Cardiovascular Intensive Care Orientation Manual

VANDERBILT HEART

Cardiovascular Intensive Care Orientation Manual

WELCOME

Welcome to the CVICU at Vanderbilt University Medical Center. We are excited to have you join our team! Our CVICU is comprised of 27 beds that support our medical cardiology and cardiothoracic surgery teams. Our nurses care for the sickest patients in the region and manage multiple high acuity therapies including: various mechanical cardiac assist devices, continuous renal replacement therapy (CRRT), and ECMO. To accommodate this acuity, we maintain a nurse to patient ratio of 1:1 or 2:1. We are proud of the care that we provide our patients and look forward to equipping you with the skills necessary to provide the excellent care for which CVICU has become known.

After completing hospital orientation, you will join us for a 9-12 week orientation to CVICU, depending on your clinical background. Our expectation during this time is that you advocate for yourself and for your patients by asking thoughtful questions and utilizing the resources provided to you. During your first day on the unit, you will meet with the unit educator to review your orientation plan and materials. In addition to your precepted time on the unit, you will complete several classes and online learning modules, including three device-specific classes and a CVICU Boot Camp that will challenge you to apply the knowledge you have learned. Throughout your orientation you will meet with your Clinical Staff Leader (CSL) and Educator to track your orientation progress and answer any questions you may have.

After you successfully complete orientation, you will continue to directly report to your CSL. Our leadership team is committed to providing you with professional development and growth opportunities not only during orientation but also throughout your career at Vanderbilt. As you develop proficiency and confidence in your nursing practice in the CVICU, we look forward to helping you grow in your own leadership capabilities and work toward your specific career goals.

Sincerely,

The CVICU Leadership Team

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CONTENTS

The VANDERBILT CULTURE

CVICU Guidelines and Expectations

In this chapter, policies and employee expectations are reviewed. For a complete list of hospital policies, please refer to Policy Tech on the Vanderbilt Nursing homepage. To find more information on unit staffing and holiday policies, please refer to the CVICU website.

WHAT IS MAGNET?

Magnet is a highly coveted designation granted by the American Nurses Credentialing Center (ANCC) to hospitals that promote nursing excellence and quality patient care (American Nurses Credentialing Center, 2011). The ANCC (2011) recognizes 4 key strategies that promote exceptional outcomes: transformational leadership, structural empowerment, exemplary professional practice, and innovation. As part of our commitment to exceptional outcomes, the CVICU leadership team actively promotes professional development, shared governance, and diversity.

SHARED GOVERNANCE

The leadership team expects all nurses to participate in unit or hospital-based committees as part of their commitment to the CVICU. These committees represent an essential part of the shared governance structure at Vanderbilt and provide an opportunity for staff to give input on administrative decisions. While the scope of each committee may vary, all committees serve to support the continuous improvement of patient care delivery. A list of nursing-sensitive hospital committees can be found in the educator's office. Descriptions of CVICU committees and other opportunities to be involved can be found in the following sections.

Unit Board

The CVICU Unit Board provides a structure for collaborative decision making between CVICU staff and leadership. Agenda items may come from the staff, management team, physicians or other disciplines that serve the CVICU patient population. Unit Board is open to all staff and unit board decisions are made by consensus agreement. The CVICU Unit Board meets the first Tuesday of every month in the CVICU conference room. A complete copy of the Unit Board Charter can be found on the CVICU website.

Education Council

The CVICU Education Council provides a forum to identify educational needs of CVICU staff and share educational information. Education Council Co-Chairs assist with continuing education and act as a resource for CVICU staff and leadership. Education Council is open to all CVICU staff and management, and meets on the first Tuesday of every month. A complete copy of the Education Council Charter can be found on the CVICU website.

PIPS Champions

On first and third Tuesday of every month, CVICU PIPS champions assist members of the leadership team am and Wound Ostomy Care Nurse (WOCN)

team to identify patients at risk for pressure injury during the Pressure Injury Prevention Survey (PIPS). These nurses also serve as resources to staff and advocates for patients regarding pressure injury in the critically ill patient.

ATTENDANCE POLICY

All dayshift staff are expected to clock in between 0638 and 0645. Nightshift staff are expected to clock in between 1838 and 1845. The CVICU Kronos time clock must be used to clock in and out. If, for any reason, an employee is unable to work a scheduled shift, the employee must speak with the unit charge nurse before five o'clock on the designated shift. Employee time can be checked and approved by visiting the Kronos website, accessible through Quick Links on the Vanderbilt Nursing website.

Absence and Tardiness

An employee is considered absent when they are unavailable for their scheduled shift without prior approved time off. An employee is considered tardy if they clock in later than the approved times, leave work prior to the end of the assigned shift without prior approval, or fail to clock in at the designated time clock.

Occurrences

An occurrence is documented as an absence, tardy or missed time clock in/out. Leadership uses the grid in *Box 1.1* as a guideline when addressing occurrences. Occurrences are tracked on a rolling 12-month period, provided that the reason for an occurrence is not covered by FMLA.

SCHEDULING GUIDELINES

All full-time staff are required to work four weekend shifts per six-week schedule. Full-time staff are also required to work one weekend call shift and one weekday call shift per schedule. Staff may not work more than five consecutive shifts in direct patient care without manager approval.

Requesting Time Off

On dayshift, up to five staff members may be on va-cation per week. On nightshift, up to five staff mem-bers can be on vacation per week. Staff may request up to 14 days (six shifts) off at a time. PTO is granted by first request at a 6 month rolling time frame. During summer months, two weeks of PTO is permitted in non-consecutive weeks.

