Chapter

20 ANESTHETIC MONITORING

James Szocik, Magnus Teig, and Kevin K. Tremper

INTRODUCTION

Anesthesiologists have long been at the forefront of patient monitoring. This has been of necessity, because we are responsible for continuously assessing the patient's physiologic status and the effects of surgery and anesthetic drugs. The following is an introduction to the basic function and utility of the wide array of monitors employed in modern anesthesia care. Monitoring devices will be organized by the organ or organ system that they are monitoring, not the physical property or technique on which that monitor derives its information. Because the monitors for each organ system may employ the same physical properties, such as light absorption or pressure transduction, each monitor will be described as it is used for a specific organ, but the description of the principle may refer to another section within the chapter. For an in-depth review of these principles the reader is referred to a more comprehensive text.[1]

Overview

In 1986 the American Society of Anesthesiologists (ASA) established a set of basic monitoring standards, stating that the patient's oxygenation, ventilation, circulation, and temperature shall be continually evaluated.[2] These standards, the first of their kind (last affirmed in 2015), should be viewed as a minimum requirement and many situations will require additional monitoring. All of the organ systems monitored are perfused by the circulatory system (Fig. 20.1). Monitoring the patient permits the anesthesia provider to continuously assess if the patient's state is "normal" or "abnormal" and to correct the cause of the abnormality, or at least treat the abnormal number generated by the monitor. However, the limitations of monitors and how to use

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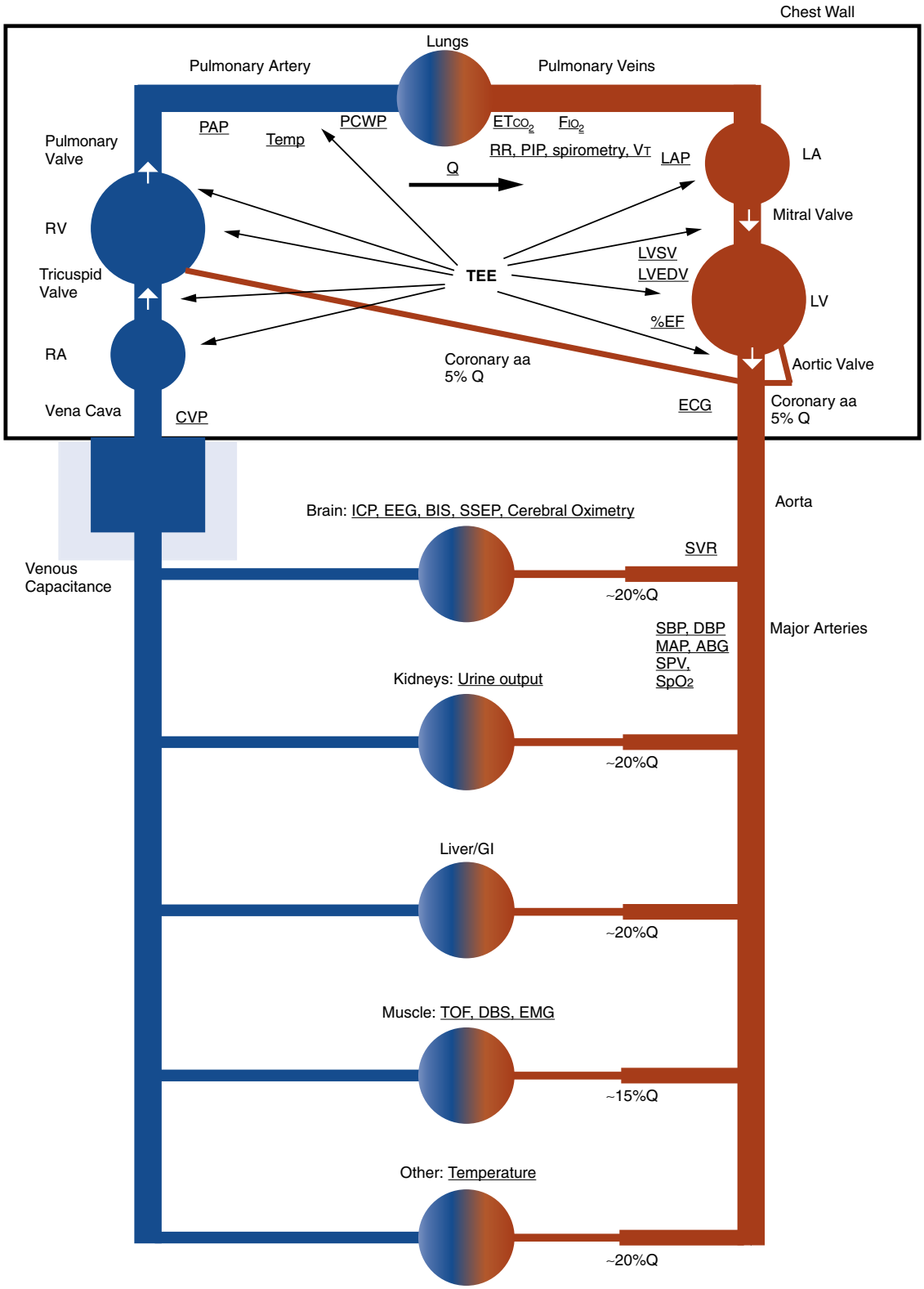

Chest Wall
Lungs
Pulmonary Artery
Pulmonary Veins
PAP
Temp
PCWP
ETCO2
FIO2
RR, PIP, spirometry, VT
LAP
LA
Pulmonary Valve
Q
RV
Mitral Valve
LVSV
LVEDV
Tricuspid Valve
TEE
LV
%EF
RA
Coronary aa 5% Q
Aortic Valve
Vena Cava
CVP
ECG
Coronary aa 5% Q
Aorta
Brain: ICP, EEG, BIS, SSEP, Cerebral Oximetry
SVR
Venous Capacitance
~20%Q
SBP, DBP MAP, ABG SPV, SpO2
Major Arteries
Kidneys: Urine output
~20%Q
Liver/GI
~20%Q
Muscle: TOF, DBS, EMG
~15%Q
Other: Temperature
~20%Q

data from multiple devices must be understood in order to confirm the diagnosis and follow the prescribed treatment.

RESPIRATORY SYSTEM

Oxygen (O_2) is a colorless, odorless gas critical for cellular respiration. Lack of delivery of oxygen to tissues will result in cellular death. Carbon dioxide is a consequence of cellular metabolism and must be removed from the tissues to maintain acid-based homeostasis. This section will review monitors of patient oxygenation and patient ventilation.

Oxygenation

Inspired Oxygen

Inspired oxygen content (or fraction of inspired O_2, Fio_2) can be measured by a variety of methods. Anesthesia machines most commonly use an amperometric sensor to measure O_2 in the fresh gas flow. Calibration is recommended, as the sensor, which is basically a fuel cell that consumes oxygen and generates current, has "drift"; that is, the readings in a constant concentration of oxygen will not be constant. It is a slow responding device, meaning that it cannot be used to measure inspired/expired oxygen, which rapidly changes. An alternative method of measuring inspired oxygen uses the fact that oxygen is paramagnetic. A paramagnetic oxygen sensor can be autocalibrating, using room air as a source of 21% O_2. The gradient between the sample and the room air can be measured by a pressure transducer or a torsion wire. The fast response time allows the measurement of both inspired and expired oxygen content. Measuring expired O_2 (Feo_2) concentration during preoxygenation (just prior to induction of anesthesia) also allows the determination of complete preoxygenation/denitrogenation.

Pulse Oximetry

The pulse oximeter provides a continuous noninvasive estimate of arterial hemoglobin saturation (Sao_2) by analyzing red and infrared light transmitted through living tissue, such as a fingertip or earlobe (Fig. 20.2).[3] It uses the physical principle known as *Beer's law*, which relates the concentration of a dissolved substance to the log of the ratio of the incident and transmitted light intensity through a known distance. Because of the differing amounts of red and infrared light absorbed by oxyhemoglobin and reduced hemoglobin the device makes this estimate using only two wavelengths of light emitted by light-emitting diodes, or LEDs (red at 660 nm and infrared at 940 nm) detected by a photodiode. The device determines the signal related to arterial hemoglobin saturation by analyzing the pulsatile component of the absorbents, hence the name pulse oximeter (Fig. 20.3). The device continuously determines the ratio of pulse-added red to pulse-added infrared light absorbance:

Eq. 1

$$R = \frac{AC_{red}/DC_{red}}{AC_{IR}/DC_{IR}}$$

This ratio (R) of absorbance is empirically calibrated to estimate Sao_2. That is, the device uses Sao_2 data derived from human volunteers to determine the relationship between the pulse oximeter saturation (Spo_2) and the ratio of light absorbance (Fig. 20.4).

Dyes and Dyshemoglobins

Standard pulse oximeters using two wavelengths of light can determine functional saturation, that is, the percent of oxyhemoglobin (HbO_2) over HbO_2 plus reduced hemoglobin (Hb). Two equations are used to solve for two unknowns:

Eq. 2

$$Sao_2 = \frac{HbO_2}{HbO_2 + Hb}$$

Functional saturation

Eq. 3

$$So_2 = \frac{HbO_2}{COHb + MetHb + HbO_2 + Hb}$$

Fractional saturation

Fig. 20.1 A summary of monitors and the circulation. Anatomic features are listed around the periphery, with monitored variables central and underlined (see Table 20.1 for normal values of monitored variables). The blood flows in a circuit with a cardiac output of roughly 20% each to the brain, kidneys, liver, GI tract, muscle mass, and other organs (skin, etc.). The systemic vascular resistance (SVR) is a calculated variable, reflecting the totality of blood flow and pressure. Roughly 70% of the blood is on the venous side. The venous capacitance is highly variable and acts as a buffer for changes in volume. Some variables may be measured or derived, depending on methodology. *aa*, Arteries; *ABG*, arterial blood gas; *BIS*, bispectral index; *CVP*, central venous pressure; *DBP*, diastolic blood pressure; *DBS*, double burst stimulation; *ECG*, electrocardiogram; *EEG*, electroencephalography; *EF*, ejection fraction; *EMG*, electromyography; *$ETco_2$*, end-tidal CO_2; *Fio_2*, fraction of inspired oxygen; *GI*, gastrointestinal; *ICP*, intracranial pressure; *LA*, left atrium; *LAP*, left atrial pressure; *LV*, left ventricle; *LVEDV*, left ventricular end-diastolic volume; *LVSV*, left ventricular systolic volume; *MAP*, mean arterial pressure; *PAP*, pulmonary artery pressure; *PCWP*, pulmonary capillary wedge pressure; *PIP*, peak inspiratory pressure; *Q*, cardiac output; *RA*, right atrium; *RR*, respiratory rate; *RV*, right ventricle; *SBP*, systolic blood pressure; *Spo_2*, arterial O_2 saturation; *SPV*, systolic pressure variation; *SSEP*, somatosensory evoked potential; *TEE*, transesophageal echocardiography; *TOF*, train-of-four; *Vt*, tidal volume.

