Chapter 10

Intraoperative Echocardiography

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Few areas in cardiac anesthesia have developed as rapidly as the field of intraoperative echocardiography. In the early 1980s, when transesophageal echocardiography (TEE) was first used in the operating room, its main application was the assessment of global and regional left ventricular (LV) function. Since that time there have been numerous technical advances: biplane and multiplane probes; multifrequency probes; enhanced scanning resolution; color flow, pulsed wave, and continuous wave Doppler; automatic edge detection; Doppler tissue imaging; three-dimensional (3D) reconstruction; and digital image processing. With these advances, the number of clinical applications of TEE has markedly increased. The common applications of TEE include (1) assessment of valvular anatomy and function, (2) evaluation of the thoracic aorta, (3) detection of intracardiac defects, (4) detection of intracardiac masses, (5) evaluation of pericardial effusions, (6) detection of intracardiac air and clots, and (7) assessment of biventricular systolic and diastolic function. In many of these evaluations, TEE is able to provide unique and critical information that was not previously available in the operating room (Box 10-1).

BOX 10-1   *Common Applications of Transesophageal Echocardiography*

- Assessment of valvular anatomy and function
- Evaluation of the thoracic aorta
- Detection of intracardiac defects
- Detection of intracardiac masses
- Evaluation of pericardial effusions
- Detection of intracardiac air and clots
- Assessment of biventricular systolic and diastolic function

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BASIC CONCEPTS

Properties of Ultrasound

In echocardiography, the heart and great vessels are insonated with ultrasound, which is sound above the human audible range. The ultrasound is sent into the thoracic cavity and is partially reflected by the cardiac structures. From these reflections, distance, velocity, and density of objects within the chest are derived.

Wavelength, Frequency, and Velocity

An ultrasound beam is a continuous or intermittent train of sound waves emitted by a transducer or wave generator. It is composed of density or pressure waves and can exist in any medium with the exception of a vacuum. Ultrasound waves are characterized by their wavelength, frequency, and velocity. *Wavelength* is the distance between the two nearest points of equal pressure or density in an ultrasound beam, and *velocity* is the speed at which the waves propagate through a medium. As the waves travel past any fixed point in an ultrasound beam, the pressure cycles regularly and continuously between a high and a low value. The number of cycles per second (Hertz) is called the *frequency* of the wave. Ultrasound is sound with frequencies above 20,000 Hz, which is the upper limit of the human audible range. The relationship among the frequency (f), wavelength (λ), and velocity (v) of a sound wave is defined by the formula:

$$v = f \cdot \lambda$$

Piezoelectric crystals convert the energy between ultrasound and electrical signals. When presented with a high-frequency electrical signal, these crystals produce ultrasound energy, which is directed toward the areas to be imaged. Commonly, a short ultrasound signal is emitted from the piezoelectric crystal. After ultrasound wave formation, the crystal "listens" for the returning echoes for a given period of time and then pauses before repeating this cycle. This cycle length is known as the *pulse repetition frequency* (PRF). This cycle length must be long enough to provide enough time for a signal to travel to and return from a given object of interest. Typically, PRFs vary from 1 to 10 kHz, which results in 0.1- to 1.0-ms intervals between pulses. When reflected ultrasound waves return to these piezoelectric crystals they are converted into electrical signals, which may be appropriately processed and displayed. Electronic circuits measure the time delay between the emitted and received echo. Because the speed of ultrasound through tissue is a constant, this time delay can be converted into the precise distance between the transducer and tissue.

Imaging Techniques

M Mode

The most basic form of ultrasound imaging is M-mode echocardiography. In this mode, the density and position of all tissues in the path of a narrow ultrasound beam (i.e., *along a single line*) are displayed as a scroll on a video screen. The scrolling produces an updated, continuously changing time plot of the studied tissue section, several seconds in duration. Because this is a timed *motion display* (normal cardiac tissue is always in motion), it is called M mode. Because only a very limited part of the heart is being observed at any one time and because the image requires considerable interpretation, M mode is not currently used as a primary imaging technique. This mode is, however, useful for the precise timing of events within the cardiac cycle and is often used in combination with color flow Doppler imaging for the timing of abnormal flows.

Two-Dimensional Mode

By rapid, repetitive scanning along *many different radii* within an area in the shape of a fan (sector), echocardiography generates a 2D image of a section of the heart. This image, which resembles an anatomic section and, thus, can be more easily interpreted, is called a *two-dimensional scan.* Information on structures and motion in the plane of a 2D scan is updated 30 to 60 times per second. This repetitive update produces a "live" (real-time) image of the heart. Scanning 2D echo devices image the heart by using either a mechanically steered transducer or, as is common in many of the modern devices, an electronically steered ultrasound beam (phased-array transducer).

Doppler Techniques

Most modern echocardiographic scanners combine Doppler capabilities with their 2D imaging capabilities. After the desired view of the heart has been obtained by 2D echocardiography, the Doppler beam, represented by a cursor, is superimposed on the 2D image. The operator positions the cursor as parallel as possible to the assumed direction of blood flow and then empirically adjusts the direction of the beam to optimize the audio and visual representations of the reflected Doppler signal. At the present time, Doppler technology can be used in at least four different ways to measure blood velocities: pulsed wave, high repetition frequency, continuous wave, and color flow.

Color Flow Mapping

Advances in electronics and computer technology have allowed the development of color flow Doppler ultrasound scanners capable of displaying real-time blood flow within the heart as colors while also showing 2D images in black and white. In addition to showing the location, direction, and velocity of cardiac blood flow, the images produced by these devices allow estimation of flow acceleration and differentiation of laminar and turbulent blood flow.

A location in the heart where the scanner has detected flow toward the transducer (the top of the image sector) is assigned the color red. Flow away from the direction of the top is assigned the color blue. This color assignment is arbitrary and determined by the equipment manufacturer and the user's color mapping. In the most common color flow coding scheme, the faster the velocity (up to a limit), the more intense is the color. Flow velocities that change by more than a preset value within a brief time interval (flow variance) have the color green added to either the red or the blue. Both rapidly accelerating laminar flow (change in flow speed) and turbulent

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BOX 10-2 *Diagnostic Applications for Contrast Echocardiography*

- Assessment of congenital heart disease
- Enhancement of endocardial borders for qualitative assessment of wall motion abnormalities
- Measurement of left ventricular function
- Quantification of valvular regurgitation
- Enhancement of color flow Doppler signals
- Assessment of myocardial perfusion
- Measurements of perfusion area after coronary artery bypass graft surgery
- Assessment of quality of coronary bypass grafts and cardioplegia distribution
- Correct assessment of the results of surgery for ventricular septal defect

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flow (change in flow direction) satisfy the criteria for rapid changes in velocity. In summary, the brightness of the red or blue colors at any location and time is usually proportional to the corresponding flow velocity while the hue is proportional to the temporal rate of change of the velocity.

Contrast Echocardiography

Normally, red blood cells scatter ultrasound waves weakly, resulting in their black appearance on ultrasound examination. Contrast echocardiography is performed by injecting nontoxic solutions containing gaseous microbubbles. These microbubbles present additional gas-liquid interfaces, which substantially increase the strength of the returning signal. This augmentation in signal strength may be used to better define endocardial borders, optimize Doppler envelope signals, and estimate myocardial perfusion.

Contrast echocardiography has been used to image intracardiac shunts, valvular incompetence, and pericardial effusions. In addition, LV injections of hand-agitated microbubble solutions have been used to identify semiquantitative LV endocardial edges, cardiac output, and valvular regurgitation (Box 10-2).

Contrast agents are microbubbles, consisting of a shell surrounding a gas. Initial contrast agents were agitated free air in either a saline or blood/saline solution. These microbubbles were large and unstable, so they were unable to cross the pulmonary circulation; they were effective only for right-sided heart contrast. Because of their thin shell, the gas quickly leaked into the blood with resultant dissolution of the microbubble. Agents with a longer persistence were subsequently developed.[1]

EQUIPMENT

All of the currently available TEE probes employ a multifrequency transducer that is mounted on the tip of a gastroscope housing. The majority of the echocardiographic examination is performed using ultrasound between 3.5 and 7 MHz. The tip can be directed by the adjustment of knobs placed at the proximal handle. In most adult probes there are two knobs; one allows anterior and posterior movement, and the other permits side-to-side motion. Multiplane probes also include a control to rotate the echocardiographic array from 0 to 180 degrees. Thus, in combination with the ability to advance and withdraw the probe and to rotate it, many echocardiographic windows are possible. Another feature common to most probes is the inclusion of a temperature sensor to warn of possible heat injury from the transducer to the esophagus.

> **BOX 10-3** *Complications from Intraoperative Transesophageal Echocardiography*
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> - Injury from direct trauma to the airway and esophagus
>   - Esophageal bleeding, burning, tearing
>   - Dysphagia
>   - Laryngeal discomfort
>   - Bacteremia
>   - Vocal cord paralysis
> - Indirect effects
>   - Hemodynamic and pulmonary effects of airway manipulation
>   - Distraction from patient

Currently, most adult echocardiographic probes are multiplane (variable orientation of the scanning plane), whereas pediatric probes are either multiplane or biplane (transverse and longitudinal orientation, parallel to the shaft). The adult probes usually have a shaft length of 100 cm and are between 9 and 12 mm in diameter. The tips of the probes vary slightly in shape and size but are generally 1 to 2 mm wider than the shaft. The size of these probes requires the patient to weigh at least 20 kg. Depending on the manufacturer, the adult probes contain between 32 and 64 elements per scanning orientation. In general, the image quality is directly related to the number of elements used. The pediatric probes are mounted on a narrower, shorter shaft with smaller transducers. These probes may be used in patients as small as 1 kg.

An important feature that is often available is the ability to alter the scanning frequency. A lower frequency, such as 3.5 MHz, has greater penetration and is more suited for the transgastric view. It also increases the Doppler velocity limits. Conversely, the higher frequencies yield better resolution for detailed imaging. One of the limitations of TEE is that structures very close to the probe are seen only in a very narrow sector. Newer probes may also allow a broader near-field view. Finally, newer probes may possess the ability to scan simultaneously in more than one plane.

COMPLICATIONS

Complications resulting from intraoperative TEE can be separated into two groups: injury from direct trauma to the airway and esophagus and indirect effects of TEE (Box 10-3). In the first group, potential complications include esophageal bleeding, burning, tearing, dysphagia, and laryngeal discomfort. Many of these complications could result from pressure exerted by the tip of the probe on the esophagus and the airway. Although in most patients even maximal flexion of the probe will not result in pressure above 17 mm Hg, occasionally, even in the absence of esophageal disease, pressures greater than 60 mm Hg will result.

Further confirmation of the low incidence of esophageal injury from TEE is apparent in the few case reports of complications. In the world's literature, there are only a few reports of a fatal esophageal perforation and benign Mallory-Weiss tear after intraoperative TEE.

The second group of complications that result from TEE includes hemodynamic and pulmonary effects of airway manipulation and, particularly for new TEE operators, distraction from patient care. Fortunately, in the anesthetized

patient there are rarely hemodynamic consequences to esophageal placement of the probe and there are no studies that specifically address this question. One potential hemodynamic effect of TEE, even in the well-anesthetized patient, is direct cardiac irritation from the probe with resultant atrial and ventricular arrhythmias. More important for the anesthesiologist are the problems of distraction from patient care. Although these reports are infrequent in the literature, the authors know of several endotracheal tube disconnections that went unnoticed to the point of desaturation during TEE. Additionally, there have been instances in which severe hemodynamic abnormalities have been missed because of fascination with the images or the controls of the echocardiograph machine. Clearly, new echocardiographers should enlist the assistance of an associate to watch the patient during the examination. This second anesthesiologist will become unnecessary after sufficient experience is gained. It is also important to be sure that all of the respiratory and hemodynamic alarms are activated during the echocardiographic examination.

SAFETY GUIDELINES AND CONTRAINDICATIONS

To ensure the continued safety of TEE, the following recommendations are made. The probe should be inspected before each insertion for cleanliness and structural integrity. If possible, the electrical isolation should also be checked. The probe should be inserted gently and, if resistance is met, the procedure aborted. Minimal transducer energy should be used and the image frozen when not in use. Finally, when not imaging, the probe should be left in the neutral, unlocked position to avoid prolonged pressure on the esophageal mucosa.

Absolute contraindications to TEE in intubated patients include esophageal stricture, diverticula, tumor, recent suture lines, and known esophageal interruption. Relative contraindications include symptomatic hiatal hernia, esophagitis, coagulopathy, esophageal varices, and unexplained upper gastrointestinal bleeding. Despite these relative contraindications, TEE has been used in patients undergoing hepatic transplantation without reported sequelae.

III

TECHNIQUE OF PROBE PASSAGE

The passage of a TEE probe through the oral and pharyngeal cavities in anesthetized patients may be challenging at times. The usual technique is to place the well-lubricated probe in the posterior portion of the oropharynx with the transducer element pointing inferiorly and anteriorly. The remainder of the probe may be stabilized by looping the controls and the proximal portion of the probe over the operator's neck and shoulder. The operator's left hand then elevates the mandible by inserting the thumb behind the teeth, grasping the submandibular region with the fingers, and then gently lifting. The probe is then advanced against a slight but even resistance, until a loss of resistance is detected as the tip of the probe passes the inferior constrictor muscle of the pharynx. This usually occurs 10 cm past the lips in neonates to 20 cm past the lips in adults. Further manipulation of the probe is performed under echocardiographic guidance.

Difficult TEE probe insertion may be caused by the probe tip abutting the pyriform sinuses, vallecula, posterior tongue, or an esophageal diverticulum. Overinflation of the endotracheal tube cuff could also obstruct passage of the probe. Maneuvers that might aid the passage of the probe include changing the neck position, realigning the

TEE probe, and applying additional jaw thrust by elevating the angles of the mandible. The probe may also be passed with the assistance of laryngoscopy. The probe should never be forced past an obstruction. This could result in airway trauma or esophageal perforation.

