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KEY POINTS

- Perioperative applications of transesophageal echocardiography (TEE) include monitoring, diagnosis, and procedural guidance. The role of TEE in cardiac surgery, noncardiac surgery, interventional procedures, and critical care continues to evolve.
- In noncardiac surgery, intraoperative TEE serves as a routine monitor or as a rescue tool in life-threatening emergencies.
- TEE aids in decision making during cardiac surgery, and three-dimensional (3-D) TEE adds incremental value in intraoperative assessments.
- TEE is integral to structural heart interventions and is a key component of multimodality imaging.
- Echocardiography plays a vital role in the diagnosis and management of shock.
- Applications of transthoracic echocardiography (TTE) in the perioperative setting are expanding.
- Focused cardiac ultrasound is performed and interpreted at the point of care, and addresses specific questions relevant to the clinical context.
- Critical care societies have proposed distinct competencies and guidelines for critical care echocardiography beyond those included in perioperative TEE guidelines. The skill set of the intensivist performing echocardiography overlaps with that of the intraoperative echocardiographer and includes other unique components.
- Basic knowledge of echocardiography is an expectation of anesthesiology training.
- Simulation is an effective training tool when integrated within a multimodal, curriculum-based approach to echocardiography education.

Introduction

Echocardiography is an invaluable tool used throughout the perioperative period. Image acquisition and interpretation occur at the point of care, enabling real-time integration of the findings in the context of the patient's clinical condition. Transesophageal echocardiography (TEE) has broad functionality in the perioperative environment. TEE serves as an intraoperative monitor, provides detailed structural and functional information, facilitates diagnosis of pathology, and guides percutaneous interventions. Applications of TEE have expanded within cardiac surgery, and into the realms of noncardiac surgery, catheter-based procedures, and critical care. As the use of clinical ultrasound grows in modern anesthetic practice, perioperative clinicians are also beginning to embrace transthoracic echocardiography (TTE). Critical care echocardiography shares many similarities with intraoperative echocardiography, but is simultaneously evolving under the larger umbrella of critical care ultrasonography. This chapter provides a historical synopsis and summary of imaging techniques and echocardiographic views and discusses applications of TEE and TTE in perioperative care.

History of Perioperative Echocardiography

INTRAOPERATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Decades of evolution and advancement have led to the current state of perioperative TEE (Fig. 37.1).¹ Clinical

echocardiography first emerged in the 1950s with the use of motion mode (M-mode) ultrasound to record signals from cardiac structures.² Nearly two decades later, the first intraoperative echocardiography was performed with epicardial imaging.³ The first practical clinical application of TEE was reported in 1976.⁴ Soon after, a group reported the first use of intraoperative TEE⁵ and described its utility in monitoring left ventricular (LV) function during cardiac surgery.⁶ In the 1980s, the mounting of two-dimensional (2-D) transducers onto modified flexible gastroscopes^{7,8} created more versatile probes, and pioneering anesthesiologists and cardiologists began to demonstrate the usefulness of TEE as an intraoperative monitor.⁹⁻¹¹ Further advancements such as transducers with combined 2-D and Doppler capabilities,^{12,13} biplane imaging,¹⁴ multiplane imaging,¹⁵ and pediatric probes¹⁶ unlocked the diagnostic potential of intraoperative TEE. Real-time three-dimensional imaging (RT-3D) entered the scene in the mid-2000s,¹⁷ and since then its intraoperative use has grown exponentially. Advances in probe technology and improved computing power continue to further the practicality of three-dimensional (3-D) image acquisition.

CRITICAL CARE ECHOCARDIOGRAPHY

Prior to the introduction of TEE in 1976, significant advances in 2-D transthoracic imaging occurred, leading to better visualization of cardiac structures.¹⁸⁻²² Production of commercially available instruments soon followed. In the intensive care units (ICUs), some clinicians incorporated echocardiography in the evaluation of cardiac function in acute respiratory distress syndrome and sepsis.^{23,24}

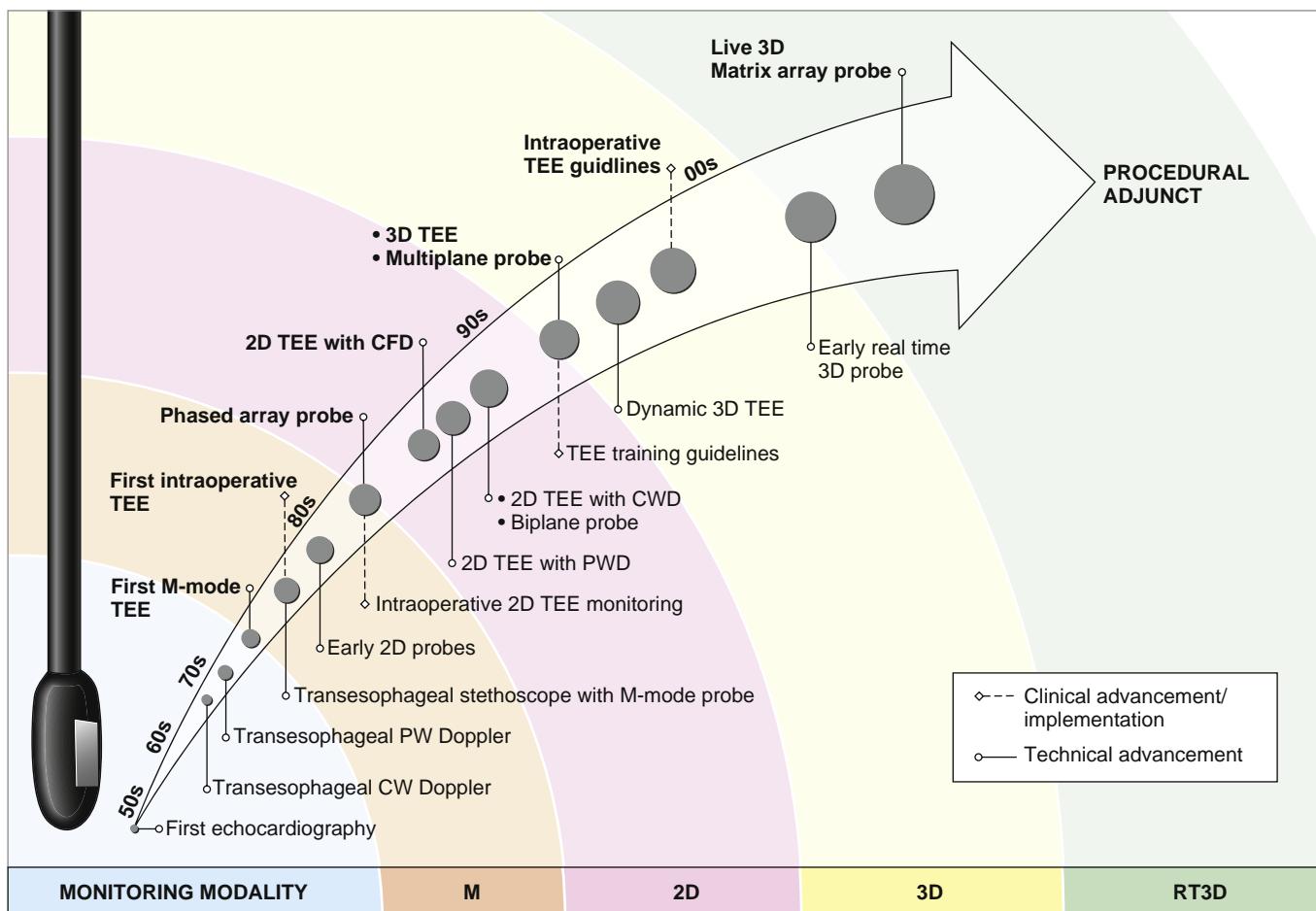


Fig. 37.1 Evolution of transesophageal echocardiography (TEE) over the years from a monitoring modality to procedural adjunct. 2D, two-dimensional; 3D, three-dimensional; CFD, color flow Doppler; CWD, continuous wave Doppler; RT3D, real-time 3D. (Modified with permission from, Mahmood F, Sherman SK. Perioperative transoesophageal echocardiography: current status and future directions. *Heart*. 2016;102(15):1159–1167.)

TTE was not widely disseminated in ICUs, however, as the intensivist's tool of choice at the time was the pulmonary artery catheter.^{25–27} During the late 1990s to 2000s, the use of pulmonary artery catheters declined,^{28–32} and TTE simultaneously gained more traction in the ICUs. With the miniaturization of equipment and emphasis on point-of-care ultrasound, use of echocardiography as a hemodynamic monitor and diagnostic tool in the ICU continues to increase.

Principles of Ultrasound

While a thorough discussion of ultrasound physics is beyond the scope of this chapter, a basic understanding of fundamental concepts is requisite for any echocardiographer. Sound is a mechanical, longitudinal wave of vibrations propagated through a medium. Several parameters describe sound waves including frequency, wavelength, amplitude, and propagation speed. Ultrasound waves are sound waves with frequencies above the audible human range (20–20,000 hertz [Hz]). Echocardiography typically uses frequencies between 2 and 12 megahertz (MHz). Ultrasound transducers use piezoelectric elements to convert

ultrasound energy into electrical energy and vice versa. Transducers function both as transmitters and receivers of ultrasound signals. 2-D echocardiography uses a phased-array transducer, which has a row of electrically interconnected piezoelectric elements.

As ultrasound energy propagates through tissue, interactions between the wave and tissue result in absorption, divergence, reflection, and scattering of the energy. Reflection of ultrasound energy at tissue interfaces forms the basis of image generation. These sound-tissue interactions reduce the intensity of the ultrasound signal, and this attenuation limits the depth of imaging. Transmitted energy at tissue interfaces often undergoes refraction and changes directions, contributing to imaging artifacts.

The accurate display of an image depends on imaging resolution, components of which include spatial resolution, temporal resolution, and contrast resolution (Table 37.1). Spatial resolution can be described according to the three beam dimensions: axial, lateral, and elevational. Image formation involves a tradeoff between spatial resolution and depth of penetration. Higher frequency (shorter wavelength) transducers provide superior axial resolution, but limited penetration depth due to attenuation. Lower frequencies (longer wavelengths) penetrate more deeply at the expense of axial resolution. Higher

TABLE 37.1 Components of Imaging Resolution

Description		Practical points
Spatial resolution	The ability to discriminate between two closely-spaced objects The minimum distance necessary between two structures so that they are displayed as separate objects	Determines image details Factors that improve spatial resolution often come at the expense of decreased temporal resolution and vice versa
Axial	Differentiates between objects along the length of the ultrasound beam	Higher frequency transducers have shorter wavelengths and better axial resolution Axial resolution is superior to lateral resolution
Lateral	Discriminates side-to-side (or horizontal) relative to the direction of the beam	Determined by the beam width Best at the focal point of the beam
Elevational	Discriminates between objects vertical to the plane of the ultrasound beam	Determined by the beam height Best at the focal point of the beam
Temporal resolution	The ability to accurately display a structure's motion with respect to time	The higher the frame rate, the better the temporal resolution Factors that decrease the time to scan the sector (e.g., decreasing the imaging depth or creating a narrower imaging sector) improve temporal resolution
Contrast resolution	The ability to resolve subtle differences in echogenicity, which are displayed as different shades of gray	Improved by harmonic imaging, use of contrast agents, B-color maps, and post-processing controls

frequencies are better for imaging superficial structures; lower frequencies are better for deeper structures.

Formation of ultrasound images relies on several assumptions: (1) ultrasound energy propagates in a straight line; (2) all returning echoes originate from the central beam, which is extremely thin; (3) echoes return to the transducer after one reflection; (4) attenuation is constant; and (5) the speed of sound is constant, therefore the depth of a reflector is proportional to the round-trip transit time.³³ Violations of these assumptions result in imaging artifacts. Spectral and color flow Doppler (CFD) and 3-D images are also susceptible to imaging artifacts.

Ultrasound Modalities

Key ultrasound modalities used in echocardiography include motion mode (M-mode), 2-D imaging, Doppler (spectral and color flow), and Doppler tissue imaging (DTI). Table 37.2 highlights important aspects of these techniques. Additional techniques may be applied in specific contexts and are introduced in the next section.

STRAIN AND STRAIN-RATE IMAGING

Strain and strain-rate imaging techniques (also called cardiac mechanics or myocardial deformation) are used to quantify global and regional ventricular function. Strain is the relative change in shape or size of an object as a result of an applied force. In the context of the myocardium, it is the fractional change in the length of a segment relative to its baseline and expressed as a percentage. Positive strain indicates lengthening or thickening and negative strain represents shortening or thinning. Three axes are relevant to myocardial deformation: longitudinal, circumferential, and radial. Normal deformation patterns during systole are longitudinal shortening (negative strain), circumferential shortening (negative strain), and radial thickening (positive strain).

Methods for strain analysis include DTI and the more commonly used speckle tracking echocardiography (STE). DTI measures velocities at two points in the myocardium and derives the strain and strain rate. STE, in contrast, tracks unique acoustic speckles in the myocardium over a series of frames and derives strain and strain rate from the change in distance. Duncan and colleagues have written an excellent review on perioperative strain and strain-rate imaging.³⁴

HARMONIC IMAGING

Harmonic imaging is a processing technique used to improve the quality of 2-D images.³⁵ A sound wave propagates non-linearly through tissue, distorting the wave's shape. This produces harmonic frequencies, which are integer multiples of the fundamental frequency originally transmitted by transducer. Signals return to the transducer at both the fundamental frequency and harmonic frequencies. Harmonic imaging typically creates images from the second harmonic frequency and filters the fundamental frequency signals. Harmonic imaging increases contrast resolution, improves signal-to-noise ratio, and decreases some artifacts. Disadvantages include slightly decreased spatial resolution and thickened appearance of some cardiac structures.^{36,37}

CONTRAST ECHOCARDIOGRAPHY

Contrast echocardiography can be used to improve diagnostic evaluation and enhance suboptimal images.^{38,39} Two methods of contrast echocardiography include injection of agitated saline or injection of commercially available contrast agents. Agitated saline can be used to identify the presence of intracardiac right-to-left shunt, because microbubbles created by agitation do not cross the pulmonary circulation.⁴⁰ Commercially available contrast agents use encapsulated microbubbles of high-density gas and are able to traverse the pulmonary circulation, allowing for opacification of the left side of the heart. Perioperative applications

TABLE 37.2 Ultrasound Modalities

Modality	Description/Features
Brightness Mode (B-mode)	<ul style="list-style-type: none"> Depicts the strength of returning sound signals using corresponding degrees of brightness M-mode and 2D imaging are modifications of original B-mode imaging
Motion Mode (M-mode)	<ul style="list-style-type: none"> Displays a one-dimensional image of the motion of cardiac structures relative to time Very high temporal resolution Provides limited spatial information Greatest utility in providing information about rapidly moving structures (e.g., valves, walls) in relation to the timing of the cardiac cycle
Two-Dimensional (2D)	<ul style="list-style-type: none"> The mainstay of the echocardiographic examination Scanning of repeated pulses generates an image representative of the cardiac structures with real-time motion Images are produced in a sector format Temporal resolution is less than that of M-mode
Doppler	<ul style="list-style-type: none"> Ultrasound energy scattered from moving red blood cells shifts to a higher frequency when moving toward the transducer and a lower frequency when moving away Doppler modes analyze this frequency shift and use the Doppler equation to estimate velocities of blood flow Velocity (v) can be calculated as follows: $v = \frac{c * \Delta f}{2f_i * \cos\theta}$ <p>Where c = the propagation speed of sound in blood (1540 m/s), Δf is the difference between the transmitted frequency (f_i) and the received frequency, and $\cos\theta$ is the angle of incidence between the ultrasound beam and blood flow</p> <ul style="list-style-type: none"> Angle-dependent Maximum Doppler shift occurs when blood flow is directly parallel to the beam. When the incident angle is ≤ 20 degrees, the percentage error in estimating the velocity is $\leq 6\%$. At greater incident angles the error significantly increases. Includes spectral (pulsed-wave, continuous wave) and color flow Doppler
Pulsed-Wave Doppler (PWD)	<ul style="list-style-type: none"> Samples blood flow velocities at a specific location The frequency of sampling determines the maximum velocity that can be detected and depends on imaging depth This maximum velocity is half of the pulse repetition frequency and referred to as the Nyquist limit Above the Nyquist limit, <i>aliasing</i> occurs. The Doppler information is displayed ambiguously, displaying both positive and negative velocities
Continuous Wave Doppler (CWD)	<ul style="list-style-type: none"> Transducer continuously transmits and receives ultrasound energy along the length of the ultrasound beam Allows measurement of higher velocity flows, such as those that are encountered with stenotic or regurgitant lesions The location of the high velocity flow may occur anywhere along the length of the beam, a limitation referred to as <i>range ambiguity</i>
Color Flow Doppler (CFD)	<ul style="list-style-type: none"> A form of pulsed-wave Doppler The direction and velocity of flow are measured in multiple sample volumes within an imaging sector. The direction and velocity information are displayed according to a color scale Flow data are superimposed on the corresponding 2D image Limited by aliasing Lower temporal resolution than 2D imaging
Doppler Tissue Imaging (DTI)	<ul style="list-style-type: none"> Used to assess myocardial motion Eliminates the high-velocity, low-amplitude signals from red blood cells and displays the low-velocity, high-amplitude signals from tissue Angle-dependent Does not distinguish between translational motion and contraction

of contrast echocardiography include improved endocardial border delineation for assessment of function, assessment of regional wall motion, and exclusion of intracardiac thrombus or mass.

Three-Dimensional Image Acquisition

3-D imaging provides information that complements the findings from the 2-D examination. Transducers with 3-D capabilities contain thousands of piezoelectric elements in a rectangular (or matrix) array configuration and create

pyramidal-shaped images. The optimal 3-D image involves tradeoffs between temporal resolution, spatial resolution, and sector size. 3-D imaging is susceptible to the same artifacts as 2-D imaging and additional artifacts unique to 3-D image construction. We encourage the interested reader to refer to published 3-D echocardiography guidelines⁴¹ as well as a practical overview focused on intraoperative 3-D image acquisition.⁴²

IMAGING MODES

Two-Dimensional Multiplane Acquisition

Matrix array (3-D) transducers have multiplane imaging capabilities and can simultaneously display two or

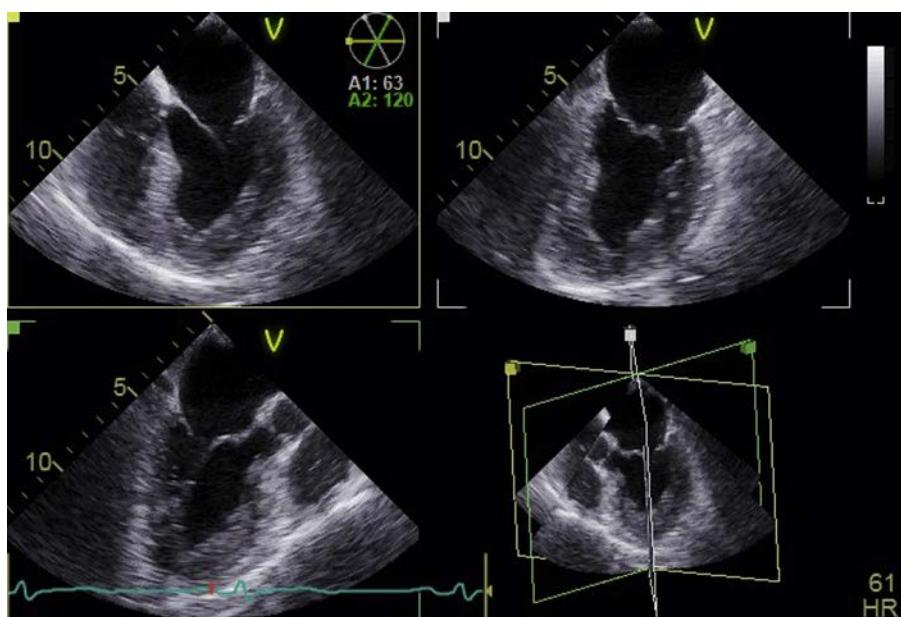


Fig. 37.2 Simultaneous display of multiple two-dimensional scan planes by multiplane imaging. The upper left panel (yellow panel) displays the primary reference imaging plane. The circular icon in this panel indicates the positions of the secondary imaging planes. Images from the secondary planes are displayed at the top right (white panel) and bottom left (green panel). A three-dimensional representation of the imaging planes and their angulation is displayed at the bottom right.

BOX 37.1 Perioperative Applications of Multiplane Imaging

- Simultaneous visualization of segmental wall motion
- Characterization of the mechanism and origin of valvular regurgitation
- Procedural guidance during percutaneous closure of atrial septal defect
- Guidance of transseptal puncture for transcatheter left atrial appendage and mitral valve procedures
- Evaluation of the left atrial appendage morphology and exclusion of thrombus

more live 2-D imaging planes. In biplane imaging, the first image serves as the reference view, and the second image is obtained by rotating the scanning plane around the longitudinal axis of the reference view. The secondary image can also be adjusted by tilting the imaging plane in the elevational or lateral axis. Multiplane imaging allows simultaneous display of multiple rotatable imaging planes (Fig. 37.2). **Box 37.1** lists common perioperative applications of multiplane imaging.

Real-Time Three-Dimensional Imaging

3-D images can be displayed live or in “real time.” RT-3D imaging acquires data over a single heartbeat; some authors refer to this as 4-D imaging. The proprietary nomenclature varies, but there are three main RT-3D modes of differing sector size:

- Narrow sector: this mode displays a pyramidal volume with the best temporal and spatial resolution of the live modes. The major limitation is that it often does not capture an entire structure of interest (Fig. 37.3).
- Wide sector: this mode “zooms in” on a selected region of interest. There is a decrease in temporal and spatial resolution compared to narrow sector imaging. Images

are easy to acquire. This mode is ideal for real-time image manipulation (see Fig. 37.3).

- Full-volume: full-volume mode has the largest imaging sector. Live full-volume images have reduced temporal and spatial resolution. Ideally, acquisition of full-volume images occurs over multiple beats. Live full-volume mode has utility when multiple-beat acquisition is not feasible.

Gated Acquisition

Gated acquisition divides the imaging volume into multiple narrow subvolumes that are acquired over a specified number of heartbeats. “Stitching” the subvolumes together creates the final image. In order to obtain the subvolumes at the same time in the cardiac cycle, image acquisition is gated to the R-wave of the electrocardiogram (ECG). Gated acquisition requires normal cardiac rhythm and the absence of electrical interference and respiratory variation.

- Wide sector: multiple-beat gated acquisition using wide sector mode significantly improves the temporal resolution of the “zoomed in” view. Spatial resolution marginally improves.
- Full-volume: this mode has the largest sector size with optimal spatial resolution and high temporal resolution (Fig. 37.4; Video 37.1).

Color Flow Doppler

CFD may be incorporated with any of the modalities, but leads to a reduction in temporal resolution. Optimal CFD images are obtained using R-wave gated multiple beat acquisitions.

QUANTITATIVE ANALYSIS

Multiplanar Reformatting

Multiplanar reformatting enables alignment of orthogonal planes to accurately measure linear dimensions and areas (e.g., planimetry of stenotic orifice, annular area).

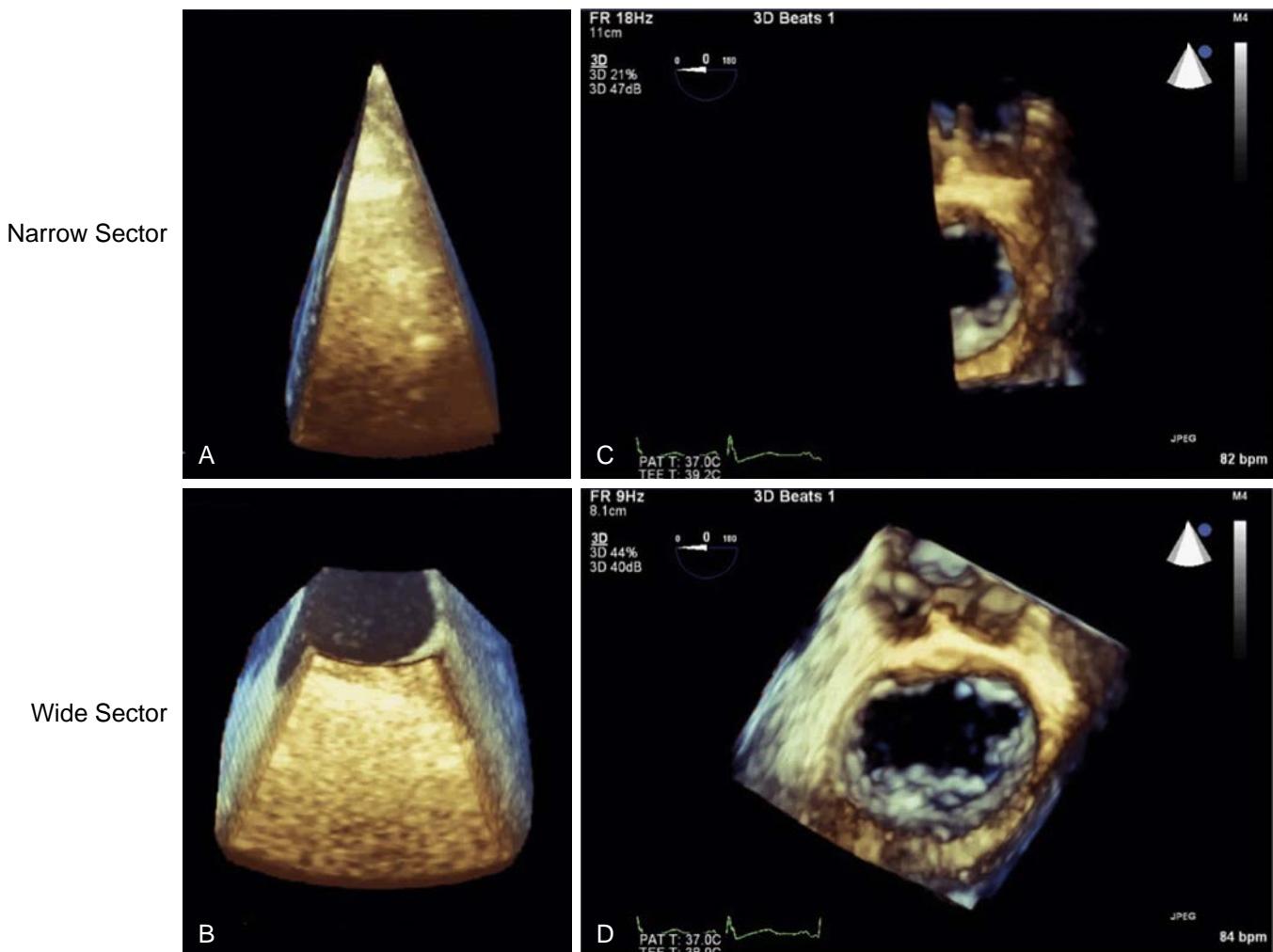


Fig. 37.3 Real-time three-dimensional imaging using narrow sector (top) and wide sector (bottom) modes. (A) Narrow sector imaging displays a narrow, pyramidal volume. (B) Wide sector imaging displays a defined region of interest selected from a larger pyramidal volume. (C) Narrow sector image of the mitral valve after cropping and rotating. Only a portion of the mitral valve structure is visualized. (D) Wide sector image of the mitral valve after cropping and rotating. The entire mitral valve structure is visualized, but at the expense of decreased spatial and temporal resolution.

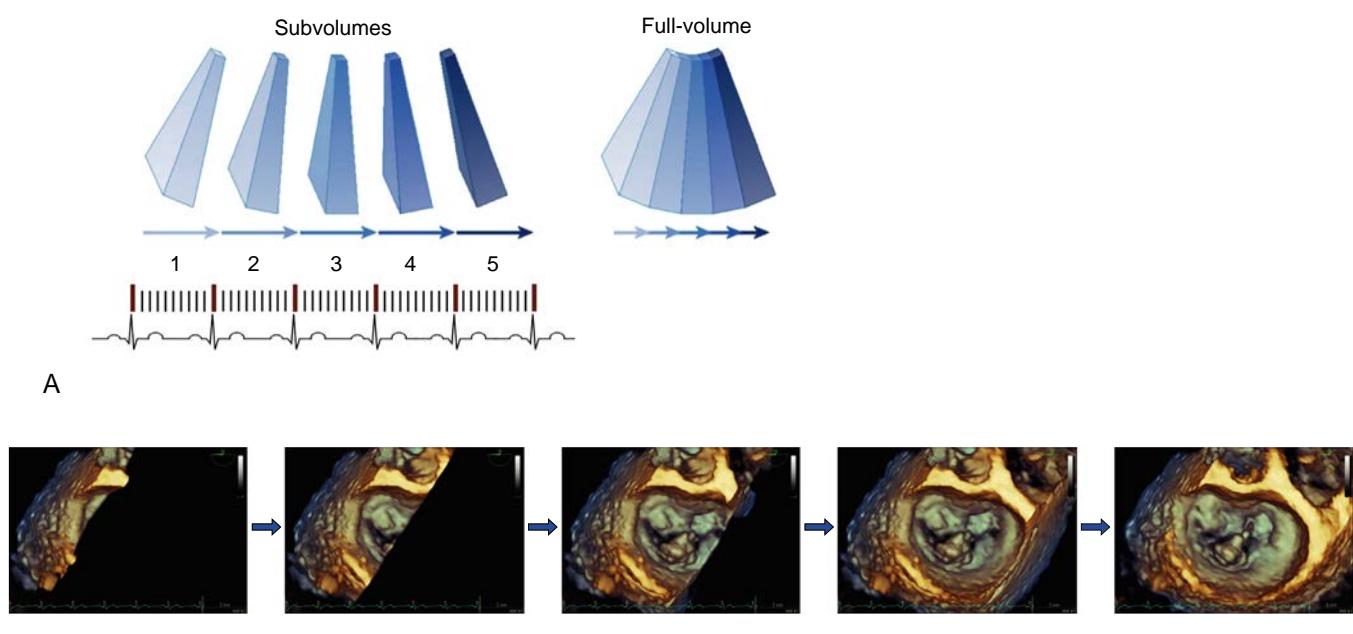


Fig. 37.4 (A) Schematic of multibeat gated full-volume image acquisition. Subvolume acquisition is gated to the R-wave of the electrocardiogram. In this example, subvolume acquisition occurs over five consecutive heartbeats. The individual subvolumes are then synchronized and "stitched" together to create a larger full-volume three-dimensional image. (B) Creation of the three-dimensional (3D) full-volume image from narrow subvolumes. (Modified from Desjardins G. Perioperative echocardiography. In: Miller R, ed. *Miller's Anesthesia*. 8th ed. Philadelphia, PA: Elsevier/Saunders; 2015:1396–1428.)

Advanced Applications

Quantitative applications exist for 3-D imaging and eliminate some of the geometric assumptions required in 2-D imaging calculations. Example analyses include calculation of ventricular ejection fraction (EF), analysis of mitral valve structure, and dynamic annular measurements. The degree of automation and need for post-processing manipulation vary. Some analyses are performed exclusively offline, making them useful in research, but less applicable to intraoperative decision making.

Indications and Practice Guidelines

PERIOPERATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

The American Society of Anesthesiology (ASA) and Society of Cardiovascular Anesthesiologists (SCA) issued perioperative TEE practice guidelines in 1996, categorizing indications for TEE in various clinical settings.⁴³ An updated version in 2010 recommended the indications for perioperative TEE based on literature review and expert opinion.⁴⁴ In the absence of contraindications, TEE should be used in all adult open heart and thoracic aortic surgical procedures. It should be considered in coronary artery bypass graft surgeries to confirm diagnostic information, detect pathology, influence the anesthetic or surgical plan, and assess surgical results. It is recommended that TEE be considered on a case-by-case basis in small children undergoing cardiac surgery due to unique risks in this population. For catheter-based intracardiac procedures, the 2010 guidelines state TEE may be used. Separate guidelines focus specifically on the role of echocardiography in interventional and catheter-based procedures.^{45,46} In noncardiac surgery, TEE is indicated in the setting of persistent unexplained life-threatening circulatory instability. If the surgical procedure or the patient's known or suspected pathology could result in severe hemodynamic, pulmonary, or neurologic compromise, TEE may be used. TEE is recommended in critical care if the diagnostic information is expected to alter management and cannot be obtained by other modalities expediently. European guidelines offer similar recommendations for the use of perioperative TEE,⁴⁷⁻⁴⁹ while the American Heart Association (AHA) and American College of Cardiology (ACC) make more limited recommendations regarding specific indications for intraoperative TEE.^{50,51}

In addition to describing indications for perioperative TEE, the 1996 ASA/SCA guidelines also distinguish between basic and advanced levels of training.⁴³ In 2002, an American Society of Echocardiography (ASE)/SCA Joint Task Force authored guidelines for training in perioperative echocardiography, defined as TEE, epicardial echocardiography, or epiaortic ultrasonography performed in surgical patients immediately before, during, or after surgery.⁵² These guidelines do not include TTE. Expectations for cognitive and technical skills of both basic and advanced training are outlined with recommendations for the minimum numbers of echocardiographic examinations performed. Further discussion of training and certification occurs at the end of the chapter.

TABLE 37.3 List of Contraindications to Transesophageal Echocardiography

Absolute Contraindications	Relative Contraindications
Esophageal pathology <ul style="list-style-type: none"> ■ Diverticulum ■ Laceration ■ Stricture ■ Tumor 	History of: <ul style="list-style-type: none"> ■ Dysphagia ■ Radiation to neck and chest ■ Upper gastrointestinal surgery
Active upper gastrointestinal bleeding	Recent upper gastrointestinal bleeding
History of esophagectomy	Active peptic ulcer disease, esophagitis
Perforated viscus	Esophageal varices Barrett's esophagus Symptomatic hiatal hernia Restricted cervical spine mobility <ul style="list-style-type: none"> ■ Atlantoaxial instability ■ Severe cervical arthritis Coagulopathy or thrombocytopenia

Adapted from Hahn RT, Abraham T, Adams MS, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr*. 2013;26(9):921–964.

CRITICAL CARE

Critical care societies have further defined competencies and applications of echocardiography in critical care beyond that established by the ASA/SCA guidelines. Several internationally based expert panels have proposed training objectives and competency-based training standards in consensus statements. Similar to the distinction between basic and advanced training in perioperative echocardiography, these statements distinguish between basic and advanced critical care echocardiography,⁵³⁻⁵⁶ or a basic and expert skill set.⁵⁷ The Society of Critical Care Medicine has provided evidence-based recommendations for the use of bedside echocardiography (TEE and TTE) in the evaluation of the critically ill patient.⁵⁷

Transesophageal Echocardiography Examination

CONTRAINDICATIONS AND COMPLICATIONS

TEE is a safe and relatively minimally invasive procedure, although echocardiographers should be aware of potential complications resulting from probe insertion and manipulation. Table 37.3 lists absolute and relative contraindications to TEE. The risks and benefits must be carefully considered in patients with oral, esophageal, or gastric disease. Hilberath and associates provide an excellent summary on the safety of TEE, with reviewed studies reporting an overall complication rate in adults of 0.18% to 2.8% and major complication rate of 0.2% to 1.2%.⁵⁸ A 2017 retrospective study reviewed recent data regarding complications in a contemporary cardiac surgery cohort.⁵⁹ Of the 7948 patients that underwent

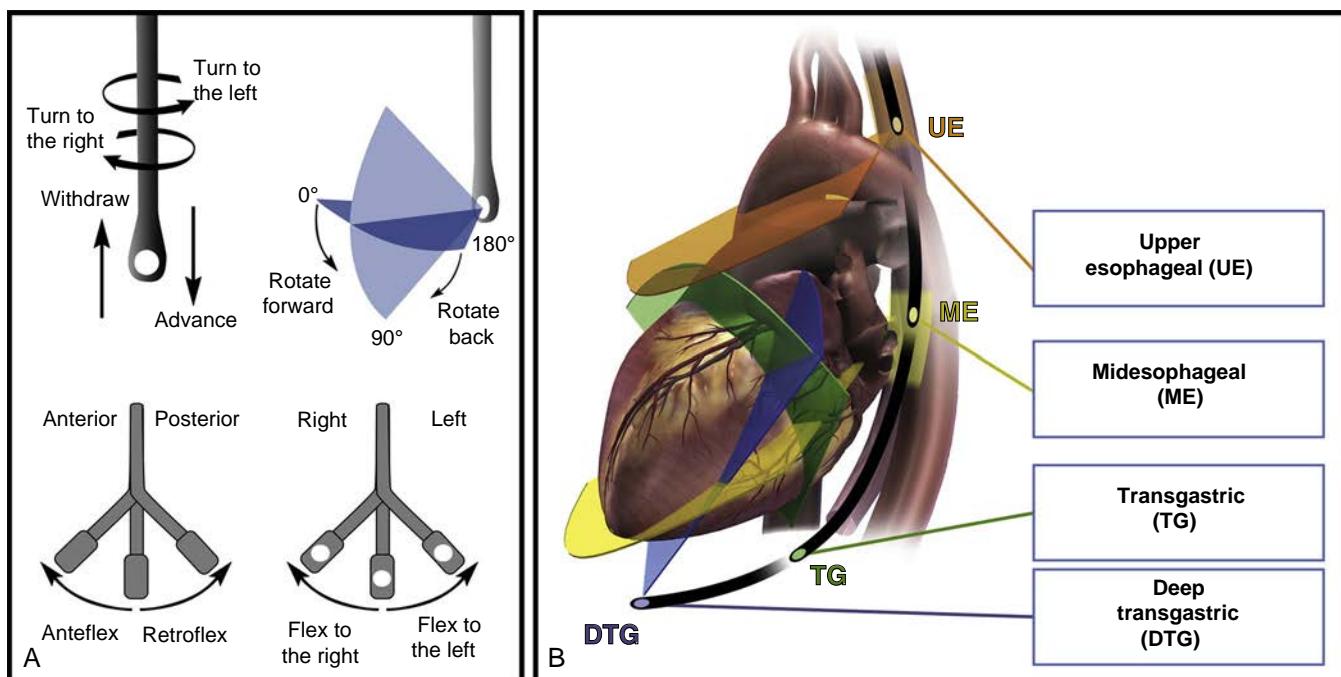


Fig. 37.5 Terminology used to describe manipulation of the transesophageal echocardiographic probe during image acquisition. (A) Terminology used for the manipulation of the transesophageal echocardiographic probe. (B) Four standard transducer positions within the esophagus and stomach and the associated imaging planes. (From Hahn RT, Abraham T, Adams MS, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr*. 2013;26(9):921–964.)

TEE, 111 (1.4%) were identified as having complications likely attributable to TEE (no other cause identified). Incidence of significant dysphagia was 0.3% and esophageal or gastric complications 0.9%. Independent risk factors associated with complications were age, body mass index, previous stroke, procedure other than isolated coronary artery bypass grafting (CABG), cardiopulmonary bypass time, and return to the operating room for any reason.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY IMAGE ACQUISITION

Probe Manipulation and Imaging Planes

TEE probe manipulation includes withdrawal/advancement, turning left/right, and angle adjustment. The large knob on the handle controls anteflexion/retroflexion and the smaller knob allows flexion to the left and right. TEE image acquisition occurs at four different levels: upper esophageal (UE), midesophageal (ME), transgastric (TG), and deep transgastric (DTG) (Fig. 37.5).

Basic Examination

The basic perioperative TEE examination serves as an intraoperative monitoring tool used to identify cardiac causes of hemodynamic or respiratory instability.⁶⁰ The basic perioperative examination includes 11 views suited to evaluating hemodynamic instability. A suggested examination sequence progresses through the 11 views as follows: ME four-chamber (ME 4C), ME two-chamber (ME 2C), ME long axis (ME LAX), ME

ascending aorta long-axis, ME ascending aorta short-axis, ME aortic valve short-axis (ME AV SAX), ME right ventricular inflow-outflow (ME RV Inflow-Outflow), ME bicaval, TG midpapillary short-axis (TG SAX), descending aorta short-axis, and descending aorta long-axis.

Comprehensive Examination

A comprehensive TEE examination enables practitioners with appropriate training to fully utilize the diagnostic capabilities of TEE. Twenty-eight views (including the 11 basic views) are recommended for comprehensive assessment (Fig. 37.6). Technical aspects of image acquisition have been extensively reviewed elsewhere.⁶¹ Echocardiographers should complete their examinations systematically to avoid missing key findings. As a result of individual patient anatomy and the time constraints of the intraoperative environment, not all views are obtained in every examination.

Epicardial and Epiaortic Imaging

While epicardial echocardiography ushered in the era of intraoperative echocardiography, its use was supplanted by TEE. In the setting of contraindications to TEE or the need for better visualization of anterior structures, epicardial echocardiography remains an alternative modality.⁶²

Epicardial ultrasound (EAU) serves as an imaging adjunct, allowing visualization of the ascending aorta and aortic arch, regions that are unable to be visualized on TEE. Detection of ascending aorta atheroma is superior with

Imaging Plane	3D Model	2D TEE Image	Acquisition Protocol	Structures Imaged	Imaging Plane	3D Model	2D TEE Image	Acquisition Protocol	Structures Imaged
Midesophageal Views									
1. ME 5-Chamber View			Transducer Angle: ~ 0 - 10° Level: Mid-esophageal Maneuver (from prior image): NA	Aortic valve LVOT Left atrium/Right atrium Left ventricle/Right ventricle/IVS Mitral valve (A ₁ A ₂ -P ₁ P ₂) Tricuspid valve	16. TG Basal SAX View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Advance ± Anteflex	Left ventricle (base) Right ventricle (base) Mitral valve (SAX) Tricuspid valve (short-axis)
2. ME 4-Chamber View			Transducer Angle: ~ 0 - 10° Level: Mid-esophageal Maneuver (from prior image): Advance ± Retroflex	Left atrium/Right atrium IAS Left ventricle/Right ventricle/IVS Mitral valve (A ₁ A ₂ -P ₁ P ₂) Tricuspid valve	17. TG Mid-Papillary SAX View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Advance ± Anteflex	Left ventricle (mid) Papillary muscles Right ventricle (mid)
3. ME Mid-Commissural View			Transducer Angle: ~ 50 - 70° Level: Mid-esophageal Maneuver (from prior image): NA	Left atrium Coronary sinus Left ventricle Mitral valve (P ₁ -A ₁ A ₂ -P ₂) Papillary muscles Chordae tendinae	18. TG Apical SAX View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Advance ± Anteflex	Left ventricle (apex) Right ventricle (apex)
4. ME 2-Chamber View			Transducer Angle: ~ 80 - 100° Level: Mid-esophageal Maneuver (from prior image): NA	Left atrium Coronary sinus Left atrial appendage Left ventricle Mitral valve (P ₁ -A ₁ A ₂ -P ₂)	19. TG RV Basal View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Anteflex	Left ventricle (mid) Right ventricle (mid) Right ventricular outflow tract Tricuspid Valve (SAX) Pulmonary Valve
5. ME Long Axis View			Transducer Angle: ~ 120 - 140° Level: Mid-esophageal Maneuver (from prior image): NA	Left atrium LVOT RVOT Mitral valve (P ₁ -A ₁) Aortic valve Proximal ascending aorta	20. TG RV Inflow View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Right-flex	Right atrium Right ventricle Right ventricular outflow tract Pulmonary valve Tricuspid Valve
6. ME AV LAX View			Transducer Angle: ~ 120 - 140° Level: Mid-esophageal Maneuver (from prior image): Withdrawal ± anteflex	Left atrium LVOT RVOT Mitral valve (A ₁ -P ₁) Aortic valve Proximal ascending aorta	21. TG RV 4-chamber View			Transducer Angle: ~ 0 - 20° Level: Transgastric Maneuver (from prior image): Left-flex, Advance, Anteflex	Left ventricle Left ventricular outflow tract Right ventricle Aortic valve Aortic root Mitral Valve
7. ME Ascending Aorta LAX View			Transducer Angle: ~ 90 - 110° Level: Upper-Esophageal Maneuver (from prior image): Withdrawal	Mid-ascending aorta Right pulmonary artery	22. TG 2-chamber View			Transducer Angle: ~ 90 - 110° Level: Transgastric Maneuver (from prior image): Neutral flexion, Withdraw	Left ventricle Left atrium/appendage Mitral valve
8. ME Ascending Aorta SAX View			Transducer Angle: ~ 0 - 30° Level: Upper-Esophageal Maneuver (from prior image): CW	Mid-ascending aorta Main/bifurcation pulmonary artery Superior vena cava	23. TG RV Inflow View			Transducer Angle: ~ 90 - 110° Level: Transgastric Maneuver (from prior image): CW	Right ventricle Right atrium Tricuspid valve
9. ME Right Pulmonary Vein View			Transducer Angle: ~ 0 - 30° Level: Upper-Esophageal Maneuver (from prior image): CW, Advance	Mid-ascending aorta Superior vena cava Right pulmonary veins	24. TO LAX View			Transducer Angle: ~ 120 - 140° Level: Transgastric Maneuver (from prior image): CCW	Left ventricle Left ventricular outflow tract Right ventricle Aortic valve Aortic root Mitral valve
10. ME AV SAX View			Transducer Angle: ~ 25 - 45° Level: Mid-esophageal Maneuver (from prior image): CCW, Advance, Anteflex	Aortic valve Right atrium Left atrium Superior IAS RVOT Pulmonary Valve	25. Descending Aorta SAX View			Transducer Angle: ~ 0 - 10° Level: Transgastric to Mid-esophageal Maneuver (from prior image): Neutral flexion	Descending aorta Left thorax Hemiazygous and Azygous veins Intercostal arteries
11. ME RV Inflow-Outflow View			Transducer Angle: ~ 50 - 70° Level: Mid-esophageal Maneuver (from prior image): CW, Advance	Aortic valve Right atrium Left atrium Superior IAS Tricuspid Valve RVOT Pulmonary Valve	26. Descending Aorta LAX View			Transducer Angle: ~ 0 - 10° Level: Transgastric to Mid-esophageal Maneuver (from prior image): Neutral flexion	Descending aorta Left thorax
12. ME Modified Bicaval TV View			Transducer Angle: ~ 50 - 70° Level: Mid-esophageal Maneuver (from prior image): CW	Left atrium Right atrium Left atrial appendage IAS Superior vena cava Inferior vena cava/coronary sinus	27. TO Aortic Arch LAX View			Transducer Angle: ~ 0 - 10° Level: Upper-Esophageal Maneuver (from prior image): Withdrawal	Aortic arch Innominate vein Mediastinal tissue
13. ME Bicaval View			Transducer Angle: ~ 90 - 110° Level: Mid-esophageal Maneuver (from prior image): CW	Left atrium Right atrium/appendage IAS Superior vena cava Inferior vena cava	28. TO Aortic Arch SAX View			Transducer Angle: ~ 70 - 90° Level: Transgastric to Mid-esophageal Maneuver (from prior image): NA	Aortic arch Innominate vein Pulmonary artery Pulmonary valve Mediastinal tissue
14. UE Right and Left Pulmonary Vein View			Transducer Angle: ~ 90 - 110° Level: Upper-esophageal Maneuver (from prior image): Withdraw, CW for the right veins, CCW for the left veins	Pulmonary vein (upper and lower) Pulmonary artery					
15. ME Left Atrial Appendage View			Transducer Angle: ~ 90 - 110° Level: Mid-esophageal Maneuver (from prior image): Advance	Left atrial appendage Left upper pulmonary vein					

Fig. 37.6 The 28 suggested views of the comprehensive transesophageal echocardiographic (TEE) examination. Each view is shown as a 3D image, the corresponding imaging plane, and a 2D image. The acquisition protocol and the structures imaged in each view are listed in the subsequent columns. The green boxes indicate the 11 views of the basic TEE examination. (Modified from Hahn RT, Abraham T, Adams MS, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr*. 2013;26(9):921-964.)

EAU compared to that of TEE or manual palpation.^{63–65} EAU impacts surgical strategy, but data as to whether this translates into improved clinical outcomes are limited and heterogeneous.^{63–65} Examination guidelines and example imaging views are available.⁶⁶

Transthoracic Echocardiography Examination

TRANSTHORACIC ECHOCARDIOGRAPHY IMAGE ACQUISITION

Three elements describe transthoracic sonographic views: (1) transducer position or window, (2) imaging plane, and (3) structures imaged. When feasible, patients should turn to their left side with the left hand resting behind the head. The main windows are parasternal, apical, subcostal, and suprasternal. Imaging motions include translation (sliding), tilting, angulation, and rotation.

Focused Cardiac Ultrasound Versus Limited Examination

With increasing use of ultrasonography at the point of care, it is important for the clinician to recognize the distinction between focused cardiac ultrasound (FoCUS, FCU) and limited TTE examination. Guidelines endorsed by several societies describe the key differences between the two examinations.^{67,68} FoCUS is a simplified, point-of-care ultrasound examination that complements the physical examination (Box 37.2). FoCUS addresses specific questions within a given clinical context. Relevant abnormalities are characterized as present or absent, and questions are answered in a yes/no format. In contrast, limited TTE is broad in scope. Interpretation includes normal, pathological, and incidental findings, and quantitative techniques may be employed. Fewer images are acquired in a limited examination compared to a comprehensive examination.⁶⁷ Limited TTE requires a practitioner with advanced training and expertise.

Abnormalities identified by FoCUS require follow-up with formal comprehensive echocardiography. If FoCUS does not reveal pathology but there is clinical suggestion of cardiac disease, formal comprehensive echocardiography should also be pursued. Many FoCUS protocols exist, most including five key views: parasternal long-axis (PLAX), parasternal short-axis (PSAX), apical four-chamber (A4C), subcostal four-chamber (SC4), and subcostal inferior vena cava (SIVC) (Fig. 37.7 through 37.10; Video 37.2). Protocols often incorporate lung ultrasound. For those interested in details of image acquisition, we refer the reader to a practical introductory primer.⁶⁹

Comprehensive Examination

A minority of anesthesiologists have the necessary training required to perform and/or formally interpret comprehensive TTE examinations. Standard imaging views occur at the parasternal, apical, and subcostal windows, plus the suprasternal notch. In addition to the views included in FoCUS, a comprehensive TTE examination includes the

BOX 37.2 Features of Focused Cardiac Ultrasound

- Goal-directed
- Problem-oriented
- Limited in scope
- Simplified
- Time sensitive and repeatable
- Qualitative or semiquantitative
- Performed at the point of care
- Usually performed by clinicians

FoCUS, Focused Cardiac Ultrasound.

Adapted from Via G, Hussain A, Wells M, et al. International evidence-based recommendations for focused cardiac ultrasound. *J Am Soc Echocardiogr.* 2014;27(7):e681–e683 e633.

PLAX right ventricular outflow, PLAX right ventricular inflow, PSAX apical, PSAX mitral (basal), PSAX aortic, apical right ventricle-focused, apical five-chamber (A5C), apical two-chamber (A2C), apical three-chamber (A3C), and suprasternal notch long axis views.⁷⁰

Qualitative Assessment

VENTRICULAR SIZE AND FUNCTION

Left Ventricle

Qualitative visual assessment of LV systolic function is the mainstay of perioperative echocardiography. Visual estimation of EF by TTE correlates well with quantitative methods^{71,72} and real-time intraoperative TEE interpretation can reasonably estimate function compared to formal offline methods.⁷³ Wall thickening is symmetric and vigorous when LV systolic function is normal (Video 37.3). The key TEE views for global assessment of LV function include the TG SAX (Fig. 37.11), ME 4C, ME 2C, and ME LAX views (Fig. 37.12). Analogous TTE views are PSAX, A4C, A2C, and PLAX views. The TG SAX and PSAX views display distributions of all three coronary arteries.

Assessment of regional function focuses on the thickening and shortening of individual segments. Translational motion, tethering, or dyssynchrony due to conduction delay or pacing can make interpretation challenging. A 17-segment model divides the heart into six basal segments (from the mitral valve annulus to the tips of the papillary muscles), six midpapillary level segments (from the tips to the base of the papillary muscles), four apical (or distal) segments (from the base of the papillary muscles to the LV apex), and the apical cap.⁷⁴ The apical cap is difficult to visualize with TEE due to foreshortening. Segmental thickening should be assessed in the context of coronary distribution, recognizing variability among individuals. The left anterior descending artery (LAD) consistently supplies the anterior and anteroseptal segments and apical cap (Fig. 37.13). The right coronary artery (RCA) supplies the right ventricle (RV) and the basal inferior, basal inferoseptal, and mid-inferior and possibly the mid-inferoseptal segments, the latter of which may be supplied by the LAD. The left circumflex typically supplies the lateral segments, although

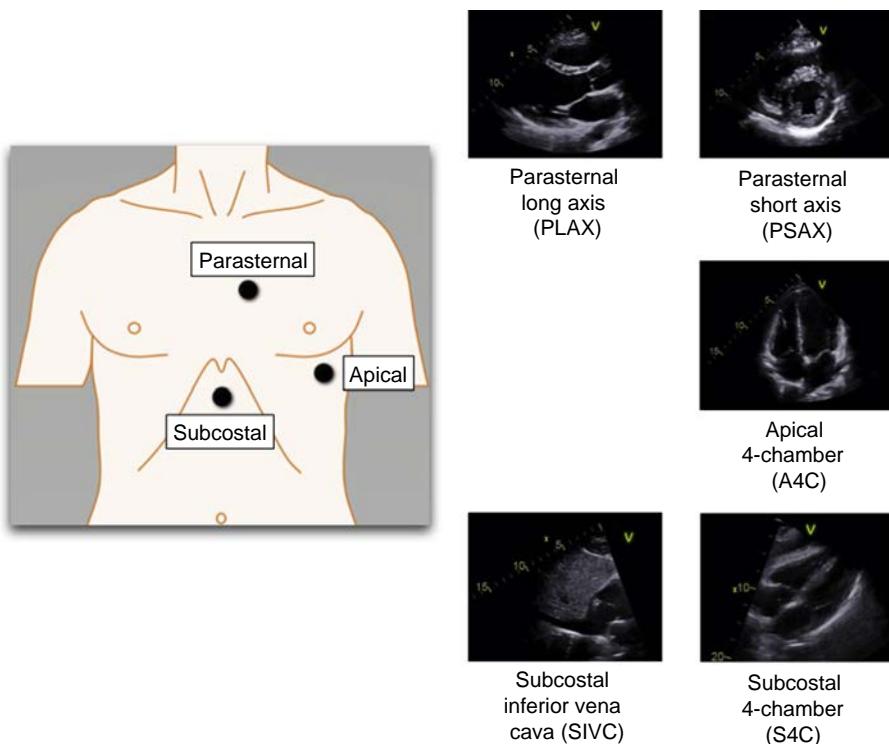


Fig. 37.7 Views included in the focused cardiac ultrasound (FoCUS) examination: parasternal long axis (PLAX), parasternal short axis (PSAX), apical 4-chamber (A4C), subcostal 4-chamber (S4C), and subcostal inferior vena cava (SIVC). The parasternal window is between the third through fifth intercostal space to the left of the sternum. The apical window is near the point of maximal impulse along the mid-axillary line, often near the fifth intercostal space. The subcostal window is just below the xiphoid, along the midline or slightly to the patient's right. (Adapted from Via G, Hussain A, Wells M, et al. International evidence-based recommendations for focused cardiac ultrasound. *J Am Soc Echocardiogr*. 2014;27(7):683 e681–683 e633.)