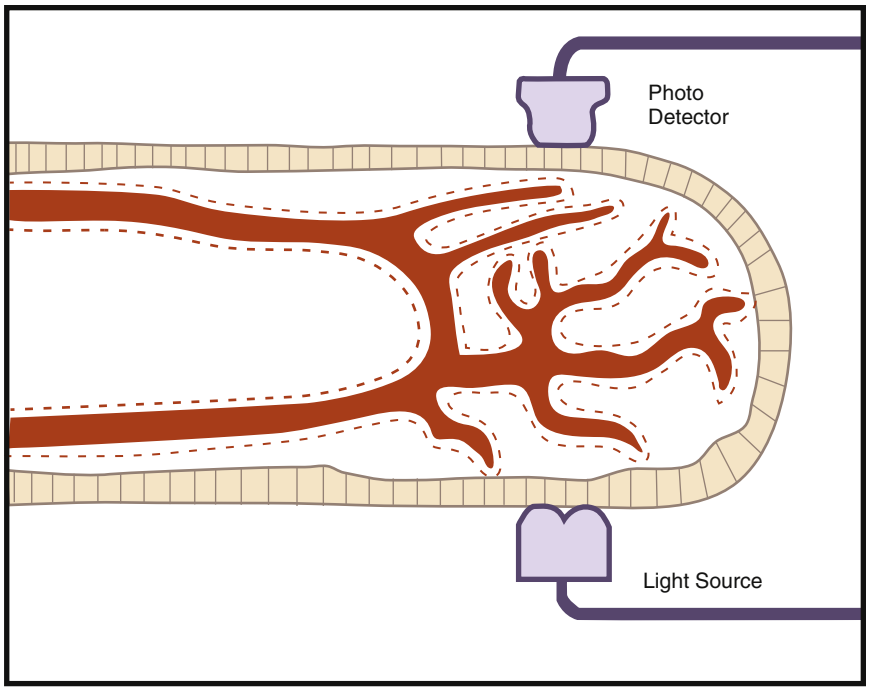


Fig. 20.2 Pulse oximeter. Pulse oximeters (Spo_2) provide an estimate of arterial hemoglobin saturation (Sao_2) by analyzing the pulsatile absorbance of two frequencies of light (660nm and 940 nm) emitted by light-emitting diodes (LEDs), the light source, and detected by a photodiode on the opposite side of the tissue bed of the finger. The photodiode generates a current when it detects any light: red or infrared, or room light. For that reason, the photodiode alternates a pulse of red light and room light with a pulse of infrared light and the room light. Then, when both LEDs are off, it measures room light alone, then subtracts the room light signal from the previous two signals, continuously correcting for changes in room light. It thereby derives a signal associated with the pulsing LED signals. The signal may be improved by decreasing ambient light by covering the probe with an opaque material.

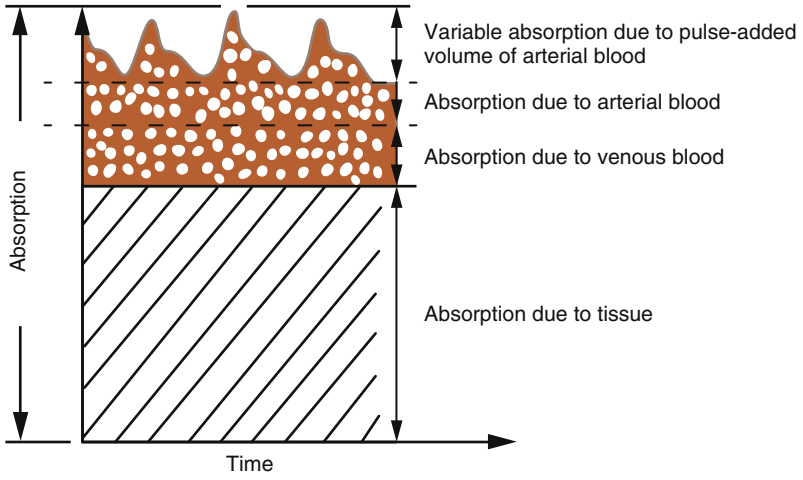


Fig. 20.3 Tissue absorbances. As light is transmitted through tissues and detected by the photodiode it is absorbed by all the tissues between the light source and the detector, that is, skin, muscle, bone, and blood. Because the pulse oximeter wants to determine a signal related only to arterial blood, it analyzes only the pulsatile absorbance noted at the top of the figure. The pulse oximeter, therefore, makes the assumption that whatever is pulsing must be arterial blood. In most cases this is true, but in some situations (e.g., patient motion) there can be large venous pulsations that can produce erroneously low saturation values.

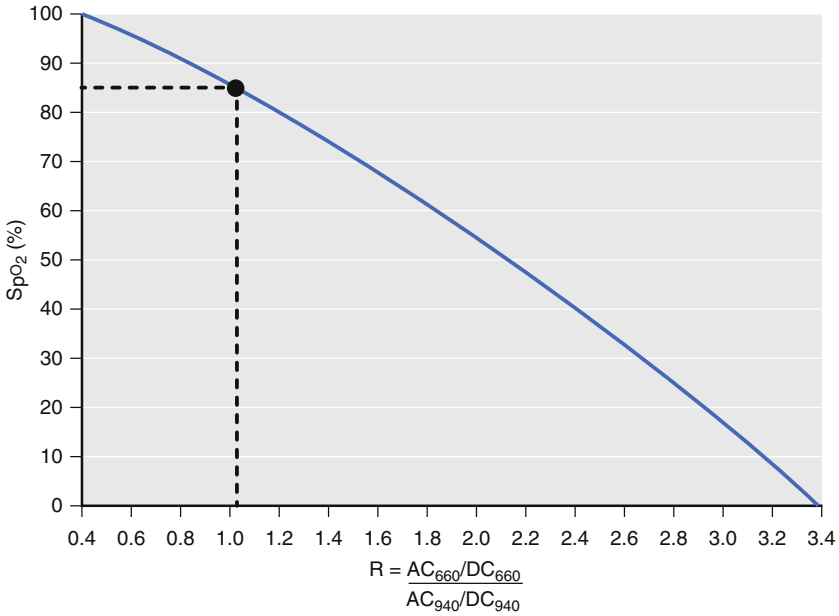


Fig. 20.4 Pulse oximeter calibration curve. Because of all the absorbances between the light source and the photo detector, the concentrations of oxyhemoglobin and reduced hemoglobin cannot be measured specifically; that is, the exact path length of the light is unknown. Using the pulse-added absorbance from both the infrared and red light source, a ratio of these pulse-added absorbances (see Eq. 1) can be empirically related to Spo_2. That is, volunteer subjects breathe low inspired oxygen concentrations to produce desaturation while blood samples are obtained for Sao_2 measurement. These Sao_2 measurements are calibrated to the ratio of red to infrared pulsatile absorbance to develop the calibration curve, which is incorporated into the device. Note the ratio ranges from approximately 0.4 to 3.4 as the saturation decreases from 100% to 0%. The volunteer data are only available from 100% saturation down to 75% and all values below that are extrapolated from the data. Note that at approximately Spo_2 85% the ratio of the two absorbances is 1.0. Therefore, any situation that causes the ratio of pulse-added red to pulse-added infrared light to tend toward a ratio of 1.0 produces a saturation of approximately 85%. This occurs with motion artifact, dyes, and methemoglobin toxicity. *AC*, Alternating current; *DC*, direct current.

Pulse oximeters are calibrated using human volunteers who have little carboxyhemoglobin (COHb) or methemoglobin (MetHb). Therefore, if either carboxyhemoglobin (carbon monoxide poisoning) or methemoglobin (methemoglobin toxicity from benzocaine, for example) is present the devices will produce an erroneous saturation value. In the case of carboxyhemoglobin, because it is red and absorbs red light similarly to that of oxyhemoglobin, the pulse oximeter will give a reading approximately equal to the sum of carboxyhemoglobin and oxyhemoglobin, giving the impression the patient is adequately saturated with oxyhemoglobin even when he has severe carboxyhemoglobin toxicity. In the case of methemoglobin, which is dark and absorbs both red and infrared light to a high degree, it causes the ratio of absorbance to tend toward one. From the calibration curve it can be seen that a ratio of 1 will produce an Spo_2 of 85% (see Fig. 20.4 [calibration curve]). Therefore, if there is a significant (>20%) amount of methemoglobin present, the pulse oximeter value will tend toward 85%. Thus, it will produce falsely low values when the patient has high Sao_2, and falsely "high" values of 85% when the patient is severely hypoxemic. Dyes produce similar errors, as does methemoglobin; that is, they force the saturation toward 85%, although because they are cleared from the circulation quickly this error is only transient. Newer eight-wavelength pulse oximeters are available that can detect all saturations: oxyhemoglobin, carboxyhemoglobin, and methemoglobin.[4] Motion artifact will also cause the Spo_2 value to tend toward 85% because the motion artifact produces noise in the numerator and denominator, the ratio R is forced toward 1.0, as occurs with methemoglobin. In fact, any situation that results in a small signal-to-noise ratio may cause the Spo_2 to trend toward 85%.[3]

Ventilation

The respiratory rate, pattern, and depth are all important descriptors of ventilation. Qualitatively, ventilation depth and pattern can be observed by chest rise, auscultation, or reexpansion of the rebreathing bag on the anesthesia machine. In any acute situation in which adequacy of ventilation is an issue, eliminating monitoring devices altogether and going to the source by listening for bilateral clear breath sounds with a stethoscope should be done immediately. This may rule out tension pneumothorax, acute bronchospasm, endobronchial intubation, pulmonary edema, or absence of ventilation altogether.

Airway Pressures

Increases in peak airway pressure, also called *peak inspiratory pressure (PIP),* merit investigation as they imply an acute increase in airflow resistance or reduction in lung/chest wall compliance. Setting the ventilator to produce an end-inspiratory pause will allow measurement of a plateau pressure, which will be a reflection of lung/chest wall compliance only. The difference between peak and plateau pressure will be a reflection of airway resistance only. If peak airway pressure is increased and plateau pressure is increased a similar amount, this signifies reduced lung/chest wall compliance, which can be caused by conditions such as tension pneumothorax or pulmonary edema. Other clinical findings can help to determine the specific cause, such as accompanying arterial hypotension with tension pneumothorax or visible frothy fluid in the endotracheal tube (ETT) with pulmonary edema. External obstruction of an ETT (from a patient biting on the tube or tube kinking) can cause an increase in PIP with a lesser increase in the plateau pressure. This can be easily ruled out by passing a suction catheter down the ETT. A loss or abrupt decrease in airway pressure is not specific but can indicate a variety of major problems, including circuit disconnections, leaks, extubation of the trachea, failure to deliver fresh gases, failure to set the ventilator properly, excess scavenging, and other anesthesia machine issues.[5] Airway pressure can be measured with analog gauges or electronic pressure transducers.

Tidal Volume

One large study demonstrated improved pulmonary outcomes after major abdominal surgery by using tidal volume of 6 to 8 mL/kg of ideal body weight (based on height and gender) as well as recruitment maneuvers and positive end-expiratory pressure (PEEP).[6] These settings are similar to those associated with improved outcomes in patients with acute respiratory distress syndrome (also see Chapter 41). Once these tidal volumes are set, the respiratory rate should be adjusted to maintain an end-tidal CO_2 ($ETco_2$) in the normal range of 35 to 40 mm Hg. Modern ventilators use a variety of modes to achieve this tidal volume (Fig. 20.5). Most ventilators have pressure limits that will alert when peak pressures are exceeded owing to increased airway resistance in the circuit or in the patient (Fig. 20.6). Monitoring the tidal volume and peak airway pressure together will enable the practitioner to quickly detect any changes in resistance to airflow due to resistance in the system or decreased compliance in the lung or chest wall (Fig. 20.7). Tidal volumes can be measured by mechanical vanes rotating in the gas stream, pressure gradients across a flow restriction (fixed or variable), and hot wire anemometers.

All anesthesia machines require a "disconnect" alarm, usually tied to the airway pressure reading. Inadequate ventilation can occur despite a nominally normal pressure. When using pressure-controlled ventilation, a significant change in ventilator volume can occur without an alarm condition occurring. Mechanical alarms and indicators of ventilation do not ensure tracheal intubation. An esophageal intubation can return "adequate" pressures and volumes and, with transmission of sounds, appear to have bilateral breath sounds. With an intact circulation, measurement of expired CO_2 is the best monitor of ventilation as discussed in detail in the next section.

Capnography/End-Tidal CO_2

Capnography is the analysis of the continuous waveform of expired CO_2. Gas is continuously sampled from the ventilator circuit just on the patient side of the Y connector. The gas sample is drawn through a small tube into an infrared analyzer and the CO_2 waveform is displayed on the physiologic monitor (Figs. 20.8 and 20.9). Carbon dioxide generated in the tissues is delivered to the right side of the heart through the venous system into the lungs via the pulmonary arteries. Exchange of the carbon dioxide into the alveolar space is fairly efficient because CO_2 has 20 times the solubility in water as does oxygen. Therefore, well-perfused alveoli achieve equilibrium with carbon dioxide in the blood. During expiration alveolar gas leaves the lungs, exiting the trachea through the ETT where the aspirated gas is sampled by the capnometer, producing a peak expired CO_2 close to the arterial carbon dioxide tension ($Paco_2$) in healthy patients ($ETco_2$ is usually 3 to 5 mm Hg less than $Paco_2$ during general anesthesia).

