

Back to Basics: Cardiovascular Anatomy, Physiology, and Hemodynamic Monitoring for Anesthesia Technologists



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Author Note: *The author reports no relevant financial relationships, conflicts of interest, or commercial affiliations related to the content of this educational activity.*

ABSTRACT

A solid understanding of cardiovascular anatomy, physiology, and monitoring is essential for Certified Anesthesia Technologists. Perioperative care can markedly alter cardiovascular function via anesthetic agents, positive-pressure ventilation, surgical stimulation, fluid shifts, and vasoactive therapies. Anesthesia technologists play an essential role in preparing, assisting with placement, maintaining, and troubleshooting cardiovascular monitors, as well as recognizing abnormal values and waveforms that may signal patient instability.

This educational review reinforces core concepts of cardiac anatomy, physiology, and the electrical conduction system before progressing to common invasive and noninvasive hemodynamic monitors used in the perioperative environment. Emphasis is placed on normal values, clinical interpretation, indications for monitoring, basic treatment principles, and situations requiring heightened concern. Additional focus is given to pharmacologic support of hemodynamics and recognition of life-threatening ECG rhythms relevant to ACLS. The goal is to maintain clinical relevance and promote safe, informed practice.

Keywords: anesthesia technology, cardiovascular monitoring, hemodynamics, perioperative care, electrocardiography

LEARNING OBJECTIVES

Upon completion of this continuing education activity, the anesthesia technologist should be able to:

- Identify major components of cardiac anatomy and coronary circulation
- Describe fundamental cardiovascular physiology, including preload, afterload, contractility, and cardiac output
- Explain the cardiac electrical conduction system and basic ECG interpretation
- Recognize normal values and the clinical significance of common hemodynamic parameters

- Describe indications, basic insertion concepts, and waveform interpretation for invasive cardiovascular monitors
- Identify abnormal monitoring findings and critical ECG rhythms that warrant prompt communication with the anesthesia provider

INTRODUCTION

Cardiovascular monitoring is foundational to perioperative anesthesia care. Anesthesia technologists play an essential role in monitor setup, troubleshooting, waveform recognition, and communication of critical findings. A strong understanding of cardiovascular physiology enhances patient safety and supports effective interdisciplinary collaboration.

Cardiac Anatomy: Structural Foundations

The heart is a four-chambered muscular organ located in the mediastinum. Its primary function is to generate forward blood flow to support tissue oxygenation and metabolic demand. The right heart pumps deoxygenated blood to the lungs, while the left heart pumps oxygenated blood to the systemic circulation (Miller et al., 2020).

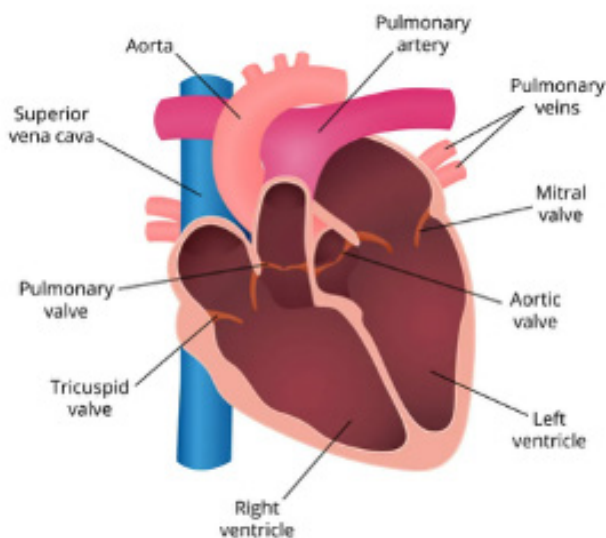


Figure 1: Heart Anatomy

Note. Adapted from *Anatomy and Physiology* (2023), by OpenStax, Rice University. CC BY 4.0. openstax.org

Chambers and Valves

The heart's four chambers and valves coordinate unidirectional blood flow and pressure generation essential for perfusion (Miller et al., 2020).

Chambers

- **Right atrium (RA):** Receives deoxygenated blood from the superior and inferior vena cava and the coronary sinus
- **Right ventricle (RV):** Pumps blood through the pulmonic valve into the pulmonary circulation
- **Left atrium (LA):** Receives oxygenated blood from the pulmonary veins
- **Left ventricle (LV):** Pumps oxygenated blood through the aortic valve into systemic circulation; its thick muscular wall generates the high pressures required for systemic perfusion

Atrioventricular Valves

- **Tricuspid valve:** Between the RA and RV
- **Mitral (bicuspid) valve:** Between the LA and LV
- **Semilunar Valves**
 - **Pulmonic valve:** Between the RV and the pulmonary artery
 - **Aortic valve:** Between the LV and the aorta

Clinical Note

Valves ensure one-way flow. Valvular dysfunction (stenosis or regurgitation) alters pressure gradients and chamber volumes, which can affect waveform interpretation and cardiac output. For example, severe aortic stenosis produces high LV pressures with relatively low systemic pressures, while mitral regurgitation can elevate left atrial and pulmonary pressures and blunt the reliability of wedge pressure as a preload estimate (Miller et al., 2020).