ANATOMY AND TRANSESOPHAGEAL ECHOCARDIOGRAPHY VIEWS

Multiplane Transesophageal Echocardiography Probe Manipulation: Descriptive Terms and Technique

The process of obtaining a comprehensive intraoperative multiplane TEE examination begins with a fundamental understanding of the terminology and technique for probe manipulation (Fig. 10-1).[2] Efficient probe manipulation minimizes esophageal injury and facilitates the process of acquiring and sweeping through 2D image planes. Horizontal imaging planes are obtained by moving the TEE probe up and down (proximal and distal) in the esophagus at various depths relative to the incisors (upper esophageal: 20 to 25 cm; midesophageal: 30 to 40 cm; transgastric: 40 to 45 cm; deep transgastric: 45 to 50 cm) (Table 10-1). Vertical planes

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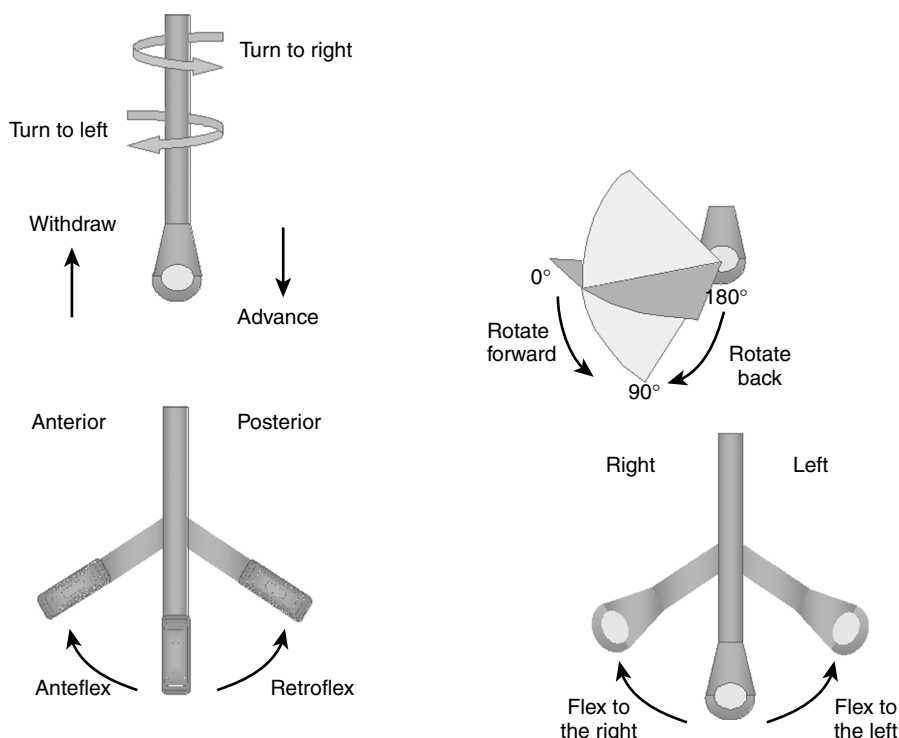
Figure 10-1 Ways to adjust the probe. *Top left,* Probe movement in the esophagus. *Top right,* Scanning angles obtained by crystal rotation. *Bottom left,* Movement of the tip forward and back. *Bottom right,* Movement of the tip from side to side.

are obtained by manually turning the probe to the patient's left or right. Further alignment of the imaging plane can be obtained by manually rotating one of the two control wheels on the probe handle, which flexes the probe tip to the left or right direction or in the anterior or posterior plane. Multiplane probes may further facilitate interrogation of complex anatomic structures, such as the mitral valve (MV), by allowing up to 180 degrees of axial rotation of the imaging plane without manual probe manipulation.

The Comprehensive Intraoperative Transesophageal Echocardiography Examination: Imaging Planes and Structural Analysis

Left and Right Ventricles

The LV should be carefully examined for global and regional function using multiple transducer planes, depths, and rotational and angular orientations (Fig. 10-2). Analysis of segmental function is based on a qualitative visual assessment that includes the following grading system of both LV wall thickness and motion (endocardial border excursion) during systole: 1 = normal (>30% thickening); 2 = mild hypokinesis (10% to 30% thickening); 3 = severe hypokinesis (<10% thickening); 4 = akinesis (no thickening); 5 = dyskinesis (paradoxical motion). The *midesophageal (ME) four-chamber view* at 0 to 20 degrees (see Fig. 10-2A) and *two-chamber* view at approximately 80 to 100 degrees (see Fig. 10-2B) enable visualization of the septal and lateral as well as the inferior and anterior segments at the basal, mid, and apical level segments, respectively. The *ME long-axis (LAX) view* at 120 to 160 degrees (see Fig. 10-2C) allows evaluation of the remaining anteroseptal and posterior LV segments. Because the LV is usually oriented inferiorly to the true horizontal plane, slight retroflexion of the probe tip may be required to minimize LV foreshortening. The *transgastric (TG) mid short-axis (SAX) view* at 0 to 20 degrees (see Fig. 10-2D) is the most commonly used view for monitoring LV function because it allows a midpapillary assessment of the LV segments supplied by the corresponding coronary arteries (right [RCA], left circumflex [CX], and left anterior descending [LAD]). This view also enables qualitative and quantitative evaluation of pericardial effusions. Advancing or withdrawing the probe at the transgastric depth enables LV evaluation at the respective apical and basal levels (*TG basal short-axis view;* see Fig. 10-2F). Further evaluation of the LV can be obtained at the midpapillary transgastric depth by rotating the probe forward to the *TG two-chamber view* (80 to 100 degrees) (see Fig. 10-2E) and *TG long-axis view* (90 to 120 degrees) (see Fig. 10-2J).

Right ventricular (RV) regional and global function can be assessed from the ME four-chamber view (see Fig. 10-2A), which allows visualization of the septal and free walls. Although a formal segmental scheme has not been developed for the RV free wall, regional assessment of the septum can be performed. Turning the probe to the right and advancing slightly from the midesophageal depth allows visualization of the tricuspid valve (TV), coronary sinus (CS), and RV apex. Rotating the probe between 60 and 90 degrees reveals the *ME RV inflow-outflow view* (see Fig. 10-2M), in which the RA, TV, inferior RV free wall, right ventricular outflow tract (RVOT), pulmonic valve (PV), and main pulmonary artery (PA) can be viewed "wrapping around" the centrally oriented AV. This view often allows optimal Doppler beam alignment to evaluate the TV and can also be helpful for directing PA catheter floating and positioning. The *TG mid short-axis view*

Table 10-1   The Comprehensive Intraoperative Multiplane Transesophageal Echocardiographic Examination

Probe Tip Depth (from lips): Upper Esophageal (20 to 25 cm)

View	*Aortic arch: long axis*
Multiplane angle range	0°
Anatomy imaged	Aortic arch; left brachiocephalic vein; left subclavian and carotid arteries; right brachiocephalic artery
Clinical utility	Ascending aorta and arch pathology: atherosclerosis, aneurysms, and dissections
	Aortic CPB cannulation site evaluation
View	*Aortic arch: short axis*
Multiplane angle range	90°
Anatomy imaged	Aortic arch; left brachiocephalic vein; left subclavian and carotid arteries; right brachiocephalic artery; main pulmonary artery and pulmonic valve
Clinical utility	Ascending aorta and arch pathology: atherosclerosis, aneurysms and dissections; pulmonary embolus; pulmonary valve evaluation (insufficiency, stenosis, Ross procedure)
	Pulmonary artery catheter placement

Probe Tip Depth: Midesophageal (30 to 40 cm)

View	*Four chamber*
Multiplane angle range	0° to 20°
Anatomy imaged	Left ventricle and atrium
	Right ventricle and atrium
	Mitral and tricuspid valves
	Interatrial and interventricular septa
	Left pulmonary veins: slight probe withdrawal and turning to left
	Right pulmonary veins: slight probe withdrawal and turning to right
	Coronary sinus: slight probe advancement and turning to right
Clinical utility	Ventricular function: global and regional
	Intracardiac chamber masses: thrombus, tumor, air; foreign bodies
	Mitral and tricuspid valve evaluation: pathology, pathophysiology
	Congenital or acquired interatrial and ventricular septal defects evaluation
	Hypertrophic obstructive cardiomyopathy evaluation
	Ventricular diastolic evaluation via transmitral and pulmonary vein Doppler flow profile analysis
	Pericardial evaluation: pericarditis; pericardial effusion
	Coronary sinus evaluation: coronary sinus catheter placement; dilation secondary to persistent left superior vena cava
View	*Mitral commissural*
Multiplane angle range	60° to 70°
Anatomy imaged	Left ventricle and atrium
	Mitral valve

Table continued on following page

Table 10-1   The Comprehensive Intraoperative Multiplane Transesophageal Echocardiographic Examination (Continued)

Probe Tip Depth: Midesophageal (30 to 40 cm)

Clinical utility	Left ventricular function: global and regional
	Left ventricular and atrial masses: thrombus, tumor, air; foreign bodies
	Mitral valve evaluation: pathology, pathophysiology
	Ventricular diastolic evaluation via transmitral Doppler flow profile analysis
View	*Two chamber*
Multiplane angle range	80° to 100°
Anatomy imaged	Left ventricle, atrium and atrial appendage
	Mitral valve
	Left pulmonary veins: turning probe to left
	Coronary sinus (short axis or long axis by turning probe tip to left)
Clinical utility	Left ventricular function: global and regional
	Left ventricular and atrial masses: thrombus, tumor, air; foreign bodies
	Mitral valve evaluation: pathology, pathophysiology
	Ventricular diastolic evaluation via transmitral and pulmonary vein Doppler flow profile analysis
	Coronary sinus evaluation: coronary sinus catheter placement; dilation secondary to persistent left superior vena cava
View	*Long axis*
Multiplane angle range	120° to 160°
Anatomy imaged	Left ventricle and atrium
	Left ventricular outflow tract
	Aortic valve
	Mitral valve
	Ascending aorta
Clinical utility	Left ventricular function: global and regional
	Left ventricular and atrial masses: thrombus, tumor, air; foreign bodies
	Mitral valve evaluation: pathology, pathophysiology
	Ventricular diastolic evaluation via transmitral Doppler flow profile analysis
	Aortic valve evaluation: pathology, pathophysiology
	Ascending aorta pathology: atherosclerosis, aneurysms, dissections
	Hypertrophic obstructive cardiomyopathy evaluation
View	*Right ventricular inflow-outflow ("wrap-around")*
Multiplane angle range	60° to 90°
Anatomy imaged	Right ventricle and atrium
	Left atrium
	Tricuspid valve
	Aortic valve
	Right ventricular outflow tract
	Pulmonic valve and main pulmonary artery
Clinical utility	Right ventricular and right and left atrial masses: thrombus, embolus, tumor, foreign bodies
	Pulmonic valve and subpulmonic valve: pathology; pathophysiology
	Pulmonary artery catheter placement
	Tricuspid valve: pathology; pathophysiology
	Aortic valve: pathology; pathophysiology

III

Probe Tip Depth: Midesophageal (30 to 40 cm)

View	*Aortic valve: short axis*
Multiplane angle range	30° to 60°
Anatomy imaged	Aortic valve
	Interatrial septum
	Coronary ostia and arteries
	Right ventricular outflow tract
	Pulmonary valve
Clinical utility	Aortic valve: pathology; pathophysiology
	Ascending aorta pathology: atherosclerosis, aneurysms and dissections
	Left and right atrial masses: thrombus, embolus, air, tumor,
	Foreign bodies
	Congenital or acquired interatrial septal defects evaluation
View	*Aortic valve: long axis*
Multiplane angle range	120° to 160°
Anatomy imaged	Aortic valve
	Proximal ascending aorta
	Left ventricular outflow tract
	Mitral valve
	Right pulmonary artery
Clinical utility	Aortic valve: pathology; pathophysiology
	Ascending aorta pathology: atherosclerosis, aneurysms and dissections
	Mitral valve evaluation: pathology, pathophysiology
View	*Bicaval*
Multiplane angle range	80° to 110°
Anatomy imaged	Right and left atrium
	Superior vena cava (long axis)
	Inferior vena cava orifice: advance probe and turn to right to visualize inferior vena cava in the long axis, liver, hepatic and portal veins
	Interatrial septum
	Right pulmonary veins: turn probe to right
	Coronary sinus and thebesian valve
	Eustachian valve
Clinical utility	Right and left atrial masses: thrombus, embolus, air, tumor, foreign bodies
	Superior vena cava pathology: thrombus, sinus venosus atrial septal defect
	Inferior vena cava pathology (thrombus, tumor)
	Femoral venous catheter placement
	Coronary sinus catheter placement
	Right pulmonary vein evaluation: anomalous return, Doppler evaluation for left ventricular diastolic function
	Congenital or acquired interatrial septal defects evaluation
	Pericardial effusion evaluation
View	*Ascending aortic: short axis*
Multiplane angle range	0° to 60°
Anatomy imaged	Ascending aorta
	Superior vena cava (short axis)
	Main pulmonary artery
	Right pulmonary artery
	Left pulmonary artery (turn probe tip to left)
	Pulmonic valve

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Table 10-1 **The Comprehensive Intraoperative Multiplane Transesophageal Echocardiographic Examination** (Continued)

Probe Tip Depth: Midesophageal (30 to 40 cm)

Clinical utility	Ascending aorta pathology: atherosclerosis, aneurysms and dissections
	Pulmonic valve: pathology; pathophysiology
	Pulmonary embolus/thrombus evaluation
	Superior vena cava pathology: thrombus, sinus venosus atrial septal defect
	Pulmonary artery catheter placement
View	*Ascending aorta: long axis*
Multiplane angle range	100° to 150°
Anatomy imaged	Ascending aorta
	Right pulmonary artery
Clinical utility	Ascending aorta pathology: atherosclerosis, aneurysms and dissections
	Anterograde cardioplegia delivery evaluation
	Pulmonary embolus/thrombus
View	*Descending aorta: short axis*
Multiplane angle range	0°
Anatomy imaged	Descending thoracic aorta
	Left pleural space
Clinical utility	Descending aorta pathology: atherosclerosis, aneurysms and dissections
	Intra-aortic balloon placement evaluation
	Left pleural effusion
View	*Descending aorta: long axis*
Multiplane angle range	90° to 110°
Anatomy imaged	Descending thoracic aorta
	Left pleural space
Clinical utility	Descending aorta pathology: atherosclerosis, aneurysms and dissections
	Intra-aortic balloon placement evaluation
	Left pleural effusion

Probe Tip Depth: Transgastric (40 to 45 cm)

View	*Basal short axis*
Multiplane angle range	0° to 20°
Anatomy imaged	Left and right ventricle
	Mitral valve
	Tricuspid valve
Clinical utility	Mitral valve evaluation ("fish-mouth view"): pathology, pathophysiology
	Tricuspid valve evaluation: pathology, pathophysiology
	Basal left ventricular regional function
	Basal right ventricular regional function
View	*Mid short axis*
Multiplane angle range	0° to 20°
Anatomy imaged	Left and right ventricle
	Papillary muscles
Clinical utility	Mid left and right ventricular regional and global function
	Intracardiac volume status
View	*Two chamber*
Multiplane angle range	80° to 100°

Probe Tip Depth: Transgastric (40 to 45 cm)

Anatomy imaged	Left ventricle and atrium
	Mitral valve: chordae and papillary muscles
	Coronary sinus
Clinical utility	Left ventricular regional and global function (including apex)
	Left ventricular and atrium masses: thrombus, embolus, air, tumor, foreign bodies
	Mitral valve: pathology and pathophysiology
View	*Long axis*
Multiplane angle range	90° to 120°
Anatomy imaged	Left ventricle and outflow tract
	Aortic valve
	Mitral valve
Clinical utility	Left ventricular regional and global function
	Mitral valve: pathology and pathophysiology
	Aortic valve: pathology and pathophysiology
View	*Right ventricular inflow*
Multiplane angle range	100° to 120°
Anatomy imaged	Right ventricle and atrium
	Tricuspid valve: chordae and papillary muscles
Clinical utility	Right ventricular regional and global function
	Right ventricular and atrium masses: thrombus, embolus, tumor, foreign bodies
	Tricuspid valve: pathology and pathophysiology

Probe Tip Depth: Deep Transgastric (45 to 50 cm)

View	*Long axis*
Multiplane angle range	0° to 20° (anteflexion)
Anatomy imaged	Left ventricle and outflow tract
	Interventricular septum
	Aortic valve and ascending aorta
	Left atrium
	Mitral valve
	Right ventricle
	Pulmonic valve
Clinical utility	Aortic valve and subaortic pathology and pathophysiology
	Mitral valve pathology and pathophysiology
	Left and right ventricular global function
	Left and right ventricular masses: thrombus, embolus, tumor,
	Foreign bodies
	Congenital or acquired interventricular septal defect evaluation

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(see Fig. 10-2D) displays the crescent-shaped, thinner-walled RV to the left of the LV (i.e., to the right side of the LV).

