accurate and reliable measure of the oxyhemoglobin concentration in the central arterial blood. However, this measure is inherently related to the exponential nature of the oxyhemoglobin dissociation curve and therefore provides no warning until the O_2 tension has fallen below 90 mm Hg, at which time the hypoxic event is already in progress. The pulse oximeter will not detect anemic, toxic, or ischemic hypoxia because, in these conditions, the redness of the blood is maintained. Consequently, though the pulse oximeter is unsurpassed for the detection of central hypoxemic hypoxia, under certain conditions it may mislead.

Transcutaneous O_2

Considerable controversy still exists regarding the role of monitoring transcutaneous O_2. Tremper et al [10, 11] found that it correlated with O_2 delivery and reflected local tissue perfusion. Other users also state that it furnishes the best measure of peripheral oxygenation [12]. However, its acceptance has been impeded by the technical difficulties inherent in obtaining the measurement.

Oxygraphy

The first rapid response O_2 analyzer (90% in 100 ms at a 500-ml flow rate) was the Beckman OM11, which was introduced in 1971. It provided a digital display of the changing O_2 concentrations during the respiratory cycle, but it did not provide a waveform unless a recorder was connected. Its value for measuring O_2 consumption was rapidly recognized and it was incorporated into a metabolic cart. Recently, however, rapid response (90% in 150 ms) paramagnetic and magneto-acoustic O_2 analyzers that continously display an O_2 waveform have been introduced for clinical use. These waveforms, which will be referred to as oxygraphy, furnish an indication of the changing dynamics of O_2 in the circuit and in the lungs. Since the introduction of oxygraphy is recent, it is appropriate to consider the information provided and its clinical significance.

Oxygraphy refers to the graphic portrayal of the changing concentration of inhaled and exhaled O_2 during the respiratory cycle. The data are presented as a waveform that furnishes information regarding F_IO_2 and expired O_2 (F_EO_2) concentrations, end-tidal O_2, and the $F_IO_2 - F_EO_2$ differences in real time. In addition, by summing all the gases in the circuit to 100%, the partial pressures of nitrogen and nitrous oxide can be approximated.

Just as capnography is based on the fact that all mammalian cells must continuously use O_2 to burn glucose to its end products of combustion, CO_2 and H_2O, oxygraphy is based on the same equation but deals with the role of O_2. Oxygraphy reflects the balance of alveolar O_2 available during inspiration minus the O_2 uptake secondary to pulmonary perfusion. If the mixed venous O_2 is below normal, the O_2 uptake from the alveoli will be greater. Since O_2 can be obtained only from the alveoli and is only consumed in the cell, any significant change in alveolar O_2 supply, diffusion, O_2 uptake, circulation, metabolism, ventilation, or circuit integrity during the respiratory cycle will be reflected in the O_2 waveform.

Oxygraphy measures alveolar O_2 tensions further down the O_2 cascade, and the waveform characteristics are affected by the volume of O_2 supplied by the effective ventilation, as well as by the O_2 demands. The latter are in turn influenced by metabolism, pyrexia, shivering, convulsions, hypocapnic alkalosis, etc. It is this measure of the O_2 supply-demand relationship that distinguishes oxygraphy from other noninvasive monitors.

ALVEOLAR VENTILATION. Alveolar ventilation is well known to be a major factor influencing alveolar P_{O_2}

tension. However, the (P_{O_2}) relationship is not linear but hyperbolic. As ventilation increases, the alveolar P_{O_2} approaches but never reaches the inspired P_{O_2}. As ventilation decreases, the alveolar P_{O_2} falls along an ever-steepening curve with dramatic changes occurring in response to small changes in ventilation over the critical ranges in ventilation [13]. Oxygraphy continuously tracks these changes.

The O_2 cascade.—The P_{O_2} gradient from atmosphere to mitochondrion has been called the O_2 cascade. During anesthesia the major variable influencing the F_IO_2 – F_EO_2 difference is clearly the alveolar ventilation. An increased O_2 consumption in the presence of a fixed low alveolar ventilation will increase the difference and may lead to a catastrophic fall in alveolar P_{O_2} [13].