Coronary Circulation

The right and left coronary arteries originate from the aortic root just above the aortic valve and supply the myocardium. In the left coronary system, most myocardial perfusion occurs during diastole because systolic contraction compresses intramyocardial vessels. As a result, aortic diastolic pressure (and thus aortic root pressure) is a key determinant of myocardial oxygen delivery. Hypotension, tachycardia (shortened diastole), and elevated LV end-diastolic pressure can each reduce coronary perfusion and increase the risk of ischemia (Miller et al., 2020).

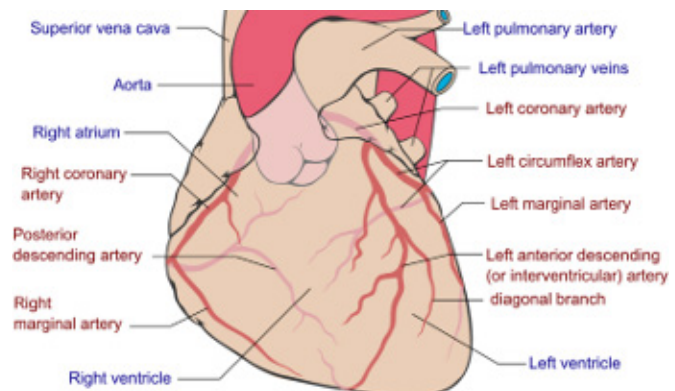


Figure 2 : Coronary Circulation

Note. Adapted from *Medical Terminology: An Open Educational Resource* (2020), by eCampusOntario Pressbooks. CC BY. ecampusontario.pressbooks.pub

CARDIAC PHYSIOLOGY: PRESSURE, FLOW, AND PERFORMANCE

Cardiac Output and Related Indices (Miller et al., 2020; Marino, 2024)

- **Cardiac output (CO):** Volume of blood pumped by the heart per minute.

$$CO = HR \times SV.$$

- **Cardiac index (CI):** CO adjusted for body surface area (BSA).
 $CI = CO/BSA.$

- **Stroke volume (SV):** Volume of blood ejected from the ventricle per beat.

$$SV = CO/HR.$$

Table 1: Normal Hemodynamic Values

Parameter	Normal Adult Value
Cardiac output (CO)	4–8 L/min
Cardiac index (CI)	2.5–4.0 L/min/m ²
Stroke volume (SV)	60–100 mL/beat

Note. Adapted from Miller's anesthesia (9th ed.; 2020), by Miller et al., Elsevier.

Clinical Pearl for Technologists

- High MAP with low CO → likely high SVR
- Low MAP with high CO → likely vasodilation
- Rising CVP with hypotension and tachycardia → consider RV failure or tamponade
- Narrow pulse pressure → low stroke volume
- Wide pulse pressure → low SVR or high stroke volume

Stroke volume is influenced by three primary determinants: preload, afterload, and contractility (Miller et al., 2020).

Preload

- **Definition:** Ventricular end-diastolic volume or pressure—how much the ventricle is filled before contraction
- **Clinical surrogates:** Clinical surrogates for preload include right atrial pressure or central venous pressure (CVP) for estimation of right ventricular (RV) preload and pulmonary artery occlusion pressure (PAOP, or wedge pressure) for estimation of left ventricular (LV) preload. These measurements are most reliable when significant mitral valve disease, pulmonary vascular disease, or elevated intrathoracic pressure (e.g., high positive end-expiratory pressure [PEEP]) are not present.
- **Relevance:** Within normal physiologic limits, increasing

preload typically increases stroke volume through the Frank–Starling mechanism, meaning the heart pumps more effectively when it is filled with an appropriate amount of blood before contraction. However, excessive preload can overwhelm the heart's ability to compensate, leading to pulmonary congestion, worsening oxygenation, or right ventricular (RV) failure (Miller et al., 2020).

Afterload

- **Definition:** The resistance the ventricle must overcome to eject blood; for the LV, closely related to systemic vascular resistance (SVR) and aortic impedance.
- **Clinical surrogates:** Clinical surrogates for afterload include systemic vascular resistance (SVR), arterial blood pressure, and the presence of left ventricular (LV) outflow obstruction, all of which help estimate the resistance the ventricle must overcome to eject blood into the systemic circulation.
- **Relevance:** High afterload (e.g., severe hypertension, vasoconstriction) raises myocardial oxygen demand and can reduce stroke volume; low afterload (e.g., sepsis, vasodilatory shock) can cause hypotension despite normal or high cardiac output.

Contractility

- **Definition:** Intrinsic myocardial contractile strength independent of preload and afterload.
- **Relevance:** Depressed contractility (e.g., cardiomyopathy, ischemia, some anesthetics) reduces stroke volume and cardiac output; inotropes can augment contractility when indicated (Miller et al., 2020).