Mitral Valve

The echocardiographic evaluation of the MV requires a thorough assessment of its leaflets (anterior and posterior), annulus, and the subvalvular apparatus (chordae tendineae, papillary muscles, and adjacent LV walls) to locate lesions and define the etiology and severity of the pathophysiology. The mitral leaflets can be further divided into posterior leaflet scallops—lateral (P1), middle (P2), and medial (P3)—that correspond with respective anterior leaflet sections—lateral third (A1), middle third (A2), and medial third (A3). The leaflets are united at the anterolateral and
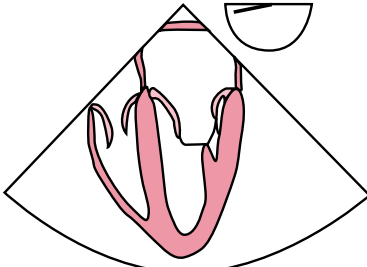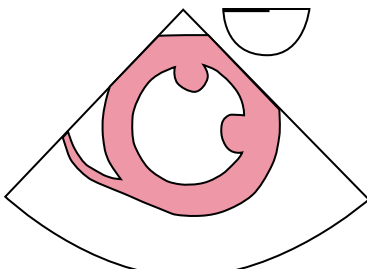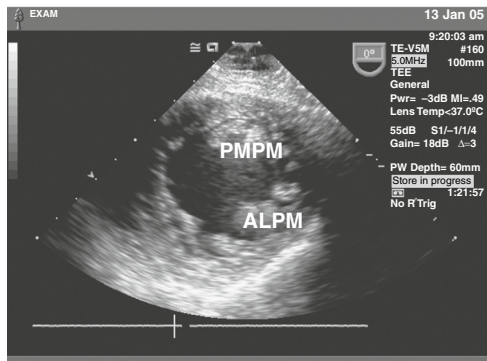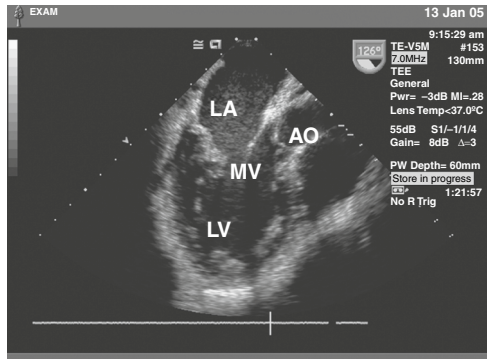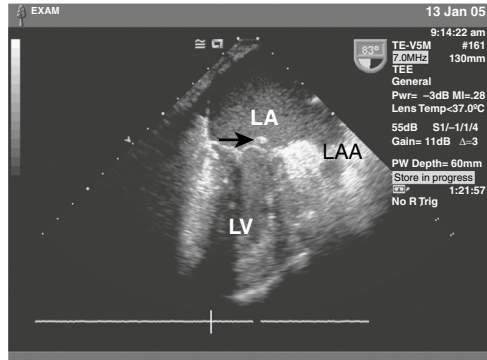
A   ME four chamber
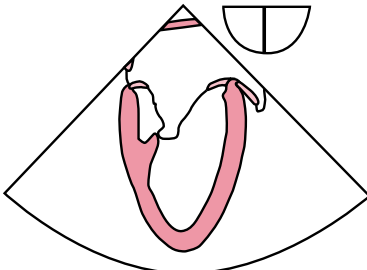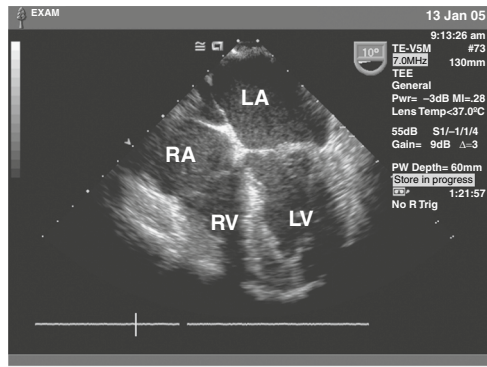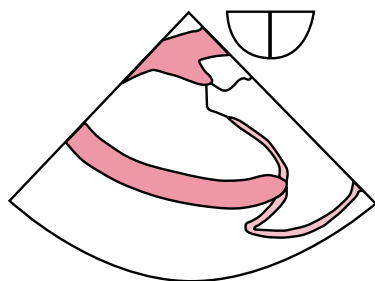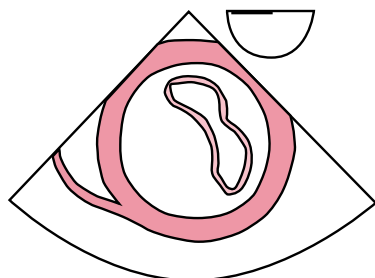

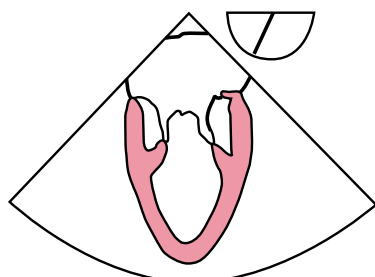B   ME two chamber
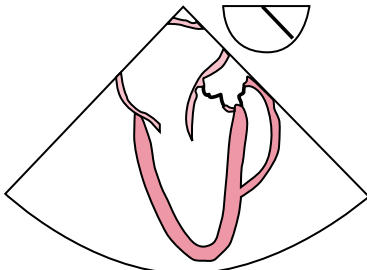
C   ME LAX

D   TG mid SAX

Figure 10-2   Schematic drawings and corresponding echo images of the comprehensive TEE examination. **A,** ME four-chamber view. **B,** ME two-chamber view; the *arrow* points to a prolapsing posterior mitral leaflet. **C,** ME LAX view. **D,** TG mid SAX view.
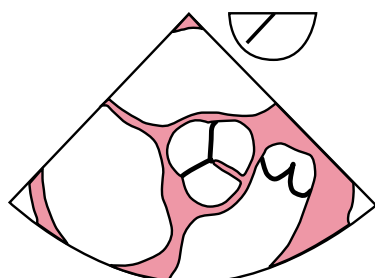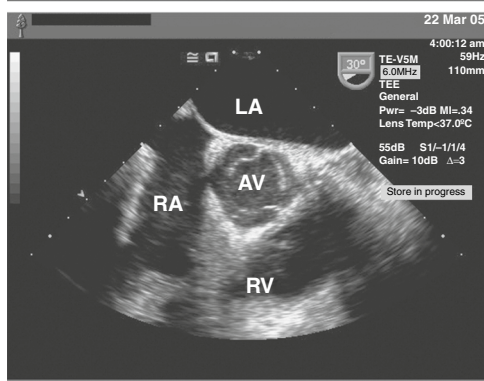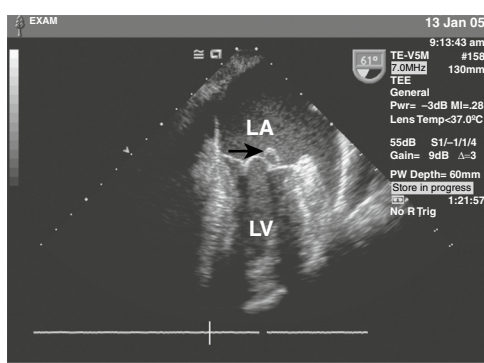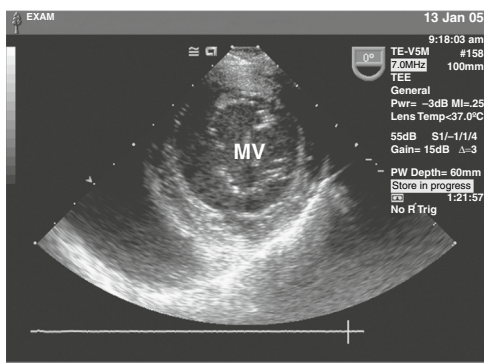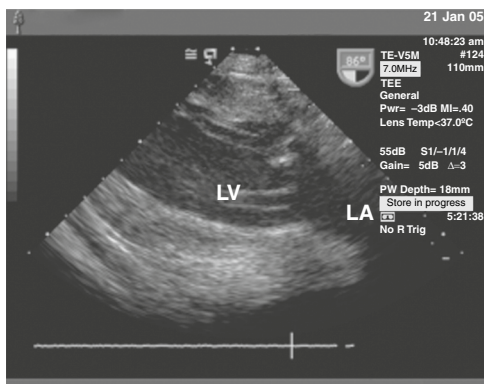
E   TG two chamber

F   TG basal SAX

G   ME mitral commissural

10

H   ME AV SAX

E, TG two-chamber view. **F,** TG basal SAX view. **G,** ME mitral commissural view; the *arrow* points to a prolapsing posterior mitral leaflet. **H,** ME AV SAX view.   *Continued*

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I   ME AV LAX

J   TG LAX

K   Deep TG LAX
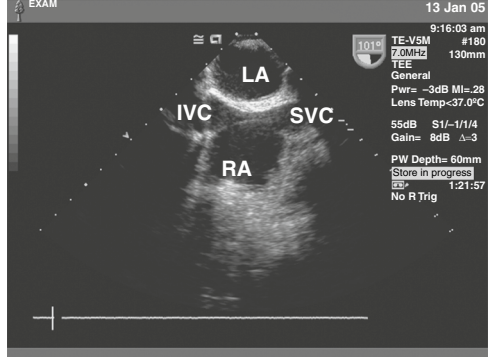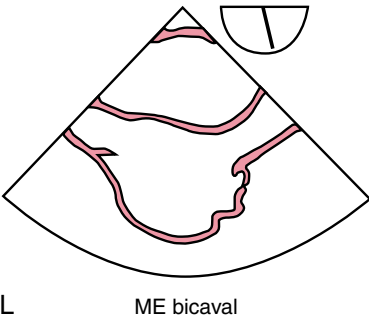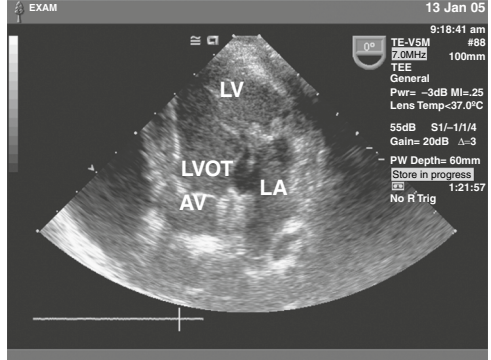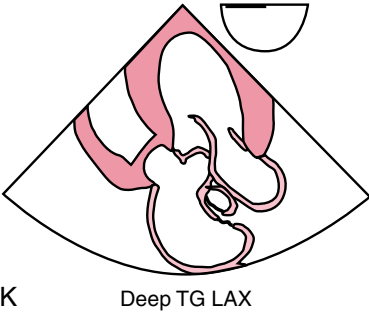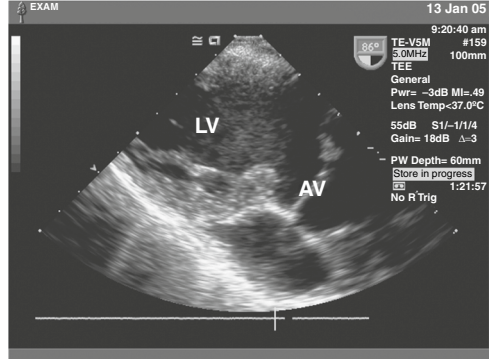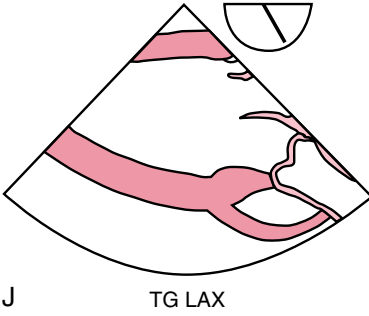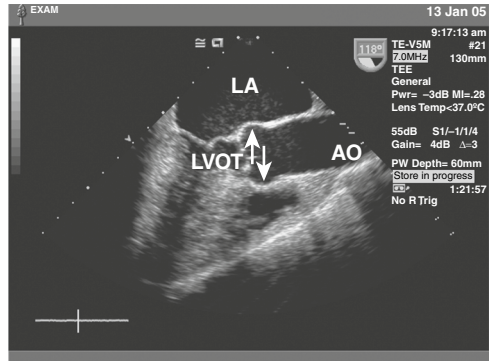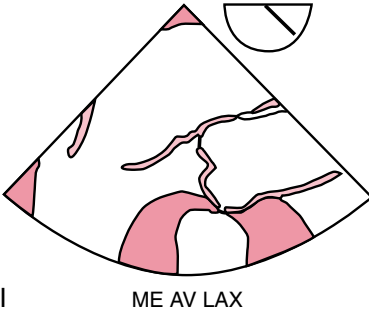
L   ME bicaval

Fig. 10-2, cont'd I, ME AV LAX view; the *arrows* point to the aortic sinuses. **J,** TG LAX view. **K,** Deep TG LAX view. **L,** ME bicaval view.
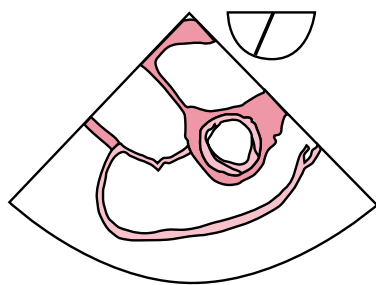
M   ME RV inflow-outflow
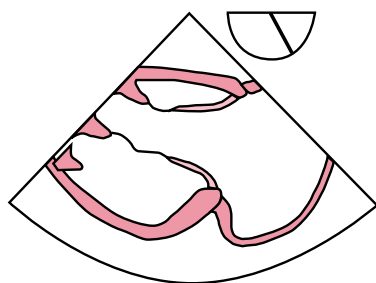
N   TG RV inflow
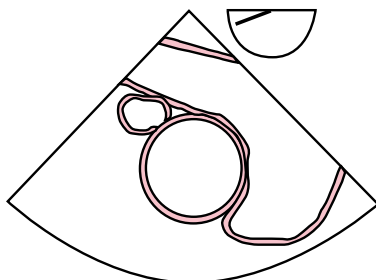
O   ME asc aortic SAX
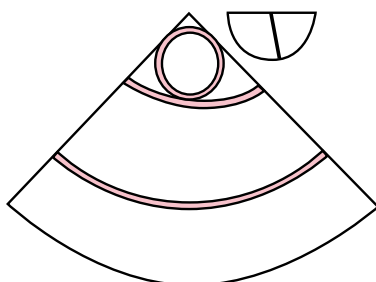
P   ME asc aortic LAX
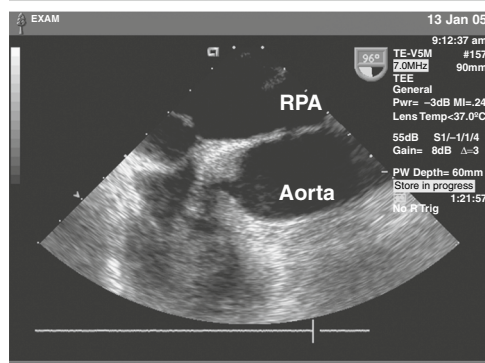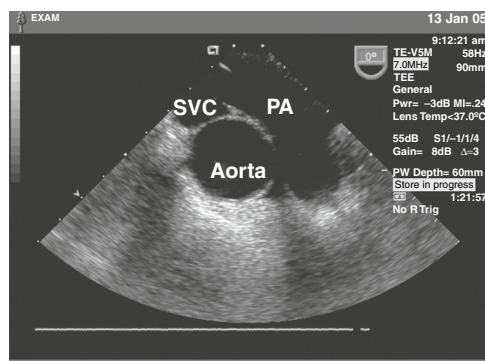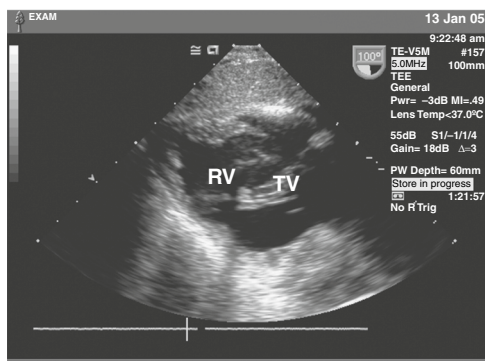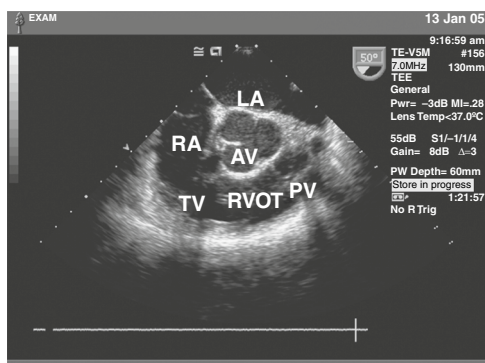
M, ME RV inflow-outflow view. **N,** TG RV inflow view. **O,** ME asc aortic SAX view. **P,** ME asc aortic LAX view.

