CASE STUDIES IN CRITICAL CARE MEDICINE

Second Edition

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PREFACE

Critically ill patients differ from other patients, not because of specific pathologies or therapies, but because of their unstable clinical condition which mandates the frequent reevaluation of the patient's physiologic homeostasis and adjustment of therapy. Critical care practitioners are continually required to integrate a plethora of physiologic data and make frequent therapeutic decisions. In our opinion, conventional texts do not readily convey to the reader this process of decision analysis. The case study format of this book represents an attempt to reflect the process of decision making and hopefully better instruct the reader in the application of this process in the management of critically ill patients.

We have chosen topics that describe the major problems encountered in adult intensive care units. Case histories were selected to present a mix of surgical and medical patients though obviously the principles of therapy and monitoring apply equally well to all critically ill patients. This text makes no pretense of addressing the entire body of knowledge of critical care medicine. Rather, we have focused on clinically relevant material and its application in decision analysis and management of critically ill patients.

In this second edition the range of topics has been expanded to cover those subjects in which newer technology has added new dimensions to the care of critically ill patients (clinical blood gas monitoring, alternate forms of mechanical ventilatory support). New chapters on pain control and symptom management, non-traumatic coma, acute endocrine emergencies, and ethical aspects of critical care medicine have been added. The discussion of neurosurgical problems, myocardial ischemic conditions, and coagulopathies in the critically ill patient have been expanded.

With these additions, the topics cover problems that occur with frequency in critically ill patients and in which there is, in most instances, a reasonable body of clinically relevant information.

The earlier chapters deal with therapeutic modalities that have an application to various pathologies and with simpler clinical problems involving single organ systems. The later chapters are devoted to more complex specific therapies and the clinical problem of multi-organ system disease.

Pediatric critical care has been excluded due to limitations of space and the existence of other pediatric case study texts. We chose not to address multiple trauma or burns as specific subjects because outside of the elements of cardiopulmonary support and fluid therapy, the management of these patients is a surgical, not critical care problem. These elements of therapy

and support have been discussed in this text, although not necessarily in the context of patients with traumatic disease.

We wish to thank the contributing authors for their chapters. Thanks are due to our colleagues, in particular Drs. Edward Brunner, Roy Patterson, Jeff Glassroth, Chris Chomka, Bill Peruzzi, and Dan Fintel for their unfailing support of our endeavours. We thank Kelly Quinn, Xenia Ruiz, and Joan Woods for secretarial assistance in the preparation of the manuscript.

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CONTENTS

1 / Clinical Blood Gas Monitoring

CHRISTINA CHOMKA, M.D.

The practice of critical care medicine is rapidly changing as new technology and more elaborate monitors are introduced into the marketplace. The usefulness of any patient monitor depends on its ability to provide accurate values and rapidly respond to changes, allowing for early detection of fluctuations from baseline. The dangers of hypoxemia, hypercarbia, and hyperoxia are well recognized in the intensive care setting, as can be attested to by the frequency of arterial blood gas sampling. Although long recognized as the "gold standard" of assessment of oxygen and carbon dioxide tensions, the test provides for only intermittent determinations, frequently missing transient but potentially harmful fluctuations.

In recent years much emphasis has been placed on the use of continuous, noninvasive monitoring.[1] Pulse oximeters, transcutaneous oxygen and carbon dioxide electrodes, and end-tidal CO_2 monitors have been marketed as being essential for assessing the adequacy of oxygenation or ventilation. In order to properly interpret the values generated by these monitors, one must be familiar with their mode of action, values that are actually being measured, and limitations of the system.[2]

Our ultimate goal is to maintain cellular viability by ensuring adequate oxygen delivery as well as carbon dioxide elimination. Because oxygen delivery is equal to oxygen content [$(Hb \times O_2\ sat \times 1.34) + (0.003 \times Pao_2)$] times the cardiac output, it is apparent that multiple factors can be responsible for cellular hypoxia. Individual monitors can inform us of some aspect of this equation. By integrating the results of multiple monitors, one can potentially have an on-line, real-time assessment of the adequacy of oxygen delivery.