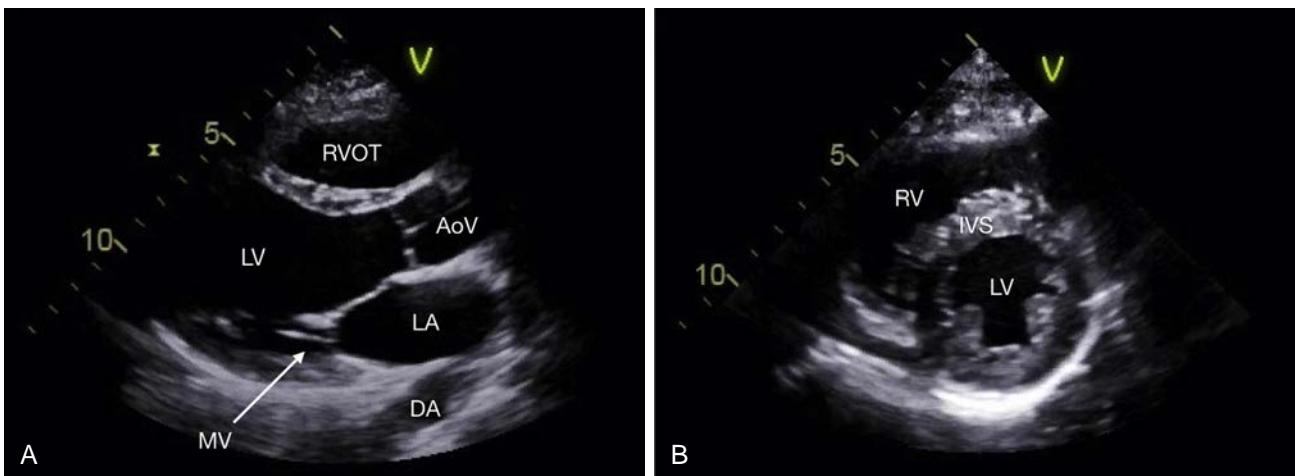


Fig. 37.8 Transthoracic parasternal views included in focused cardiac ultrasound. (A) The parasternal long axis view is shown. This view is analogous to the transesophageal midesophageal long axis view. In transthoracic imaging, anterior structures are closest to the transducer (and therefore displayed at the top of the image), while in transesophageal imaging, posterior structures are in closest proximity to the transducer (and displayed at the top of the image). (B) The parasternal short axis view is shown. This view is analogous to the transesophageal transgastric mid-papillary short axis view. AoV, Aortic valve; DA, descending aorta; IVS, interventricular septum; LA, left atrium; LV, left ventricle; MV, mitral valve; RV, right ventricle; RVOT, right ventricular outflow tract.

contributions from the LAD to the anterolateral segments or RCA to the inferolateral segments are not uncommon.⁷⁵ Each segment should be evaluated as normal/hyperkinetic, hypokinetic (reduced thickening), akinetic (minimal or absent thickening), or dyskinetic (thinning, stretching, or aneurysmal). Echocardiography is more sensitive than the ECG in detecting ischemic changes.⁷⁶

Right Ventricle

The complex geometry of the RV makes assessment more challenging. Qualitatively, the RV should appear approximately two-thirds the size of the left ventricle, assessed in ME 4C and A4C views. Typically the right ventricle does not extend to the LV apex. RV hypertrophy suggests chronic pulmonary hypertension or the presence of cardiomyopathies.

The interventricular septum provides additional information about RV pathology. Normally, the septum curves toward the RV, and the LV cavity appears circular in short axis. In states of RV *volume* overload, the septum shifts away from the RV and flattens resulting in a D-shaped LV cavity mainly in *mid-to-late diastole* (Video 37.4). RV *pressure* overload leads to leftward septal shift and septal flattening throughout the cardiac cycle, with the changes most pronounced at *end-systole* (Video 37.5).⁷⁷

VALVULAR FUNCTION

Qualitative assessment of valvular function begins with sequential assessment of the valve in multiple imaging planes, noting the motion of the leaflets, presence of leaflet

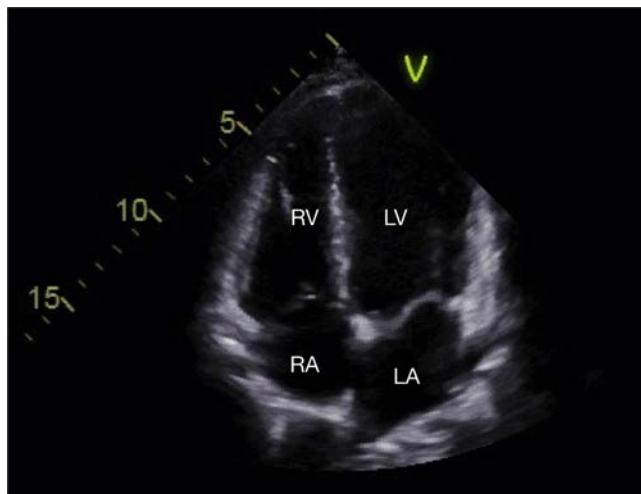


Fig. 37.9 Transthoracic apical four-chamber view. This view is analogous to the midesophageal four-chamber view in transesophageal echocardiography. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

thickening or calcification, vegetations or masses, and additional structural abnormalities (Fig. 37.14). Malcoaptation and annular dilation help define the etiology of dysfunction in regurgitant lesions. 3-D imaging modalities clarify the location of structure abnormalities.

CFD provides visual information about flow through valves. Aliasing or turbulence of forward flow through a valve should prompt evaluation for additional signs of stenosis. Further characterization of stenotic lesions is primarily quantitative in nature. Color Doppler imaging also detects

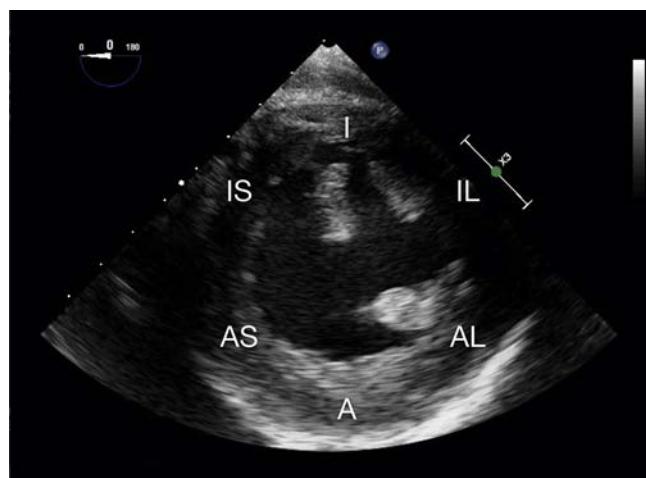


Fig. 37.11 The transesophageal transgastric mid-papillary short axis view (TG SAX) is a common view for qualitative evaluation of global left ventricular systolic function. Basal and apical segments are not visualized in this view, so midesophageal or TG LAX views should also be obtained to provide a more complete picture of the overall function. The TG SAX view includes myocardial segments supplied by each of the coronary arteries, making it useful for detection of new ischemia. The TG SAX view displays the mid inferior (*I*), mid inferolateral (*IL*), mid anterolateral (*AL*), mid anterior (*A*), mid anteroapical (*AS*), and mid inferoseptal (*IS*) segments.

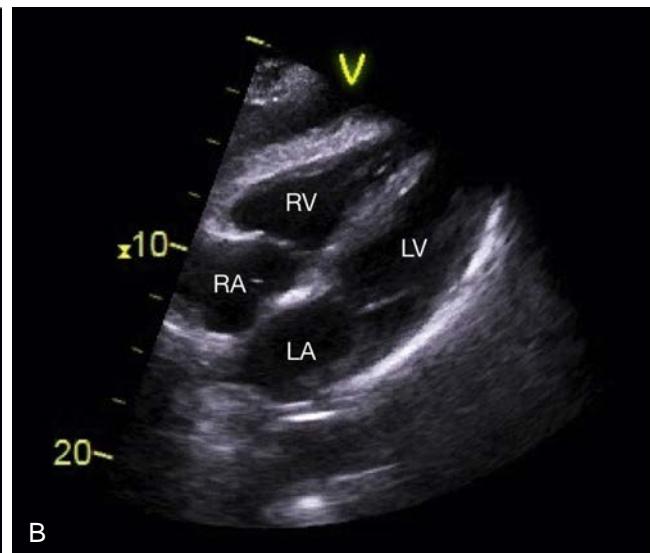
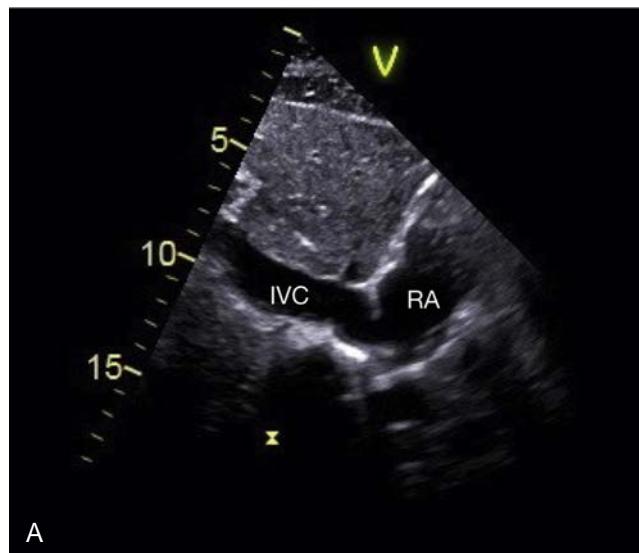


Fig. 37.10 Transthoracic subcostal views included in focused cardiac ultrasound. (A) The subcostal inferior vena cava (IVC) view is shown. The IVC diameter and collapsibility index are measured in this view to estimate right atrial pressure in spontaneously breathing individuals (see text). (B) The subcostal four-chamber view is shown. This view is useful for ultrasonographic examination during cardiac arrest, because it can be performed without interrupting chest compressions. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

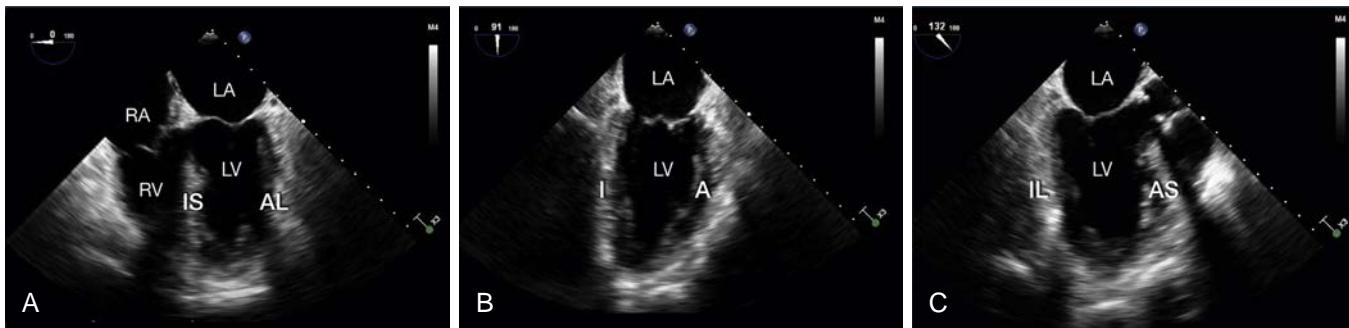


Fig. 37.12 Midesophageal views for assessing left ventricular function using transesophageal echocardiography. (A) The midesophageal four-chamber (ME 4C) view displays the inferoseptal (IS) and anterolateral (AL) walls. (B) The midesophageal two-chamber (ME 2C) view displays the inferior (I) and anterior (A) walls. (C) The midesophageal long axis view (ME LAX) displays the inferolateral (IL) and anteroseptal (AS) walls. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

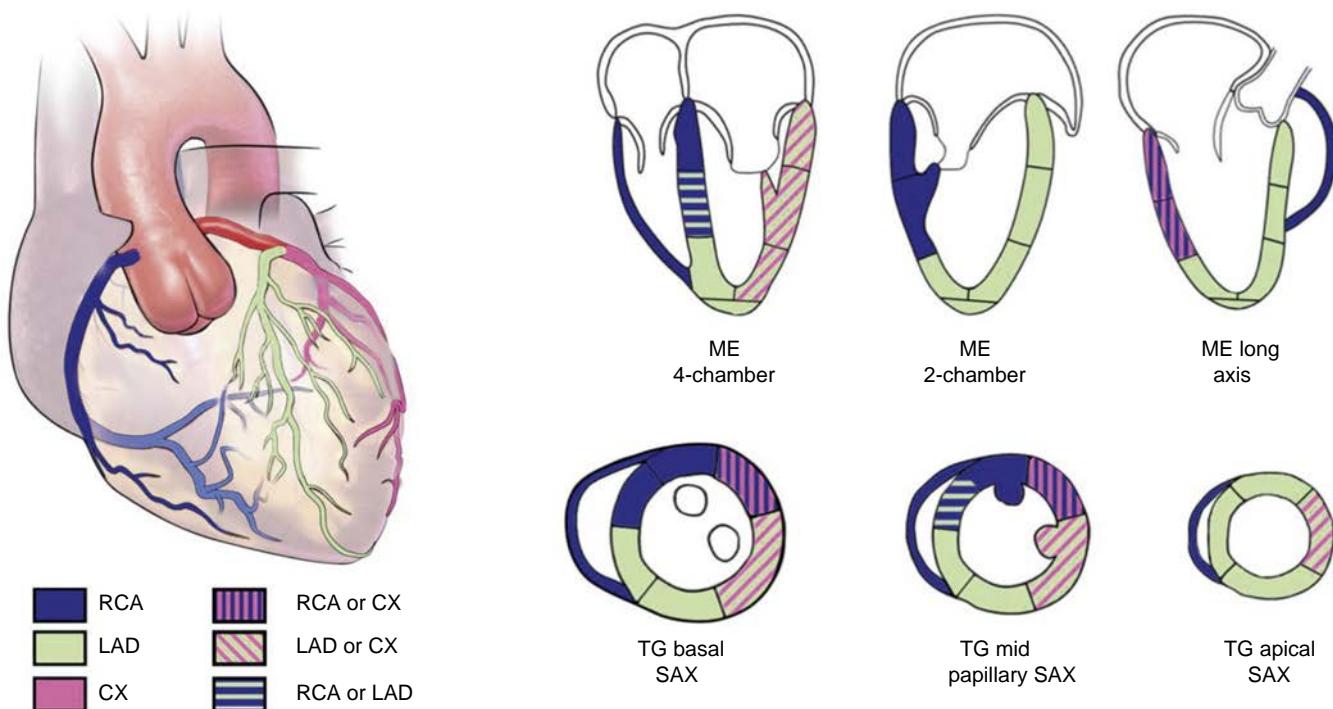


Fig. 37.13 Typical distributions of the right coronary artery (RCA), the left anterior descending coronary artery (LAD), and the circumflex coronary artery (CX) from transesophageal views of heart. The arterial distribution varies among patients. Some segments have variable coronary perfusion. ME, Mid-esophageal; TG, transgastric; SAX, short axis. (Modified from Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr.* 2015;28(1):1–39 e14.)



Fig. 37.14 Qualitative assessment of the aortic valve (zoomed-in transesophageal aortic valve short axis view). (A) There are three valve leaflets of normal thickness. Leaflet excursion appears normal. (B) There are three aortic valve leaflets which are severely thickened. Leaflet excursion is reduced. These qualitative findings should prompt additional quantitative assessment of valvular stenosis. L, Left coronary cusp; LA, left atrium; N, non-coronary cusp; R, right coronary cusp; RA, right atrium.

the presence of valvular regurgitation. The three color Doppler components of a regurgitant jet are proximal flow convergence, vena contracta, and jet area (Figure. 37.15). As blood approaches the regurgitant orifice, it accelerates, forming a series of hemispheric shells of increasing velocity and decreasing area. This flow convergence region provides qualitative information about regurgitation severity and is also utilized in quantitative assessments. The vena contracta is the narrowest portion of a regurgitant jet at or immediately downstream of the regurgitant orifice.^{78,79} The jet area provides information about the mechanism of regurgitation. For example eccentric regurgitation often arises from structural abnormalities while centrally originating regurgitation may be secondary in nature. The size of the jet area is subject to both technical and hemodynamic limitations, so visual estimation of jet area alone is not a reliable means of assessing regurgitation severity.

Atrial and ventricular dilation suggest chronicity of severe regurgitation; normal chamber size would be unusual in the presence of chronic severe regurgitation. Density and shape of the continuous-wave Doppler (CWD) recording of the regurgitant jet provide additional qualitative information.⁷⁹

An important limitation of intraoperative assessment of valvular regurgitation is the hemodynamic consequences of general anesthesia, resulting in reduced preload, afterload, and contractility. General anesthesia commonly reduces the severity of MR. Pharmacologic increase of afterload may provide more representative measurements, but may also lead to significant overestimation of regurgitation severity.⁸⁰

Quantitative Assessment

VENTRICULAR SIZE AND FUNCTION

Left Ventricle

Most indices of systolic function used in routine evaluation are load-dependent, necessitating interpretation in context and serial assessments due to changing intraoperative conditions. Global systolic function is assessed by measuring the difference between an end-diastolic and end-systolic value of a parameter and dividing by the end-diastolic value. Fractional shortening uses length measurements and fractional area change (FAC) uses area measurements. Both approaches have limitations in the setting of regional wall motion abnormalities (RWMA). Ejection fraction (EF) is the difference between the end-diastolic volume (EDV) and end-systolic volume (ESV), divided by EDV:

$$EF = \frac{(EDV - ESV)}{EDV}$$

The recommended method for 2-D calculation of EF is the biplane method of disks (modified Simpson rule).⁸¹ This requires tracing the endocardial border in two perpendicular views and assumes the LV volume consists of a stack of elliptical disks. The upper limit of normal for EDV is 74 mL/m² for males and 61 mL/m² for females; ESV is 31 mL/m² for males and 24 mL/m² for females.⁸¹ Table 37.4 presents updated reference ranges for LVEF.⁸¹ Assessment of EF using 3-D echocardiography (Fig.

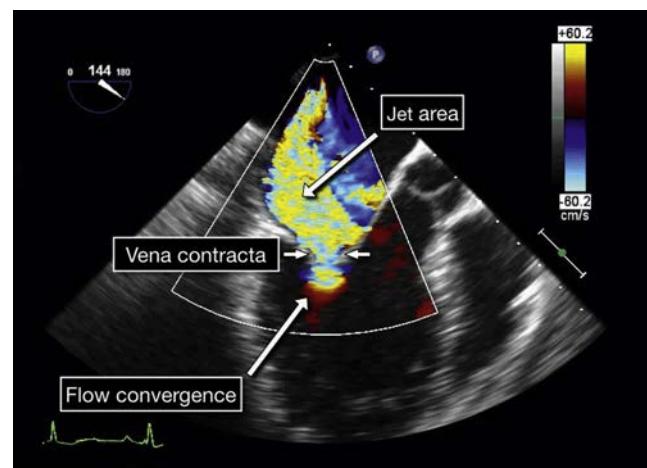


Fig. 37.15 Mitral regurgitation displaying the three components of a regurgitant jet: (1) flow convergence, (2) the vena contracta, and (3) the regurgitant jet area.

TABLE 37.4 Classification of Left Ventricular Systolic Function According to Ejection Fraction

Systolic Function	Male	Female
Normal	52%–72%	54%–74%
Mild dysfunction	41%–51%	41%–53%
Moderate dysfunction	30%–40%	30%–40%
Severe dysfunction	<30%	<30%

From Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;28(1):1–39 e14.

37.16) is more accurate than 2-D echocardiography.⁴¹ In the presence of significant valvular regurgitation, myocardial contractility may be depressed and forward cardiac output low despite a normal EF.

Additional indices of LV systolic function include DTI of the mitral annular systolic velocity (s'), myocardial performance index (MPI), and the change in LV pressure over time (dP/dt). Pulsed-wave DTI of the myocardium at the level of the mitral annulus yields a myocardial velocity profile (Fig. 37.17). The systolic velocity wave (s') reflects the apically-directed motion of the myocardium and correlates with EF. Normal s' reference ranges vary based on sex, age, and mitral annulus measurement size.⁸² MPI assesses both systolic and diastolic performance and is calculated by the sum of isovolumetric contraction and relaxation times divided by ejection time. MPI identifies impaired global function and has prognostic value, however it is not routinely part of the perioperative examination. The change in LV pressure over time (dP/dt) derived from the CWD signal of MR provides information about LV contractility. Normal LV dP/dt is greater than 1200 mm mmHg/s; dP/dt less than 800 mmHg/s is consistent with severe dysfunction.⁸³ Global longitudinal strain is emerging as a technique with utility in identifying systolic dysfunction prior to decrement in EF. Normal values vary across vendor platforms, but in general, a value of –20% (or more negative) would be expected in normal systolic function (Fig. 37.18).

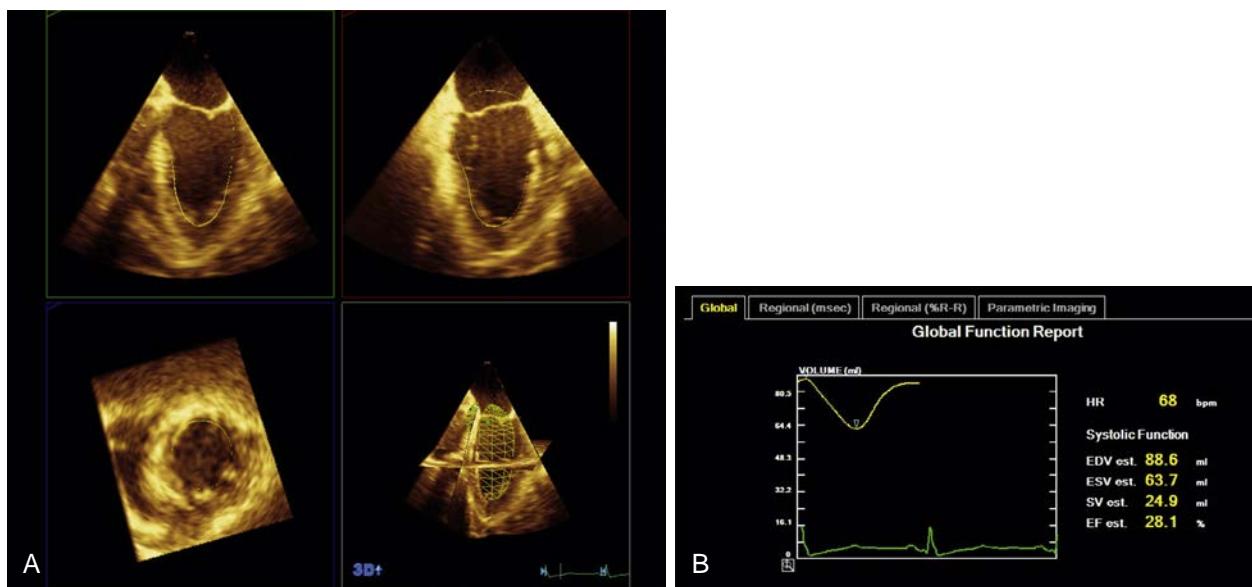


Fig. 37.16 Three-dimensional (3D) transesophageal echocardiographic measurement of left ventricular ejection fraction. (A) A full-volume data set of the left ventricle is displayed using multiplanar reconstruction. After manual tracing of the endocardial borders at end-diastole and end-systole in the planes displayed in the top two panels, a semi-automated endocardial border detection algorithm tracks the borders in the remaining frames. (B) The yellow line displays the left ventricular volume over the duration of the cardiac cycle. Measurements reported include end-diastolic volume, end-systolic volume, stroke volume, and ejection fraction. Segmental changes in volume over the course of the cardiac cycle can also be displayed.

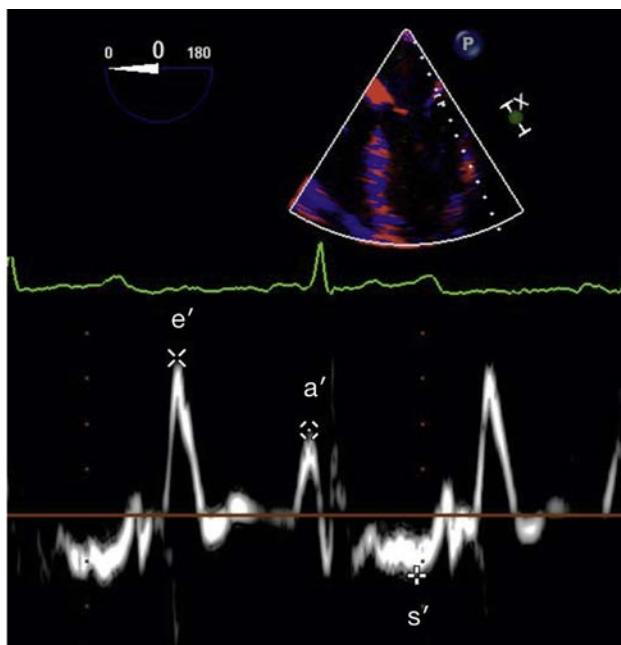


Fig. 37.17 Spectral Doppler tissue imaging obtained at the lateral mitral annulus in the midesophageal four-chamber view. The systolic waveform (s') corresponds to the systolic tissue velocity. The two diastolic waveforms correspond to the early diastolic tissue velocity (e') and the late (atrial) diastolic tissue velocity (a'). When obtained using TEE, s' is a negative waveform (directed away from the transducer), while e' and a' are positive waveforms. Transthoracic acquisition of mitral annular velocity is performed in the apical four-chamber view. In TTE acquisition, the s' waveform is positive, while e' and a' are negative.

Right Ventricle

Parameters for assessment of RV function include FAC, volumetric assessments (EF), tricuspid annular plane systolic excursion (TAPSE), DTI of the tricuspid annular systolic

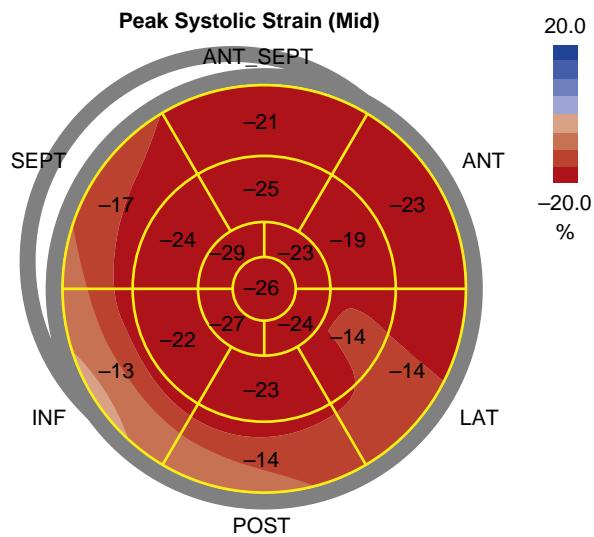


Fig. 37.18 A transthoracic “bulls-eye” plot of speckle tracking-derived global longitudinal strain obtained with transthoracic echocardiography. The image depicts the 17 segments of the left ventricle and the peak segmental systolic strain for each segment. The dark red areas represent normal strain, while the light red and pink areas represent abnormal strain. The average global longitudinal strain in this example is normal (-20.8% , not displayed in the image).

velocity (s'), and MPI. RV FAC is obtained by tracing the endocardial border in diastole and systole, from the annulus along the free wall (excluding trabeculations) to the apex, from the apex to the annulus along the interventricular septum, and then returning to the annular starting point. RV FAC is calculated as:

$$\text{RV FAC} = \frac{\text{RV EDA} - \text{RV ESA}}{\text{RV EDA}} * 100$$

RV FAC is expressed as a percentage and has a lower limit of normal of 35%.⁷⁷ Estimation of RV EF using 2-D echocardiography requires geometric assumptions, and guidelines do not recommend its use.⁷⁷ When expertise is available 3-D RV EF is recommended, with less than 45% usually representing abnormal function.⁸¹

TAPSE is commonly used in TTE for evaluating RV longitudinal function, measured by aligning the M-mode cursor parallel to the tricuspid annular motion in the A4C view.⁸¹ TAPSE less than 17 mm is consistent with RV systolic dysfunction. Parallel alignment of the tricuspid annulus with the M-mode cursor is difficult in TEE, and ME 4C measurements of TAPSE correlate poorly with TTE measurements. Alternative methods for measuring tricuspid annular motion with TEE have been explored. Anatomical M-mode measurements in ME 4C and DTG 4-chamber 0-degree views demonstrate agreement with TTE TAPSE.⁸⁴ TEE speckle tracking of tricuspid annular motion has also been demonstrated to correlate well with TTE TAPSE^{85,86}; whether this will have practical perioperative applications requires further investigation. Reduction in TAPSE following cardiac surgery is well described. Whether reduced TAPSE reflects a true decrement in RV systolic function or rather geometric alteration from pericardiotomy remains controversial.^{87,88}

An s' of less than 9.5 cm/s is consistent with RV dysfunction, but like TAPSE is an index of longitudinal excursion and may be reduced after cardiac surgery. A right ventricular myocardial performance index (MPI) of greater than 0.43 by pulsed-wave Doppler (PWD) or greater than 0.54 by DTI reflects abnormal RV function.⁸¹ RV strain and strain rate are emerging as prognostic parameters in the cardiology literature, but reference values are vendor specific and measurements are not yet routinely part of the perioperative examination.

Diastolic Function

Many complex interactions contribute to ventricular filling during diastole, but the echocardiographer should have a basic understanding of diastolic physiology and the progression of LV filling abnormalities along a continuum of impaired relaxation and decreased compliance. Myocardial relaxation primarily affects LV filling during early diastole (isovolumetric relaxation and early rapid filling), whereas the effects of ventricular compliance predominate in late diastole (diastasis and atrial contraction).

Transmitral PWD flow patterns reflect changes in LV filling and are used to characterize the stages of diastolic dysfunction.

Two waveforms correspond to early rapid filling (E wave) and atrial contraction (A wave), respectively. (Fig. 37.19). The E:A ratio changes with progressive diastolic dysfunction (Fig. 37.20), and traditionally, other parameters such as isovolumetric relaxation time, deceleration time, pulmonary venous flow profiles, and propagation velocities have provided supportive information when grading diastolic dysfunction. Under normal conditions, most ventricular filling occurs during early diastole (resulting from relaxation and suction forces), and the E:A is ratio greater than 1. Grade 1 diastolic dysfunction manifests as *impaired relaxation* with decreased early diastolic filling and a compensatory increase in late diastolic filling (E < A). Relaxation remains impaired in more advanced degrees of diastolic dysfunction. In grade 2 diastolic dysfunction (*pseudonormal pattern*), decreasing LV compliance leads to a rise in LV end-diastolic pressure (LVEDP). Ultimately an increase in left atrial pressure (LAP) increases driving pressures across the mitral valve, increasing early filling, and “normalizing” the E:A ratio. With progression to a *restrictive pattern* (grade 3 dysfunction), markedly decreased LV compliance and significantly elevated LVEDP and LAP result in very elevated peak E-wave velocity (E:A > 2) and rapid equilibration of LV and LAPs.

DTI is an important technique for evaluating diastolic function. DTI of mitral annular myocardial velocities produces two diastolic waveforms: e' (early diastolic tissue velocity) and a' (late diastolic tissue velocity), as shown in Fig. 37.17. Preload conditions have less effect on e' than on the transmitral E wave. DTI e' is reduced when impaired relaxation is present, making

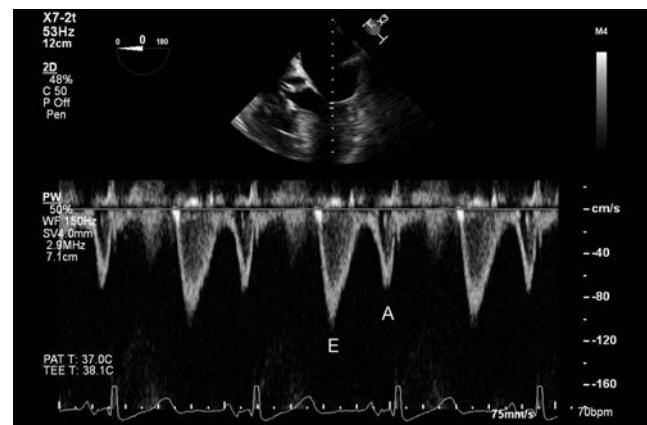


Fig. 37.19 Transmitral pulsed-wave Doppler profile demonstrating the peak early filling (E-wave) and flow due to atrial contraction (A-wave).

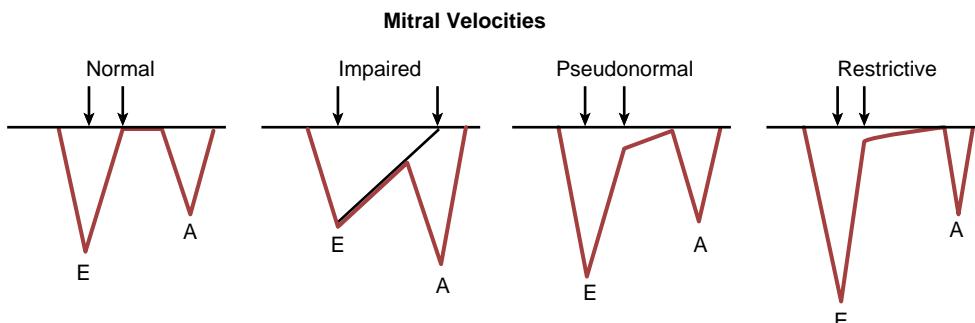


Fig. 37.20 Line drawings representing simultaneous transesophageal pulsed-wave Doppler recordings from the mitral annulus are shown for normal, impaired, pseudonormal, and restrictive left ventricular diastolic function. The black arrows mark the deceleration time of early filling, which is the interval between the peak E velocity and the point where the deceleration slope intersects the baseline. (Modified from Fig. 46.32, Desjardins G. Perioperative echocardiography. In: Miller R, ed. *Miller's Anesthesia*. 8th ed. Philadelphia, PA: Elsevier/Saunders; 2015:1396–1428.)

this a useful parameter for assessing whether diastolic dysfunction is present or absent. Much has been published about site of measurement (septal vs. lateral vs. averaged) and corresponding normal thresholds. A lateral e' velocity of 10 cm/s or greater virtually excludes diastolic dysfunction (pericardial constriction being an exception). The E:e' ratio provides information about LV filling pressures: usually filling pressures are normal when the average E:e' is less than 8, whereas average E:e' greater than 14 usually reflects elevated filling pressures. However, many factors limit the interpretation of e', including mitral stenosis, significant mitral annular calcification, mitral valve prosthesis or ring, left bundle branch block, ventricular pacing, and significant MR.⁸⁹

Updated ASE guidelines address the perceived complexities of diastolic function assessment and propose a simplified approach to evaluation. In patients with preserved EF, four parameters should be assessed initially: e', E:A ratio, left atrial volume index, and peak tricuspid jet annular velocity.⁹⁰ These updates reflect an appreciation that increased LA size serves as a marker of chronically elevated LVEDP. In patients with reduced EF, characterization of diastolic dysfunction begins with transmural E:A ratio. These updated guidelines may not be generalizable to the perioperative period, and moreover, TEE cannot reliably assess LA size. There are several algorithms relevant to the perioperative period that can be referenced for a more in-depth review of diastolic dysfunction.⁹¹⁻⁹³

VALVULAR FUNCTION

Stenosis

Aortic and mitral stenosis are the most common stenotic lesions in the perioperative setting. Fundamental to the quantitative evaluation of valvular stenosis is Doppler echocardiography. Two key concepts underlying Doppler hemodynamic measurements are the continuity principle and the pressure-velocity relationship (see *Hemodynamic measurements* section). Recommended parameters for the assessment of aortic stenosis include peak aortic velocity, mean transvalvular gradient, and valve area calculation by continuity equation (Table 37.5).⁹⁴ The peak gradient derived using echocardiography estimates the instantaneous peak pressure difference across the valve, whereas conventional catheterization data report the difference between peak LV and peak aortic pressures (peak-to-peak gradient). Dimensionless index and 3-D planimetry corroborate findings obtained by recommended methods. Discordant grading of aortic stenosis by valve area and mean gradient requires additional evaluation of LV function, stroke volume (SV), and assessment of flow reserve. Recommended parameters for assessing mitral stenosis include gradient measurements, planimetry, and pressure half-time.⁹⁵ The continuity equation and the proximal isovelocity surface area (PISA) method may be used for additional information.

Limitations exist with quantitative methods. Geometric assumptions made when using the continuity equation may lead to an underestimation of calculated valve area. Loading conditions impact flow, and as a result, impact peak velocities and calculated pressure gradients. Under general anesthesia, gradients may underestimate the severity of stenosis. In high-flow states, elevated gradients may reflect high cardiac output.

TABLE 37.5 Parameters for Evaluating Severity of Aortic Stenosis

Parameter	SEVERITY OF STENOSIS		
	Mild	Moderate	Severe
Peak velocity (m/s)	2.6–2.9	3.0–4.0	≥4.0
Mean gradient (mm Hg)	<20	20–40	≥40
AVA (cm ²)	>1.5	1.0–1.5	<1.0
Indexed AVA (cm ² /m ²)*	>0.85	0.6–0.85	<0.6

*In patients with small body size, aortic valve area (AVA) should be indexed to body surface area. A small valve area in a small patient may only represent moderate stenosis. Indexing to very large body surface area is controversial.

Adapted from: Baumgartner H, Hung J, Bermejo J, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2017;30(4):372–392.

Regurgitation

Recall that the vena contracta is the narrowest portion of a regurgitant jet at or downstream of the regurgitant orifice. The vena contracta width is a semiquantitative parameter for grading regurgitation severity, with cutoff values differing between valves. Pulsed-wave interrogation of flow patterns provides additional semiquantitative information. Pulmonary vein systolic flow reversal is specific for severe mitral regurgitation (MR) and hepatic vein systolic flow reversal is specific for severe tricuspid regurgitation⁷⁸.* Holodiastolic flow reversal in the descending aorta with velocities sustained above 20 cm/s is specific for severe aortic insufficiency.

Quantitative methods for grading regurgitation include calculation of regurgitant volume, regurgitant fraction, and effective regurgitant orifice area (EROA). Approaches include the stroke volume method, ventricular volumetric method, and the proximal isovelocity surface area (PISA) method. The radius of the previously mentioned flow convergence region is included in the PISA calculation. The details of these approaches are beyond the scope of this chapter. 3-D echocardiography allows for measurement of vena contracta area, which may be advantageous in the case of multiple jets or noncircular regurgitant orifices.

HEMODYNAMIC MEASUREMENTS

Cardiac Output

Doppler-based methods can be used to estimate SV and cardiac output (Fig. 37.21). Volumetric flow or SV can be calculated as a cylindrical volume as follows:

$$SV \text{ (cm}^3\text{)} = CSA \text{ (cm}^2\text{)} * VTI \text{ (cm),}$$

where CSA is the cross-sectional area through which the blood flows and VTI is the velocity time integral of blood flow at that location. The VTI represents the distance the average blood cell travels during one cardiac cycle, and is

* in the absence of conditions such as atrial fibrillation, atrioventricular dissociation, or pacing with ventriculoatrial conduction.

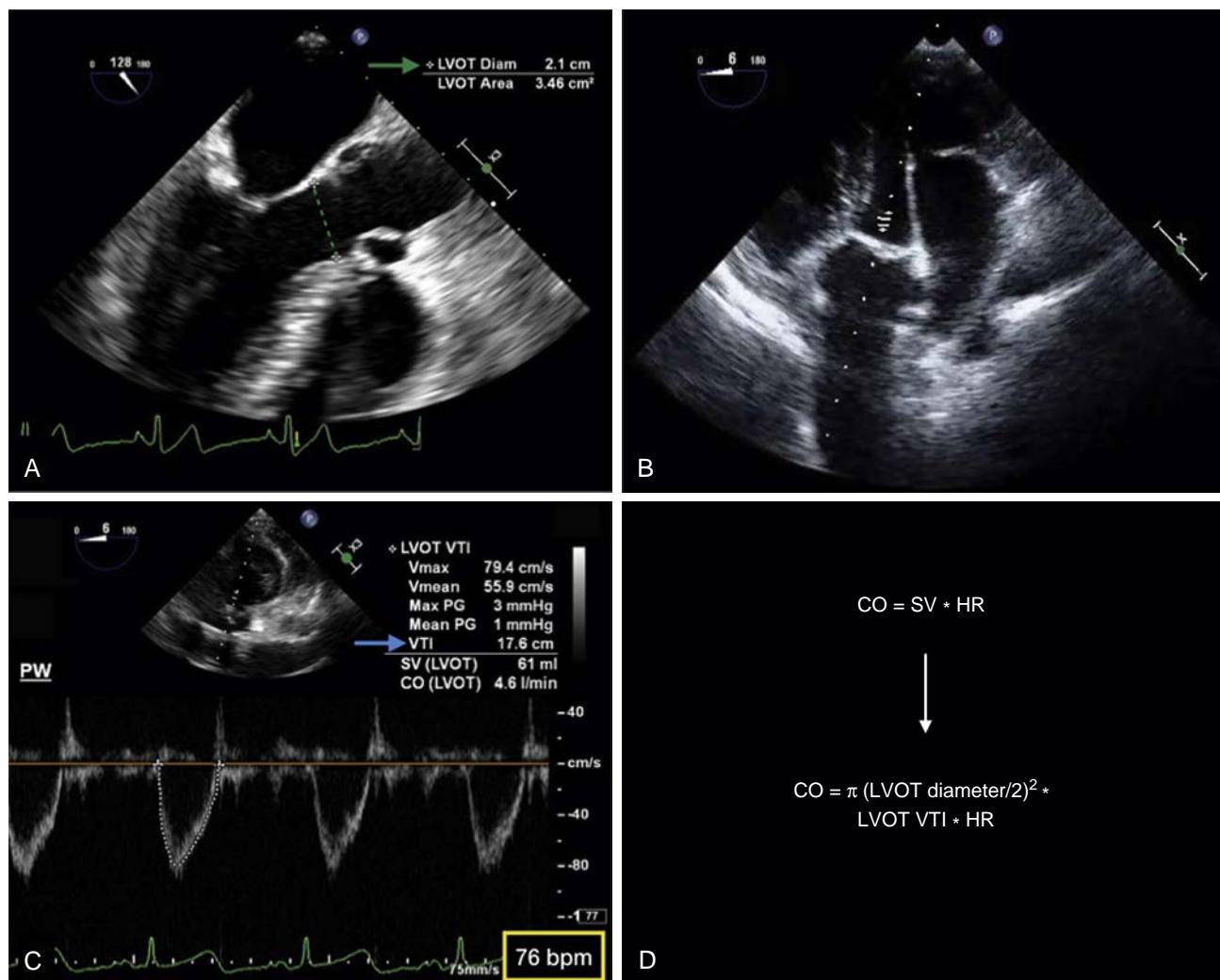


Fig. 37.21 Calculation of cardiac output (CO) using transesophageal echocardiography. (A) The left ventricular outflow tract (LVOT) diameter is measured in the midesophageal aortic valve long axis view. The cross-sectional area (CSA) of the LVOT is assumed to be circular, and is calculated as: $CSA_{LVOT} = \pi \left(\frac{LVOT \text{ Diameter}}{2} \right)^2$. In this example, $CSA_{LVOT} = 3.14 * \left(\frac{2.1 \text{ cm}}{2} \right)^2 = 3.46 \text{ cm}^2$. (B) Velocity of blood flow through the LVOT is measured using pulsed-wave Doppler in the deep transgastric view. The velocity and diameter measurements should be performed at the same anatomic location. (C) The spectral envelope is traced to determine the area under the curve, which is equal to the velocity time integral (VTI). This represents the distance the column of blood travels with each beat and is sometimes referred to as "stroke distance." Stroke volume (SV) is the product of the cross-sectional area of the LVOT and the LVOT VTI, or $SV = CSA_{LVOT} * LVOT \text{ VTI}$. (D) CO is derived by multiplying the stroke volume (SV) by heart rate (HR), or $CO = 3.46 \text{ cm}^2 * 17.6 \text{ cm} * 76 \text{ bpm} = 4,628 \frac{\text{ml}}{\text{min}}$. In the example provided, the machine is configured to automatically calculate stroke volume and cardiac output as displayed in panel C.

the area under the spectral Doppler envelope. Cardiac output is the product of SV and heart rate.

Cardiac output calculation is typically performed at the LV outflow tract (LVOT). The diameter is measured in the ME AV LAX view using TEE and the parasternal LAX view using TTE. The LVOT VTI is obtained in the Deep TG LAX using TEE and the A5C using TTE. Potential sources of error include inaccurate measurement of the LVOT diameter (with a squaring of any error when calculating the cross-sectional area), poor Doppler alignment with the blood flow, and measurement of the area and velocity measurements at different anatomic locations.

Many studies in surgical and ICU patients have compared Doppler-derived cardiac output (CO) to CO by thermodilution. Some authors interpret the literature to indicate Doppler-derived measurements have good agreement with CO by thermodilution.⁹⁶ Other authors, however, highlight the

methodological limitations and heterogeneity of studies, and argue echocardiographic and thermodilution-derived CO cannot be used interchangeably.⁹⁷ Stronger evidence supports the use of echocardiographic-derived CO for assessing trends over time.⁹⁷

Continuity Principle

The continuity principle states that flow rate proximal to a restricted orifice equals the flow rate through the orifice. Applying this principle to pulsatile intracardiac flow, the stroke volumes proximal to (SV_1) and at a restricted orifice (SV_2) are the same.

$$SV_1 = SV_2$$

and therefore

$$CSA_1 * VTI_1 = CSA_2 * VTI_2$$

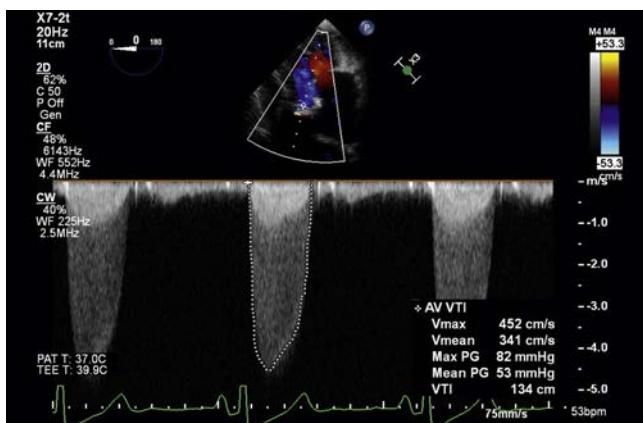


Fig. 37.22 The transvalvular pressure gradient across a stenotic aortic valve is estimated using continuous wave Doppler in the transesophageal deep transgastric view. The peak gradient is derived from the peak velocity of the spectral Doppler signal using the simplified Bernoulli equation. The mean gradient is the average of the instantaneous peak gradients throughout systole and is obtained by tracing the Doppler envelope. The ultrasound system will automatically calculate the mean pressure gradient from the tracing. The measurements in this example are consistent with severe aortic stenosis.

Common clinical applications of this equation include calculation of stenotic valve areas, prosthetic valve areas, and regurgitant orifice areas.

Bernoulli Equation

Pressure cannot be measured directly using echocardiography, however applications of the Bernoulli principle allow for estimation of gradients from velocity information (Fig. 37.22). This technique is commonly applied to quantify the severity of stenosis. The modified Bernoulli equation estimates the transvalvular pressure gradient (ΔP) as follows:

$$\Delta P = 4(V_2^2 - V_1^2)$$

where V_2 represents the peak velocity at the valve and V_1 represents the peak velocity proximal to the valve. V_1 is usually much lower than V_2 and can be ignored, simplifying the equation to:

$$\Delta P = 4V^2$$

If V_1 exceeds 1.5 m/s it should be included in the calculation.⁹⁴

Intracardiac Pressure Estimates

Right Atrial Pressure. In spontaneously breathing individuals, right atrial pressure (RAP) can be estimated from the inferior vena cava (IVC) diameter and collapsibility of the IVC during inspiration ("sniff" maneuver). The IVC collapsibility index (cIVC) is defined as:

$$cIVC = \frac{(D_{\max} - D_{\min})}{D_{\max}} * 100\%$$

where D_{\max} is the maximum diameter on expiration and D_{\min} is the minimum diameter on inspiration. Table 37.6 presents a simplified approach to estimating RAP using IVC diameter and cIVC recommended by Rudski and associates.⁷⁷ In cases of intermediate RAP estimates, it is

TABLE 37.6 Echocardiographic Estimation of Right Atrial Pressures*

RAP	Estimated RAP (Range) (mm Hg)	IVC Diameter (cm)	cIVC
Normal	3 (0–5)	≤2.1	>50%
Intermediate	8 (5–10)	≤2.1	<50%
Intermediate	8 (5–10)	>2.1	>50%
Elevated	15 (10–20)	>2.1	<50%

*In spontaneously breathing individuals.

In the case of intermediate estimates, the presence or absence of secondary indices of elevated RAP can be used to better characterize the pressure estimate. cIVC, IVC collapsibility index (see text for definition); IVC, inferior vena cava; RAP, right atrial pressures.

As proposed by/adapted from Table 3 in Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr.* 2010;23(7):685–713; quiz 786–688.

recommended to use secondary indices of elevated RAP to downgrade or upgrade the estimate.⁷⁷

Chamber Pressures. Velocity measurements of valvular regurgitation or shunt can be used to estimate chamber pressures using the simplified Bernoulli equation. The pressure gradient (ΔP) reflects the pressure difference between the chamber where blood flow originates and blood flow is received. This is expressed as:

$$P_{OC} - P_{RC} = 4V^2$$

where P_{OC} is the pressure in the originating chamber and P_{RC} is the pressure in the receiving chamber. The equation can then be rearranged to:

$$P_{OC} = 4V^2 + P_{RC}$$

A common application is estimation of pulmonary artery systolic pressure (PASP) from the maximal tricuspid regurgitant jet velocity (Fig. 37.23). Pulmonary hypertension is defined as a mean pulmonary arterial pressure (mPAP) of 25 mm Hg or greater, which corresponds to a PASP of 38 mm Hg. mPAP can be estimated from PASP as follows: $mPAP = (0.61 \times PASP) + 2$ mm Hg.⁹⁸ Alternatively, mPAP can be estimated using the simplified Bernoulli equation and the peak velocity of pulmonary regurgitation.

Hemodynamic Failure and Shock

Hemodynamic instability and circulatory failure may occur at any phase in a patient's perioperative care, and echocardiography provides valuable information to the operating room anesthesiologist and intensivist alike. Echocardiography enables characterization of the mechanism of shock (cardiogenic, hypovolemic, distributive, and/or obstructive)⁹⁹ and can be used for serial assessment of response to therapies.¹⁰⁰ Most clinicians will apply qualitative techniques during echocardiographic assessment; quantitative approaches also add valuable information when used by appropriately trained individuals.

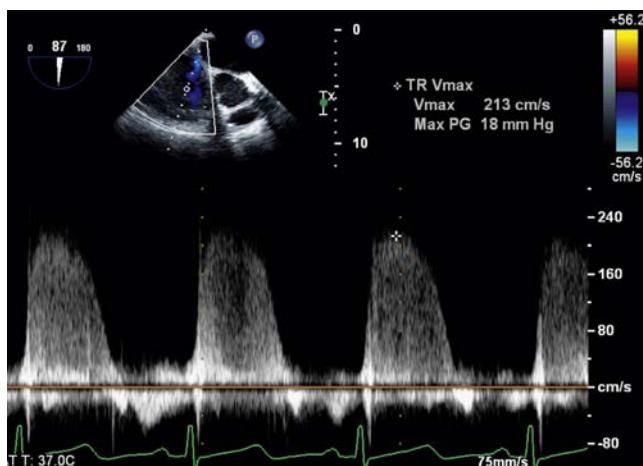


Fig. 37.23 A midesophageal modified bicaval view aligns the Doppler cursor with the tricuspid regurgitant jet. Continuous wave Doppler measures the peak systolic velocity of the tricuspid regurgitation (V_{TR}). V_{TR} reflects difference between the right ventricular systolic pressure (RVSP) and right atrial pressure (RAP), so $RVSP = 4 V_{TR}^2 + RAP$. In the absence of right ventricular outflow tract obstruction or pulmonary stenosis, RVSP and pulmonary artery systolic pressure (PASP) are essentially equal. In this example, the estimated PASP = 18 mm Hg + RAP.