Staff may request preferred off (P-OFF) days when they would like to be off on a certain day without taking PTO. Preferred off requests will be consid-ered until 1600 on the Wednesday before the schedule process opens. The scheduling timeline is posted on the CSL office door. Staff may request up to six P-OFF days per scheduling period.

BOX 1.1	OCCURRENCE/ DAYS	DISCIPLINE AND ACTION
Occurrence 1 Occurrence is: * 1 Absence * 2 Tardies * 2 Missed Clocks	4 Occurrences 6 Occurrences 8 Occurrences 10 Occurrences	Verbal Warning Written Warning Final Warning Termination
Days Absent * Consecutive * Non–Consecutive	6 Days 9 Days 12 Days 15 Days	Verbal Warning Written Warning Final Warning Termination
No Call/ No Show	1 Occurrence 2 Occurrences 3 Occurrences	Written Warning Final Warning Termination

Scheduling Groups

There will be 3 scheduling groups (Group A, Group B and Group C). The groups will rotate who schedules first during the self scheduling window. The first group to sign up during the scheduling process will be the last group to be moved from their requests, the second group will be second to be moved and the third group the first to be moved.

Holiday Scheduling

All full-time staff are required to work two major and two minor holidays. Holiday schedules are determined by staff seniority. Major holidays (Thanksgiving, Christmas Eve, Christmas Day, New Years Eve, and New Years Day) and Minor holidays (Easter, Memorial Day, Fourth of July, and Labor Day) are considered. Staff are placed in A or B holiday groups, which rotate yearly. In addition to the actual holiday, staff will be assigned to work the day or days surrounding the scheduled holiday. Staff with greater than five years experience on days and greater than three years experience on nights may request PTO during winter holidays. Otherwise, vacations will not be granted during the week of a major or minor holiday.

Placed on Call

Staff may be placed on call due to low census or acuity. Nurses may request to be placed on call (POC) by submitting a first-off request in Vandy-works. First-off requests can be placed as early at 1700 on the Saturday before the week of the shift you are requesting. If a nurse places a first-off re-quest within 14 hours of the requested shift, they must verbally notify the charge nurse prior to that shift. If a nurse is granted a first-off request, then they will go to the bottom of the list for first-off all other days of the same week. Staff placed on call for low holiday census will not be eligible to take call for their next scheduled holiday unless they are called in within four hours.

Scheduled on Call

When optimally staffed, CVICU will have two nurses scheduled on call (OCN) at all times. On call nurses will be utilized after available float pool nurses have been assigned to the unit. If neither OCN has been called in during the current schedule, the least senior staff member will be called in first.

Floating to Other Units

CVICU nurses may float to other units depending on patient census or acuity. Nurses with less than six months seniority or working their on call shift will not be floated. Nurses who have not floated to other units will be floated first. If all scheduled nurses have floated, then the determination will be made according to last float dates.

STANDARDS OF CARE

The CVICU Standards of Care define the minimum amount nursing care that a patient receives while admitted to the CVICU at Vanderbilt. Nurses may give care that exceeds the practices outlined in the Standard of Care. Stepdown Standards of Care may be applied to patients with transfer orders. A complete copy of the standards of care can be found on the CVICU website and in Learning Exchange. The Standards of Care Quick Guide can be found in *Box 1.2.*

PRECEPTOR PEARLS

The Standards of Care should also guide how nurses chart the care they provide. Use the quick guide to make sure that all charting is completed accurately each shift.

BEDSIDE REPORT

Bedside report is expected to be completed during each shift handover. Bedside report should include the patient and/or family member and review: code status, fall risk, restraint use, pertinent history and a full system assessment. Nurses should use this opportunity to complete a visual inspection of all wounds, incisions, drains or other skin issues, including pressure ulcers. Orders should be reviewed with the off-going shift at this time. Visual inspection of all IV infusions, including confirmation of IV concentrations and rates should be reconciled with the orders prior to the off-going nurse leaving. The oncoming nurse is expected to trace all lines from the pump to the point of entry and ensure that all lines are labeled and within the expiration date.

ESCALATING ISSUES

Occasionally issues arise that must be addressed. It is the expectation of the leadership team that any patient safety or nursing care issue is addressed professionally with the involved staff member(s) at the time that the concern is noted. If the issue is not able to be resolved, then the staff member should escalate the concern to the CSL or RSL on duty. For provider teams, nurses may utilize the CVICU Urgent Needs Escalation Pathway (Figure 1.2) to guide the escalation of acute or trending changes that, if left unattended, could potentially result in patient harm.

Just Culture

Vanderbilt subscribes to the *Just Culture* philosophy to improve the working environment for staff and patient safety. *Just Culture* promotes professional accountability between the leadership team and front-line staff by improving system errors, utilizing mistakes as learning tools, and promoting a blame-free environment (Boysen, 2013). To this end, staff are encouraged to report or self-report any errors that occur so that the leadership team can identify any underlying process issues that may have contri-

BOX 1.2

STANDARDS OF CARE QUICK GUIDE

- COMPLETED Q SHIFT & PRN
  - Complete Assessment
  - RASS/CAM ICU scores
  - Fall Risk Assessment
  - Braden Skin Assessment
  - EKG Rhythm, Temporary Pacemaker Settings
  - Alarm Limits
  - Priority Problems
  - Plan of Care
- COMPLETED Q4 HOURS & PRN
  - Cardiac Output, Index and SVR on PA Cath patients
  - Transducers Zeroed
  - Temperature Assessment
  - Peripheral Pulse Checks
  - Mouth Care
- COMPLETED Q2 HOURS & PRN
  - Focused Re-assessment
  - IABP Unassisted numbers
  - Pain Assessment
  - Restraint Assessment
- COMPLETED Q1 HOUR & PRN
  - Vital Signs
  - Intake and Output
  - Device 'vital signs'

-uted to the error. Unless there is reason to suspect otherwise, the leadership team operates under the assumption that any medical errors are non-malicious in intent and that process improvement or education can prevent future errors of the same type.