The respiratory tidal volume is composed of alveolar gas volume and dead space. Approximately one third of the tidal volume in healthy patients is dead space (see Fig. 20.8 for details). Because the inspired gas contains no carbon dioxide (unless the CO_2 absorber is malfunctioning and allowing rebreathing of CO_2 to occur), dead space gases will not contain carbon dioxide. When expiration begins in the respiratory cycle, the first gas detected is apparatus dead space, followed by the anatomic dead space. Neither of these spaces contains carbon dioxide,

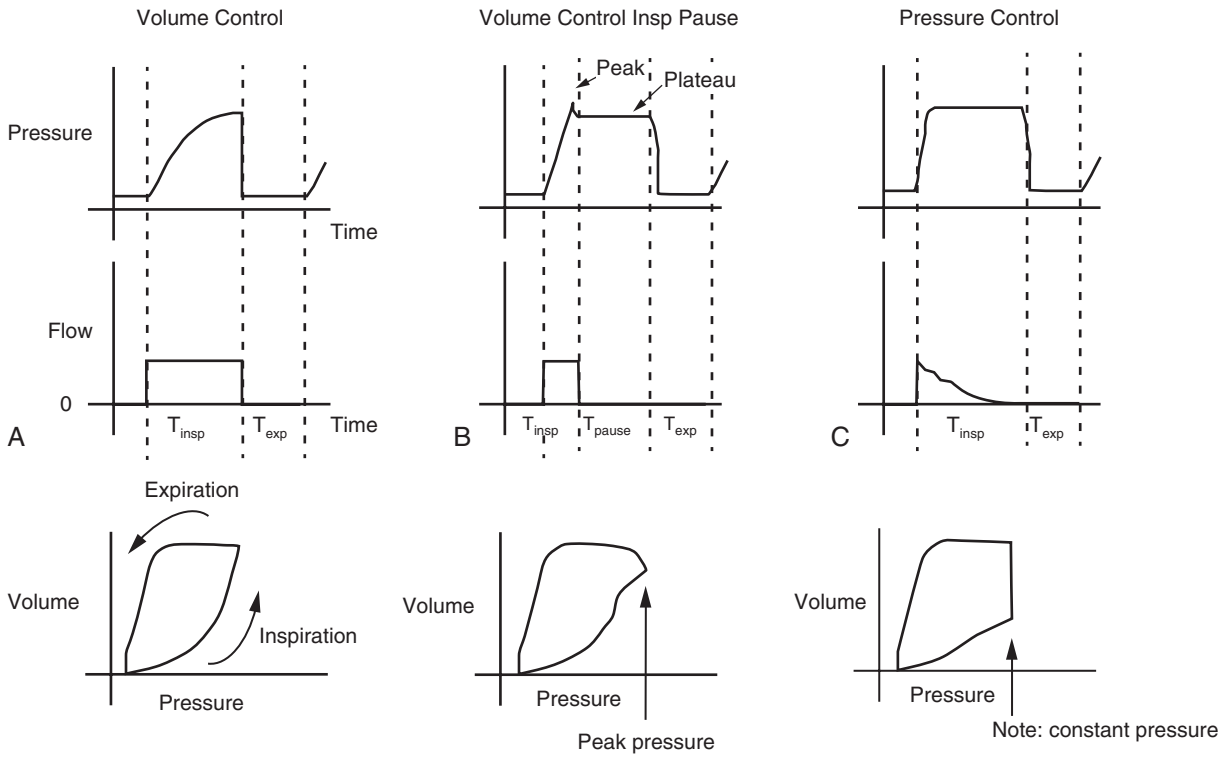


Fig. 20.5 Ventilator pressure time curves. Three commonly employed modes of ventilation generate characteristic curves. (A) In volume-controlled ventilation, the pressure and volume smoothly increase until expiration (which is passive). (B) With the addition of an inspiratory pause, the pressure drops with minimal change in volume. (C) In pressure-controlled ventilation, the pressure is constant as volume increases, until expiration. Only four variables determine volume-based mechanical ventilation: (1) inspiratory time (T_{insp}), (2) inspiratory pause time (T_{pause}), (3) expiratory time (T_{exp},) and (4) inspiratory flow rate. In ventilators that have control loops, faulty monitoring can lead to inadequate or hazardous ventilation. The compliance of the lung can be measured by dividing the tidal volume by the distending pressure (peak or plateau pressure minus PEEP). Dynamic compliance reflects the compliance during airflow, so it includes the resistance of the endotracheal tube as well as the compliance of the lungs. With an inspiratory pause (B), both the dynamic compliance and the static compliance (of the lungs and chest wall) can be measured by using either the peak pressure or the plateau pressure, respectively. The pressure-volume loops are different for the various ventilation modes as well. *PEEP,* Positive end-expiratory pressure.

so the capnogram will remain at zero during the initial phase I of the capnogram (see Fig. 20.9). As the gas from the alveolar space (well perfused) and the alveolar dead space mix and are detected at the sampling tube, the carbon dioxide waveform will increase from zero up to a plateau value producing a rough square wave until inspiration begins and the CO_2 waveform immediately returns to zero. The final plateau value of the capnogram ($ETco_2$) will approximately equal the arterial CO_2 value if there is no alveolar dead space. The $ETco_2$ value will always be less than the $Paco_2$ value: the degree of this gradient will be in direct proportion to the amount of alveolar dead space in the expired volume, relative to the alveolar gas. The larger the proportion of dead space, the smaller the $ETco_2$ value. Common abnormalities of the capnogram are depicted in Fig. 20.10.

Alveolar dead space may be increased in chronic obstructive lung disease (also see Chapter 41) in which large emphysematous areas of the lung increase the alveolar dead space and produce a large gradient between the $ETco_2$ and $Paco_2$. In other situations, acute changes in alveolar dead space occur. The classic case involves pulmonary emboli that completely obstruct blood flow to some capillaries, causing an acute increase in alveolar dead space and resulting in an acute decrease in the $ETco_2$ value (Fig. 20.10D). Increased dead space can also occur when there is a ventilation-perfusion mismatch causing decreased perfusion to well-ventilated areas of the lung. For example, when a patient is placed in the lateral position (see Chapter 19) the dependent lung is well perfused and ventilated but the elevated lung is less well perfused and therefore has more alveolar dead

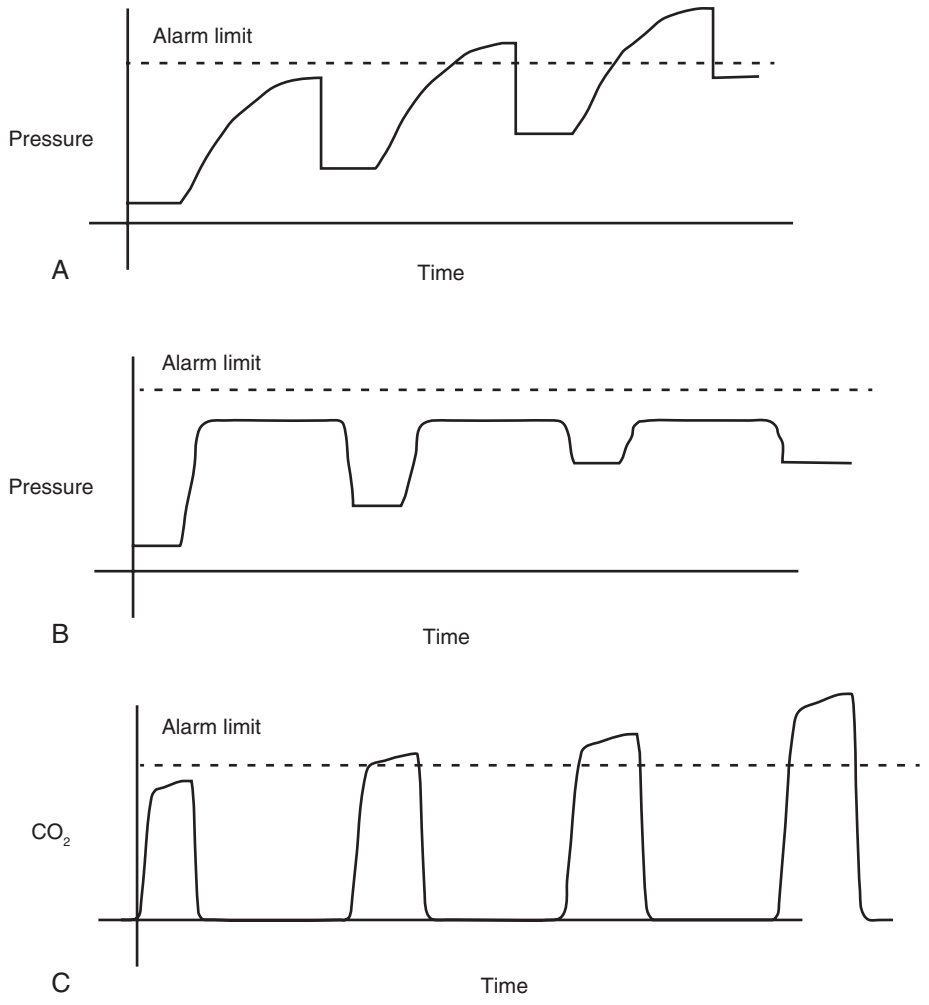


Fig. 20.6 Stacking breaths. In both volume control (A) and pressure control (B) ventilation, insufficient expiratory time leads to "stacking" of breaths and changes in the pressure waveform. In the case of volume control ventilation, the pressure can increase, triggering an alarm. With pressure control ventilation, tidal volumes decrease and pressure remains constant (this may trigger a high PEEP alarm). (C) The capnogram also demonstrates decreased ventilation (increasing CO_2) and a change in the shape of the CO_2 curve. *PEEP*, Positive end-expiratory pressure.

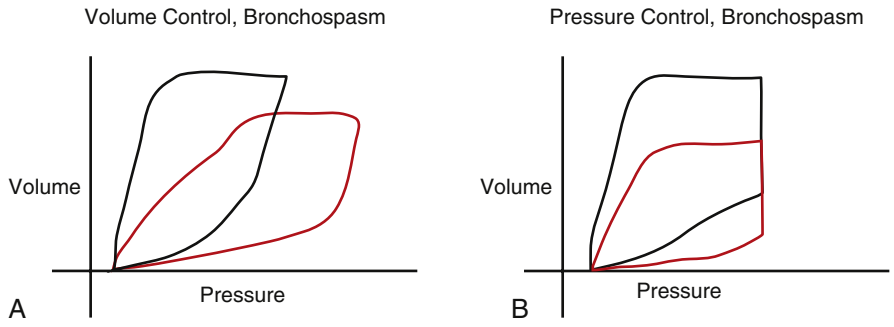


Fig. 20.7 Bronchospasm. With volume control ventilation (A), the set tidal volume is attempted to be delivered, with an increase in pressure. This results in the pressure volume loop being shifted to the right and flattened. In pressure control ventilation (B), the stiffness of the lung results in a decreased tidal volume, without a change in the pressure (because that is the ventilator setpoint).