VENTRICULAR DYSFUNCTION

Qualitative evaluation of biventricular systolic function is central to identifying the etiology of shock or hemodynamic instability. As previously mentioned, visual estimation of LVEF by experienced individuals correlates well with quantitative methods,^{71,72} and can be learned easily with proper feedback.¹⁰¹ A focused examination should identify whether significant myocardial dysfunction is present. Further assessment may suggest myocardial stunning or ischemia, especially if in a segmental distribution or acute in onset. This is not without limitations however, as acute decreases in preload can cause new RWMA in the absence of ischemia.¹⁰²

Takotsubo cardiomyopathy, a form of stress cardiomyopathy thought to result from excess catecholamine stimulation, may occur with a perioperative incidence of 1 in 6700.¹⁰³ Classically described as apical ballooning and basal hyperkinesis in the absence of obstructive coronary disease (Video 37.6), it derives its name from the similarly appearing Japanese takotsubo octopus pot trap. Additional patterns of regional involvement in stress cardiomyopathy include midventricular, basal, localized, or global.¹⁰⁴ Echocardiography allows evaluation of LV morphology and segmental wall motion, right ventricular involvement, and complications such as MR, LVOT obstruction, intracardiac thrombus, and cardiac rupture.¹⁰⁴

SEVERE HYPOVOLEMIA

Hypovolemia leads to a decrease in LV end-diastolic area (EDA) and end-systolic area (ESA), but at baseline there is wide variability in the normal ranges.¹⁰⁵ For practical purposes, qualitative assessment of chamber size provides the most useful information if monitored serially in the operating room or in the setting of significant hypovolemia, where

EDA will be markedly reduced. It is important to distinguish hypovolemia from distributive shock, where ESA is also decreased but the EDA area will be normal.¹⁰⁶ End-systolic cavity obliteration (sometimes described as “kissing walls”) is frequently associated with decreased EDA and hypovolemia, but may also occur in high inotropic or vasodilatory states.¹⁰⁵

As previously described, IVC diameter and cIVC can be used to estimate the RAP in spontaneously breathing individuals.⁷⁷ Central venous pressure, however, is a poor predictor of fluid responsiveness.¹⁰⁷ Whether cIVC predicts fluid responsiveness during spontaneous ventilation is an area of active debate,^{108–111} and has been examined in several studies.^{112–117} In many instances, spontaneously breathing patients with cIVC of more than 40% to 50% will respond to fluid administration,^{112,115,117} but this is not found consistently,^{113,114} and does not necessarily identify nonresponders.^{112,115} We would encourage the reader to read the primary literature to gain a more in-depth understanding of the topic prior to attempting to use cIVC in the assessment of hypovolemia.

Predictors of fluid responsiveness applicable in patients who undergo passive mechanical ventilation, typically in the setting of critical illness, are covered in the *Critical Care* section of the chapter.

LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION

LVOT obstruction may precipitate significant hemodynamic compromise, creating a perplexing scenario of worsening instability despite aggressive inotropic resuscitation. LVOT obstruction and systolic anterior motion (SAM) of the mitral valve are classically associated with hypertrophic cardiomyopathy. In the perioperative setting, LVOT obstruction and SAM often occur following mitral valve repair. The presence of LVOT obstruction in other susceptible populations is likely underappreciated, and probably results from a combination of anatomic predisposition and precipitating hemodynamic factors associated with enhanced LV contractility (hypovolemia, hyperkinesis, inotropes). Anatomic factors include small LV cavity, basal septal hypertrophy, and an anteriorly-displaced mitral valve coaptation zone with redundant leaflet tissue.¹¹⁸ One series observed LVOT obstruction in over 20% of patients admitted in septic shock, and the presence of LVOT obstruction was associated with higher mortality.¹¹⁹ SAM was present in only two-thirds of the cases of LVOT obstruction, underscoring the complex mechanisms leading to clinically relevant obstruction. In a series of perioperative rescue echocardiograms, LVOT obstruction was present in 3.6% of cases.¹²⁰

LVOT obstruction produces high-velocity flow in the LVOT. Interrogation of the LVOT with CFD reveals flow acceleration and turbulence. Blood flow velocity exceeds the Nyquist limit of PWD, so CWD is required for quantification. Visual assessment of CFD and gradual movement of the PW sampling volume through the LV cavity and LVOT can be used to estimate the location of the obstruction. In the presence of LVOT obstruction, the spectral Doppler profile of systolic blood flow across the LVOT is dagger-shaped and mid-to-late peaking (Fig. 37.24). The

M-mode of the aortic valve will demonstrate premature leaflet closure during mid-systole (Fig. 37.25B). Findings consistent with SAM include anterior displacement of the mitral leaflet during systole with entrainment in the LVOT (Fig. 37.26; Video 37.7). Variable degrees of MR may be present.

CARDIAC TAMPONADE

Echocardiography readily detects pericardial fluid, which appears as an echolucent space adjacent to the heart. The physiologic significance of a pericardial effusion depends on both the volume and rate of accumulation. A large effusion may develop slowly with minimal consequence, whereas a rapidly accumulating effusion of small volume can have

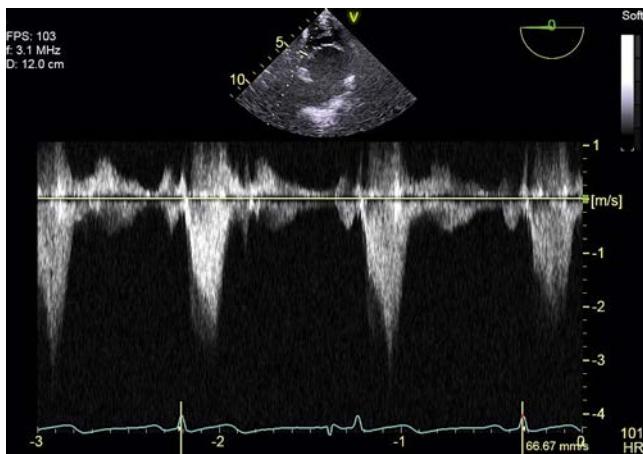


Fig. 37.24 A continuous wave Doppler signal characteristic of left ventricular outflow tract obstruction. The signal appears “dagger-shaped,” with the peak velocity occurring in mid-to-late systole. In the perioperative setting, obstruction may be dynamic and unmasked by precipitating factors in susceptible individuals.

profound hemodynamic impact. Cardiac tamponade develops when intrapericardial pressure exceeds cardiac chamber pressures, thereby compressing the cardiac chambers. This impairs chamber filling ultimately compromising cardiac output. Hemodynamic effects of tamponade occur along a continuum.¹²¹

Echocardiographic findings can support the diagnosis of cardiac tamponade in the context of relevant clinical findings. When intrapericardial pressure exceeds cardiac chamber pressures, chamber collapse may occur during the respective relaxation phases. The thin right atrium typically has the lowest pressure and usually is the first chamber to show collapse or inversion, which is observed in ventricular systole (Fig. 37.27A; Video 37.8).^{122,123} Importantly, transient right atrial collapse may be present in the absence of tamponade, and therefore is a nonspecific finding. A longer duration of atrial collapse improves the specificity of this finding.¹²² RV diastolic collapse (Video 37.9; see Fig. 37.27B) occurs later than right atrial collapse in the progression of tamponade, and is a more specific finding. In a series of patients with moderate or large pericardial effusions, the absence of any right-sided chamber collapse had a high negative predictive value for clinical tamponade.¹²⁴ Collapse of right heart chambers may not occur in settings where baseline right-sided pressures are elevated, such as in patients with pulmonary hypertension. Following cardiac surgery, echocardiographic findings in the setting of tamponade may be atypical, including localized compression of cardiac chambers and accumulation of clot.

In spontaneously breathing patients, a dilated IVC with less than a 50% decrease in diameter with deep inspiration is a sensitive sign for tamponade. This combination of findings has poor specificity, however, because it reflects elevated central venous pressure.¹²⁵ During spontaneous ventilation in the absence of pathology, there is a normal

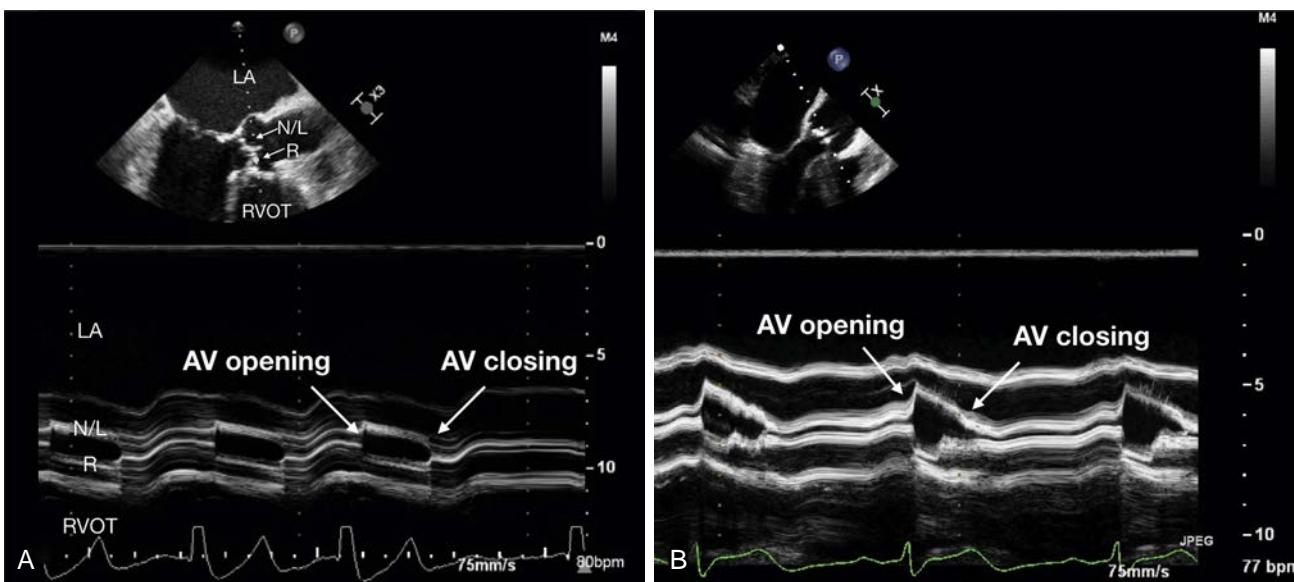


Fig. 37.25 M-mode interrogation of the aortic valve in the midesophageal aortic valve long axis view. The cursor passes through the left atrium (LA), posterior aortic root, the non or left coronary cusp (N/L), the right coronary cusp (R), the anterior aortic root, and the right ventricular outflow tract (RVOT). (A) In the absence of obstruction to flow (or other aortic valve pathology), the motion of the aortic valve leaflets during systole creates a rectangular shape. (B) In the presence of left ventricular outflow tract obstruction, the aortic valve opens normally, but closes prematurely.

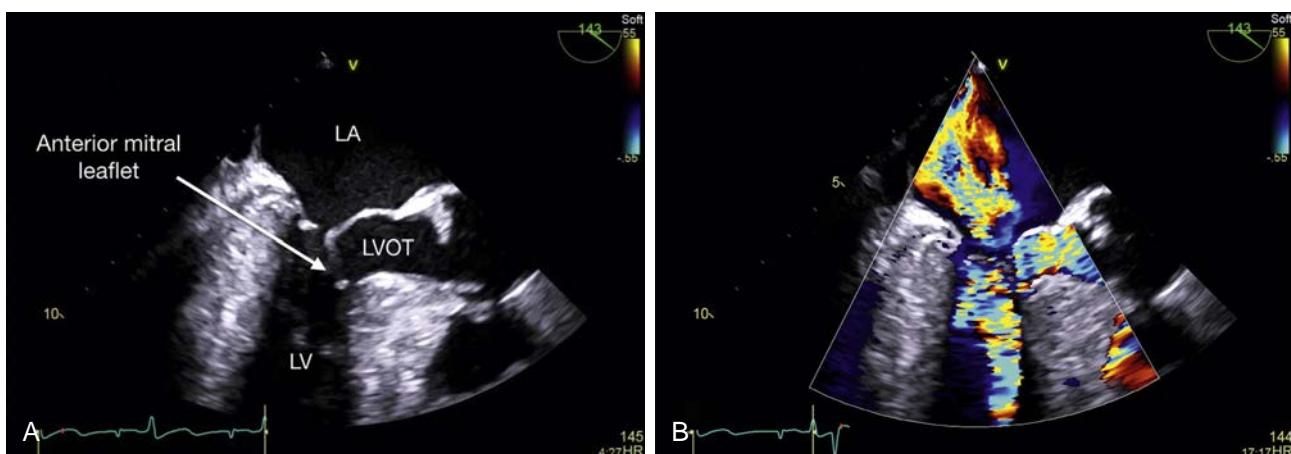


Fig. 37.26 Midesophageal long axis views without (A) and with color (B) demonstrating systolic anterior motion of the mitral valve (SAM). (A) Instead of coapting normally, the anterior mitral leaflet (arrow) moves into the left ventricular outflow tract (LVOT) during systole. This narrows the effective outflow tract and can lead to dynamic obstruction to flow. (B) Turbulent flow is visualized by color Doppler in the LVOT. In this example, there is significant mitral regurgitation associated with SAM. LA, Left atrium; LV, left ventricle.

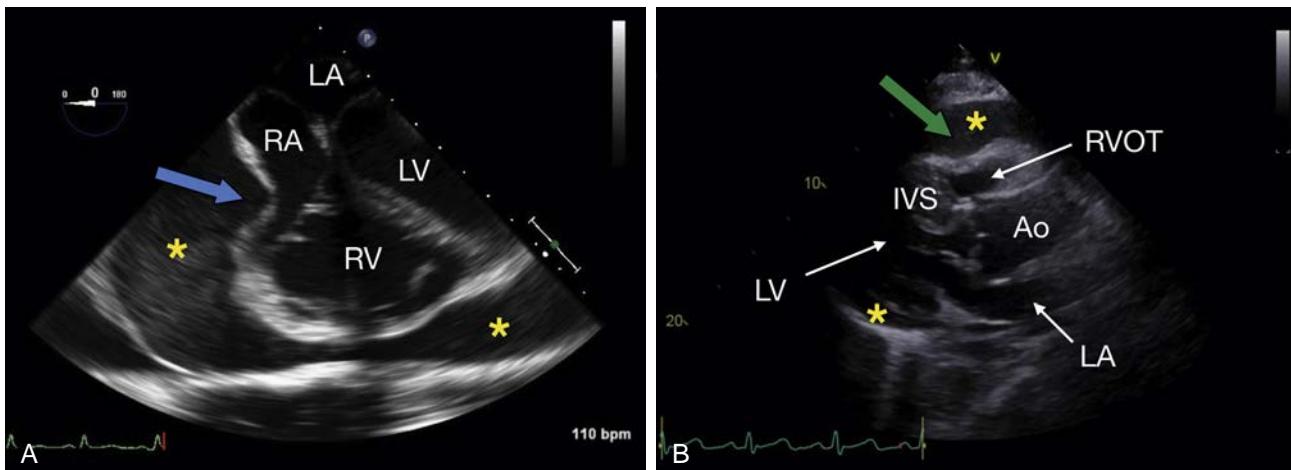


Fig. 37.27 Echocardiographic features supporting the clinical diagnosis of tamponade. (A) Transesophageal midesophageal four-chamber view demonstrating a large echoluent pericardial effusion (*). Right atrial collapse (blue arrow) is present during ventricular systole (note the red marker on the ECG tracing after the R-wave). (B) Transthoracic parasternal long axis view also demonstrating a large pericardial effusion (*). Right ventricular collapse (green arrow) during diastole is present (note the red marker on the p-wave of the ECG tracing). Right ventricular collapse is more specific for tamponade than right atrial collapse. Ao, Ascending aorta; IVS, interventricular septum; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

variation in the peak transvalvular flow velocities, with tricuspid velocities increasing during inspiration and mitral velocities decreasing. Exaggerated respiratory variation in transvalvular flow velocities occurs in tamponade, calculated as a percentage change from expiration velocity.¹²¹ In tamponade, there is usually a greater than 30% variation in mitral inflow velocities, and greater than 60% (absolute) variation in tricuspid inflow velocities. Exaggerated respirophasic changes in transvalvular velocities alone are insufficient to support a diagnosis of tamponade. Additionally, these findings have not been validated in patients receiving positive pressure ventilation. In fact, in an experimental animal model of tamponade, the greatest variation in mitral inflow velocities was observed during the control phase, with attenuation of the variation during

tamponade.¹²⁶ Abnormal venous flow patterns in the superior vena cava (SVC) and hepatic veins can also provide supportive evidence of tamponade; however, analysis may not be feasible in at least one-third of patients due to the presence of TR, atrial fibrillation, paced rhythm, or technical inadequacy.¹²⁴ Echocardiography frequently guides pericardiocentesis procedures.

PULMONARY EMBOLISM

Acute pulmonary embolism (PE) is classified into three subgroups: high-risk (massive), intermediate-risk (submassive), and low-risk.^{127,128} The presence of sustained hypotension or shock (not explained by other causes) defines massive PE. Patients with intermediate- and low-risk PE by

definition are normotensive. The groups are further delineated according to RV dysfunction, myocardial injury, and other clinical features. When PE is suspected, the recommended first-line imaging modality is computed tomography angiography (CTA).¹²⁸ Echocardiography, in contrast, is not recommended in the initial diagnostic evaluation of PE.¹²⁹ When the diagnosis of PE is established, echocardiography has utility in risk stratification, prognostication, and response to therapy.^{128,130}

In cases of suspected PE with hemodynamic instability or shock, obtaining a CTA may not be feasible. In this context, echocardiography has a role in refining the diagnostic assessment and management decisions.^{128,131} Freely mobile intracardiac thrombus, or “clot in transit,” is visualized rarely on echocardiography (<4% of cases), but more commonly in high-risk PE (22%) and is associated with worse prognosis.¹³² Indirect signs of PE include RV dilation, RV dysfunction, and the 60/60 sign, which is a pulmonary ejection acceleration time of less than 60 ms with a mid-systolic notch and a TR peak gradient between 30 and 60 mm Hg.¹³⁰ Several parameters may be used to assess RV dysfunction, including “McConnell’s sign,” TAPSE, increased end-diastolic RV-to-LV diameter ratio (≥ 0.9 or 1.0), and interventricular septal flattening during systole, indicative of pressure overload.^{128,130} Myocardial deformation as a marker of RV dysfunction is not widely used but is an area of investigation.^{130,133}

“McConnell’s sign” is an echocardiographic finding of RV hypokinesis or akinesis with apical sparing (Video 37.10). It was originally reported as having a high sensitivity and specificity for acute PE,¹³⁴ however several subsequent studies have demonstrated this finding to lack sensitivity.¹³⁵ The absence of McConnell’s sign does not exclude acute PE. In many populations, McConnell’s sign is highly specific. Important exceptions include patients with RV infarction¹³⁶ or pulmonary hypertension,¹³³ where McConnell’s sign may be observed in the absence of acute PE (i.e., a nonspecific finding).

Few studies focus specifically on echocardiographic findings in the high-risk subgroup. However, in a series of 511 patients with confirmed PE, 16 of the patients met high-risk criteria.¹³⁷ Interestingly, all 16 patients had RV enlargement and hypokinesis, and at least one of the following: McConnell’s sign, 60/60 sign, or right heart thrombus. Flattened interventricular system, RV free wall hypokinesis plus a RV-to-LV diameter ratio greater than 0.9, McConnell’s sign, and right heart thrombus were observed significantly more frequently in patients with high-risk PE than those without.

Recent work has sought to better define the diagnostic utility of echocardiography in PE (including all risk categories), especially in light of the increasing use of point-of-care ultrasound. Meta-analysis of both formal and point-of-care TTE examinations in patients with clinical concern for PE found that echocardiographic signs of right heart strain (variably defined) had a sensitivity of 53% and a specificity of 83%.¹³⁵ In the previously mentioned series of 511 patients, over one-third of the non-high-risk patients demonstrated completely normal RV morphology and function.¹³⁷

Intraoperative Transesophageal Echocardiography - Noncardiac Surgery

In noncardiac surgery, TEE serves two main roles: an intraoperative monitor and a rescue tool. TEE has been used in diverse operative settings including vascular, orthopedic, and transplant surgery. A comprehensive review on the topic has been published by Mahmood and colleagues.¹³⁸ The impact of TEE tends to be greater when it is used as a rescue tool versus as a monitor.¹³⁹ The extent of the therapeutic impact is difficult to quantify, however, in part because interpretation of “impact” is not uniform. Supportive or confirmatory information adds substantial value in many contexts. Therapeutic modifications reported as a result of TEE findings include initiation of pharmacologic therapy, fluid administration, and alteration in anesthetic plan or surgical procedure.^{138,140,141} Continuation of patient management without changes may also be considered a relevant impact.

MONITORING ROLE

Myocardial Ischemia

Echocardiographic changes of myocardial ischemia manifest earlier than ECG changes, suggesting a potential role for TEE in the early detection of intraoperative ischemia. Current practice is to perform TEE monitoring selectively in patients with known risk factors or undergoing high-risk procedures. Early research demonstrated a 20% incidence of new or worsening RWMA in high-risk patients.¹⁴² Intraoperative RWMA were infrequently associated with postoperative myocardial infarction (MI) and overall complication rate was low. Another study in the 1990s concluded that changes detected by intraoperative TEE added little incremental value in predicting risk of postoperative ischemic outcomes (death, nonfatal MI, unstable angina) compared with preoperative data and two-lead ECG monitoring.¹⁴³ The imaging capabilities of current transducers are vastly enhanced compared to those used in earlier decades, and may not reflect TEE’s utility as a monitor of ischemia in the current era. A study of 54 high-risk patients undergoing vascular surgery demonstrated new RWMA on intraoperative TEE in 43% of cases.¹⁴⁴ Overall incidence of postoperative MI was 11%, and all patients with MI demonstrated intraoperative RWMA. Intraoperative TEE was a sensitive indicator of risk for postoperative MI, although there still were a substantial number of patients with “false positive” echocardiographic changes. Many questions surrounding the optimal use and potential impact of TEE as a monitor for ischemia in high-risk settings remain unanswered. In the absence of patient-specific or procedural risk factors, the routine use of TEE during noncardiac surgery to monitor for myocardial ischemia is not recommended.⁵¹

Liver Transplantation

Hemodynamic management during orthotopic liver transplantation is complex, with frequent periods of instability from potentially numerous causes.^{145,146} At baseline, patients often have high cardiac output and low systemic

vascular resistance with significant comorbidities. In the preanhepatic phase (from incision to occlusion of vascular inflow to the liver), hemodynamic alterations may result from an abrupt change in preload due to drainage of large volume ascites, hemorrhage, or surgical caval compression. TEE examination during this phase allows for characterization of dynamic changes, as well as baseline evaluation of biventricular and valvular function. During the anhepatic phase (occlusion of inflow to the liver to unclamping of the portal vein), preload to the heart is decreased. If an end-to-end interposition of the IVC is performed, complete caval occlusion is required and venovenous bypass may be used to improve preload and decrease venous congestion. A piggyback technique only requires partial caval occlusion and venous return to the heart is usually adequate without bypass. TEE findings of ventricular dysfunction, diminished cardiac output, hypovolemia, and intracardiac thrombi may be present regardless of the surgical technique. The reperfusion phase begins with release of the cross clamp from the portal vein, resulting in infusion of cold, hyperkalemic, acidotic blood. Profound instability may result and even progress to cardiac arrest. TEE provides real-time visualization of cardiac function and filling. Over 40% of liver transplant patients demonstrate evidence of inducible LVOT obstruction on preoperative dobutamine stress echocardiography,¹⁴⁷ making this an important diagnosis to exclude in the setting of refractory hypotension. Inspection of the IVC for high-velocity or turbulent flow is also possible with TEE.

Esophageal varices are a relative contraindication to TEE and are a relevant concern in the liver transplant population. However, the reported rate of TEE complications in liver transplantation is low, even in patients with known esophageal varices or history of upper gastrointestinal bleeding. A recent review suggests TEE to be safe in patients without grade 3 varices or active gastrointestinal bleeding.¹⁴⁶ Approaches to minimize risk include limited probe manipulation during the anhepatic phase and avoidance of TG and DTG views.

Lung Transplantation

Intraoperative TEE provides key information in each phase of lung transplantation in a patient population with impaired cardiopulmonary reserve.¹⁴⁸ Initial assessment includes evaluation for RV dilation, hypertrophy, and dysfunction, which result from long-standing pulmonary hypertension. Identification of a patent foramen ovale (PFO) may have clinical consequence if right-sided pressures exceed left-sided pressures, impacting the surgical plan. Common problems during one-lung ventilation and graft implantation include hypoxemia, hypercapnia, and elevated pulmonary vascular resistance, which may precipitate acute RV failure. Recognition of deteriorating RV function by TEE prompts initiation of inotropes or pulmonary vasodilators. Should profound instability result, TEE may provide information leading to initiation of cardiopulmonary bypass or help guide cannula placement for extracorporeal membrane oxygenation (ECMO). After reperfusion, in addition to continued assessment of biventricular function, attention turns to the pulmonary artery anastomosis and the pulmonary veins¹⁴⁹ for findings suggestive of kinking, thrombus, or stenosis.

Vascular Surgery/Endovascular Procedures

Patients undergoing vascular procedures are at increased risk of perioperative cardiovascular morbidity and mortality. Applications of TEE in this setting include monitoring for ischemia, assessment of systolic and diastolic function, and guidance for fluid resuscitation. In open abdominal aortic aneurysm repair, significant increases in afterload and wall tension occur with application of the cross clamp. After aortic occlusion, LVEF significantly decreases and EDV and ESV increase.¹⁵⁰ New or worsening RWMA^s have been reported in up to one-third of patients, and occur more frequently with suprarenal versus infrarenal clamping.¹⁵¹ Persistent RWMA^s after several hours have been associated with postoperative MI.¹⁵¹ With unclamping of vessels and ensuing instability, TEE serves as an adjunctive monitor as resuscitation proceeds. Over the past two decades, there has been a dramatic increase in endovascular repairs and a decline in open vascular procedures, consequently impacting the anticipated anesthetic concerns. Potential profound instability may be dramatically reduced, diminishing the value of intraoperative TEE in that context. However, TEE may be used to identify aortic pathology, identify landing zones, and evaluate for endoleaks.¹³⁸

RESCUE ROLE

TEE used in the setting of unanticipated hemodynamic instability or cardiopulmonary arrest, also called “rescue” TEE, rapidly provides valuable information. Rescue TEE identifies causes of shock described earlier in the chapter (e.g., ventricular dysfunction, severe hypovolemia, LVOT obstruction, tamponade, and PE). Embolic phenomena relatively unique to the operative setting include air emboli during upright neurosurgical procedures and fat or cement emboli in orthopedic and spine surgeries.

Several series report the value of rescue echocardiography, describing its ability to identify a cause for instability or confirm expected diagnoses.¹⁵²⁻¹⁵⁴ TEE aided or impacted clinical management in the majority of the cases. Rescue TEE commonly implicates LV dysfunction and hypovolemia in instability, although no diagnosis occurs with enough frequency to be predictive without performing an echocardiographic examination. In fact, in one series the most frequent finding was a normal examination or demonstration of known pathology (48%).¹⁵⁵ Despite a “negative” result, the TEE findings influenced management in more than half of these cases, suggesting the impact of TEE is not confined to cases demonstrating pathology. The largest reported study includes 364 rescue studies (96% TEE) performed throughout the perioperative period (55% intraoperatively).¹²⁰ The most common findings intraoperatively were hypovolemia (32%) and LV dysfunction (11%), whereas postoperatively RV (24%) and LV (22%) systolic dysfunction predominated. The findings influenced management in 59% of cases. Information can be obtained quickly (<5 minutes)¹⁵⁴ and may make an immediate impact. The sequence of examination and included views may vary among providers, but these should provide information swiftly. One proposed rescue protocol includes a sequence of five views: ME 4C, ME AV LAX, ME bicaval, TG SAX, and descending aorta SAX.¹⁵⁵

Intraoperative Transesophageal Echocardiography - Cardiac Surgery

Numerous observational studies demonstrate the impact of intraoperative TEE on decision making in cardiac surgery. In a retrospective study of 12,566 patients, new echocardiographic findings on TEE prior to cardiopulmonary bypass (CPB) led to changes in surgical management in 7% of cases.¹⁵⁶ After CPB, TEE findings prompted return to CPB in 2.2% of cases. When analyzed according to surgical procedure, influential new findings were demonstrated in 5.4% of 3853 isolated CABG cases pre-CPB, and in 1.5% of CABG cases post-CPB with 0.8% requiring graft revision. A study of 521 patients undergoing CABG demonstrated new pre-CPB findings influencing the surgical plan in 11.9% of cases, and in 0.7% of cases post-CPB.¹⁵⁷ In valvular surgery, an analysis of 8 studies including 15,540 patients demonstrated the influence of TEE findings on surgical decision making in 11% of cases pre-CPB and 4% of cases post-CPB.⁶⁴

When considering the impact of TEE findings on surgical interventions, limitations should be recognized. Selection bias for the use of TEE may be present in cases where it is anticipated to alter decision making, especially in CABG. Also, many studies included PFO as a diagnosis influencing the surgical plan. In the absence of high risk for hypoxemia and right-to-left shunting, optimal management of PFO when the surgical procedure does not involve atriotomy is unclear.¹⁵⁸ In an observational study of patients undergoing cardiac surgery, newly diagnosed PFO was not associated with increased perioperative morbidity or mortality.¹⁵⁹ PFO closure did not influence long-term survival, but was associated with higher risk of intraoperative stroke.

Intraoperative TEE findings provide prognostic value. Detection of RWMA by TEE at any time during CABG is an independent predictor of postoperative MI.¹⁶⁰ Deterioration of regional wall motion on intraoperative TEE following revascularization is associated with higher risk of both short-term¹⁶¹ and long-term¹⁶² adverse cardiovascular events.

A general framework is useful when conducting pre-CPB and post-CPB examinations. Prior to initiation of CPB, evaluation includes assessment of overall biventricular function, aortic pathology that may affect cannulation strategy, presence of aortic regurgitation, and identification of unanticipated findings (e.g., severe valvular lesions, intracardiac thrombus). In the case of valvular surgery, diagnosis is confirmed and additional information regarding the mechanism of dysfunction is communicated to the surgeon. In minimally invasive cardiac surgery, TEE enables visualization of wires for cannulae placement and can guide percutaneous coronary sinus catheter placement. Post-CPB examination includes assessment of global and regional ventricular function, appropriate seating and function of newly implanted prosthetic valves,¹⁶³ identification of any new valvular abnormalities requiring intervention, and exclusion of iatrogenic aortic injury. Applications of TEE in the context of specific pathology and surgical procedures are outlined in the sections that follow.

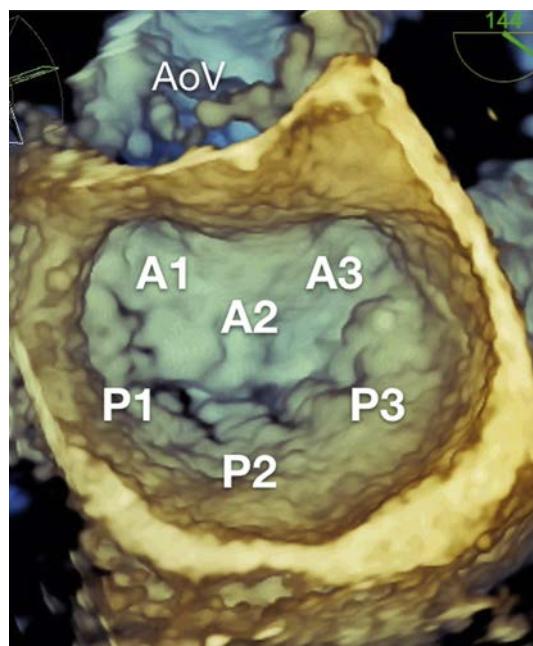


Fig. 37.28 En face display of a three-dimensional image of the mitral valve. This orientation is analogous to the viewpoint of the surgeon, and facilitates communication regarding the location of structural pathology. The scallops of the anterior (A1, A2, A3) and posterior (P1, P2, P3) mitral leaflets are labeled. AoV, Aortic valve.

MITRAL VALVE REPAIR

TEE imaging of the mitral valve has transformed surgical planning, particularly when assessing the suitability of attempted repair versus valve replacement for MR. Assessment of the regurgitant valve begins with examination of the structure of the valve leaflets and valvular apparatus. Primary (degenerative) MR involves abnormality of the leaflets or valvular apparatus, whereas in secondary (functional) regurgitation, the valve is structurally normal with insufficient coaptation resulting from ventricular remodeling. The mechanism of MR is classified according to Carpentier's classification of leaflet motion: type I-normal leaflet motion, type II-excessive leaflet motion, and type III-restricted leaflet motion (IIIa-restricted in systole and diastole, while IIIb is restricted in systole).¹⁶⁴ Quantitative assessment is performed, recognizing that intraoperative conditions often lead to underestimation of the severity of regurgitation.⁸⁰ 3-D TEE offers enhanced spatial resolution and superior accuracy in localizing leaflet pathology compared to 2-D TEE.¹⁶⁵⁻¹⁶⁷ "En face" display of 3-D images analogous to the surgeon's intraoperative view facilitates communication regarding structural details (Fig. 37.28). The origin of the regurgitation can be precisely identified using multiplanar reformatting 3-D technology. Vena contracta area, regurgitant volume, and EROA can be quantified using 3-D techniques with potentially improved accuracy. At present, 3-D quantitative analyses are labor-intensive and time consuming, making them largely impractical for routine intraoperative assessments.

Valve repair is often feasible in degenerative disease with isolated prolapse or flail, and moderate or less annular dilation.⁷⁹ Bileaflet disease (Barlow disease), severe annular dilation, rheumatic deformities, and severe mitral annular

calcification are predictors of unsuccessful repair.¹⁶⁸ Regurgitation recurs in a substantial proportion of patients with ischemic MR following repair. LV remodeling and leaflet tethering with apical displacement of the coaptation point impact the feasibility of repair of ischemic MR. Quantitative measurements include assessment of the tenting height (the perpendicular distance from the plane of the mitral annulus to the point of leaflet coaptation) and systolic tenting area (area enclosed by the mitral annular plane and closed leaflets). Tenting height of 11 mm or greater is associated with recurrent MR. On intraoperative TEE, mitral diastolic annular diameter of 37 mm or greater, tenting area of 1.6 cm² or greater, and severe MR are associated with high probability of repair failure.¹⁶⁸

Additional considerations in the pre-bypass echocardiographic evaluation include predictors of post-repair SAM of the anterior mitral leaflet, which commonly reflect anterior displacement of the coaptation point. This may take the form of reduction in distance between the coaptation point and the septum (C-sept distance < 2.5 cm) or a relatively elongated posterior leaflet (anterior to posterior leaflet ratio of ≤ 1.3 or posterior leaflet height > 1.5 cm), and narrow aorto-mitral angle.^{169,170} Other SAM predictors include basal septal hypertrophy (>15 mm), small LV cavity, and anterior displacement of the papillary muscles.

Immediately following separation from CPB, echocardiography assesses for significant regurgitation, stenosis, or dynamic LVOT obstruction due to SAM. Stability of the ring and normal leaflet motion should be demonstrated. Any residual regurgitation should be no more than mild and transvalvular. Should SAM occur, management steps include administration of fluid and discontinuation of any inotropes. If SAM persists, the next therapeutic maneuvers are administration of beta blockers and increase in afterload. The effect of these interventions can be readily observed on TEE. SAM will improve or resolve in most patients, and long-term follow-up demonstrates low incidence of SAM, favoring conservative management.¹⁷¹ Persistence of significant SAM requires revision. Assessment of mitral inflow typically involves measurement of transvalvular gradient, recognizing this is dependent on cardiac output.

TRICUSPID VALVE REPAIR

Increased recognition of the adverse prognostic effects of TR has stimulated interest in tricuspid valve pathology. After surgical correction of left-sided valvular heart disease, severe secondary TR often fails to improve. Even mild or moderate secondary TR may progress if uncorrected. Current AHA/ACC guidelines recommend tricuspid valve repair for severe TR at the time of left-sided valve surgery.⁵⁰

The decision to intervene upon less than severe TR at the time of left-sided valve surgery is more complex. When not intervened upon at the time of left-sided valve surgery, mild or moderate TR progresses in approximately 25% of individuals.⁵⁰ Annular dilation strongly factors in the decision to intervene early. Individuals with mild or greater TR and either annular dilation (diastolic diameter of > 40 mm or > 21 mm/m²) or evidence of prior right-sided heart failure can benefit from concomitant tricuspid valve repair (type IIa recommendation). In the absence

of tricuspid annular dilation, tricuspid valve repair may be considered during left-sided surgery when moderate TR and pulmonary hypertension are present (type IIb recommendation).

The structure of the tricuspid valve is complex. Because the annulus is nonplanar and the valve leaflets unequal in size, it can be difficult to visualize all three leaflets in the same 2-D imaging plane. The anterior location of the valve (far field in the scanning plane) and the thin leaflets also limit 2-D TEE imaging. Measurement of annular diameter by 2-D TTE typically occurs in the A4C view,¹⁷² however this one measurement may not accurately reflect the degree of dilation and has underestimated maximal diameter compared to 3-D TEE.¹⁷³ Studies of 3-D TEE have enhanced our understanding of annular dilation in the septal-to-lateral dimension, the progression from an oval to more circular shape, and the accompanying changes in annular dynamism.^{174,175} Use of 3-D color may improve quantitative assessment of regurgitation severity,¹⁷² however in the intraoperative environment, underestimation of TR is not unexpected. Technologies using 3-D TEE will continue to fill a larger role in the evaluation of TR.

AORTIC DISSECTION

Acute type A aortic dissection has high morbidity and mortality. Accurate diagnosis is paramount to rapid treatment. The preferred diagnostic approach under most circumstances is CTA, which identifies dissection with a sensitivity of 100% and specificity of 98%.¹⁷⁶ In some cases, TEE may be performed more readily than CTA. TEE performs with slightly lower sensitivity (86%-100%) and specificity (90%-100%), although in many studies TEE performs with comparable specificity to CTA.^{176,177} TEE imaging challenges include a blind spot in visualization of the distal ascending aorta and proximal arch due to interposition of the left mainstem bronchus between the aorta and esophagus. Imaging artifacts in the near field are common because of reverberation and refraction (especially in the presence of a pulmonary artery catheter) and may be erroneously interpreted as an intimal flap.

Intraoperative TEE during surgical intervention further defines features of the dissection. Measurements of the aortic annulus and root are performed, effacement of the sinotubular junction excluded, and structure and function of the aortic valve assessed. Mechanisms of aortic insufficiency include regurgitation due to a bicuspid valve, extension of the intimal flap to the annulus causing asymmetric leaflet prolapse, malcoaptation due to root dilation, and prolapse of the intimal flap preventing complete leaflet closure.¹⁷⁸ This mechanistic information influences whether valve repair is feasible and may lead to modification of the planned surgical procedure.¹⁷⁹ Further assessment includes examination of the true and false lumens. Blood flow may be visualized across the intimal flap. Usually the true lumen will expand during systole, which can be appreciated using M-mode echocardiography. The false lumen often demonstrates diastolic expansion and spontaneous echo contrast. Due to the complex nature of some dissections, it may be difficult to determine the true and false lumens accurately.

MECHANICAL CIRCULATORY SUPPORT (MCS)

Durable Mechanical Circulatory Support

Approximately 2500 durable left ventricular assist devices (LVADs) are implanted annually in North America, nearly half of which are destination therapy.¹⁸⁰ Intraoperative TEE confirms known pathology and is integral for identifying abnormalities that may require additional intervention.¹⁸¹⁻¹⁸³ Prior to implantation, echocardiographic assessment includes examination of biventricular size and function, and evaluation for intracardiac shunt (including PFO) and intracardiac thrombus. Preexisting valvular dysfunction is characterized. Greater than mild aortic insufficiency, moderate or greater mitral stenosis, and moderate or greater tricuspid insufficiency may require additional valvular procedures. An existing mechanical aortic valve may require replacement due to the risk for thrombus formation secondary to decreased transvalvular flow after LVAD implantation. Echocardiographic predictors of RV failure following LVAD implantation have been an area of considerable investigation, including the newer myocardial deformation techniques. At this time no measures reliably predict the need for biventricular mechanical support.¹⁸³

During device implantation, TEE guidance facilitates appropriate apical positioning of the inflow cannula. Post-implantation assessment includes LV size, degree of LV decompression, RV function, reassessment for PFO, and frequency and extent of aortic valve opening. Assessment of LV unloading includes determination of overall chamber size and the position of the interventricular septum, which should be midline rather than bowing toward either chamber. As LVAD speeds are increased, parameters are frequently reassessed, especially if there is concern for marginal RV function. An appropriately positioned inflow cannula should be in the apex, directed toward the mitral valve, and should not interfere with the subvalvular apparatus. Acute angulation of the inflow cannula toward the septum may lead to cannula obstruction. Typical inflow velocity is 1.5 m/s or less. Imaging artifacts with newer-generation LVADs may preclude spectral Doppler and color flow assessment of the cannula. The outflow graft-ascending aorta anastomosis may be visualized in ME or UE views. An outflow velocity of 2 m/s or greater raises concern for obstruction, although normal values for newer generation devices may be higher.^{183,184} The mid-portion of the outflow graft can also be visualized along the right side of the heart, but should not cause compression.

Temporary Mechanical Circulatory Support

Common percutaneous devices for temporary MCS include intraaortic balloon pumps, ECMO, Impella percutaneous ventricular assist devices (Abiomed, Danvers, MA), and TandemHeart percutaneous ventricular assist devices (CardiacAssist, Pittsburgh, PA). Surgically implanted devices are options when longer duration of temporary support is anticipated. Appropriate device position depends on the specific technology and may be facilitated by TEE guidance.^{183,185} When assessing for myocardial recovery, TEE may add to the information provided by hemodynamics and has been incorporated into some venoarterial extracorporeal membrane oxygenation weaning protocols.¹⁸⁶

CONGENITAL HEART SURGERY

In congenital heart surgery, TEE provides diagnostic information, contributes to surgical planning, identifies residual defects after repair, and influences postoperative medical management.¹⁸⁷ As TEE has been more widely applied in congenital heart surgery, contemporary studies report pre-CPB findings altering the surgical procedure in approximately 1% to 9% of cases and residual lesions requiring surgical revision in approximately 4% to 6% of cases, with greater impact in more complex cases.¹⁸⁸⁻¹⁹¹ When including return to CPB for brief ventricular support or an assist device, TEE has impacted surgical interventions in close to 13% of cases.¹⁸⁹ TEE further influences pharmacologic management and provides new diagnostic information. Epicardial imaging may provide complementary information to TEE when assessing pulmonary arteries and coronary arteries during pediatric congenital heart surgery.¹⁹² There is a limited body of literature suggesting that intraoperative TEE in pediatric cardiac surgery has cost-saving benefits.¹⁹³

OTHER SURGICAL PROCEDURES

The intraoperative echocardiographer encounters many scenarios where valvular disease requires surgical intervention, including native and prosthetic valve dysfunction. TEE confirms the preprocedure diagnosis and may refine the location of structural abnormalities. Surgical intervention for endocarditis prompts TEE evaluation for vegetations on other valves and the presence of abscess cavities. Other cardiac surgical procedures that commonly rely on intraoperative TEE include minimally invasive approaches (valve repair/replacement and CABG), cardiac mass removal, heart transplantation, and pulmonary thromboendarterectomy.

Intraprocedural Transesophageal Echocardiography- Structural Heart Interventions

Innovations in percutaneous technologies have led to exciting growth in the management of structural heart disease, extending treatment to patients with previously limited therapeutic options. The section that follows elaborates on the role of echocardiography in a few of these percutaneous procedures.

TRANSCATHETER AORTIC VALVE REPLACEMENT

First approved for commercial use in Europe in 2007 and the United States in 2011,¹⁹⁴ transcatheter aortic valve replacement (TAVR) has revolutionized the treatment paradigm for aortic stenosis. TAVR is indicated for the treatment of extreme- and high-risk populations with severe symptomatic aortic stenosis and it is a reasonable alternative in patients with intermediate surgical risk.¹⁹⁵ The number of procedures performed annually continues to grow.¹⁹⁶ Comprehensive resources are available detailing the periprocedural echocardiographic imaging for TAVR.^{197,198}

Valve Sizing

Annular dimensions guide appropriate sizing of the transcatheter valve. The preferred imaging modality for annular size measurements is multidetector computed tomography (MDCT).¹⁹⁹ As compared to MDCT, 2-D TTE and TEE routinely underestimate annular size because of the elliptical shape of the aortic valvular complex.^{200,201} 3-D TEE yields larger annular size measurements than 2-D TEE.^{202,203} Measurements obtained by 3-D TEE may underestimate²⁰⁴⁻²⁰⁶ or be comparable to^{200,203,207} MDCT measurements. Both MDCT^{208,209} and 3-D TEE^{205,210} have been used to predict postimplantation paravalvular regurgitation. Automated 3-D TEE software is under investigation, demonstrating reproducibility and good agreement with MDCT-based prosthesis sizing.^{211,212}

Intraprocedural Examination

At the beginning of the procedure, echocardiographic assessment includes not only evaluation of the aortic valve, but also determination of biventricular function, assessment of other valvular abnormalities, characterization of any preexisting pericardial effusion, and identification of other features of procedural significance, including small LV cavity and basal septal hypertrophy. When the procedure is underway, wire and device position can be guided with echocardiography, although often fluoroscopy is the primary tool. Ideal valve position prior to deployment depends on valve design (e.g. self-expanding or balloon-expandable).

Following implantation of the valve, an integrated assessment using fluoroscopy, invasive hemodynamics, and echocardiography provides information about valve position, severity of paravalvular regurgitation, and transvalvular gradients. Early recognition of unfavorable results allows for further interventions such as balloon dilation or implantation of a second device. Echocardiography excludes other complications, including functional mitral stenosis, new pericardial effusion, new RWMA (potentially due to coronary obstruction), and aortic injury.

Transesophageal Echocardiography Versus Transthoracic Echocardiography

Active debate surrounds the optimal anesthetic approach²¹³ and intraprocedural imaging strategy²¹⁴ for transfemoral TAVR. Original TAVR clinical trials were performed under general anesthesia with intraprocedural TEE.^{215,216} However technology has improved and procedures are of shorter duration, making procedures under monitored anesthesia care or with nursing-administered sedation feasible. High-volume centers employ these techniques along a spectrum, ranging from 100% general anesthesia, to using both sedation and general anesthesia, to nearly 100% of cases with sedation.²¹⁷ In the United States from 2012 to 2015, use of general anesthesia decreased from 97.6% to 82.6%, while use of moderate sedation increased from 2.2% to 16.6%.¹⁹⁶ In a 2015 survey of 250 centers representing 38 countries, general anesthesia was the most common technique in 60.1% of centers, with 39.5% of the total centers reporting exclusive use of general anesthesia.²¹⁸ Forty-six percent of centers reported systematic use of intraprocedural TEE; 62% and 31% of centers reported use of TEE and TTE, respectively, for assessment of aortic regurgitation

following device implantation.²¹⁸ Despite the increasing shift away from general anesthesia and consequently TEE, use of intraprocedural TEE may be preferable for monitoring patients at high risk for coronary occlusion or annular rupture, reducing intravenous contrast exposure in high-risk patients, and providing secondary measurements for valve sizing.²¹⁹

Because intraprocedural TEE is often performed in the setting of general anesthesia, it is difficult to assess the independent effects of general anesthesia or echocardiographic modality on outcomes. In a study comparing general anesthesia to nongeneral anesthesia in transfemoral TAVR, the majority of patients receiving general anesthesia had intraprocedural TEE, while TEE was performed in a minority of cases without general anesthesia. Propensity-matched analysis adjusted for type of anesthetic did not demonstrate significant differences in rates of successful valve deployment, more-than-mild aortic regurgitation, or other complications in patients that had an intraprocedural TEE versus those that did not.²²⁰ A retrospective study of 454 consecutive patients undergoing transfemoral TAVR with either TTE under moderate sedation ($n = 234$) or TEE under general anesthesia ($n = 220$) showed no difference in the incidence of at least mild paravalvular regurgitation at hospital discharge (33% in TTE group vs. 38% TEE group, respectively $P = .326$).²²¹ The TTE group did however have a significantly higher incidence of second valve implantation (7% vs. 2%, $P = .026$) and post-dilation (38% vs. 17%, $P < .001$).²²¹ Many retrospective studies are confounded by chronology bias—use of primarily TEE early on with a transition to TTE occurring after the development of procedural expertise. Additionally, findings with earlier generations of valves may not be applicable to the current generation used in clinical practice. While current studies do not allow definitive conclusions to be drawn about TEE versus TTE in TAVR, they represent initial steps in tackling these questions.

TRANSCATHETER MITRAL VALVE REPAIR

Percutaneous treatment of MR with the MitraClip procedure (Abbott Vascular-Structural Heart, Menlo Park, CA) uses a clip to grasp and appose the free edges of the anterior and posterior mitral leaflets at the site of regurgitation, creating a double orifice similar to that of the surgical Alfieri technique. In the United States, MitraClip was initially approved for the treatment of severe primary (degenerative) MR in patients with prohibitive surgical risk. In Europe, percutaneous valve repair has also been used for the treatment of secondary (functional) MR. In both patient populations, impact of treatment and long term outcomes are areas of active investigation.²²² Several randomized trials now include patients with secondary MR²²³⁻²²⁵, and the body of literature is rapidly growing. As of 2019, indications for MitraClip therapy in the United States have expanded to include patients with significant secondary MR despite optimal medical therapy.

Echocardiographic imaging is essential in determining the suitability for the procedure, providing intraprocedural guidance, and evaluating procedural success.²²⁶ Preoperative echocardiography characterizes the anatomic features of the valve, examines the subvalvular apparatus, and provides qualitative and quantitative assessments of the severity of the regurgitation. Optimal morphologic features

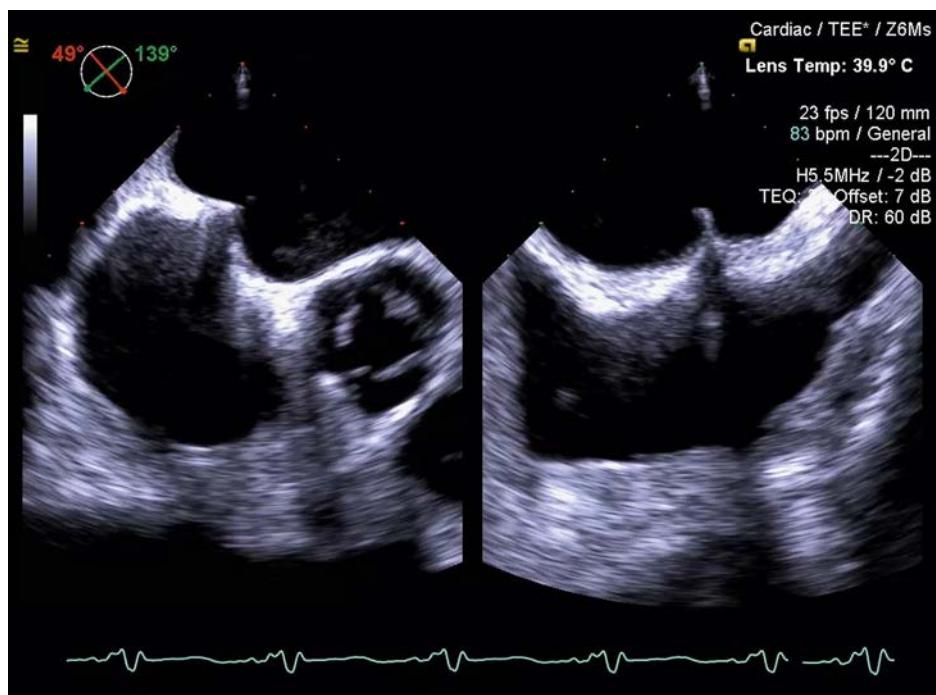


Fig. 37.29 Biplane imaging of modified midesophageal aortic valve short axis (left) and bicaval (right) views. Live biplane imaging is an essential tool in guiding puncture of the interatrial septum in catheter-based procedures entering the left side of the heart.

are based on the EVEREST study inclusion and exclusion criteria, and include regurgitation central in origin (A2/P2), lack of calcification in grasping area, mitral valve area greater than 4 cm^2 , small flail gap (<10 mm), and narrow flail width (<15 mm).²²⁷ Intraprocedural TEE, in combination with fluoroscopy, guides interatrial septal puncture and advancement of the catheter into the LA (Fig. 37.29). Under TEE guidance, the clip is aligned above the leaflets with the arms perpendicular to the line of coaptation. Once satisfactory position is obtained, the device system is advanced into the left ventricle (Fig. 37.30). Live echocardiographic imaging is often utilized as the system is slightly withdrawn so the clip grasps the leaflets; confirmation of bileaflet capture and assessment of the regurgitation severity is performed prior to clip deployment. Presence of significant residual or worsened MR, or evidence of mitral stenosis by echocardiography may prompt repositioning of the clip. Regurgitation and stenosis are quantified after deployment; placement of a second clip may be indicated if significant MR remains.