Veritas

Veritas is Vanderbilt's incident reporting tool and can be accessed on any clinical work station. Veritas provides a system in which interdepartmental leadership teams can collaborate to find a solution to complaints or problems.

CODE ROLES AND RESPONSIBILITES

Codes in the CVICU should be organized with clear delegation of roles. If a primary nurse needs to code a patient, they should promptly press the code button and initiate chest compressions. When help arrives, the charge nurse is responsible for assuring that all code roles are accounted for. The following roles should each be assigned to separate nurses: scribing the documentation record, managing the crash cart, and pushing medications (Figure 1.1). In addition to these roles, at least two personnel should rotate chest compression every two minutes. Any staff without an official code role may be asked to step outside of the room to facilitate clear lines of communication during the code.

RECOMMENDATION LETTERS

The CVICU Leadership Team will write recommendation letters for graduate school after the employee has been in CVICU for two year and is not on any disciplinary performance plan.

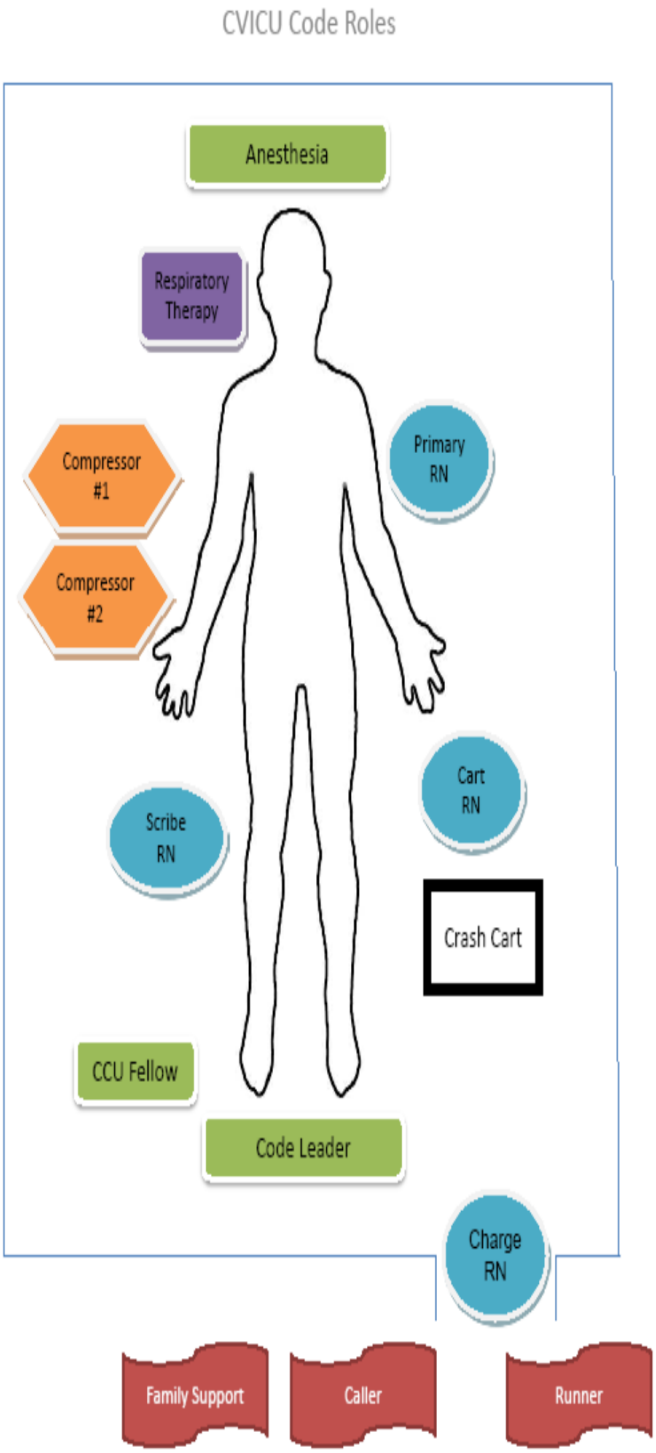

Figure 1.1

Each code should have a scribe, a nurse pulling drugs from the crash cart , and a nurse pushing medications in addition to two compressors.