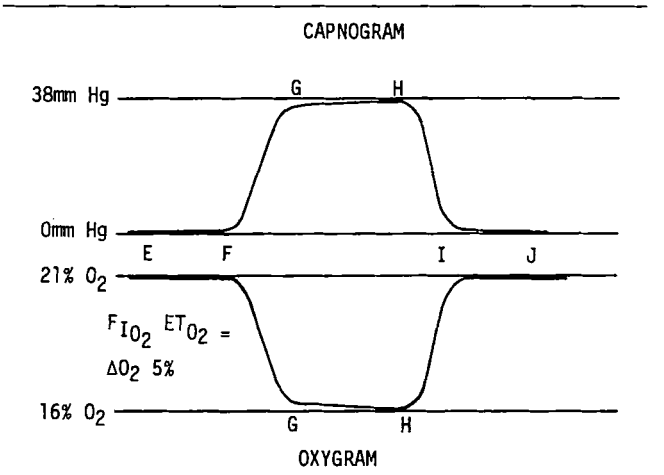

Fig 4. Normal oxygraphic waveform and its relationship to the capnogram. In the oxygraphic waveform, EF represents the beginning of expiration when the airways contain air. FG represents the decrease in oxygen (O_2) concentration that occurs as alveolar gases coming from the lungs that contain less O_2 rapidly displace the air in the anatomical dead space. GH represents the falling O_2 concentration produced by the exhaled alveolar gases and H furnishes the closest approximation to the alveolar O_2 concentration that can be obtained noninvasively. H is significant only as the ET_{O_2} if the waveform has a normal configuration. HI represents the inspiratory phase and IJ represents the air in the airways after inspiration. F_IO_2 = inspired oxygen fraction; ET_{O_2} = end-tidal oxygen. Under normal conditions the F_IO_2 – ET_{O_2} difference is 5% or less.

INSTRUMENTATION

Although only one instrument manufacturer markets a paramagnetic oximeter that furnishes clinically acceptable oxygraphy, other techniques can provide this information. These techniques include mass, acoustical, and Raman spectrometry. At this time only sidestream rapid response O_2 monitors are available. The advantages relative to weight, sampling problems, and response times of sidestream monitors are the same as for capnography.

THE O_2 WAVEFORM

The oxygram is essentially the mirror image of the capnogram. When both are presented in real time while the patient is breathing room air, the relationship of the oxygram to the capnogram is as shown in Figure 4.

The oxygram, unlike the capnogram, has more than one normal configuration because the waveform can be influenced by changes in respired gas concentrations during which O_2 parameters can still remain within normal limits.

SIGNIFICANCE OF THE F_IO_2 – F_EO_2 DIFFERENCE

Normally during spontaneous ventilation in a healthy adult, 21% O_2 is inspired and 16% O_2 is expired. The F_IO_2 – F_EO_2 difference is 4 to 5%. A value of more than 5% in the difference during steady-state anesthesia can serve as a faster, more sensitive indicator of acute hypoventilation than can end-tidal CO_2 [14].

The F_IO_2 – F_EO_2 difference also furnishes an indication of the supply-to-demand relationships of O_2 in the body. Differences of greater than 5%, after a steady state has been reached, indicate a body supply-demand imbalance that must be recognized and corrected. Oxygraphic evidence of an O_2 supply-demand mismatch precedes pulse oximetry and capnographic changes.

According to Linko and Paloheimo [15], oxygrams confirm the diagnosis of hypoventilation, and low end-tidal levels reveal inadequate fresh supplementation. They found that F_IO_2 – F_EO_2 differences increased twofold, while end-tidal CO_2 increased by only 30%, during manual hypoventilation. Further, they presented evidence that indicates that end-tidal O_2, and especially the inspiratory end-tidal O_2 concentration difference, is a faster and more sensitive indicator of acute hypoventilation than is end-tidal CO_2 concentration, which is usually used as an estimate of adequate alveolar ventilation. In addition, they maintain that a low pulse oximetry value is preceded by low end-tidal O_2 concentration and that CO_2 changes occur more slowly and are less pronounced than those of O_2.

The F_IO_2 – F_EO_2 difference furnishes the earliest warning of the development of an impending hypoxic episode. This type of warning can add valuable minutes to the initiation of resuscitative efforts. Failure to meet O_2 demand is the most common cause of organ failure, cardiac arrest, and brain damage.