OTHER PERCUTANEOUS STRUCTURAL HEART PROCEDURES

TEE serves as a standard imaging modality in numerous other catheter-based procedures, including atrial septal defect closures, ventricular septal defect closures, LA appendage occlusion, valve-in-valve therapies for failed prostheses, and paravalvular leak closures. Echocardiography during these procedures assesses the suitability of the intervention, provides real-time guidance, and assesses the effectiveness of the intervention. Transcatheter techniques under investigation include mitral valve replacement²²⁸ and therapies directed at TR, including valve annuloplasty, techniques targeting leaflet coaptation, and caval valve implantation.^{229,230}

Emerging Technologies

FUSION IMAGING

Multimodality imaging is essential for planning and execution of percutaneous structural heart procedures. Interventional cardiologists use echocardiographic and fluoroscopic data simultaneously to guide catheter manipulation and device deployment. Often these images are displayed on separate screens, which can provide challenges as the proceduralist must combine the information to reconstruct a mental 3-D representation of the structures. Fusion of echocardiographic and fluoroscopic images provides simultaneous visualization of catheter movements with cardiac structures. Currently a technology in development, the optimal applications will be better characterized in the coming years.^{231,232}

THREE-DIMENSIONAL PRINTING

3-D models of valvular structures and complex congenital heart defects can be printed from echocardiographic and other imaging data sets. Visual and tactile interactions with the models allow for improved understanding of structural interactions and abnormalities. At present, 3-D printing in this context is most relevant as a training and simulation tool, but patient-specific models can be used to optimize pre-procedural planning.²³³ Model quality depends on the quality of the imaging data. High cost, long printing times, and lack of materials capable of replicating tissue structural properties limit perioperative applications of 3-D printing.²³⁴

ARTIFICIAL INTELLIGENCE/MACHINE LEARNING

Artificial intelligence and its subfield, machine learning, permeate everyday life. The quest to apply these

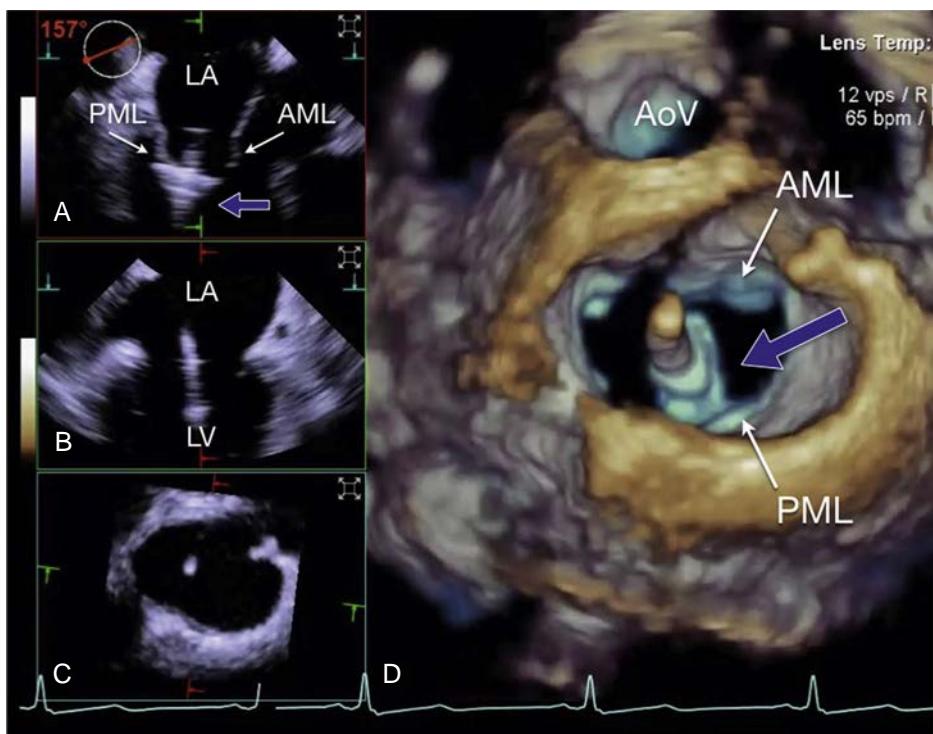


Fig. 37.30 Simultaneous live display of two-dimensional reference planes (Panels A-C) and the three-dimensional volume (Panel D) during a MitraClip procedure. The clip (blue arrow) should be aligned orthogonally to the line of coaptation of the mitral valve. Real-time imaging facilitates optimal device positioning by providing visual feedback following changes in catheter and device position. AML, Anterior mitral leaflet; AoV, aortic valve; LA, left atrium; LV, left ventricle; PML, posterior mitral leaflet.

technologies to questions in medicine is no exception. Artificial intelligence is a branch of computer science focused on development of computer systems capable of emulating human intelligence, including aspects such as learning, knowledge retention, problem solving, and reasoning.²³⁵ Machine learning focuses on the development of predictive models where rules are learned from data rather than explicitly programmed.²³⁶ Currently, many 3-D echocardiography platforms require manual, time-consuming user input for quantitative analysis, making their routine use in the operating room impractical. While not yet mainstream, automated analysis platforms using artificial intelligence in echocardiography exist, including mitral and aortic valve analysis in 3-D TEE²³⁷ (Fig. 37.31) and automated chamber quantification in 3-D TTE.²³⁸⁻²⁴⁰ Outside of the perioperative arena, machine learning has been applied to 2-D TTE assessment of LV hypertrophy phenotypes.²⁴¹ Refinement and continued development of such technologies conceivably could provide complex, quantitative valvular and ventricular analyses under dynamic conditions, improving accuracy and reducing inconsistencies in interpretation.

Perioperative Transthoracic Echocardiography and Focused Cardiac Ultrasound

While TEE has historically been the principal modality of perioperative echocardiography, TTE is emerging as a relevant and feasible counterpart. Anesthesiologists with substantial echocardiography experience have initiated

preoperative TTE consult services, providing echocardiographic examination and interpretation when indicated during preoperative evaluation.²⁴²⁻²⁴⁵ Echocardiographic findings have led to management changes in 54% to 84% of cases, including modification of the planned anesthetic approach and influencing decision making regarding the need for additional preoperative consultation or intraoperative monitoring.²⁴²⁻²⁴⁴ Interpretation of the images by cardiologists²⁴⁴ and formal TTE imaging in a subset of patients confirmed the major findings of the focused TTE examination in the majority of cases.^{242,243} Standard preoperative echocardiography in patients undergoing major surgery has failed to demonstrate an association with improved outcomes,²⁴⁶ so the value of preoperative anesthesiologist-performed TTE in guiding interventions or prognostication needs further characterization. As these consult services develop, important considerations include the intended scope of the examination and the experience of the individuals performing and interpreting the studies.

Several small investigations demonstrate the feasibility of FoCUS* prior to urgent or emergent surgical procedures, resulting in changes to the anesthetic technique or management plan.²⁴⁷⁻²⁴⁹ Specific patient populations, such as patients sustaining hip fractures, may be optimally suited for FoCUS. Delay in surgery for hip fractures is associated with increased risk of mortality,^{250,251} and preoperative echocardiographic examination through standard channels has been associated with delayed surgery.²⁵² A multi-center,

*These studies described the use of “focused transthoracic echocardiography.” To maintain consistency with terminology proposed in published guidelines, we described these studies as FoCUS.

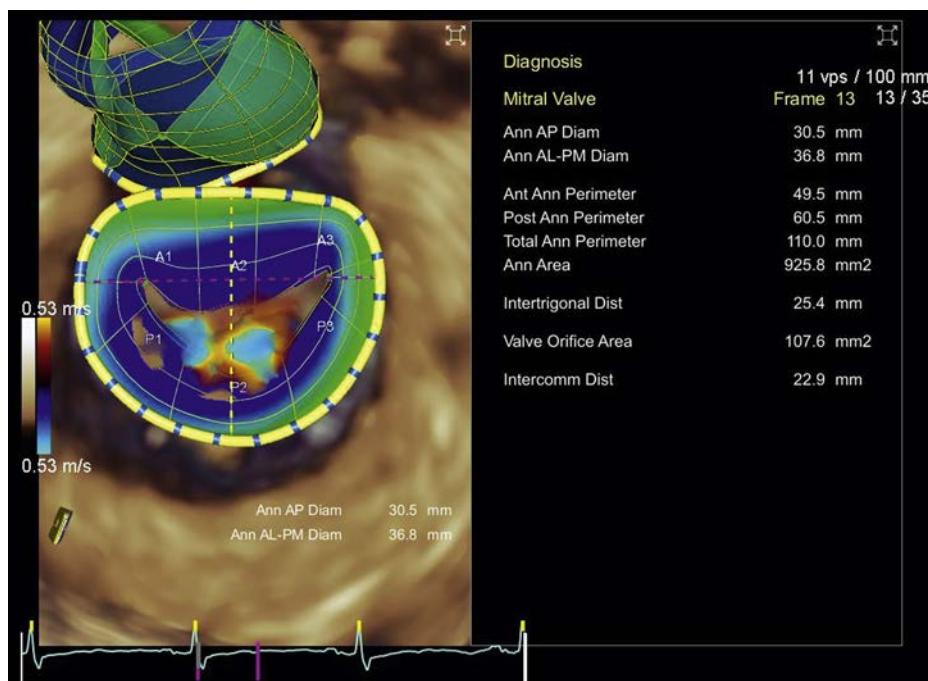


Fig. 37.31 Example of valve analysis program using artificial intelligence. Dynamic measurements of mitral annular geometry are provided throughout the cardiac cycle.

randomized pilot study of preoperative FoCUS in hip surgery has been completed, providing the groundwork for a larger, definitive randomized controlled trial.²⁵³

The operating theater, with the sterile surgical field and limited patient accessibility, is not necessarily conducive to transthoracic imaging. Despite the perceived challenges, small studies have prospectively demonstrated the feasibility of FoCUS during surgery. In a series that included stable and unstable patients undergoing a range of surgeries (orthopedics/trauma, abdominal/vascular, urology/gynecology, and head or ear, nose, and throat), focused examination was feasible in more than 90% of cases.²⁵⁴ Patients with chronic obstructive pulmonary disease and those undergoing abdominal surgery had significantly worse image quality. Subsequent studies have demonstrated feasibility in thoracic surgery,²⁵⁵ and an impact on intraoperative management in 22% to 66% of patients.^{255,256}

There is increasing interest in FoCUS and TTE for hemodynamic monitoring and diagnostic assessments in the peri-partum anesthetic management of pregnant women.²⁵⁷ The leftward displacement of the heart and left tilt to avoid aortocaval compression are favorable for parasternal and apical imaging windows.²⁵⁷ Postpartum hemorrhage, preeclampsia, and cardiomyopathy can all result in hemodynamic derangements detectable by cardiac ultrasound.²⁵⁸

Despite the recognized value of FoCUS,²⁵⁹ it is not yet a routine aspect of anesthesiology practice. A survey of cardiothoracic anesthesiologists revealed a familiarity with FoCUS (81%), but only 47% of respondents use it in their clinical practice.²⁶⁰ While the study has limitations, including low response rate, it provides insight into current practice patterns. The most frequently cited obstacle to use of FoCUS was lack of training, followed by concerns about missed diagnosis, lack of equipment, and lack of a certification process. As the majority of these anesthesiologists

had prior formal echocardiography training (TEE, TTE, or FoCUS), it is likely the obstacles would be further amplified for anesthesiologists without this exposure.

Critical Care

Echocardiography performed and interpreted at the point of care is feasible and influential in the care of critically ill surgical and nonsurgical patients. Both TTE and TEE findings impact the management of patients in shock, influence diagnostic and therapeutic management,²⁶¹⁻²⁶³ and provide prognostic information.^{264,265} In a study of 110 patients in undifferentiated shock, early resuscitation guided by echocardiography was associated with improved survival, less fluid administration, and greater inotrope use compared to 110 control patients examined retrospectively.²⁶⁶ Although this does not indicate causality, it highlights the potential value of echocardiography in resuscitation, especially in light of the association between positive fluid balance and mortality.²⁶⁷⁻²⁶⁹ TTE use is more prevalent than TEE in the ICU,²⁷⁰ and European and Australian intensivists use TEE more commonly than their North American counterparts.²⁷¹ TEE offers improved diagnostic accuracy in the setting of mechanical ventilation,²⁶¹ however several groups have obtained adequate transthoracic images for focused studies in the majority of mechanically ventilated patients.^{262,272,273} TEE provides superior imaging for the evaluation of suspected endocarditis or intracardiac mass, evaluation of great vessel pathology, and assessment of valvular dysfunction.⁵⁶

Similar to perioperative TEE, there are proposed basic and advanced applications of critical care echocardiography.^{54,57} Basic applications include assessment of global biventricular size and function, identification of

pericardial fluid or tamponade, identification of severe valvular regurgitation, and as guidance for resuscitative efforts during and after cardiac arrest. Advanced applications include quantitative assessments of function and filling pressures, evaluation of fluid responsiveness, and detection of abnormalities including cardiac source of embolism, endocarditis, acute aortic pathology or injury, intracardiac shunt, and complications of MI. Evaluation of suspected endocarditis, great vessel pathology or injury, and PE may be considered basic or advanced applications.

FLUID RESPONSIVENESS

When assessing a patient in shock, the decision to administer, withhold, or stop fluids is often challenging. Patients who are fluid responsive will demonstrate an increase in SV or cardiac output in response to a fluid challenge,¹⁰⁷ and dynamic indices (such as heart-lung interactions or the passive leg raise maneuver) temporarily induce changes in preload to assess for this response. In patients receiving passive mechanical ventilation, several echocardiographic dynamic parameters have been used to predict fluid responsiveness and aid in clinical decision making:

- *Inferior vena cava distensibility index:* During positive pressure ventilation, inspiration causes a rise in intrathoracic pressure, which decreases the venous return to the heart and leads to IVC distension. During expiration, IVC diameter is at a minimum. Two IVC distensibility indices (dIVC or ΔIVC) relating the maximum (D_{\max}) and minimum (D_{\min}) diameters during the respiratory cycle have been described:

$$\Delta\text{IVC} = \frac{D_{\max} - D_{\min}}{D_{\min}} * 100\%$$

where a threshold of 18% or greater discriminated between responders and nonresponders,²⁷⁴

and

$$\Delta\text{IVC} = \frac{D_{\max} - D_{\min}}{(D_{\max} + D_{\min})/2} * 100\%$$

where a threshold of 12% or greater discriminated between responders and nonresponders.²⁷⁵

Use of the first equation is most common.²⁷⁶ In more heterogeneous populations, ΔIVC performs less accurately (two subsequent studies report an area under the receiver operating characteristic curve of 0.43 and 0.635, respectively).^{277,278} The reported optimal thresholds for discriminating responders from nonresponders also vary. In general, ΔIVC has greater specificity than sensitivity.

Several preconditions must be met for ΔIVC to be a useful tool, including absence of spontaneous respiratory effort, 8 to 12 mL/kg tidal volumes (necessary to increase the pleural pressure to affect the IVC diameter), absence of cor pulmonale, and absence of intraabdominal hypertension.¹⁰⁸ Highlighting the importance of these conditions,

ΔIVC accurately predicts fluid responsiveness in patients ventilated with tidal volumes of 8 to 10 mL/kg and PEEP of 5 cm H₂O or less but poorly predicts fluid responsiveness in patients receiving tidal volumes less than 8 mL/kg or PEEP greater than 5 cm H₂O.²⁷⁶

- *Superior vena cava collapsibility index:* During positive pressure ventilation, the SVC collapses during inspiration due to increased intrathoracic pressure (a pattern opposite that of the IVC). An initial study of patients with septic shock evaluated the SVC collapsibility index (ΔSVC), defined as:

$$\Delta\text{SVC} = \frac{D_{\max} - D_{\min}}{D_{\max}} * 100\%$$

where D_{\max} is the maximum diameter on expiration and D_{\min} is the minimum diameter on inspiration.²⁷⁹ A threshold of more than 36% distinguished fluid responders from nonresponders with 90% sensitivity and 100% specificity. In a larger cohort including mechanically ventilated patients with any type of shock, ΔSVC of 21% or greater predicted fluid responsiveness with 61% sensitivity and 84% specificity.²⁷⁸ When compared in the same patients, ΔSVC has greater diagnostic accuracy than ΔIVC.^{1,277,278} TEE imaging is required to determine ΔSVC.

- *Variation in peak aortic/LVOT velocity:* Positive pressure ventilation results in cyclical changes in the maximum velocity of flow in the LVOT and across the aortic valve. Application of PWD at the level of the aortic valve²⁸⁰ or in the LVOT²⁷⁸ allows for measurement of the highest (V_{\max}) and lowest (V_{\min}) peak velocities over the course of the respiratory cycle. The difference between the two is divided by their mean and expressed as a percentage as follows:

$$\Delta V_{\max\text{Ao}} = \frac{V_{\max} - V_{\min}}{(V_{\max} + V_{\min})/2} * 100\%$$

A threshold of 12% or more discriminated between responders and nonresponders in initial investigations.²⁸⁰ Similar to other dynamic indices, in larger, diverse populations, this parameter does not perform as accurately as originally reported. When compared in the same cohort, $\Delta V_{\max\text{Ao}}$ had the highest sensitivity, whereas ΔSVC had the highest specificity for predicting fluid responsiveness.²⁷⁸

Additional echocardiographic assessments utilizing heart-lung interactions and dynamic parameters are an area of active research. End-expiratory occlusion maneuvers alone or in combination with end-inspiratory occlusions have been used to predict fluid responsiveness as assessed by changes in LVOT velocity time integral and/or maximum aortic velocity.^{281,282} These approaches are not currently widely applied and require further validation in varying patient populations. With any of these indices, the clinician should interpret these findings within the overall context of the patient's status and with an awareness of the limitations.

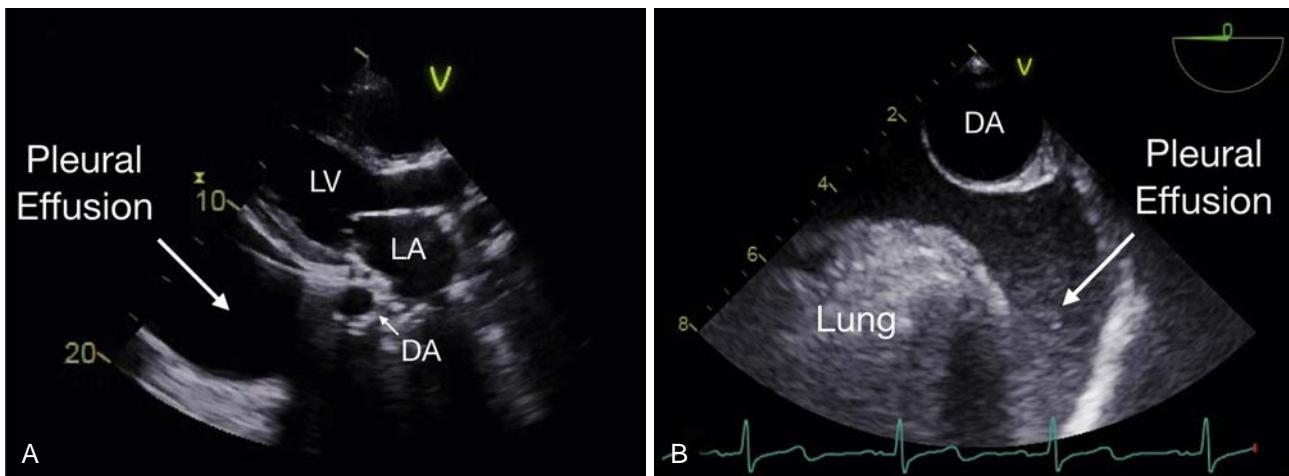


Fig. 37.32 Pleural effusions demonstrated by echocardiography. (A) This transthoracic parasternal long axis view demonstrates an echolucent space posterior to the DA, consistent with a large left pleural effusion. A pericardial effusion (not present in this image) would be seen as an echolucent space anterior to the descending aorta. (B) A transesophageal descending aorta short axis view demonstrating an echolucent space consistent with a left pleural effusion. Note the atelectatic lung at the left of the image. DA, Descending aorta; LA, left atrium; LV, left ventricle.

POST-CARDIAC SURGERY

A 2016 systematic review of 15 studies examining TTE and TEE after cardiac surgery demonstrated that echocardiography (performed by a mix of novice and expert sonographers) was feasible and that findings frequently led to management changes.²⁸³ In hemodynamically unstable postoperative cardiac surgical patients, significant discordance has been demonstrated between diagnoses obtained from continuous TEE and diagnoses obtained from hemodynamic measurements.²⁸⁴ TEE-based diagnoses have greater interobserver agreement, suggesting that echocardiography may add valuable diagnostic information even in the presence of invasive hemodynamic monitors.

TRAUMA AND RESUSCITATION

For decades, cardiac ultrasound via the subcostal window has been part of the evaluation of the trauma patient within the Focused Assessment with Sonography for Trauma (FAST) examination.²⁸⁵ FoCUS extends this cardiac examination and has been adopted as a tool to guide resuscitation of trauma patients. A trial randomizing trauma patients to FoCUS or no FoCUS as part of the initial evaluation demonstrated significantly less fluid administration, shorter time from the trauma bay to the operating room, and higher rate of ICU admissions in the FoCUS group.²⁸⁶ In blunt or penetrating chest trauma, echocardiography reveals complications such as cardiac rupture, dysfunction (due to contusion), valvular injury, hemopericardium, and aortic disruption.²⁸⁷

OTHER CLINICAL APPLICATIONS

Echocardiography may identify causes of hypoxia, such as right-to-left intracardiac shunt or pleural effusion (Fig. 37.32). Clinicians often incorporate lung ultrasound with echocardiography when evaluating cardiopulmonary failure (also see Chapter 83).²⁸⁸

Novel applications of echocardiography in critical care are emerging. Areas of active research include strain imaging in sepsis²⁸⁹ and the use of echocardiography in

evaluating patients that fail to liberate from mechanical ventilation.²⁹⁰

HANDHELD CARDIAC ULTRASOUND

With their compact size and portability, handheld echocardiography devices are ideal for point-of-care assessments.²⁹¹ Intensivists with limited echocardiography training have performed and interpreted focused handheld examinations, estimating LV systolic function with reasonable accuracy compared to formal TTE performed by a sonographer and interpreted by an echocardiographer.²⁹² When performed by intensivists experienced in echocardiography, handheld echocardiographic examinations offer similar 2-D diagnostic capabilities to full-platform TTE in mechanically ventilated, critically ill patients, with limitations due to lack of spectral Doppler.²⁹³

MINIATURE TEE PROBES

Miniature TEE probes have promise as tools for hemodynamic monitoring or characterization of cardiopulmonary failure in the critical care setting.^{294,295} Examination with miniaturized multiplane TEE probes is feasible and well tolerated in ventilated, critically ill patients.²⁹⁵ When compared to standard TEE, miniature probes enable concordant diagnostic assessments of causes of cardiorespiratory failure, albeit with lesser image quality. Consequent to the diminished image quality, common hemodynamic measurements can be performed less frequently, but when possible, the results agree with those obtained by standard TEE. Larger studies and evaluation of resulting clinical impact are necessary prior to widespread adoption of miniature TEE.

Training and Certification

KNOWLEDGE EXPECTATIONS AND TRAINING

Across the field of anesthesiology, there is a growing impetus for formalized training in perioperative ultrasound

including echocardiography. The American Council for Graduate Medical Education requires anesthesiology residents to demonstrate competency in the acquisition of standard transesophageal and transthoracic echocardiographic views.²⁹⁶ Certification by the American Board of Anesthesiology requires basic knowledge of TEE, assessed both in the written examination²⁹⁷ and the Objective Structured Clinical Exam.²⁹⁸ Accredited Anesthesiology Critical Care Medicine programs require fellows to demonstrate knowledge of ultrasound principles and proficiency in TEE and TTE.²⁹⁹

Formalized echocardiography training requires curriculum development and educational initiatives. For some trainees, exposure to ultrasound now begins in medical school. Several institutions include cardiac ultrasound in their educational programs, initially using it to teach anatomy and function, and later as an adjunct to the physical examination.³⁰⁰ Many residency programs are developing multimodal longitudinal curricula including didactic lectures, hands-on workshops, online modules, simulation, and formal echocardiography rotations.³⁰¹⁻³⁰⁴ Online virtual simulation provides users an interactive format to learn standard views and spatial relationships.^{305,306} Mannequin-based simulation is an effective method for improving the acquisition of cognitive and technical skills both in TEE³⁰⁷⁻³¹² and TTE,^{308,313,314} and may be a useful educational tool for learners early in training. In addition to these multimodal didactic approaches, practical clinical experience under supervision remains a key part of the training process. In North America, advanced training in TEE usually occurs in the context of the cardiothoracic anesthesiology fellowship.

The coming years will likely include increasing emphasis on FoCUS training in anesthesiology. In a survey of US anesthesiology residency program directors and trainees, the majority of respondents indicated FoCUS should be a standard component of training, while only a minority of the programs incorporated such training.³¹⁵ A different survey of Canadian residency program directors indicated FoCUS training should be compulsory, and while most of the programs had training opportunities, only a minority included mandatory rotations.³¹⁶ The challenge to the specialty is how to best incorporate FoCUS, to define minimum training standards and competencies, and to provide education for trainees as well as anesthesiologists already in practice. The European Association of Cardiovascular Imaging has published a reference curriculum and syllabus for FoCUS education and training.³¹⁷ Clinicians must clearly understand the narrow scope of FoCUS, recognizing the limitations and indications for expert consultation. Many examples of the applicability of perioperative FoCUS are in the hands of experienced echocardiographers—whether this translates to operators with less experience and less formal training will need to be examined. After the initial acquisition of cognitive and technical skills in ultrasound, continued use is important to ensure proficiency; when these skills are not used for over a year, significant attrition occurs.³¹⁸

CERTIFICATION

The National Board of Echocardiography offers examination and certification in basic (Basic PTEeXAM) and advanced (Advanced PTEeXAM) perioperative TEE.³¹⁹ Individuals that

pass the examination achieve testamur status. Testamurs can achieve basic certification via supervised training, practice experience, or extended continuing medical education. Advanced PTEeXAM testamurs can receive certification by completing an accredited cardiothoracic anesthesiology pathway; a practice experience pathway is only available to individuals that completed training prior to 2009. There is no certification for perioperative TTE, however, qualified individuals can take the Examination of Special Competence in Adult Comprehensive Echocardiography (ASCeXAM) and achieve testamur status. In Europe, the European Association of Cardiovascular Imaging and European Association of Cardiothoracic Anaesthesiology jointly offer certification in adult TEE.³²⁰ General critical care ultrasound and basic critical care echocardiography are now considered to be within the scope of practice of intensivists, while certification has been proposed for those obtaining advanced training.^{55,56} As of 2019, the National Board of Echocardiography now offers an Examination of Special Competence in Critical Care Echocardiography (CCEeXAM).³²¹

Future Directions

The evolution of perioperative echocardiography is at a transformative juncture. The landscape is one of growth, innovation, and accessibility. From its perioperative origin as a monitor in cardiac surgery decades ago, TEE has become integral to the practice of cardiac anesthesia and surgical decision making. More sophisticated and automated platforms will pave the way for increasingly complex, quantitative analyses to be performed in real time. Catheter-based procedures push the boundaries of treatable pathologies, and echocardiography is central to the multimodal imaging techniques employed by structural heart teams. Many cardiac anesthesiologists have an interest in TTE, which complements and builds upon their already established cognitive and technical skills.

Outside of the cardiac arena, anesthesiologists use TEE as an intraoperative monitor in high-risk procedures or populations. Basic knowledge of TEE is now expected of anesthesiology trainees. In the future, knowledge of transthoracic imaging in the form of FoCUS may too become a requirement. Increased portability of ultrasound machines and newer technologies like capacitive micromachined ultrasound transducers make the potential for widespread point-of-care imaging an imminent reality. With the growing interest and investment in ultrasonography, a basic skill set may soon be within the purview of all anesthesiologists. Opportunities for research into education and the impact of anesthesiologist-performed examinations abound. Critical care training objectives now include basic proficiency in echocardiography in the larger context of critical care ultrasound. Similar to advanced perioperative TEE, proficiency in advanced critical care echocardiography requires additional dedicated training and allows comprehensive evaluation of cardiac structure and function, and hemodynamic assessments in the context of the critically ill patient. Whether applied as a routine monitor, a more advanced diagnostic tool, or used in procedural guidance, echocardiography is an essential component of the modern perioperative physician's armamentarium.

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Video 37.1 3D Imaging—multiple beat full-volume acquisition. Multiple beat full-volume acquisitions create three-dimensional images by acquiring individual data subvolumes over consecutive heartbeats and then “stitching” the data together to create the larger full-volume image.

Video 37.2 Echocardiographic views in focused cardiac ultrasound (FoCUS): Parasternal long axis (PLAX), parasternal short axis (PSAX), apical four-chamber (A4C), subcostal four-chamber (SC4), and subcostal IVC (SIVC). *A*, Anterior wall; *AML*, anterior mitral leaflet; *AoV*, aortic valve; *DA*, descending aorta; *I*, inferior wall; *IVC*, inferior vena cava; *IVS*, interventricular septum; *L*, lateral wall; *LA*, left atrium; *LV*, left ventricle; *PML*, posterior mitral leaflet; *RA*, right atrium; *RV*, right ventricle; *RVOT*, right ventricular outflow tract; *S*, septal wall.

Video 37.3 Transesophageal echocardiographic views demonstrating qualitatively normal left ventricular systolic function. *AoV*, Aortic valve; *LA*, left atrium; *LV*, left ventricle; *MV*, mitral valve; *RA*, right atrium; *RV*, right ventricle.

Video 37.4 Transthoracic parasternal short axis view demonstrating flattening of the interventricular septum in *diastole*, a finding observed in states of right ventricular volume overload. The septal curvature remains normal and the left ventricular cavity appears circular during systole. Of note, both left and right ventricular systolic function are reduced. *IVS*, interventricular septum; *LV*, left ventricle; *RV*, right ventricle.

Video 37.5 Transthoracic parasternal short axis view of a patient with right ventricular pressure overload. The leftward shift and flattening of interventricular septum occurs throughout the cardiac cycle, but is most pronounced at *end-systole*. *IVS*, Interventricular septum; *LV*, left ventricle; *RV*, right ventricle.

Video 37.6 Takotsubo cardiomyopathy. These clips demonstrate the classic pattern of apical ballooning with preserved basal contractility seen in Takotsubo (stress-induced) cardiomyopathy. Additional patterns of regional involvement may also be observed.

Video 37.7 Systolic anterior motion of the mitral valve (SAM). The anterior mitral leaflet is visualized entering the left ventricular outflow tract and contacting the interventricular septum during systole. Turbulent flow is visualized in the left ventricular outflow tract (LVOT) and significant mitral regurgitation (MR) is present. Treatment of SAM includes discontinuation (or avoidance) of inotropes, increasing preload, increasing afterload, and decreasing the heart rate. With therapeutic interventions, the SAM decreases, there is less turbulence in the LVOT, and the MR decreases.

Video 37.8 Tamponade-right atrial systolic collapse. A large echoluent pericardial effusion is demonstrated (*). Within the effusion, there are areas of increased density suggesting organization. Right atrial collapse (*blue arrow*) during ventricular systole supports the clinical diagnosis of tamponade. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

Video 37.9 Tamponade-right ventricular diastolic collapse. Transthoracic parasternal long axis views displaying a large pericardial effusion (*). Right ventricular diastolic collapse (*green arrow*) is present, and supports the clinical diagnosis of tamponade. Left atrial collapse during ventricular systole, often observed later than right-sided chamber collapse, is also present (*red arrow*). *Ao*, Ascending aorta; *IVS*, interventricular septum; *LA*, left atrium; *LV*, left ventricle; *RVOT*, right ventricular outflow tract.

Video 37.10 McConnell's sign is an echocardiographic finding of right ventricular hypokinesis or akinesis with apical sparing. The apparent preservation of apical contractility actually reflects translational motion due to tethering of the apical fibers to the left ventricle. McConnell's sign may be observed in acute pulmonary embolism or right ventricular infarction. *IVS*, Interventricular septum; *RA*, right atrium; *RV*, right ventricle.

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KEY POINTS^a

- Identify the type of cardiovascular implantable electronic device (CIED) (pacemaker, transvenous defibrillator, subcutaneous defibrillator), as well as the generator manufacturer and model of the CIED.
- Contact the physician or clinic managing the patient's CIED in the preoperative period to obtain appropriate records and a perioperative recommendation (Heart Rhythm Society [HRS]).
- Obtain a copy of this interrogation and the perioperative recommendation from the CIED physician (HRS). Ensure that the implantable cardioverter-defibrillator (ICD) treatment settings are appropriate and that the CIED will pace the heart.
- Consider replacing any CIED near its elective replacement period in a patient scheduled to undergo either a major surgical procedure or a surgical procedure where monopolar electrosurgery will be used within 15 cm of the generator.
- Determine the patient's underlying rate and rhythm to determine the need for backup (external) pacing support.
- Ensure that all magnet behavior (pacing, suspension of shock therapy) is appropriate if magnet use is planned.
- Program minute ventilation rate responsiveness "off," if present.
- Consider disabling all rate enhancements to prevent misinterpretation of cardiac rhythm.
- Consider increasing the lower rate limit to provide optimal oxygen delivery for major procedures.
- If electromagnetic interference is likely, (1) disable antitachycardia therapy if a defibrillator is present and (2) consider asynchronous pacing for some pacing-dependent patients. Magnet application may be acceptable for some ICDs (disable antitachycardia therapy) or pacemakers (provide asynchronous pacing). Asynchronous pacing from an ICD always requires reprogramming.
- Monitor cardiac rhythm with the pulse oximeter (plethysmography) or arterial waveform analysis.
- Use a bipolar electrosurgical unit (ESU), if possible; if not possible, then pure "cut" electrosurgery is better than "blend" or "coag," and ESU should be applied in short bursts (<4 seconds) separated by at least 2 seconds.
- Place the ESU dispersive electrode in such a way to prevent electricity from crossing the generator-heart circuit.
- If the ESU causes ventricular oversensing with pacing quiescence or atrial oversensing with inappropriate ventricular pacing, limit the effect by using short ESU bursts, relocating the dispersive electrode, or placing a magnet over the pacemaker (not indicated for ICDs).
- Some patients require postoperative interrogation, especially if preoperative reprogramming took place. For "low-risk cases," HRS (but not ASA) states that this interrogation can take place in an ambulatory setting up to 1 month postoperatively. Some rate enhancements can be reinitiated, and a determination of optimum heart rate and pacing parameters should be made. Any patient with disabled antitachycardia therapy must be monitored until the antitachycardia therapy is restored.

^aAdapted from the American Society of Anesthesiologists (ASA) Practice Advisory (2005, revised 2011) and the Heart Rhythm Society (HRS), formerly the North American Society of Pacing and Electrophysiology (NASPE) and ASA Consensus Statement (2011) for Perioperative Management of Patients With a Pacemaker or Defibrillator.

Introduction

Cardiac implantable electronic devices (CIEDs) refer to a permanently implanted cardiac pacemaker, an implantable cardioverter-defibrillator (ICD), or a cardiac resynchronization therapy (CRT) device. Evolving technology for CIEDs and their widespread use for bradyarrhythmias, tachyarrhythmias, and congestive heart failure management have made the perioperative management of these devices critical for anesthesiologists. It is estimated that more than 3 million people in the United States have a pacemaker and more than 300,000 people have an ICD.^{1,2} With approximately 1 million patients worldwide receiving a pacemaker or ICD every year, patients with CIEDs are a growing population in the perioperative arena. The prevalence of cardiovascular disease in an aging population is an important driver for the increased utilization of CIEDs.

Historically, there was little guidance for anesthesiologists caring for patients with CIEDs because of the conflicting consensus statements regarding the perioperative management of CIEDs. In 2011, the Heart Rhythm Society (HRS)/American Society of Anesthesiologists (ASA) published an Expert Consensus Statement on the perioperative management of patients with CIEDs. This statement was in collaboration with the American Heart Association (AHA) and the Society of Thoracic Surgeons (STS).³ This article provides information and a guided team approach to best manage this patient population, and it has become an important piece of literature for anesthesiologists. In this chapter, we will review basic CIED function, the perioperative management of these devices, and emerging technology of CIEDs.

Basic Cardiac Implantable Electronic Device Function

PACEMAKERS

Pacemakers are devices placed for bradyarrhythmias, and they remain the only effective treatment for ameliorating symptomatic bradycardia due to sinus node dysfunction (e.g., sick sinus syndrome) or a failure of impulse propagation (e.g., complete heart block). Advances in technology and understanding of cardiac conduction physiology have led to the development of more physiologic pacing. Pacemakers have become sophisticated at maintaining the normal atrial-ventricular activation over various heart rate ranges, varying the heart rate in response to metabolic demands, and preserving natural ventricular activation. Pacemakers have many additional features that correspond to the changing needs of patients throughout

the day, including rate responsiveness to increase pacing during times of increased physical exertion and sleep functions to decrease pacing rate during times of rest. Standard pacemakers have either one or two (atrial and ventricular) leads. A patient is considered to be pacemaker dependent if they suffer significant symptoms or even cardiac arrest upon the cessation of pacing.^{4,5}

The dual chamber pacemakers are capable of pacing and sensing in both the ventricle and the atrium. Such capability permits the pacemaker to ensure not only an adequate ventricular rate, but to preserve the atrial contraction before each ventricular contraction. These pacemakers guarantee a minimum atrial rate and also ensure that a ventricular contraction occurs within a specified amount of time after each atrial contraction. Limitations on ventricular rate are usually built in the circuitry and are programmable. Most pacemakers have the capability of varying the pacing rate. In the rate adaptive mode, the pacemakers sense the patient's level of activity and accordingly adjust the pacing rate using sensors that are typically piezoelectric and detect body motion transmitted from underlying muscles. Another method of determining the presence of physical activity utilizes detection of the respiratory rate and/or volume using bioimpedance sensors.

All pacemakers generate a pulse of current to depolarize a small region of the myocardium; the wave then spontaneously spreads to the rest of the myocardium. The pacing capture threshold is the minimum electrical energy needed to consistently capture the heart outside of the refractory period and is determined by (1) the intrinsic excitability of the myocardium, (2) the current density at the electrode-tissue interface, and (3) the duration of the electric pulse.

The North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG) initially published a generic pacemaker code (NBG code) in 1987. In 2002 the NBG code was subsequently revised (Table 38.1).⁶ Common perioperative pacing modes include dual chamber, adaptive-rate pacing (DDDR), dual chamber pacing without atrium synchronous ventricular pacing (DDIR), and dual chamber asynchronous pacing with no rate modulation or multisite pacing (DOO).

1. DDDR pacing defines a pacemaker programmed to pace the atrium and/or ventricle, sense the atrium and/or ventricle, inhibit or trigger pacing output in response to a sensed event, and have a rate responsive sensor that is able to alter paced rates due to changes in perceived metabolic demand. DDDR is a very common program configuration for patients with sick sinus syndrome and/or heart block.

TABLE 38.1 2002 NASPE/BPEG Generic Code for Antibradycardic Pacing

Position	I	II	III	IV	V
Description	Chamber Paced	Chamber Sensed	Response to Sensing	Rate Modulation	Multisite Pacing
Possible designations	D = Dual (A+V) A = Atrium V = Ventricle O = None	D = Dual (A+V) A = Atrium V = Ventricle O = None	D = Dual (T+I) T = Triggered I = Inhibited O = None	R = rate modulation O = None	D = Dual (A+V) A = Atrium V = Ventricle O = None

2. DDIR is a common pacing mode for patients with supraventricular tachyarrhythmias (SVTs). If a patient is set to DDD, the ventricle response depends on the atrial rate. During SVT in DDD mode, rapid ventricular pacing may occur. In DDI, the pacemaker paces and senses both the atrium and ventricle; however, the device will not pace the ventricle at an identical rate if the patient has SVT. The response to a fast rate in the atrium will lead to inhibition of pacing in the ventricle—hence the *I* in the third designation. Most modern pacemakers have built-in automatic mode switching during episodes of SVT. They will switch from DDD to DDI to avoid SVT with a rapid ventricular response.
3. For some perioperative care, devices will be placed in an asynchronous mode like DOO. An asynchronous mode or nontracking mode will pace the atrium and ventricle at a set rate, regardless of the underlying rate and rhythm. This is advantageous in the perioperative environment in order to avoid the pacemaker from oversensing the monopolar electrocautery as intrinsic cardiac conduction. Asynchronous modes avoid oversensing (and under pacing) hearts that are pacemaker dependent and inhibited from pacing due to monopolar electrocautery (Table 38.2).

IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS

ICDs are implanted in patients for primary or secondary prevention of cardiac arrest. Primary prevention refers to ICD placement for patients who have not had any episodes of ventricular arrhythmias but who are at risk for future events. Secondary prevention refers to ICD placement for patients who have had prior ventricular arrhythmias. There is strong evidence that ICDs implanted for primary prevention improve mortality in high-risk patients, including patients with a left ventricular ejection fraction less than 40% who are on optimal medical therapy.⁷⁻¹¹ For patients with ischemic and nonischemic cardiomyopathy, ICDs reduce mortality approximately 23% to 55%.^{12,13} Certain groups of patients do not benefit from

ICD implantation, including patients with recent myocardial infarction and patients who have received coronary artery bypass grafting.¹⁴ In addition, many of the patients in the decisive trials for ICD placement were younger (mean or median age between 58 and 67 years for the MADIT-II, CABG-PATCH, DINAMIT), whereas many patients currently receiving ICDs are over the age of 70.¹⁵

ICDs have four main functions. They sense atrial or ventricular electrical activity, classify these signals to various programmed “heart rate zones,” deliver tiered therapies to terminate ventricular tachycardia (VT) or fibrillation, and pace for bradycardia. All modern ICDs are pacemakers, and this has important perioperative applications. Although ICDs improve survival in many patients, unnecessary shocks are very detrimental. They are proarrhythmic, can lead to anxiety and depression, and decrease patient quality of life. Inappropriate shocks are common (30% to 50% of all shocks) as a result of inappropriate treatment of SVT, oversensing physiologic T waves, or lead fracture.¹⁶ The discrimination between VT and SVT is critical for ICDs to avoid inappropriate therapy. There are several methods by which ICDs discriminate between SVT and VT. Single chamber ventricular ICDs utilize ventricular–ventricular timing intervals and QRS morphology. Dual chamber ICDs use atrial–atrial timing intervals and the chamber of onset. Subcutaneous ICDs assess surface electrocardiogram (ECG). The sensitivity and specificity for VT detection by QRS morphology is more than 90%.¹⁶

ICDs terminate ventricular arrhythmias by either anti-tachycardiac pacing (ATP) or defibrillation. ATP terminates reentrant VT by blocking reentry and it terminates slow VT (<188 to 200) approximately 90% of the time. ATP is desirable because it reduces inappropriate shocks and prolongs battery life. For VT that is not terminated by ATP or for ventricular fibrillation (VF), defibrillation is the treatment of choice. The energy for defibrillation may be incrementally increased or set to maximum energy for each shock.¹⁷

TABLE 38.2 Common Pacemaker Settings

Examples	Chamber Paced	Chamber Sensed	Response to Sensing	Rate Modulation	Multisite Pacing	Common Clinical Utilization
AAI	Atrium	Atrium	Intrinsic atrial beat inhibits atrial pacing	None	None	Sick sinus syndrome with intact atrioventricular conduction
DDDR	Both	Both	Intrinsic beat will inhibit output; atrial beat will trigger ventricular pacing if lack of intrinsic ventricular beat	Present	None	Atrioventricular block
VVIRV	Ventricle	Ventricle	Intrinsic ventricular beat will inhibit ventricular pacing	Present	Multisite ventricular pacing	Heart failure with prolonged QRS
DOO	Both	None	None	None	None	Perioperative asynchronous setting to avoid electromagnetic interference

CARDIAC RESYNCHRONIZATION THERAPY DEVICES

CRT plays an important role in the management of heart failure and is becoming a device commonly encountered by anesthesiologists because of the large prevalence of heart failure in this country. These devices are indicated in select patients with heart failure, systolic dysfunction, and a prolonged QRS. Conduction abnormalities are frequently seen in systolic heart failure, with approximately 25% to 40% of these patients having a prolonged QRS complex (>120 ms).¹⁸ In these patients, cardiac depolarization spreads slowly through the myocardium without a healthy Purkinje conduction system, leading to intraventricular dyssynchrony. During intraventricular dyssynchrony, the left ventricular (LV) septal wall contracts earlier than the lateral wall, which leads to less efficient ejection from the left ventricle in addition to decreased diastolic filling. The goal of CRT is to restore synchronous contraction of the left ventricle and to optimize timing of LV and right ventricular (RV) ejection. This is accomplished through biventricular pacing using a standard RV lead and an LV lead placed adjacent to the lateral wall via the coronary sinus. Biventricular pacing in the right ventricle and the left ventricle leads to improved hemodynamic variables, including systolic blood pressure, stroke volume, cardiac output, and rate of rise of LV pressure (dP/dt). In contrast to pharmacologic means of improving systolic function, CRT improves cardiac performance with reductions rather than with increases in myocardial metabolic demand. In addition, CRT has been shown to improve mitral regurgitation (MR) and New York Heart Association (NYHA) function class because of reverse ventricular remodeling over time.¹⁹ Standard indications for CRT are LV ejection fraction (LVEF) less than 35% with QRS greater than 120 ms, sinus rhythm, and NYHA class III or IV after optimal medical therapy. Left bundle branch block is the most common conduction abnormality in patients undergoing CRT. Approximately 30% of patients meeting selection criteria for CRT do not respond to biventricular pacing. Risk factors for failure to respond to CRT include ischemic cardiomyopathy, sustained VT, severe MR, and dilated LV cavity.²⁰ CRT has been shown to reduce mortality, heart failure symptoms, and also heart failure hospitalizations. Patients with CRT should be considered pacemaker-dependent because of the constant pacing they undergo to synchronize the ventricle.

Perioperative Considerations

PREOPERATIVE ASSESSMENT

As perioperative physicians, the preoperative assessment of patients with CIEDs is critical to safe and time-efficient care. The key to the preoperative assessment is timely communication between the anesthesia provider and the CIED team that usually manages the device programming and function. The CIED team may include a cardiologist, an electrophysiologist, and/or a physician extender such as a nurse or nurse practitioner. The CIED team needs to know various information about the surgical case and postoperative disposition in order to create an individualized care plan for

the patient. The anesthesiology provider must communicate the specifics of perioperative care to the CIED team in order to ensure a proper plan specific to that patient and the surgical case. Unfortunately, there is no single prescription for CIED management that can be applied to all patients coming for surgery. In addition, it is not recommended that an industry-employed allied profession determine the perioperative plan for a particular CIED.²¹

The perioperative team must convey to the CIED team important information regarding the presence of electromagnetic interference (EMI), the likelihood of cardioversion or defibrillation, patient position, location of surgery that may damage or encroach upon the CIED leads, and postoperative disposition. The CIED team must communicate with the anesthesiology team the date of last interrogation, the type of CIED device, the indication for CIED placement, the battery life, pacemaker dependence, and magnet response. Each of these vital perioperative parameters are discussed in detail in this chapter.

The goal of a thorough preoperative evaluation between the CIED and anesthesia teams is to avoid CIED complications in the perioperative period, such as damage to the device, inability of the device to deliver pacing or shocks, lead-tissue interface damage, changes in pacing behavior, electrical reset to the backup pacing mode, or inappropriate ICD therapies. Any of these complications can lead to poor patient outcomes, including hypotension, arrhythmias, and myocardial ischemia.

PREOPERATIVE INFORMATION TRANSFER

The ACCF/AHA/HRS 2011 guidelines emphasize that a team approach should be utilized when caring for patients with CIEDs. Patients coming for surgery with CIEDs do not need routine interrogations prior to surgery unless there has been a change in clinical presentation. For patients with a pacemaker, they should have an interrogation report within the last 12 months; patients with an ICD or CRT should have a report within the previous 6 months. The different time courses for interrogations between pacemakers and ICDs and CRT devices reflect the overall cardiovascular comorbidities of these respective patient populations. By definition, patients with ICDs and CRT devices have decreased systolic function and heart failure, and they are more likely to have clinical decompensation compared to patients with isolated pacemakers. Worsening cardiovascular function might negatively impact CIED performance. The preoperative evaluation begins with general considerations, such as establishing whether a patient has a CIED and defining the type of device. A focused history and physical, including rhythm strips and ECGs, can usually answer important basic questions about the patient's CIED. After these are completed, it is important to define the type of the device to be used. If the patient has been seen by the CIED team, they will leave a detailed note.

Within the interrogation report, it is important to note the type of device. Patients with pacemakers have different clinical risk profiles compared with those with ICDs and CRTs. In addition, the goals of perioperative management for these devices are different. Most pacemakers encourage intrinsic cardiac conduction because this maintains atrial–ventricular synchrony, LV–RV synchrony, and septal

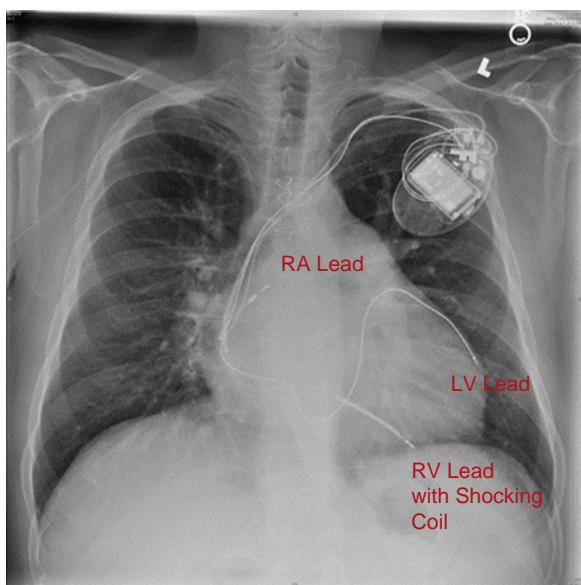


Fig. 38.1 Chest radiograph: the pulse generator is located in the left deltopectoral groove. There are three leads visualized: a lead in the right atrium (RA), a lead in the left ventricle (LV) via the coronary sinus, and a lead in the right ventricle (RV). The RV lead has a thicker diameter toward the tip, indicating it is an implantable cardioverter-defibrillator lead.

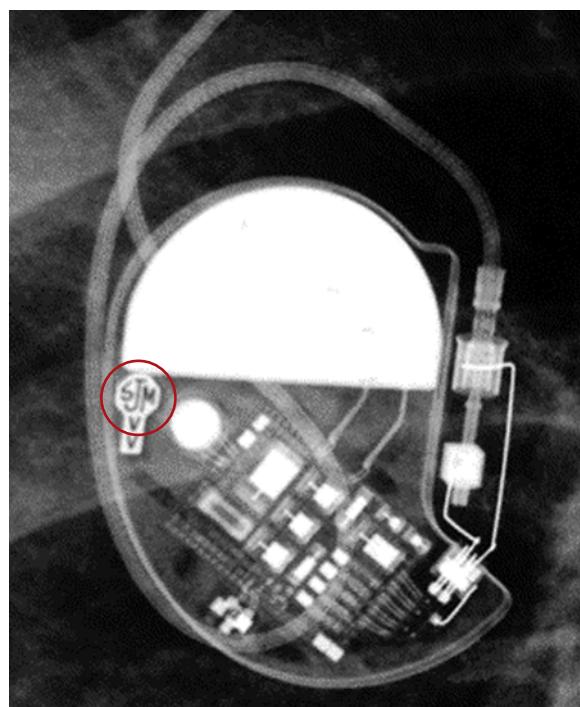


Fig. 38.2 Chest radiograph of the cardiovascular implantable electronic device generator. The letter designation on the generator allows the physician to identify this manufacturer as St. Jude Medical.

LV-lateral LV synchrony. Although DDD pacing of both atrium and ventricle in a dual-chamber pacemaker maintains atrioventricular (AV) synchrony, it creates RV-LV dysynchrony and LV-LV dyssynchrony because pacing begins in the right ventricle and crosses the septum before activating the LV lateral wall. Preserved synchrony optimizes stroke volume, which is desirable during the perioperative environment. Therefore, in the perioperative environment, it is advantageous to maintain intrinsic cardiac conduction as much as possible if the surgical procedure and use of EMI permits. In contrast, CRT devices attempt to ensure pacing 100% of the time in order to optimize stroke volume.

The manufacturer and model should be noted, along with the indication for placement of the CIED, in order to troubleshoot problems. Theoretically all patients with CIEDs should carry identification cards that identify the type of CIED, the manufacturer, the model, and date of implantation. In practice, many patients do not carry their CIED cards, and it is left to the anesthesia provider to identify these parameters. There are several methods for obtaining this information. Frequently an interrogation note from the CIED team will identify all these parameters. Occasionally, patients present for elective surgery without CIED team communication. In this subset of patients, there are several options. One option for identifying the type of device and manufacturer is the use of chest radiographs (Fig. 38.1). Chest radiographs can identify pacemakers versus ICDs, due to the increased thickness of the shocking coil in ICDs. Chest radiographs can also identify CRT devices due to the lead in the coronary sinus. With some training, the manufacturer can also be identified via chest radiograph (Fig. 38.2). In situations where the patient does not have his or her card, or there is no CIED note and there is no chest radiograph available or it cannot be interpreted, then contact with the CIED manufacturers can be initiated, since they all keep clinical records of their

patients. Communicating directly with the manufacturer can disclose information about the type of CIED, date of implantation, and original settings. Unfortunately, current information about pacemaker dependence and battery life is not usually available by phone.

Battery longevity is important to consider as an anesthesia provider. Three months of battery duration is reasonable in the perioperative period. This time point is chosen because battery life depends on the amount of pacing and the number of shocks delivered for pacemakers and ICDs, respectively. The burden of pacing in the perioperative period may be very different than the patient's normal requirements. Sinoatrial (SA) and AV nodal suppressing agents such as opioids, beta-blockers, and calcium channel blockers administered in the hospital may increase pacing requirements; therefore it is prudent to have several months of battery life remaining. In addition, there may be unexpected postoperative complications leading to the increased duration of hospitalization. If a patient is presenting for elective surgery with a diminished battery life, the generator should be replaced prior to the surgery.