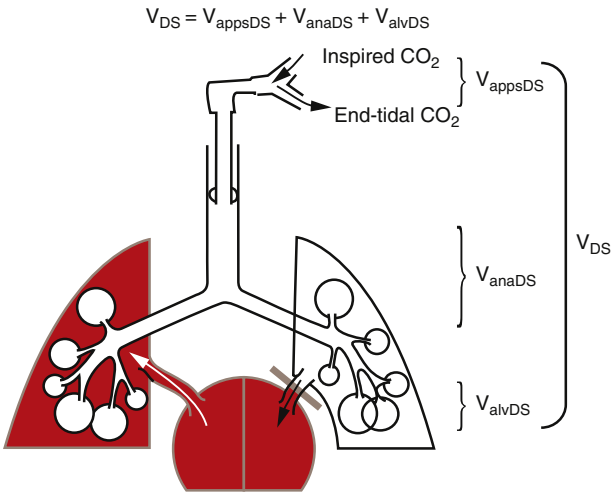


Fig. 20.8 Apparatus, anatomic, and alveolar dead space. To interpret the capnogram one must first understand alveolar dead space and its components. This schematic shows the heart, lung, and ventilator circuit up to the Y connector. Dead space volume (V_{DS}) is defined as any portion of the tidal volume that does not participate in gas exchange. V_{DS} is further divided into three components: apparatus dead space (V_{appsDS}), anatomic dead space (V_{anaDS}), and alveolar dead space (V_{alvDS}). Apparatus dead space is the volume of gas between the Y connector and the end of the endotracheal tube. Anatomic dead space is the dead space of the trachea and all connecting airways down to the alveoli. In this figure the lung on the right has no blood flow so all those alveoli are not perfused and at the end of expiration will have zero carbon dioxide. The lung on the left is well perfused and those alveoli can be assumed at end of expiration to equilibrate to the arterial carbon dioxide ($Paco_2$) value. The expired mixture of the alveolar gas ($Paco_2$) and alveolar dead space gas (no CO_2) produces the end-tidal CO_2 ($ETco_2$).

space, again producing a decrease in the $ETco_2$ value compared to the $Paco_2$. Finally, a progressive increase in alveolar dead space may occur because of a global lack of perfusion when the cardiac output decreases (see Fig. 20.10D). For example, if cardiac output suddenly decreases from 5 L/min to 2.5 L/min with alveolar ventilation remaining constant, less blood will flow per unit time to perfuse the same number of ventilated alveoli. The result is an increase in alveolar dead space and a decrease in $ETco_2$. For this reason the $ETco_2$ capnogram is often referred to as the "poor man's measure of cardiac output." Any significant decrease in cardiac output is associated with a decrease in $ETco_2$ (see Fig. 20.10D). In the most acute situation of cardiac arrest when cardiopulmonary resuscitation (CPR) is initiated (also see Chapter 45), the most important monitor to follow to assure the adequacy of chest compressions during CPR is the capnogram. A capnogram showing $ETco_2$ greater than 20 mm Hg with every ventilated breath during CPR ensures both ventilation and perfusion of the lung. If the capnogram shows $ETco_2$ less than 20 mm Hg during chest compressions, it is likely that cardiac output is inadequate. In this situation, the CPR should be adjusted until $ETco_2$ is more than 20 mm Hg. The other advantage of monitoring the capnogram during CPR is that there is no motion artifact associated with a capnogram unlike nearly every other monitor during CPR or chest compressions, such as electrocardiogram (ECG) and the pulse oximeter. Because of the utility of the continuous capnogram waveform assuring that there is intact ventilation and perfusion (i.e., cardiac output), some consider the capnogram to be the most important monitor used during general anesthesia.

Although the sampling tube can be placed on nasal cannula or around the mouth in patients whose tracheas are not intubated, a reliable capnographic waveform is achieved only in a patient whose trachea is intubated. In nonclosed systems (where the sampling tube is placed by the airway under a mask or a nasal cannula), there may be aspiration of room air (with no carbon dioxide), which will dilute the capnographic sample.

CIRCULATORY SYSTEM

Multiple characteristics of the circulation can be measured, including the heart rate, ECG, blood pressure, urine output, central venous pressures (CVPs), pulmonary artery pressures (PAPs), cardiac output, and systolic pressure variation (SPV) (Table 20.1). Some of these are difficult to measure and all require interpretation. Many important variables cannot be measured, such as venous capacitance, organ blood flow/perfusion, and circulating blood volume. Other values are derived from combinations of measured values (e.g., stroke volume, vascular resistance). No single characteristic determines adequacy of perfusion, and a solid understanding of the underlying physiology is necessary to interpret even the simplest monitor.

Measurement of the Electrocardiogram

Continuous monitoring of the ECG is one of the standards of the ASA, yielding information on heart rate and rhythm. Simply, the ECG is the electrical activity of the heart, measured at the body surface. Technically, it is the net dipole moment of the heart displayed on the vertical axis in millivolts versus time on the horizontal axis. The operating room is an electrically noisy environment and subtle ECG changes can be obscured by the filtering, and artifacts (false positives) can be introduced. ECG monitors in the operating room have a filtering mode that reduces the electrical interference, but they may produce artifacts that look like concerning ECG changes, such as T-wave changes. These monitors also have a "diagnostic mode" that removes all filtering and the artifacts it may induce.

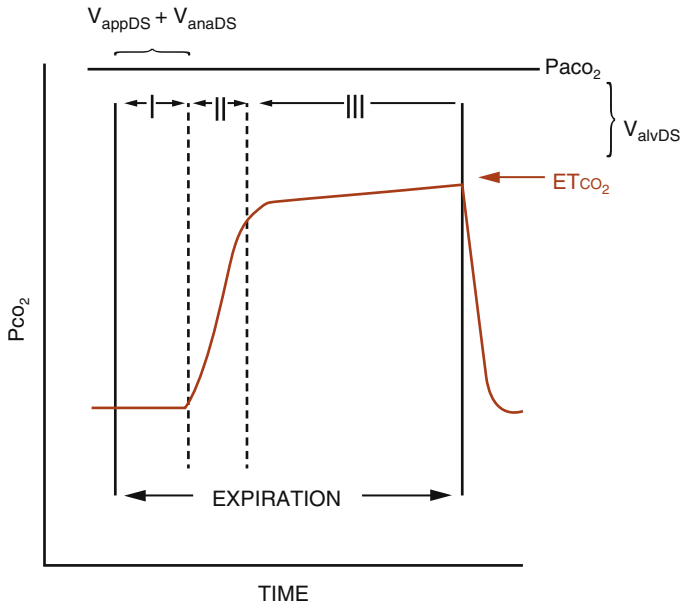


Fig. 20.9 Normal capnogram. A capnogram is a continuous tracing of the carbon dioxide concentration sampled at the Y connector on an intubated, ventilated patient and plotted versus time during the inspiratory and expiratory cycle. It can be divided into three phases. Phase I is the beginning of expiration when the apparatus dead space (V_{appDS}) and anatomic dead space (V_{anaDS}) are being sampled, both of which have zero carbon dioxide. Phase II starts when the mixed alveolar gases are detected and the capnogram rises up and reaches a plateau value. Phase III has only a slight rise as the mixed alveolar gases are sampled during the end of the expiratory cycle. With the initiation of inspiration the CO_2 value drops to zero and stays at zero until the next expiration. Note the end peak value is the end-tidal CO_2 ($ETco_2$). The $ETco_2$ is always lower than the $Paco_2$; the magnitude of this gap is directly proportional to the ratio of alveolar dead space gas to alveolar gas.

Therefore, if the ECG on the monitor looks different from the preoperative ECG, it is best to switch the filters off and place the monitor in the diagnostic mode to see if those changes are real. A three-lead system, which uses electrodes placed on both shoulders and left abdomen below the rib cage, provides leads I, II, and III. The preferred method is a five-lead system, using a single precordial lead placed in the V_5 position (Fig. 20.11). A majority of the dysrhythmias and ischemia seen during anesthesia can be detected by a combination of monitoring leads II and V_5.[7] Monitoring the ECG allows dysrhythmias, such as heart block, atrial fibrillation, ventricular fibrillation, bradycardia, asystole, and tachycardia to be diagnosed (and treatment evaluated). The ECG can also aid in diagnosing myocardial ischemia and electrolyte disturbances (Table 20.2).

Blood Pressure and Flow

The primary utility of the circulatory system is to maintain a constant supply of blood flow to all organs to allow them to function and maintain aerobic metabolism. This system is composed of a basic pump, the heart; conduits, the blood vessels; and resistance as blood flows through the microcirculation. This is an Ohm's law system, V = IR, where V (blood pressure) equals blood flow (cardiac output) multiplied by resistance (systemic vascular resistance). The pressure difference across the circulation of any organ is defined as the perfusion pressure, that is, the pressure on the upstream side of that system minus the pressure on the downstream side. For the systemic circulation, the pressure difference is the mean arterial pressure (MAP) minus the CVP; and for the pulmonary circulation it is the mean pulmonary artery pressure (MPAP) minus the left atrial pressure, usually estimated by a pulmonary artery wedge pressure (PAWP). Mean pressure is approximated by the formula:

Eq. 4

$$\text{Mean BP} = \text{Diastolic BP} + \frac{(\text{Systolic BP} - \text{Diastolic BP})}{3}$$

For the most vital of organs, the brain and heart, these perfusion pressures are slightly different. For the brain it is the MAP minus the intracranial pressure (ICP) (see "Intracranial Pressure Monitoring") and for the heart

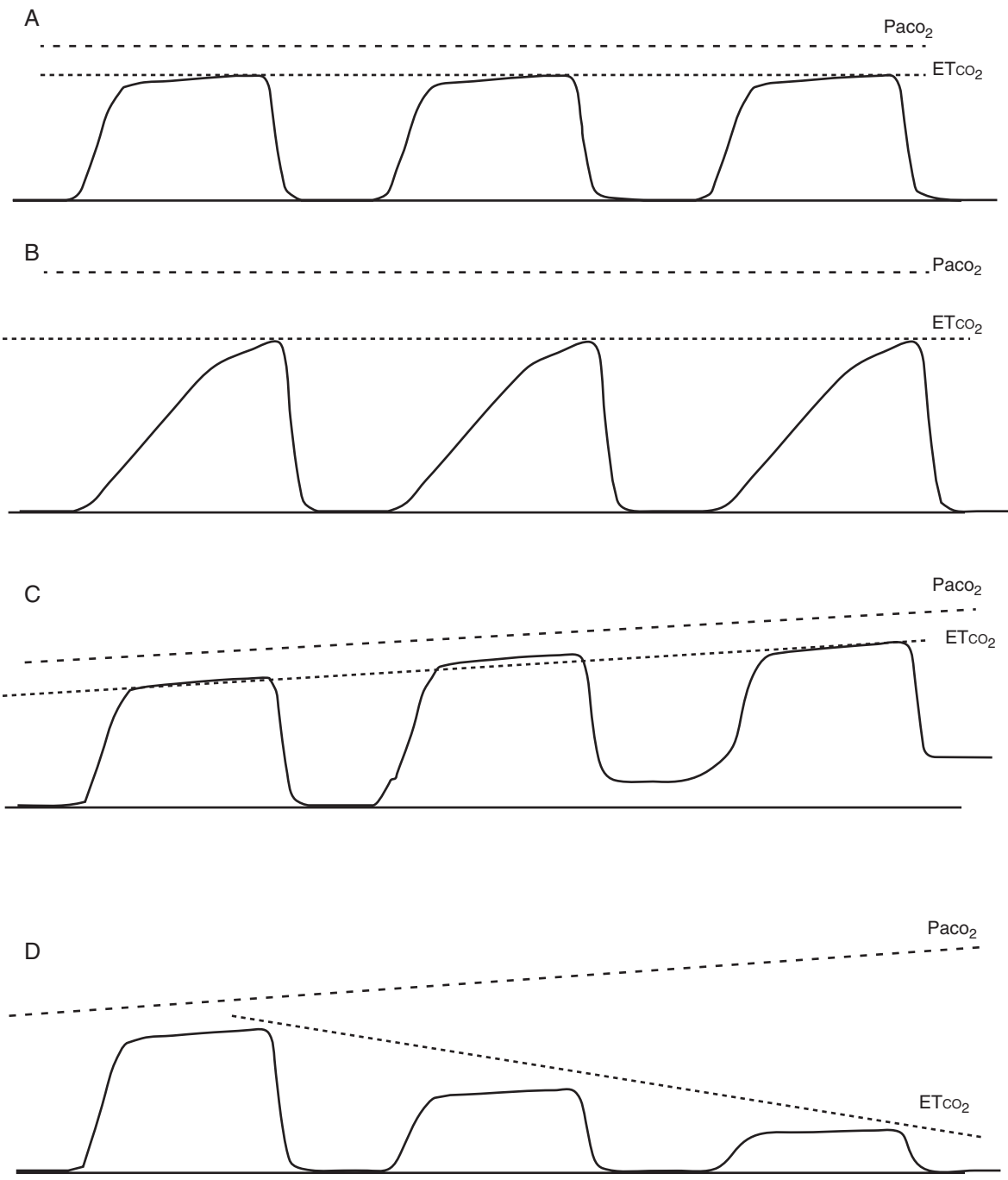


Fig. 20.10 Capnogram abnormalities. (A) The normal $Paco_2$ to $ETco_2$ gradient is 2 to 5 mm Hg. (B) This rightward slant of the initiation of the alveolar gas detection is seen when there is the presence of asthma or chronic obstructive pulmonary disease. The greater the slant to the right, the worse the expiratory airway resistance. The gradient of $Paco_2$ to $ETco_2$ has increased. (C) This waveform shows a progressive rise in the baseline CO_2 value; that is, there is a progressive increase in inspiratory carbon dioxide, noting a CO_2 rebreathing most commonly due to an exhausted CO_2 absorber. (D) This waveform signifies a progressive drop in the $ETco_2$, that is, a decrease in the height of the waveform. This form is noted whenever there is abrupt reduction in pulmonary blood flow (cardiac output), as occurs with a pulmonary embolism or a cardiac arrest. *$ETco_2$*, End-tidal carbon dioxide.