ALVEOLAR ARTERIAL P_{O_2} [(A − a)O_2] DIFFERENCE

The normal (A − a)O_2 gradient varies between 8 and 24 mm Hg, increasing linearly with age. This widening results solely from decreases in arterial rather than alveolar P_{O_2} [16]. The main factors responsible for an increase in the difference are (1) increased P_{O_2} in the F_{IO_2}, (2) venous admixture secondary to a low ventilation-to-perfusion ratio or to a true shunt, and (3) reduced P_{O_2} on the venous blood [8]. An increased (A − a)O_2 gradient during steady-state ventilation often signals pending arterial hypoxemia [13]. The first indirect evidence that an (A − a)O_2 difference greater than 5% exists may be furnished by oxygraphy.

During anesthesia and mechanical ventilation, the most important factors affecting the gradient are the alveolar P_{O_2} and the venous admixture resulting from a ventilation/perfusion inequality.

(A − a)O_2 differences are often increased as a result of hyperventilation of sufficient magnitude to produce hypocapnic alkalosis. Hypocapnia can decrease arterial P_{O_2} by decreasing cardiac output, increasing O_2 consumption, shifting the oxyhemoglobin dissociation curve to the left, increasing airway resistance, and decreasing lung compliance. Hypocapnia at a $PaCO_2$ level of 20 mm Hg will double O_2 consumption by simultaneously increasing O_2 demand and decreasing O_2 supply [17]. Accumulating evidence seems to indicate that hypocapnia is far from benign and that our attitudes toward it must be seriously reconsidered.

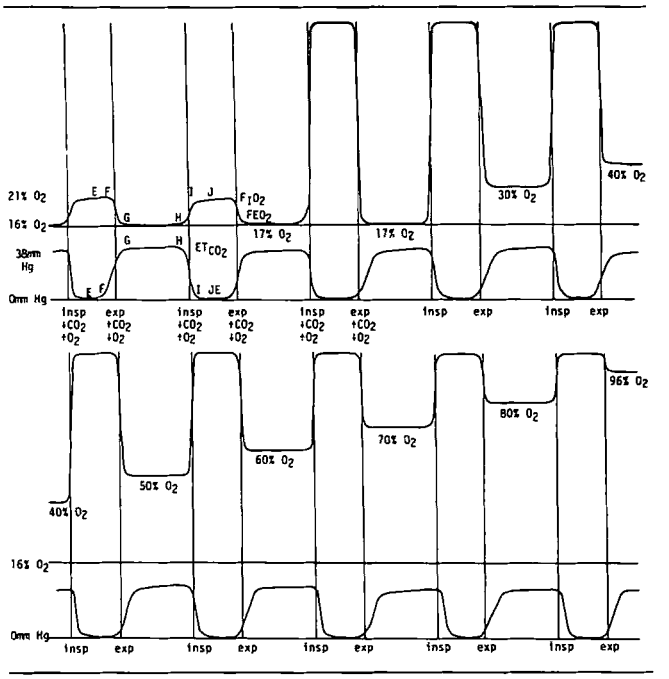

Fig 5. Stylized time sequence of simultaneous capnography and oxygraphy indicating the progression of denitrogenation with 100% oxygen (O_2). The first two breaths are recorded with the patient breathing room air, then 100% O_2 is introduced into the circuit. The sequence, which actually represents about 5 minutes, shows the continual elimination of body nitrogen. When the inspired (F_{IO_2}) minus expired (F_{EO_2}) O_2 difference reaches 4 to 5%, all the nitrogen has been eliminated. insp = *inspiration;* exp = *expiration;* CO_2 = *carbon dioxide;* $ETCO_2$ = *end-tidal carbon dioxide.*

PREOXYGENATION

Preoxygenation takes on new meaning when oxygraphy is used. A common recommendation is to ask the patient to take 4 to 10 breaths of 100% O_2 to ensure an adequate oxyhemoglobin saturation. In this situation the pulse oximeter will usually rapidly confirm that the O_2 saturation has increased from 95 or 96% to 99 or 100%. The oxygraph, however, will indicate that F_{IO_2} may be 100%, while the F_{EO_2} initially will read 30 or 40%, indicating that the functional residual capacity still contains a significant concentration of nitrogen, which is being cleared from the lungs and tissues. Obviously the higher the concentration of O_2 in the lungs the greater will be the O_2 reserve. The margin of safety can be increased significantly because the O_2 reserve of the lung can be changed from about 700 to 2000 ml or more of O_2.