It is important to establish if the patient is pacemaker-dependent, which is defined as the absence of a perfusing rhythm without pacing. In order to establish pacemaker dependence, a history and chart review may identify an episode of bradyarrhythmia leading to syncope. In addition, for patients with refractory SVT, they will occasionally have an AV nodal ablation. This is a therapeutic procedure that establishes complete heart block to eliminate rapid ventricular response to SVT. This patient population is pacemaker-dependent. Patients with CRT devices can be considered pacemaker-dependent even if they have a normal sinus rate and intact AV conduction due to

TABLE 38.3 Summary of Information Anesthesia Team Should Identify Prior to Surgery

Perioperative Considerations	Comments
Last CIED interrogation	12 months for pacemakers 6 months for ICD/CRT
Type of device	Pacemaker ICD CRT Loop recorder
Manufacturer	Obtained via patient card, history, CIED note, chest radiograph, or telephone call to manufacturer
Battery life	Suggested >3 months
Pacemaker dependence	Underlying rhythm CRT devices should be paced 100%
Magnet response	Differs by manufacturer and battery life
Pacing threshold	Should be set several times higher for safety

CIED, Cardiac implantable electronic devices; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter-defibrillator.

the hemodynamic deterioration they suffer with dyssynchrony of the RV-LV and LV-LV. Pacemaker dependence can be established by the CIED team preoperatively. They can decrease the pacing rate in a stepwise fashion to search for intrinsic electrical activity. If no intrinsic rate is present around 40 to 45 beats/min or the patient develops symptoms, the patient may be considered pacemaker-dependent. Some patients who are not pacemaker-dependent may become pacemaker-dependent in the perioperative period due to SA and AV nodal blocking agents; therefore, it is important to be prepared to address this clinical scenario. If the patient is deemed to be pacemaker-dependent, a secondary method for pacing the patient should be considered in the event of a pacemaker failure. Alternative methods of pacing patients intraoperatively include transesophageal pacing, transcutaneous pacing, or transvenous pacing through a pacing pulmonary artery catheter or through a temporary transvenous pacing wire. Transesophageal pacing activates the left atrium, which is in close proximity to the esophagus, so this type of pacing depends on intact AV node conduction. It will not work for heart block. Transcutaneous pacing depends on correct pad position and high pacing outputs, which can be very uncomfortable in a nonsedated patient. Transvenous pacing and pacing pulmonary artery catheters can be time consuming, and they are not ideal choices for emergent pacing. Whatever method is chosen, it is important to have the necessary equipment and support organized and available prior to beginning the procedure. A summary of important information for the anesthesia team to identify is summarized in Table 38.3.

ELECTROMAGNETIC INTERFERENCE

EMI in the preoperative setting can interfere with the functioning of both pacemakers and ICDs.^{22,23} EMI may inhibit pacing, damage the lead-tissue interface, damage the pulse

generator, and/or trigger an electric reset mode, particularly if the source of EMI is within 6 inches (15 cm) of the CIED generator.²⁴ Although there are several potential sources of EMI, the most commonly encountered source is monopolar electrocautery. Monopolar electrocautery is the most frequently used type of electrocautery because it has both cutting as well as coagulation capabilities. Monopolar electrocautery creates a current that passes from the probe to the tissue and returns through the patient to the return pad to create an electrical circuit. Bipolar electrocautery is usually not a concern²⁵, the electrical current field is small and limited to the two poles at the end of the electrode. Unfortunately, bipolar is less frequently used compared with monopolar electrocautery and is usually reserved for neurosurgery, ophthalmology, and head and neck procedures. Bipolar electrocautery does not cause EMI of CIEDs unless it is applied directly to a CIED, and there are minimal clinical circumstances where that would be indicated. The vast majority of electrocautery encountered in the operating suite is monopolar due to its versatility.

Modern CIEDs have evolved to produce better shielding from EMI, and adverse events from EMI are much less common with improvements in CIED technology.²⁴⁻²⁷ Shielding reduces the potential to induce oversensing in the CIED. Oversensing in a pacemaker means the pacemaker “sees” the EMI-generated artifacts as intrinsic cardiac electrical activity and therefore does not initiate a paced rhythm. Such failure to initiate a paced rhythm (inhibition of the pacemaker) can compromise hemodynamics in a pacemaker-dependent patient. If oversensing occurs in an ICD, EMI may be misinterpreted by the ICD as a malignant tachyarrhythmia, which may cause the patient to receive an inappropriate shock. It is important to note that it is not only the location of monopolar electrocautery that is critical to proper CIED function, but it is also the location of the grounding pad that is critical to avoid CIED malfunction. The HRS/ASA Expert Consensus Statement suggests that due to the decreased likelihood of EMI-related interference seen when surgery is below the umbilicus, the patient should proceed to surgery with no magnet application to the device or reprogramming. This assumes that the monopolar electrocautery grounding pad or return pad is also placed below the level of the umbilicus. If current is traveling above the level of the umbilicus, there is significant risk of EMI of the CIED. For procedures above the umbilicus with no EMI from the surgical procedure, the patient may also proceed for surgery without routine magnet use or device reprogramming. For all patients presenting for surgery with CIEDs, there should be a magnet available with the magnet function of the device known in case of a change in the surgical plan or unexpected EMI. Magnet function is discussed in the next section.

MAGNETS

Magnet applications for CIEDs were originally intended to investigate battery life of the device, and they were not created for perioperative management of CIEDs, although that is probably their most common utilization at this time. When magnets are applied to the pacemaker, the effect on the device depends on the manufacturer, in addition to the battery life. For example, when a magnet is applied

to a Medtronic pacemaker, the device will pace asynchronously. If the Medtronic device is a single-chamber pacemaker, it will pace that single chamber asynchronously. If the Medtronic device is a dual-chamber pacemaker, it will pace both chambers asynchronously. However, the rate at which the pacemaker will pace depends on battery life. If the Medtronic pacemaker has adequate battery life, the device will pace at 85 beats/min. However, if the battery life is at the elective replacement interval, the device will pace at 65 beats/min. The change in rate is binary, meaning that the rate goes from 85 to 65 beats/min when the battery enters its elective replacement interval. Different manufacturers have different responses to magnet application. St. Jude pacemakers will pace asynchronously during magnet application if the magnet response is turned on. It is important to note that magnet application is programmable—that is, with some devices, magnet application will have no effect. The effect of magnet response is an important piece of information to obtain from the CIED team. For a St. Jude pacemaker with magnet response programmed on, magnet application will program the device to pace asynchronously at a rate of 100 beats/min if the battery life is normal or a rate of 85 beats/min if the device is at its battery elective replacement interval. Unlike Medtronic devices, the rate change from 100 to 85 beats/min is not binary; it is a gradual reduction from 100 to 85 beats/min.³

Magnets continue to be very popular in everyday practice for the management of CIEDs. This is a relatively easy way to render the pacemaker asynchronous, with the added benefit of the device reverting back to its programmed settings once the magnet is removed. Importantly, the asynchronous rate provided by magnet placement may not be appropriate for the patient for a particular procedure. At times, patients require a higher heart rate in order to increase tissue oxygen delivery. To the contrary, most magnet rates are typically around 90 beats/min, which may not be appropriate for a large subset of patients—that is, those with aortic stenosis or coronary artery disease. These variables make it important to confirm the magnet effect on each individual patient's device before the procedure, and to have an individualized plan for each patient.

The choice of reprogramming versus magnet application largely depends on the type of surgery, patient position, accessibility of a programmer, and knowledge of magnet function of a particular patient's device. It should be emphasized that a magnet has different effects when applied to a pacemaker or to an ICD. In a majority of pacemakers, magnet application induces an asynchronous mode. Magnet application to an ICD inhibits tachyarrhythmia detection, thus preventing therapy (i.e., delivery of shocks). However, it does *not* change the mode of the underlying pacemaker. Therefore, a magnet placed over an ICD will not induce asynchronous mode in the underlying pacemaker. For patients with an ICD who are pacemaker dependent, reprogramming is the preferred option if EMI is a significant concern. One advantage of reprogramming are that if the patient is not in the supine position, it may be difficult to maintain the magnet in a location over the device to render it asynchronous. This is particularly true in the prone position. If the patient has a normal sinus rate, adequate chronotropy, and intact AV node conduction, magnet application may compete with the patient's own

heart rate if surgical stimulation increases the sinus rate. This can lead to significant arrhythmias caused by depolarization of the ventricle by the pacemaker during a refractory period, due to intrinsic electrical activity. The principal disadvantage of reprogramming versus magnet application is that the changes made with the programmer are not as easily reversed. If the patient develops an arrhythmia or has an increased sinus rate, it may be difficult to reprogram the device intraoperatively, depending on programmer availability and CIED team presence. The other disadvantage of reprogramming a CIED compared with magnet application involves the human factor. Failure to re-enable tachyarrhythmia therapy following surgery can lead to catastrophic consequences for patients with CIEDs. It is important to understand the risks and benefits of magnet application versus reprogramming, and create a perioperative plan that suits the patient.

ELECTROMAGNETIC INTERFERENCE DAMAGE TO THE CARDIAC IMPLANTABLE ELECTRONIC DEVICE

EMI is an important consideration for anesthesiology providers because of oversensing. It is uncommon for modern CIEDs to be damaged by EMI from the operative environment; however, some particular complications will be discussed. Monopolar electrocautery that is done in very close proximity to the pulse generator may directly damage the myocardium at the interface with the CIED lead. This can trigger arrhythmias or loss of pacing capture due to increases in pacing thresholds.²⁸ Therefore, it is recommended that bipolar electrocautery should be used when electrocautery is performed in close proximity to the pulse generator. Another complication that may happen due to ionizing radiation is device reset.^{29,30} It is uncommon for even monopolar electrocautery to cause reset. In the perioperative setting, this very rarely occurs when an energy surge directly contacts the pulse generator, resulting in a major hardware/software failure. The reset mode is unique to each manufacturer and serves as a safety backup in the case of catastrophic failure. Unfortunately, magnet application and device reprogramming will not prevent electrical reset. The best way to prevent reset is to ensure that EMI is as far as possible from the pulse generator (ideally >15 cm). If a patient's CIED does go into reset mode, the pacemaker will commonly revert to a ventricular demand pacing (VVI) mode between 65 and 70 beats/min. Magnet response in reset mode will also vary among manufacturers. ICDs will have a wide range of rate cutoffs for VT detection in the reset mode. ICD pacing will be typically in a VVI mode again around 65 to 70 beats/min. Electrical reset should prompt consultation with a CIED team to ensure proper device functionality.

INTRAOPERATIVE MANAGEMENT

The intraoperative management of CIEDs naturally evolves from a thoughtful preoperative plan. A pathway for management of CIEDs during nonemergent surgery is outlined in Fig. 38.3. Pacemaker patients who are pacemaker-dependent and who are having surgery with monopolar electrocautery within 15 cm of the generator should have their

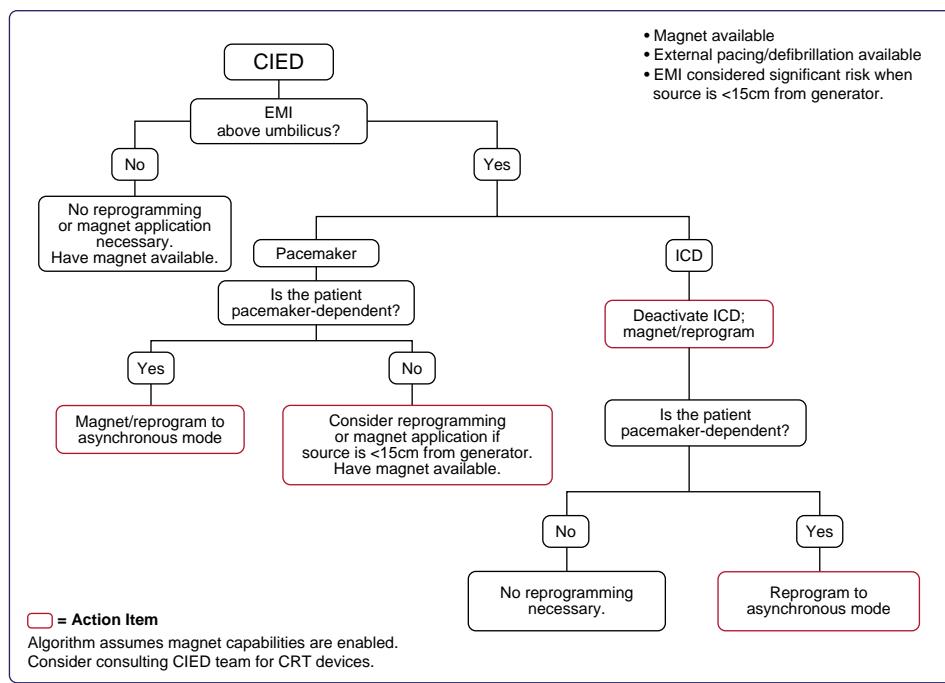


Fig. 38.3 Cardiovascular Implantable Electronic Device pathway for nonemergent surgery. CIED, Cardiac implantable electronic devices; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter-defibrillator; EMI, electromagnetic interference. Thompson A, Neelankavil JP, Mahajan A. Current Anesthesiology Reports, 2013, Vol 3, Issue 3, 139–143. (Courtesy Dr. Annemarie Thompson.)

device reprogrammed to avoid pacemaker oversensing of electrocautery as intrinsic cardiac function. This could lead to underpacing and hemodynamic collapse in pacemaker-dependent patients. For patients in the supine position with known magnet function who have magnet rates appropriate for their concurrent comorbidities, placement of a magnet over a pacemaker is a reasonable approach for patients at risk for monopolar electrocautery oversensing. Whether the prescription plan is reprogramming or magnet application, the rate responsive features of the device should be disabled. The rate response is the fourth letter in the 2002 NAPSE guidelines and refers to increasing the heart rate above the lower rate limit when the patient has increased metabolic demand (e.g., exercise). Rate response sensors commonly use minute ventilation or thoracic impedance to increase rate, and both may be affected intraoperatively. Patients on mechanical ventilation will see increases in heart rate if the minute ventilation is increased with these sensors. For patients with thoracic impedance sensors, electrocautery may lead to increases in heart rate. Although these increases in heart rate may be transient, they may be detrimental for patients in whom strict heart rate control is optimal. In addition, these changes in heart rate may be distracting for the anesthesia provider, who may perceive the heart rate fluctuations as CIED malfunction. Patients with ICDs should have their tachyarrhythmia therapies disabled to avoid inappropriate shocks from monopolar cautery oversensing by either reprogramming or magnet application if EMI (or the return pad) is used above the umbilicus. Patients with ICDs who are pacemaker dependent with close EMI to the generator are a special group that must have their devices reprogrammed. Magnet application is not a reasonable strategy in these patients, since the magnet will not make the pacemaker asynchronous. Patients who have CRT devices are another special classification of

patients presenting for surgery. Again, most dual-chamber pacemakers are programmed to minimize RV pacing. CRT devices are programmed to achieve ventricular pacing 100% of the time to improve cardiac output, systolic, and diastolic function. Even though CRT devices are pacing the ventricle 100% of the time, they will not have the characteristic wide complex QRS found in RV pacing alone. Programming the CRT device to an asynchronous mode above the intrinsic rate is a reasonable intraoperative management strategy. Since almost all CRT devices are ICDs, magnet application to the device will deactivate tachyarrhythmia therapy but will not make the CRT device asynchronous. It is critical to reprogram CRT devices to an asynchronous mode if EMI is used above the umbilicus. Brief periods of monopolar electrocautery may be tolerated in patients with CRT devices who have a reasonable sinus rate and normal conduction if the device is not reprogrammed. However, prolonged use of monopolar electrocautery will lead to hemodynamic deterioration due to a loss of synchrony between the RV and LV and the LV septal and lateral walls.

Intraoperative monitoring mirrors standard American Society of Anesthesiology (ASA) guidelines. Plethysmographic pulse oximetry is invaluable for patients with CIEDs undergoing surgery because it is the best clinical indicator that the pacemaker is capturing and creating cardiac output. With monopolar electrocautery, monitoring for pacemaker capture with ECG can be challenging. Due to ECG technology, the ECG rate will sometimes double count the heart rate due to counting of a pacemaker spike and the resulting QRS complex. Double counting of the heart rate may lead to erroneous patient management. In addition, pacemaker spikes may be difficult to see due to their low voltage. Pacemaker spikes may be accentuated with various patient monitors; however, they may inaccurately identify a pacemaker spike. It is important to ensure that each pacemaker

spike is capturing the myocardium. Ensuring the paced rate of the device is the same as the pulse oximetry rate ensures proper capture of the myocardium and appropriate cardiac output. While arterial lines accurately display pacemaker capture and cardiac output, patients with CIEDs do not need routine arterial line placement unless patient acuity or surgical complexity deems it necessary.

If a patient requires central venous access or a pulmonary artery catheter, it is important to be mindful of CIED leads placed within 1 to 2 months prior to the planned surgical procedure. The risk for dislodging new leads in the right atrium and right ventricle is higher until the leads become fixed within the myocardium. Patients with CRT devices have a coronary sinus lead that can be more easily dislodged when compared with a chamber lead, since there is no mechanism of lead fixation in the coronary sinus.

POSTOPERATIVE MANAGEMENT

Patients with CIEDs should be appropriately monitored in a clinical environment that is tailored to their postoperative risk. There does not need to be escalation of postoperative disposition based on the presence of a CIED alone. The patient acuity and operative course should be the major drivers for postoperative disposition. For example, patients presenting for outpatient surgery require routine postoperative continuous monitoring until they meet discharge criteria. Most patients with CIEDs do not require routine interrogation following surgery; however, there are notable exceptions. It is reasonable to evaluate patients with CIEDs within 1 month of their surgery on an outpatient basis.³ For some patients, they must have their device interrogated prior to leaving a monitored setting, whether that is the postanesthesia care unit or a monitored bed. Patients who have had their tachyarrhythmia therapy disabled for surgery must have their device reprogrammed and their tachyarrhythmia therapy re-enabled prior to leaving a monitored setting. In addition, patients who have undergone hemodynamically significant procedures with large volume shifts should have their device interrogated to ensure proper sensing and capture. Patients receiving cardioversion, defibrillation, and therapeutic radiation are at risk for device reset, and should have interrogations as well in the immediate postoperative period.³ Patients who fall into high-risk categories are more likely to have a change in CIED functionality. Part of the preoperative communication between the CIED team and the anesthesia team should address the need for CIED interrogation postoperatively. Identifying patients who need CIED interrogation prior to discharge avoids lapses in communication in the postoperative period.

EMERGENCY PROTOCOL

Patients with CIEDs may present for urgent or emergent surgery. In these instances, the bidirectional communication between the CIED and anesthesiology teams may not be feasible. When these clinical scenarios present themselves, it is important for the anesthesiology team to identify several components of the CIED in a time efficient manner (Table 38.4). As described previously, the anesthesiology team can get this information from the patient's CIED wallet card, medical records, or CIED team note, or from the

TABLE 38.4 Cardiovascular Implantable Electronic Device Information for Emergency Surgery

Important Information to Obtain for Emergency Surgery	How to Obtain Information
Type of device	Wallet card Medical record Chest radiograph Manufacturer 1-800 number Device programmer
Pacemaker dependence	Medical record Electrocardiogram Rhythm strip Device programmer
Risk of electromagnetic interference	Discussion with surgeon
Magnet function	Medical record Device programmer

TABLE 38.5 Manufacturer Contact Information

Manufacturer	Phone Number
Medtronic	1-800-633-8766
St. Jude	1-800-722-3423
Boston Scientific	1-800-227-3422
Biotronik	1-800-547-0394

chest radiograph. If these methods do not provide pertinent information, the anesthesiology team should call the device manufacturer to obtain basic information regarding the type of device and date of implantation (Table 38.5).

During an emergency procedure, it is important to establish whether the patient is pacemaker dependent. In the absence of a CIED note, a 12-lead ECG or rhythm strip can be examined to look for pacemaker spikes. If the majority of QRS complexes are preceded by a pacing spike, the patient should be treated as pacemaker dependent. The risk of EMI to the CIED should be established using the criteria previously presented, including the use of monopolar versus bipolar electrocautery, in addition to the location of EMI and the return pad. For pacemaker patients who are pacemaker dependent, it is reasonable to use a magnet for surgeries requiring EMI above the umbilicus. However, it is important to remember that a minority of pacemakers will have the magnet function turned off. Therefore, the anesthesiologist should monitor the pulse oximeter to ensure that there is no oversensing of the EMI leading to a lack of pacing in pacemaker-dependent patients, even when the magnet is applied to the device. For patients who are not pacemaker dependent, it is reasonable to have a magnet available in case the patient requires asynchronous pacing. For patients with ICDs, defibrillator pads should be placed on the patient during emergency procedures. Magnet application should be applied for cases with EMI above the level of the umbilicus. Since this will not render the pacemaker asynchronous, if monopolar EMI is used above the umbilicus, it is critical for the surgeon to use short bursts (<5 seconds) of monopolar electrocautery to avoid pacemaker

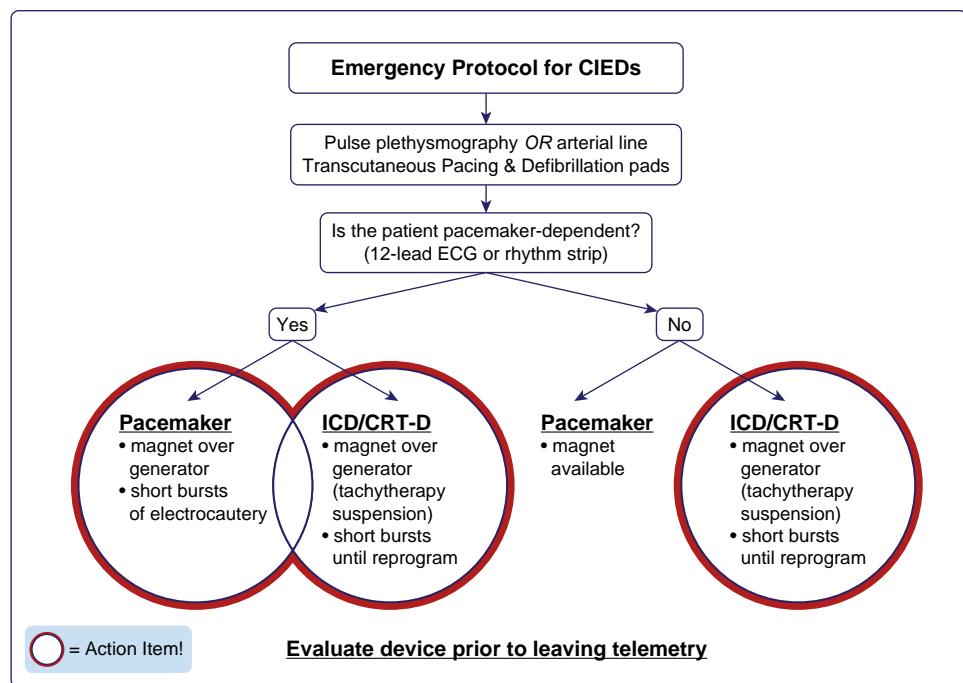


Fig. 38.4 Cardiac implantable electronic device (CIED) pathway for emergency surgery. CRT, Cardiac resynchronization therapy; ECG, electrocardiogram; ICD, implantable cardioverter-defibrillator. (Courtesy Dr. Annemarie Thompson.)

oversensing. Interrogation of the CIED under the direction of a physician knowledgeable in the function of these devices should be done as soon as possible. The emergency protocol is described in the HRS/ASA Expert Consensus Statement. A suggested emergency algorithm is shown in [Fig. 38.4](#).

It may be challenging for anesthesiology providers to feel comfortable reprogramming CIEDs in the perioperative environment. Having a structured CIED team that is willing to work with anesthesiologists is a great way to learn how to reprogram devices. Even if one does not feel comfortable reprogramming CIEDs in the perioperative environment, it may be very important for the anesthesiologist to know how to turn on a programmer to obtain important information about a device during an emergency procedure. Interrogating a device and simply reading the home screen can provide critical information regarding the type of device (pacemaker, ICD, CRT), the pacemaker mode, histograms of percentage of time the patient is being paced, battery life, and lead impedance. This is relatively easy to do if one has access to a device programmer in one's institution. Sample images of home screens are shown in Fig. 38.5.

SPECIFIC SURGICAL PROCEDURES

The general perioperative recommendations as outlined before cover the majority of clinical scenarios encountered by anesthesia providers caring for patients with CIEDs. However, there are specific surgical procedures that convey increased risk for CIED malfunction or damage due to high therapeutic energy transfer to the CIED.

Cardioversion

External cardioversion may occur as a planned procedure or as part of advanced cardiovascular life support (ACLS)

to treat unstable arrhythmias. Historically, there was increased concern for CIED function because of the use of unipolar leads. With these older leads, the current traveled from the generator (one pole) to the tip of the lead (second pole). There were reports of loss of capture and electrical reset when the cardioversion pads were placed in an anterior and lateral orientation.^{31,32} With newer leads, which have a bipolar configuration (both poles located in the tip of the lead), it is uncommon for cardioversion to affect CIED function. In a study with 44 patients, no CIED malfunction was observed during cardioversion with an anterior–posterior pad orientation with the anterior pad more than 8 cm from the CIED generator.³³

Radiofrequency Ablation

Many patients with CIEDs present to the electrophysiology laboratory for electrophysiology studies and catheter ablation. It is uncommon to have CIED malfunction with radio-frequency ablation (RFA); however, if complications occur, they involve electrical reset, oversensing, and undersensing due to energy delivery near CIEDs. Recommendations include avoiding direct contact between the ablation catheter and the pulse generator and leads, and keeping the path of radio-frequency current (electrode tip to current return pad) as far away from the pulse generator and leads as possible.^{34,35}

Lithotripsy

Extracorporeal shockwave lithotripsy is used for disintegrating renal calculi. From the initial use of lithotripsy, there was theoretical concern that the shockwaves and EMI would damage CIEDs; therefore, CIED presence was originally a contraindication to lithotripsy. As technology has evolved, current lithotripsy requires less energy transfer to the patient with improved focus of energy, as well to the renal calculi. Guidelines from Canada recommend a

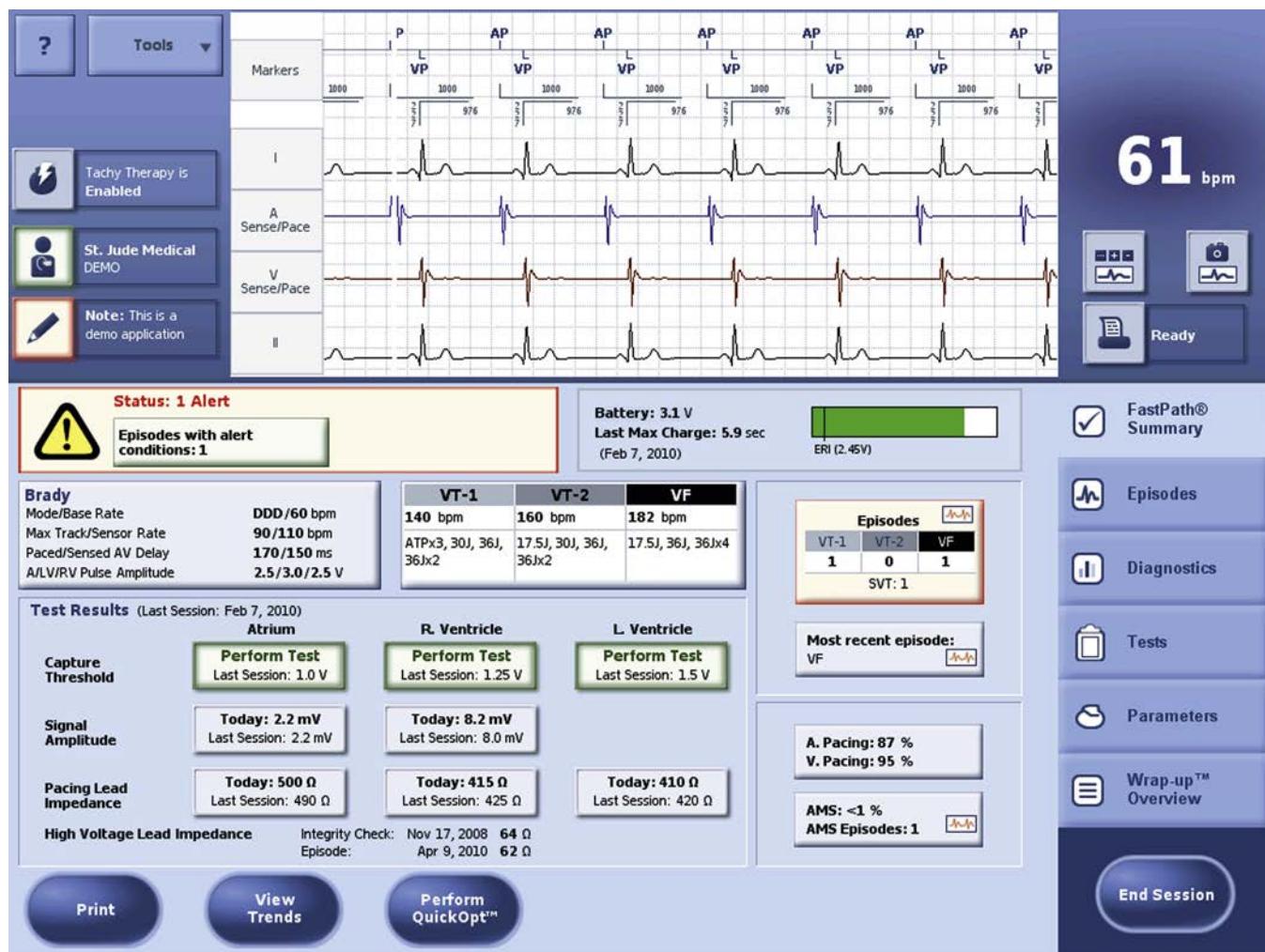


Fig. 38.5 Representative image of pacemaker programmer. Battery life, pacemaker settings, implantable cardioverter-defibrillator therapy, and percentage of atrial and ventricular pacing are readily found on the home screen.

distance of 15 cm from the device to treatment area and that abdominal CIEDs are contraindicated.³⁶ Another study suggests that with improved technology for lithotripsy, most CIEDs do not have complications; however, if complications arise, the device should be reprogrammed immediately by a member of the CIED team.³⁷

Radiation

Diagnostic radiation usually does not significantly affect CIEDs. On the other hand, therapeutic radiation can significantly affect CIED function if directed onto the pulse generator.³⁸⁻⁴⁰ If radiation therapy is performed, position the device outside the field of radiation. The concern with therapeutic radiation and CIEDs is that the circuit of the CIED may be damaged directly by the radiation. Direct radiation of CIEDs should be avoided, and accumulated doses should be kept under 5 Gy.³ In some instances, the pulse generator will require surgical relocation prior to commencing radiation.

Electroconvulsive Therapy

Electroconvulsive therapy (ECT) involves direct electric current to the brain for the treatment of depression. There are no reports of direct damage to the CIED; however, the concern is pacemaker oversensing of the electric current leading to underpacing in pacemaker-dependent patients. The medical

decision to use ECT should consider the duration of its application. If the therapy is going to be short (<5 seconds), it is unlikely that hemodynamically significant oversensing will occur. It is also unlikely that the patient will receive an inappropriate shock. If prolonged ECT exposure is required, the device should be placed in asynchronous pacing mode and antitachyarrhythmia function should be disabled. A discussion between the CIED team and the psychiatrist may facilitate treatment plans, since these patients usually undergo multiple treatments of ECT. ECT may lead to significant hemodynamic effects unrelated to the CIED, including sinus tachycardia from the seizure and ventricular arrhythmias. A magnet should be available for all patients undergoing ECT in the event that oversensing of myopotentials or inappropriate therapy for sinus tachycardia occurs.³

Endoscopy

Most upper and lower endoscopies do not use electrocautery, and for those cases, CIED management is straightforward. However, occasionally, electrocautery is used for some patients. There are case reports of inappropriate ICD therapy in these patients. When electrocautery is planned for endoscopy, the same guidelines as previously presented apply with regard to CIED team communication and the assessment of EMI risk to the CIED.

Emerging Technologies

The past couple of years have seen the advent of a new, leadless pacemaker. The impetus for the development of a leadless pacemaker stems from the fact that many pacemaker complications are related to the leads themselves, including lead fracture and infection. Currently, the Medtronic Micra is the only leadless pacemaker approved for use in the United States. The Micra is a single-chamber device placed in the right ventricle via the femoral vein. Its modes include VVIR, VVI, VOO, and OVO. Because these devices are so new, there is very little data on how to manage these patients in the perioperative period. Because of their small size, these devices do not have a magnet sensor and thus will not respond to a magnet. It is recommended that these devices be reprogrammed to VOO mode to reduce oversensing when EMI is anticipated.⁴¹ It should be noted that these devices use the same programmer as their full-size counterparts.

A new type of ICD is now on the market, and its use continues to grow: the subcutaneous ICD (S-ICD) manufactured by Boston Scientific Inc. A driving force for the design of this pacemaker is similar to the Micra pacemaker. Transvenous systems such as the most common CIEDs on the market may be challenging in patients with difficult vascular anatomy. In addition, the removal of transvenous systems can be complicated in the setting of long-standing CIED placement, requiring a laser lead extraction. This system was initially approved in 2012 as defibrillation therapy and is being used in patients at risk for malignant ventricular arrhythmias who do not have a need for bradycardia pacing, or antitachycardia pacing to manage VT.⁴² Although this device is not able to provide long-term pacing, it is capable of pacing at 50 pulses/min for 30 seconds after a defibrillatory shock, should the patient become profoundly bradycardiac posttreatment.⁴³

The S-ICD consists of a pulse generator and a single subcutaneous lead. Both the pulse generator and the lead are implanted in the subcutaneous tissue and are extrathoracic.⁴⁴ Currently, the S-ICD can only be implanted in the left chest. The pulse generator is usually implanted between the anterior and midaxillary lines at the level of the sixth intercostal space. The lead is then tunneled medially from the pulse generator pocket to the xiphoid process and then superiorly along the left parasternal border.

Although different in its makeup, the S-ICD has the same response to a magnet as a traditional ICD. That is, magnet application over the pulse generator will turn off the antiarrhythmic features of the device, and removing the magnet will revert the device to its prior programmed state. A feature the S-ICD has that ensures the magnet is properly positioned is a beeping sound that indicates that arrhythmia detection and shock therapy have been suspended. If the beep is not heard with magnet application, the magnet should be repositioned over the device until a beep is elicited. Due to the location of the ICD at the midaxillary line, it is best to reprogram this device in the perioperative setting to turn off the antitachycardic function. When the device is reprogrammed, defibrillator pads should be placed on the patient.

Conclusions

Anesthesiologists should have a basic understanding of CIEDs and also the nuances of managing this subset of

patients in the perioperative period. As technology continues to evolve, and as the population lives longer and indications for CIED therapy continue to grow, perioperative physicians will encounter this patient population more frequently. It should also be noted that at times, trained CIED experts (cardiologists, manufacturer representatives) are not available, making it the anesthesiologist's duty as a true perioperative physician to be able to fully care for these patients.

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KEY POINTS

- There are four key principles of intraoperative neurologic monitoring.
 - The pathway at risk during the surgical procedure must be amenable to monitoring.
 - The monitor must provide reliable and reproducible data.
 - If evidence of injury to the pathway is detected, there must be some intervention possible.
 - If changes in the neurologic monitor are detected, and no intervention is possible, although the monitor may be of prognostic value, it does not have the potential to provide direct benefit to the patient from early detection of impending neurologic injury.
- There are few randomized prospective studies evaluating the efficacy of neurologic monitoring modalities.
- Anesthesiologists can improve the efficacy of monitoring by maintaining good physiologic homeostasis and stable levels of anesthesia during parts of the surgical procedure that place the nervous system at greatest risk.
- Based on clinical experience and nonrandomized studies, four practice patterns for use of neurologic monitoring have emerged:
 - There are procedures for which monitoring is recommended and used by most centers.
 - There are procedures for which monitoring is used frequently in some centers, but not in others.
 - There are procedures for which there is no clear clinical experience or evidence indicating that monitoring is useful at all (experimental use).
 - There are procedures in which monitoring is used selectively for patients believed to be at higher-than-usual risk for intraoperative neurologic injury.
- Good communication between surgeon, anesthesiologist, and neurophysiologist is essential to optimizing the utility of monitoring.

Neurologic monitoring during anesthesia care for a patient spans a wide spectrum of techniques, diverse procedures, and various intraoperative or even postoperative settings. Techniques for monitoring fall into two broad categories: techniques to assess metabolic integrity of the nervous system, which typically entail either global or regional determinations of blood flow or oxygenation; or techniques to assess functional integrity, which likewise may be global or focused on specific anatomic pathways or structures of the nervous system. Neurologic monitoring implies that the data for the assessment of the integrity of the nervous system are acquired on a continuous or frequent intermittent basis rather than just at the beginning and end of a procedure.

The procedures and settings in which neurologic monitoring is typically applied all share the characteristic that changes in the monitored parameters can be corrected or minimized by either modifying the surgical approach or manipulating parameters under the control of the anesthesiologist. Monitored procedures range from procedures where monitoring dictates the surgical approach, such as localization of the motor strip during tumor surgery or the neurologic examination during an “awake” craniotomy, to procedures that by their nature put parts of the nervous system at increased risk.

In many procedures that require neurologic monitoring, the anatomical target is also susceptible to drugs

administered in the course of an anesthetic. The anesthesiologist and the surgeon need to be aware not only of limitations inherent in individual monitoring techniques, but also of nonsurgical factors that influence the monitoring results. The monitoring approach ideally should anticipate nonsurgical factors by providing a degree of redundancy that helps distinguish a localized surgical trespass from a systemic event.

The application of neurologic monitoring to a surgical procedure may be organized in different ways. It may simply consist in setting up a piece of monitoring equipment, such as a facial nerve monitor that provides acoustic feedback to the surgeon. At the other end of the spectrum, it may require the dedicated services of a technologist and a neurophysiologist in addition to the surgical and anesthesia teams as would be the case during the mapping phase of the resection of a brain tumor. Between those extremes are a variety of delivery models for intraoperative neurophysiologic monitoring that may entail components of telemedicine. Regardless of the organizational structure, the utility of intraoperative neuromonitoring will depend on a shared understanding of surgical objectives, anesthetic constraints, and limitations of monitoring. This shared understanding will then need to be supported by open lines of communications by all involved, particularly during critical phases of an operation.¹

For some procedures, neurologic monitoring is a marker of the quality of care and is routinely employed because outcome data support its use. Examples include correction of scoliosis and resection of vestibular schwannomas. More frequently, the approach to monitoring is based on local conventions and surgical expectations. In this latter case, monitoring utility depends even more on a good understanding of the technique's capabilities and limitations by anesthesiologists, surgeons, and the intraoperative monitoring team; on good communication; and on their mutual collaboration to allow corrective action in the face of changing signals or to prevent false alarms that disrupt surgery.

This chapter first discusses individual monitoring modalities in isolation so that the clinician can appreciate the inherent strengths and weaknesses of each. Subsequent sections apply this information by describing suitable approaches to various clinical settings that combine and integrate individual techniques to optimize neurologic outcome for patients. The chapter ends with a brief discussion of how neurologic monitoring is believed to be useful today, and where more work is needed to determine whether monitoring has a role in surgical patients in the future.

Monitoring Modalities

MONITORS OF ADEQUACY OF NERVOUS SYSTEM BLOOD FLOW

Adequacy of cerebral blood flow (CBF) can be monitored by two principal methods. The first method assesses blood flow itself with the implicit assumption that "normal" flow provides adequately for the metabolic needs of the brain. The second approach assesses oxygen delivery either locally or globally with the implicit assumption that normal oxygen delivery at the site of measurement reflects adequate blood supply throughout the central nervous system (CNS). To illustrate the limitations imposed by such implicit assumptions, let us examine global or hemispheric CBF in the context of a patient's disease process.

In normal brain, values of hemispheric CBF of approximately 50 mL/100 g/min reflect adequate oxygen delivery for maintaining structural integrity and function. Values of less than 20 to 25 mL/100 g/min are associated first with failure of function and on further decrease with structural damage.² In neurosurgical patients, structural integrity and function may be altered by disease processes and anesthetics, which have an impact on the interpretation of measured CBF. A CBF of 40 mL/100 g/min in a patient in barbiturate coma after resection of an arteriovenous malformation may represent hyperemia (because metabolic demand is very low), whereas the same CBF in a patient with a mass lesion may reflect a modest decrease in cerebral perfusion pressure secondary to increasing intracranial pressure. Thus the clinical response to an abnormal value requires context and clinical judgement.

Global Blood Flow Monitoring Techniques (Noninvasive)

Intravascular Tracer Compounds. Direct measurement of CBF is possible by determining kinetics of wash-in and/or wash-out of an inert tracer compound, a method originally described by Kety and Schmidt.³ A widely used modern

variation of the same concept is the imaging of the first passage of intravascular contrast agents during computer tomographic or magnetic resonance imaging for determining loco-regional blood flow (Fig. 39.1). These techniques share the limitation of providing a snapshot of CBF in time instead of continuous assessment of flow over time.

Transcranial Doppler Ultrasound. Transcranial Doppler (TCD) ultrasound is a technique that infers CBF from measurements of the blood flow velocity in the large conducting arteries of the brain. The TCD probe transmits pulses of sound waves through the thin temporal bone in a variation of the pulsed wave Doppler technique with which anesthesiologists may be familiar from echocardiography. When these sound waves are reflected off the red blood cells back toward the TCD probe, the velocity of the reflected sound waves is changed because the blood cells themselves are in motion toward or away from the probe. This phenomenon is known as the "Doppler shift," and is directly related to flow velocity and flow direction of the blood cells. Blood flow is faster during systole and in the center of a vessel whereas it is slower in diastole and near the vessel wall. TCD records a spectrum of flow velocities whose outline resembles an arterial waveform tracing. These concepts are illustrated in Fig. 39.2.

Intraoperatively, TCD measurements are most commonly and easily made by continuous monitoring of the middle cerebral artery for the purpose of detecting either significant changes in flow velocity or the presence of particulate emboli.⁴ As a diagnostic study, segments of all proximal intracranial arteries and the internal carotid artery of the neck can be insonated. An important limitation of TCD results from the fact that most of the examination is done through the temporal bone, which may be thick enough to preclude an adequate examination in 10% to 20% of patients.^{5,6}

Two assumptions that are intuitive and plausible, but ultimately unproven, must be made for TCD-measured blood flow velocity to have a direct relationship to CBF. First, blood flow velocity is directly related to blood flow only if the diameter of the artery where the flow velocity is measured and the measurement angle of the Doppler probe remain constant. In practical terms, the difficulty with this assumption lies in finding a means to affix the TCD probe in a way that prevents dislodgment or movement during monitoring. The second assumption requires that CBF in the basal arteries of the brain is directly related to cortical CBF. Because TCD monitoring is typically done using the middle cerebral artery, this assumption may be invalid if collateral blood flow by leptomeningeal collaterals from anterior and posterior cerebral artery territories is adequate. Although these two assumptions constrain the utility of TCD as a stand-alone monitor of CBF, the changes in flow velocity seen in typical applications (discussed subsequently) are large enough to provide useful clinical information.

More importantly, TCD is the only continuous neurologic monitoring technique that provides early warning for hyperperfusion and for the number of emboli delivered to the brain during various phases of an operation. Because of their high echogenicity, emboli show up in the TCD spectrum as high-intensity transient signals (see Fig. 39.2) and are easily identified as brief beeps or chirps within the background of the Doppler sounds.

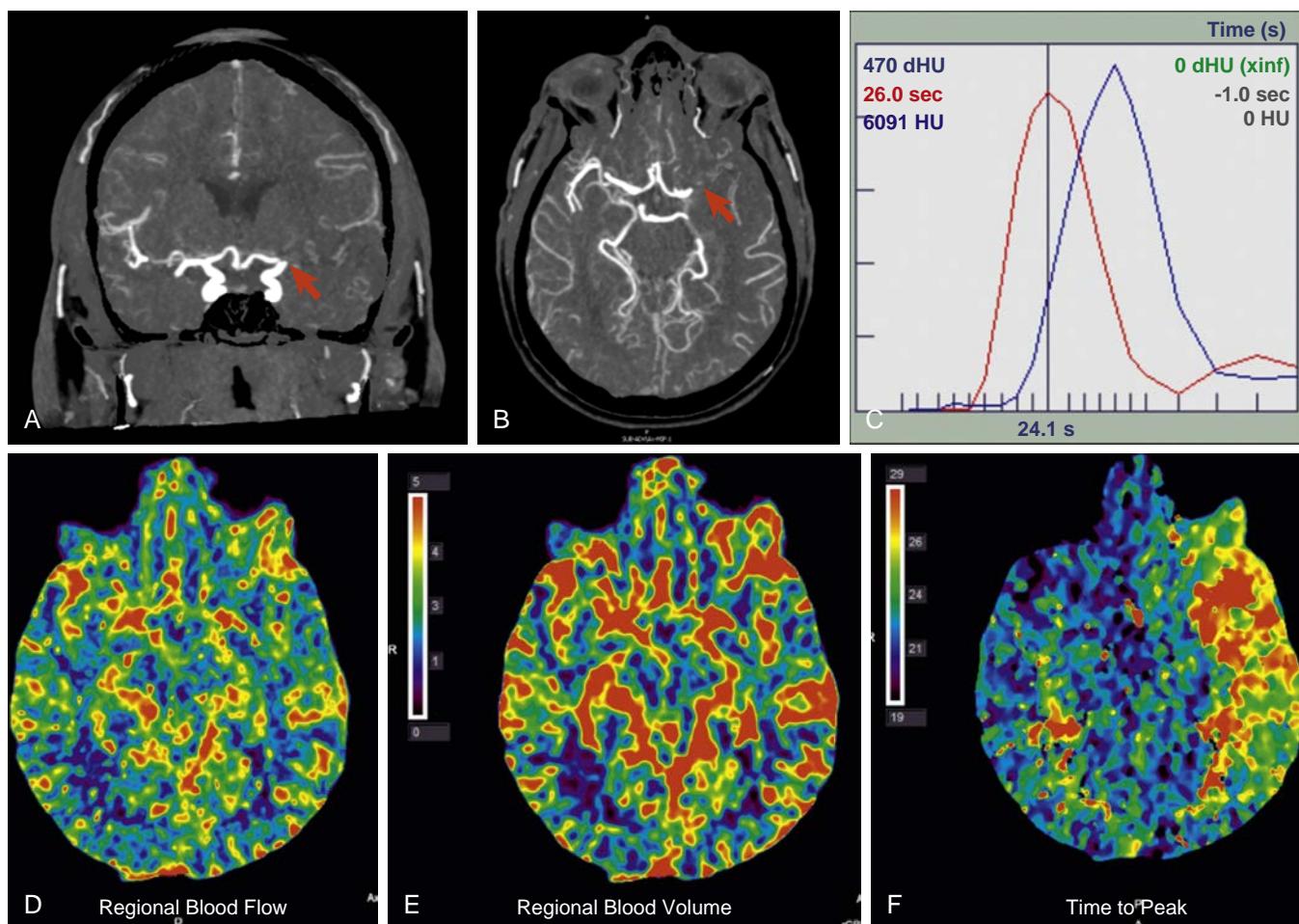


Fig. 39.1 Determination of cerebral blood flow by means of an intravascular tracer. Shown are computer tomographic images of a patient 90 minutes after onset of a left middle cerebral artery stroke. The site of the occlusion in coronal (A) and axial (B) planes is indicated by a red arrow. Panel C shows the arterial inflow function and venous outflow function derived by repeated imaging of the passage of radiopaque contrast through a volume element (voxel) representative of arterial and venous territory, respectively. The A2 segment of the anterior cerebral artery is typically chosen as a voxel for the arterial inflow function and the superior sagittal sinus for the venous outflow function. Based on these functions, blood flow, volume, and flow kinetics can be calculated for other areas of the image. The cerebral blood flow map (D) shows symmetrical flow in both hemispheres, with warmer colors indicating areas of higher flow consistent with grey matter. Blood volume (E) is symmetrical as well, but the time to the peak concentration of contrast (F) is significantly delayed for brain affected by the stroke.

Jugular Bulb Venous Oxygen Saturation. The degree of oxygen extraction by an organ can be monitored by following the oxygen saturation of the mixed venous blood that drains that organ. In the case of the brain, jugular bulb venous oxygen saturation ($Sjvo_2$) is believed to measure the degree of oxygen extraction by the brain and to represent the balance between cerebral oxygen supply and demand. To monitor $Sjvo_2$, a fiberoptic catheter is placed in a retrograde fashion into the jugular bulb through the internal jugular vein under fluoroscopic guidance. Correct tip placement is crucial to minimize admixture of extracranial venous blood. To decrease the risk of complications, usually only one side is monitored.

Several theoretical limitations of the technique must be borne in mind to interpret $Sjvo_2$ values and trends properly. Although nearly all blood from the brain drains via the jugular veins, intracranial mixing of venous blood is incomplete and may result in differences between right-sided and left-sided measurements. The dominant jugular vein (i.e., the right for most patients) drains predominantly cortical venous

blood, whereas the contralateral jugular vein drains more of the subcortical regions.⁷ Despite such regional differences, $Sjvo_2$ must be considered a monitor of global cerebral oxygenation because inadequate perfusion to a focal brain region may not decrease $Sjvo_2$ values below the normal range of 55% to 75%. Because $Sjvo_2$ represents the balance between supply and demand, interpretation of the absolute value of $Sjvo_2$ must take the clinical circumstances into account.

Cerebral Oximetry. Cerebral oximetry is a noninvasive technique that, similar to $Sjvo_2$, uses reflectance oximetry to measure the oxygen saturation of the tissues underneath the sensor. Typically, two sensors are applied to both sides of the forehead. The light passes not only through parts of the frontal brain, but also through the overlying skull and scalp. Contamination of the oximetry signal by extracranial blood sources is a serious concern, although the use of two sensing diodes with different distances from the light source within one sensor patch and adjustments of the algorithm of the oximeter may minimize this problem.^{8,9}

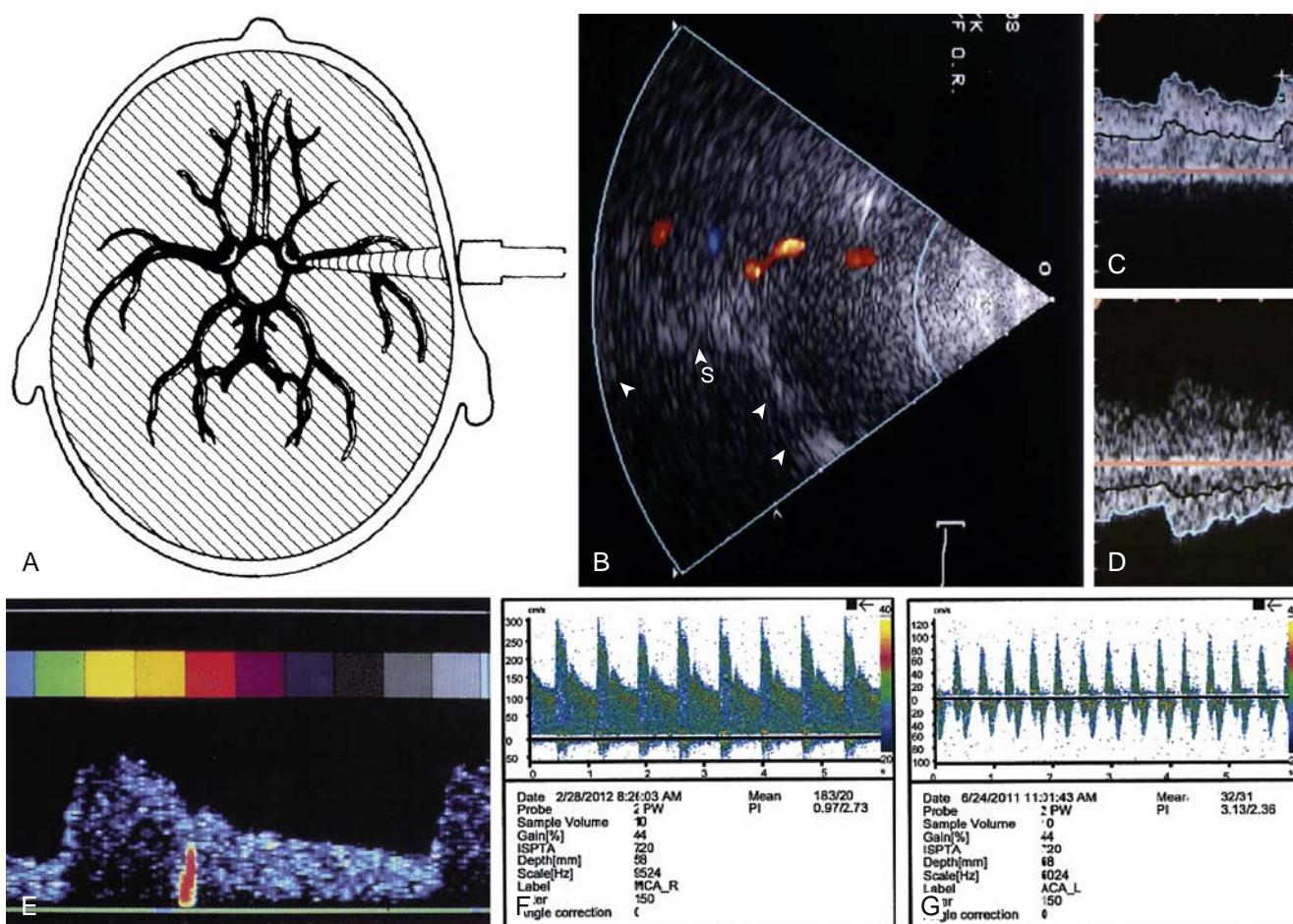


Fig. 39.2 (A) Transcranial Doppler monitoring is done by insonating the arteries at the base of the brain through a thin part of the temporal bone. (B) If this procedure is performed with an imaging probe, some intracranial structures, such as the cerebral peduncles (white triangles) or the sella complex (white triangle labeled "S"), can be visualized. The captured Doppler signals originate from the right middle, right anterior, and left anterior cerebral arteries. (C) Normal Doppler spectrum obtained from the middle cerebral artery. By convention, flow toward the probe is displayed as a waveform above the baseline. (D) Doppler profile of the bifurcation of the terminal internal carotid artery as it branches into the middle cerebral artery (flowing toward the transducer) and the anterior cerebral artery (flowing away). This flow signal can be obtained if the transducer is focused as shown in A. (E–G) Examples of three clinical applications of transcranial Doppler. (E) Emboli are highly echogenic and appear as high-energy transient signals. On the audible output, these emboli are easily noticed as brief beeps or chirps. (F) Doppler profile of a middle cerebral artery in a patient with severe vasospasm after an aneurysmal subarachnoid hemorrhage (compare with C). (G) Transcranial Doppler examination consistent with intracranial circulatory arrest. There is a brief systolic inflow followed by retrograde flow during diastole.