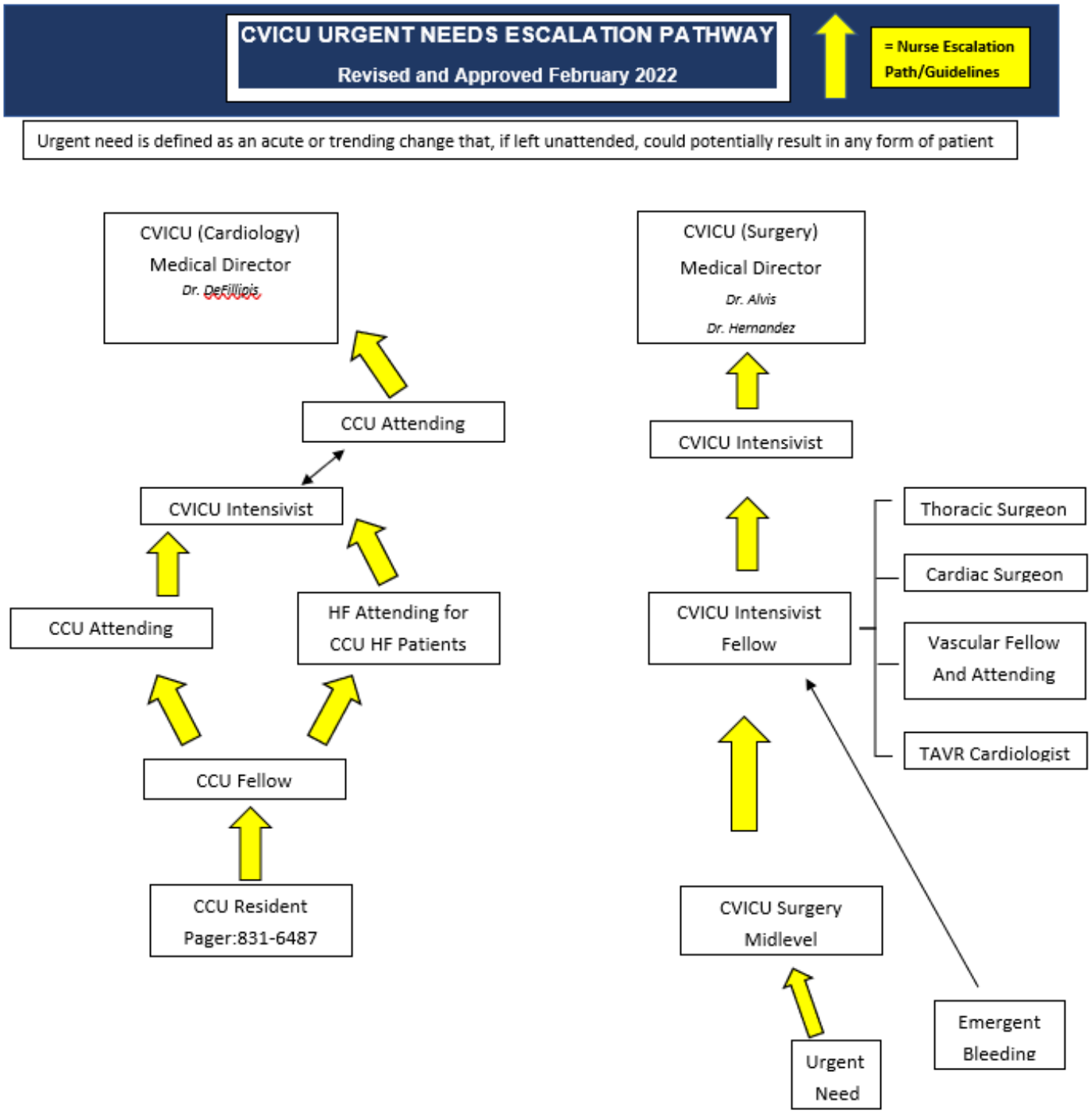

Figure 1.2

On the medical cardiology service the nurse should address issues with the resident followed by the fellow. If the issue is not resolved, notify the CCU attending, and, lastly, the CVICU Cardiology Medical Director. On the surgical service the nurse should escalate concerns through the nurse practitioner or physician assistant, followed by the intensivist fellow, intensivist and, finally, the CVICU Surgery Medical Director.

2 CRITICAL CARE *FOUNDATIONS*

Care of the Critically Ill Cardiac Patient

Nurses working in cardiovascular intensive care must have a proficient understanding of cardiac anatomy, physiology and hemodynamics. This chapter will review basic cardiac structures and physiology in addition to hemodynamic monitoring devices and parameters. Relevant pulmonary physiology, ventilator modalities, and ABGs will be reviewed.

CARDIAC STRUCTURAL ANATOMY

The normal human heart is a muscular organ that contains four chambers: two atria in the upper heart and two ventricles in the lower heart. The right atrium receives blood from the systemic circulation via the superior and inferior vena cava. The right atrium then contracts to propel the blood into the right ventricle. The right ventricle's subsequent contraction propels blood through the pulmonary arteries into the capillary-rich lung beds for oxygenation. On the left side of the heart, the left atrium receives oxygenated blood from the lungs and propels this blood into the left ventricle for systemic circulation via the aorta.

Valves

Four valves separate the chambers of the heart, two semilunar valves (aortic and pulmonic) and two atrioventricular valves (tricuspid and mitral). Appropriately named, the pulmonic valve separates the right ventricle from the lungs while the aortic valve separates the left ventricle from the aorta. Both the aortic and pulmonic semilunar valves are relatively small in diameter and receive a high velocity of blood ejecting from the left and right ventricle.

The atrioventricular valves open during diastole to allow filling from the atria to the ventricles and close during systole to prevent retrograde blood flow into the atria during ventricular ejection (Figure 2.2). The atrioventricular valves are held in position by the papillary muscles within both ventricles. Valve closure is responsible for the heart sounds heard on auscultation with S1 representing the closure of the atrioventricular valves and S2 representing the closure of the semilunar valves.