PULSE OXIMETRY

Pulse oximeters are presently used widely to monitor arterial oxygen saturation. Of all of the monitors currently available to monitor oxygenation, the pulse oximeter is probably the easiest to use; because it is factory precalibrated and noninvasive, it can be put into use instantly without the need for warm-up time.[3] Oximetry relies upon absorption or reflectance spectroscopy and incorporates the principles of the Beer-Lambert law for determination of oxyhemoglobin saturation because the absorption spectra of oxygenated and reduced hemoglobin are different.[4]

Developed in the 1940s by Millikan, the initial oximeter required exsanguination of the site to be monitored in order to establish a baseline and subsequent heating of the site to arterialize the blood.[5] Hemoglobin saturation was measured by transilluminating a capillary bed with two wavelengths. A photodetector measured the transmitted light. The saturation of the arterial blood was related to the difference between the absorbent signal of the arterialized sample and the baseline. Because of technical difficulties in their usage, the initial oximeters had limited clinical applications.[6, 7]

In the 1970s Nakajima and colleagues made a discovery that transformed the oximeter into a clinically useful tool.[8] They recognized that the pulsatile absorbancies of the red and infrared light transmitted correlated with hemoglobin saturation, with red light being more absorbed at 660 nm by reduced hemoglobin and infrared light being more absorbed by oxygenated hemoglobin at 990 nm. By positioning any vascular bed between a two-wavelength light source and detector, hemoglobin saturations can be measured. The baseline is determined by absorption due to nonpulsatile arterial blood, venous and capillary blood, and tissue. Arterial absorption is a calculation of the ratio of the pulse-added absorption to the baseline absorbancies at the two wavelengths.[9]

With only two wavelengths being measured, the pulse oximeter is only able to distinguish two hemoglobin moieties within the blood—oxyhemoglobin and reduced hemoglobin. If other hemoglobins are present within the blood (i.e., methemoglobin, carboxyhemoglobin), they will be incorporated into the oxyhemoglobin and reduced hemoglobin measurement, thereby generating erroneous saturations.[10, 11] In fact, any substance within the blood that absorbs light at the red and infrared wavelengths of the pulse oximeter may cause measurement error. Dyes such as methylene blue, indigo carmine, and indocyanine green can all cause transient reductions in oxygen saturation.[12, 13] Pulse oximeter saturation can also be affected by room light interference.[14, 15] Although measures have been taken to correct for this interference by rapid averaging and subtraction of the ambient light signal, unrecognized errors can occur. The easiest remedy of this situation would be an opaque protective covering of the sensor site. Additional problems can be caused by motion artifact or weak signals caused by thready pulses.[16] To more accurately interpret the values, a pulse waveform or signal strength indicator should be incorporated into the monitors so that erroneous readings can be ignored.

The use of oximetry probes is not entirely without risk, although they are significantly safer than transcutaneous or intra-arterial probes. Skin damage, with the extreme situation being necrosis of the digit to which the probe had been applied, has been reported in situations of prolonged use with poor peripheral circulation secondary to low-flow states or the use of vasoconstricting drugs.[17]

Currently available pulse oximeters have a reported accuracy of ±2% in the 70% to 100% saturation range and ±3% down to the 60% saturation range, assuming minimal concentrations of dyshemoglobin species.[18–20] Below this range saturations correlate poorly with those obtained with in vitro co-oximeter measurements, significantly overestimating the actual oxygen saturation. Studies have revealed superior accuracy of the ear oximeter to that of the finger oximeter, which in part may be related to differences in transit time.

The greatest advantages of pulse oximetry are its ease of application and ability to provide continuous saturation readings. Because significant reductions in arterial saturation may occur before cyanosis becomes clinically apparent, the pulse oximeter can act as an early-warning monitor of potentially life-threatening desaturation. The primary limitations of this monitoring device, aside from the technical aspects, relate to the fact that oxygen saturation, and not oxygen tension or oxygen delivery, is measured. In view of the sigmoidal shape of the oxyhemoglobin dissociation curve, major changes in oxygen tension may occur (especially in a patient receiving supplemental oxygen) with minimal fluctuation of the oxygen saturation. In addition, one cannot equate an acceptable saturation with adequate perfusion because the device will function as long as a pulse is present. A signal strength indicator or pulse waveform may help qualitate peripheral perfusion. Vascular pulsations that are markedly reduced or absent will incapacitate the monitor. Pulse oximetry can be applied to the ear, digits, bridge of the nose, nasal septum, or temple over the temporal artery.