Systemic and Pulmonary Vascular Resistance

- **Systemic vascular resistance (SVR):** Reflects left ventricular afterload in the systemic circulation (Marino, 2024).

$$SVR = (MAP - CVP) \times 80 / CO$$

- **Pulmonary vascular resistance (PVR):** Reflects right ventricular afterload in the pulmonary circulation (Marino, 2024).

$$PVR = (MPAP - PAOP) \times 80 / CO$$

Table 2: Normal Systemic and Pulmonary Vascular Resistance Values

Parameter	Normal Adult Value
Systemic Vascular Resistance (SVR)	800–1200 dyn·s/cm ⁵
Pulmonary Vascular Resistance (PVR)	100–250 dyn·s/cm ⁵

Note. Adapted from Miller's Anesthesia (9th ed.), by Miller et al., 2020.

Elevated PVR (e.g., hypoxia, acidosis, pulmonary embolism) increases RV afterload and can precipitate RV failure—particularly during positive-pressure ventilation and in patients with pre-existing pulmonary hypertension (Marino, 2024).

ELECTRICAL CONDUCTION SYSTEM

The heart's electrical conduction system coordinates organized electrical activation and mechanical contraction of the myocardium. Precise timing of atrial and ventricular depolarization is essential for maintaining effective cardiac output and adequate systemic perfusion (Barash et al., 2023).

Components

- **Sinoatrial (SA) node:** Primary pacemaker located in the right atrium; intrinsic rate 60–100 bpm
- **Atrioventricular (AV) node:** Delays conduction from atria to ventricles, allowing ventricular filling before contraction
- **His–Purkinje system (bundle of His, right and left bundle branches, Purkinje fibers):** Rapid ventricular conduction producing near-simultaneous depolarization and efficient contraction

Clinical relevance

Disruptions of conduction can cause bradyarrhythmias, tachyarrhythmias, or varying degrees of heart block. On ECG, these present as changes in rate or rhythm, PR interval, QRS width, or ectopy. Significant conduction abnormalities can compromise cardiac output and blood pressure and warrant prompt recognition and communication with the anesthesia provider (Barash et al., 2023).

ELECTROCARDIOGRAPHY (ECG)

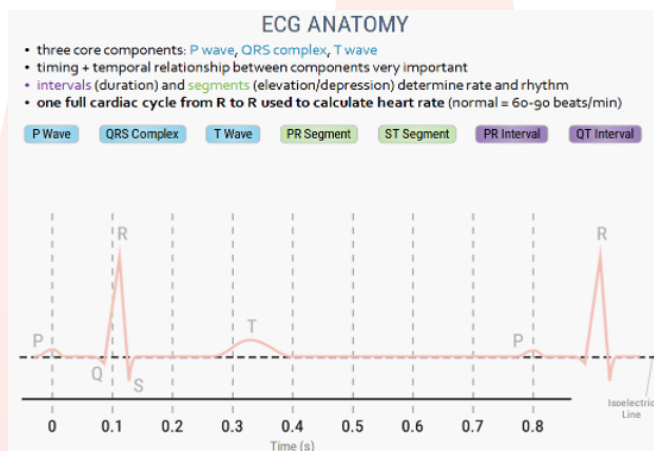


Figure 3: ECG Waveform

Note. Schematic ECG with labeled intervals. Adapted conceptually from *Conquering the ECG in Cardiology Explained*.

Purpose of ECG monitoring

- **Arrhythmia detection:** Identify bradycardias, tachycardias, and conduction blocks
- **Ischemia monitoring:** Detect ST-segment and T-wave changes that may indicate myocardial ischemia
- **Rate and rhythm:** Provide continuous assessment of heart rate and rhythm during anesthesia and recovery (Barash et al., 2023)

Basic waveform interpretation

- **P wave:** Atrial depolarization
- **PR interval:** Time from atrial depolarization to ventricular depolarization (AV conduction); normal 0.12–0.20 seconds
- **QRS complex:** Ventricular depolarization; normal duration <0.12 seconds
- **ST segment:** Early ventricular repolarization; deviations suggest ischemia or injury
- **T wave:** Ventricular repolarization (Barash et al., 2023)

When to Be Concerned

- **ST-segment depression or elevation** may suggest myocardial ischemia or infarction, especially when the changes are new, evolving, or accompanied by hemodynamic instability.
- **New-onset arrhythmias**, including atrial fibrillation with rapid ventricular response, ventricular tachycardia, or frequent premature ventricular contractions (PVCs), can significantly impair hemodynamic stability and cardiac output.
- **Prolonged PR intervals or widened QRS complexes** may indicate conduction system disease, medication effects, myocardial ischemia, or electrolyte abnormalities requiring prompt evaluation (Barash et al., 2023)

RECOGNITION OF CRITICAL ACLS RHYTHMS FOR TECHNOLOGISTS

While anesthesia technologists do not direct ACLS interventions, they must rapidly recognize life-threatening rhythms and alert the anesthesia provider or code team. Always verify ECG lead placement and signal quality before assuming a true arrhythmia—artifact from electrocautery, shivering, or loose electrodes can mimic ventricular arrhythmias (American Heart Association, 2020).