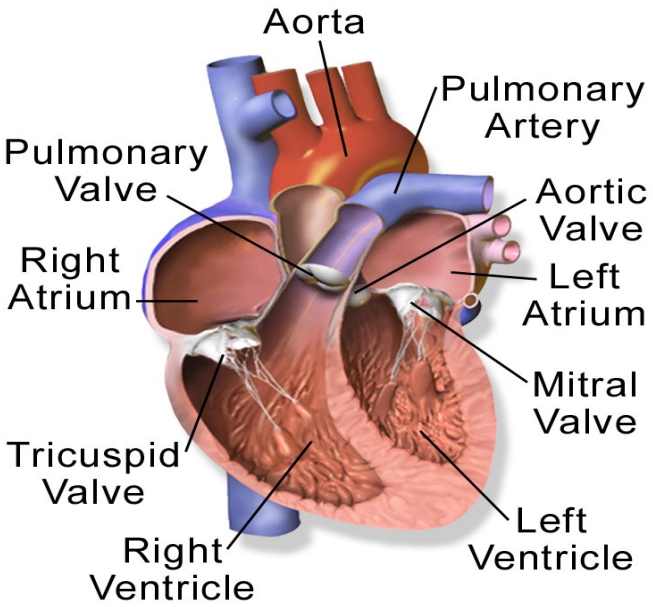

Figure 2.1 Normal Cardiac Anatomy. Image by Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2).

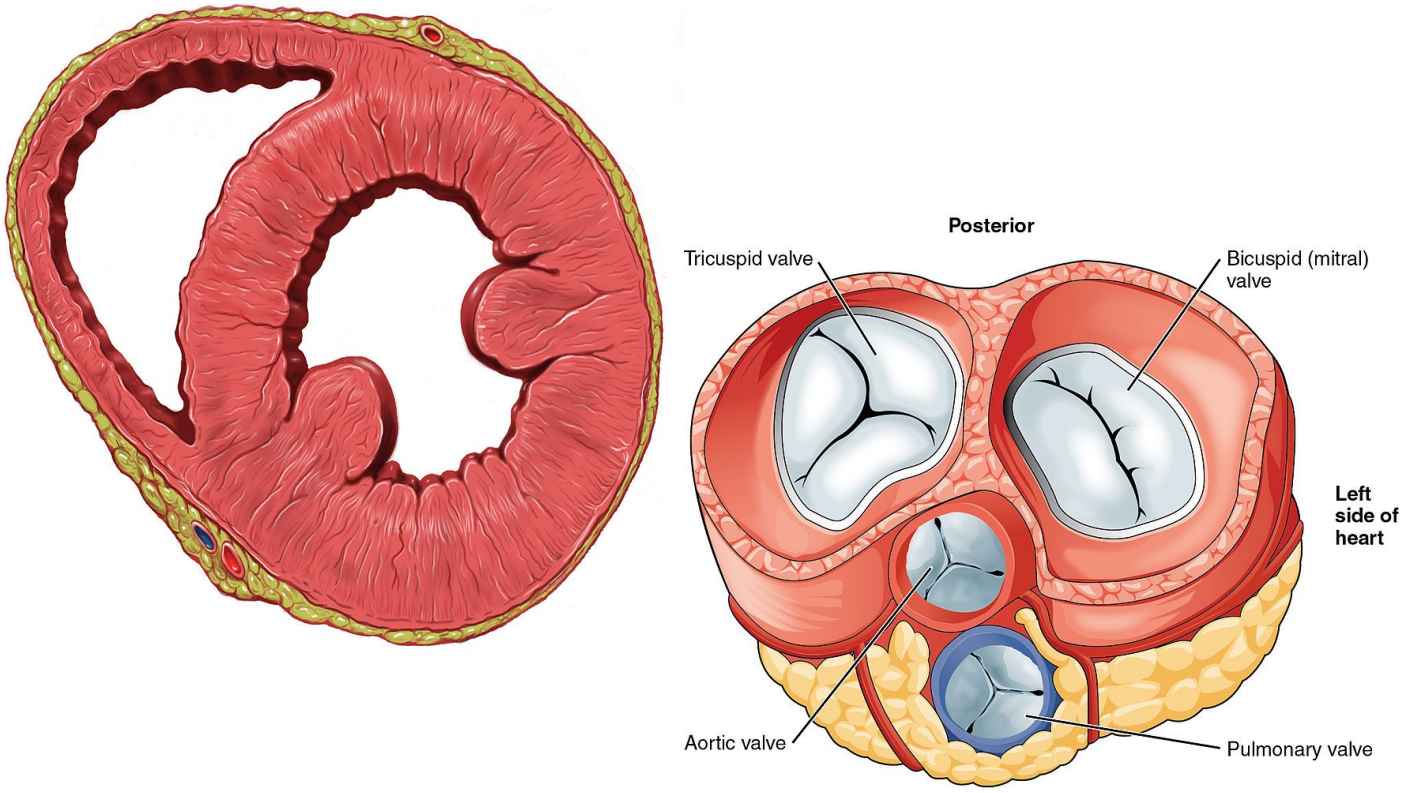

Figure 2.2 The Heart in Cross Section. Images by **OpenStax College - Anatomy & Physiology, Connexions Web site. http://cnx.org/content/col11496/1.6/, Jun 19, 2013., CC BY 3.0, https://commons.wikimedia.org/w/index.php?curid=30148207 and By Patrick J. Lynch, medical illustrator - Patrick J. Lynch, medical illustrator, CC BY 2.5, https://commons.wikimedia.org/w/index.php?curid=1490819**

Myocardium and Associated Structures

The muscle of the heart, or myocardium, is encased in the fibrous pericardial sac. This pericardial sac holds the heart in position in the thoracic cavity and secretes pericardial fluid to lubricate the movement of the heart in the chest. The ventricular myocardium is thicker on the left side of the heart, approximately 6-11 mm, to generate high pressures needed to circulate blood systemically. Conversely, the right ventricle is relatively thin-walled and measures only 2-4mm, generating blood flow to the low pressure of the pulmonary vasculature (Figure 2.2).