Continued
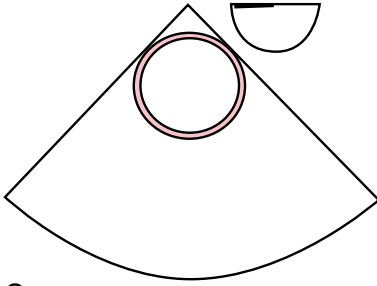
Q   Desc aortic SAX
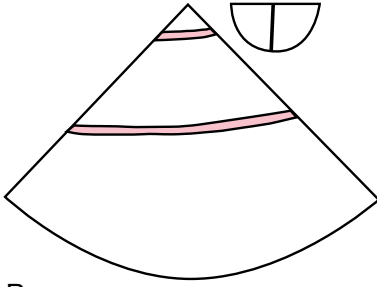
R   Desc aortic LAX
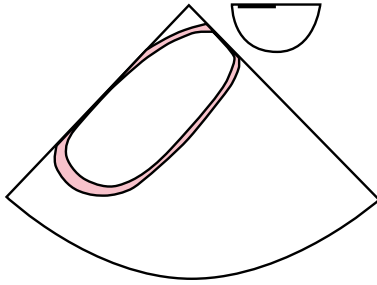
S   UE aortic arch LAX
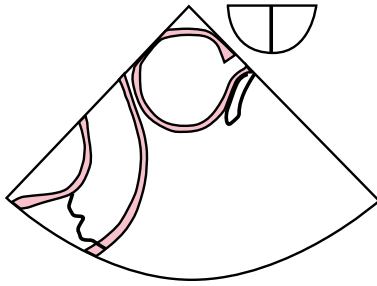
T   UE aortic arch SAX
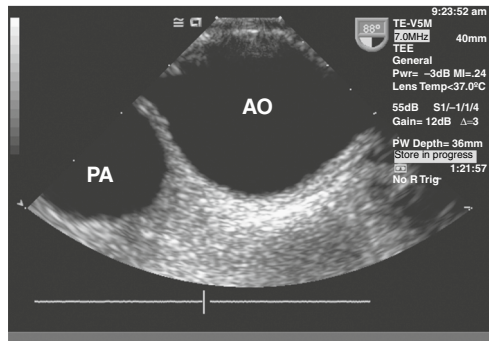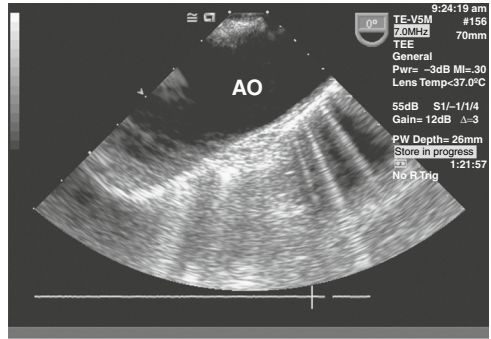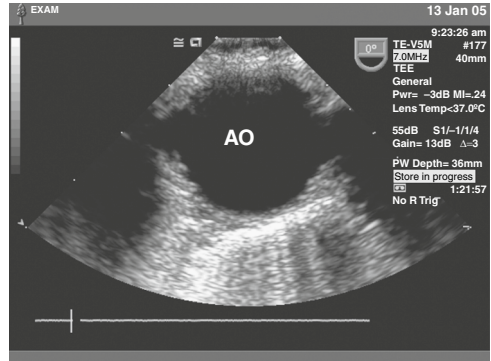
Fig. 10-2, cont'd Q, Desc aortic SAX view. **R,** Desc aortic LAX view. **S,** UE aortic arch LAX view. **T,** UE aortic arch SAX view. ME=mid-esophageal; SAX=short axis; LAX=long axis; AV=aortic valve; RV=right ventricle; TG=transgastric; UE=upper esophageal; asc=ascending aorta; desc=descending aorta; LA=left atrium; MV=mitral valve; LVOT=left ventricular outflow tract; RA=right atrium; LV=left ventricle; TV=tricuspid valve; PV=pulmonic valve; RVOT=right ventricular outflow tract; ALPM=anterior lateral papillary muscle; PMPM=posterior medial papillary muscle; IVC=inferior vena cava; SVC=superior vena cava.

posteromedial commissures. The ME four-chamber view (see Fig. 10-2A) displays the larger appearing anterior leaflet (A3) to the left of the posterior leaflet (P1). Anteflexing the probe provides imaging of the anterior aspect of the MV, while gradual advancement of the probe and retroflexion shifts the image plane to the posterior aspect of MV. Maintaining the probe at the ME depth and rotating the multiplane angle forward to 60 to 70 degrees develops the *ME mitral commissural view* (see Fig. 10-2G) in which A2 is flanked by P1 on the right and P3 on the left, giving A2 the appearance of a "trap door" as it moves in and out of the imaging plane throughout the cardiac cycle.

Aortic Valve, Aortic Root, and Left Ventricular Outflow

The three cusps of the semilunar AV are best visualized simultaneously in the *ME AV short-axis view* (see Fig. 10-2H), which is obtained by rotating the probe forward to 30 to 60 degrees. The noncoronary cusp is superior, lying adjacent to the atrial septum, the right cusp is inferiorly imaged, and the left cusp lies to the right pointing in the direction of the LA appendage (LAA). This view permits planimetry of the AV orifice, evaluation of congenital anomalies of the AV (e.g., bicuspid AV), and qualitative assessment of aortic regurgitation (AR) when color flow Doppler imaging is used. The *ME AV long-axis view* (see Fig. 10-2I) can be obtained at the same depth while rotating the probe to 120 to 160 degrees, allowing for visualization of the left ventricular outflow tract (LVOT), AV annulus and leaflets (right and either noncoronary or left), sinuses of Valsalva, sinotubular junction, and proximal ascending aorta. This view is particularly useful for evaluating AR with color flow Doppler imaging, systolic anterior motion of the MV, and proximal aortic pathology (dissections, aneurysms). Rotating the probe back to 90 to 120 degrees and advancing into the stomach to the transgastric level develops the *TG long-axis view* (see Fig. 10-2J). In this view, the LVOT and AV are oriented to the right and inferiorly in the displayed image, thereby providing an optimal window for parallel Doppler beam alignment for the assessment of flows and pressure gradients (aortic stenosis, hypertrophic obstructive cardiomyopathy). Rotating the probe back further to 0 to 20 degrees, advancing deep into the stomach and anteflexing the tip so that it lies adjacent to the LV apex allows for the development of the *deep TG long-axis view* (see Fig. 10-2K), which also provides optimal Doppler beam alignment for measuring transaortic valve and LVOT flow velocities and may also provide an additional window for assessing flows through muscular VSDs and LV apical pathology (thrombus, aneurysms).

Tricuspid Valve

The echocardiographic evaluation of the TV requires a thorough assessment of its three leaflets (anterior, posterior, and septal), annulus, chordae tendineae, papillary muscles, and corresponding RV walls. In the ME four-chamber view (see Fig. 10-2A), the septal TV leaflet is displayed on the right side and the posterior TV leaflet on the left side of the annulus. Rotating the multiplane angle to 60 to 90 degrees develops the ME RV inflow-outflow view (see Fig. 10-2M), which displays the posterior TV leaflet

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on the left side of the image and the anterior TV leaflet on the right side of the image adjacent to the AV. The *TG RV inflow view* (see Fig. 10-2N) is obtained by advancing the probe into the stomach and rotating to 100 to 120 degrees. This view is ideal for visualizing the chordae tendineae and papillary muscles in the RV. Rotating back to the TG mid short-axis view at 0 to 20 degrees and slightly withdrawing the probe provides a cross-sectional view of the TV, displaying the anterior leaflet in the far field, the posterior leaflet to the left in the near field, and the septal leaflet on the right side of the image.

Pulmonic Valve and Pulmonary Artery

The pulmonic valve (PV) is a trileaflet, semilunar valve. The ME AV short-axis view (see Fig. 10-2H) displays the transition between the RVOT and PV. Rotating the probe back toward 0 degrees and withdrawing slightly develops the *ME ascending aortic short-axis view* (see Fig. 10-2O), displaying the transition between the RV and main PA and its bifurcation. Although the right PA is usually easy to visualize by turning the probe to the right, the left PA is often obscured by the interposing, air-filled, left main-stem bronchus. This view can be used in the Doppler echocardiographic assessment of PV pathophysiology because of the parallel alignment of the beam relative to the flow and can also be used to locate pulmonary emboli. The ME RV inflow-outflow (see Fig. 10-2M) view can also be used to assess RV, PV, and main PA.

Left Atrium, Left Atrial Appendage, Pulmonary Veins, and Atrial Septum

The LA is the closest cardiac structure to the TEE probe when positioned in the esophagus. Consequently, the LA is usually easily displayed in the superior aspect of the 2D image sector. The ME four-chamber view (see Fig. 10-2A) displays the LA almost in its entirety with the LAA oriented to its superior and lateral aspect when the probe is slightly withdrawn. The muscular ridges of the pectinate muscles within the LAA should not be confused with thrombi. Slight further withdrawal of the probe and turning it to the left allows the left upper pulmonary vein (LUPV) to be imaged as it enters the LA from the anterior to posterior direction separated from the lateral border of the LAA by the "warfarin ridge." In contrast to the LUPV, which is usually optimally aligned for parallel Doppler beam alignment, the left lower pulmonary vein (LLPV) enters the LA just below the LUPV in a lateral-to-medial direction and is more perpendicularly aligned. Pulmonary venous Doppler flow-velocity profiles are useful for the qualitative and quantitative assessment of LV diastolic function. Turning the probe to the right at this depth reveals the right upper pulmonary vein (RUPV) entering the LA in an anterior-to-posterior direction. The right lower pulmonary vein (RLPV) can sometimes be visualized as it enters perpendicular to the long axis of the LA, by slightly advancing the probe.

The interatrial septum (IAS), consisting of thicker limbus regions flanking the thin fossa ovalis, can also be imaged in the ME four-chamber view. Benign lipomatous hypertrophy of the IAS must be distinguished from pathologic lesions such as atrial myxomas. The patency of the IAS and presence of PFO or congenital atrial septal defects (ASDs) should be assessed with Doppler echocardiography and intravenous injections of agitated saline or other contrast agents. Advancing and rotating the probe to 80 to 100 degrees develop the ME two-chamber view (see Fig. 10-2B), which allows for further imaging of the LA from left to right. The LAA and LUPV can be seen by turning the probe slightly to the left. Rotating the probe to the right at this level and adjusting the multiplane angle to 80 to 110 degrees develop the *ME bicaval view* (see Fig. 10-2L), which delineates the superior vena cava (SVC) entering the RA

to the right of the image and the inferior vena cava (IVC) entering from the left. The IAS can be seen in the middle of the image separating the LA and RA.

Right Atrium and Coronary Sinus

The RA can be most easily visualized in the ME four-chamber view (see Fig. 10-2A) by turning the probe to the patient's right side. In this view, the entire RA can be visualized for size, overall function, and the presence of masses (thrombi, tumors). Rotating the multiplane angle to 80 to 110 degrees develops the *ME bicaval view* (see Fig. 10-2L), which displays the RA and its internal structures (eustachian valve, Chiari network, crista terminalis). The SVC can be imaged entering the RA on the right, superior to the right atrial appendage (RAA), and the IVC enters the RA on the left of the display. Advancing and turning the probe to the right allow for a qualitative evaluation of the intrahepatic segment of the IVC and hepatic veins. Pacemaker electrodes and central venous catheters for hemodynamic monitoring or cardiopulmonary bypass (CPB) can be easily imaged in this view.

The coronary sinus (CS) lies posteriorly in the atrioventricular groove, emptying into the RA at the inferior extent of the atrial septum. The CS can be viewed in long axis entering the RA just superior to the tricuspid annulus by advancing and slightly retroflexing the probe from ME four-chamber view (see Fig. 10-2A). The CS can be imaged cross-sectionally in short axis in the ME two-chamber (see Fig. 10-2B) view in the upper left of the display. Turning the probe to the left in this view often allows visualization of the CS in long axis as it traverses the atrioventricular groove. The CS and thebesian valve can also be visualized in the *ME bicaval* view (see Fig. 10-2L) on the upper right of the image as the CS enters the RA at an obtuse angle, by turning the probe to leftward simultaneously with retro- and leftward flexion. Echocardiographic visualization of the CS can be useful for directing the placement of CS catheters used for CPB with retrograde cardioplegia.

Thoracic Aorta

The proximal and mid-ascending thoracic aorta can be visualized in short axis in the *ME ascending aortic short-axis view* (see Fig. 10-2O). Advancing and withdrawing the probe should enable visualization of the thoracic aorta from the sinotubular junction to a point 4 to 6 cm superior to the AV and allow inspection for aneurysms and dissections. Rotating the multiplane angle to 100 to 150 degrees develops the *ME ascending aortic long-axis view* (see Fig. 10-2P), which optimally displays the parallel anterior and posterior walls for measuring proximal and mid-ascending aortic diameters. This view can also be obtained from the ME AV long-axis view (see Fig. 10-2I) by slightly withdrawing and turning the probe to the left.

TEE imaging of the aortic arch is often obscured by the interposing, air-filled trachea. The most optimal views of the aortic arch are obtained by withdrawing the probe from the ME ascending aortic short-axis view at 0 degrees (see Fig. 10-2O) and rotating to the left to obtain the *upper esophageal aortic arch long-axis view* (see Fig. 10-2S), which displays the proximal arch followed by the mid arch, the great vessels (brachiocephalic, left carotid and left subclavian artery), and distal arch before it joins the proximal descending thoracic aorta imaged in cross section. Alternatively, rotating the probe to 90 degrees develops the *upper esophageal aortic arch short-axis view* view (see Fig. 10-2T). Turning the probe to the left, this view delineates the transition of the distal arch with the proximal descending thoracic aorta. Turning the probe to the right and slightly withdrawing it allows for the mid arch and great vessels to be imaged on the right side of the screen, followed by the distal ascending aorta when the probe is subsequently advanced

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and rotated forward to 120 degrees (ME ascending aortic long-axis view [see Fig. 10-2P]). Epiaortic scanning may be particularly useful for assessing the extent of ascending aortic and arch pathology (e.g., aneurysms, dissection, atherosclerosis) to determine cross-clamping and cannulation sites for CPB.