Shockable Rhythms

• Ventricular Fibrillation (VF)

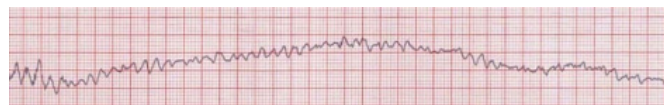


Figure 4: Ventricular fibrillation

Note. Adapted from "Ventricular Fibrillation (VF)," by E. Burns and R. Buttner, *Life in the Fast Lane ECG Library*, 2021. CC BY-NC-SA 4.0. litfl.com

- » **ECG Appearance:** Chaotic, irregular waveform with no identifiable P waves, QRS complexes, or T waves.
- » **Clinical Significance:** No effective cardiac output; the patient is pulseless and unresponsive. Requires immediate defibrillation and high-quality CPR.

• Pulseless Ventricular Tachycardia (VT)

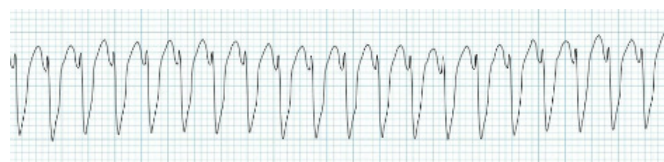


Figure 5: Ventricular tachycardia

Note. Adapted from "Ventricular Tachycardia—Monomorphic VT," by R. Buttner and E. Burns, *Life in the Fast Lane ECG Library*, 2021. CC BY-NC-SA 4.0. litfl.com

- » **ECG Appearance:** Wide-complex tachycardia, often regular, typically >150 bpm; may be monomorphic or polymorphic
- » **Clinical Significance:** If pulseless, treat as a shockable arrest rhythm with defibrillation and CPR

Nonshockable Rhythms

• Asystole

- » **ECG Appearance:** Near-flat line with no discernible electrical activity (confirm in more than one lead and check connections)
- » **Clinical Significance:** No cardiac output; treat with CPR and medications (e.g., epinephrine), not defibrillation (American Heart Association, 2020)

• Pulseless Electrical Activity (PEA)

- » **ECG appearance:** Organized electrical activity (e.g., sinus rhythm or bradycardia) without a palpable pulse
- » **Clinical Significance:** Pulseless electrical activity represents electromechanical dissociation, meaning organized electrical activity is present without effective mechanical contraction. Immediate high-quality CPR and rapid evaluation for reversible causes are required (American Heart Association, 2020).

Reversible Causes

Table 3: Reversible Causes of Cardiac Arrest

Hs	Ts
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade, cardiac
Hydrogen ion (acidosis)	Toxins
Hypo-/hyperkalemia	Thrombosis, pulmonary
Hypothermia	Thrombosis, coronary

Note. Didactic summary; content informed by American Heart Association (2020).

Technologists should be comfortable recognizing these rhythms on the monitor, verifying leads and signal quality, recalling the Hs and Ts, and immediately notifying the anesthesia provider when a life-threatening rhythm is suspected (American Heart Association, 2020).

BLOOD PRESSURE MONITORING

Noninvasive Blood Pressure (NIBP)

Noninvasive blood pressure (NIBP) is typically measured using oscillometric cuffs and provides systolic, diastolic, and mean arterial pressure (MAP) values (Vincent & De Backer, 2013; American Society of Anesthesiologists [ASA], 2023).

MAP Calculation (Approximate)

$$MAP \approx (2/3 \times DBP) + (1/3 \times SBP)$$

Normal MAP

In adults, a MAP of at least 65 mmHg is commonly targeted to maintain adequate organ perfusion, although individualized goals may vary depending on patient comorbidities, chronic hypertension, and surgical context.

NIBP monitoring is suitable for most low-risk procedures; however, it may be insufficient during rapidly changing or unstable hemodynamic states (American Society of Anesthesiologists [ASA], 2023).

Invasive Arterial Blood Pressure Monitoring (Arterial Line)

Arterial catheters, commonly referred to as arterial lines or "A-lines," are typically inserted into the radial, femoral, or brachial arteries to provide continuous blood pressure monitoring and facilitate frequent arterial blood gas sampling (Barash et al., 2023).

Indications

- Beat-to-beat blood pressure monitoring for hemodynamically unstable patients or those at risk for rapid physiologic changes related to fluid shifts or blood loss

- Frequent arterial blood gas sampling in ventilated or critically ill patients
- Continuous monitoring during vasoactive medication infusions requiring tight blood pressure control

Waveform and Setup Tips

- Level and zero at the phlebostatic axis (4th intercostal space, mid-axillary line)
- Recognize damping artifacts (overdamped/underdamped) and assess for air bubbles, clots, loose connections, or tubing kinks (Barash et al., 2023)

Waveform Components

- Systolic Upstroke
 - » Reflects rapid ejection of blood from the left ventricle
- Systolic Peak
 - » Represents the highest arterial pressure during systole
- Dicrotic Notch
 - » Represents closure of the aortic valve
- Diastolic Runoff
 - » Reflects the decline in arterial pressure during diastole as blood flows into the peripheral circulation

Overdamped or underdamped waveforms may distort systolic and diastolic pressure measurements. Anesthesia technologists should recognize abnormal waveform characteristics and assess the monitoring system for air bubbles, loose connections, blood clot formation, or tubing kinks (Barash et al., 2023).