The Conduction System

Cardiac muscle cells have a resting potential of approximately –90mV across their cellular membrane (Sidebotham, McKee, Gillham & Levy, 2007). Specialized pacemaker myocytes spontaneously generate electrical impulses to depolarize this resting potential, diffusing ions across the cell membrane and forcing muscular contraction. The primary cluster of pacemaker myocytes are housed in the right atria, known as the Sinoatrial (SA) node.

PRECEPTOR PEARLS

Understanding valve position during the cardiac cycle can inform your clinical assessment. For example, a systolic murmur may indicate an incompetent atrioventricular valve or a stenotic aortic valve. Both create turbulent blood flow as the heart ejects blood in systole.

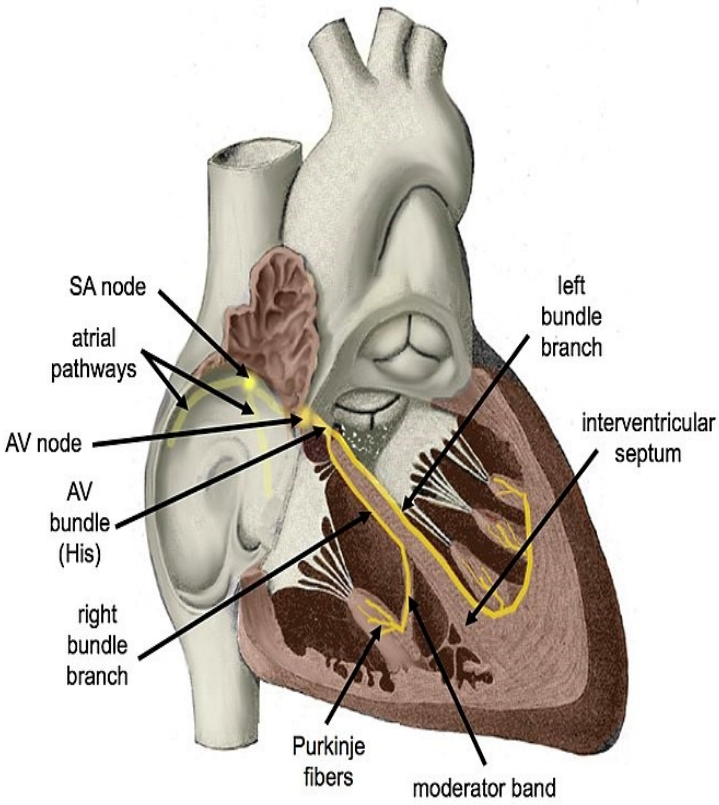

Figure 2.3 The Conduction System. Image by Cypressvine - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=80381713

Under normal circumstances, the SA node initiates and conducts this electricity through intermodal tracts in the atria to the AV node. The AV Node subsequently slows this conduction to allow the atria to contract, filling the ventricles with blood prior to ventricular depolarization. Finally, the electrical impulse passes quickly through the bundle of HIS, bifurcating into the bilateral bundle branches to allow for a swift, coordinated contraction of the bilateral ventricles. It is important to note that any disruption of blood supply to or the anatomic structures around the SA or AV node can cause conduction abnormalities. Further discussion regarding conduction abnormalities and their treatment can be found in Chapter 6.

Coronary Arteries

The coronary arteries provide oxygenated blood to the myocardium. Branching from the coronary ostia at the base of the aorta, each coronary artery branches to perfuse unique areas of the heart (Figure 2.4). Importantly, the Left Anterior Descending (LAD) artery supplies approximately 55% of the blood flow to the left ventricle (LV). For this reason, the LAD is commonly nicknamed "the widowmaker". This artery branches into the diagonals and septal perforators, also supplying oxygen to the ventricular septum. Approximately 20% of the LV perfusion is supplemented by the Left Circumflex artery and its branches . The Right Coronary artery supplies blood to the right ventricle (RV) and the remainder of the LV. Notably, the Right Coronary artery branches into the acute marginal, posterior descending and posterior lateral branches to provide oxygen to the inferior and posterior of the heart as well as the AV node.

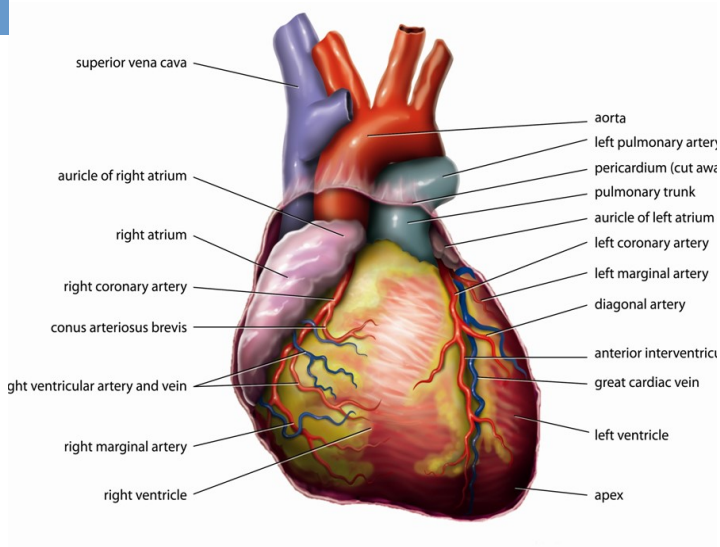

Figure 2.4 Coronary Arteries, Anterior View. Image by Tvanbr - Own work, Public Domain, https://commons.wikimedia.org/w/index.php?curid=11645241