A short-axis image of the descending thoracic aorta is obtained by turning the probe leftward from the ME four-chamber view (see Fig. 10-2A) to produce the *descending aortic short-axis* view (see Fig. 10-2Q). Rotating the multiplane angle of the probe from 0 degrees to 90 to 110 degrees produces a long-axis image, the *descending aortic long-axis* view (see Fig. 10-2R). The descending thoracic aorta should be interrogated in its entirety, beginning at the distal aortic arch by continually advancing the probe and turning slightly to the left until the celiac and superior mesenteric arteries are visualized branching tangentially from the anterior surface of the abdominal aorta when the probe is in the stomach. Thorough examination of the descending thoracic aorta may be necessary to evaluate the distal extent of an aneurysm or dissection. In addition, the descending aortic short- and long-axis views can be useful for confirming appropriate intra-aortic balloon positioning.

CLINICAL APPLICATIONS

Ventricular Function

Cardiovascular function, such as the global indices of muscle contraction or regional indices described by segmental wall motion, is assessed by analyzing moving echocardiographic images. Assessment of global and regional ventricular function has become the cornerstone for evaluating patients with ischemic heart disease (IHD). The dynamic assessment of ventricular function with echocardiography is based on derived indices of muscle contraction and relaxation. Echocardiographic indices of LV function that incorporate endocardial border outlines and Doppler techniques can be used to estimate CO, stroke volume (SV), ejection fraction (EF), and parameters of ventricular relaxation and filling.

Global LV performance is directly related to preload, contractility, and afterload. Cardiac output reflects systolic function and is an important factor in oxygen delivery. Alteration in LV diastolic function may result from systolic dysfunction or, in as many as 40% of patients, may be the primary and main etiology of cardiac failure.

Visual Estimation of Function

In routine clinical work, the anesthesiologist-echocardiographer must make visual estimates of ventricular wall motion and overall function. These depend on the ability of the individual to interpret the images and correctly "quantify" subjective assessments of ventricular function. The clinician may be aided by video recordings of prior function or video images stored on split screens. These estimations of global ventricular function are usually performed in the short-axis view of the LV, but additional information can also be gained by assessing the long-axis views of the LV. In this assessment, the observer examines the end-diastolic image and compares it with the end-systolic frame to determine the degree of ejection. The rate of ejection is also estimated.

Regional wall motion abnormalities (RWMAs) may be identified on visual inspection of echocardiographic images by anesthesiologists. Several studies have documented that an educated visual analysis of echocardiographic images provides a better estimation of LVEF than more sophisticated and time-consuming computer techniques.

Preload/Diastolic Function

End-Diastolic Dimensions

Whereas in conventional hemodynamics preload is often estimated by measuring left-sided heart filling pressures (pulmonary capillary wedge pressure [PCWP], left atrial pressure [LAP], or LV end-diastolic pressure [LVEDP]), in echocardiography it can be determined by measuring LV end-diastolic dimensions. End-diastolic dimensions provide a better index of preload than the PCWP. When PCWP and end-diastolic volume (EDV), derived from short-axis areas at the level of the papillary muscles, were compared as predictors of cardiac index (CI) in patients undergoing coronary artery bypass grafting (CABG), a strong correlation was observed between end-diastolic area (EDA), or EDV, and CI, whereas no significant correlation was found between PCWP and CI.

TEE is often, for practical reasons, limited to a single short-axis view at the level of the papillary muscles. Some evidence suggests that short-axis EDAs measured at this level correlate reasonably well with measurements obtained by on-heart echocardiography and with EDVs measured simultaneously using radionuclides. There are two main echocardiographic signs of decreased preload: (1) decrease in EDA (<5.5 cm^2/m^2) invariably reflects hypovolemia; and (2) obliteration of the end-systolic area (ESA) ("the kissing ventricle sign") accompanies the decrease in EDA in severe hypovolemia.

Myocardial Ischemia Monitoring

Regional Wall Motion and Systolic Wall Thickness

The relationship of echocardiographic indices of regional myocardial function to ischemia has been compared with changes that occur with the surface ECG, PCWP, and the onset of chest pain. As early as 1935 it was recognized that acute myocardial ischemia results in abnormal inward motion and thickening of the affected myocardial region. Since then, RWMAs have been shown to occur within seconds of inadequate blood flow or oxygen supply. These abnormal contraction patterns typically occur at the same time as regional lactate production.

Diagnosis of Ischemia

The precise sequence of functional changes that occur in the myocardium after interruption of flow has been studied in models of acute ischemia, including percutaneous transluminal coronary angioplasty (PTCA). Abnormalities in diastolic function usually precede abnormal changes in systolic function. Normal function is critical for LV filling and is dependent on ventricular relaxation, compliance, and atrial contraction. Diastolic ventricular function can be assessed by monitoring the rate of filling associated with changes in the chamber dimensions. Regional systolic function can be estimated by echocardiographic determination of wall thickening and wall motion during systole in both long- and short-axis views of the ventricle. The short-axis view of the LV at the papillary muscle level displays myocardium perfused by the three main coronary arteries and is, therefore, very useful. However, because the short-axis view does not image the ventricular apex and this is a very common location of ischemia, the long-axis and longitudinal ventricle views are also clinically important.[3]

As the myocardial oxygen supply/demand balance worsens, graded RWMAs progress from mild hypokinesia to severe hypokinesia, akinesia, and finally dyskinesia. *Normal contraction* is defined as greater than 30% thickening of the ventricular wall. *Mild hypokinesia* refers to inward contraction that is slower and less vigorous than normal during systole, with ventricular wall thickening of 10% to 30%.

BOX 10-4   *Limitations of Transesophageal Echocardiography*

Incompatibility: induction, laryngoscopy, intubation, emergence, extubation
Artifact interference: electronic, septum, bundle-branch block
Lack of specificity: tethering effect, scar, afterload changes, stunned myocardium

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Severe hypokinesia is defined as less than 10% wall thickening. The precise distinction between varying degrees of hypokinesia can be difficult. *Akinesia* refers to the absence of wall motion or no inward movement of the endocardium during systole. *Dyskinesia* refers to paradoxical wall motion or movement outward during ventricular systole.

Limitations

Although TEE appears to have many advantages over traditional intraoperative monitors of myocardial ischemia, there remain potential limitations as well (Box 10-4). The most obvious limitation of TEE monitoring is the fact that ischemia cannot be detected during critical periods, such as induction, laryngoscopy, intubation, emergence, and extubation. In addition, the adequacy of RWMA analysis may be influenced by artifact.

The septum in particular must be given special consideration with respect to wall motion and wall thickness assessment. The septum is composed of two parts: the lower muscular portion and the basal membranous portion. The basal septum does not exhibit the same degree of contraction as the lower muscular part. At the most superior basal portion, the septum is attached to the aortic outflow track. Its movement at this level is normally paradoxical during ventricular systole. The septum is also a unique region of the LV, because it is a region of the RV as well and is therefore influenced by forces from both ventricles. In addition, sternotomy, pericardiotomy, and CPB have been found to alter the translational and rotational motion of the heart within the chest, which may cause changes in ventricular septal motion.

Another potential problem of RWMA assessment is evaluation of the dyssynchronous contraction that occurs as a result of a bundle-branch block or ventricular pacing. In these situations, the system used to assess RWMAs must compensate for global motion of the heart and evaluate not only regional endocardial wall motion but also myocardial thickening.

Not all RWMAs are indicative of myocardial ischemia or infarction. Clearly, under normal conditions, all hearts do not contract in a homogeneous and consistent manner. It is reasonable to assume, however, that most of the time an acute change in the regional contraction pattern of the heart during surgery is likely attributable to myocardial ischemia. An important exception to this rule may apply in models of acute coronary artery occlusion. In these models, it has been established that myocardial function becomes abnormal in the center of an ischemic zone, but it is also true that the myocardial regions adjacent to the ischemic zones become dysfunctional as well. Several studies have reported that the total area of dysfunctional myocardium commonly exceeds the area of ischemic or infarcted myocardium. The impairment of function in nonischemic tissue has been thought to be caused by a "tethering effect." Tethering, or the attachment of noncontracting tissue that is normally perfused, probably accounts for the consistent overestimation of infarct size by echocardiography when compared with postmortem studies.

Another limitation of RWMA analysis during surgery is that it does not differentiate stunned or hibernating myocardium from acute ischemia, nor does it differentiate the cause of ischemia between increased oxygen demand and decreased

oxygen supply. Finally, it should be noted that areas of previous ischemia or scarring may become unmasked by changes in afterload and appear as new RWMAs. This is particularly important in vascular surgery, in which major abrupt changes in afterload occur.
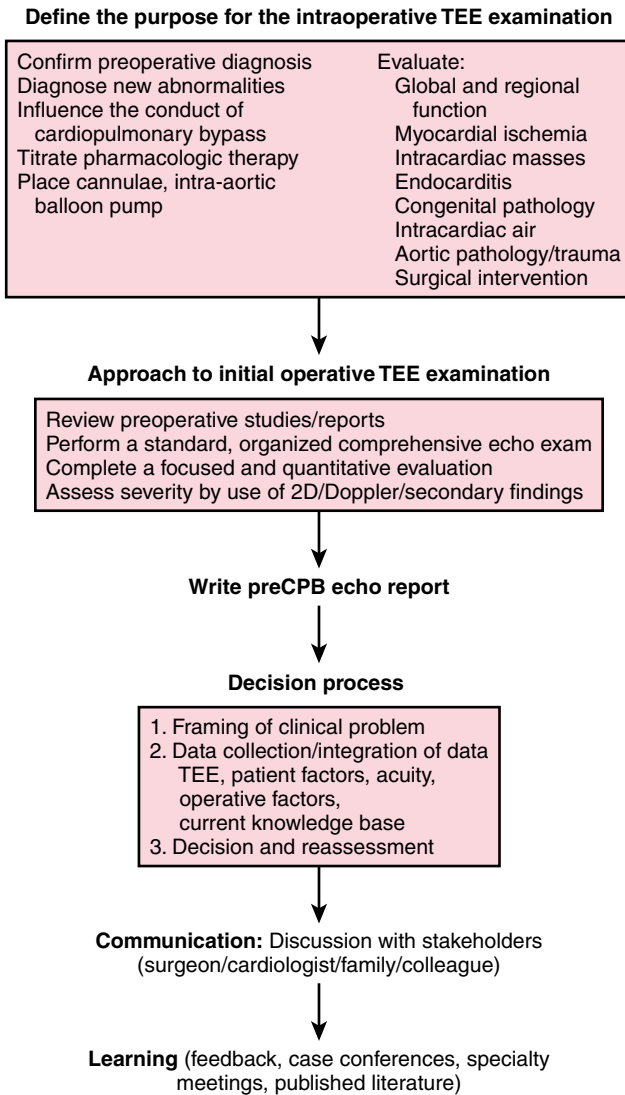
Outcome Significance

Data regarding the significance of intraoperative detection of RWMAs suggest that transient abnormalities unaccompanied by hemodynamic or ECG evidence of ischemia may not represent significant myocardial ischemia and are usually not associated with postoperative morbidity. Hypokinetic myocardial segments appear to be associated with minimal perfusion defects compared with the significant perfusion defects that accompany akinetic or dyskinetic segments. Hence, hypokinesia may be a less predictive marker for postoperative morbidity.[4]

Intraoperative TEE has helped predict the results of CABG. Following CABG of previously dysfunctional segments, immediate improvement of regional myocardial function (which is sustained) has been demonstrated. In addition, prebypass compensatory hypercontracting segments have been reported to revert toward normal immediately after successful CABG. Persistent RWMAs after CABG appear to be related to adverse clinical outcomes, and lack of evidence of RWMAs after CABG has been shown to be associated with a postoperative course without cardiac morbidity.

A formalized approach to the acquisition of data and decision-making (Fig. 10-3) enhances the quality of the intraoperative echocardiogram, its interpretation, and the confidence with which the findings are communicated to other members of the operative and nonoperative teams. The broad database that is required to formulate an intelligent decision includes provider- and patient-specific data. Patient-specific data include history and demographics, preoperative diagnostic examinations, admitting diagnosis and comorbidities, the patient's wishes, recommendations of referring physicians, and intraoperative data. Intraoperative data include hemodynamic data, visual inspection, surgical input, and the TEE examination. A systematic TEE examination of the heart and great vessels permits the acquisition and interpretation of qualitative and quantitative echocardiographic data applied to intraoperative decision-making. The provider-specific data are composed of an accumulated database of knowledge acquired from training, experience, and continuous medical education.

INTRAOPERATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY: INDICATIONS

The first decision by the echocardiographer is whether TEE is indicated. Application of intraoperative TEE in the care of the patient with mitral disease is widely accepted. Even in this area, however, there is a paucity of data supporting an improved outcome for intraoperative patients cared for with TEE compared with no TEE. The decision to perform TEE during cardiac surgery is substantiated by practice expectations and consensus opinion.[5] In an attempt to develop an evidence-based approach to this expanding technology, the American Society of Anesthesiologists (ASA) and the Society of Cardiovascular Anesthesiologists (SCA) co-sponsored a task force to develop guidelines for defining the indications for perioperative TEE.[2,6] Despite the scarcity of outcome data to support the application of TEE in the perioperative period, TEE had rapidly been adopted by cardiac surgeons and cardiac anesthesiologists as a routine monitoring

Define the purpose for the intraoperative TEE examination

Confirm preoperative diagnosis	Evaluate:
Diagnose new abnormalities	Global and regional
Influence the conduct of	function
cardiopulmonary bypass	Myocardial ischemia
Titrate pharmacologic therapy	Intracardiac masses
Place cannulae, intra-aortic	Endocarditis
balloon pump	Congenital pathology
	Intracardiac air
	Aortic pathology/trauma
	Surgical intervention

Approach to initial operative TEE examination

Review preoperative studies/reports
Perform a standard, organized comprehensive echo exam
Complete a focused and quantitative evaluation
Assess severity by use of 2D/Doppler/secondary findings

Write preCPB echo report

Decision process

1. Framing of clinical problem
2. Data collection/integration of data
   TEE, patient factors, acuity,
   operative factors,
   current knowledge base
3. Decision and reassessment

Communication: Discussion with stakeholders
(surgeon/cardiologist/family/colleague)

Learning (feedback, case conferences, specialty
meetings, published literature)

Figure 10-3 An algorithm for the decision-making process.

and diagnostic modality during cardiac surgery. In 1996, the task force published their guidelines designed to establish the scientific merit of TEE and justification of its use in defined patient cohorts. The indications were grouped into three categories based on the strength of the supporting evidence/expert opinion that TEE improves outcome (Box 10-5). Category I indications suggested strong evidence/expert opinion that TEE was useful in improving clinical outcome. Category II indications suggested there was weak evidence/expert opinion that TEE improves outcome in these settings. Category III indications suggested there was little or no scientific merit or expert support for the application of TEE in these settings.