Because 66% to 80% of the cerebral blood volume is venous blood, cerebral oximetry determines predominantly "local venous oxygen saturation." The simplicity of its use, and the familiarity with the principles of treating decreases in systemic mixed venous oxygen saturation, have made cerebral oximetry a popular trend monitor in operations that potentially cause decreases in blood flow to the vessels of the head.¹⁰ There are some significant limitations, however, of the use of cerebral oximetry during such procedures. First, adequacy of global cerebral perfusion is inferred from measurements over the frontopolar brain. Second, normative data on normal values or expected changes for cerebral oximetry are largely absent, but preoperative application of the sensors allows the start of a trend in conjunction with a neurologic baseline examination.¹¹

An example of how these limitations play out is provided by a study of the use of cerebral oximetry during 100 carotid endarterectomies in awake patients.¹² Cerebral oximetry was able to identify 97.4% of patients with adequate CBF as indicated by the absence of clinical symptoms. The monitor

frequently indicated inadequate CBF, defined as a 20% decrease in cerebral oxygen saturation from the pre-clamp baseline, although the patient had no clinical symptoms of inadequate CBF. The false-positive rate of 66.7% may simply illustrate the fact that oxygen extraction increases before function fails. The real problem is that the lower limit for acceptable regional oxygen saturation is unknown in a large population of patients.¹³ Acceptable values may be different from patient to patient, and addition of anesthetic drugs that influence cerebral metabolism may confuse the picture further.

Tissue-Level Blood Flow Monitoring Techniques (Invasive)

Tissue-level monitoring for the brain is by definition invasive. All monitors in current clinical or research use are implanted through a burr hole, extend either into the white matter or ventricular system, and typically use a bolt for stabilization. They all share a 1% to 2% risk of bleeding, infection, or ischemia owing to the implantation procedure.¹⁴

A second shared feature is their limited spatial resolution (i.e., each monitoring probe monitors only a limited area of brain surrounding the probe). When these monitors were first developed, there was considerable debate regarding the optimal placement of the device given such limited spatial resolution. Based on today's appreciation for the impact of secondary neurologic insults on the ultimate outcome, there is growing agreement that tissue-level monitoring is best performed in morphologically and functionally normal tissue that is part of the penumbra or vulnerable zone of interest.¹⁵⁻¹⁷

Of the tissue-level monitors, tissue partial pressure of oxygen (P_{O_2}) monitoring has undergone sufficient refinement to be in wider clinical use. Thermal diffusion blood flow measurement and laser Doppler flow measurement are experimental and not in widespread clinical use.

Tissue Partial Pressure of Oxygen Monitoring

Localized monitoring of tissue P_{O_2} is based on an oxygen-sensitive electrode originally described by Clark.¹⁸ The diffusion of oxygen molecules through an oxygen-permeable membrane into an electrolyte solution causes an electric current that is proportional to P_{O_2} . Currently available catheter-based electrodes placed into the subcortical white matter provide stable recording conditions over long periods.

Most of the data on brain tissue oxygen levels (P_{BrO_2}) come from studies in patients with head trauma.¹⁹ Comparison with stable xenon CT for assessment of CBF and studies during temporal clipping in aneurysm surgery show good correlation between P_{BrO_2} and CBF.^{20,21} Similarly, the time course of changes in P_{BrO_2} after traumatic brain injury resembles that of CBF.^{22,23} Critics of the technique argue that P_{BrO_2} values are highly influenced by the partial pressure of arterial oxygen (P_{aO_2}) and are merely an elaborate indicator of the quality of patient ventilation. This view is supported by the observation that increasing the fraction of inspired oxygen (F_{iO_2}) increases P_{BrO_2} , but likely represents an oversimplification.²⁴ Concurrent microdialysis studies have shown that increasing F_{iO_2} not only increases P_{BrO_2} , but also decreases tissue lactate levels, suggesting a true improvement in the metabolic milieu of the brain tissue itself.^{25,26} While decreases in P_{BrO_2} are associated with worse outcomes in traumatic brain injury patients, the role of P_{BrO_2} -directed therapy is still investigational.²⁷

MONITORS OF NERVOUS SYSTEM FUNCTION

The most commonly used monitors of function are the electroencephalogram (EEG), sensory-evoked responses (SERs), motor evoked responses, and the electromyogram (EMG). The EEG is a surface recording of the summation of excitatory and inhibitory postsynaptic potentials spontaneously generated by the pyramidal cells in the cerebral cortex. The signals are very small, and each recording electrode records information both directly beneath the electrode and information volume conducted from deeper tissue.²⁸ Monitoring the EEG is usually directed toward one or more of four perioperative uses. First, the EEG is used to help identify inadequate blood flow to the cerebral cortex caused by either a surgically induced or anesthetic-induced reduction in blood flow or retraction on cerebral tissue. Second, the EEG may be used

to guide an anesthetic-induced reduction of cerebral metabolism either in anticipation of a loss of CBF or in the treatment of high intracranial pressure, when a reduction in CBF and blood volume is desired. Third, the EEG may be used to predict neurologic outcome after a brain insult. Finally, the EEG may be used to gauge the depth of the hypnotic state of the patient under general anesthesia (see [Chapter 40](#)).

More than 50 years of experience monitoring the EEG has led to many known correlations of EEG patterns with clinical states of the normal and diseased cerebral cortex. The electroencephalographer can accurately identify consciousness, unconsciousness, seizure activity, stages of sleep, and coma. In the absence of significant changes in anesthetic technique, the electroencephalographer also can accurately identify inadequate oxygen delivery to the brain (from either hypoxemia or ischemia). By using high-speed computerized EEG analysis and statistical methods, EEG patterns in the continuum from awake to deeply anesthetized are becoming, with few exceptions, much better understood. In addition, computer advances have made possible high-speed mathematical manipulation of the EEG signal to present the data in a manner more suitable to continuous trends for use during surgical or anesthetic monitoring.

Evoked potentials are electrical activity generated in response to either a sensory or a motor stimulus. Measurements of evoked responses may be made at multiple points along an involved nervous system pathway. The evoked responses are generally smaller than other electric activity generated in nearby tissue (muscle or brain) and are readily obscured by these other biologic signals. In the case of SERs, repeated sampling and sophisticated electronic summation and averaging techniques are needed to extract the desired evoked potential signal from background biologic signals. Motor-evoked responses are generally larger and commonly do not require averaging.

SERs are the most common type of evoked potentials monitored intraoperatively. During the last three decades, much research has been done on the use of intraoperative motor-evoked potentials (MEPs), and use of MEPs during both intracranial and spinal surgery is now no longer considered experimental. There are three basic types of SERs: somatosensory-evoked potentials (SSEPs), brainstem auditory-evoked potentials (BAEPs), and visual-evoked potentials (VEPs).

Electroencephalogram

Basic Unprocessed Electroencephalogram Concepts.

The EEG is produced by a summation of excitatory and inhibitory postsynaptic potentials produced in cortical gray matter. Because the EEG signal is generated only by postsynaptic potentials and is much smaller than action potentials recorded over nerves or from heart muscle, extreme care must be taken when placing electrodes to ensure proper placement and excellent contact with the skin to avoid significant signal loss. Alternatively, subdermal needle electrodes may be used, particularly when sterile application of an electrode close to a surgical field is necessary. When electrodes are applied directly to the surface of the brain, impedance is minimized by close electrode contact and saturation of the area with an electrolyte solution.

EEG electrodes generally are placed according to a mapping system that relates surface head anatomy to underlying

brain cortical regions. The placement pattern of recording electrodes is called a montage. Use of a standard recording montage permits anatomic localization of signals produced by the brain and allows development of normative EEG patterns and comparison of EEG recordings made at different times. The standard EEG "map" is called the 10 to 20 system for EEG electrode placement (Fig. 39.3). This system is a symmetric array of scalp electrodes placed systematically based on the distance from the nasion to the inion and from

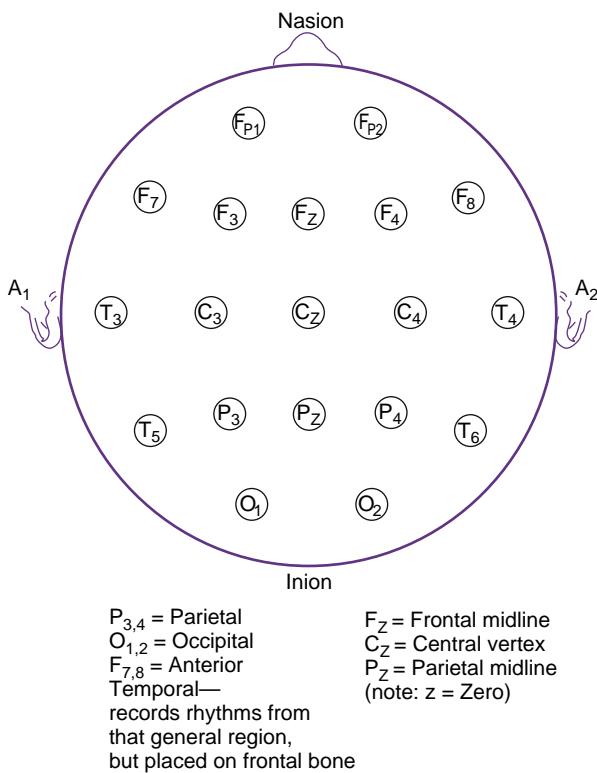


Fig. 39.3 International 10 to 20 system of electrode placement for recording electroencephalogram and sensory-evoked responses. (From Hughes JR. *EEG in Clinical Practice*. 2nd ed. Newton, MA: Butterworth-Heinemann; 1994.)

the pretragal bony indentations associated with both temporomandibular joints. Based on 10% or 20% of these distances, recording electrodes are placed systematically over the frontal (F), parietal (P), temporal (T), and occipital (O) regions at increasing distances from the midline. Left-sided electrodes are given odd number subscripts, and right-sided electrodes are given even number subscripts. Increasing numbers indicate a greater distance from the midline. Midline electrodes are designated with a "z" subscript. The standard diagnostic EEG uses at least 16 channels of information,²⁹ but intraoperative recordings have been reported using 1 to 32 discreet channels.

The intraoperative EEG is most commonly recorded from electrodes placed on the scalp. Recordings also may be made from electrodes placed on the surface of the brain (electrocorticography), or from microelectrodes placed transcutaneously to record from individual neurons (e.g., during surgery for Parkinson disease).^{30,31} The EEG signal is described using three basic parameters: amplitude, frequency, and time. Amplitude is the size, or voltage, of the recorded signal and ranges commonly from 5 to 500 μ V (vs. 1-2 mV for the electrocardiogram signal). Because neurons are irreversibly lost during the normal aging process, EEG amplitude decreases with age. Frequency can be thought of simply as the number of times per second the signal oscillates or crosses the zero voltage line. Time is the duration of the sampling of the signal; this is continuous and real time in the standard paper or digital EEG, but is a sampling epoch in the processed EEG (see later).

Normal Electroencephalogram. Normal patterns seen on the EEG vary among normal individuals, but are consistent enough to allow for accurate recognition of normal and pathologic patterns. The usual base frequency in an awake patient is the beta range (>13 Hz). This high-frequency and usually low-amplitude signal is common from an alert attentive brain and may be recorded from all regions. With eye closure, higher amplitude signals in the alpha frequency range (8-13 Hz), seen best in the occipital region, appear (Fig. 39.4). This "eyes closed" resting pattern is the

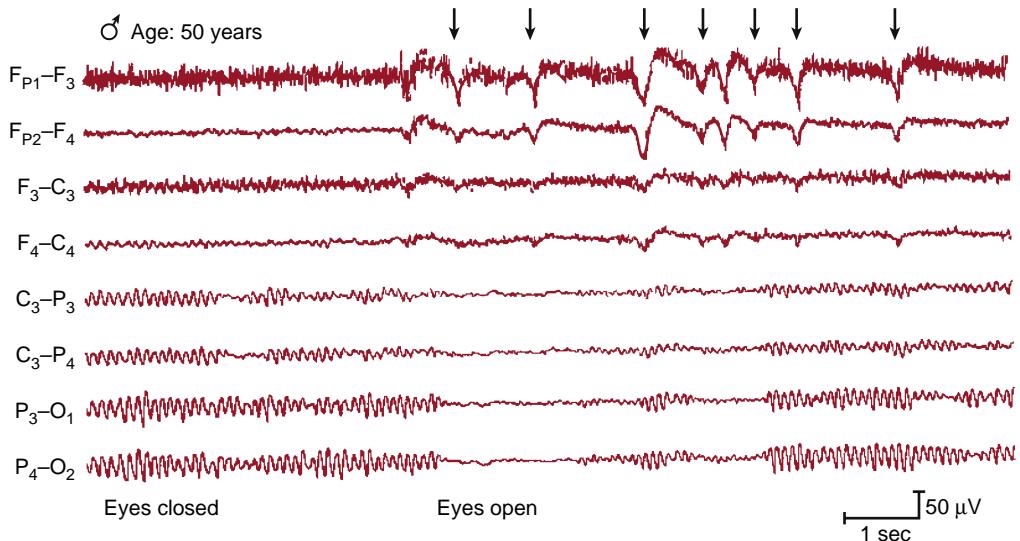


Fig. 39.4 The loss and return of alpha activity as the eyes open and close can be seen. The large spikes (!) are muscle artifact from eye blinks and hence are best visible in channels that incorporate frontal electrodes (designated F).

baseline awake pattern used when anesthetic effects on the EEG are described. When events that lead the brain to produce higher frequencies and larger amplitudes occur, the EEG is described as “activated,” and when slower frequencies are produced (theta = 4-7 Hz, and delta = <4 Hz), the EEG is said to be “depressed.” The EEG during natural sleep may contain all of these frequencies at various times. The slower frequencies occur during deep natural sleep with “sleep spindles” (Fig. 39.5), but during light sleep or rapid eye movement sleep, the EEG becomes activated, and the eye muscle EMG appears on the EEG.

In the normal EEG in awake and asleep patients, patterns recorded from corresponding electrodes on each hemisphere are symmetric in terms of frequency and amplitude, the patterns are predictable if clinical states are known, and spike (epileptic) waveforms are absent. In most cases, normal EEG patterns are associated with normal underlying brain function in awake and asleep patients.

Abnormal Electroencephalogram. General characteristics of the “abnormal” EEG include asymmetry with respect to frequency, amplitude, or both, recorded from corresponding electrodes on each hemisphere, and patterns of amplitude and frequency that are unpredictable or unexpected in the normal recording. These abnormal patterns reflect either anatomic or metabolic alterations in the underlying brain. Regional asymmetry can be seen with tumors, epilepsy, and cerebral ischemia or infarction. Epilepsy may be recognized by high-voltage spike and slow waves, whereas cerebral ischemia manifests first with EEG slowing with preservation of voltage. Further slowing and loss of voltage occurs as ischemia becomes more severe. Factors affecting the entire brain may produce symmetric abnormalities of the signal. Identifying pathologic abnormal patterns in the global EEG signal is very important, although sometimes quite difficult, in the clinical situation. Many of the normal global pattern changes produced by anesthetic drugs are similar to pathologic patterns produced by ischemia or

hypoxemia. Control of anesthetic technique is very important when the EEG is being used for clinical monitoring of the nervous system.

Processed Electroencephalogram Concepts. Numerous limitations are introduced when moving from the raw EEG domain to the processed EEG domain. First, artifact is processed in many cases along with desired signal leading to a perfectly believable processed EEG display that is materially incorrect. Second, the standard 16-channel EEG montage provides more information than can be practically analyzed or displayed by most processed EEG monitors and perhaps more than is needed for routine intraoperative use. Most available processed EEG devices used by anesthesiologists use four or fewer channels of information—translating to at most two channels per hemisphere. Processed EEG devices generally monitor less cerebral territory than a standard 16-channel EEG. Third, some intraoperative changes are unilateral (e.g., regional ischemia owing to carotid clamping), and some are bilateral (e.g., EEG depression by bolus administration of an anesthetic). Display of the activity of both hemispheres is necessary to delineate unilateral from bilateral changes. An appropriate number of leads over both hemispheres is needed. Most early studies validating intraoperative EEG monitoring used continuous visual inspection of a 16- to 32-channel analog EEG by an experienced electroencephalographer—such monitoring was considered the gold standard.^{32,33} Adequate studies comparing the processed EEG with fewer channels with this gold standard across multiple uses and operations have not been done, although limited data using processed EEG monitoring during carotid surgery suggest that two- or four-channel instruments would detect most significant changes,^{34,35} provided that the electrodes are appropriately placed over watershed areas of blood supply.

Devices. EEG processing for intraoperative monitoring is typically based on power analysis of a segment of raw EEG, referred to as an epoch. Power analysis uses Fourier transformation to convert the digitized raw EEG signal into component sine waves of identifiable frequency and amplitude. The raw EEG data, which is a plot of voltage versus time, is converted to a plot of frequency and amplitude versus time. Many commercially available processed EEG machines display power (voltage or amplitude squared) as a function of frequency and time. These monitors display the data in two general forms, either compressed spectral array or density spectral array. In compressed spectral array, frequency is displayed along the x axis, and power is displayed along the y axis with height of the waveform equal to the power at that frequency. Time is displayed along the z axis. Tracings overlap each other, with the most recent information in front (Fig. 39.6). Density spectral array also displays frequency along the x axis, time is displayed along the y axis, and power is reflected either by the density of the dots at each frequency or by a spectrum of colors. Each display format provides the same data, and choice depends on the preference of the user.

Many changes that occur during anesthesia and surgery are reflected as changes in amplitude, frequency, or both. These changes can be clearly seen in these displays if adequate and appropriate channels are monitored. Power

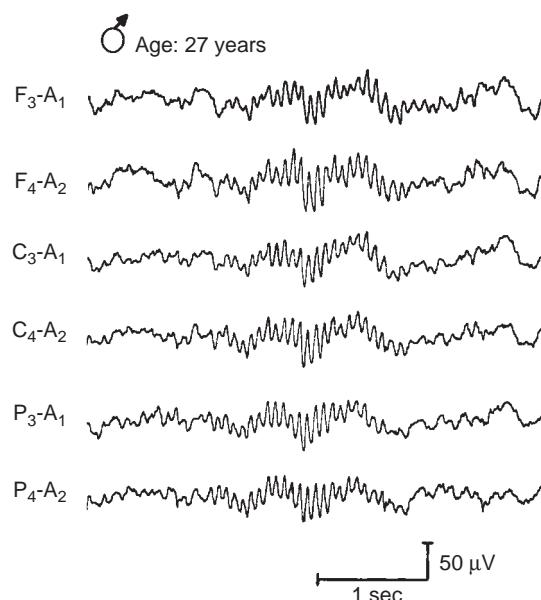


Fig. 39.5 Characteristic sleep spindles in normal sleep are shown in the center.

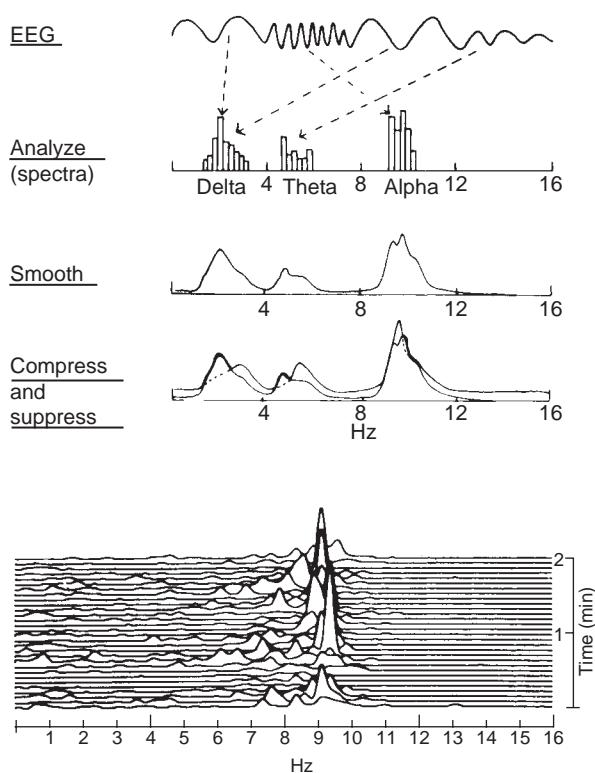


Fig. 39.6 Diagram of a technique used to generate compressed spectral array. Example at the bottom of the figure shows compressed spectra of the alpha rhythm from a normal subject. *EEG*, Electroencephalogram. (From Stockard JJ, Bickford RG. The neurophysiology of anaesthesia. In: Gordon E, ed. *A Basis and Practice of Neuroanaesthesia*. New York: Elsevier; 1981:3.)

analysis has been used clinically for many years as a diagnostic tool during procedures with risk for intraoperative cerebral ischemia, such as carotid endarterectomy and cardiopulmonary bypass (CPB). Power analysis has proven to be a sensitive and reliable monitor in the hands of experienced operators using an adequate number of channels. In addition, parameters obtained from power analysis have been investigated as monitors for depth of anesthesia.³⁶⁻³⁹

Data Acquisition Period. An important consideration in the processed EEG is time. Raw EEG is continuous in real time. The processed EEG samples data over a given time period (epoch), processes the data, and then displays information in various formats. There is a relationship between epoch length and spectral resolution. If a long epoch length is chosen, the waveform can be described precisely, but the time required for data processing is long and not real time. If a short length of data is sampled, analysis may be done in near real time, but the epoch chosen for analysis may not be representative of the overall waveform (i.e., the condition of the patient). There also may be insufficient data points for meaningful Fourier transformation. This issue, as related to the use of intraoperative EEG for analysis of anesthetic depth, has been studied by Levy.⁴⁰ A longer epoch may produce less epoch-to-epoch variability and allow more precise description of frequency and power; however, the longer epoch increases the delay before new information is processed and displayed, reducing the amount and timeliness of information available for clinical decision making. In

studying EEG epochs of 2 to 32 seconds, Levy⁴⁰ concluded that 2-second epochs are appropriate during general anesthesia. Many commercially available devices have used 2-second epoch lengths, updated at varying user-selected intervals. With better and faster computers, continuous monitoring of 2-second epochs and now even longer epochs is possible.

Evoked Potentials

Basic Concepts Common to All Modalities. EEG signals provide information about cortical function, but little to no information about subcortical neural pathways crucial to normal neurologic function. Intraoperative monitoring of SERs has gained increasing popularity over the last 35 years because it provides the ability to monitor the functional integrity of sensory pathways in an anesthetized patient undergoing surgical procedures placing these pathways at risk. Because motor pathways are often adjacent anatomically to these sensory pathways or supplied by the same blood vessels, or both, function of motor pathways may be inferred, albeit imperfectly, from the function of these sensory pathways. Today, MEPs are monitored together with SERs to provide direct information about function of motor pathways. SERs are typically 100-fold smaller in amplitude than the EEG. Recording SERs in an environment such as an operating room with its myriad electrical devices is challenging and requires substantial technical expertise.

Sensory-Evoked Responses. SERs are electric CNS responses to electric, auditory, or visual stimuli. SERs are produced by stimulating a sensory system and recording the resulting electric responses at various sites along the sensory pathway up to and including the cerebral cortex. Because of the very low amplitude of SERs (0.1-10 μ V), it is often impossible to distinguish SERs from other background biologic signals, such as the EEG or EMG, which may be considered in this case undesirable “noise.” To extract the SER from the background noise, the recorded signal is digitized, and signal averaging is applied. With this technique, signal recording is time-locked to the application of the sensory stimulus. For example, during intraoperative posterior tibial nerve SER monitoring, after nerve stimulation at the ankle, only signal information occurring less than 90 ms after the stimulus is recorded (Fig. 39.7). The SER occurs at a constant time after the stimulus application; other electric activity, such as spontaneous EEG, occurs at random intervals after the sensory stimulus. The averaging technique improves the SER signal-to-noise ratio by eliminating random elements and enhancing the SER. This enhancing effect increases directly with the square root of the number of responses added into the averaged response.

SER recordings are of two general types determined by the distance of the recording electrode from the neural generator of the evoked response. SERs recorded from electrodes close to the neural generators (within approximately 3-4 cm in the average adult) are termed “near-field potentials.”⁴¹ Near-field potentials are recorded from electrodes placed very close to the actual signal generator site,⁴² and the morphology is directly affected by electrode location.⁴¹ Far-field potentials are recorded from electrodes located a greater distance from the neural generator and are conducted to the recording electrode through a volume

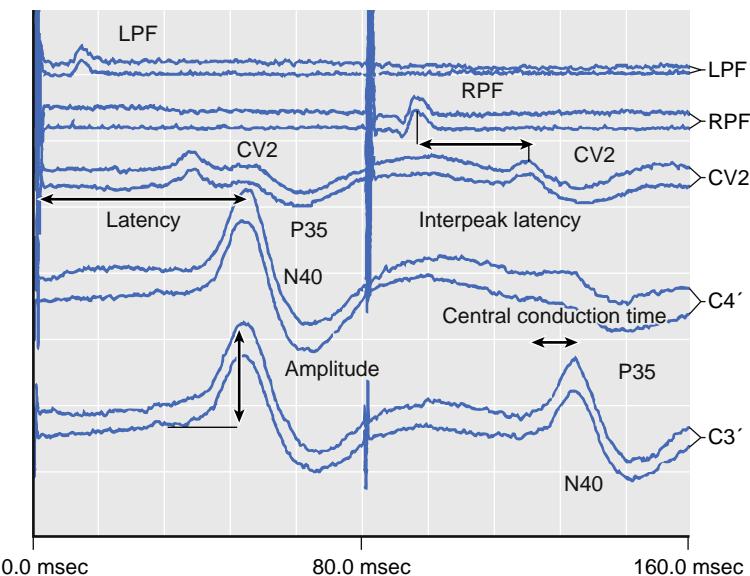


Fig. 39.7 Sensory-evoked responses are described in terms of latency and amplitude. Interpeak latency is the measured time between two peaks. Interpeak latency may be measured between two peaks in the same channel or between peaks in different channels (shown in figure). Note that the polarity of peaks is displayed contrary to standard convention (see text). The example shows a recording of posterior tibial somatosensory-evoked potentials. Each tracing is reproduced twice because reproducibility of a waveform helps distinguish signal from artifact. Left and right posterior tibial nerves are stimulated at 0 and 90 ms, respectively. The first evoked response is recorded from left and right popliteal fossa (LPF and RPF, respectively). The peak labeled CV2 represents the brainstem response recorded at the craniocervical junction. As a far-field potential, the potential looks similar for right- and left-sided stimulation. The primary cortical responses are recorded from the contralateral hemisphere (labeled P35 and N40).

conductor (brain, cerebrospinal fluid, and membranes). Because the current spreads diffusely throughout the conducting medium, it is more difficult to locate the source of the recorded signal and the electrode position has little effect on the morphology of the recorded evoked potential (see Fig. 39.7).^{41,42} As the distance between the recording electrode and the neural generator increases, the recorded SER becomes smaller. More responses have to be averaged to record far-field potentials (several thousand) than near-field potentials (50–100).^{41,42}

SERs also may be described as cortical or subcortical in origin. Cortical SERs are generated by the arrival at the cortex of the volley of action potentials generated by stimulating the sensory system. Because they are recorded as a near-field potential, they are typically easy to identify by elapsed time, waveform morphology, and amplitude. Subcortical responses may arise from many different structures depending on the type of response, including peripheral nerves, spinal cord, brainstem, thalamus, cranial nerves, and others. Cortical SERs are usually recorded from scalp electrodes placed according to the standard 10 to 20 system for EEG recordings (see Fig. 39.3). Subcortical evoked responses also may be recorded as far-field potentials from scalp electrodes or, as appropriate, from electrodes placed over the spinal column or peripheral nerve.

Evoked potentials of all types (sensory or motor) are described in terms of latency and amplitude (see Fig. 39.7). Latency is defined as the time measured from the application of the stimulus to the onset or peak (depending on convention used) of the response. The amplitude is simply the voltage of the recorded response. According to convention, deflections below the baseline are labeled “positive (P),” and deflections above the baseline are labeled “negative (N).” Because amplitude and latency change with recording

circumstances, normal values must be established for each neurologic monitoring laboratory, and may differ from values recorded in other laboratories.

SERs used for intraoperative monitoring include SSEPs, BAEPs, and rarely VEPs. For all these techniques, cortical recording electrodes are placed on the scalp, using the same standard 10 to 20 system as for recording the EEG, whereas recordings for subcortical and peripheral signals are placed in various standardized anatomic locations. The surgical incision and the need for sterility may necessitate nonstandard electrode placements. Such deviations must be considered when interpreting baseline and subsequent SERs. In the case of MEPs, stimulating electrodes also are placed according to the 10 to 20 system of electrode placement, but over the motor cortex instead. Recording electrodes may be placed over the spinal column, peripheral nerve, and (most commonly) innervated muscle.

One of the most important principles of recording SERs intraoperatively is that reproducible, reliable tracings must be obtained at baseline before any intervention likely to cause changes in the evoked response. If good-quality tracings with identifiable waveforms cannot be recorded and reproduced at baseline, evoked-response monitoring would be of little use in monitoring the integrity of the CNS intraoperatively. If significant variability exists, or waveforms are difficult to identify, it will be impossible intraoperatively to distinguish SER changes that are clinically significant from a preexisting baseline variability of waveforms. When good, reproducible responses cannot be recorded at baseline, monitoring should not be used for clinical decision making. It is helpful to comment on the quality of the baseline SERs during the preincision time out so that the whole team is better positioned to put changes in SERs into context.

Somatosensory-Evoked Potentials. SSEPs are recorded after electric stimulation of a peripheral mixed nerve using needles or surface gel electrodes. SSEP responses consist of short-latency and long-latency waveforms. Cortical short-latency SSEPs are most commonly recorded intraoperatively because they are less influenced by changes in anesthetic drug levels. The pathways involved in the generation of upper extremity short-latency SSEPs include large-fiber sensory nerves with their cell bodies in the dorsal root ganglia and central processes traveling rostrally in the ipsilateral posterior column of the spinal cord synapsing in the dorsal column nuclei at the cervicomedullary junction (first-order fibers), second-order fibers crossing and traveling to the contralateral thalamus through the medial lemniscus, and third-order fibers from the thalamus to the frontoparietal sensorimotor cortex. These primary cortical-evoked responses, which are recordable with most anesthetic techniques, result from the earliest electric activity generated by the cortical neurons and are thought to arise from the postcentral sulcus parietal neurons. The longer-latency secondary cortical waves are thought to arise in the association cortex. These responses have much greater variability in an awake patient,⁴² habituate rapidly on repetitive stimulation,⁴¹ and are only poorly reproducible during general anesthesia. Cortical SSEPs other than the primary cortical response are not monitored or interpreted intraoperatively because they are severely altered by general anesthesia.⁴¹

Although most evidence indicates that upper extremity evoked potentials are conducted rostrally in the spinal cord through dorsal column pathways, some data suggest that lower extremity SSEPs are conducted at least partially by the lateral funiculus.⁴³ Stimulation of the posterior tibial nerve or common peroneal nerve at or above motor threshold activates group I fibers that synapse and travel rostrally through the dorsal spinocerebellar tract. After synapsing in nucleus Z at the spinomedullary junction, the pathway crosses and projects onto the ventral posterolateral thalamic nucleus.⁴⁴ This pathway difference is important because the dorsal lateral funiculus is supplied primarily by the anterior spinal artery, the artery that also supplies the descending motor pathway and neurons in the spinal cord. Manipulations, such as distraction of the spinal column to correct scoliosis, which may secondarily compress or distort radicular blood supply to the anterior spinal cord, should cause changes in the SSEP in the event blood supply is reduced to critical levels. This hypothesis is verified by the very low, but not zero, incidence of postoperative paraplegia on awakening without any intraoperative changes in SSEPs.

For SSEP recordings with median nerve stimulation, recording electrodes are first placed at Erb point, just above the midpoint of the clavicle. This point overlies the brachial plexus, and signals recorded here assure the clinician that the stimulus is actually being delivered properly to the patient. The next electrode is placed midline posteriorly over the neck at level of the second cervical vertebra, relatively near the dorsal column nuclei. Signals recorded here ensure proper transmission of the response from the peripheral nervous system into the spinal cord and rostral along the spinal cord to the lower medulla. The final electrodes are placed on the scalp overlying the sensory (parietal) cortex

TABLE 39.1 Generators of Somatosensory-Evoked Potentials After Median Nerve Stimulation

Peak	Generators
N9 (EP)	Brachial plexus*
N11	Posterior columns or spinal roots
N13/P13	Dorsal column nuclei*
N14, 15	Brainstem or thalamus
N19/P22	Parietal sensory cortex*

*Indicates sites commonly recorded during surgical procedures. All other waveforms indicated are not commonly monitored.

contralateral to the stimulated limb. Signals recorded here ensure the integrity of the pathway through the brainstem, thalamus, and internal capsule, and may assess adequacy of CBF in this area of the cortex.⁴⁵⁻⁴⁹

To record SSEPs after posterior tibial nerve stimulation, electrodes are placed first over the popliteal fossa to ensure proper stimulus delivery to the nervous system. Electrodes also may be placed over the lower lumbar spine to ensure proper transmission of the signal into the spinal cord itself, but this site is not commonly used because of the proximity of sterile surgical incisions. Cervical spine and scalp recording electrodes are placed in a similar fashion as described previously, although different locations may be used as required by the surgical incision. More invasive recording methods, such as epidural electrodes, also may be used intraoperatively.

Purported generators for short-latency SSEPs are listed in Table 39.1 and shown in Fig. 39.8.^{41,50} Induction of anesthesia, a patient's neurological disease or age, and use of different recording electrode locations (montage), necessitated by the surgical incision, may significantly alter the appearance of the SSEP. In these cases, attribution of a particular generator to a given wave on the tracing may be quite difficult. During neurologic monitoring, such precision is not needed, and recorded waveforms are compared with tracings obtained at baseline and during earlier portions of the surgical procedure. After lower limb stimulation, absolute latencies are increased because of the greater distance the response to stimulation must travel along the peripheral sensory nerve and spinal cord. Interpeak latencies (see Fig. 39.7) also are evaluated to assess specific conduction times, such as N9 to N14 conduction time, reflecting transmission time from the brachial plexus to brainstem, or N14 to N20 conduction time, reflecting transmission time between the dorsal column nuclei and the primary sensory cortex.⁵¹ Latencies are also significantly affected by age at both extremes of life and many neurologic disorders.

Brainstem Auditory-Evoked Potentials. BAEPs are produced in the diagnostic laboratory by delivering repetitive clicks or tones via headphones. Headphones are not practical for surgical monitoring of neurosurgical procedures, and click stimuli are delivered using foam ear inserts attached to stimulus transducers (Fig. 39.9). Stimulus intensity is usually set at 60 to 70 dB above the patient's click-hearing threshold, although, practically speaking, many intraoperative laboratories establish monitoring after induction of anesthesia and instead begin with a stimulus

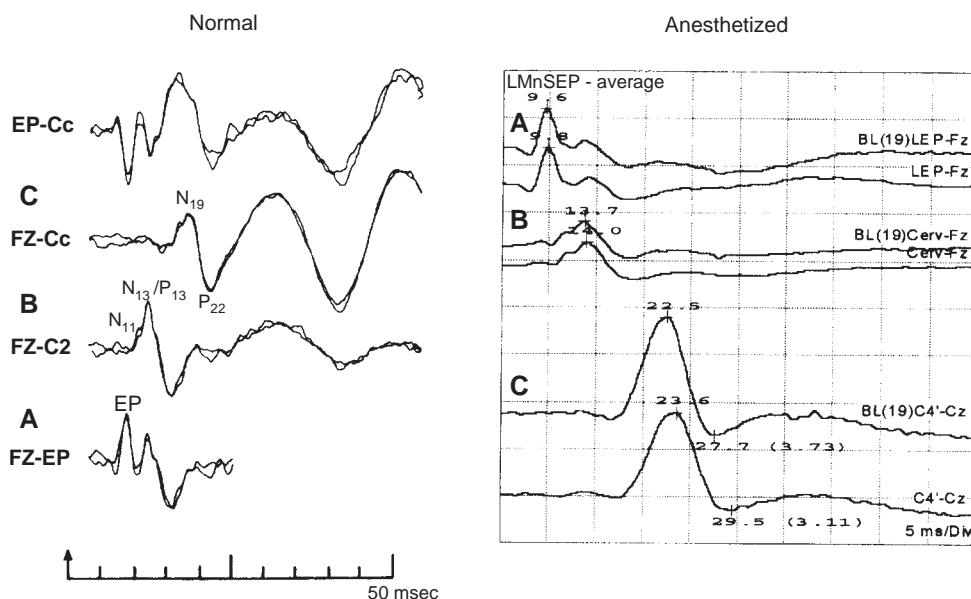


Fig. 39.8 Short-latency somatosensory-evoked potentials (SEP) produced by stimulation of the left median nerve (*LMn*) at the wrist. The ability to identify each of the labeled peaks shown in the tracing from the awake patient is compromised by the anesthetic state and use of different recording electrode locations (A–C). Corresponding tracings are labeled with the same letter. (From Chiappa KH, Ropper AH. Evoked potentials in clinical medicine. *N Engl J Med*. 1982;306:1205.)

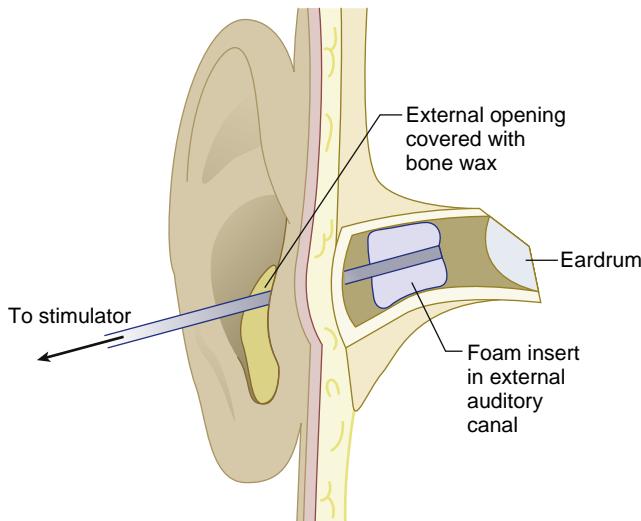


Fig. 39.9 Schematic of brainstem auditory-evoked potential-stimulating apparatus. Loud click stimuli are delivered directly to the eardrum through the ear insert.

intensity of 90 dB nHL (normal hearing level). The duration of the click is approximately 100 μ s, and the stimulus is given usually 10 to 15 times per second. Clicks are delivered using different “polarities”—that is, the click may cause initial movement of the tympanic membrane away from the transducer (rarefaction) or toward the transducer (condensation). Use of these two different methods commonly produces very different waveforms, amplitudes, and latencies in individual patients, and the method that produces the largest reproducible response is chosen. If stimulus artifact is a serious problem, clicks of alternating polarity may be used to decrease the artifact, but the waveforms produced are an average of those produced by either stimulating technique alone and may be more difficult to monitor.

Rate and intensity of stimulus delivery affect BAEPs.^{41,52} Unilateral stimulation is used because responses from the other ear, which may remain normal during surgery, may obscure any abnormal responses from the monitored ear. Recording electrodes are placed on the lobe of the stimulated ear and on the top of the head (vertex).⁵² White noise may be delivered to the contralateral ear to prevent bone conduction from stimulation of the monitored ear from producing an evoked response from the contralateral ear. On average, 500 to 2000 repetitions are required because BAEPs recorded from the scalp are far-field potentials and extremely small (often <0.3 μ V).^{41,52}

Peaks in recordings of BAEPs are labeled I through VII; the purported neural generators for these peaks and the auditory pathway are shown in Fig. 39.10. The anatomic auditory pathway would predict that BAEP monitoring would be most useful for surgical procedures in the posterior fossa that risk hearing or structures in the upper medulla, pons, and midbrain. As with other SERs, amplitude, absolute latencies, and interpeak latencies are evaluated to assess integrity of the auditory system, localize the functional defect when it occurs, and assess peripheral and central conduction times. Because waves VI and VII are inconsistent and variable, they are not routinely monitored,⁵² and most articles reporting use of BAEP for surgical monitoring in the operating room monitor waves only up to wave V.^{53–55}

Visual-Evoked Potentials. VEPs are recorded after monocular stimulation with recording electrodes over the occipital, parietal, and central scalp.⁵⁶ Flash stimulation of the retina using light-emitting diodes embedded in soft plastic goggles through closed eyelids or as needed via contact lenses with built-in light-emitting diodes is provided. VEPs are cortical SERs, which vary with the type of stimulus, part of the retina stimulated, degree of pupil

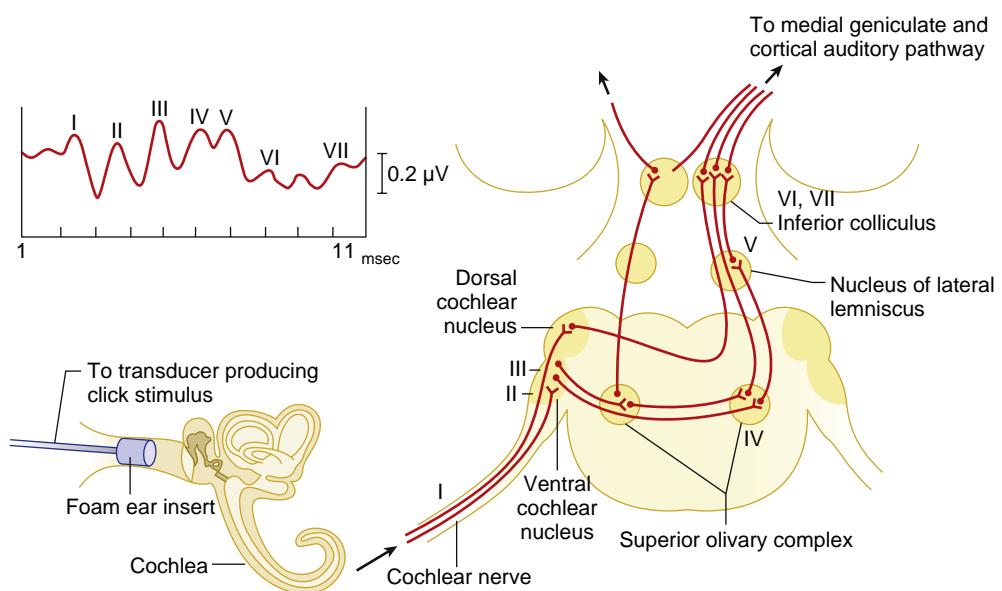


Fig. 39.10 Schematic of auditory neural pathway. The brainstem auditory-evoked potential is initiated by stimulation of the cochlea with a broadband click stimulus given through an ear insert in the external auditory canal. Neural generators of the brainstem auditory-evoked potential peaks are shown.

dilation, and patient's attention level.⁴¹ Because some of these factors change commonly and even constantly during the course of every anesthetic, VEPs would be expected to be highly variable during surgery even when no surgical trespass on the visual system occurs. VEPs are the least commonly used evoked-response monitoring technique intraoperatively. However, some investigators have recently been able to generate reproducible intraoperative VEPs using multiple red light-emitting diodes embedded in a soft silicone disk placed directly on the cornea for stimulating the retina.⁵⁶ This technique may allow for further studies which will determine the clinical utility of this modality. However, flash stimuli generate potentials in all areas of the primary visual cortex at once making it difficult to detect an injury to a small area of cortex.

Motor-Evoked Potentials. MEPs are generated most commonly by the application of a transcranial train of electric stimuli, and responses are recorded at various points along the spinal column, peripheral nerve, and innervated muscle.

TRANSCRANIAL MOTOR-EVOKED POTENTIALS. Monitoring of the integrity of the motor tracts within the spinal cord is a technique with great potential benefit, and even during the relative short history of MEP monitoring, there are reported cases of loss of MEPs with preservation of the SSEP.⁵⁷⁻⁶² This technique is used extensively in spinal surgery, in which transmission across the operative field can be assessed, and in aortic surgery, with the potential for impairment of the blood supply to the vulnerable anterior spinal cord. Relative to SER monitoring, MEP monitoring is quite invasive and, in the case of transcranial stimulation, uses much higher stimulus intensity (≥ 400 V). Special stimulation techniques may be used to obtain transcranial MEPs (tcMEPs) from young children or adults with some degree of neurologic compromise at baseline.

Several variants of MEP monitoring exist. The most common method involves transcranial electrical stimulation. During transcranial electric MEP monitoring, stimulating electrodes (usually small, metallic screw-type electrodes similar to those used in fetal monitoring) are placed into the scalp overlying the motor cortex, and a train of electric stimuli (usually around 400–500 V) is applied to the scalp. This stimulation definitely activates muscles of mastication, and bilateral bite-blocks must be placed to prevent serious damage to the tongue during stimulation. Alternatively, if the precentral gyrus or motor strip is exposed during surgery, stimulating electrodes may be placed directly onto the cortex. Because approximately 90% of the transcranial stimulus dissipates across scalp and skull, typical stimulus intensities of direct cortical stimulation are 40 to 50 V.

Both stimulating methods also activate surrounding cortical structures and subcortical white matter pathways (sensory and motor). In fact, tcMEPs can frequently be recorded from patients with cerebral palsy, despite the disruption of their cortical neuronal architecture. Distal antidromic propagation of the transcranially applied stimulus is blocked by synapses in all of the ascending sensory pathways. The stimulus is propagated easily orthodromically through descending motor pathways. It is important to note that only the largest diameter fibers, 2% to 3% of all fibers in the corticospinal tract, propagate the impulses associated with tcMEPs. The evoked responses may be recorded over the spinal cord, the peripheral nerve, and, most commonly, the muscle itself. To enhance the MEP, these responses may be averaged in the same manner as SERs, but averaging is almost always unnecessary. Activation of the corticospinal tract well below the motor cortex may limit the utility of tcMEP to assess the adequacy of CBF to the motor cortex, because activation may be distal to the site of ischemia and part of a different vascular bed.

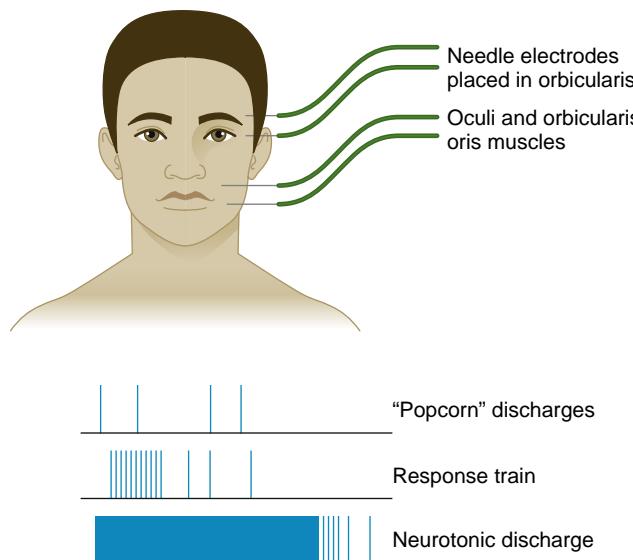


Fig. 39.11 Schematic of facial nerve monitoring and typical responses seen during surgery.

Electromyography. Intraoperative monitoring of EMG responses generated by cranial and peripheral motor nerves allows early detection of surgically induced nerve damage and assessment of level of nerve function intraoperatively. In these cases, the ability of a nerve to produce a response in the innervated muscle is used to assess the health of a cranial or peripheral nerve at risk during surgery. Recordings are made from either surface (electrocardiogram or gold cup) electrodes or needle electrodes placed directly in the innervated muscle of interest. Sensitivity of EMG recordings is best if recordings are made from needles within the muscle itself. Surface electrodes and, to a lesser extent, subdermal needle electrodes may completely miss neurotonic discharges that indicate damage to a nerve.⁶³ The most experience with this monitoring modality has been obtained during facial nerve monitoring.

EMG monitoring may be either active or passive. During active monitoring, a cranial or peripheral nerve is stimulated electrically, and the evoked EMG (compound muscle action potential) response from the muscle is recorded. Stimulation of the nerve proximal to the operative area or tumor can be used to assess functional integrity of the nerve.⁶⁴ Nerve function also may be assessed by noting the intensity of nerve stimulus needed to evoke a muscle response and by the morphology of the compound muscle action potential. Nerve function may be monitored passively during surgery with continuous recording of all generated responses from innervated muscle groups. “Popcorn” EMG discharges are produced by simple, benign contact with the monitored nerve. Response trains are produced with more significant nerve irritation. Neurotonic discharges are produced by significant nerve irritation or damage or both (Fig. 39.11).⁶⁵ When these EMG responses reach a certain voltage threshold, they are usually converted into audible signals that provide immediate feedback to the surgeon and warn of impending nerve damage in real time. Real-time feedback is key because density and frequency of neurotonic discharges may correlate with degree of postoperative

nerve dysfunction, as shown by data obtained from patients undergoing resection of acoustic tumors.⁶⁴ One caveat of EMG monitoring is that sharp section of a nerve may produce no EMG discharge at all.

Intraoperative monitoring of the motor component of other cranial nerves also has been successfully performed. EMG monitoring of the trigeminal nerve can be accomplished with electrodes placed over or in the temporalis or masseter muscles. Trigeminal nerve motor monitoring has been used during nerve section for tic douloureux to ensure preservation of the motor branch of the trigeminal nerve and in combination with facial nerve monitoring during resection of large posterior fossa lesions.⁶⁵ Using recording electrodes placed in or over the trapezius or sternocleidomastoid muscles, the spinal accessory nerve has been successfully monitored during resection of large meningiomas, glomus jugulare tumors, and neck carcinomas.⁶⁵ EMG monitoring of the hypoglossal nerve with needle electrodes placed in the tongue has been infrequently used for large posterior fossa lesions and clivus tumors.⁶⁵ EMG monitoring of the eye muscles can be performed using tiny hook wires for recording; however it is rarely used in practice.

Monitoring of peripheral motor nerves has been performed by placing needle electrodes in or over the muscles innervated by nerves that traverse the operative area and are at risk from the planned surgical procedure. Auditory feedback from EMG monitoring can warn the surgeon of unexpected surgical trespass of the nerve, help locate a nerve within the field (e.g., during untethering of the spinal cord), and localize the level of any conduction block or delay. Because radiculopathies have been reported to occur after spine surgery, particularly because of incorrect pedicle screw placement, EMG monitoring of peripheral nerves has been used in patients undergoing spine surgery to decrease the risk of nerve root injury during the procedure.⁶⁵ During pedicle screw placement, the surgeon can stimulate the screw directly with a small amount of current. If EMG responses are produced with only a small amount of current, the screw has likely been placed outside the bony pedicle.

Reactions to Intraoperative Changes in Monitored Responses

Intraoperative changes in evoked responses, such as decreased amplitude, increased latency, or complete loss of the waveform, may result from surgical trespass, such as retractor placement or ischemia, or they may reflect systemic changes, such as changes in anesthetic drug administration, temperature changes, or hypoperfusion. When these changes are detected and considered to be significant, the surgeon or anesthesiologist can make changes to relieve or lessen the insult to the monitored pathway (and presumably surrounding neural structures). Interventions by the anesthesiologist are directed at improving perfusion to the nervous tissue at risk and include increasing arterial blood pressure, especially if induced hypotension is used, or if the patient's blood pressure has decreased significantly from preoperative values; transfusion, if significant anemia is present; volume expansion; augmentation of cardiac output; and normalization of abnormal arterial blood gas tensions. Changes in evoked potentials after retractor

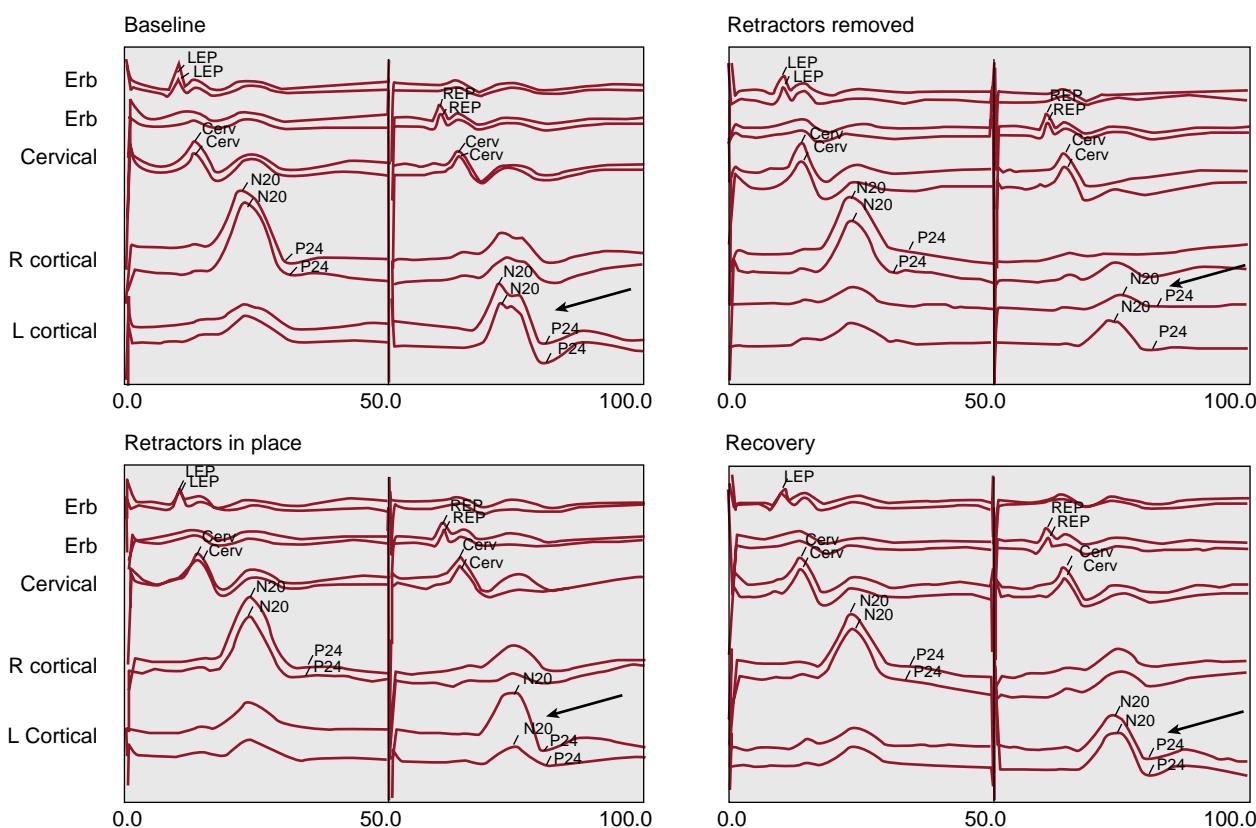


Fig. 39.12 Somatosensory-evoked potentials during aneurysm clipping. Responses generated by the cortex at risk are indicated by arrows. Baseline, after retractor placement, after retractor removal, and recovery traces are shown. The initial evoked response change occurred 4 minutes after retractor placement. Note loss of voltage of cortical-evoked response caused by inadvertent compression of the middle cerebral artery. Cerv, Cervical; LEP, left Erb point; REP, right Erb point.

placement during a craniotomy or after compression of the blood supply to the spinal cord from spinal column distraction promptly allow the surgeon and the anesthesiologist to make appropriate changes to the operative procedure and anesthetic management that may prevent or minimize any postoperative neurologic deficit (Fig. 39.12). It is imperative that clear communication occur between all parties in the operating room when significant intraoperative changes in evoked potentials occur. If expected results of an intervention are not observed the underlying hypothesis as to the cause of the changes must be reevaluated. This can only happen when lines of communication are open between all participants.⁶⁶

Tolerance limits for degree of change in evoked response signals or duration of complete loss of waveform before permanent neurologic dysfunction are becoming better defined, and position statements have been issued by the American Society of Neurophysiologic Monitoring (<http://www.asnm.org/page/PositionStatements>). Tolerance limits for degree of change are especially unclear for tcMEPs. Such ambiguity is common among intraoperative monitors. Although we do know that increased frequency and duration of ST segment depression during coronary bypass surgery is associated with an increased risk of perioperative infarction, exact limits for degree and duration of ST segment depression for surgery do not exist, and likely vary significantly from patient to patient. The same problem may be exhibited with neurologic monitors.