Table 4: Common Arterial Line Waveform Abnormalities

Finding	Likely Cause
Flattened waveform	Air bubble, clot, or tubing kink
Exaggerated systolic peak	Underdamped monitoring system
Loss of dicrotic notch	Overdamped monitoring system
Abnormal SBP/DBP with preserved MAP	Damping artifact
Hypothermia	Thrombosis, coronary

Note. Common arterial waveform troubleshooting findings.

CENTRAL VENOUS PRESSURE (CVP)

Definition and Normal Values

Central venous pressure (CVP) reflects right atrial pressure and is influenced by venous return, intravascular volume

status, right ventricular (RV) function, and intrathoracic pressure (Marik & Cavallazzi, 2013). Normal CVP values typically range from 2 to 8 mmHg.

Clinical Use

CVP monitoring is commonly used as a rough estimate of right ventricular preload; however, it is an imperfect measurement and should always be interpreted within the broader clinical context, including ventilation mode, positive end-expiratory pressure (PEEP), intrathoracic pressure, and right ventricular function. Isolated CVP measurements have limited predictive value for fluid responsiveness and should not be used as the sole determinant of volume status (Marik & Cavallazzi, 2013).

Trends in CVP, when combined with clinical assessment and additional hemodynamic parameters, may assist providers in guiding fluid administration, vasopressor therapy, or diuresis.

Insertion and Technologist Responsibilities

Central venous catheters are commonly inserted through the internal jugular, subclavian, or femoral veins using sterile technique and ultrasound guidance when available (Miller et al., 2020).

Anesthesia technologists may assist with sterile field preparation, ultrasound setup, patient positioning, transducer setup, pressure tubing preparation, leveling and zeroing of the monitoring system, and verification of appropriate CVP waveform characteristics.

PULMONARY ARTERY (PA) CATHETER MONITORING

Pulmonary artery (PA) catheters, commonly referred to as Swan–Ganz catheters, provide detailed hemodynamic information but are used selectively because of their invasive nature (Marino, 2024). Although use has declined in some practice settings, pulmonary artery catheters remain valuable in select high-acuity patients requiring advanced cardiovascular assessment.

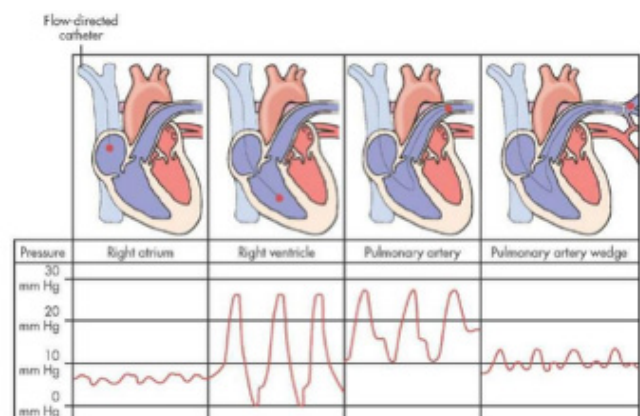


Figure 6: Pulmonary Artery Catheter Placement

Note. Adapted from *The ICU Book* (5th ed.), by Marino, 2024.

Pulmonary artery catheters provide several advanced hemodynamic measurements, including the following:

- **Right Atrial Pressure (central venous pressure [CVP])**
 - » **Normal CVP:** approximately 2–8 mmHg
 - » Reflects right atrial pressure and serves as an estimate of right ventricular preload and intravascular volume status
- **Pulmonary Artery Pressure (PAP)**
 - » **Normal Systolic PAP:** 15–30 mmHg
 - » **Normal Diastolic PAP:** 5–15 mmHg
- **Pulmonary Artery Occlusion Pressure (PAOP),** also referred to as pulmonary capillary wedge pressure (**PCWP**)
 - » **Normal PAOP:** approximately 5–12 mmHg

Wedge measurements are performed selectively because excessive balloon inflation or prolonged wedging may increase the risk of pulmonary artery rupture.

- **Cardiac Output (CO)**
 - » Commonly measured using thermodilution techniques
 - » Assesses overall cardiac performance and systemic blood flow

Indications

Pulmonary artery catheters may be used in patients requiring advanced hemodynamic assessment, including the following:

- Severe valvular heart disease
- Pulmonary hypertension
- Complex or unclear volume status
- Severe heart failure requiring vasoactive medication titration
- Major cardiac, vascular, or transplant procedures requiring detailed intraoperative and postoperative monitoring

Waveform Interpretation

During catheter advancement, characteristic waveforms help identify catheter location within the cardiovascular system.