TRANSCUTANEOUS MONITORS

Transcutaneous gas exchange has been recognized for more than 100 years, since the initial work of von Gerlach in measuring oxygen and carbon dioxide exchange between the skin and air.[21] Numerous obstacles had to be overcome such as compensating for the oxygen consumption of the skin, variations in gas exchange across the epidermis, and regulating oxygen delivery to the skin before transcutaneous measurements of oxygen tension could be relied upon to reflect arterial oxygen tension. Even with all of the refinements, multiple factors can adversely affect cutaneous blood flow and result in erroneous tension readings. It is the recognition of the factors that affect transcutaneous readings that makes this a clinically useful tool. Unlike oximeters that monitor hemoglobin saturation, transcutaneous monitors measure actual gas tensions. A falling $PtcO_2$ at high oxygen tensions may be indicative of undesirable changes long before arterial hemoglobin saturation actually changes.

Clark's discovery of the polarographic oxygen electrode in 1956 along with the introduction of the Severinghaus carbon dioxide electrode in 1958 provided the technologic basis for continuous transcutaneous measurement of oxygen and carbon dioxide that diffuse to the skin surface from the dermal capillary bed beneath. Currently utilized transcutaneous electrodes are essentially miniaturized versions of both the Clark and Severinghaus electrodes.[22] After electrode calibration and special site preparation allowing for airtight electrode positioning, diffusion of gases to the surface of the skin is produced through local hyperemia secondary to heating of the electrode site to 43 to 45°C. Cutaneous blood flow is temperature dependent, with the greatest increases in flow occurring at skin temperatures between 35 and 45°C and maximal dilatation occurring around 45°C. Above this temperature complications to the skin are significant without further increases in blood flow.[23] In addition, the stratum corneum, which provides the mechanical strength of the epidermis, liquefies at temperatures >41°C, and this increases the diffusion of gases between 100 and 1,000 times through this barrier.[24]

Fortunately, the initial clinical application of the transcutaneous oxygen electrode was in the neonatal population, in which, after heating of the skin to 44°C, close correlation was observed between transcutaneous and arterial oxygen tensions. It is only in the neonatal population that arterial blood actually comes in contact with the epidermis; in adults, perpendicular capillary loops approach the epidermis, and oxygen delivery occurs by diffusion. Subsequent studies in hemodynamically unstable neonates revealed that $PtcO_2$ significantly underestimated PaO_2.[25] Application to the adult population proved less rewarding. In hemodynamically stable adult patients, $PtcO_2$, although trending PaO_2, was only 80% of the actual value.[26] Under conditions of hemodynamic stability, rapid alterations in arterial PO_2 will be qualitatively tracked by $PtcO_2$; however, a 95% response time ranging from 8 to 90 seconds has been reported, depending on the magnitude of the change.[27] Because the electrodes measure oxygen that diffuses through the skin, skin tissue rather than arterial oxygen tension is measured. Factors influencing the oxygen delivery to the skin, such as cardiac output and regional perfusion, will be reflected in the transcutaneous measurement as well as the PaO_2. Human and animal studies confirm this fact inasmuch as it has been documented repeatedly that $PtcO_2$ tracks PaO_2 during hypoxemia and cardiac output during shock.[28, 29]

This improved understanding of the physiology of transcutaneous monitoring has prompted an increased use in adults. A monitor that at one time was considered useless in situations of altered perfusion is now being applied more frequently to critically ill patients, specifically those at risk for cardiopulmonary compromise, or to regional alterations in perfusion (e.g., skin flaps, extremities distal to balloon pump insertion).[30, 31]