Many centers using intraoperative SER monitoring define decreases in amplitude of 50% or more from baseline associated with a less than 10% prolongation in latency as clinically significant SER changes. Uncorrected, such changes are associated in clinical series and in case reports with onset of new postoperative neurologic deficits. As a result, such changes are immediately investigated. In practice, any SER changes directly associated with a surgical event are considered clinically significant, however, even if the magnitude of change is less than just described. Changes in SER that do not progress to complete loss of the waveform are less likely to be associated with a major new postoperative neurologic deficit. Complete loss of the SER waveform intraoperatively without recovery is highly likely to be associated with a major new deficit. If the SER recovers either spontaneously or after intraoperative interventions, the likelihood of neurologic injury depends on the procedure, the duration of the SER loss, and on whether the SER is mainly used to judge integrity of adjacent unmonitored structures. These problems are illustrated in one study of intracranial aneurysm surgery in which SSEPs were monitored to predict/prevent postoperative motor deficits. Loss of the SSEP waveform for less than 15 minutes was not associated with a new permanent neurologic deficit, whereas complete loss of the SSEP for longer periods was increasingly likely to reflect permanent neurologic injury, even if the response recovered completely to its intraoperative baseline during surgery.⁶⁷

Clinical Applications of Neurologic Monitoring

NEUROVASCULAR SURGERY (ALSO SEE CHAPTERS 56 AND 57)

Extracranial Neurovascular Surgery: Carotid Vascular Surgery (Monitors: Electroencephalogram, Somatosensory-Evoked Potentials, Transcranial Doppler, Cerebral Oximetry)

Carotid vascular surgery typically requires transient interruption of blood flow to the brain through the affected carotid artery. Although the risk of stroke associated with carotid vascular surgery has been declining,⁶⁸ the residual risk varies greatly based on the indication for the procedure. It is lowest for asymptomatic patients and highest after a recent revascularization for ischemic stroke.⁶⁹

Electroencephalogram. The use of the EEG as a monitor of the adequacy of hemispheric blood flow during carotid endarterectomy has been established for many years. In a large series of patients undergoing carotid endarterectomy at the Mayo Clinic,³³ the EEG was compared with regional CBF using the Xe washout method. This study validated the EEG as an indicator of the adequacy of regional CBF.

Normal CBF in gray and white matter averages 50 mL/100 g/min. With most anesthetic techniques, the EEG begins to become abnormal when CBF decreases to 20 mL/100 g/min. Cellular survival is not threatened until CBF decreases to 12 mL/100 g/min. The difference in blood flow between when the EEG becomes abnormal and the blood flow at which cellular damage begins to occur provides a rational basis for monitoring the EEG during carotid surgery. In many cases, prompt detection of EEG changes may allow intervention (e.g., shunting, increasing cerebral perfusion pressure) to restore CBF before onset of permanent neurologic damage.

Serious intraoperative reduction in cerebral oxygen supply may result from surgical factors (e.g., carotid cross-clamping) that are usually beyond the anesthesiologist's control, and from factors that the anesthesiologist can correct. Reduction in CBF produced by hyperventilation, hypotension, or temporary occlusion of major blood vessels may be corrected by reducing ventilation, by restoring normal blood pressure, or, in the case of temporary vessel occlusion, by increasing blood pressure above normal. Because the EEG may readily detect cerebral ischemia, continuous EEG monitoring may be used to evaluate the effectiveness of therapy instituted to correct ischemia.

Should the patient undergoing carotid endarterectomy have EEG monitoring? That question cannot be answered based on available data. EEG monitoring provides information about CBF that would not otherwise be available. The clinician has an opportunity to intervene to increase inadequate blood flow when it occurs. Anecdotally, many clinicians have found such monitoring useful and use it routinely. Population studies do not support routine use, reflecting the fact that strokes in carotid vascular surgery are infrequent and the majority are caused by embolism, which may not be adequately treated by simply increasing CBF.

In a large series of patients undergoing carotid endarterectomy with selective shunting who were monitored with 16-channel unprocessed EEG, no patient awakened with a new neurologic deficit that was not predicted by EEG.⁷⁰ Transient, correctable EEG changes were not associated with stroke. Persistent changes were associated with stroke. This study had no comparison group, however, analyzing stroke rate when EEG monitoring was not used during surgery. Conversely, in the North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial, retrospective comparisons of patients receiving EEG monitoring and patients not receiving EEG monitoring failed to show a significant difference in outcome.^{71,72}

Even more difficult to prove is that EEG monitoring is useful when all patients are shunted during carotid clamping. Such monitoring has detected correctable shunt malfunction, and investigators have described hypotension-related EEG changes in patients with critical stenoses and poor collateral circulation.⁷³ Advocates of selective shunting based on EEG (or other monitoring) criteria claim that inserting a shunt unnecessarily through a region of diseased vessel would surely increase embolization. A multicenter study of 1495 carotid endarterectomies provides some evidence that shunting of patients without evidence of decreased cerebral perfusion increases the incidence of stroke more than sixfold.⁷⁴ Although this study and other more recent studies⁷⁵⁻⁷⁷ advocate that selective shunting using some form of monitoring of the adequacy of CBF should improve perioperative stroke rate, an analysis by the Cochrane Stroke Group⁷⁸ failed to show sufficient evidence to advocate for routine shunting, selective shunting, or even no shunting at all. In addition, this review failed to demonstrate that any type of monitoring for cerebral ischemia was superior to another.

Processed EEG also has been used during carotid vascular surgery. Two issues affect the efficacy and reliability of processed EEG as a monitor for cerebral ischemia. First, what is the minimum number of channels (or areas of the brain) to be monitored? Clinical experience and clinical investigations suggest that four channels (two per side) are the minimum number of channels for adequate sensitivity and specificity.³⁴ When a limited number of channels were compared with 16-channel EEG monitoring, 100% sensitivity and specificity were obtained using 2 channels per hemisphere, provided that those channels monitored the middle cerebral artery territory. These results were obtained with a frontoparietal channel combined with a frontotemporal channel.³⁴

The second issue is the experience level of the observer monitoring the processed EEG. Is a dedicated, experienced technician or electroencephalographer needed? In a study addressing this question, the 16-channel unprocessed EEG monitored by a dedicated technician was compared with a processed EEG reviewed by three anesthesiologists of differing levels of experience with processed EEG.³⁵ The three anesthesiologists interpreted the tracings without knowledge of the case. They were presented only with the written trace with an indication of the point at which the carotid artery was clamped. In these cases, the most important interpretation pitfall to avoid is the "false-negative" pattern. If the clinician interprets the EEG as showing adequate CBF when in fact it does not, the surgeon may fail to shunt

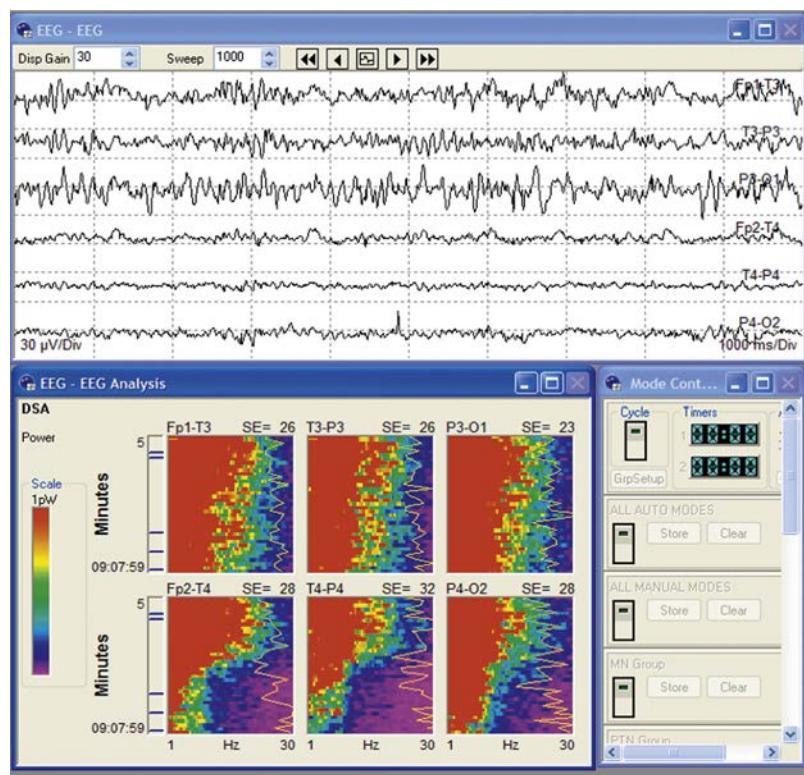


Fig. 39.13 Hemispheric ischemia after clamping of the internal carotid artery. The top panel shows three channels of electroencephalogram (EEG) for each hemisphere. The right-sided channels (bottom three tracings) show nearly suppressed EEG activity due to ischemia. The bottom panel shows the corresponding density spectral array, where EEG power in each frequency is coded in color, with red indicating greater power. The oldest data is at the top of each field and the most recent at the bottom. The lower row of three density spectral array (DSA) panels corresponds to the right-sided EEG leads. Halfway through the panel there is a dramatic reduction in EEG power after clamp placement. The bottom spectrum corresponds to the raw EEG shown tracings in the top panel. (Image courtesy of Reza Gorji, MD.)

an ischemic patient. A false-positive result may be less of a problem because that patient is not ischemic but is given a shunt anyway. In this case, only the risk of emboli from the “unnecessary” shunt is incurred. The positive predictive value of the anesthesiologist correctly interpreting the trace as unchanged after clamping was 91% to 98%, which indicates that the device can be used by novice interpreters with fair accuracy to determine the presence of cerebral ischemia at the time of carotid occlusion (Fig. 39.13).

Somatosensory-Evoked Potentials. SSEP monitoring also has been used to gauge adequacy of CBF to the cerebral cortex and subcortical pathways during carotid vascular surgery.^{49,79,80} SSEPs have been found in the laboratory to have a similar but slightly lower threshold for electrical failure compared with the EEG when CBF is reduced. SSEPs are generally intact until the cortical blood flow decreases to less than 15 mL/100 g/min.⁴⁷ A recent review (meta-analysis) of shunting during carotid vascular surgery suggests that selective shunting based on SSEP monitoring results in a perioperative stroke rate similar to when EEG monitoring is used as the basis for shunting.⁸¹ However, logic would also suggest that changes in SSEP are unlikely, for example, with ischemia involving the anterior portions of either the frontal or temporal lobe, which could readily be detected by an appropriately placed EEG electrode. There is even less outcome evidence to support the use of SSEPs during carotid surgery than there is for the EEG, but the authors

and others have found SSEPs to be useful as a simultaneous monitor with EEG to detect subcortical ischemia.⁸²

Transcranial Doppler. TCD monitoring during carotid vascular surgery is based on measurement of two primary parameters—blood flow velocity in the middle cerebral artery and the number of emboli detected in the same artery. The hypothesis justifying the use of this monitoring during carotid vascular surgery has two components: blood flow velocity correlates with CBF, and increasing numbers of emboli increase the likelihood of embolic stroke. Intraoperative use of TCD has not been widely adopted for many reasons. As previously noted, good TCD signals cannot be obtained in up to 20% of individuals who could benefit from monitoring. Also, TCD probe motion during surgery causes major problems with loss of signal or with angle of insonation-induced changes in the relationship between blood flow velocity and blood flow. Nonetheless, multiple series of carotid endarterectomies with TCD monitoring have been reported with good success, quoting a critical blood flow velocity reduction around 50% as indicative of inadequate CBF requiring intervention (shunt or increased blood pressure or both).⁸³⁻⁸⁹ The relationship between emboli count and stroke is better established. Multiple studies conducted in the preoperative, intraoperative, and postoperative periods indicate that higher emboli counts are associated with higher stroke risk and warrant intervention.⁸³⁻⁸⁹ TCD is the only monitor able to detect dangerous

hyperemia, also known as normal perfusion pressure breakthrough, after removal of a severe, flow-limiting carotid stenosis. Typically, a sustained doubling of flow velocity after unclamping should prompt the anesthesiologist to consider lowering the blood pressure. There are no good outcome data supporting TCD use intraoperatively, but data regarding emboli count and risk of stroke suggest that if technical issues with probe attachment to the patient can be overcome, TCD may be useful as a predictor of impending stroke in the preoperative, postoperative, and perhaps intraoperative periods.

Cerebral Oximetry (Near-Infrared Spectroscopy).

Near-infrared spectroscopy (NIRS) is an attractive monitor during carotid endarterectomy because of its ease of application and lack of training required for interpretation. The hypothesis governing its use is very simple: as oxygen delivery to the brain decreases, oxygen extraction from arterial blood increases, and the oxygen saturation in cerebral venous blood decreases. Multiple case reports and series document the use of cerebral oximetry during neurovascular surgery, but several major questions surrounding use of NIRS during carotid surgery remain unanswered.

The first and most important question is what degree of decrease in oxygen saturation can be tolerated before intervention is necessary. Because most interventions involve some risk (e.g., shunt → emboli, increased BP → myocardial ischemia), the answer to this question is important. The answer does not yet exist, however, and may vary from patient to patient. Two studies in awake patients showed that the saturation value at which any patient would develop symptoms varied from patient to patient,^{12,13} and an absolute value that required shunting could not be determined. Another study showed that cerebral oxygen saturation decreased before the EEG developed changes, and the authors used this observation to claim superiority for NIRS monitoring of the brain during carotid surgery.⁹⁰ This finding should not be surprising, however, because function of the brain (in this case, electric function) does not fail until increased extraction of oxygen no longer meets metabolic demands for the tissue. If metabolic demands are being met by increased extraction, it is unclear that intervention is needed.

Finally, a study by Friedell and colleagues⁹¹ compared NIRS with EEG and SSEP monitoring during carotid surgery. In 24 of 323 patients, significant differences were observed between NIRS and monitors of electric function. Seventeen patients showed no changes in electric function with significant decreases in cerebral oxygen saturation. In seven patients, no change in cerebral oxygen saturation occurred despite significant change in the EEG and SSEP. The latter finding may be due to the fact that NIRS and EEG/SSEP monitor different vascular territories. These data in combination with the data from studies in awake patients suggest that use of NIRS alone during carotid vascular surgery may be inappropriate. In addition, an aggregate of studies and case reports available in the literature suggests that there is no clear cutoff value of regional oxygen saturation which would mandate shunt placement or increasing the cerebral perfusion pressure.

Intracranial Neurovascular Surgery (Monitors: Somatosensory-Evoked Potentials, Motor-Evoked Potentials)

Somatosensory-Evoked Potentials. SSEPs have been extensively studied during cerebral aneurysm surgery. During these procedures, the surgical incision and brain retraction diminishes the utility of placing EEG scalp or brain surface electrodes that could detect cerebral ischemia in at-risk cortex. Recording electrodes placed on the surface of the brain have been used successfully, but they are commonly considered “in the way” by neurosurgeons. Thus EEG is typically used only to confirm metabolic suppression during temporary clip placement. Scalp electrodes for SSEP monitoring may be placed more easily, although the recording montage is frequently not the same as that used in an awake patient.

For aneurysms involving the anterior cerebral circulation, SSEP monitoring has an excellent, but not perfect, record for predicting postoperative neurologic function. Most patients without surgically induced SSEP changes during the procedure awaken with an unchanged neurologic examination. Patients with significant SSEP changes that do not revert to normal awaken with a new neurologic deficit. Patients with significant intraoperative SSEP changes that return to normal may show at least a transient postoperative deficit, with the severity and duration of that deficit increasing as the severity of the SSEP change increases. Many authors have reported significant utility of SSEP monitoring in detecting improper aneurysm clip placement (see Fig. 39.12) and in guiding intraoperative blood pressure management, particularly in patients already showing, or at significant risk for, vasospasm after subarachnoid hemorrhage.^{48,92-97} The same success cannot be reported, however, for posterior circulation aneurysms. In these cases, many areas of the cortex and subcortical structures are at risk for damage that cannot be monitored at all by somatosensory pathway function. A significant false-negative monitoring pattern exists for these patients, but changes can still be detected when a surgical insult is sufficiently severe to involve large portions of the brain.⁹⁸⁻¹⁰¹

An important concern during aneurysm surgery is injury to perforating vessels near the aneurysm, which risks damage to subcortical pathways in the internal capsule and may account for half the new neurologic deficits immediately postoperatively.¹⁰² Anatomically, the motor pathway runs anterior to the sensory pathway in the internal capsule and is at greater risk if the anterior choroidal artery, lenticulostriate perforator vessels, or even perforating branches of the anterior cerebral artery are injured during dissection and clip placement. Two concerns need to be addressed if tcMEP is to be incorporated successfully in these cases. First, motion caused by stimulation needs to be minimized to not interfere with the surgery. Second and more importantly, stimulus parameters need to be set to limit deep current spread that would activate the corticospinal tract distal to the internal capsule and obscure ischemia of the proximal pathway. The use of longer stimulus trains at intensities close to the motor threshold addresses both concerns¹⁰³ and have made tcMEP monitoring a useful adjunct during aneurysm surgery in many centers.¹⁰⁴

SUPRATENTORIAL INTRACRANIAL NONVASCULAR SURGERY (MONITORS: AWAKE PATIENT, ELECTROENCEPHALogram, SOMATOSENSORY-EVOKED POTENTIALS)

The most comprehensive approach to the problem of functional localization of brain structures that need to be preserved to achieve a good outcome is to perform entire segments of a supratentorial craniotomy in an awake patient, who is undergoing repeated neurologic examinations targeted to assess the eloquent area at risk. Such procedures are typically divided into exposure, mapping, and resection phases, and can be done with the patient entirely awake or awake only during periods when the neurologic examination needs to be assessed.¹⁰⁵ Common to all these approaches is the need for meticulous locoregional anesthesia of the scalp at the craniotomy site and the pin sites of the head holder. A second requirement is a patient who is well informed about the awake parts of the procedure and willing and able to cooperate. Dexmedetomidine, propofol, and remifentanil are the agents most frequently incorporated into the anesthetic regimens for awake craniotomy.¹⁰⁶ Complications of awake craniotomy include nausea and vomiting, respiratory problems, and “tight” brain, but are typically mild and occur in less than 10% of cases in experienced centers. Seizures triggered by cortical stimulation can be stopped by the application of iced saline to the exposed cortex or a small amount of barbiturate or propofol.

Seizure Focus Localization Surgery

Patients with epilepsy who have seizures that generalize from an anatomically distinct focus may benefit greatly from the surgical resection of that seizure focus.¹⁰⁷ Precise localization of a seizure focus is important for achieving the therapeutic objective of seizure control and for minimizing complications from the resection. With sensitive magnetic resonance tomography techniques, neuro-navigation, and recordings of typical seizure activity in the awake patient after placement of subdural and depth electrodes, the anatomic location and the appropriate extent of the resection frequently can be determined preoperatively.¹⁰⁸ These developments have diminished the role of intraoperative recordings from the epileptogenic zone using electrocorticography.

Electrocorticography is done by placing a grid of subdural electrodes onto the exposed brain surface and recording spontaneous electric activity. Electrocorticography is constrained by several limitations. The time for such recordings is limited to a few minutes; recordings are limited to interictal discharges, which may not correlate with the epileptogenic focus; and recordings need to be obtained from a brain that is under the effects of general anesthetic agents, which alter the EEG.

To provide good conditions during the recording, the level of anesthesia is lightened (e.g., by use of a strict nitrous oxide–narcotic technique or low concentrations of volatile anesthetic drugs). Provocative techniques, such as hyperventilation or administration of a small dose of methohexitol, may be useful to activate the seizure focus. Intraoperative seizure mapping requires the involvement of an expert electroencephalographer familiar with this technique.

Motor Strip Localization

Electrophysiologic monitoring of the somatosensory system in anesthetized patients can provide a simple anatomic guide to the location of the rolandic fissure, which separates the parietal primary sensory and frontal primary motor cortex. The fissure is located by recording cortical SSEPs from a subdural strip electrode that is placed perpendicular to the presumed location of the fissure. The exact location of the fissure is characterized by a reversal in the polarity of the primary cortical response between the electrodes straddling the fissure, as illustrated in the clinical example in Fig. 39.14. Subsequent placement of the electrode strip onto the primary motor area of the precentral gyrus allows subsequent monitoring of the corticospinal tract through direct cortical stimulation.

POSTERIOR FOSSA SURGERY (MONITORS: BRAINSTEM AUDITORY-EVOKED POTENTIALS, CRANIAL NERVE MONITORING, SOMATOSENSE-EVOKED POTENTIALS, MOTOR-EVOKED POTENTIALS)

Besides the cerebellum, the posterior fossa contains within the narrow space of the brainstem many crucial neural structures, including the following: the ascending and descending sensorimotor pathways; cranial nerve nuclei; cardiorespiratory centers; the reticular activating system; and the neural networks that underlie crucial protective reflexes, such as eye blink, swallowing, gag, and cough. Posterior fossa surgery is not undertaken lightly, and even small injuries can leave significant neurologic deficits. Although some of these neural structures, such as the sensory, voluntary motor, or auditory pathway, can be monitored consistently, intraoperative integrity of other neural structures is frequently only inferred from the well-being of neighboring structures amenable to monitoring.

Microvascular Decompression of Cranial Nerves V, VII, and IX

Microvascular decompression is done most frequently for trigeminal neuralgia (cranial nerve V) in patients who present acceptable medical risks for a posterior fossa craniotomy. More rarely, the same approach is used to treat hemifacial spasm or neurovascular compromise of lower cranial nerves. The surgery entails dissecting along the intracranial portion of the nerve, identifying offending blood vessels that encroach on the nerve, and placing an insulating Teflon pad between vessel and nerve. The surgery risks ischemic damage to perforating vessels arising from the offending arteries and cerebellar retraction-related damage to cranial nerves. The facial and vestibulocochlear nerves are at particular risk for stretch-induced injury caused by medial retraction of the cerebellum. Retraction-induced stretch produces a prolongation of the interpeak latency between peaks I and V of the BAEP waveform, ultimately leading to a complete loss of all waves beyond wave I (Fig. 39.15). Failure to release retraction in a timely manner results in postoperative hearing loss. Such monitoring increases the chances for preserved hearing after microvascular decompression.¹⁰⁹⁻¹¹³ During microvascular decompression of the facial nerve for hemifacial spasm, recent developments using EMG monitoring

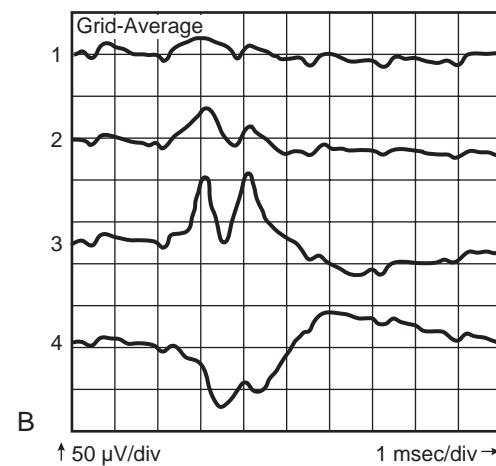
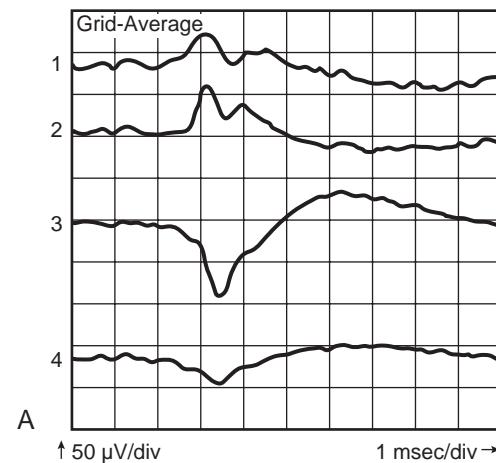
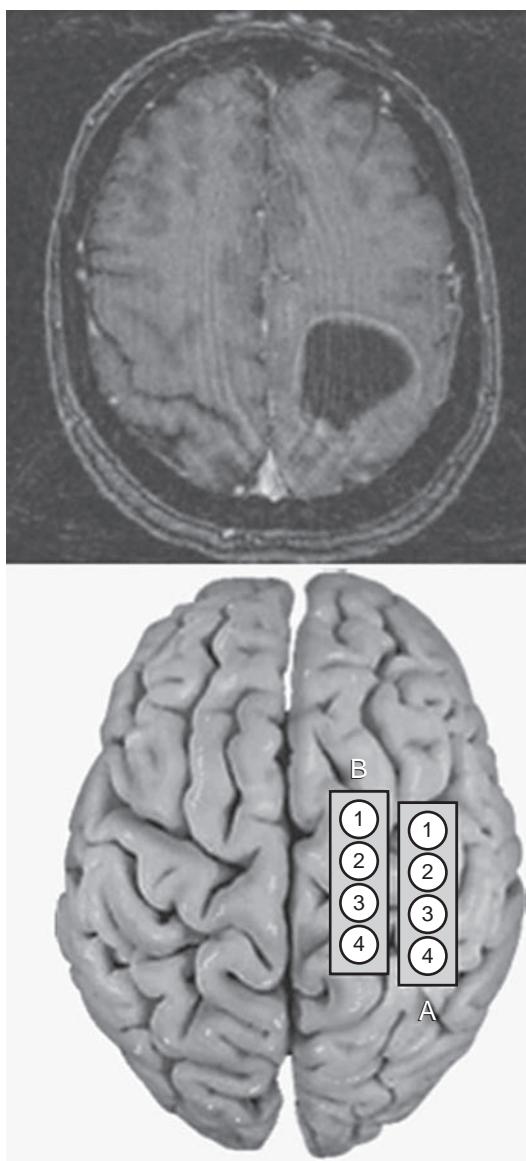


Fig. 39.14 Intraoperative localization of the rolandic fissure separating the primary sensory and motor cortex. The clinical example is from a patient with a large parietal tumor shown in the scan. Two of the recordings made from a four-contact subdural electrode strip are shown. The relative positions of the strip electrode are labeled B and A. In recording A, the primary cortical response from the electrodes anterior to the rolandic fissure shows an upward deflection, whereas the response from electrodes posterior to the fissure shows a downward deflection. Moving the strip electrode anteriorly (recording B) moves this "phase reversal" between electrodes 3 and 4.

have enabled better documentation of the adequacy of nerve decompression and lessen the likelihood of persistence or recurrence of hemifacial spasm postoperatively. This new technique monitors the so-called lateral spread response (LSR) of the facial nerve. A peripheral branch of the facial nerve is stimulated. In the normal patient, this stimulation would not result in a recordable EMG response in a muscle innervated by a different branch of the facial nerve. In patients with hemifacial spasm, EMG responses can be recorded in muscles innervated by a different branch of the facial nerve (an LSR) indicating abnormal crossover of electrical activity. Decompression of the facial nerve has been shown in multiple studies to result in a great decrease or elimination of this LSR, and studies have indicated that elimination of the LSR is highly predictive of immediate postoperative relief of hemifacial spasm.¹¹⁴

Vestibular Nerve Schwannoma

Vestibular nerve schwannomas are the most common tumors located in the cerebellopontine angle. Because of the common origin of the cochlear component of cranial nerve VIII and the essentially identical intracranial trajectory of the facial nerve, hearing loss and facial nerve palsy are concerns during surgical resection of these tumors. Size and preoperative auditory function are the best predictors of postoperative hearing.¹¹⁵ For tumors up to about 1.5 cm in diameter, monitoring of BAEPs can increase the chances of preserving hearing.¹¹⁶ In addition to BAEPs, the facial nerve is monitored through spontaneous and stimulated EMG. Prospective trials have shown a higher percentage of patients with a functional facial nerve 1 year after surgery if facial nerve monitoring was used. Tonic discharges warn of impending damage caused by stretch or heat

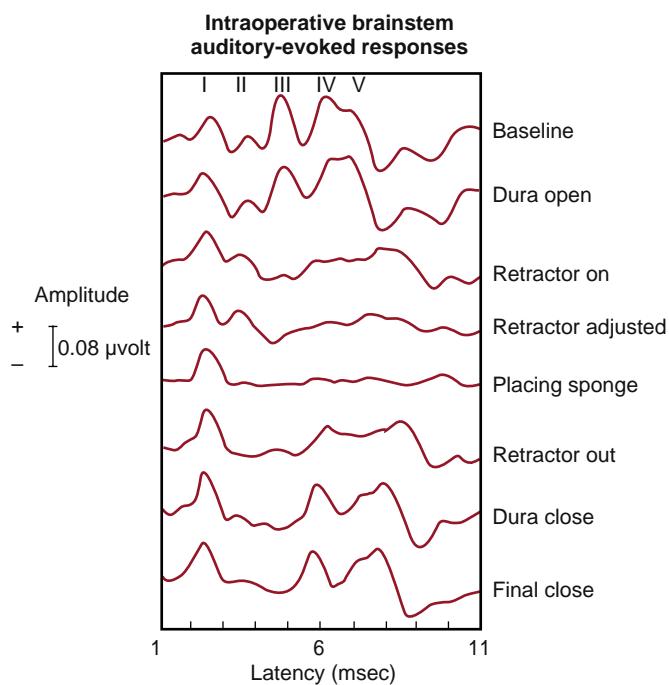


Fig. 39.15 Intraoperative monitoring of brainstem auditory-evoked responses during microvascular decompression. The baseline recording shows the typical five waves of the brainstem auditory-evoked potential response. Intraoperative events are designated to the right of each trace. Placement of the retractor causes a severe increase in latency of wave V even after adjustment of retraction. During placement of the sponge, all waves subsequent to wave I, which originates in the inner ear, are nearly completely lost. Removal of the retractor causes brainstem auditory-evoked potentials to revert toward baseline.

(e.g., electrocautery). Sharp section of the nerve may elicit no discharge, and neuromuscular blockade may eliminate the ability to monitor. If the course of the nerve is displaced by the tumor, the surgeon can map its course with a handheld stimulator and real-time auditory feedback.

Other Posterior Fossa Neoplasms

Monitoring for operations on other neoplasms located in the brainstem typically is individualized to each particular case or to the particular surgical approach. EMG and compound muscle action potentials may be recorded not only from the territory of the facial nerve, but also from the tongue to monitor the hypoglossal nerve, and from the glottis through electrodes embedded into a specialized endotracheal tube to monitor the vagus nerve. Such a setup can be used to map the floor of the fourth ventricle functionally, if it is distorted by a tumor.¹¹⁷ Such monitoring may be insufficient to preserve vital reflexes because only the efferent limb of these reflexes is monitored by recording EMG from innervated muscle. MEPs can be recorded from orbicular oris, or mentalis, to assess the integrity of the facial nerve motor nucleus as well as the proximal facial nerve when tumors prevent visualization of the proximal portion of cranial nerve VII. MEPs can also be recorded from tongue as well. Obtaining these responses relies on placement of additional stimulating electrodes and optimization of stimulating conditions.

Use of neurologic monitoring for brainstem ischemia, although done in some centers, is not well documented or supported by clinical studies. Global well-being of the brainstem may be monitored by combining multiple modalities of

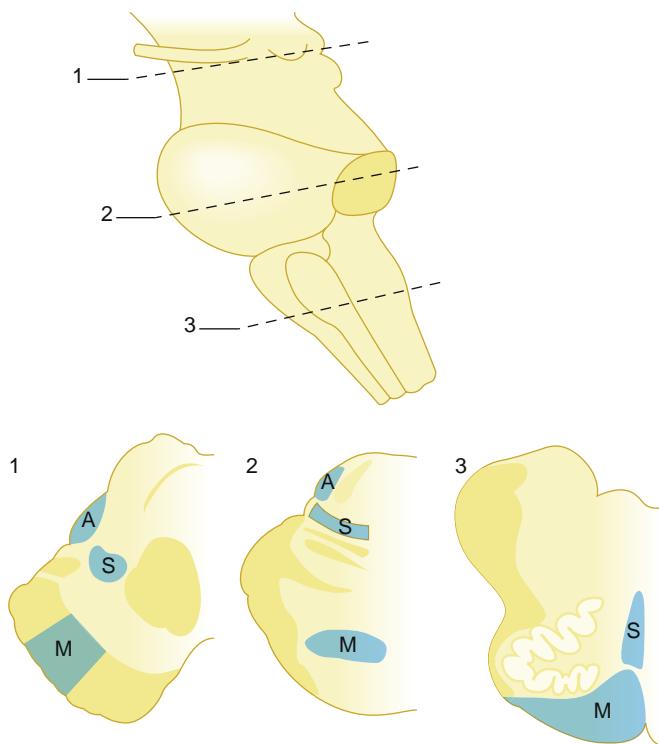


Fig. 39.16 Monitoring of the brainstem with evoked potentials. Evoked potentials monitor specific tracts that encompass defined areas in the brainstem. This is shown in three transverse sections approximately at the levels indicated in the drawings. The areas directly monitored by a given modality are indicated in blue and labeled M (motor), S (somatosensory), and A (auditory). Conclusions about the well-being of the remainder of the brainstem are made by inference from the monitored areas.

evoked potentials, such as BAEPs, SSEPs, and MEPs. Each modality monitors a function whose integrity would be considered important in its own right for the functional outcome of an individual patient. As illustrated in **Fig. 39.16**, the cross-section monitored by combining all these modalities still leaves out crucial areas. Given that perfusion occurs through perforating vessels, it is easy to see that monitoring may indicate that all is well or, more likely, that a therapeutic intervention was helpful in restoring function, when clinically the patient is still left with a significant deficit. This occurrence invalidates neither monitoring nor the therapeutic intervention, but indicates only that the monitored pathway was not located in an area at risk from the surgical procedure. Because of such obligatory “false-negative” results, few studies address the utility of such monitoring. Given that each individual monitoring modality comes with its own constraints, such an approach typically requires a dedicated neurophysiologist for interpretation and troubleshooting.

SPINAL COLUMN AND SPINAL CORD SURGERY (MONITORS: SOMATOSENSORY-EVOKED POTENTIALS, MOTOR-EVOKED POTENTIALS, ELECTROMYOGRAM, AND BULBOCAVERNOSUS REFLEX)

Intraoperative monitoring of SSEPs has been used most extensively in patients undergoing surgical procedures involving the spinal column or spinal cord, or both. Extensive experience has been gained in patients who have

decompressive laminectomies or who have undergone corrective procedures for scoliosis. Intraoperative changes in SSEPs have been noted in 2.5% to 65% of patients undergoing surgical procedures on the spine or spinal cord.¹¹⁸⁻¹²¹ When these changes are promptly reversed either spontaneously or with interventions by the surgeon or anesthesiologist (e.g., lessening the degree of spine straightening in scoliosis surgery or increasing arterial blood pressure), the patients most often have preserved neurologic function postoperatively. When these changes persisted, however, the patients most often awakened with worsened neurologic function.

False-negative (rare) and false-positive (common) results have been reported with SSEP monitoring during spine surgery. Patients with intact SSEPs throughout the procedure have awakened with a new significant neurologic deficit, but the total reported incidence of this finding is far less than 1% of all cases monitored. Patients with no postoperative neurologic deficit commonly experience significant changes in intraoperative SSEPs.⁷⁰ This monitoring pattern is most commonly caused by failure to control for other, nonpathologic factors that may alter the SSEP. Overall, the reliability of properly performed SSEP monitoring to predict the postoperative sensory and motor function has been reported to be excellent.^{41,121,122} Motor tracts are not directly monitored by SSEPs, however. In addition, the blood supply to the dorsal columns of the spinal cord, which carries all of the upper extremity SSEPs and at least a portion of the lower extremity SSEPs, is derived primarily from the posterior spinal arteries. The blood supply to motor tracts and neurons is derived primarily from the anterior spinal artery. It is possible for a significant motor deficit to develop postoperatively in patients with intact SSEPs throughout the operative course. Such events have been reported.^{123,124}

In operations on the spinal column and after acute spinal cord injury, the sensory and motor changes generally correlate well;⁴¹ however, in patients with neurologic dysfunction after thoracic aortic vascular surgery, frequently posterior spinal cord function (proprioception, vibration, light touch) is left intact when motor and other sensory functions (pain, temperature) are impaired. This result occurred in 32% of patients with neurologic injury after aortic aneurysm repair in one series,¹²⁵ with similar results in many other series. Intraoperative SSEP monitoring in these patients carries a significant risk for false-negative results, and as a result, such monitoring is not widely used.

Multiple anecdotal reports and an increasing number of case series suggest that MEP monitoring during surgery on the spine or its blood supply is useful. Several series have reported significant changes in MEPs without changes in SSEPs. These series suggest that combined use of SSEP monitoring and MEP monitoring may eliminate false-negative monitoring patterns during spine surgery.¹²⁶⁻¹³¹ In a consensus statement, the American Society of Neurophysiologic Monitoring concluded that use of MEP monitoring in combination with SSEP monitoring is well established to prevent injury to sensory and motor tracts during spinal column surgery.¹³² In the case of monitoring paraplegia risk during thoracoabdominal aneurysm surgery, the literature shows mixed but improving support for the use of MEP monitoring. Two earlier studies suggested that MEPs

may not be as effective as hoped. The first study recorded MEPs from the lumbar spinal cord in dogs produced by transcranial electric stimulation.¹³³ Elmore and associates found that these spinally recorded potentials did not accurately predict postoperative motor function. In a second study, Reuter and colleagues¹³⁴ recorded MEPs at the spinal cord and the peripheral nerve level in dogs produced by transcranial electric stimulation. They also found that the spinally recorded responses were inaccurate in predicting motor function postoperatively. The peripheral nerve responses disappeared in all animals and were not present 24 hours later regardless of whether the animal could move its lower extremities.

These studies suggest that the spinally recorded MEP likely represents a response generated by the descending corticospinal tract. This white matter pathway is resistant to ischemia compared with the more metabolically active anterior horn cells (gray matter). Recovery of this white matter-generated MEP response could occur after reperfusion of the cord, whereas the gray matter might not recover. Responses recorded from the peripheral nerve would reflect postsynaptic anterior horn cell function, but lower extremity ischemia occurring after aortic cross-clamping may preclude recording this or the response from muscles during surgery.

More recent clinical series have shown much greater success with MEP monitoring during aortic vascular surgery in correctly detecting inadequate spinal cord blood flow and in improving operative outcome. The technique has proven useful, particularly when operative strategies such as reimplantation of crucial intercostal vessels based on results of MEP monitoring, alteration of spinal cord perfusion pressure (blood pressure increase or cerebrospinal fluid drainage or both), spinal cord cooling, and other methods are used.¹³⁵ Additional studies, particularly with the use of endovascular stent repair of thoracoabdominal aneurysms, have found MEPs very useful in guiding therapy and improving outcome.^{136,137}

For surgeries involving the conus medullaris and sacral nerve roots such as untethering of the spinal cord or resection of a lipoma of the filum terminale, tcMEPs can be recorded from the anal sphincter. In addition, the reflex arc of the bulbocavernosus reflex can be recorded, by stimulating the pudendal nerve and recording a motor response from the anal sphincter.¹³⁸

PERIPHERAL NERVE SURGERY (MONITORS: ELECTROMYOGRAM, NERVE ACTION POTENTIAL)

Neurologic monitoring for surgeries involving peripheral nerves can be done in two different settings. In the first setting, the peripheral nerve is intact, but threatened by the surgery. Examples would be an intrinsic nerve tumor, such as a schwannoma or an extensive soft tissue tumor, particularly if it displaces the normal anatomic course of a nerve. Monitoring of spontaneous and stimulated muscle responses from muscle groups innervated by the nerve in question can be used to guide the resection. Spontaneous EMG discharges can be generated by stretch or compression of the nerve, by local heating from electrocautery, or from ischemia. Two caveats apply to the monitoring

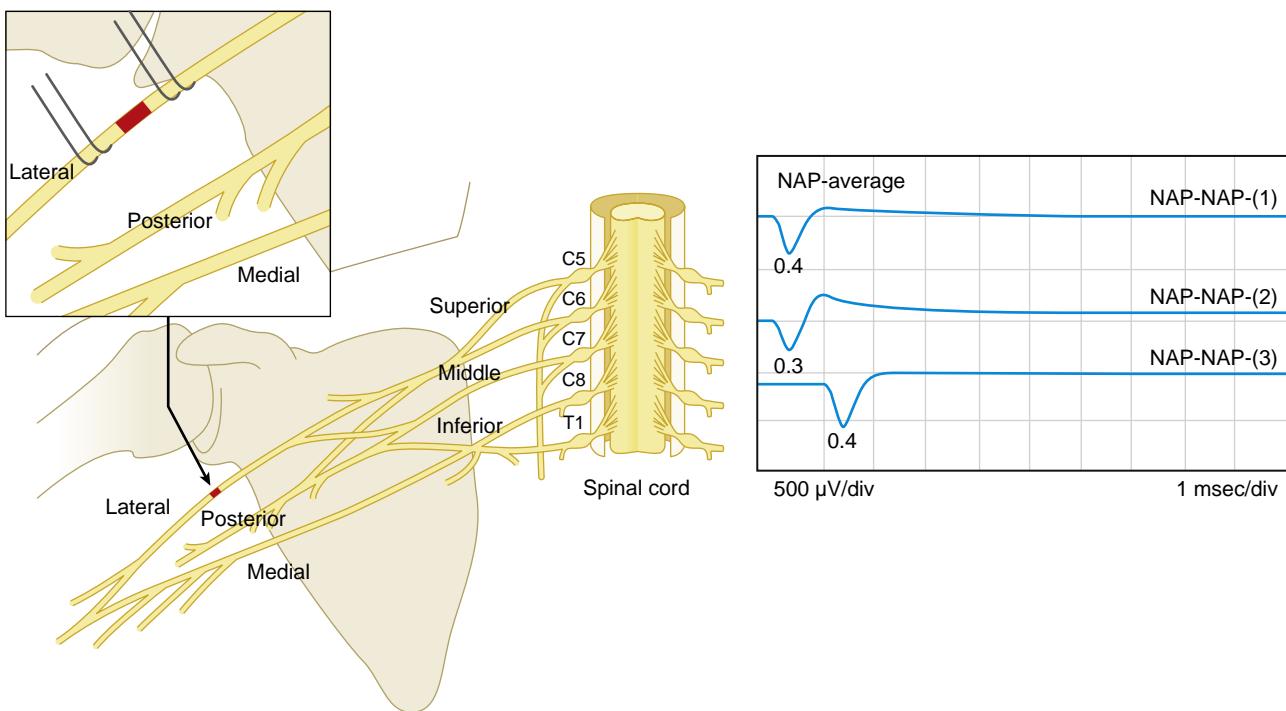


Fig. 39.17 Recording a nerve action potential (NAP) during a brachial plexus exploration. The site of the injury on the lateral fascicle is indicated in red. As shown in the inset, the surgeon places hook electrodes on either side of the exposed part of the nerve. If the injury is limited to an axonotmesis, proximal stimulation will result in a distal NAP similar to the ones shown in the recording to the right. The delay in the response in the third tracing is simply the result of a change in technical settings.

of spontaneous EMG. First, the neuromuscular junction is part of the monitored pathway, and muscle relaxation decreases/abolishes the sensitivity of monitoring in a dose-dependent manner. Second, sharp section of the nerve may not result in a noticeable discharge. To search out the course of the nerve intraoperatively, the surgeon may stimulate the wound area with a hand-held probe and listen for stimulated EMG or palpate for muscle contraction. The underlying concept should be familiar to anesthesiologists from the use of nerve stimulators in regional anesthesia.

A variation of this technique of monitoring that is in widespread use, because it is conceptually simple, is monitoring of pedicle screw placement during spinal instrumentation with the aim of avoiding nerve root injuries owing to malpositioned pedicle screws.^{139,140} The aim is to avoid malpositioned screws that weaken the construct or cause postoperative radicular pain. Typically, the pilot hole or, less desirable, the shank of an implanted pedicle screw is stimulated repetitively with increasing current to determine the threshold for eliciting a dermatomal compound muscle potential. The interpretation of responses is complicated by the fact that the anatomical relationship between pedicles and nerve roots depends on the level of the spinal cord because the spinal cord is shorter than the bony spinal column. Thus a medially misplaced screw in the lumbar region will come to lie next to a nerve root, whereas in the thoracic spine, a medial misplacement puts the screw next to the corticospinal tract, which cannot be activated by single stimuli. Because thresholds vary among cervical, thoracic, and lumbar spine, as well as between healthy and diseased nerve roots, this technique has limitations but is widely thought to be useful.¹⁴¹

A second setting where monitoring of peripheral nerves is used is in patients with prolonged weakness and sensory loss after nerve injury undergoing nerve exploration.¹⁴² The aim is to determine whether nerve reconstruction may improve outcome. The area of the lesion is determined by preoperative nerve conduction studies. Intraoperatively, the nerve is first stimulated proximal to the lesion, and a recording of the nerve action potential is made directly from the nerve distal to the lesion, as illustrated in Fig. 39.17. If there is nerve conduction across the lesion, lysis of scar is performed, and the incision is closed. Natural recovery by means of axonal regrowth produces the best outcome. If conduction does not occur across the lesion, resection of the damaged nerve and nerve cable grafting is performed.^{65,143,144}

APPLICATIONS OF INTRAOPERATIVE MONITORING TO PEDIATRIC PATIENTS

In recent years many of the techniques described above have been used during surgery on children as young as 6 weeks of age. Very young children present special challenges due to the immaturity of the CNS. Incomplete myelination of specific tracts that carry sensory or motor signals is the primary source of the challenges. While light anesthesia (0.5 minimum alveolar concentration [MAC] volatile anesthetic) allows MEPs to be elicited from healthy adolescents, infants and young children are exquisitely sensitive to the effects of volatile anesthetic drugs. Therefore total intravenous anesthesia (TIVA) techniques are the anesthetics of choice when recording evoked potentials from these patients. In addition, adaptive strategies need to be applied by the monitoring team to overcome the effects of myelination delays and other developmental factors (Table 39.2).

TABLE 39.2 Additional Considerations When Monitoring Infants and Young Children

Modality	Observation	Developmental Neurophysiology	Compensation Strategy
SSEPs—MN & UN SSEP—PTN	Prolonged Erb's to cortical or cervical to cortical inter-peak latency Difficult to obtain; low amplitude	Incomplete myelination of medial lemniscus and thalamocortical pathways Incomplete myelination of dorsal columns; asynchronous volleys	Avoid volatile anesthetic drugs, avoid burst suppression, reduce stimulation rate As above, increase pulse length; optimize signal:noise ratio
tcMEP		Cortical and spinal cord motor neurons very susceptible to effects of volatile agents. Variance in conduction velocity of large CST fibers exceeds mean conduction velocity	TIVA, may need low-dose ketamine to help support BP. Double-train stimulation with unequal train lengths; optimizing inter-train interval
D-wave	Difficult to obtain in children under 24 months of age	Signal dispersion due to large variance in conduction velocities	Electrodes with larger surface area may be helpful
ABR	Low quality signals with volatile agents	uncertain	TIVA for ABR for less than 24 months
Bulbocavernosus Reflex	Very susceptible to effects of volatile agents	Oligosynaptic reflex; sensitivity to volatile agents persists	Double pulse train with large inter-train interval

ABR, Auditory brainstem response; *D*-wave, positive or negative deflection recorded from electrodes placed directly over the spinal cord; SSEPs, somatosensory-evoked potentials; tcMEP, transcranial motor-evoked potential; TIVA, total intravenous anesthesia.

Modified from Francis L, Busso V, McAuliffe JJ. Intraoperative neuromonitoring in pediatric surgery. In: Koth A, Sloan TB, Toleikis JR, eds. *Monitoring the Nervous System for Anesthesiologists and Other Health Care Professionals*. Cham: Springer Int; 2017.

Some monitored surgical procedures are performed almost exclusively in children. One example is selective dorsal rhizotomies for relief of spasticity associated with cerebral palsy. This procedure involves interrogation of lower extremity dorsal root subdivisions (rootlets) and evaluating the compound action potentials generated in response. This technique is best performed with a TIVA technique. Infant/toddler hearing evaluations may require the use of auditory brainstem response tests under anesthesia. Experience at the Cincinnati Children's Hospital Medical Center (CCHMC) suggests that a propofol infusion anesthetic yields a more reliable assessment of wave V than a sevoflurane-based anesthetic.

It is important to recognize that the EEG patterns associated with an anesthetic state in adults do not apply to infants under anesthesia. Infants less than 3 months have almost no change in their EEGs when transitioning from a quiet resting state to an anesthetized state as slow wave patterns dominate both states. Alpha and theta patterns emerge under anesthesia at 4 months of age but differ from those of older children and adults.¹⁴⁵

In summary, infants and young children can benefit from intraoperative neurophysiological monitoring if both monitoring and anesthetic techniques are appropriately modified.

NONNEUROLOGIC SURGERY THAT RISKS DAMAGE TO THE CENTRAL NERVOUS SYSTEM (MONITORS: ELECTROENCEPHALogram, TRANSCRANIAL DOPPLER, CEREBRAL OXIMETRY, JUGULAR VENOUS OXYGEN SATURATION)

Cardiopulmonary Bypass

Electroencephalogram. In humans, changes that occur with the institution of CPB may alter the EEG by multiple different mechanisms. Plasma and brain concentrations of anesthetic drugs may be altered by CPB or by anesthetic drugs commonly given during CPB, alterations in arterial carbon dioxide tension and arterial blood pressure may occur, and hemodilution with hypothermic perfusate nearly always

occurs. These effects, all of which may produce EEG changes similar to pathologic changes seen with ischemia, make it difficult to interpret EEG changes occurring during CPB.

Levy and others^{146,147} tried to distinguish the normal effects of hypothermia from other events occurring at the institution and conclusion of CPB. Initially, Levy concluded that only a qualitative relationship could be determined, but later, with the use of a much more sophisticated EEG analysis technique (approximate entropy), EEG changes associated with changes in temperature could be quantified.

Chabot and colleagues¹⁴⁸ and Edmonds and colleagues¹⁴⁹ have attempted to use quantitative (processed, multiple-channel) EEG during CPB to detect cerebral hypoperfusion and relate these changes to postoperative neurologic function. In addition, some minimal work has been done with intervention after detection of cerebral hypoperfusion using quantitative EEG. Although the data seem promising, only a few patients have been studied, with very few corroborating studies. In addition, this type of monitoring is extremely costly in time, personnel, and equipment. Given the lack of convincing outcome data, the cost-to-benefit ratio is unclear at best. Some other investigators failed to show any convincing relationship between intraoperative EEG parameters and postoperative neurologic function, especially in small infants and children.^{150,151} Whether the processed, quantitative EEG provides useful information for clinical management of patients during CPB is not clear. None of the currently available studies and recommendations would support an evidence-based justification for their routine application. However, use of EEG to document electrocortical silence and maximum reduction of cerebral metabolism prior to institution of circulatory arrest seems logical.