Cardiac Physiology and the Cardiac Cycle

The cardiac cycle has two primary phases: systole and diastole. The closure of the atrioventricular valves, heard as S1 on auscultation, marks the onset of cardiac systole. Electrical conduction through the ventricle prompts ventricular contraction and increases the pressure within the ventricles, known as isovolumetric contraction. As this pressure overcomes aortic and pulmonary artery pressure, the aortic and pulmonary valves open against the favorable pressure gradient and allow for a rapid ejection of blood. Systolic blood pressure, therefore, is an indirect indicator of cardiac output.

Diastole, or ventricular filling, begins with the closure of the semilunar valves as the pressure in the ventricle falls below the resting aortic pressure. The closing of the semi lunar valves is heard as S2 on auscultation. Simultaneously, the low pressure of the ventricle in diastole allows the atrioventricular valves to open against a favorable gradient, allowing for rapid inflow of blood from the atria. Ventricular relaxation promotes blood flow into the coronary arteries, perfusing the myocardium during diastole. At a normal resting heart rate, diastole represents two-thirds of the cardiac cycle, promoting adequate oxygenation and resting of the mycocardium.

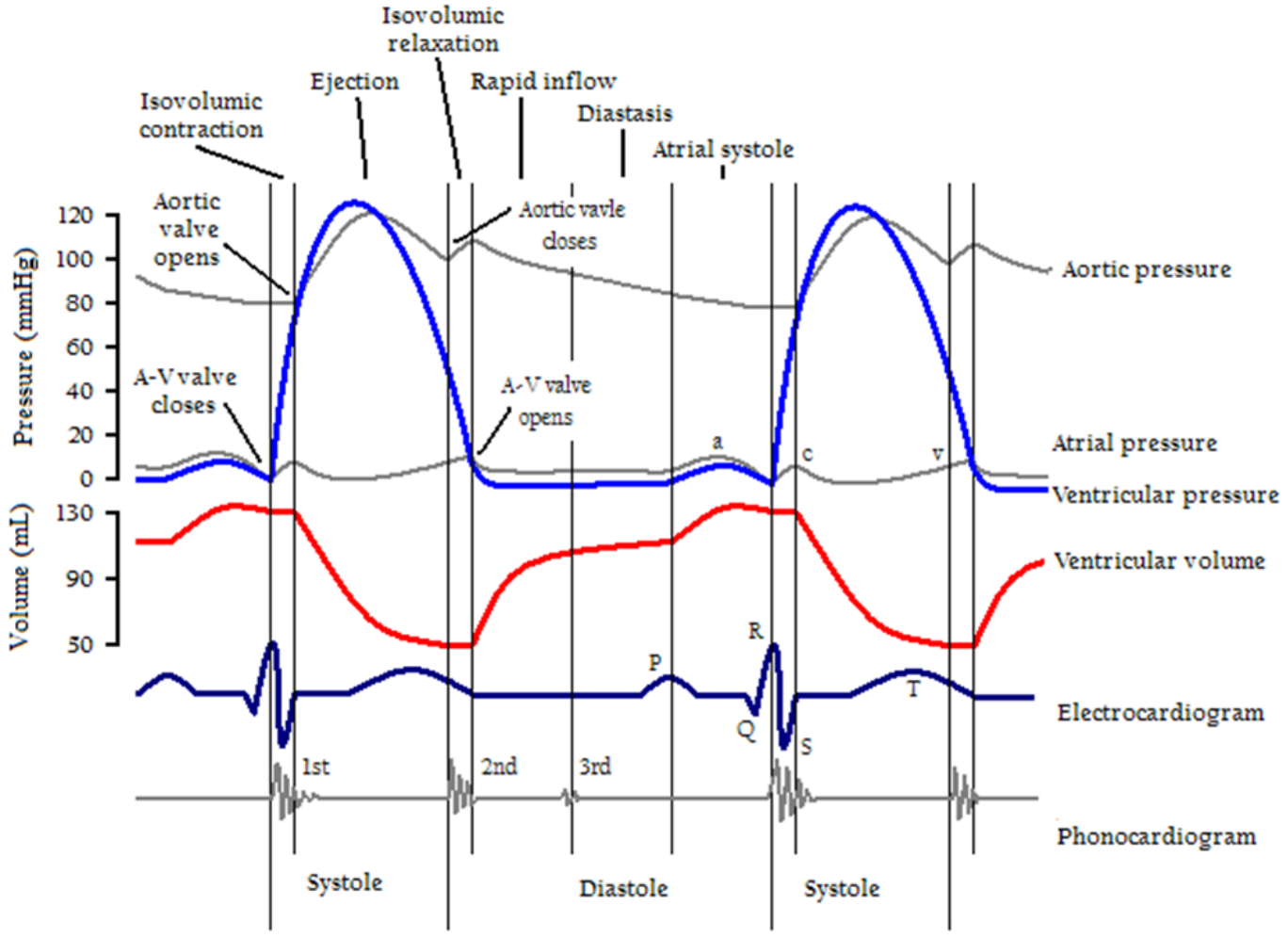

Figure 2.5 Wigger's Diagram. Public Domain.