Initial work performed by Tremper and Shoemaker in 1980 in critically ill patients found that $PtcO_2$ followed PaO_2 until the cardiac index (CI) fell below 2 L/min/m^2, at which point the $PtcO_2$ values became flow dependent.[32] He subsequently defined a transcutaneous index ($PtcO_2/PaO_2$) to assess the degree of peripheral perfusion deficit. Patients with a CI $>$ 2.2 L/min/m^2 had a transcutaneous index of 0.79 $\pm$ 0.12; as the CI fell to 2.0 L/min/m^2, the index decreased to 0.49; and at a CI of 1.0 L/min/m^2 the transcutaneous index was 0.12.[32] It was apparent that as the CI fell to shock levels, the $PtcO_2$ values correlated with the CI.

The transcutaneous CO_2 electrode was also used initially as a neonatal monitor. Although transcutaneous electrodes respond to $PaCO_2$ changes when heated to a lower temperature than required for $PtcO_2$ monitoring, analysis at 44°C allows for a faster response time, with 95% response occurring at 3.5 minutes.[33] Heating of the skin by the sensor increases the PCO_2 at the skin surface by approximately 23 $\pm$ 11 mm Hg, but despite this difference in absolute value, the $PtcCO_2$ trends the $PaCO_2$ accurately.[34] Because the $PtcCO_2$ averages 140% to 160% of the $PaCO_2$, many manufacturers have programmed correction factors into the $PtcCO_2$ monitors, thereby producing transcutaneous CO_2 values comparable to arterial CO_2 values. As with $PtcO_2$ values, $PtcCO_2$ becomes flow dependent and does not trend $PaCO_2$ at a CI below 1.5 L/min/m^2.

Transcutaneous CO_2 electrodes are effective monitors of the adequacy of ventilation, without needing to have the patient intubated as with capnography. Rapid titration of ventilator changes can also be performed. More recent applications include

continuous, noninvasive monitoring of fetal acid-base status during labor and delivery with electrode placement on the fetal scalp.

Combined electrodes measuring both $PtcO_2$ and $PtcCO_2$ are currently available. Sensor sites should be changed every 4 hours to minimize the risk of first-degree skin burns. Whereas $PtcCO_2$ electrode calibration should be carried out every 4 hours, $PtcO_2$ electrode calibration is required only every 24 hours; however, a drift check should be performed every 4 hours.

END-TIDAL CO_2

Capnography, the measurement of carbon dioxide in the respiratory gases, currently can be measured either by infrared absorption or mass spectrometry. The infrared analyzer passes a beam with a wavelength of 4.3 μm through a sample of gas, and the transmitted light intensity is compared with that of a reference beam. Carbon dioxide's maximal absorption for infrared light occurs around 4.3 μm; thus, to measure the carbon dioxide concentration accurately, the infrared beam must be filtered carefully to avoid measurement of other gases (i.e., N_2O) with absorption peaks close to that of carbon dioxide.[35]

Most infrared analyzers are sidestream analyzers aspirating small amounts of gas from the breathing circuit close to the patient and passing the gas through small-bore tubing to a remote analyzer. The sampling rate is usually 150 cc/min with an average delay of approximately 100 ms. This sampling method adds little weight or dead space to the breathing circuit, but care must be taken to prevent clogging of the sampling tubing with secretions or condensation.

Gas analysis can also occur within the breathing circuit itself by using a mainstream analyzer, which places a detector adjacent to the endotracheal tube and allows for very rapid CO_2 determinations (averaging 10 ms). Although rarely affected by secretions because of the large internal diameter used to minimize airway resistance, the analyzer is bulky and heated to prevent condensation; thus endotracheal tube displacement and patient burns can occur if the system is not well supported.[36]

An alternate means of CO_2 analysis is through mass spectrometry. The gas sample is aspirated into a high-vacuum chamber in which an electron beam ionizes and fragments its components. These are then accelerated through an electric field into a strong magnetic field that deflects the ionic fragments into an arch, the diameter of which is dependent on the charge-to-mass ratio of the fragments. Detector plates are positioned according to the deflection arches of the substances to be measured. As fragments hit the detector, an electric current is generated from which the concentration of the gas to be measured can be calculated. Not only CO_2 but multiple gas components as well can be simultaneously analyzed.[37]