BOX 10-5 *Indications for the Use of Transesophageal Echocardiography*

Category I

- Heart valve repair
- Congenital heart surgery
- Hypertrophic obstructive cardiomyopathy
- Endocarditis
- Acute aortic dissection
- Acute, unstable aortic aneurysm
- Aortic valve function in the setting of aortic dissection
- Traumatic thoracic aortic disruption
- Pericardial tamponade

Category II

- Myocardial ischemia and coronary artery disease
- Increased risk of hemodynamic disturbances
- Heart valve replacement
- Aneurysms of the heart
- Intracardiac masses
- Intracardiac foreign bodies
- Air emboli
- Intracardiac thrombi
- Massive pulmonary emboli
- Traumatic cardiac injury
- Chronic aortic dissection
- Chronic aortic aneurysm
- Detection of aortic atheromatous disease as a source of emboli
- Evaluating the effectiveness of pericardiectomies
- Heart-lung transplantation
- Mechanical circulatory support

Category III

- Other cardiomyopathy
- Emboli during orthopedic procedures
- Uncomplicated pericarditis
- Pleuropulmonary disease
- Placement of intra-aortic balloon pump, pulmonary artery catheter
- Monitoring the administration of cardioplegia

Modified from published guidelines of the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists.

CASE STUDIES OF INTRAOPERATIVE TEE

Case Study 1	CARDIAC FUNCTION AND REGIONAL WALL MOTION ABNORMALITIES

FRAMING

Ventricular function is a predictor of outcome after heart surgery and a predictor of long-term outcome in patients with cardiovascular disease. Patients with compensated congestive heart failure may have severely decreased EF with minimal

symptoms. Regional ventricular dysfunction is most commonly caused by myocardial ischemia or infarction. Hence, there is an imperative to detect ventricular dysfunction and institute treatment in an attempt to prevent acute or long-term consequences.

Is ventricular function normal or abnormal? Is the abnormal function global or regional? What is the coronary distribution that relates to an RWMA? Is the ventricle big or small? Is the myocardium thinned or hypertrophied? Is the abnormal function new or old? Does the medical or surgical intervention improve or deteriorate ventricular function?

DATA COLLECTION

LV systolic function is assessed echocardiographically based on regional and global wall motion. Methods of assessment include changes in regional wall thickness, radial short-ening with endocardial excursion, fractional area change (FAC), and systolic displacement of the mitral annulus. Off-line measurements of EF can be calculated using Simpson's rule. FAC is the most common metric used to assess global LV function. Other measures include end-diastolic area (EDA), end-systolic area (ESA), and meridional wall stress.

Regional assessment provides an index of myocardial well-being that can be linked to coronary anatomy and blood flow. Although the measurement of coronary blood flow is not achieved by TEE, the perfusion beds and corresponding myocar-dium for the left anterior descending (LAD), left circumflex (LCX), and right coronary (RCA) arteries are relatively distinct and can be scrutinized by TEE using multiplane imaging. The transgastric and long-axis imaging views of the LV are the most widely used for evaluating wall motion abnormalities. Digital archival systems have gained popularity for their ability to capture a single cardiac cycle that can then be exam-ined more closely as a continuous cine loop. Cine loops can also permit side-by-side display of images obtained under varying conditions (e.g., prebypass and postby-pass). Regional myocardial ischemia produces focal changes in the corresponding ventricular walls before changes occur on the ECG. Changes progress from normal wall motion to hypokinesis or akinesis. Dyskinesis, thinning, and calcification of the myocardium suggest a nonacute process, likely a prior infarction.

DISCUSSION

Preexisting ventricular dysfunction suggests increased risk for surgery and poorer long-term outcome. The presence of such ventricular dysfunction may deteriorate intra-operatively, requiring the need for marked pharmacologic or mechanical support. A patient with a preoperative EF of 10% scheduled for coronary artery bypass grafting (CABG) and MV repair is at increased risk of intraoperative ischemia, acute heart fail-ure, and difficulty maintaining hemodynamic stability during the immediate postby-pass period. Anticipating such problems, placement of an intra-aortic balloon pump or femoral arterial catheter is considered during the prebypass period (Fig. 10-4). The same patient is likely to benefit from the administration of inotropic agents.

A marked decrement or unexpected decrease in global cardiac function after release of the aortic cross-clamp can be caused by poor myocardial preservation dur-ing cross-clamping or distention of the heart during bypass. The risk of such inci-dents can be reduced by the monitoring of the electrical activity of the heart and pulmonary artery pressures and for distention of the RV and LV. Effective venting of the heart is often difficult to discern by visual inspection alone, especially with the use of minimally invasive surgery through small incisions. TEE imaging can diagnose ventricular distention produced by AV insufficiency.

Not all preexisting regional wall motion abnormalities benefit from coronary revascularization. Regions of akinesia and dyskinesia are usually the result of a myo-cardial infarction and may reflect nonviable myocardium, although "hibernating" myocardium is possible. Hypokinetic segments are generally viable and may represent active ischemia. Preoperative positron emission tomography (PET) scanning can detect hibernating myocardium and may be cost-effective to guide CABG.[7] The detection of hibernating myocardium in an area of chronic ischemia and regional hypokinesis will direct the surgeon to revascularize the corresponding stenosed coronary artery. In contrast, an occluded coronary artery with downstream infarction may not ben-efit from revascularization, as contractile function may be irreversibly lost. However, in this latter scenario, revascularization postinfarction may provide some benefit in decreasing the risk of ventricular aneurysm formation.

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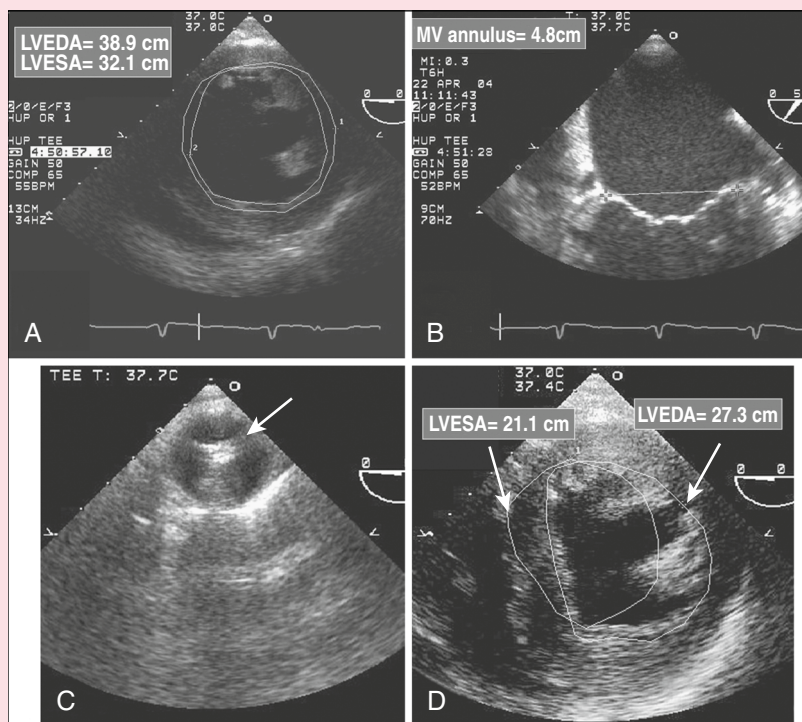
Figure 10-4   The prebypass transesophageal echocardiography (TEE) examination may have predictive value for postbypass circulatory management. A 63-year-old woman with a past medical history of hypertension, congestive heart failure, pulmonary edema, dilated cardiomyopathy, diabetes, and obesity was scheduled for coronary artery bypass grafting (CABG) and mitral valve (MV) repair. The preoperative evaluation documented moderate to severe mitral regurgitation (MR) with reversal of systolic pulmonary vein blood flow velocity. The prebypass TEE mid-esophageal four-chamber view showed a markedly dilated left ventricle (LV) and mildly dilated right ventricle (RV) with mildly decreased global dysfunction. **A)** The transgastric view was characterized by severe global dysfunction and a LV end-diastolic diameter of 6.6 cm. The fractional area change (FAC) was 17% [FAC = (LV end-diastolic area − LV end-systolic area)/LV end-diastolic area × 100]. Revascularization alone was unlikely to significantly improve MV function. **B)** The mid-esophageal bicommissural view of the MV demonstrated marked dilation of the MV annulus (major axis = 4.8 cm) and tethering of the leaflets below the valve plane that was caused by LV chamber dilation. A femoral arterial catheter was inserted for monitoring of central aortic pressure and/or possibly placing an intra-aortic balloon pump. The patient underwent a CABG × 3 and MV annuloplasty for moderate MR. The separation from bypass was difficult, requiring milrinone, epinephrine, vasopressin, and placement of an intra-aortic balloon pump. **C)** TEE, which was used to initially confirm the location of the femoral guidewire, was later used to position the balloon pump just downstream to the left subclavian artery. **D)** Worsening of RV function that was characterized by increased central venous pressure, new-onset tricuspid regurgitation, and a hypokinetic RV can be appreciated by ventricular septal flattening and dilation of the RV. The LV ejection fraction did not decrease as might be expected; after correcting MR, the FAC improved slightly from 17% to 22% post-bypass. Cardiac function continued to improve, and the counterpulsation device was removed without complication on the first day after surgery. The infusions of milrinone and epinephrine were continued for several days.

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If the intraoperative examination reveals new ventricular dysfunction, the intra-operative team must determine the etiology and severity and plan a treatment. Other causes of RWMAs such as conduction abnormalities (left bundle-branch block or ventricular pacing) can be difficult to distinguish. Treatment of myocardial ischemia may include optimizing hemodynamics; administering anticoagulants, nitrates, calcium channel blockers, or β-blockers; inserting an intra-aortic balloon pump; or instituting CPB and coronary revascularization. The presence of a new-onset RWMA after separation from bypass is worrisome for myocardial ischemia. Even the patient without coronary artery disease remains at risk because of hypotension, shower of air or debris into the coronary circulation, or coronary spasm. The patient with coronary artery disease undergoing coronary revascularization may have all the above risks, technical difficulties at the anastomotic site, injury to the native coronary (e.g., stitch caught the back wall), or occlusion of the coronary graft by thrombosis or aortic dissection. The coronary arteries, grafts, and anastomoses should be carefully inspected for patency and flow. Graft patency in the operating room is difficult to determine. Techniques include manual stripping and refill, measuring coronary flow by hand-held Doppler, or administration of echocardiographic contrast agents. A new RWMA in the distribution of a new coronary graft can prompt the decision-making strategies listed in Table 10-2.

Table 10-2   Management Strategies for New-Onset Myocardial Ischemia Post Bypass

Diagnosis	Plausible Treatment
Coronary graft occlusion	Revise coronary graft
Coronary air emboli	Increase coronary perfusion pressure, administer coronary dilators
Coronary calcium/atheroma emboli	Support circulation
Dissection of the aortic root	Repair dissection
Coronary spasm	Administer coronary dilators

Case Study 2   MANAGEMENT OF ISCHEMIC MITRAL REGURGITATION

FRAMING

Ischemic heart disease is the most common cause of mitral insufficiency in the United States. Mechanisms of valve incompetence are varied and include annular dilatation, papillary muscle dysfunction from active ischemia or infarction, papillary muscle rupture, or ventricular remodeling from scar, often leading to a tethering effect of the subvalvular apparatus. Mitral regurgitation leads to pulmonary hypertension, pulmonary vascular congestion, and pulmonary edema with functional disability. Ventricular function deteriorates as the LV becomes volume overloaded with corresponding chamber dilatation. Left untreated, severe MR from ischemic heart disease has a poor prognosis, hence the imperative for diagnosis and treatment.[8] Patients presenting for surgical coronary revascularization often have concomitant MR of a mild or moderate degree. The intraoperative team is confronted with the decision of whether to surgically address the MV during the coronary operation.

Does MR warrant mitral surgery? What is the mechanism of the regurgitation? What is the grade of the MR? Is the MR likely to improve by coronary revascularization alone?

DATA COLLECTION

Pertinent data, including preoperative functional status and evaluation, need to be considered to appropriately interpret and place the intraoperative data in context. The preoperative echocardiogram and ventriculogram need to be reviewed. The

intraoperative hemodynamic data are coupled with TEE information to complete the dataset needed to move forward with the decision-making process. The severity of MR on TEE is measured by the vena contracta, maximum area of the regurgitant jet, regurgitant orifice area, and pulmonary vein blood flow velocities. Wall motion assessment and the ECG are used for detecting reversible myocardial dysfunction that may benefit from revascularization. The hemodynamic and TEE data are coupled with provocative testing of the MV in an attempt to emulate the working conditions of the MV in an awake, unanesthetized state. It is not uncommon that preoperative mild to moderate MR with a structurally normal valve totally resolves under the unloading conditions of general anesthesia.[9]

DISCUSSION

Most cases of ischemic MR are categorized as "functional" rather than structural. In a study of 482 patients with ischemic MR, 76% had functional ischemic MR, compared with 24% having significant papillary muscle dysfunction. The mechanism of ischemic MR is attributed to annular dilatation, secondary to LV enlargement and regional LV remodeling with papillary muscle displacement, causing apical tethering and restricted systolic leaflet motion. The importance of local LV remodeling with papillary muscle displacement as a mechanism for ischemic MR has been reproduced in an animal model.

The MR is prioritized in accordance with principal diagnosis (e.g., coronary artery disease), comorbidities, functional disability, and short- and long-term outcome. Ischemic MR is quantitated and the mechanism of valve dysfunction is defined. Intraoperative MR is compared with preoperative findings. Discrepancies between the preoperative and intraoperative assessment of the valve may reflect the pressure and volume unloading effects of general anesthesia. In patients with functional ischemic MR who have 1 to 2+ MR, the MV is often not repaired or replaced. However, the need for surgical intervention in patients with 2+ MR under anesthesia remains a point of debate and has not been definitively answered by prospective studies. MV surgery is typically recommended to improve functional status and long-term outcome for patients with 3+ ischemic MR or greater. Ignoring significant ischemic MR at the time of CABG can limit the functional benefit derived from surgery.

The risks to the patient of not surgically altering the MV and anticipated residual regurgitation is weighed against the risk of atriotomy, mitral surgery, extending cardiopulmonary and aortic cross-clamp times, and the likelihood that the coronary surgery will be successful at decreasing the severity of MR. Added risk includes commitment to a mechanical prosthesis should a reparative procedure prove unsuccessful. MR due to acute ischemia may resolve after restoration of coronary blood flow (Fig. 10-5). The reversibility of the regurgitation is difficult to predict: factors supporting reversibility (and hence no immediate need to surgically address the valve) include a structurally normal MV, normal LA and LV dimension, including the mitral annulus, and RWMAs associated with transient regurgitation and pulmonary edema. Revascularization of the culprit myocardium with improvement in regional function may be all that is necessary to restore normal mitral coaptation.[10] Myocardial infarction with a fixed wall motion defect or aneurysm, chronically dilated left-sided heart, dilated annulus, or other structural abnormalities that are not reversible (ruptured papillary muscle or chordae, leaflet prolapse, leaflet perforation) suggest myocardial revascularization is unlikely to correct the valvular incompetence.

The decision to proceed or not to proceed with mitral surgery in the setting of ischemic heart disease is institution and surgeon dependent. Centers may elect to surgically address any degree of MR detected during the preoperative or intraoperative workup of a patient scheduled for coronary surgery. Less aggressive sites elect to proceed with coronary revascularization, followed by repeat scrutiny of the ventricular wall motion and MV. If revascularization has not corrected the MR, the surgeon proceeds with CPB and mitral surgery. With the advent of off-pump coronary artery bypass surgery, this process has gained another level of complexity, because decisions to proceed with mitral repair will commit the patient to CPB. Off-pump mitral surgical procedures may be possible in the near future.