Table 20.1 Normal Values

Measured Variable (Abbreviation)	Normal Value
Systolic blood pressure (SBP)	90-140 mm Hg
Diastolic blood pressure (DBP)	60-90 mm Hg
Mean arterial pressure (MAP)	70-105 mm Hg
Systolic pressure variation (SPV)	5 mm Hg
Pulse pressure variation (PPV)	10%-13%
Central venous pressure (CVP)	2-6 mm Hg
Right ventricular pressure	15-30/2-8 mm Hg
Pulmonary artery pressure (PAP)	15-30/5-15 mm Hg
Mean pulmonary artery pressure	9-20 mm Hg
Pulmonary capillary wedge pressure (PCWP)	6-12 mm Hg
Left atrial pressure (LAP)	4-12 mm Hg
Heart rate (HR)	60-90 beats/min
Arterial O_2 saturation (Spo_2)	95%-100%
Cardiac output (Q or CO)	4-8 L/m
Cardiac index (CI)	2.4-4.0 L/min/m^2
Ejection fraction (EF)	55%-70%
End-diastolic volume	65-240 mL
Calculated Values	
Stroke volume (SV), stroke volume index (SVI)	50-100 mL/beat, 33-47 mL/m^2/beat
Systemic vascular resistance (SVR)	800-1300 dynes · sec/cm^5
Pulmonary vascular resistance (PVR)	<250 dynes · sec/cm^5
Respiratory Parameters	
Respiratory rate (RR)	12-20 breaths/min
Peak inspiratory pressure (PIP)	15-20 cm H_2O
Tidal volume (V_T)	6-8 mL/kg ideal body weight
End-tidal CO_2 ($ETco_2$)	35-40 mm Hg
Cerebral Parameters	
Intracranial pressure (ICP)	5-15 mm Hg
Electroencephalography (EEG)	Waveform varies by state of consciousness
Somatosensory evoked potential (SSEP)	Normal amplitude and latency
Bispectral index (BIS)	80-100 awake
Muscle Parameters	
Train-of-four (TOF)	4 twitches present
TOF ratio	>0.9
Double burst stimulation (DBS)	No fade
Tetany	No fade
Electromyography (EMG)	Depends on stimulus

The range of normal values for monitored and measured variables in clinical practice are shown in this table. Indices are commonly obtained by dividing the value by the body surface area (BSA).

it is the systemic diastolic pressure minus the right side of the heart, or coronary sinus pressure. Because the heart perfuses itself during diastole, the diastolic pressure is used as the upstream pressure head. In all these systems, blood pressure correlates directly to blood flow assuming the resistance is constant. Unfortunately, in some situations the pressure may be normal but the flow may be reduced because of a high resistance. The reverse is certainly true: as the arterial blood pressure decreases, the blood flow to that organ, or the body in general, will eventually be insufficient to perfuse organs adequately. Therefore, the purpose of constant and repeated measurements of arterial blood pressure is to ensure that hypotension is not occurring. Fig. 20.12 presents a decision tree for the diagnosis of hypotension.

Blood Pressure: Hypotension

Documentation of pulse rate and arterial blood pressure at least every 5 minutes is one of the ASA standards. Yet, despite this long history of measuring arterial blood pressure at frequent intervals, the definition of hypotension based on clinical outcomes was determined relatively recently. In 2009, an association between a MAP

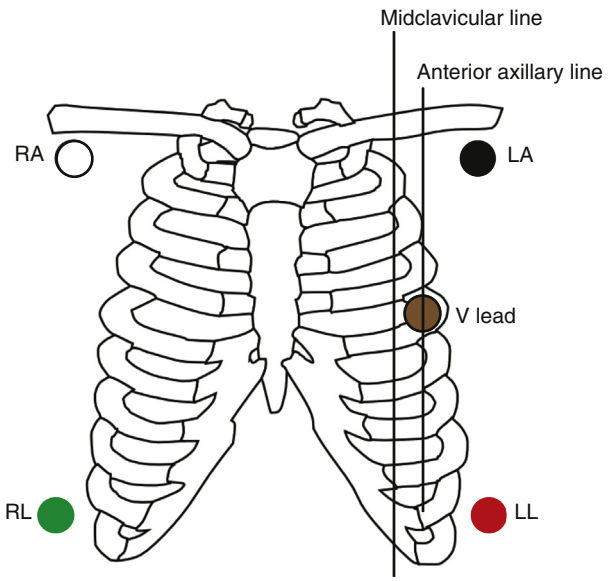


Fig. 20.11 Electrocardiograph lead placement. The limb leads (RA, LA, RL, LL) are placed peripherally on the chest (or on the limbs if available). The V lead is placed in the fifth intercostal space in the anterior axillary line (between the middle of the clavicle and the middle of the axilla).

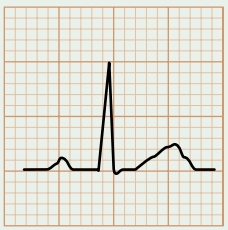
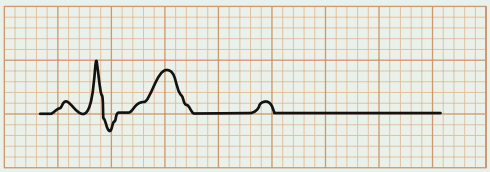
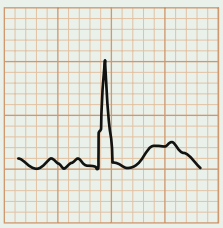
of less than 50 mm Hg or a 40% decrease in MAP from the preoperative arterial blood pressure for more than 10 minutes was associated with an increased incidence of postoperative cardiac events (i.e., troponin increases).[8] In 2013, the cumulative time with a mean MAP less than 55 mm Hg was noted to be associated with progressively increasing incidences of postoperative renal and cardiac injury (increased creatinine and troponin in the postoperative period).[9] In 2015, it was noted that mean MAPs less than approximately 50 mm Hg and less than 60 mm Hg for as short a duration as 5 and 10 minutes, respectively, were associated with an increased 30-day postoperative mortality rate.[10] Therefore, intraoperative hypotension for adults can be defined as mean MAP between 55 to 60 mm Hg.

Noninvasive Blood Pressure

The use of an automatic noninvasive cuff, which measures blood pressure by the oscillometric method, is the routine in anesthetic care. The cuff inflates beyond systolic pressure and slowly deflates until it detects a pulse, continues deflating until it reaches maximal pulsations (the MAP), and further deflates until a pulse is not detected. Although it presents systolic and diastolic blood pressures, the most accurate pressure of an oscillometric cuff is the MAP

III

Table 20.2 Electrocardiographic Monitoring

Situation	Condition	Comments	ECG Display
Normal ECG	P wave, QRS complex, T wave	Function of normal electrolytes and conduction	
Dysrhythmia	Heart block	Drug effect or injury to conduction system	
	Atrial fibrillation	Atrial overdistention, intrinsic disease	

Continued

Table 20.2 Electrocardiographic Monitoring—cont'd

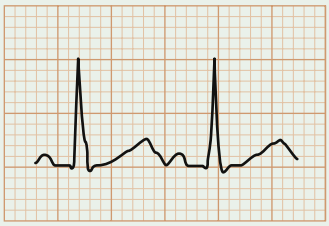
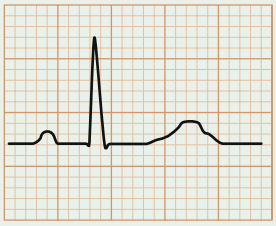
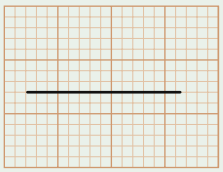
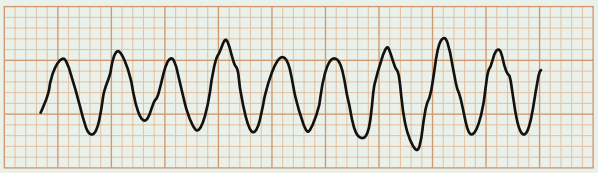
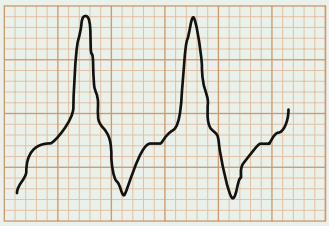
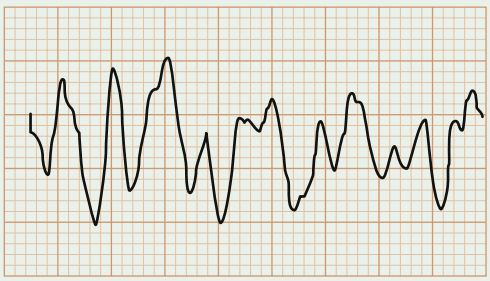
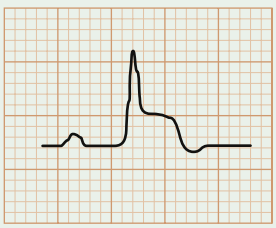
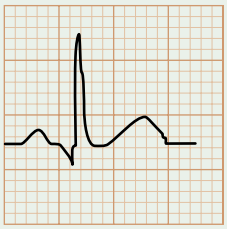
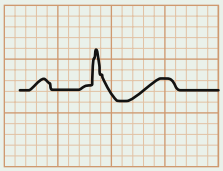
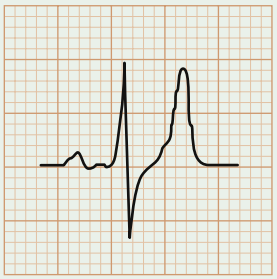
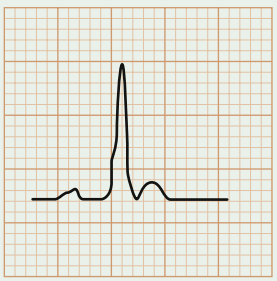
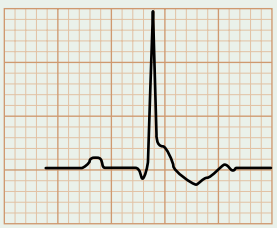
Situation	Condition	Comments	ECG Display
	Sinus tachycardia	Hypovolemia, light anesthesia, hypoxia, hypercarbia	
	Sinus bradycardia	Excess vagal tone, drug effects, hypoxia	
	Asystole	Extreme vagal tone, extreme hypoxia	
	Torsades	Genetic ion channel differences, long QT syndrome, drugs	
	Ventricular tachycardia	Coronary artery disease, mechanical irritation from central line	
	Ventricular fibrillation	Intrinsic myocardial disease	
Active ischemia	ST-segment changes	Ischemia, demand or supply	

Table 20.2 Electrocardiographic Monitoring—cont'd

Situation	Condition	Comments	ECG Display
Completed old infarction	Q waves	Localized to area of injury	
Electrolyte	Hypokalemia	Depressed T wave, U wave	
	Hyperkalemia	Peaked T waves, sinusoidal ECG in the extreme	
	Hypercalcemia	Shortened QT interval, possible J wave	
Temperature	Hypothermia	Osborne J wave	

The electrocardiogram (ECG) changes induced in many physiologic conditions are neither sensitive nor specific but may be confirmatory. The ECG changes for heart rhythm are diagnostic.