Mass spectrometers tend to be large and expensive; thus they are frequently used to monitor more than one patient, thereby allowing for only intermittent monitoring. Because they are sidestream analyzers, they are subject to the same line blockage problems associated with the sidestream infrared capnometers.[38] The exhaled CO_2 waveform in a healthy individual is nearly a square wave and can be divided into three phases: phase 1, exhaled gas from anatomic dead space; phase 2, exhaled gas

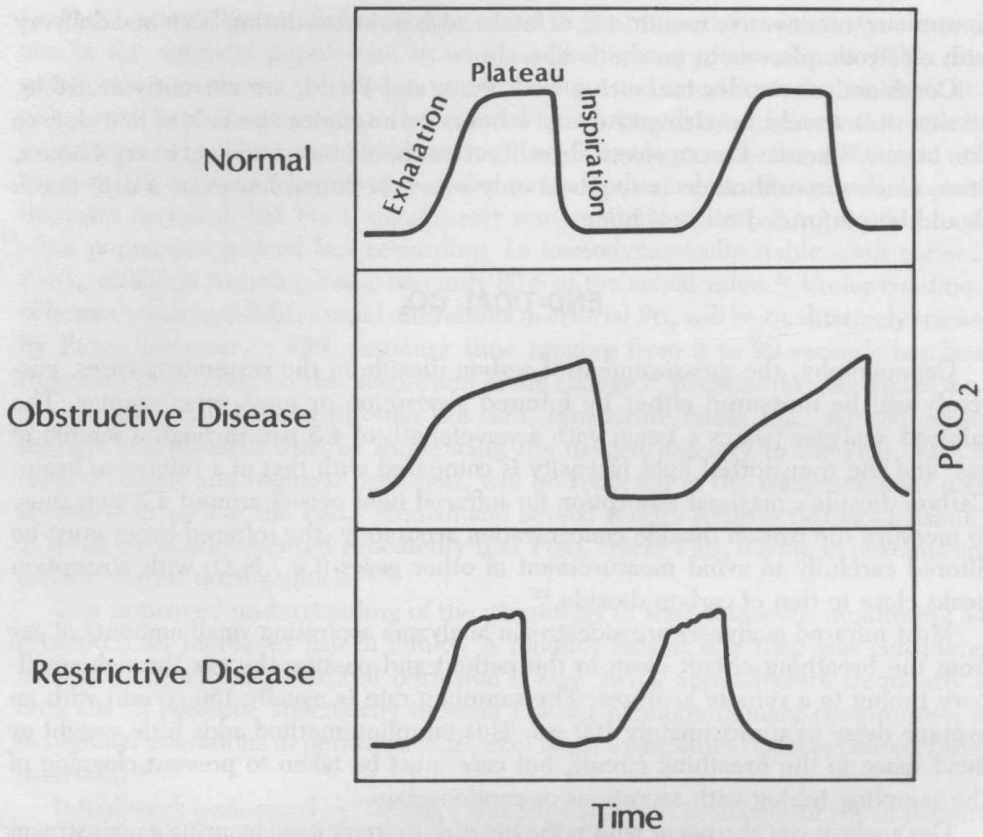

FIG 1–1.
The normal capnograph pattern shows a sharply rising exhalation slope with a clearly identifiable plateau. Inspiration is seen as an abrupt decrease in CO_2 to baseline. Obstructive disease generally produces a prolonged exhalation slope with a difficult-to-identify plateau. Restrictive disease generally provides a "choppy" plateau. (From Shapiro BA, Harrison RA, Cane RD, et al: *Clinical Application of Blood Gases,* ed 4. Chicago, Year Book Medical Publishers, Inc, 1989. Used by permission.)

composed of progressively more alveolar gas; and phase 3 (the alveolar plateau), exhaled gas from alveoli, both perfused and nonperfused.