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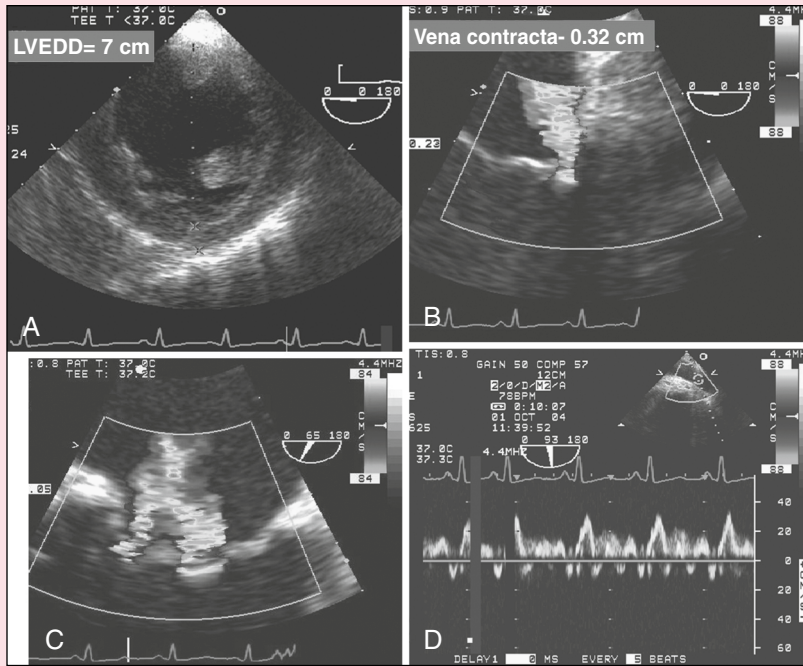
Figure 10-5 Evaluation of mitral regurgitation (MR) in a patient undergoing coronary artery bypass grafting. A 63-year-old man was scheduled to undergo off-pump coronary artery revascularization. The patient had a history of progressive congestive heart failure without evidence of acute pulmonary edema. The physical examination was significant for diffuse laterally displaced point of maximum impulse (PMI) and a systolic murmur at the apex that radiated to the axilla. The patient received an intraoperative transesophageal echocardiography (TEE) examination to evaluate the severity of MR. **A)** The left ventricle (LV) was significantly dilated with an LV end-diastolic dimension of 7 cm and had depressed systolic function with an estimated ejection fraction of 40%. The MR was characterized by color-flow Doppler imaging to be a central jet of mild to moderate severity. **B)** The grading of MR was based on the area of the regurgitant jet and the vena contracta viewed in a bicommissural view. The pathogenesis of MR was believed to be functional and resulted from restricted leaflet mobility caused by the dilated LV. **C)** The coaptation of the anterior and posterior leaflets was below the valve plane. **D)** The absence of reversal of pulmonary vein blood flow measured in the left lower pulmonary vein supported the assessment of moderate MR. Because the annulus was not significantly dilated (the minor axis measured 2.97 cm) and the MR graded as only mild to moderate, the surgeon proceeded with his initial plan of off-pump coronary artery bypass grafting. The MR decreased immediately after revascularization, and the patient's symptoms were expected to further improve with afterload reduction.

Case Study 3   MANAGEMENT OF PREVIOUSLY UNDIAGNOSED AORTIC VALVE DISEASE

FRAMING

A relatively common clinical scenario for the echocardiographer is to assess the significance of previously unrecognized AV pathology. This discussion has pertinence for the echocardiographer faced with the new diagnosis of a bicuspid valve, AS, or insufficiency.

    What are the symptoms that brought the patient to medical attention? What is the patient's baseline function? What is the anatomy of the AV? What is the severity

of AR or of AS? How do the intraoperative findings of AV disease differ from the preoperative assessment? Would surgical repair or replacement of the AV benefit the patient's short- or long-term outcome? What is the planned procedure, and how would the risks be changed if the procedure was altered to address the new finding? Does another health care provider need to be involved in the decision of whether to surgically address the valve? Is the pathology of the AV significant enough to require surgical intervention at this time?

DATA COLLECTION AND CHARACTERIZATION OF THE AORTIC VALVE

Multiplane TEE permits an accurate assessment of AV area, valvular pathology, severity of regurgitation and stenosis, and detection of secondary cardiac changes. In the case of AS, the severity of valvular dysfunction is determined by measuring the transvalvular pressure gradient, by calculating the AV area using the continuity equation, and by planimetry of the AV systolic orifice. Planimetry of the AV orifice with TEE is more closely correlated with the catheterization-determined valve area (using the Gorlin formula) than the value derived from TTE ($r = 0.91$ vs. 0.84). The severity of AR by TEE is generally graded with color flow Doppler imaging with measurement of the width of the regurgitant jet relative to the width of the LV outflow tract. TEE is sensitive to even the most trivial amount of AR. Jet areas measured by TEE tend to be larger, and their severity is graded as greater compared with AR assessed by TTE. Determining the clinical significance of AR typically requires assessment of more than just regurgitant grade, although severe 4+ AR is never left unaddressed.

The etiology and extent of AV disease can be best delineated by TEE, as shown in Figure 10-6. The relatively high resolution of the AV and associated structures in the near field of the midesophageal short- and long-axis views permits an accurate assessment of the severity and mechanism of valvular disease. The aortic leaflets should be inspected in the midesophageal long-axis view for the presence of vegetations, perforation, restriction, thickening/calcification, malcoaptation, and leaflet prolapse. The presence of subvalvular disease, such as a discrete fibrous subaortic membrane, can also be reliably excluded. The ascending aorta from the valve to the right pulmonary artery also should be viewed in long axis. This view is usually optimal for examining associated pathology of the aortic root and ascending aorta (e.g., aortoannular ectasia, bicuspid valve, type A aortic dissection).

AS is caused by calcification of the AV and rheumatic heart disease. Bicuspid AVs are at greater risk compared with the general population. AS produces a systolic pressure gradient between the LV and aorta. Secondary findings are dependent on where the patient's condition is along the natural course of the disease. Secondary findings often contribute to the decision-making process, because they infer the

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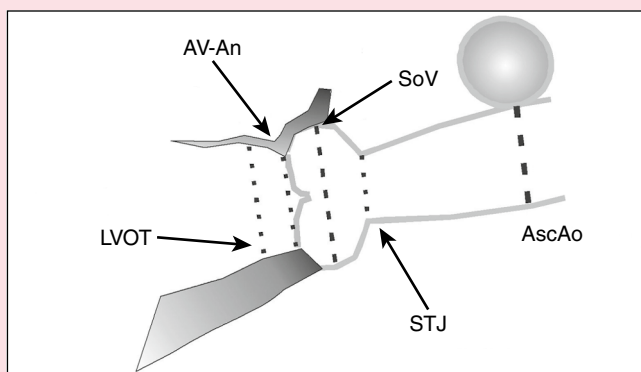
Figure 10-6 Anatomy of the aortic root. This schematic figure of the aortic valve in long axis shows the components of the aortic root, which include sinotubular junction (STJ), sinus of Valsalva (SoV), and the annulus of the aortic valve (AV-An). LVOT = left ventricular outflow tract; AscAo = ascending aorta.

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effects or consequences of the disease. AS is commonly associated with LV hypertrophy and abnormal filling of the LV. The diastolic function is often impaired owing to a thickened, noncompliant LV. Hence, MV and pulmonary vein blood flow velocities would demonstrate a blunted passive filling phase of the ventricle. Systolic function is often normal or hyperdynamic. The LV chamber size is normal or small. However, long-standing AS results in progressive ventricular systolic dysfunction and heart failure. The LV becomes dilated with compromised contractile function. As the ventricle fails, CO decreases with a resultant decrease in trans-AV pressure gradient. Hence, the pressure gradient across an AV may be misleading as a measure for severity of AS.

NATURAL COURSE OF AORTIC STENOSIS

The natural course of AS in the adult begins with a prolonged asymptomatic period associated with minimal mortality. Progression of the disease is manifested by a reduction in the valve area and an increase in the transvalvular systolic pressure gradient. The progression is quite variable, exhibiting a decrease in effective valve area ranging from 0.1 to 0.3 cm^2/yr. AV calcification, as depicted by echocardiography, has been suggested to be an independent predictor of outcome. Patients with no or mild valvular calcification, compared with those with moderate or severe calcification, had significantly increased rates of event-free survival at 1 and 4 years (92% vs. 60% and 75% vs. 20%, respectively). Decisions regarding valve replacement for mild or moderate AV disease in the setting of cardiac surgery for another cause are complicated by the variability in the natural progression of the disease. The pathogenesis of AS is an active process having many similarities to the progression of atherosclerosis. AV calcification is not a random degenerative process but an actively regulated disease associated with hypercholesterolemia, inflammation, and osteoblast activity. More aggressive medical control of these processes might be expected to have a positive impact on outcome by retarding the degenerative process.

ASSESSMENT OF MILD AND MODERATE AORTIC STENOSIS

The intraoperative management of mild to moderate AS at the time of cardiac surgery remains controversial. A patient arrives in the operating room scheduled for a CABG but is discovered to also have mild or moderate AS that was unappreciated preoperatively. The operative team must decide whether to surgically address the AV. The ACC/AHA task force recommends valve replacement at the time of coronary surgery if the asymptomatic patient has severe AS but acknowledges there are very limited data to support intervention in the case of mild or moderate AS. It is in this exact scenario that the rate of progression of AS is of value but it is rarely obtainable. A rapidly calcifying valve in a young patient that is becoming rapidly stenotic would sway the operative team to perform an aortic valve replacement (AVR). A combined double cardiac procedure (CABG/AVR) increases the initial perioperative risk, as well as those risks associated with long-term prosthetic valve implantation. A delay in AVR and commitment to a second heart operation in the future subjects the patient to the risk of a repeated sternotomy in the setting of patent coronary grafts and its associated morbidities. If the AV is not operated on during the initial presentation for CABG, the development of symptomatic AS may be quite delayed or may not happen.

A review of 1,344,100 patients in the national database of the Society of Thoracic Surgeons having CABG, CABG/AVR, or AVR alone culminated in a decision paradigm recommendation. The study assumed rates of AV disease progression (pressure gradient of 5 mm Hg/yr), valve-related morbidity, and age-adjusted mortality rates that were obtained from published reports.[11] The authors proposed three factors in the consideration of CABG or AVR/CABG: age (life expectancy), peak pressure gradient, and rate of progression of the AS (if known). Since the latter is difficult to discern, the analysis assumed an average rate of disease progression and recommended patients should undergo AVR/CABG when the pressure gradient exceeds 30 mm Hg. The threshold (AS pressure gradient) to perform both procedures is increased for patients older than 70 years of age because the reduced life expectancy diminishes the likelihood that they will become symptomatic from the AV disease. Whether to perform a concomitant AVR at the time of revascularization was also addressed by Rahimtoola, who advocated a less aggressive approach.[12] One problem with both studies is that they analyzed the transvalvular pressure gradient, which may be a misleading measure of the degree of stenosis of the AV, as its value is dependent

on CO. A low CO and flow rate will produce a low transvalvular pressure gradient, even in the setting of a severely stenotic AV. However, in the setting of preserved ventricular systolic function and mild or moderate AS, a pressure gradient is a useful metric. The variable rate of disease progression and the controversy regarding the indications for "prophylactic" AVR preclude a simple algorithm for dealing with this patient cohort. Increased age, lack of symptoms, minimal LV hypertrophy, with a valve area suggesting milder disease, and a pressure gradient less than 30 mm Hg would sway the decision to not replace the AV. In an asymptomatic young patient, a severely calcified valve, bicuspid valve, and LV hypertrophy in the setting of moderate stenosis, and a pressure gradient greater than 30 mm Hg would suggest that an AVR might be beneficial in the long term. It is often useful to include the patient's primary cardiologist and family in the decision-making process.

ASSESSMENT OF LOW PRESSURE GRADIENT AORTIC STENOSIS

Patients with LV dysfunction and decreased CO in the setting of AS often present with only modest transvalvular pressure gradients (<30 mm Hg). Distinguishing patients with a low CO and severe AS from patients with mild to moderate AS can be challenging (Fig. 10-7). The standard for assessing severity of AS is AV area, typically calculated using either a continuity method or by planimetry. Patients with low-gradient AS with severe LV dysfunction who received an AVR had improved survival and functional status compared with patients who did not have a valve replacement.[13]

A low pressure gradient related to LV dysfunction may not open the AV to its maximum capacity. Dobutamine challenge in a patient with low pressure gradient AS can be useful in establishing true AV area. The ability to distinguish between true AV stenosis and a state of "pseudostenosis" relies on characteristic changes in hemodynamic and structural measurements in response to the augmented CO. The test is not typically performed in the operative setting but rather as a preoperative evaluation. The increase in calculated AV area is related to the increase in the CO and is attributed to partial reversal of primary cardiac dysfunction. If dobutamine improves CO and increases AV area, it is likely the baseline calculations overestimated the severity of the AS. The dobutamine challenge is conducted as follows: patients with low-gradient AS receive intravenous dobutamine at 5 μg/kg/min with stepwise increases in dose. Patients may exhibit a significant increase in AV area (0.8 cm^2 to 1.1 cm^2) and a decline in valve resistance after dobutamine challenge. Patients with fixed, high-grade AS would demonstrate no change in valve area and an increase in valve resistance. The 2003 ACC/AHA/ASE Task Force gave a class IIb recommendation (usefulness/efficacy is less well established by evidence/opinion) for the use of dobutamine echocardiography in the evaluation of patients with low-gradient AS and ventricular dysfunction.[14] In addition to its role in distinguishing between true stenosis and pseudostenosis, low-dose dobutamine echocardiography is helpful in risk stratifying of patients with severe true AS. Patients with augmented contractile function after dobutamine administration have an improved outcome after surgery.[15]

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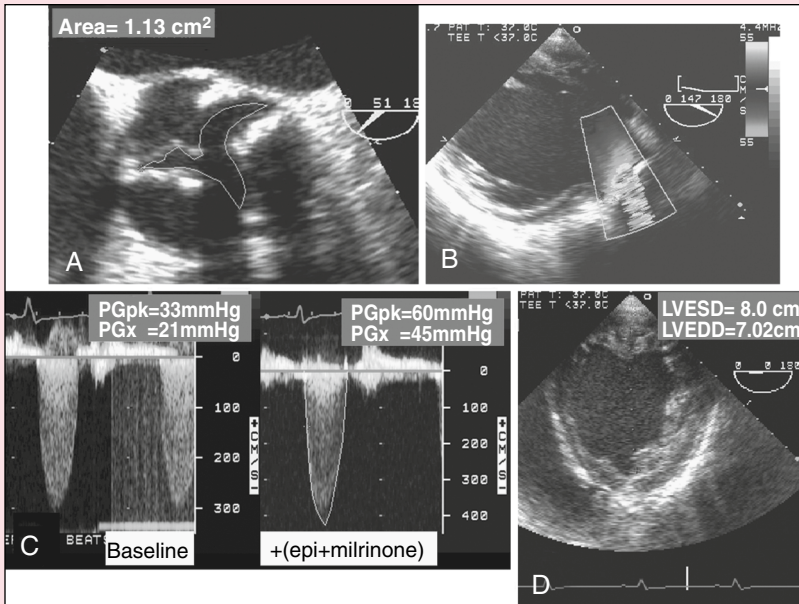
Figure 10-7 Low pressure gradient severe aortic stenosis. A 76-year-old cachetic man was scheduled to undergo corrective surgery for severe mitral regurgitation (MR) and possibly clinically significant aortic stenosis (AS). **A)** The midesophageal short-axis view of the aortic valve (AV) showed a highly calcified trileaflet valve with restricted mobility. The measurement of AV area, 1.13 cm^2, which was obtained by planimetry, was believed to underestimate the severity of AS because of the shadowing artifacts related to the severity of calcification. **B)** The transgastric long axis view of the left ventricle (LV) was obtained, and the velocity profiles of blood flow within the LV outflow tract and the AV were measured. **C)** Although the patient had a diagnosis of severe AS, the maximal and mean pressure gradients were 33 mm Hg and 21 mm Hg, respectively. The area of the AV was calculated to be 0.83 cm^2 using the continuity equation. **D)** The LV function was characterized by a severe dilated cardiomyopathy with an ejection fraction of 8%, LVESD of 7 cm, and LVEDD of 8 cm. The diagnosis of low pressure gradient AS was considered, and infusions of epinephrine and milrinone were started. Cardiac performance improved from 2.4 L/min to 4.5 L/min, and the pressured gradients increased to 60 mm Hg, peak, and 45 mm Hg, mean. Although the calculated valve area that was recorded under conditions of inotrope support slightly increased to 0.9 cm^2, transesophageal echocardiography (TEE) clarified that the marked increase in the pressure gradient was consistent with a diagnosis of low-gradient AS and confirmed the presence of cardiac reserve.