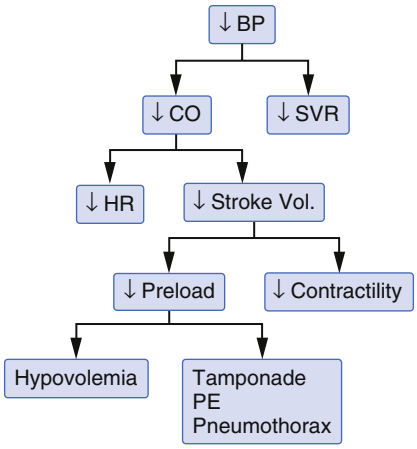


Fig. 20.12 Decision tree diagnosis of acute hypotension. Given that the cardiovascular system is a pressure = flow × resistance "circuit," the diagnosis and acute management of hypotension should follow the principles outlined in the text. This schematic does not account for increasing venous compliance. Decreases in blood pressure (BP) must be due to a drop in either resistance or cardiac output (CO). If there is no obvious reason for an acute drop in resistance (e.g., a sympathectomy from spinal anesthesia), then the CO drop must be due to a decrease in either heart rate (HR) or stroke volume. If the HR has not decreased, then the decrease in stroke volume must be due to a decrease in either preload or contractility. If there is no reason for a drop in contractility and the preload is decreased, it is most commonly due to a lack of relative volume, frequently due to the increase in venous capacitance with anesthetics. One should always keep in mind the three acute mechanical obstructions to blood flow: cardiac tamponade, pulmonary embolism (PE), and tension pneumothorax. *SVR*, Systemic vascular resistance.

(Fig. 20.13).[11] The size of the blood pressure cuff will influence the resultant blood pressure measurement. If the cuff is properly sized its width will be approximately 40% of the circumference of the arm. If the cuff is too small the blood pressure measurement will be too high, if it is too large the measurement will be too low. The oldest noninvasive method of determining blood pressure is the Riva-Rocci technique, which uses a cuff to occlude the arterial flow, slowly deflating the cuff, and noting the pressure when the flow returns (as determined by palpation, Doppler, or any other method). By using a Doppler probe, this method can be successful in patients with hypotension or nonpulsatile flow, including patients with a left ventricular assist device (LVAD).

Invasive Arterial Blood Pressure Monitoring

In cases in which a patient has significant cardiovascular disease or the procedure is expected to have large fluid shifts, a continuous arterial blood pressure measurement from an invasive catheter (usually in the radial artery) is of great value (also see Chapter 41). An arterial line provides beat-to-beat blood pressure measurement, allows for blood sampling of hematocrit, analysis of arterial blood gases, glucose and other blood constituents, and assessment of the intravascular status volume by measuring SPV or other measures of volume responsiveness (see following section, "Measures of Intravascular Volume Responsiveness"). The radial artery is most commonly used because it has the least associated risk and is most easily palpable. Other sites such as the brachial, femoral, or dorsalis pedis arteries may be used. Table 20.3 provides a comparison of different techniques of blood pressure measurement and sites of arterial cannulation. The arterial line is connected to a pressure transducer, which converts the mechanical energy of the arterial pulse into an electrical signal. This fluid-filled tube/transducer setup is an underdamped system that can cause amplification artifact of the systolic blood pressure. This artifact is worsened by an increasing pulse rate and increasing amount of fluid (length of tubing) in the system; however, the MAP should remain accurate.[1] Placement of an arterial line is a sterile procedure, with many technical variations on placement. Some institutions have created protocols to be followed for any line placement.

Measures of Intravascular Volume Responsiveness

Systolic Pressure Variation

The gold standard for determining the adequacy of intravascular volume and cardiac function is transesophageal echocardiography (TEE). Although TEE is extremely useful for diagnosing and in some cases monitoring cardiac performance, it is not necessary or practical during use of most anesthetics. Yet, there are many procedures that cause intravascular volume shifts and lead to questions about cardiac performance, resulting in a need for more information than is available with standard monitors. In situations in which CVP monitoring is not necessary or feasible, substantial information can be derived from analyzing the variations of a continuous arterial pressure waveform associated with positive-pressure ventilation. Measuring the degree to which a positive-pressure breath can result in a decrease in the systolic pressure can predict the responsiveness of a patient to an intravascular fluid challenge[12] (Table 20.4).[13] *Responsiveness* is typically defined as an improvement in stroke volume, blood pressure, or cardiac output.[12] SPV is defined as the difference between maximum and minimum systolic blood pressure during a positive-pressure respiratory cycle. SPV may be manually calculated by freezing the arterial waveform on the physiologic monitor and scrolling up and down, subtracting the average value of arterial peak pressures between positive-pressure breaths from the lower peak arterial pressure values during the breaths (Fig. 20.14). The decrease in arterial pressure associated with positive-pressure ventilation is due in part to the positive intrathoracic pressure transiently impeding venous return to the right side of the heart (see Fig. 20.1). This in turn reduces right-sided heart stroke volume, which in turn reduces left-sided heart stroke volume and arterial blood pressure.

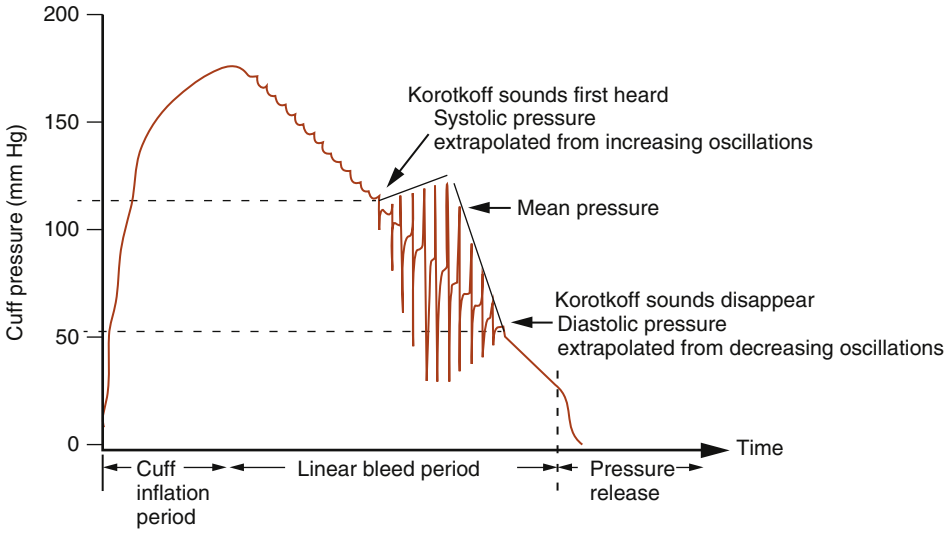


Fig. 20.13 Oscillometric cuff and Korotkoff sounds. Initial Korotkoff sounds correlate with the increasing cuff oscillations. The magnitude of the oscillations increase progressively to a peak, and then decrease. The peak in oscillations is a measure of the mean arterial pressure, which is the most accurate measurement in an oscillometric cuff. The oscillometric systolic and diastolic pressures are inferred from the slope of the envelope around the oscillations. The decreasing oscillations correlate with the diastolic pressure and disappearance of Korotkoff sounds. (Adapted from Ehrenwerth J, Eisenkraft J, Berry J. *Anesthesia Equipment: Principles and Applications.* 2nd ed. Philadelphia: Elsevier Saunders; 2013.)

Table 20.3 Arterial Blood Pressure Measurement

Method	How Obtained	Advantage/Benefit/Indication	Disadvantage/Risk
Riva-Rocci	Palpate pulse, inflate cuff, slowly deflate until pulse returns	Can be used without a stethoscope, by palpation of pulse or Doppler flow detection	Only gives a systolic pressure, can work with nonpulsatile flow
Korotkoff	Auscultate over antecubital fossa, inflate cuff, slowly deflate, noting first auscultation sounds and last sounds	Gives diastolic as well as systolic pressure	Needs stethoscope, quiet environment
Noninvasive blood pressure (NIBP)	Choose correct cuff size, initiate cuff inflation	Can be automated, for routine monitoring, measures mean pressure, interpolates systolic and diastolic pressure	Does not work with severe hypotension, motion artifact, or patient with left ventricular assist device
Invasive	Connect intra-arterial catheter to transducer	Wide range of pressure, measures a mean pressure, systolic and diastolic pressure Useful when there is hemodynamic instability, vasopressor administration Can serve as access route for blood draws	Invasive, potential for amplification artifact, dampening, hemorrhage, hematoma, infection, injury to artery or distal areas
	Radial	Most commonly used as generally accessible; hand typically has dual blood supply	Can produce artificially low values with severe systemic vasoconstriction
	Brachial	Sometimes available when radial site is not	No redundant blood supply, uncomfortable, cannot flex arm
	Femoral	Large vessel, can give accurate values with profound vasoconstriction	Prone to infection, affected by prone positioning
	Dorsalis pedis	May be an accessible site when others are not	Some amplification of waveform

Table 20.4 Measures of Volume Responsiveness to Intravenous Fluid Bolus[a]

	Fluid Responsive	Not Fluid Responsive
SPV	>10 mm Hg	<5 mm Hg
PPV	>15%	<7%
SVV	<15%	<5%

The three measures of volume responsiveness are systolic pressure variation (SPV), pulse pressure variation (PPV), and stroke volume variation (SVV). As the table indicates, there is a "gray zone" between levels of responsiveness where it is unclear if the patient would benefit from the treatment.[13]

[a]Defined as improved stroke volume or end-diastolic area as assessed by transesophageal echocardiography.

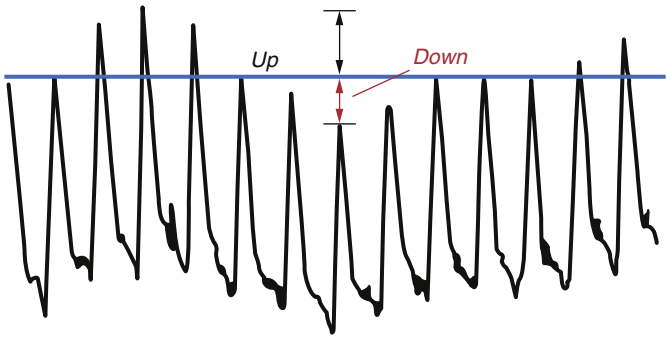


Fig. 20.14 Systolic pressure variation. A positive-pressure breath can result in a transient decrease in the systolic blood pressure. The mechanism is predominantly related to the positive intrathoracic pressure causing a decrease in venous return and subsequent decrease in right-sided heart stroke volume, and ultimately left-sided heart stroke volume, which causes the systolic pressure to decrease. The magnitude of the drop in systolic blood pressure can predict the responsiveness of a patient to an intravascular fluid challenge

The SPV is an indirect assessment of the venous capacitance, which when abnormal, indicates a potential of the blood pressure to improve with fluid administration.

One of the major limitations of SPV is that the patient must have a regular heartbeat, that is, it cannot be used in atrial fibrillation, which itself causes irregular variations in systolic blood pressure. The values may also be affected by increased lung or chest wall compliance, prone positioning, and high PEEP and will not be usable when there is an open thoracic cavity. The newer generation of physiologic monitors automatically calculates SPV.

Pulse Pressure Variation

Another method of deriving similar information is measuring the relative changes in pulse pressure during the respiratory cycle, known as *pulse pressure variation (PPV)*. In this situation, the pulse pressure between breaths minus the pulse pressure during the positive-pressure breath is subtracted and then divided by the mean pulse pressure times 100%.

Eq. 5

$$\text{PPV}\,\% = \frac{\text{PP}_{\text{max}} - \text{PP}_{\text{min}} \times 100}{\frac{\text{PP}_{\text{max}} + \text{PP}_{\text{min}}}{2}}$$

PPV can be used to predict response to an intravenous fluid bolus, similar to the SPV.[13]

Stroke Volume Variation

Stroke volume variation is another technique using the arterial waveform to assess volume responsiveness. In this situation, a pulse contour algorithm is employed to estimate the stroke volume from the arterial pulse wave.[14] These estimates of arterial pulse volume are compared during the respiratory cycle in an analogous fashion to SPV and PPV described previously. The percent reduction in estimated stroke volume associated with positive-pressure ventilation is used to assess whether the patient would benefit from additional fluid. All of these measures of volume responsiveness require positive-pressure ventilation in a closed chest with a regular cardiac rhythm without excessively high levels of PEEP (see Table 20.4).