Transcranial Doppler. TCD ultrasound can monitor the cerebral circulation during CPB. Anecdotal reports and case series document the use of TCD for determining adequacy of CBF, detection of emboli, and detection of improper cannula placement.¹⁵² Only very limited outcome data exist, and use of TCD ultrasound during CPB does not stand up to evidence-based examination, primarily because of lack

of information. Probe placement instability and inability to obtain signals in some patients also have limited the use of this monitor intraoperatively. Finally, although the hypothesis that cerebral microemboli are at least part of the pathogenesis of postoperative cognitive decline following CPB is attractive, a recent study failed to demonstrate any relationship between CPB counts of presumed emboli and postoperative cognitive dysfunction.¹⁵³

Cerebral Oximetry and Jugular Venous Oxygen Saturation. As is the case with EEG monitoring during CPB, there are multiple case reports and several series that advocate the use of NIRS or jugular venous oxygen saturation ($Sjvo_2$) as indicators of adequate perfusion of the brain during CPB.^{154,155} Incorrect placement of CPB cannulae has been detected clinically and in laboratory studies. One more recent series of patients undergoing CPB for coronary bypass surgery showed a higher incidence of major organ system dysfunction and longer hospital stays in patients with lower baseline and intraoperative cerebral oxygen saturation values.¹⁵⁶ The same questions exist for this application of NIRS, however, as do for the use of NIRS during carotid vascular surgical procedures. Although a recent systematic review of the use of NIRS during CPB showed that data are insufficient to conclude that interventions based on low cerebral oxygen saturation values prevent either stroke or postoperative cognitive dysfunction,¹⁵⁷ several more recent studies suggest significant promise for this modality when used in combination with TCD ultrasound. Use of NIRS and TCD ultrasound together identified a significant percentage of patients undergoing CPB with impaired autoregulation who were at higher risk for postoperative cognitive dysfunction and/or stroke.¹⁵⁸⁻¹⁶⁰ This patient population may benefit from higher mean arterial pressure (MAP) during CPB. Use of NIRS during CPB clearly provides information that would not otherwise be available, but more work is needed before we fully understand the role of this monitor during CPB.

$Sjvo_2$ is very invasive. Although data from case reports and studies suggest that $Sjvo_2$ may have utility in detecting inadequate CBF, lack of outcome data, lack of clearly defined critical values at different temperatures during CPB, and availability of less invasive modalities (EEG, cerebral oximetry) have resulted in only limited use of this monitoring method during CPB. Based on current information, no neurologic monitoring techniques, either alone or in combination, are clearly useful in improving outcome during surgical procedures requiring CPB. Further research is needed before the cost in personnel and equipment of neurologic monitoring during CPB can be justified.

INTENSIVE CARE APPLICATIONS OF NEUROLOGIC MONITORING (MONITORS: ELECTROENCEPHALOGRAM, EVOKED POTENTIALS, TRANSCRANIAL DOPPLER, JUGULAR VENOUS OXYGEN SATURATION)

Secondary injury to the CNS has been recognized in past decades as a major modifiable risk factor in patients with CNS disease. Aneurysmal subarachnoid hemorrhage, stroke, and traumatic brain injury are examples of CNS

insults in which secondary injury has important implications for the ultimate functional outcome.¹⁶¹⁻¹⁶³ These same diseases frequently result in a primary insult to the CNS that severely constrains the utility of the clinical neurologic examination because of the need for mechanical ventilation and sedation. Many techniques of neurologic monitoring discussed earlier are used in the intensive care unit. Generally, however, techniques that require the continued presence of skilled technologists, such as monitoring of evoked potentials, are prohibitively expensive and of less practical value than techniques that provide data that easily integrates into the physiologic support provided through intensive care or techniques that can be performed as daily assessments. Some of this neurophysiologic data can provide important prognostic information in comatose patients and guide decision making.

Continuous Electroencephalogram Monitoring

Continuous EEG monitoring may be of benefit in comatose patients. It facilitates timely intervention for specific diagnoses, such as nonconvulsive seizures, as the underlying cause of a fluctuating neurologic status, or point to focal problems such as regional ischemia due to vasospasm after subarachnoid hemorrhage.¹⁶⁴ The perceived utility of continuous EEG monitoring has led to a degree of standardization in indications and the logistics of the application of this technique in the intensive care unit.^{165,166}

Cerebral Ischemia

Cerebral ischemia is an important cause of secondary injury to the CNS. It can be difficult to detect in patients who are either comatose or sedated, but can occur even in patients with adequate cerebral perfusion pressure.^{167,168} Three techniques may provide intensivists with additional information about cerebral perfusion. None of the monitors is considered “standard of care.” As with all monitors, the impact of the monitor on outcome depends on the quality of the therapeutic interventions that result from integration of the additional data into the clinical management of a given patient.

$Sjvo_2$ monitoring is used most extensively in the intensive care unit to monitor patients with traumatic brain injury. The data have been used to guide blood pressure and ventilatory management to optimize blood flow. $Sjvo_2$ monitoring has had a major effect on ventilatory management of head-injured patients and has significantly reduced the routine use of hyperventilation in neurosurgical patients.¹⁶⁹⁻¹⁷² $Sjvo_2$ values of less than 50% generally indicate cerebral ischemia. Increases in $Sjvo_2$ may occur in response to therapy, or they may be an ominous sign if the increase is caused by falling demand because of neuronal death.

Similar to $Sjvo_2$ monitoring, monitoring of P_{BrO_2} and blood flow is used most frequently in patients with traumatic brain injury. P_{BrO_2} performs well in clinical practice. Decreases to less than 10 to 15 mm Hg are associated with worsening outcome,^{22,173} whereas P_{BrO_2} -targeted treatment strategies may improve outcome.¹⁷⁴

TCD ultrasound is widely used in the intensive care unit to document the presence and severity of cerebral vasospasm after subarachnoid hemorrhage. As the

major cerebral arteries narrow, flow velocity within the lumen must increase if blood flow is to be maintained. Such narrowing occurs 12 to 24 hours before the onset of clinical symptoms, thus allowing therapy to be initiated before the onset of clinical symptoms.¹⁷⁵⁻¹⁷⁹ Mean flow velocities of more than 120 cm/s seem to correlate well with angiographic vasospasm,^{180,181} although intracranial pressure and concurrent therapy to raise cerebral perfusion pressure modify the flow velocity. The latter two factors result in characteristic changes of the TCD waveform, however, and preserve the utility of the examination.

Prognosis in Coma and Determination of Brain Death

EEG monitoring may help to assess the clinical course and the prognosis of comatose patients. Assessment of prognosis must be separated from the insult that precipitated the coma by more than 24 hours. If not, the EEG may reflect predominantly the effect of the insult and may not predict prognosis. More than 24 hours after the insult, spontaneous sustained burst suppression correlates strongly with severe irreversible brain injury.¹⁸² Absence of EEG variability portends a high likelihood of persistent vegetative state or death,^{182,183} whereas spontaneous variability, reactivity to external stimuli, and typical sleep patterns are associated with more favorable outcomes.¹⁸⁴⁻¹⁸⁶

A specific indication for EEG monitoring is the therapeutic induction of a coma by barbiturate administration. Because neither blood nor cerebrospinal fluid concentrations of barbiturates reliably predict burst suppression and near-maximal reduction in cerebral metabolic rate of oxygen consumption,¹⁸⁷ and because barbiturate administration usually requires an increase in cardiovascular support, documentation of a burst suppression pattern on EEG allows the use of the minimal effective dose of barbiturate.

Similar to EEG, evoked potential studies have a place in predicting prognosis in comatose patients.¹⁸⁸ The presence of normal SSEPs bilaterally is an excellent prognostic sign, whereas the absence of any SSEP cortical response is a poor prognostic indicator. The degree of bad outcome can be predicted by BAEPs. Intact and normal BAEPs with absent cortical SSEPs predict a best outcome of a chronic vegetative state. Outcome may be worse, however, because BAEPs commonly deteriorate later with rostral-to-caudal deterioration. Absent BAEP responses beyond wave I predict a high likelihood of brain death. Present but abnormal SSEPs are associated with outcomes intermediate between good to high function and a chronic vegetative state.¹⁸⁹⁻¹⁹⁸

TCD ultrasound also has been used in the intensive care unit as an aid to the diagnosis of brain death. As intracranial pressure increases, the pulsatility of the TCD waveform increases, accentuating the systolic peak and diminishing flow during diastole. With further increases in intracranial pressure, a characteristic to-and-fro pattern of flow is established, which is consistent with clinical brain death.¹⁹⁹ TCD studies are easily performed at the bedside and can minimize the need for unnecessary transports of the patient for definitive radiologic studies.

Nonsurgical Factors Influencing Monitoring Results

ANESTHESIA AND THE ELECTROENCEPHALOGRAPH

Anesthetic drugs affect the frequency and amplitude of EEG waveforms. Although each drug class and each specific drug has some specific, dose-related EEG effects (Table 39.3), some basic anesthesia-related EEG patterns may be described. Subanesthetic doses of intravenous and inhaled anesthetics usually produce an increase in frontal beta activity and abolish the alpha activity normally seen in the occipital leads in an awake, relaxed patient with the eyes closed. As the patient loses consciousness with general anesthesia, the brain waves become larger in amplitude and slower in frequency. In the frontal areas, small beta activity seen in an awake patient slows to the alpha range and increases in size. In combination with the loss of the occipital alpha activity, this phenomenon produces the appearance of a “shift” of the alpha activity from the posterior cortex to the anterior cortex. Further increases in the dose of inhaled or intravenous anesthetic drugs produce further slowing of the EEG. Some anesthetic drugs suppress EEG activity totally (see Table 39.3). Other anesthetic drugs never produce burst suppression or an isoelectric EEG, despite increasing dose, either because they are incapable of completely suppressing the EEG (e.g., opioids, benzodiazepines) or because cardiovascular toxicity of the drug (e.g., halothane) prevents administration of a large enough dose.

Intravenous Anesthetic Drugs

Barbiturates, Propofol, and Etomidate. Despite widely varying potencies and durations of action, barbiturates, propofol, and etomidate produce similar EEG patterns (Fig. 39.18 shows EEG effects of thiopental). These drugs all follow the basic anesthesia-related EEG pattern described previously with initial EEG activation (see Fig. 39.18A), followed by dose-related depression. As the patient loses consciousness, characteristic frontal spindles are seen (see Fig. 39.18B), which are replaced by polymorphic 1- to 3-Hz activity (see Fig. 39.18C) as the drug dose is increased. Further increases in dose result in lengthening periods of suppression interspersed with periods of activity (burst suppression). With a very high dose, EEG silence results. All of these drugs have been reported to cause epileptiform activity in humans, but epileptiform activity is clinically significant only after methohexitol and etomidate when given in subhypnotic doses.

Ketamine. Ketamine does not follow the basic anesthesia-related EEG pattern. Anesthesia with ketamine is characterized by frontally dominant rhythmic, high-amplitude theta activity. Increasing doses produce intermittent polymorphic delta activity of very large amplitude interspersed with low-amplitude beta activity.²⁰⁰ Electrocortical silence cannot be produced with ketamine. EEG activity may be very disorganized and variable at all doses. This disorganization of the EEG with ketamine is responsible for the failure of the bispectral index (BIS) to be useful in looking at the effect of ketamine on consciousness.²⁰¹ Recovery of normal EEG

TABLE 39.3 Anesthetic Drugs and Electroencephalogram

Drug	Effect on EEG Frequency	Effect on EEG Amplitude	Burst Suppression?
Isoflurane, Sevoflurane, Desflurane			Yes, >1.5 MAC
Subanesthetic	Loss of alpha, ↑frontal beta		
Anesthetic	Frontal 4-13 Hz activity		
Increasing dose >1.5 MAC	Diffuse theta and delta → burst suppression → ↓0		
Nitrous oxide (alone)	Frontal fast oscillatory activity (>30 Hz)	↓, especially with inspired concentration >50%	No
Barbiturates			Yes, with high doses
Low dose	Fast frontal ↑ beta activity	Slight	
Moderate dose	↑Frontal alpha frequency spindles		
Increasing high dose	Diffuse delta → burst suppression → silence	↓0	
Etomidate			Yes, with high doses
Low dose	Fast frontal beta activity		
Moderate dose	Frontal alpha frequency spindles		
Increasing high dose	Diffuse delta → burst suppression → silence	↓0	
Propofol			Yes, with high doses
Low dose	Loss of alpha; frontal beta		
Moderate dose	Frontal delta; waxing/waning alpha		
Increasing high dose	Diffuse delta → burst suppression → silence	↓0	
Ketamine			No
Low dose	Loss of alpha, variability	↓	
Moderate dose	Frontal rhythmic delta		
High dose	Polymorphic delta; some beta	Beta is low amplitude	
Benzodiazepines			No
Low dose	Loss of alpha; increased frontal beta activity		
High dose	Frontally dominant delta and theta		
Opioids			No
Low dose	Loss of beta; alpha slows	None	
Moderate dose	Diffuse theta, some delta		
High dose	Delta, often synchronized		
Dexmedetomidine	Moderate slowing, prominent spindles		No

Alpha = 8-13 Hz frequency; beta = >13 Hz frequency; delta = <4 Hz frequency; theta = 4-7 Hz frequency. EEG, Electroencephalogram; MAC, minimum alveolar concentration.

activity even after a single bolus dose of ketamine is slow compared with barbiturates. There is no information available about the relationship between emergence reactions after ketamine and the EEG. Ketamine also has been associated with increased epileptiform activity.²⁰⁰

Benzodiazepines. Despite varying potencies and durations of action, benzodiazepines also follow the basic anesthesia-related EEG pattern. As a class, however, these drugs are incapable of producing burst suppression or an isoelectric EEG.

Opioids. As a class, opioids do not follow the basic anesthesia-related EEG pattern. Opioids generally produce a dose-related decrease in frequency and increase in amplitude

of the EEG. If no further doses of opiates are given, alpha and beta activity return as drug redistribution occurs. The rapidity of return depends on the initial dose and on the drug. In rats, remifentanil is associated with the most rapid return to normal.²⁰² Complete suppression of the EEG cannot be obtained with opioids. Epileptiform activity occurs in humans and in animals receiving large to supraclinical doses of opioids. Sharp wave activity is common after induction of anesthesia with fentanyl, with 20% of patients showing this phenomenon after 30 µg/kg; 60%, after 50 µg/kg; 58%, after 60 µg/kg; and 80%, after 70 µg/kg.²⁰³ Alfentanil bolus has been used clinically to activate seizure foci during epilepsy surgery.²⁰⁴ This epileptiform activity is mainly noted in the frontotemporal region.

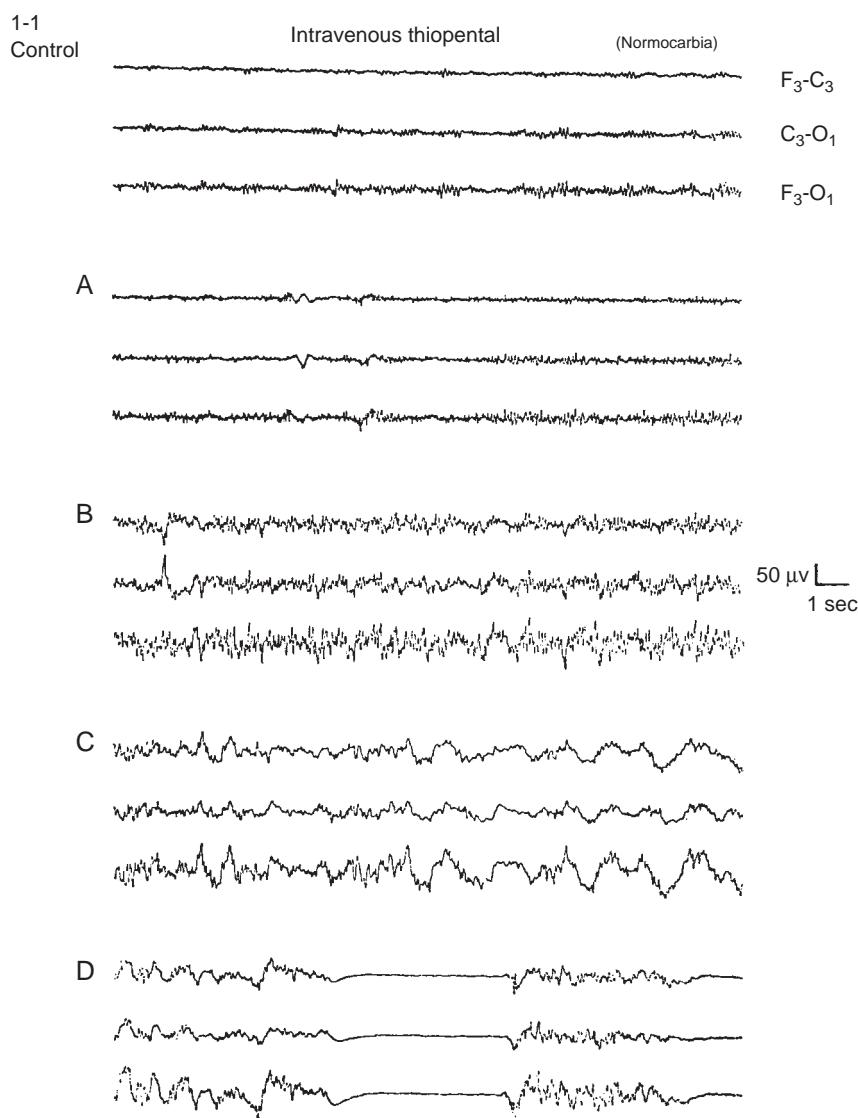


Fig. 39.18 Electroencephalogram effects of intravenous administration of thiopental in humans. (A) Rapid activity. (B) Barbiturate spindles. (C) Slow waves. and (D) Burst suppression. (From Clark DL, Rosner BS. Neurophysiologic effects of general anesthetics. *Anesthesiology*. 1973;38:564.)

Dexmedetomidine. Dexmedetomidine is being increasingly used for sedation in the operating room and in the intensive care unit, and is also used for sedation during diagnostic EEG studies in children. EEG studies of patients undergoing sedation with dexmedetomidine alone show patterns similar to those seen in normal human sleep with increased slow-wave activity and sleep spindles prominent.²⁰⁵ Burst suppression or an isoelectric EEG pattern cannot be produced even with high doses of the drug. Level of sedation with dexmedetomidine can be effectively monitored using processed EEG parameters and has been reported using BIS and entropy techniques.²⁰⁶ Interestingly, BIS values at comparable levels of sedation appear to be lower with dexmedetomidine than propofol.²⁰⁷

Inhaled Anesthetics

Nitrous Oxide. Used alone, nitrous oxide causes a decrease in amplitude and frequency of the dominant occipital alpha rhythm. With the onset of analgesia and depressed consciousness, frontally dominant fast oscillatory activity

(>30 Hz) is frequently seen.²⁰⁸ This activity may persist to some extent for 50 minutes after discontinuation of nitrous oxide. When nitrous oxide is used in combination with other agents, it increases the effects that would be associated with the agent alone clinically and with respect to the EEG pattern seen.

Isoflurane, Sevoflurane, and Desflurane. Potent inhaled anesthetic drugs, including halothane and enflurane, which are no longer available, follow the basic anesthesia-related EEG pattern. Isoflurane initially causes an activation of the EEG followed by a slowing of the EEG activity that is more marked with increasing dose. Isoflurane begins to produce periods of EEG suppression at 1.5 MAC, which become longer with increasing dose until electric silence is produced at 2 to 2.5 MAC. Isolated epileptiform patterns sometimes can be seen during intersuppression activity at 1.5 to 2 MAC isoflurane.²⁰⁹ Sevoflurane causes similar dose-dependent EEG effects. Equi-MAC concentrations of sevoflurane and isoflurane

TABLE 39.4 Ability of an Individual Anesthetic Drug to Produce a Change in Sensory- and Motor-Evoked Potentials That Could Be Mistaken for a Surgically Induced Change

Drug	SSEPs		BAEPs		VEPs		TRANSCRANIAL MEPS	
	LAT	AMP	LAT	AMP	LAT	AMP	LAT	AMP
Isoflurane	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Nitrous oxide*	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Barbiturates	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Etomidate	No	No	No	No	Yes	Yes	No	No
Propofol	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Diazepam	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Midazolam	Yes	Yes	No	No	Yes	Yes	Yes	Yes
Ketamine	No	No	No	No	Yes	Yes	No	No
Opioids	No	No	No	No	No	No	No	No
Dexmedetomidine	No	No	No	No	No	ND	ND	No

*Increases the anesthetic effect of the drug or drugs with which it is used.

Note. This table is not quantitative in any way. "Yes" or "No" designations indicate whether an individual drug is capable of producing an effect on any portion of the evoked response that could be mistaken for a surgically induced change.

AMP, Amplitude; BAEPs, brainstem auditory-evoked potentials; LAT, latency; MEPS, motor-evoked potentials; ND, no data available from the literature; SSEPs, somatosensory-evoked potentials; VEPs, visual-evoked potentials.

cause similar EEG changes.²¹⁰ Epileptiform activity has been induced by administration of sevoflurane in patients without epilepsy, and seizure activity on EEG, but not clinical seizure activity, has been reported in pediatric patients with a history of epilepsy during induction of anesthesia with sevoflurane.^{211,212} Despite these observations, sevoflurane, similar to other inhalation drugs, is not suitable for use during electrocorticography for localization of seizure foci.²¹³ EEG patterns seen with enflurane are similar to the patterns seen with isoflurane except that epileptiform activity is considerably more prominent. At 2 to 3 MAC, burst suppression is seen, but virtually all intersuppression activity consists of large spike/wave pattern discharges. Hyperventilation with high concentrations of enflurane increases the length of suppression, decreases the duration of bursts, but increases the amplitude and main frequency component of the intersuppression epileptiform activity. Frank EEG seizures also may occur with enflurane that produce the same cerebral metabolic effects as pentylenetetrazol, a known convulsant.

Halothane also produces EEG patterns similar to those of isoflurane, but dosages of halothane that would produce burst suppression in the EEG (3-4 MAC) are associated with profound cardiovascular toxicity. Desflurane produces EEG changes similar in nature to equi-MAC concentrations of isoflurane. In limited clinical studies, there has been no evidence of epileptiform activity with desflurane, despite hyperventilation and 1.6 MAC dosage,²¹⁴ and desflurane has been used as a treatment of refractory status epilepticus.²¹⁵

Clinical studies have shown that the EEG effects of inhaled anesthetic drugs are influenced by age and baseline EEG characteristics. Older patients and patients with EEG slowing at baseline were more sensitive to the EEG effects of isoflurane and desflurane. As anesthesia was deepened, similar EEG pattern changes were noted, but these changes occurred at lower end-tidal anesthetic concentrations.²¹⁶

BOX 39.1 Guidelines for Choosing Anesthetic Techniques During Procedures in Which Sensory-Evoked Responses Are Monitored

1. Intravenous drugs have significantly less effect than "equipotent" doses of inhaled anesthetics
2. Combinations of drugs generally produce "additive" effects
3. Subcortical (spinal or brainstem) sensory-evoked responses are very resistant to the effects of anesthetic drugs. If subcortical responses provide sufficient information for the surgical procedure, anesthetic technique is not important, and effects on cortically recorded responses may be ignored

ANESTHESIA AND SENSORY-EVOKED RESPONSES

Volatile Anesthetics

Multiple drugs used in the perioperative period can influence the ability to monitor SERs accurately (Table 39.4). An extensive review from 2003 provides the interested clinician with a detailed analysis of all drug effects on SERs,²¹⁷ which is beyond the scope of this chapter. Table 39.4 does not quantify drug effects, but rather lists whether an individual drug is capable of producing a change in any part of an evoked response that could be mistaken for a surgically induced change. A "no" designation in this table does not mean that there are no effects of a given drug on SERs. The "no" designation indicates that any effects that do occur would not be called clinically significant by *clinicians experienced in intraoperative monitoring*. Several general concepts (Box 39.1) help the clinician who is trying to determine the best choice of drugs for use during monitored cases.

The volatile anesthetics isoflurane, sevoflurane, desflurane, enflurane, and halothane have similar effects in differing degrees on all types of SERs. VEPs are the most sensitive

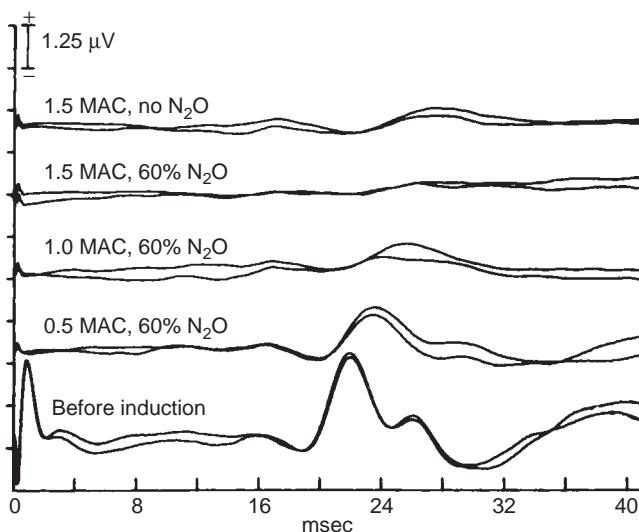


Fig. 39.19 Representative somatosensory-evoked potential cortical responses (C-3, C-4-FPz) at various minimum alveolar concentration (MAC) levels of isoflurane. (From Peterson DO, Drummond JC, Todd MM. Effects of halothane, enflurane, isoflurane, and nitrous oxide on somatosensory-evoked potentials in humans. *Anesthesiology*. 1986;65:35.)

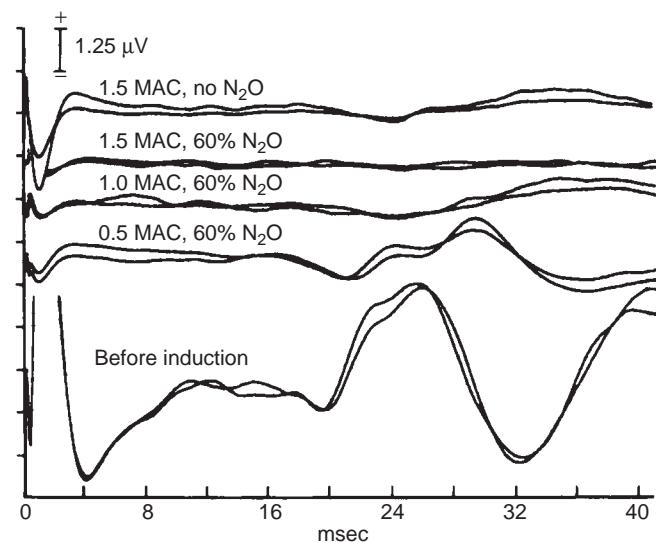


Fig. 39.20 Representative somatosensory-evoked potential cortical responses (C-3, or C-4-FPz) at various minimum alveolar concentration (MAC) levels of enflurane. (From Peterson DO, Drummond JC, Todd MM. Effects of halothane, enflurane, isoflurane, and nitrous oxide on somatosensory-evoked potentials in humans. *Anesthesiology*. 1986;65:35.)

to the effects of volatile anesthetics, and BAEPs are the most resistant to anesthetic-induced changes. Spinal and subcortical SSEP responses are significantly less affected than cortical potentials.²¹⁸⁻²²⁰

SSEPs, because they are the most widely used intraoperative SER technique, are the most completely studied with respect to the effects of anesthetic drugs. The effects of the currently used volatile anesthetics on cortical SSEPs are dose-dependent increases in latency and conduction times and a decrease in amplitude of cortically, but not subcortically recorded signals.²²¹ When comparing the different volatile agents, studies have reported conflicting results.^{218,220} None of these differences are clinically important and may be ignored by the practicing clinician. With respect to the newer agents, desflurane and sevoflurane seem to have qualitatively and quantitatively similar effects on SERs as isoflurane.²²²⁻²²⁵ In neurologically normal patients, 0.5 to 1 MAC of any of the potent inhaled agents in the presence of nitrous oxide is compatible with monitoring of cortical SSEPs (Figs. 39.19–39.21).²¹⁷ Neurologically impaired patients may show a significantly greater sensitivity to inhaled agents, even to the point of not tolerating any recordable level of inhaled agent. Generally, better monitoring conditions are obtained, however, with narcotic-based anesthetics with less than 1 MAC total (nitrous oxide plus potent agent) end-tidal inhaled anesthetic concentration.

The volatile anesthetics result in increases in latency of BAEPs without significantly affecting the amplitude.²²⁶⁻²²⁸ Volatile anesthetics cause increases in latency and decreases in amplitude in the early (middle latency) cortical responses after auditory stimulation,²²⁷ however, and these middle latency responses are now being used to monitor the hypnotic component of general anesthetics.²²⁸ Adequate monitoring of BAEPs is possible with any clinically useful concentrations of inhaled agents (with or without nitrous oxide) (Figs. 39.22 and 39.23).

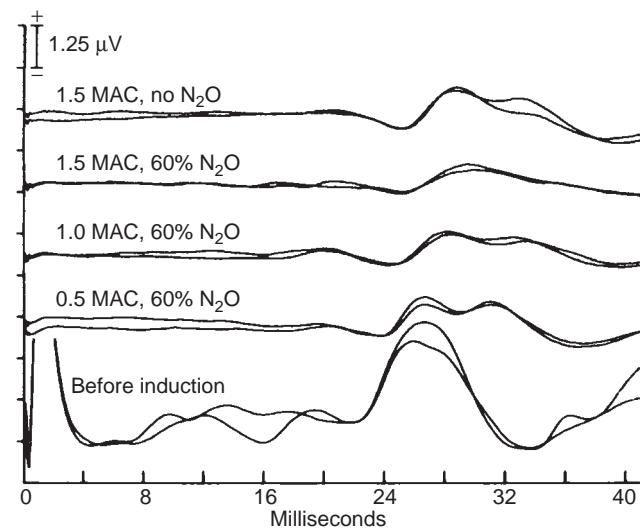


Fig. 39.21 Representative somatosensory-evoked potential cortical responses (C-3, C-4-FPz) at various minimum alveolar concentration (MAC) levels of halothane. (From Peterson DO, Drummond JC, Todd MM. Effects of halothane, enflurane, isoflurane, and nitrous oxide on somatosensory-evoked potentials in humans. *Anesthesiology*. 1986;65:35.)

Use of the volatile anesthetic drugs during monitoring of VEPs results in dose-dependent increases in latency with or without changes in amplitude.²²⁹ Isoflurane results in dose-dependent increases in latency and decreases in amplitude up to 1.8% in 100% oxygen, at which time the waveform is lost.²²¹ More recent studies report some success in recording intraoperative VEPs from patients with normal eyesight, but waveform variability and potent depression of waveforms by volatile agents remain concerns.^{230,231} In the opinion of many experts, the variability of VEPs in anesthetized patients is so great that satisfactory monitoring is impossible using any anesthetic technique.

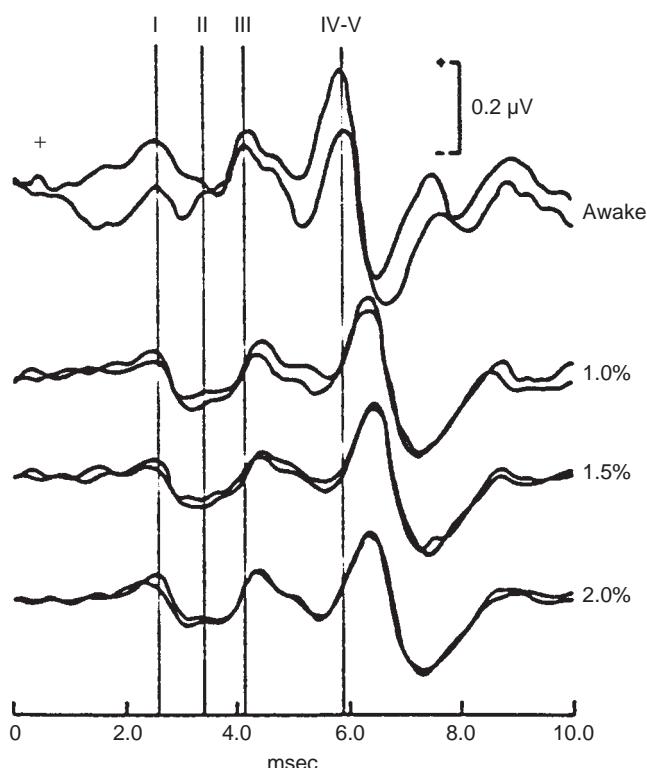


Fig. 39.22 Influence of isoflurane alone on brainstem auditory-evoked potential in a typical subject. Latency of peaks III and IV to V increased at 1.0% but stabilized with increasing anesthetic depth. (From Maninen PH, Lam AM, Nicholas JF. The effects of isoflurane–nitrous oxide anesthesia on brainstem auditory evoked potentials in humans. *Anesth Analg*. 1985;64:43.)

Although volatile anesthetics cause significant changes in the SER waveforms, it is possible to provide adequate monitoring intraoperatively in the presence of anesthetic doses of volatile anesthetics. Doses of anesthetic drugs causing significant depression of the response to be monitored must be prevented. In our experience, end-tidal concentrations of inhaled anesthetic drugs totaling greater than 1.3 MAC have a dose-related, increasing probability of obliterating cortical SSEPs even in neurologically normal patients. Equally important, anesthetic concentration should not be changed during the critical periods of intraoperative monitoring. Critical periods are defined as periods in which surgical interventions are most likely to result in damage to neurologic tissue and changes in the SERs. Because the volatile anesthetic-induced changes in SERs are dose dependent, increasing anesthetic dosage at a crucial point in the operative procedure can result in confusing changes in the SERs that potentially may be caused by the anesthetic, the surgical procedure, or both. The appropriate intervention is difficult to determine.

As with the volatile anesthetics, nitrous oxide causes differing effects on the SERs depending on the sensory system monitored. It causes decreases in amplitude without significant changes in latency in SSEPs when used alone or when added to a narcotic-based or volatile anesthetic.^{217,218,232} The addition of nitrous oxide to a maintenance volatile anesthetic during the monitoring of BAEPs causes no further change.²²⁶ Likewise, use of nitrous oxide alone causes no change in BAEPs, unless gas accumulates in the middle ear.²³² Use of nitrous oxide alone results in an increase in

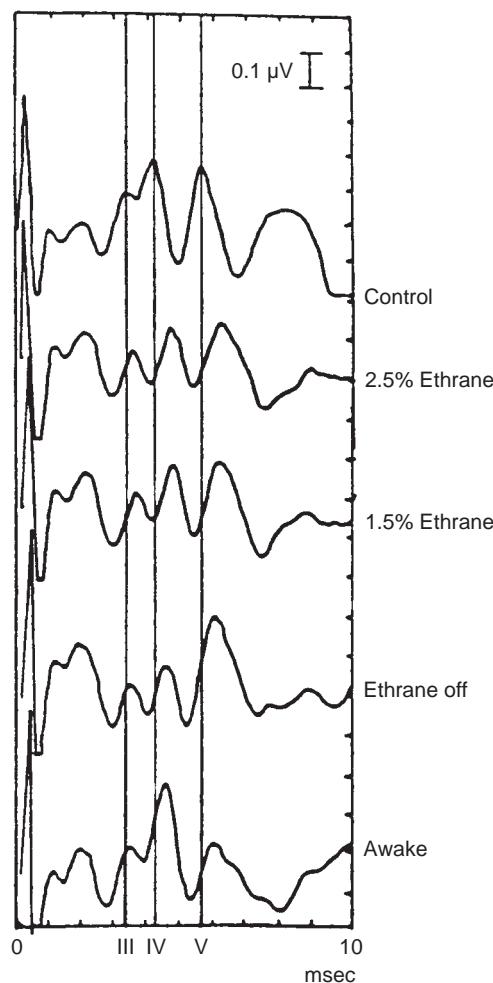


Fig. 39.23 Brainstem auditory-evoked potential recording obtained in one patient at different enflurane (Ethrane)-inspired concentrations. (From Dubois MY, Sato S, Chassy J, et al. Effects of enflurane on brainstem auditory evoked responses in humans. *Anesth Analg*. 1982;61:898.)

latency and a decrease in amplitude in VEPs, but when it is added to a volatile anesthetic technique, it causes no further changes in VEPs.^{229,232}

Intravenous Anesthetics

The effects of propofol on SERs have been studied in many different settings. At typical clinical doses required for general anesthesia, propofol has minimal effects on somatosensory-evoked responses recorded along the somatosensory pathway up to the early cortical potentials.^{233,234} Thus propofol-based TIVA is frequently used as the preferred technique to optimize signal-to-noise ratio for SSEPs and provide rapid feedback to the surgeon.²³⁵ BAEPs under propofol anesthesia show minor increases in interpeak latencies and decreases in amplitude that are insufficient to interfere with clinical monitoring of auditory function.^{236,237}

The effects of barbiturates on SERs have been studied in animals and humans. Increasing doses of thiopental in patients result in progressive dose-dependent increases in latency, decreases in amplitude of SSEPs, and progressive increases in latency of wave V in BAEPs. The changes in SSEPs are more pronounced than the changes in BAEPs, and waveforms beyond the initial primary cortical response are quickly obliterated. This finding is consistent with the theories that

barbiturates affect synaptic transmission more than axonal conduction. Early waveforms in SERs result primarily from axonal transmission, and later waves depend on multisynaptic pathways in addition to axonal transmission. At doses of thiopental far greater than doses producing an isoelectric EEG, adequate monitoring of early cortical and subcortical SSEPs and BAEPs was preserved.²³⁸ Other barbiturate compounds show similar effects.²³⁹ This observation is important, especially when attempting to monitor the adequacy of CBF during cerebrovascular surgery when the patient has been given large, "protective" doses of barbiturates. The EEG is isoelectric and not helpful for monitoring. The early cortical SSEP waveforms are still preserved, however, and may be very helpful in determining adequacy of CBF. Preserved ability to monitor SSEPs in head-injured patients receiving therapeutic thiopental infusions has been demonstrated.²⁴⁰ VEPs are much more sensitive to barbiturates. Low barbiturate doses obliterate all except the earliest waveforms. In cats, the early potentials persisted with increases in latency even to very high pentobarbital doses.²⁴¹ Except for VEPs, adequate perioperative monitoring of SERs is possible even in the presence of high-dose barbiturate therapy as long as the effects of the drug (increased latency with moderately decreased amplitude) are considered.

After bolus administration and intravenous infusions, etomidate causes increases in latency of all waves and prolongation of central conduction time in SSEPs. In contrast to virtually all other commonly used anesthetics, etomidate causes increases in amplitude of the cortical SSEP.^{242,243} This effect may be due to an alteration in the balance of inhibitory and excitatory influences or an increase in the irritability of the CNS. This effect seems to be present in the cortex, but not in the spinal cord. Etomidate infusions have been used to enhance SSEP recording in patients when it was impossible to obtain reproducible responses at the beginning of intraoperative monitoring because of the pathology (Fig. 39.24). Following baseline responses that could not be monitored, the etomidate augmentation of the SSEP allowed adequate monitoring and detection of intraoperative events leading to compromise of the spinal cord.²⁴³ The effects of etomidate on BAEPs are dose-dependent increases in latency and decreases in amplitude that are not clinically significant.²⁴⁴

Benzodiazepines also can cause changes in SERs.^{245,246} Diazepam causes increases in latency and decreases in amplitude of SSEPs, increases in latency in the cortical response after auditory stimulation, and no change in BAEPs. Midazolam causes decreases in amplitude without changes in latency of SSEPs.²⁴²

Generally, opioids cause small dose-dependent increases in latency and decreases in amplitude of SSEPs. These changes are not clinically significant. Effects on amplitude are more variable than the latency increases.^{247,248} Even at large doses of fentanyl (60 µg/kg), reproducible SSEPs can be recorded. Other opioids cause similar dose-dependent changes in SSEPs.²⁴⁹ Opioids can be used even in high doses in patients requiring intraoperative SSEP monitoring without impairment of ability to monitor neurologic function adequately. Opioid-induced changes must be taken into account, however, when evaluating the recordings. Large intravenous bolus administration of opioids should be avoided at times of potential surgical compromise to neurologic function to prevent confusing the interpretation of SEP changes if they develop. BAEPs were resistant to doses

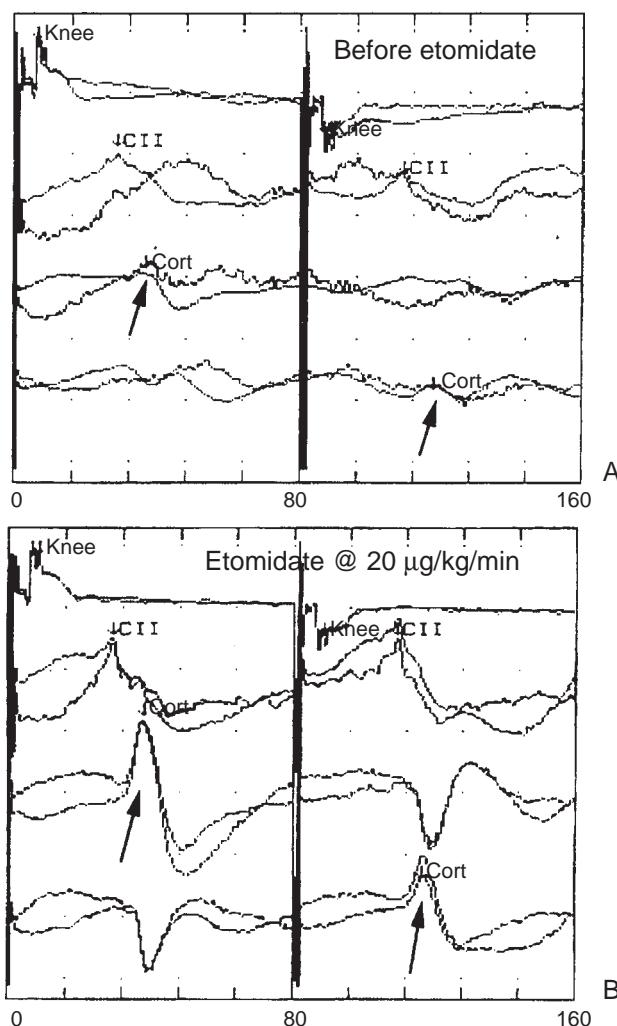


Fig. 39.24 Effects of etomidate on somatosensory-evoked potential. (A) These tracings were obtained from a mildly mentally impaired patient with severe kyphoscoliosis during the early maintenance phase of anesthesia using isoflurane and fentanyl. (B) These tracings were obtained after discontinuing isoflurane and instituting an etomidate infusion at 20 µg/kg/minute. Note dramatically increased amplitude and clarity of the signal in the cortical (Cort) channels (marked by arrows), which both are recorded with the same amplification scale.

of fentanyl of 50 µg/kg with no changes observed in absolute latency, interpeak latency, or amplitude.²⁵⁰

Based on several case reports and small series, dexmedetomidine is compatible with all types of evoked potential monitoring,²⁵¹ although data regarding MEPs are not entirely consistent. One recent study demonstrated significant attenuation of MEPs during scoliosis surgery when dexmedetomidine was used as an adjunct to propofol and remifentanil.²⁵² Data are limited, and large studies are lacking entirely. As the use of this drug increases, more data should become available, but at this time, use of dexmedetomidine does not seem to be clearly problematic.

ANESTHESIA AND MOTOR-EVOKED POTENTIALS

Effects of anesthetics on tcMEPs recorded from muscle are surprisingly profound (see Table 39.4).²⁵³⁻²⁵⁷ Anesthetic techniques typically used by most anesthesiologists for spine surgery would produce prohibitive depression of the MEP.^{258,259} Investigators showed in several studies that

intravenous anesthetic drugs produce significantly less depression, and techniques using any of a combination of ketamine, opiates, etomidate, and propofol have been described.^{255,256,260-263} The authors have had excellent experience with a combination of propofol and remifentanil, which also is supported in the literature.

Anesthetic effects on MEP responses recorded at spinal levels seem to be less serious. When responses are recorded from muscle, neuromuscular blocking drugs should be monitored quantitatively, maintaining T1 twitch height at around 30% of control values to prevent excessive movement during the operation.^{126,254} When responses are not recorded from muscle, profound relaxation is desirable because gross muscle movement produced by MEP stimulation is eliminated, facilitating the surgical procedure. More recent studies using rapid trains of stimuli with transcranial

electric and magnetic stimulus techniques have produced responses that are more resistant to the effects of anesthetic drugs, and more “traditional” techniques using inhaled anesthetics and narcotics may be used.²⁶³⁻²⁶⁵ Most studies support the use of TIVA as preferable to techniques using nitrous oxide or potent inhaled anesthetic drugs, however. Precise control of the anesthetic and avoidance of boluses during critical monitoring periods seem to be even more important than for SSEPs, and active cooperation of the anesthesia care team is essential for good, reproducible results. Fig. 39.25 shows the dramatic effect of introduction of 0.3 MAC isoflurane to a total intravenous technique using propofol and remifentanil. Given ability to monitor the hypnotic component of a TIVA technique with several different available monitors, the authors recommend a total intravenous technique without relaxant whenever possible.

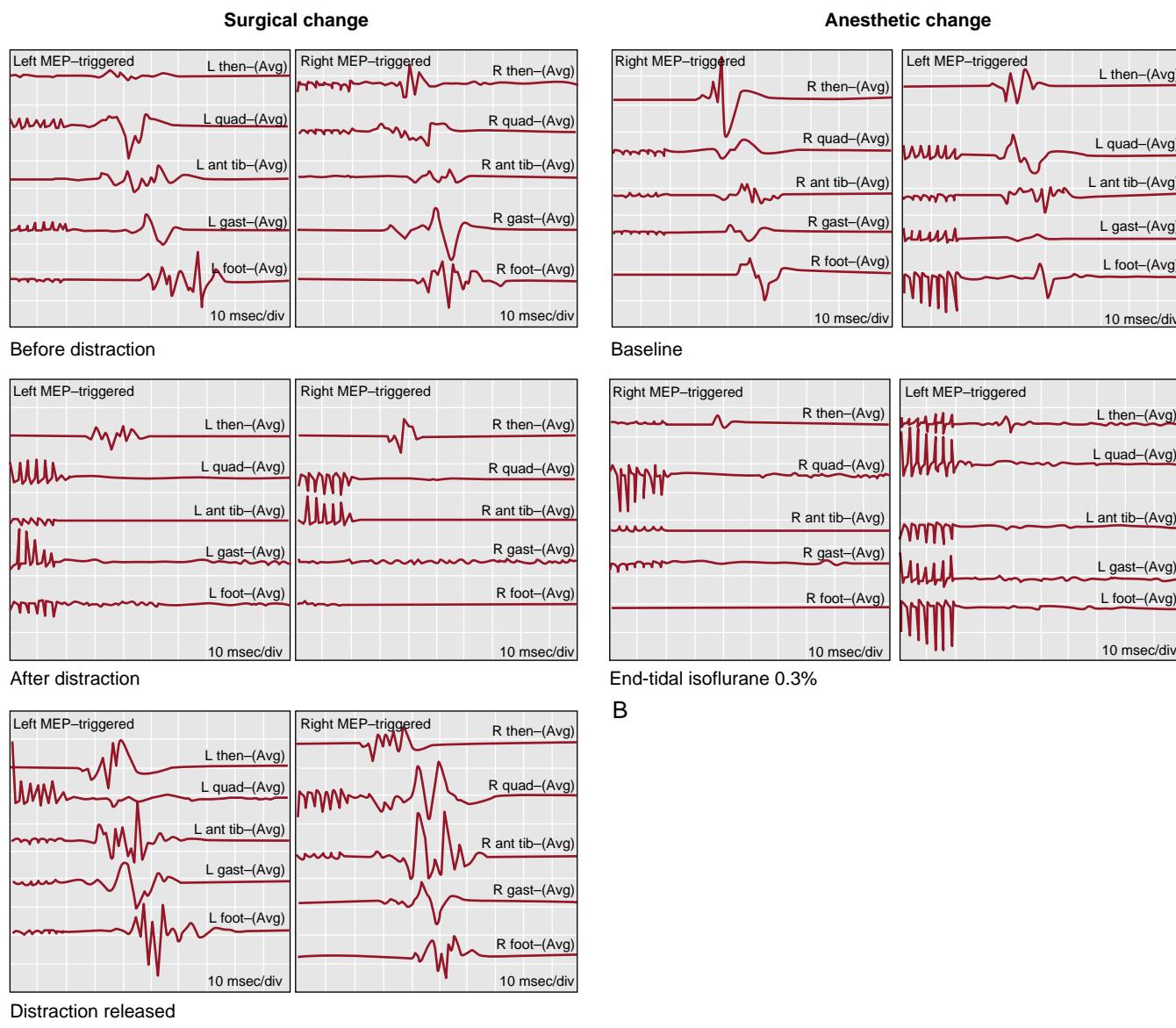


Fig. 39.25 (A) Transcranial electrical motor-evoked potential recording showing surgically induced change during spine surgery (scoliosis repair). (B) Motor-evoked potential with an anesthetic-induced change. Note similarity of the change pattern, except that in the anesthetic-induced change, the responses in the upper extremity also changed. Left-sided and right-sided responses are shown on the corresponding panel. A single upper extremity response is shown (top tracing) in each panel. Responses from four muscle groups in each lower extremity are shown directly below. *Ant tib*, M tibialis anterior; *gast*, m gastrocnemius; *L*, left; *quad*, m quadriceps femoris; *R*, right; *then*, thenar.

PATOPHYSIOLOGIC EFFECTS ON THE ELECTROENCEPHALogram

Hypoxia

Hypoxia may produce inadequate delivery of oxygen to the cerebral cortex generating EEG, and changes similar to those occurring with ischemia result. Initially, hypoxemia may not result in any EEG changes because the brain can increase blood flow to compensate. When the hypoxemia becomes severe enough, further increases in flow are impossible, and EEG changes occur. "Slowing" of the EEG during hypoxia is a nonspecific global effect. Fast frequencies are lost, and low frequencies dominate. Eventually, the EEG is abolished as the brain shuts down electric activity and diverts all oxygen delivered to maintenance of cellular integrity.

Hypotension

In a normal, awake patient, significant levels of hypotension are needed to cause the earliest of CNS signs, as measured by discrimination tests such as the flicker-fusion test. This test examines the flicker rate at which the observer perceives the light to be continuous. In the early days of deliberate hypotension, this test was part of the preoperative evaluation to judge how far the pressure could be reduced during the operation. Clear signs of confusion and inability to concentrate or respond properly to simple commands generally represent very low levels of cerebral perfusion when caused by hypotension because the normal cerebral circulation has a large capacity to vasodilate and maintain normal flow in the face of significant hypotension.

The EEG changes associated with even this level of hypotension are not dramatic, although they are clear by comparison with a previously active recording. Herein lies the problem with using intraoperative EEG to determine whether a given level of hypotension has resulted in brain ischemia. EEG changes are not very pronounced and are bilateral. These changes also are nearly identical to the changes caused by increasing doses of many anesthetic drugs. EEG changes associated with hypotension can be detected, but when the hypotension is induced slowly and associated with changes in anesthetic drugs (e.g., use of isoflurane to reduce blood pressure), the changes are very difficult to interpret. EEG changes associated with acute, severe hypotension such as may be caused by sudden arrhythmias are easier to read. Many patients undergoing surgery do not have a normal cerebral circulation, however. In these individuals, even mild hypotension may result in significant cerebral ischemia. In these individuals, monitoring the EEG during planned hypotension may be helpful, provided that other causes of similar EEG changes may be carefully controlled. There remains little literature to support the use of EEG monitoring during hypotension, but in our opinion, when the EEG is being monitored (e.g., during carotid surgery), EEG changes secondary to hypotension really do represent cerebral ischemia of a significant degree and should be considered an important finding.

Hypothermia

During cooling on CPB, the total power and peak power frequency of the high-frequency band were highly correlated with temperature using Fourier analysis and spectral edge data; however, significant variability was noted among subjects, especially during cooling.²⁶⁶ Complete EEG suppression usually develops at 15°C to 18°C. Levy and

colleagues¹⁴⁷ showed an improved ability to quantify the effects of hypothermia on the EEG using an EEG processing technique known as "approximate entropy."

Hypercarbia and Hypocarbia

Hypocapnia is known to activate excitable seizure foci, and in rare cases may produce EEG evidence of cerebral ischemia even in awake subjects.²⁶⁷ Hypercapnia, unless severe and associated with hypoxemia, has only indirect effects secondary to increased CBF. In an anesthetized patient, hypercarbia-associated increases in CBF may have similar effects to the effects seen with increasing end-tidal tension of volatile anesthetics.²⁶⁸

Untoward Events

One of the suggested reasons for monitoring the brain of an anesthetized patient is to enable detection of injuries to the nervous system that would not be otherwise apparent. Although hundreds of such case reports are in the literature, as well as many in our experience, cost-effectiveness of such monitoring is unclear. In a more recent case at our institution, severe EEG changes occurred at the beginning of a carotid endarterectomy, before surgical incision, and were unassociated with any other vital sign changes or hypotension. Immediate angiography revealed acute carotid occlusion and completely changed the operation performed with this patient, and the patient recovered completely. There are intraoperative events that could lead to CNS insult, which, if detected early, could be rapidly reversed or treated to prevent permanent injury. Given the rarity of such events, however, it is extremely unlikely that such monitoring would be shown to be beneficial in any foreseeable randomized trial. If the "at-risk" patient could be identified preoperatively, perhaps the EEG or other types of neuromonitoring could be useful in detecting untoward CNS events during anesthesia, such as a new stroke after elective general surgery.

PHYSIOLOGIC FACTORS INFLUENCING SENSORY-EVOKED RESPONSES

Numerous physiologic variables, including systemic arterial blood pressure, temperature (local and systemic), and blood gas tensions, can influence SEP recordings. With decreases in mean arterial blood pressure to below levels of cerebral autoregulation owing to either blood loss or vasoactive drugs, progressive changes in SERs have been noted. SSEP changes observed are progressive decreases in amplitude until loss of the waveform with no changes in latency.^{269,270} BAEPs are resistant to even profound levels of hypotension (MAP of 20 mm Hg in dogs).²⁶⁹ Cortical (synaptic) function necessary to produce cortical SERs seems to be more sensitive to hypoperfusion than spinal cord or brainstem, nonsynaptic transmission.²⁷⁰ Rapid decreases in arterial blood pressure to levels above the lower limit of autoregulation also have been associated with transient SSEP changes of decreased amplitude that resolve after several minutes of continued hypotension at the same level.²⁷¹ Reversible SSEP changes at systemic pressures within the normal range have been observed in patients undergoing spinal distraction during scoliosis surgery. These changes resolved with increases of systemic arterial blood pressure to slightly higher than the patient's normal pressure, suggesting that the combination of surgical manipulation with levels of hypotension generally considered "safe" could result in spinal cord ischemia.²⁷²