The quantity of O_2 in the lung is especially critical during difficult intubations, apneic periods, and in patients with cardiorespiratory impairment (abdominal distension, ascites, pregnancy, obesity, restrictive pulmonary disease, chronic obstructive pulmonary disease, etc.). During denitrogenation with 100% O_2, the F_{EO_2} will progressively increase over a period of about 5 minutes from 16 or 17% on the first breath to 96% when denitrogenation is completed, as shown in Figure 5. At this juncture, the F_{IO_2} − F_{EO_2} difference is 4 to 5% and the pulmonary reserve has a full complement of O_2. After denitrogenation, the apneic period may be more than doubled before the O_2 saturation begins to fall [18]. Thus, the use of oxygraphy provides a reliable real-time measure of the progress of denitrogenation.

After maintenance has begun, nitrous oxide is added and the O_2 concentration is reset to the desired maintenance concentration, the F_{IO_2} − F_{EO_2} difference will change until an equilibrium is reached between the patient and the circuit. When this steady state is reached, the F_{IO_2} − F_{EO_2} difference should return to about 5%. If the difference exceeds 5% thereafter, it is usually evidence of hypoventilation [15], hypoxia, a change in circuit concentrations, an air embolism, an air leak, or an increase in dead space.

During maintenance of anesthesia, if denitrogenation has been accomplished, the presence of nitrogen in the circuit may result from a circuit leak or an air embolism. During a circuit leak, F_EO_2 may exceed the FiO_2 and the oxygraph may reverse its configuration.

On emergence from anesthesia, both nitrous oxide diffusion and the second gas effect can produce alveolar hypoxia. When the nitrous oxide is turned off and 100% O_2 is delivered for a short time, the nitrous oxide rapidly diffuses into the alveoli in greater volume than the nitrogen entering, and the O_2 in the alveoli is diluted. Oxygraphy identifies the endpoint when the nitrous oxide has been eliminated, at which time the $FiO_2 - FeO_2$ difference returns to less than 5%.

COMMENTARY

Great advances in patient monitoring of the respiratory system have been made in the past 10 years. Instrumentation was made available to noninvasively measure changes in CO_2 and O_2, changes that no clinician could sense. Capnography began to mature, pulse oximetry did mature, and oxygraphy began its early gestational phase. And we began to recognize that each monitoring method fits differently into a hierarchy for the early detection of different critical events [19,20].

Capnography rapidly detects failed intubation, failed ventilation, failed circulation, and failed anesthetic circuit as they occur. In the operating room and intensive care unit, where minutes to hours can pass before respiratory obstruction and hypoventilation are recognized, capnography furnishes an early warning. Both physicians and nurses learn very rapidly that proper respiratory exchange reduces the occurrence of dysrhythmias and circulatory crises. The emphasis in medicine has been on dysrhythmia detection rather than on the inadequacy of respiratory exchange, which often initiates the hypoxia or hypercapnia that produces the cardiac dysrhythmias.

Oxygraphy often helps detect hypoventilation, hypoxia, increased O_2 uptake, a fall in Po_2 in venous blood, and the presence of nitrogren or nitrous oxide, and it continuously monitors the alveolar O_2 concentrations. The most significant clinical contributions of oxygraphy are that the $FiO_2 - FeO_2$ difference furnishes a continuous, noninvasive, approximate indication of the indirect relationship of O_2 supply to demand, as well as the amount of O_2 available in the functional residual capacity. Currently the best assessment of oxygenation is obtained by measuring the mixed venous O_2 with a pulmonary artery catheter. Oxygraphy does not furnish as fine a measure, but it does furnish a noninvasive warning of a possible developing fall in mixed venous O_2.

Both capnography and oxygraphy will detect disconnections, misconnections, and esophageal intubation. Both techniques may often detect human error, equipment failure, and lapse of vigilance.

Patients who are anesthetized, paralyzed, and mechanically ventilated are an integral part of a tightly coupled system in which a minor error can quickly cascade into a major catastrophe. Capnography, the oxygen analyzer, pulse oximetry, and oxygraphy all contribute greatly to patient safety by detecting potentially catastrophic events before hypoxia has occurred.

Yes, we have learned much in the last decade from monitoring CO_2 and O_2. Vital clinically applicable information regarding intubation, ventilation, circulation, and the circuit is now available continuously and noninvasively. Our understanding of dead space, shunting, system sampling, leaks, CO_2 and O_2 gradients, and end-tidal values has been greatly expanded. This information aids and supplements clinical experience and judgment in decision making. Much has been accomplished and much undoubtedly remains to be discovered in the next 10 years.

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