Central Venous Monitoring

As described in the preceding theoretical analysis, blood pressure alone is not a sufficient variable to evaluate perfusion (also see Chapter 25). Knowledge of CVP, PAP, and cardiac output (CO) may be helpful in guiding patient therapy. Additionally, central venous access may

Table 20.5 Central Venous Access and Pressure Measurement

Route	Indications	Risk	Benefit
Any central venous pressure (CVP) catheter	Unable to obtain peripheral access, route for potent vasoactive medications	Bleeding, infection	Stable IV access
RIJ (right internal jugular)	Unable to obtain peripheral access, route for potent vasoactive medications	Carotid artery injury	Straight path to heart for pulmonary artery (PA) catheter
LIJ (left internal jugular)	Unable to use RIJ	Carotid artery injury, thoracic duct injury, short distance to innominate vein	Can use if RIJ not available
Subclavian	Unable to use RIJ or LIJ	Pneumothorax risk, injury to brachial plexus, subclavian artery/vein	More comfortable for patient after surgery, can insert with cervical collar in place; lower infection risk
Femoral	Disease of head and neck precluding use of neck access	Increased infection risk	Can apply pressure if bleeding
PA catheter	Unstable patient	In addition to CVP risks: PA rupture, dysrhythmias	Can obtain cardiac output information

Practice guidelines for pulmonary artery catheterization have been published: American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Practice guidelines for pulmonary artery catheterization: an updated report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. *Anesthesiology*. 2003;99(4):988-1014. These guidelines highlight three areas: (1) patient disease (Does the patient have serious cardiac disease for which knowledge of the cardiac output and filling pressures may alter treatment?), (2) surgery (Is the surgery a major procedure in which there will be fluid shifts or changes that will be reflected in the monitor?), and (3) setting (Do the practitioners have the expertise to perform the procedure with minimum potential risk and maximum potential benefit?).

be necessary for administration of certain drugs and may act as secure access for administration of large volumes of resuscitation fluids. Table 20.5 lists a comparison of different sites for central line placement.

Central Venous Pressure

Information obtained from a CVP line includes the CVP pressure and waveforms (Fig. 20.15). The CVP waveform has several elements, each reflecting the underlying cardiovascular physiology. The *a wave* reflects atrial contraction against the closed tricuspid valve, the *c wave* reflects tricuspid bulging as the ventricle contracts, the *x descent* corresponds to atrial relaxation, the *v wave* occurs during atrial filling, and the *y descent* reflects atrial emptying. Despite the physiologic basis for the waveform, CVP is a minimally helpful guide for intravascular fluid therapy because of the complexity of the relationships between intravascular volume, venous capacitance, venous return, cardiac performance, and arterial blood pressure.[15]

Although CVP values are not considered to be very predictive of intravascular volume status, they may be useful in the extremes. That is, a CVP under 2 mm Hg may suggest a beneficial cardiovascular effect from intravenous fluid administration, whereas a value of more than 15 mm Hg suggests that more fluid may not be needed. This approach to assessing the utility of a physiologic variable has been described as a "gray zone analysis." That is, in the extremes the variable provides useful information, but in the range between those extremes, or the normal range, is an area where the utility of the variable is less valuable in assessing clinical status. However, even at extreme values, the decision whether to administer

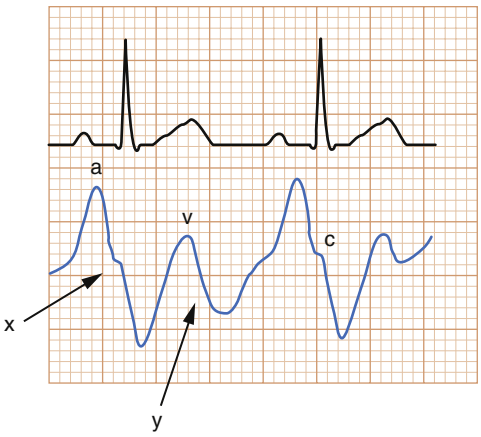


Fig. 20.15 Central venous pressure waveform. The mean central venous pressure (CVP) value can be used to assess right-sided heart filling pressure. The waveform can also be instructive.

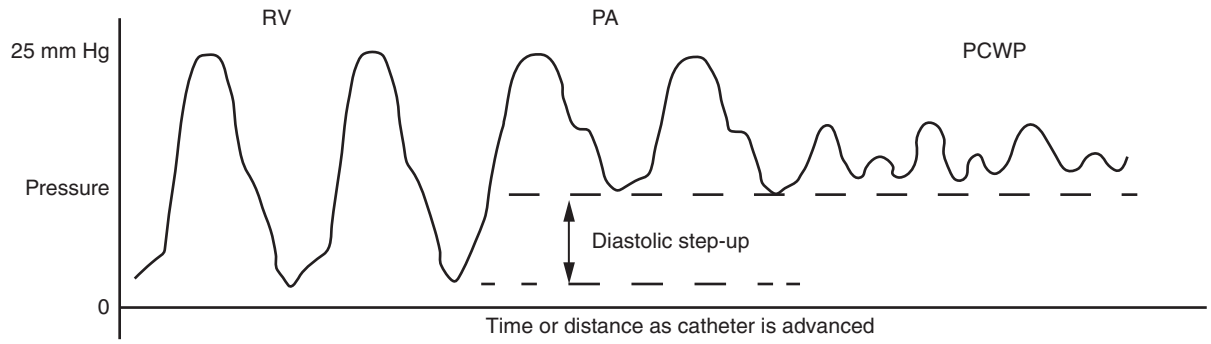


Fig. 20.16 A trace of pressure versus distance as a pulmonary artery catheter is advanced from the right atrium through the right ventricle (RV) into the pulmonary artery and ultimately resting in a wedge position in the pulmonary artery. Note as the catheter is advanced from the right ventricle into the pulmonary artery the diastolic pressure is cut off and rises to the PA diastolic, which is only slightly higher than the pulmonary artery wedge pressure. *PA,* Pulmonary artery; *PCWP,* pulmonary capillary wedge pressure.

Table 20.6 Differential Diagnosis of Severe Hypotension

Diagnosis	CVP	PAP	PCWP	CO	Airway Pressure
Pneumothorax	↑	↑	↑	↓	↑
Tamponade	↑	↑	↑	↓	↔
Pulmonary embolism	↑	↑	↓	↓	↔
Hypovolemic shock	↓	↓	↓	↓	↔
Cardiogenic shock	↑	↑	↑	↓	↔
Septic shock	↓	↓	↓	↑	↔

The changes in invasive hemodynamic and airway pressures are associated with specific causes of hypotension.
CO, Cardiac output; *CVP,* central venous pressure; *PAP,* pulmonary artery pressure; *PCWP,* pulmonary capillary wedge pressure.

fluid or remove fluid (with diuretics) should be based on the individual patient's clinical circumstances.

Central Venous Catheter Placement

Before placing a central venous catheter, informed consent must be obtained because of such risks as bleeding, infection, and potential damage to surrounding structures (nerves, lymph, vessels, lung, pneumothorax, and more), and to apprise the patient of the risks and benefits. The ASA has an excellent practice guideline for central venous access.[16]

Pulmonary Artery Pressure and Cardiac Output

A pulmonary artery catheter (PAC) is an access catheter advanced from the right atrium to the right ventricle into a wedge position in the pulmonary artery by following the pressure waveforms as noted in Fig. 20.16. Data from a PAC can be used to diagnose a variety of conditions owing to its ability to measure right- and left-sided heart filling pressures as well as cardiac output. Under normal conditions, because the pulmonary vascular system is a very low resistance system, the PA diastolic pressure and the PA wedge pressure are very similar. Table 20.6 describes a variety of acute causes of severe hypotension and the results of specific monitored parameters.

Methods of Measuring Cardiac Output

Thermodilution is a common method of measuring cardiac output. A measured amount of "cold" fluid is injected into the central circulation via the proximal port of a PAC, and a thermister measures the temperature at the distal port. These temperature readings are recorded as a curve over time. The area under the curve is proportional to the cardiac output. Typically, several measurements are averaged at different points of the respiratory cycle. Special PACs can measure and display cardiac output on a continuous basis. In spite of all the hemodynamic data provided by the PAC, there have been no studies documenting improved outcomes in surgical patients.[17] The risks are substantial, including line sepsis, thrombosis, and pulmonary artery rupture. If a PAC is utilized for the management of severe hemodynamic compromise, it should be removed as soon as

possible. Although the use of PACs in surgery and critical care has dramatically decreased, there has been an increase in echocardiography for immediate diagnosis of cardiac disease.

Transesophageal Echocardiogram Monitoring

One way to quickly determine the cardiac status is to perform a TEE (also see Chapter 25). An ultrasound probe is inserted into the esophagus and various views of the heart are obtained in real time. Information on cardiac structure (heart valves, chamber size), contractile activity (ejection fraction), systolic and diastolic dysfunction, and pericardial disease (effusion, tamponade) can all be diagnosed with a TEE. It has therefore become the gold standard in cardiac evaluation. Limitations of TEE include the need for expertise on the part of the provider, access to the head of the patient, and the risk of esophageal injury.

CENTRAL NERVOUS SYSTEM

Processed Electroencephalograph Monitoring

Although the electrical activity of the brain can be monitored with the multichannel electroencephalograph (EEG), processed EEG monitors focused on the frontal area have been developed for the sake of convenience. These devices are derived through an empirical comparison of the awake and anesthetized EEG and provide output as an index generated through a multistep process. EEG features are extracted, artifact is minimized, and an algorithm converts the EEG features to a numerical index, often ranging from 100 (awake) to 0 (isoelectric EEG). These monitors are intended to assess anesthetic depth and reduce the incidence of awareness with postoperative recall (also see Chapter 47) (avoiding subtherapeutic dosing) and minimize unnecessary anesthetic administration (avoiding supratherapeutic dosing). The incidence of recall is between 1:500 to 1:1000.[18,19] The most definitive work on these devices—in particular, the bispectral index (BIS) monitor—has demonstrated a reduction in the incidence of postoperative recall of intraoperative events, compared to no monitoring of anesthetic depth, but not to a greater degree than alerts based on the expired anesthetic concentrations[18,19] (also see Chapter 47).

Minimum Alveolar Concentration Alert Monitoring

The minimum alveolar concentration (MAC) was developed as a method to assess and compare the relative potency of inhaled anesthetics. MAC is the end-expired concentration of an anesthetic at equilibrium at which 50% of the subjects would move in response to a noxious stimulus. In clinical anesthesia it has been used to assess depth of anesthesia and randomized controlled clinical trials suggest keeping MAC above 0.5 to 0.7 to prevent recall of intraoperative events.

The results in large randomized trials suggest that in cases in which inhalational anesthetics are employed, alerts related to the expired anesthetic age-adjusted MAC (0.5 to 0.7) are equivalent to the BIS monitor in preventing awareness with postoperative recall.[20] These studies also noted that patients monitored by either method had lower incidence of recall than patients without any anesthetic depth monitoring. When total intravenous anesthesia (TIVA) is used without any inhaled anesthetic, no calculated MAC alert can be produced and use of a neurologic monitor is recommended, especially if a nondepolarizing muscle relaxant is employed[20] (also see Chapter 11).

A 2012 study demonstrated an association between the simultaneous occurrence of a low BIS (<45), a low MAC (<0.8), and a low MAP (<75)—a so-called "triple-low" state—and increased 30-day mortality rate. One of two subsequent studies of the triple-low state has demonstrated the same weak independent association, though it is unclear whether changing one of the three parameters (BIS, MAC, MAP) would alter mortality rate.[21,22]

Intracranial Pressure Monitoring

Because the brain is enclosed in a fixed cranial vault, the perfusion pressure of the brain (cerebral perfusion pressure, CPP) is defined as the MAP minus the ICP. For that reason, under conditions when cerebral edema or increased cerebrospinal fluid may dramatically increase the ICP, continuous monitoring of ICP may be useful to ensure brain perfusion. Two methods are commonly employed to monitor ICP (also see Chapter 30). The first is a ventriculostomy catheter, inserted percutaneously into a lateral ventricle of the brain. ICP is transduced with a traditional disposable transducer zeroed at the tragus of the ear. An advantage of ICP monitoring with a ventriculostomy is that cerebrospinal fluid may be removed to reduce intracranial volume and thus ICP. A second technique of measuring ICP employs a device with a fiberoptic pressure transducer on the tip of a catheter, which can be inserted into the brain parenchyma or the subdural space. These devices do not require zeroing.