- **Right Atrium (RA)**
 - » Low-pressure venous waveform
- **Right Ventricle (RV)**
 - » Elevated systolic pressure with low diastolic pressure
 - » No dicrotic notch present

- **Pulmonary Artery (PA)**
 - » Systolic pressure similar to RV pressure
 - » Higher diastolic pressure
 - » Presence of a dicrotic notch representing pulmonic valve closure
- **Pulmonary Artery Occlusion Pressure (PAOP/wedge)**
 - » Venous-like waveform reflecting left atrial pressure when the balloon is inflated and the catheter tip is wedged within the pulmonary artery

Anesthesia technologists should recognize expected waveform transitions, assist with pressure transducer setup and zeroing, help confirm appropriate catheter positioning, and promptly communicate unexpected findings such as arrhythmias during advancement, sudden increases in PAP, or loss of the wedge waveform.

EJECTION FRACTION (EF)

Definition

Ejection fraction (EF) represents the percentage of blood ejected from the ventricle during each cardiac contraction and is commonly used as an indicator of left ventricular systolic function (Miller et al., 2020).

$$EF = (SV \div EDV) \times 100$$

Normal left ventricular ejection fraction (LVEF) is generally considered to range from 50% to 70%.

Clinical Significance

Reduced ejection fraction indicates impaired systolic function and decreased cardiac reserve. Patients with reduced EF may be more susceptible to anesthetic-induced myocardial depression, hemodynamic instability, fluid shifts, and changes in afterload during the perioperative period. Severely reduced EF is associated with increased perioperative cardiovascular risk and may warrant invasive monitoring or advanced hemodynamic assessment in high-acuity surgical patients.

These patients often require closer hemodynamic monitoring, cautious titration of anesthetic agents, careful fluid management, and potential pharmacologic support with inotropes or vasopressors to maintain adequate cardiac output and end-organ perfusion.

PHARMACOLOGIC SUPPORT OF HEMODYNAMICS

Anesthesia technologists frequently encounter vasoactive, inotropic, and vasodilator medications used to support blood pressure, systemic vascular resistance (SVR), and cardiac

output during the perioperative period. Although medication selection and dosing are determined by anesthesia providers, technologists benefit from understanding the basic mechanisms of action and expected hemodynamic effects of commonly used cardiovascular medications (Barash et al., 2023; Miller et al., 2020).

Fluids

- **Crystalloids** (e.g., balanced electrolyte solutions, normal saline)
 - » Role: First-line therapy for hypovolemia and volume replacement
 - » Hemodynamic effect: Increase preload and stroke volume in preload-responsive patients
- **Colloids** (e.g., albumin)
 - » Role: Expand intravascular volume using smaller infused volumes
 - » Clinical considerations: Used selectively based on patient condition, institutional practice, and overall fluid management goals

Vasopressors

Vasopressors primarily increase systemic vascular resistance (SVR) and mean arterial pressure (MAP).

- **Phenylephrine**
 - » Mechanism: Pure alpha-1 (α_1) adrenergic agonist causing arterial and venous vasoconstriction
 - » Hemodynamic effect: Increases SVR and MAP; may reduce heart rate and cardiac output because of reflex bradycardia, particularly in preload-deficient patients
 - » Typical use: Treatment of anesthesia-induced vasodilation and hypotension in patients with relatively preserved cardiac function
- **Norepinephrine**
 - » Mechanism: Strong alpha-1 (α_1) adrenergic activity with moderate beta-1 (β_1) effects
 - » Hemodynamic effect: Increases SVR and MAP with modest increases in myocardial contractility and heart rate
 - » Typical use: Vasodilatory shock states such as sepsis or profound hypotension associated with low SVR
- **Vasopressin**
 - » Mechanism: Vasopressin-1 (V_1) receptor agonist producing vasoconstriction independent of adrenergic receptors

- » Hemodynamic effect: Increases SVR and MAP and may reduce catecholamine dose requirements
- » Typical use: Adjunct vasopressor therapy, particularly in catecholamine-resistant vasodilatory shock

Inotropes

Inotropes primarily increase myocardial contractility and may also increase heart rate.

- **Epinephrine**
 - » Mechanism: Adrenergic agonist with beta-1 (β_1), beta-2 (β_2), and alpha-1 (α_1) receptor activity; effects are dose dependent
 - » Hemodynamic effect: Increases myocardial contractility and heart rate and may increase SVR at higher doses; serum lactate levels may rise because of beta-adrenergic stimulation
 - » Typical use: Low-output states, anaphylaxis, and cardiac arrest according to Advanced Cardiovascular Life Support (ACLS) guidelines
- **Dobutamine**
 - » Mechanism: Predominantly beta-1 (β_1) adrenergic agonist with mild beta-2 (β_2) activity
 - » Hemodynamic effect: Increases myocardial contractility and cardiac output; may mildly decrease SVR because of vasodilation
 - » Typical use: Low cardiac output states associated with elevated afterload, particularly in heart failure
- **Milrinone**
 - » Mechanism: Phosphodiesterase-3 inhibitor that increases intracellular cyclic adenosine monophosphate (cAMP)
 - » Hemodynamic effect: Improves myocardial contractility while reducing SVR and pulmonary vascular resistance (PVR)
 - » Typical use: Right ventricular failure, pulmonary hypertension, or low-output states associated with elevated afterload