The end-tidal CO_2 concentration is defined as the maximal concentration of exhaled CO_2 and is usually 1 to 2 mm Hg less than the arterial CO_2 tension because it reflects the CO_2 concentrations of both perfused and nonperfused (dead space) alveoli. Abnormalities in ventilation, circulation, and metabolism can be reflected in the capnogram that is generated.[39, 40] It is therefore important that in addition to a numerical display of the end-tidal CO_2 concentration a waveform be displayed to facilitate diagnosis.

Figure 1–1 displays a normal capnogram and those associated with obstructive and restrictive lung disease. Note the gradual rise of the alveolar plateau that is produced by increased alveolar dead-space ventilation from chronic obstructive pulmonary disease (COPD) in the obstructive disease example.

Figure 1–2 shows the effect of a fall in cardiac output on the capnogram. Figure 1–3 shows a capnogram displaying cardiogenic oscillations on the tracing.[41]

Capnography provides a continuous, noninvasive means of monitoring CO_2 in intubated patients. Clinical judgment is required to ascertain whether reductions in end-tidal CO_2 are a result of excessive CO_2 elimination or increased dead-space ventilation. Capnography can also be used during cardiopulmonary resuscitation to assess the adequacy of perfusion.[42] Reductions in P_{ETCO_2} during cardiopulmonary resuscitation (CPR) are associated with comparable reductions in cardiac output, whereas increases in P_{ETCO_2} during successful resuscitation are associated with cor-