Case Study 4   ACUTE AORTIC SYNDROMES

FRAMING

The unstable patient with suspected acute aortic disease or injury is often the most challenging of TEE cases. There are few more crucially important decisions that are posed to the intraoperative echocardiographer than to quickly and accurately diagnose the nature and extent of acute aortic injury. Hypotension and respiratory distress may prevent a complete and comprehensive evaluation before surgery. Patient history is often unobtainable. The echocardiographer becomes a detective. Clues are quickly gathered from the available clinical presentation, past history, and associated physical findings. The TEE is often the only modality used to establish the diagnosis and define the surgical plan.

It is midnight on a gloomy rainy night. The hospital helicopter pilot calls in "young woman, unrestrained driver, deceleration injury, steering wheel impact, chest contusion, unconscious, hypotensive. She is intubated with bilateral breath sounds. Her

blood pressure is 70/40 mm Hg with an HR = 125 sinus tachycardia. She is being fluid resuscitated and being transported directly to the cardiac operating room." The patient is too unstable for magnetic resonance imaging (MRI) or computed tomography (CT). The patient arrives with a portable chest radiograph obtained as she traveled through the emergency department, showing a widened mediastinum. The vital signs have not changed except that she is receiving dopamine at 10 µg/kg/min. Pulses are palpable in the groins and the neck (Fig. 10-8). The attending surgeon turns to the echocardiographer-anesthesiologist and asks, "I need to know whether this is an anterior injury with heart contusion, injury to the ascending aorta, tamponade with blood in the pericardium, or a transected aorta or does the patient have a nonoperable injury? The former will require a sternotomy. The transections will require a left thoracotomy. If we make the incorrect decision, the patient will surely die." The patient is stabilized in the operating room and the TEE probe is inserted. After the diagnosis is made, the patient is positioned and prepped accordingly for the definitive surgery.

The sensitivity and specificity of TEE to detect and diagnose injury or disease of the thoracic aorta are significantly better than the sensitivity and specificity of TTE and are comparable to findings on CT and MRI.[16] TEE provides information regarding cardiac performance and the presence of other critically important sequelae that may be important in determining the approach and timing for surgical intervention. Hence, TEE is indicated even if MRI or CT has confirmed the diagnosis.

Can consent be obtained from the patient or family members? In these emergency circumstances, it may be more prudent to proceed with the TEE examination rather than delaying diagnosis and treatment in an attempt to find family members. What is the differential diagnosis of a widened mediastinum? How does TEE discriminate the different causes of a widened mediastinum? Is the TEE performed in the awake distressed patient, or is the TEE done under more controlled conditions of an anesthetized, intubated patient? Is there a risk of cervical spine injury? Is there a risk of esophageal injury? Can insertion of the TEE probe further compromise the patency of mediastinal structures? Is there fluid in the pericardium? What is the biventricular function? Is there myocardial rupture? Is there aortic rupture? Is the thoracic aorta intact? Is there an intimal flap and a dissection? Is there a transection? Is there a pleural or periaortic effusion/hematoma? What factors determine the urgency of intervention and strategies for management?

DATA COLLECTION

Because the diagnosis and cause for instability are not established, the entire mediastinum, including the left pleural space, is interrogated before definitive therapy is initiated. Rarely is there not enough time to do a complete TEE examination. The operative team can often proceed with confidence in the management of these critically ill patients with only TEE to guide the treatment. The primary event in aortic dissection is a tear and separation of the aortic intima. It is uncertain whether the inciting event is a primary rupture of the intima with secondary dissection of the media or hemorrhage within the media and subsequent rupture of the overlying intima. Systolic ejection forces blood into the aortic media through a tear that leads to the separation of the intima from the surrounding media, creating a false lumen. Blood flow may exist in both the false and true lumens through communicating fenestrations. Aortic dissections are classified by one of two anatomic schemes (the DeBakey and Stanford classifications). Transection is diagnosed through the detection of para-aortic hematoma near the isthmus and a "step-up" in the internal media wall.

DISCUSSION

Acute dissections (Stanford type A or DeBakey type I or type II) involving the ascending aorta or arch are considered acute surgical emergencies. In contrast, dissections confined to the descending aorta (distal to the left subclavian artery; Stanford type B or DeBakey type III) are treated medically unless the patient demonstrates proximal extension, hemorrhage, or malperfusion. From the International Registry of Acute Aortic Dissection (IRAD), 73% of the 384 patients with type B dissections were managed medically; in-hospital mortality was 10%.[17] The long-term survival rate after applying medical therapy is 60% to 80% at 4 to 5 years and 40% to 45% at 10 years.

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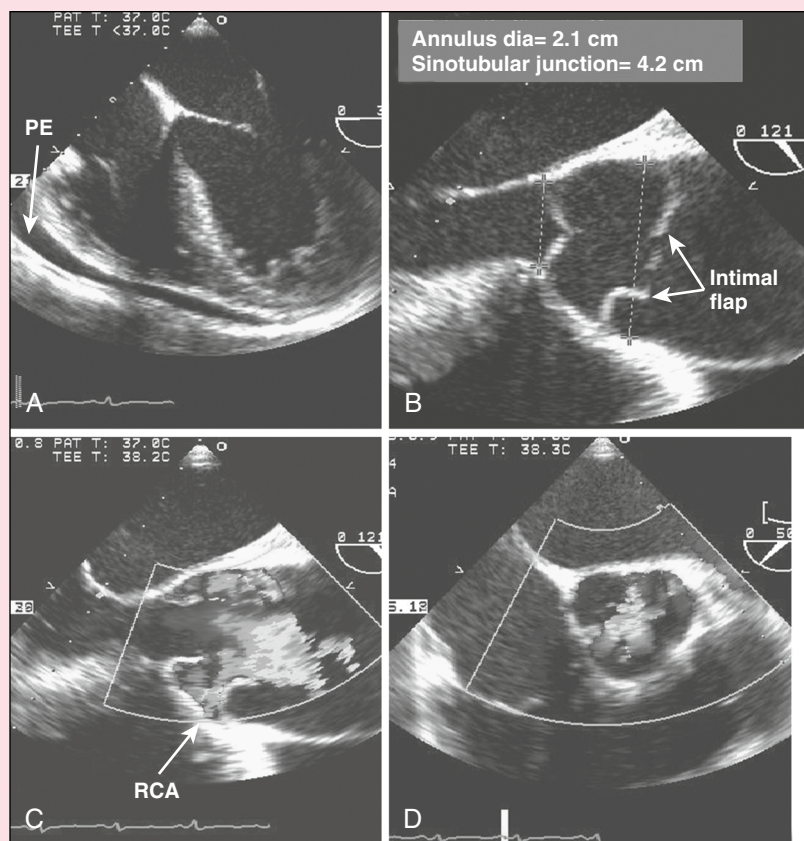
Figure 10-8 Acute aortic syndrome as the etiology of hemodynamic compromise. A 62-year-old previously healthy, unrestrained driver had a motor vehicle accident. On arrival to the emergency department, the patient was hypotensive (BP = 90/45) and tachycardic (HR = 120). He described an episode of loss of consciousness that was associated with severe chest pain but could not recall if the syncopal episode preceded the accident. The chest radiograph was significant for several fractured ribs, widened mediastinum, and a pleural effusion. The patient became progressively more unstable and was transferred to the operating room to perform diagnostic transesophageal echocardiography (TEE) and definitive surgical procedure if necessary. The echocardiographer performed a quick transthoracic echocardiographic examination that confirmed the presence of pericardial effusion with findings that were consistent for tamponade. After fluid resuscitation and induction of anesthesia, a TEE examination was performed. **A)** The midesophageal four-chamber view showed presence of a pericardial effusion (PE) that compromised right atrial filling. **B)** The mid-esophageal long-axis view of the aortic valve revealed a type A dissection that was characterized by intimal flaps within the aortic root and that extended distally into the descending thoracic aorta. The annulus of the aortic valve was of normal size, but the sinus and root were markedly enlarged (diameter of sinotubular junction = 4.22 cm). **C)** The dissection extended into the noncoronary and right coronary sinus segments, narrowing blood flow at the coronary ostia (*arrow*). Although the ECG did not show acute ischemia, the right ventricular function and inferior wall of the left ventricle were mildly hypokinetic. **D)** Although an effaced aortic root, ascending aortic aneurysm, and acute dissection in this age group are suggestive of congenital bicuspid valve, the short-axis view of the aortic valve showed a trileaflet valve with a coaptation defect with aortic insufficiency at the noncoronary cusp. The surgeon resuspended the aortic valve and replaced the ascending aorta and hemiarch with a tube graft. The valve repair was successful with only +1 aortic insufficiency and cardiac return to normal after surgery.

Survival is best in patients with noncommunicating and retrograde dissections. From the IRAD registry, in-hospital mortality for surgical patients was significantly higher (32%). The increased rate of mortality for surgically treated patients is likely influenced by selecting a cohort of patients with more advanced disease and complicated course (malperfusion, leakage, extension). The overall reported short- and long-term outcomes are similar for medically treated patients with type B dissections. Of 142 patients with type aortic dissections, there was a trend toward lower mortality with medical therapy compared with surgical treatment at 1 year (15% vs. 33%). Both groups had similar survival at 5 and 10 years (60% and 35%).

Ascending aortic dissections (involving the aortic root, ascending aorta, or arch) are acute surgical emergencies, because of the high risk for a life-threatening complication such as AR, cardiac tamponade, myocardial infarction, rupture, and stroke. The mortality rate is as high as 1% to 2% per hour early after symptom onset. Neither acute myocardial ischemia nor cerebral infarction should contraindicate urgent intervention. Although patients with stroke in progress may be at increased risk for hemorrhagic cerebral infarction due to intraoperative anticoagulation, leading to hemorrhagic stroke, the authors have seen several patients who experienced dramatic neurologic recovery. Operative mortality for ascending aortic dissections at experienced centers varies from 7% to 36%, well below the greater than 50% mortality with medical therapy.[18]

Traumatic aortic rupture is a life-threatening vascular injury that often results in lethal hemorrhage. In a multicenter trial of 274 patients, the overall mortality rate reached 31%, with 63% of deaths attributable to aortic rupture. Aortic transection and rupture usually occur at the aortic isthmus (between the left subclavian and the first intercostal arteries) and result from shear forces generated by unrestrained frontal collisions. Although aortography had been considered the gold standard for the diagnosis of transection, TEE and contrast-enhanced spiral CT and MRI are currently favored, especially for patients with renal insufficiency.[19] Intravascular ultrasonography has been proposed as a potential diagnostic tool for the identification of limited aortic injuries. Traumatic aortic rupture needs to be distinguished from an aortic dissection. Imaging of a dissected aorta typically reveals true and false lumens at multiple levels. The focal aortic injury of aortic transection is quite localized and may be overlooked when performing a cursory examination. A second potential diagnostic problem is that protuberant atherosclerotic changes of the aorta may be difficult to differentiate from partial aortic tears. The thick and irregular intraluminal flap, which corresponds to disruption of both intimal and medial aortic layers, can be imaged in both the short- and long-axis planes in the vicinity of the isthmus. In the longitudinal view, the medial flap is nearly perpendicular to the aortic wall because traumatic lesions are usually confined within a few centimeters distal to the left subclavian. The formation of a localized contained rupture of the false aneurysm is common. Color flow Doppler imaging and spectral Doppler imaging can be used to detect turbulence associated with nonlaminar flow at the aortic defect and the presence of a pressure gradient. Traditional treatment includes immediate surgical intervention using a right lateral decubitus approach and resection of the aorta with insertion of a tube graft. Deployment of endovascular stent grafts has been successful. Two series that included a total of 16 patients having aortic transection reported successful repair with no mortality or serious morbidity.[20] However, the application of this device under such conditions poses a high risk for left subclavian malperfusion and paraplegia. The decision regarding appropriate management and time course of therapy will depend on the technical availability and expertise within the institution and the forthcoming results of clinical trials that use newer, less invasive technologies.

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SUMMARY

- An ultrasound beam is a continuous or intermittent train of sound waves emitted by a transducer or wave generator that is composed of density or pressure. Ultrasound waves are characterized by their wavelength, frequency, and velocity.
- Waves interact with the medium in which they travel and with one another, and the manner in which waves interact with a medium is determined by its density and homogeneity. When a wave is propagated through an inhomogeneous medium, it is partly absorbed, partly reflected, and partly scattered.
- Doppler frequency shift analysis can be used to obtain blood flow velocity, direction, and acceleration of red blood cells, where the magnitude and direction of the frequency shift are related to the velocity and direction of the moving target.
- Doppler shifts above the Nyquist limit will create artifacts described as "aliasing" or "wraparound," and blood flow velocities will appear in a direction opposite to the conventional one. To optimize Nyquist limits, the ultrasound frequency should be low and the sampling frequency should be high.
- Normally, red blood cells scatter ultrasound waves weakly, resulting in a black appearance on ultrasonic examination. Contrast echocardiography uses gas microbubbles to present additional gas-liquid interfaces, which substantially increase the strength of the returning signal. This augmentation in signal strength may be used to better define endocardial borders, optimize Doppler envelope signals, and estimate myocardial perfusion.
- *Axial resolution* is the minimum separation between two interfaces located in a direction parallel to the ultrasound beam so that they can be imaged as two different interfaces. *Lateral resolution* is the minimum separation of two interfaces aligned along a direction perpendicular to the beam. *Elevational resolution* refers to the ability to determine differences in the thickness of the imaging plane.
- Absolute contraindications to transesophageal echocardiography in intubated patients include esophageal stricture, diverticula, tumor, recent suture lines, and known esophageal interruption. Relative contraindications include symptomatic hiatal hernia, esophagitis, coagulopathy, esophageal varices, and unexplained upper gastrointestinal bleeding.
- Horizontal imaging planes are obtained by moving the transesophageal echocardiography probe up and down (upper esophageal: 20 to 25 cm; midesophageal: 30 to 40 cm; transgastric: 40 to 45 cm; deep transgastric: 45 to 50 cm). Multiplane probes may further facilitate interrogation of complex anatomic structures by allowing up to 180 degrees of axial rotation of the imaging plane without manual probe manipulation.
- The dynamic assessment of ventricular function with echocardiography is based on derived indices of muscle contraction and relaxation. Echocardiography indices of left ventricular function that incorporate endocardial border outlines and Doppler techniques can be used to estimate cardiac output, stroke volume, ejection fraction, and parameters of ventricular relaxation and filling.
- Decision making by TEE requires "Framing" the problem, "Data collection", and a comprehensive TEE examination.

III

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