Vasodilators and Afterload/Preload Reducers

- **Nicardipine**
 - » Mechanism: Dihydropyridine calcium channel blocker producing predominantly arterial vasodilation
 - » Hemodynamic effect: Reduces SVR with minimal direct myocardial depression; reflex tachycardia may occur

- » Typical use: Perioperative hypertension and hypertensive emergencies requiring rapid intravenous (IV) titration
- Nitroglycerin (glyceryl trinitrate)
 - » Mechanism: Nitric oxide donor causing venodilation at low doses and mixed arterial and venous dilation at higher doses
 - » Hemodynamic effect: Reduces preload and myocardial oxygen demand and may improve coronary perfusion
 - » Typical use: Myocardial ischemia, acute coronary syndromes, and controlled preload reduction

Selected Antiarrhythmic and Rate-Control Medications

- Amiodarone
 - » Typical use: Ventricular tachycardia, ventricular fibrillation after defibrillation attempts, and selected supraventricular arrhythmias
- Lidocaine
 - » Typical use: Alternative therapy for ventricular arrhythmias in selected clinical situations
- Beta-blockers (e.g., esmolol, metoprolol, labetalol)
 - » Typical use: Rate control during tachyarrhythmias and treatment of hypertension or myocardial ischemia
- Clinical consideration: May reduce myocardial contractility and should be used cautiously in low-output states

Anesthesia technologists should recognize when vasoactive medications are being administered, anticipate expected hemodynamic effects on monitored parameters, and promptly communicate unexpected changes in blood pressure, heart rate, cardiac rhythm, or waveform characteristics to the anesthesia provider.

EMERGING AND ADVANCED HEMODYNAMIC MONITORING TECHNOLOGIES

Advances in perioperative monitoring have expanded beyond traditional static pressure measurements toward technologies emphasizing dynamic assessment and trending of cardiovascular performance.

Contemporary hemodynamic monitoring platforms incorporate pulse contour analysis and algorithm-assisted decision support to estimate parameters such as continuous cardiac output, stroke volume, stroke volume variation (SVV), and pulse pressure variation (PPV). These indices are most useful for assessing fluid responsiveness in mechanically ventilated patients under controlled conditions and are generally most reliable with controlled ventilation, regular rhythm, tidal volumes ≥ 8 mL/kg predicted body weight, a closed chest, and stable intra-abdominal pressure (Marik & Cavallazzi, 2013).

Although these systems may improve situational awareness and trend recognition, they do not replace foundational physiologic principles, conventional monitoring techniques, or clinical judgment. Accurate signal acquisition, proper calibration, awareness of device limitations, and interpretation within the broader clinical context remain essential responsibilities of the anesthesia technologist (Marik & Cavallazzi, 2013).

Why We Monitor and Basic Treatment Principles

Hemodynamic monitoring allows early recognition of physiologic instability and guides timely therapeutic intervention.

• Fluids for Hypovolemia

- » Purpose: Restore preload and improve stroke volume in volume-responsive patients

Table 5: Common Hemodynamic Medications and Effects

Drug	Primary Effect	Increases	Decreases	Typical Use
Phenylephrine	Vasoconstrictor	SVR, MAP	HR (reflex)	Anesthetic vasodilation
Norepinephrine	Vasoconstrictor + mild inotrope	SVR, MAP	—	Septic shock
Vasopressin	Non-adrenergic vasoconstrictor	SVR	—	Catecholamine-resistant hypotension
Dobutamine	Inotrope	CO	SVR (mild)	Low-output states
Milrinone	Inodilator	CO	SVR, PVR	RV failure
Nitroglycerin	Venodilator	—	Preload	Myocardial ischemia
Nicardipine	Arterial vasodilator	—	SVR	Hypertension

- **Vasopressors for Low SVR**

- » Purpose: Restore mean arterial pressure (MAP) and maintain organ perfusion during vasodilatory states

- **Inotropes for Impaired Contractility**

- » Purpose: Improve cardiac output in low-output states associated with myocardial dysfunction

- **Vasodilators for Excessive Afterload or Myocardial Ischemia**

- » Purpose: Reduce SVR, preload, or myocardial oxygen demand when hypertension or ischemia is present

- **Antiarrhythmics and Rate-Control Medications for Dysrhythmias**

- » Purpose: Restore organized rhythm and optimize ventricular filling and cardiac output

Anesthesia technologists support safe perioperative care by assisting with accurate monitor setup, calibration, leveling, and zeroing of pressure transducers; verifying waveform quality; and promptly communicating abnormal findings or concerning trends to the anesthesia provider.

Understanding the physiologic significance behind monitored values and waveform characteristics enhances interdisciplinary communication, improves clinical decision support, and contributes to patient safety (Vincent & De Backer, 2013). Technology enhances monitoring capability, but patient assessment and physiologic interpretation remain central to safe anesthesia care.