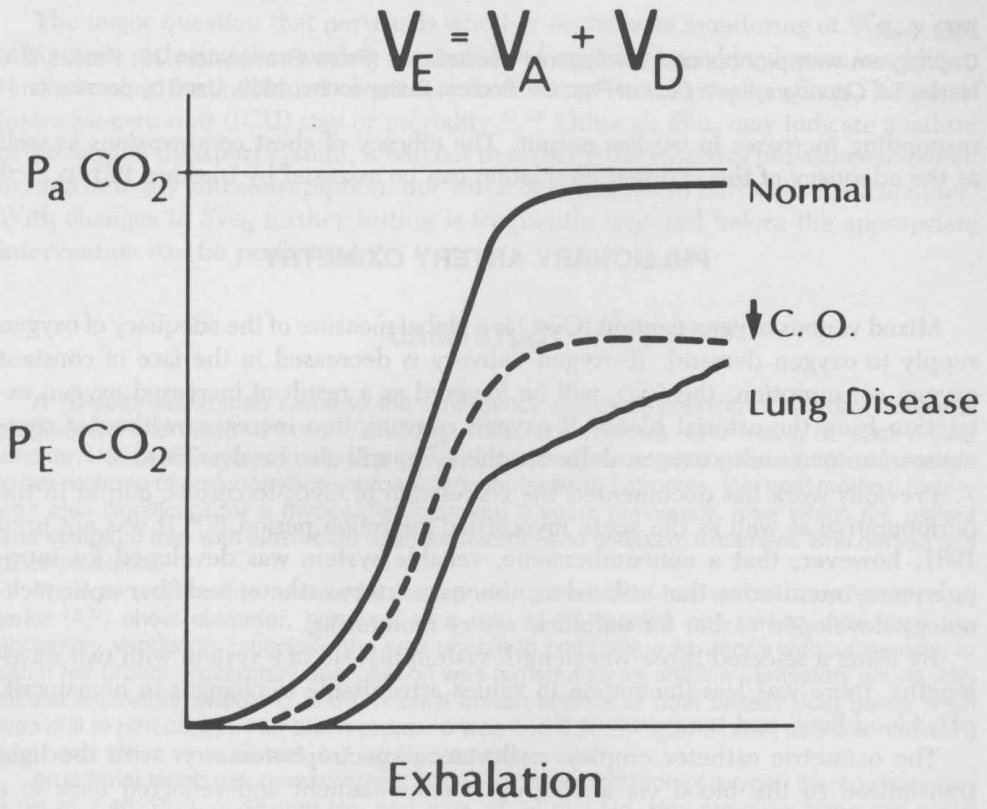

FIG 1–2.
Total ventilation (V_E) is composed of both alveolar ventilation (V_A) and dead-space ventilation (V_D). $Paco_2$ is considered the best reflection of alveolar ventilation. The end-tidal P_{CO_2} (P_{ETCO_2}) is the expired P_{CO_2} (P_{ECO_2}) at the end of the plateau. An increased V_D will be manifested as an increased $P(a\text{-}ET)CO_2$ gradient. The two most common causes of increased dead-space ventilation are decreased cardiac output and lung disease. A decreased pulmonary perfusion will result in more alveoli having a lower P_{CO_2}; the net result is a decreased expired P_{CO_2} but no change in the lung-emptying pattern. This is depicted as the *dashed curve* with a shape similar to the normal. Lung disease will involve changing lung-emptying patterns and thus a change in the curve. (From Shapiro BA, Harrison RA, Cane RD, et al: *Clinical Application of Blood Gases,* ed 4. Chicago, Year Book Medical Publishers, Inc, 1989. Used by permission.)

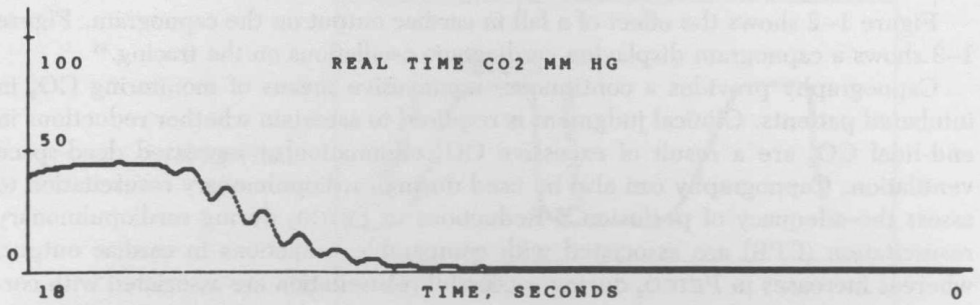

FIG 1–3.
Capnogram with pronounced cardiogenic oscillations. (From Gravenstein JS, Paulus DA, Hayes TJ: *Capnography in Clinical Practice.* Boston, Butterworths, 1989. Used by permission.)

responding increases in cardiac output. The efficacy of chest compressions as well as the adequacy of the restored circulation can be assessed by tracking P_{ETCO_2}.[43–45]

PULMONARY ARTERY OXIMETRY

Mixed venous oxygen content ($C\bar{v}O_2$) is a global measure of the adequacy of oxygen supply to oxygen demand. If oxygen delivery is decreased in the face of constant oxygen consumption, the $C\bar{v}O_2$ will be lowered as a result of increased oxygen extraction from the arterial blood. If oxygen consumption increases without a commensurate increase in oxygen delivery, the $C\bar{v}O_2$ will also be decreased.[46]

Previous work has documented the correlation of $C\bar{v}O_2$ to cardiac output in the perioperative as well as the acute myocardial infarction period.[47, 48] It was not until 1981, however, that a noncumbersome, reliable system was developed for intrapulmonary monitoring that utilized a pulmonary artery catheter and fiber-optic technology developed earlier for umbilical artery monitoring.

By using a selected three-wavelength system instead of a system with two wavelengths, there was less fluctuation in values attributable to changes in hematocrit, pH, blood flow, and temperature.[49]

The oximetric catheter employs reflectance spectrophotometry, with the light transmitted to the blood via a fiber-optic monofilament and reflected back in a separate fiber-optic monofilament to the photodetector. The percent saturation of oxyhemoglobin is calculated by microprocessor technology. In vitro calibration of the catheter is required before insertion, and in vivo calibration can be performed if blood is withdrawn from the pulmonary artery and measured in a co-oximeter as the control. The system appears stable, with a drift of less than 1% per 24 hours; therefore, once-a-day calibration appears adequate.

In healthy individuals breathing room air, the mixed venous oxygen saturation ($S\bar{v}O_2$) ranges between 68% and 77%. At lower oxygen tensions a near-linear relationship exists between saturation and tension.

Reductions in $S\bar{v}O_2$ can result from decreased arterial saturation, increased oxygen consumption, decreased cardiac output, or decreases in hemoglobin. Although $S\bar{v}O_2$