Cerebral Oximetry

The oxygenation of a portion of the brain (i.e., portion of the cerebral cortex) can be monitored with a reflectance oximeter. This device uses near infrared light in a fashion similar to a pulse oximeter. However, instead of using the pulsatile absorbance of light transmitted through tissue to estimate arterial saturation, it uses the reflected infrared light through the scalp and skull from a portion of the cerebral cortex beneath it. This parameter is called

regional oxygen saturation (rSo_2). The light is reflected predominantly from the hemoglobin in the red blood cells within the vasculature of the cerebral cortex. The device presents a number between 1% and 100% saturation, again similar to a pulse oximeter. The algorithms for determining this saturation are proprietary to the manufacturers. These devices have been used in cardiac and vascular surgical procedures when there is a concern of decreased cerebral oxygenation because of poor perfusion of the brain. One study of shoulder surgery in the beach chair position demonstrated that rSo_2 may be helpful in determining when changes in Fio_2 or ventilation may be needed.[23] rSo_2 values are usually about 70% (like mixed venous blood). Those values less than 50% or with a 20% decrease from the baseline values may be associated with decreased cerebral oxygenation.

PERIPHERAL NERVOUS SYSTEM

Neuromuscular Monitoring

The use of neuromuscular blocking drugs is an important part of many anesthetics (also see Chapter 11). Monitoring the effects of drugs that block neuromuscular transmission at the synaptic junction is vital to prevent patient motion at inopportune times during the surgery and to prevent partial paralysis with risks of awareness, aspiration of gastric contents, and hypoventilation at the end of surgery. In the past few years, residual neuromuscular blockade postoperatively has been a major concern. Whether the neuromuscular blockade has been reversed by neostigmine or sugammadex can only be confidently determined by the results of monitoring with a peripheral nerve stimulator.

Basic Physiology and Pharmacology

Normal neuromuscular transmission starts with a motor nerve impulse arriving at the end plate. Quanta of acetylcholine are released in response to the depolarization, diffuse across the neuromuscular synaptic cleft, bind to the postsynaptic nicotinic cholinergic receptor, and trigger depolarization of the nerve, opening calcium channels with subsequent activation of actin-myosin chains and muscle contraction. Most of the acetylcholine is hydrolyzed enzymatically by acetylcholinesterase. The resulting choline is recycled into the nerve terminal. Nondepolarizing neuromuscular blocking drugs act by competitively inhibiting the binding of acetylcholine with the receptor. Although the blockade is competitive, it can be overcome with additional quanta of acetylcholine. The nondepolarizing blockers demonstrate fade with repeated stimulation, thought to be due to a presynaptic $\alpha 3\beta 2$ acetylcholine receptor exhaustion.[24-26] Succinylcholine acts differently by binding to the receptor and activating it, resulting in prolonged depolarization and blockade of transmission.

Neuromuscular Blockade Monitor

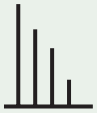
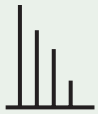
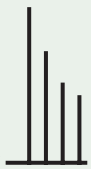
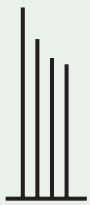
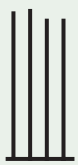
The most common method to follow the effects of a nondepolarizing neuromuscular blocking drug is to use a "twitch monitor" and follow a train-of-four (TOF) count. The TOF monitor generates four supramaximal stimuli at 0.5-second intervals (2 Hz). As the degree of blockade deepens, the twitches first fade, then are progressively lost (Table 20.7). Assessment of very deep or profound levels of blockade can be done by using a posttetanic count. A tetanic stimulus for 5 seconds primes the nerve terminal with more acetylcholine allowing a posttetanic count to be done. Even low levels of blockade may be associated with adverse results. To test for lower levels of blockade a double-burst stimulation can be performed. Despite being quantitative there is a great degree of subjectivity in the monitor. Newer monitors are more quantitative, allowing the measurement of TOF ratio and detection of lighter levels of blockade, which may still be clinically significant. Succinylcholine induces a noncompetitive blockade, which can be followed by a single twitch.[25,26]

Evoked Potential

Evoked potential (EP) monitoring is indicated for procedures in which there may be neurologic injury because of either mechanical trauma or ischemia such as spinal surgery, thoracic abdominal aneurysm, or surgery of the face or neck. Monitoring of EPs requires constant attention of trained personnel. It is important for anesthesia providers to understand their use and limitations, for they will affect the choice of anesthetic. The most commonly employed EPs are somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs). Both involve a stimulating electrode and a sensing electrode continuously assessing the function of the sensory or motor nerve track.

SSEPs involve delivering a small current to a sensory nerve and measuring the response on the sensory cortex with a scalp electrode. The response is viewed as voltage versus time plot. To reduce background noise, multiple responses are averaged to produce an SSEP waveform. Nerve injury or ischemia is associated with a decrease in amplitude and an increase in latency of the peaks in the waveform compared to the baseline waveform. Inhaled anesthetics (halogenated ethers and nitrous oxide) also produce marked decreases in amplitude and increases in latency at larger doses. Patients with preexisting compromise of brain or spinal cord integrity are especially susceptible to the effects of inhaled anesthetics. Propofol is considered the best drug choice for maintenance of unconsciousness during EP monitoring. Dexmedetomidine has also been used as an anesthetic adjunct with minimal depression of neurophysiologic signals in adults. A limitation of SSEPs for spine surgery is that they monitor sensory tracts but not motor tracts (the ventral spinal cord). Therefore, there can be false negatives; that is, procedures can have intact SSEPs when there is in fact damage to the motor tracts.

Table 20.7 Assessment of Neuromuscular Blockade by Monitor

	Stimulus					
% Blockade	**PTC**	**TOF**	**DBS**	**TOF Ratio**	**Tetany**	**Clinical Response**
>100%	0	0/4	0	N/A	0	Flaccid
>100%	0<PTC<10	0	0	N/A		
90%	PTC > 10	1 of 4 	N/A	N/A		
80%		2 of 4 		N/A		May breathe, maintain $ETco_2$, but lack airway patency
75%		3 of 4 		N/A		
0-75%	N/A	4 of 4 	Significant fade 	0.2 		
	N/A		Fade detectable 	0.4 		Risk of aspiration still present
	N/A		Some fade 	0.7 		Head lift > 5 sec
	N/A			0.9 	Fade at 50 Hz	
60%	N/A				No fade at 50 Hz, fade at 100 Hz	
30%	N/A				Fade at 200 Hz	
0%	N/A			1.0		

The table provides the responses of a neuromuscular blockade monitor as a function of block versus the stimulus. Posttetanic count (PTC), train-of-four (TOF), double-burst suppression (DBS), train-of-four ratio (TOF ratio), tetany, and the clinical response are given as a function of the percentage of neuromuscular blockade.

MEPs involve stimulating the motor cortex and detecting a response in muscle. MEPs therefore have the advantage of ensuring an intact ventral spinal cord and are more sensitive to both neural injury and anesthetic drugs. The disadvantage is that they require an intact neuromuscular junction, that is, the avoidance of neuromuscular nondepolarizing muscle relaxants during anesthetic care. MEPs are more profoundly affected by volatile anesthetics and nitrous oxide than SSEPs; therefore, intravenous anesthetics are commonly used. It is common to combine SSEP and MEP monitoring as well as electromyography (EMG) in a patient undergoing major spinal surgery. There should be close communication among the anesthesia provider, monitoring technician, and surgeon at all times during surgery.

TEMPERATURE

Anesthetics interfere with normal temperature autoregulation and can cause abrupt increases in temperature associated with malignant hyperthermia (MH).[27] Therefore, the patient's temperature is monitored to manage intraoperative hypothermia (inadvertent or desired), prevent hyperthermia, and to confirm and detect MH (although rising temperature is often a later finding in MH). Historically, core body temperature was measured orally or rectally with liquid thermometers. Although accurate, these were slow to respond, fragile, and cumbersome in an operating room environment. Infrared scanners directed at the tympanic membrane are used extensively pre- and postoperatively. The infrared response time is faster, but readings are subject to errors caused by cerumen and other obstructions to the optical path. Intraoperative, low mass, small thermisters are often used. These work by converting changes in temperature to a change in electrical resistance, which is converted and displayed. The acceptable accuracy is +/– 0.5° C.

True core temperature is measured by probes in the pulmonary artery, distal esophagus, tympanic membrane, or nasopharyngeal zones. Other sites that can approximate core temperature include oral, axillary, and bladder. Bladder temperature is highly affected by urine output, approaching true core temperature at high urine flows. Rectal and skin temperatures are highly variable relative to true core temperature (Table 20.8).

For short-duration anesthetics (under 30 minutes), the primary mechanism for the drop in core temperature is redistribution of heat from the core to the periphery.

MAGNETIC RESONANCE IMAGING AND ADVERSE CONDITIONS

Magnetic resonance imaging (MRI) uses radiofrequency pulses to change the rotation of nuclei in atoms aligned in a very strong magnetic field (also see Chapter 38). As the pulse is removed, the energy is released and can be imaged in multiple dimensions. Because different body tissues have different relaxation rates, better tissue differentiation (e.g., white vs. gray matter in the central nervous system) can be obtained.

The magnetic field strength decreases with distance from the coil. The actual rate of decrease is nonlinear and depends on multiple factors, including the shape and orientation of the magnet. A safe distance in one direction may not be a safe distance in another direction. MRI suites have demarcation lines indicating the field strength at various distances. Better designed suites have a series of rooms, so that direct access to the high magnetic environment is not possible without being screened. Equipment that functions at a certain distance may not work closer to the magnet (and may become a projectile).

All monitors are affected by the MRI environment.[28] Noise levels in an MRI suite are up to 95 dB, making auscultation

Table 20.8 Temperature Monitoring Sites

Site	Advantages	Disadvantages
Pulmonary arterial	Gives true blood temperature	Extremely invasive
Tympanic	Gives "brain" temperature	May cause injury to tympanic membrane
Esophageal	Tends to reflect core temperature	Subject to cooling by respiratory gases
Nasopharyngeal	Gives "brain" temperature	Nosebleeds, ambient cooling/heating
Oral	Comfortable in awake patient	Not easily done in sleeping patient
Bladder	Easily done if a Foley catheter is in place	Depends on urine output to reflect core temperature
Skin	Easy, noninvasive	Doesn't reflect core temperature, ambient temperature
Rectal		May not reflect true core, invasive, nonsterile area

All temperature readings are dependent upon blood flow to the area. Alterations in blood flow may result in erroneous temperature readings. Surgical site can compromise monitoring; for example, an open thorax can change esophageal temperature readings.

of any sounds difficult (breath sounds, heart tones, Korotkoff sounds). The magnetic field will interfere with ECG monitoring as well. The rapidly changing magnetic field orientation can induce a current in any loop, causing heating and burns (also applies to pulse oximetry equipment). Extended ventilation and sampling tubing helps keep sensitive equipment away from the magnet. The converse is true as well, in that monitors may affect the quality of the MRI.

MONITORS AND ALARMS

False positives are the bane of alarm settings. Too sensitive a setting and alarm fatigue ensues. A setting not sensitive enough can lead to critical states occurring without notification. For this reason, the pulse tone of the pulse oximeter (decreasing with decreasing saturation) is the only continuous audible "alarm" used in the operating room. The ventilator disconnect (a low circuit pressure) is the most commonly employed true audible alarm active in the operating room. In an attempt to try to address the alarm overdose/alarm fatigue issues associated with multiple monitoring systems, a newer generation of integrated alerting systems have been developed, such as AlertWatch.[29]

QUESTIONS OF THE DAY

1. How is the accuracy of pulse oximetry affected by abnormal hemoglobins such as carboxyhemoglobin or methemoglobin?
2. During mechanical ventilation, how should an increase in peak airway pressure be investigated to determine the clinical cause?
3. What are the advantages and disadvantages of noninvasive arterial blood pressure measurement compared to invasive arterial blood pressure measurement with an arterial line?
4. What is the rationale for using systolic blood pressure variation, pulse pressure variation, or stroke volume variation as a measure of intravascular volume responsiveness? In what clinical circumstances would the systolic pressure variation be inaccurate as a measure of intravascular volume responsiveness?
5. A patient requires a central venous line because of poor peripheral intravenous access. What factors should be used to determine the site of cannulation?
6. Which monitoring sites most closely reflect core body temperature?

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