CONCLUSION

A strong understanding of cardiovascular anatomy, physiology, and hemodynamic monitoring is foundational to anesthesia technology practice. The role of the anesthesia technologist extends beyond equipment preparation and monitor setup to include recognition of abnormal waveform patterns, validation of monitoring accuracy, troubleshooting of monitoring systems, and prompt communication of concerning findings to the anesthesia provider.

Competency in recognizing hemodynamic instability and critical Advanced Cardiovascular Life Support (ACLS) rhythms contributes to early identification of cardiovascular compromise and supports timely perioperative intervention. As perioperative monitoring technologies continue to evolve, foundational physiologic knowledge remains essential for accurate interpretation and safe patient care.

Continuous reinforcement of cardiovascular and hemodynamic principles strengthens clinical judgment, enhances interdisciplinary communication, and contributes to improved perioperative patient safety and outcomes. 

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Continuing Education Quiz

PAGE 1 of 2

To test your knowledge on this issue's article, provide correct answers to the following questions on the form below. Follow the instructions carefully.

- 1. A mechanically ventilated patient undergoing major abdominal surgery develops hypotension (MAP 55 mmHg). Arterial waveform shows a narrow pulse pressure. Cardiac output measured by thermodilution is low. CVP is 3 mmHg. Which intervention best addresses the primary physiologic problem?**

 - A) Initiate phenylephrine infusion
 - B) Administer crystalloid bolus
 - C) Start dobutamine infusion
 - D) Administer nicardipine
- 2. During PA catheter advancement, the waveform transitions from low-pressure venous tracing to a waveform with systolic pressure ~25 mmHg and diastolic pressure near 0 mmHg without a dicrotic notch. Where is the catheter tip most likely located?**

 - A) Right atrium
 - B) Right ventricle
 - C) Pulmonary artery
 - D) Wedge position
- 3. A patient with severe aortic stenosis becomes hypotensive after induction of anesthesia. Which physiologic principle explains why aggressive vasodilation may be poorly tolerated?**

 - A) Reduced coronary perfusion due to decreased diastolic pressure
 - B) Increased preload from venodilation
 - C) Decreased pulmonary vascular resistance
 - D) Increased ejection fraction
- 4. A patient's arterial waveform appears flattened with loss of systolic peak exaggeration and diminished pulse pressure, but MAP remains unchanged. What is the most likely cause?**

 - A) Acute LV failure
 - B) Underdamped system
 - C) Overdamped monitoring system
 - D) Sudden decrease in SVR
- 5. A patient with pulmonary hypertension undergoes positive-pressure ventilation. Shortly after intubation, blood pressure drops and CVP rises significantly. Which mechanism best explains this change?**

 - A) Decreased LV afterload
 - B) Increased RV afterload leading to reduced LV preload
 - C) Increased coronary perfusion
 - D) Reflex tachycardia
- 6. An ECG shows wide-complex tachycardia at 170 bpm. The patient is unresponsive and pulseless. The anesthesia technologist should immediately recognize this rhythm as:**

 - A) Supraventricular tachycardia with aberrancy
 - B) Ventricular tachycardia requiring defibrillation
 - C) Atrial fibrillation
 - D) Sinus tachycardia
- 7. A patient with septic shock demonstrates MAP 58 mmHg, cardiac output 7.5 L/min, and SVR 500 dyn-s/cm⁵. Which medication most directly targets the underlying hemodynamic abnormality?**

 - A) Dobutamine
 - B) Milrinone
 - C) Phenylephrine or norepinephrine
 - D) Nitroglycerin
- 8. Coronary perfusion pressure is best approximated by:**

 - A) MAP – CVP
 - B) SBP – HR
 - C) Aortic diastolic pressure – LVEDP
 - D) CO – SVR
- 9. A mechanically ventilated patient has stroke volume variation (SVV) of 18%, low MAP, and low CVP. This pattern most strongly suggests:**

 - A) Vasodilatory shock without volume responsiveness
 - B) Fluid responsiveness and preload dependence
 - C) Cardiogenic shock
 - D) Tamponade physiology
- 10. An anesthesia technologist notices sudden ST-segment depression in leads II and V5 during surgery. Blood pressure remains within normal limits. What is the most appropriate immediate action?**

 - A) Silence alarm and observe trend
 - B) Increase IV fluids
 - C) Verify lead placement and promptly notify anesthesia provider
 - D) Administer amiodarone

Continuing Education Quiz

PAGE 2 of 2

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The answers to the Fall 2026 "Back to Basics: Cardiovascular Anatomy, Physiology, and Hemodynamic Monitoring for Anesthesia Technologists" Quiz 2 are: (circle answers)

- | | |
|------------|-------------|
| 1. A B C D | 6. A B C D |
| 2. A B C D | 7. A B C D |
| 3. A B C D | 8. A B C D |
| 4. A B C D | 9. A B C D |
| 5. A B C D | 10. A B C D |

Quiz 2

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