Systole commences with the closure of the atrioventricular valves, heard as S1 on auscultation. Moving against a favorable pressure gradient, the aortic and pulmonary valves opens to allow for a rapid outflow of blood as the ventricles contract. Diastole commences with the closure of the aortic and pulmonary valves. Simultaneously, the atrioventricular valves reopen to allow for rapid ventricular filling.

Determinants of Adequate Cardiac Output

Cardiac output is the amount of blood that the heart is able to pump in one minute. Measured in liters/minute, cardiac output quantifies the health of the heart muscle and provides insight into other physiologic mechanisms contributing to the global patient picture. At the most basic level, cardiac output is determined by stroke volume and heart rate. Stroke volume is defined as the volume ejected with each heart beat (Sidebotham, McKee, Gillham & Levy, 2007). It is important to note, however, that many factors impact stroke volume, chief of which include the following three physiologic concepts: preload, afterload, and contractility.

Preload

Functionally, preload represents the volume of blood returning to the heart during diastole. Thus, preload is generally considered a measurement of end diastolic volume and is commonly referred to as filling pressure. Without adequate blood to fill the ventricles, the heart cannot generate an adequate stroke volume. Preload also indirectly impacts the contractility of the heart, as described by Starling's Law of the Heart. This law draws denotes that increased volume and ventricular stretch increases the force of contraction from the ventricles. However, the force of contraction does have limitations and particularly high ventricular volumes may decrease contractility of the heart. For this reason, it is clinically important to assess and optimize fluid status in the critically ill cardiac patient.

Afterload

Afterload represents the resistance against which the ventricles must eject and is also representative of ventricular wall stress. Most commonly, this resistance is secondary to systemic vascular changes and complex physiologic mechanisms. For example, a patient who is chronically hypertensive is in a chronic high afterload state. Conversely, many critically ill patients may decrease their afterload as a in stages of septic or anaphylactic shock. The diastolic blood pressure is considered to be an indirect measurement of systemic vascular resistance.

Contractility

Contractility represents the ability of the heart to contract, independent of preload and afterload. This is essentially a measure of the health of the myocardium. Unfortunately, there is no direct hemodynamic measure of contractility. However, echocardiography, patient history, and assessment of ejection fraction (EF) may inform the conclusion that the heart is not contracting effectively.

Hemodynamic Monitoring

Hemodynamic monitoring utilizes invasive lines to provide and array of data regarding the volume, pressure and flow of blood throughout the body to guide treatment decisions. However, the utility of these lines is largely based on the accuracy of the measurement. The following sections will review the types of hemodynamic monitoring provided in the CVICU with emphasis on nursing interventions to assure the accuracy of these measurements.

Arterial Blood Pressure Monitoring

Arterial lines are lines placed in the radial, brachial

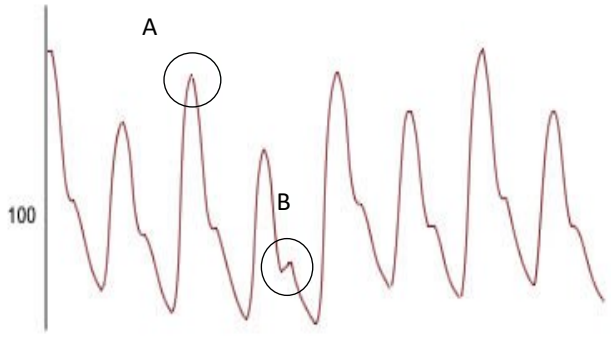

Figure 2.6 A Normal Arterial Waveform

or femoral arteries that transduce the mechanical pressure changes of systole and diastole into a pressure changes during the cardiac cycle to the Phillips monitor. Arterial lines are the gold standard of blood pressure monitoring, and provide continuous, real-time data. A normal arterial waveform should have a demonstrable systolic pressure reading (Figure 2.6, A) and a visible dicrotic notch (Figure 2.6, B), representing the closure of the aortic valve.

As with all IV lines transducing pressure, an arterial line must be transduced using non-compressible tubing attached to a transducer (Figure 2.7). To ensure accuracy, this transducer is leveled to the heart at the phlebostatic axis (5th intercostal space, mid-axillary line) and zeroed to atmospheric pressure.

Central Venous Pressure

Central Venous Pressure (CVP) can be measured by transducing an internal jugular (IJ) or subclavian line. This pressure reflects the pressure in the IJ or left atria and is largely a reflection of preload or volume status. It is important to note that, similar to an arterial line, the CVP waveform reflects mechanical changes in the vasculature. During atrial systole, the pressure increase in the atria reflects positive pressure toward the IJ catheter (Figure 2.8, A). tricuspid valve closes, a small notch can be seen (Figure 2.8, C). Finally, as the atria fills again during atrial diastole, another influx of pressure can be noted (Figure 2.8, V). Because the pressure transduced is from the venous vasculature, the pressure gradients between systole and diastole are much smaller. For this reason, a mean pressure gradient is considered accurate. Normal CVP ranges from 2-8 mm/Hg. As with all transduced pressures, accuracy is dependent upon the nurse leveling the transducer to the phlebostatic axis and zeroing the pressure to atmosphere.

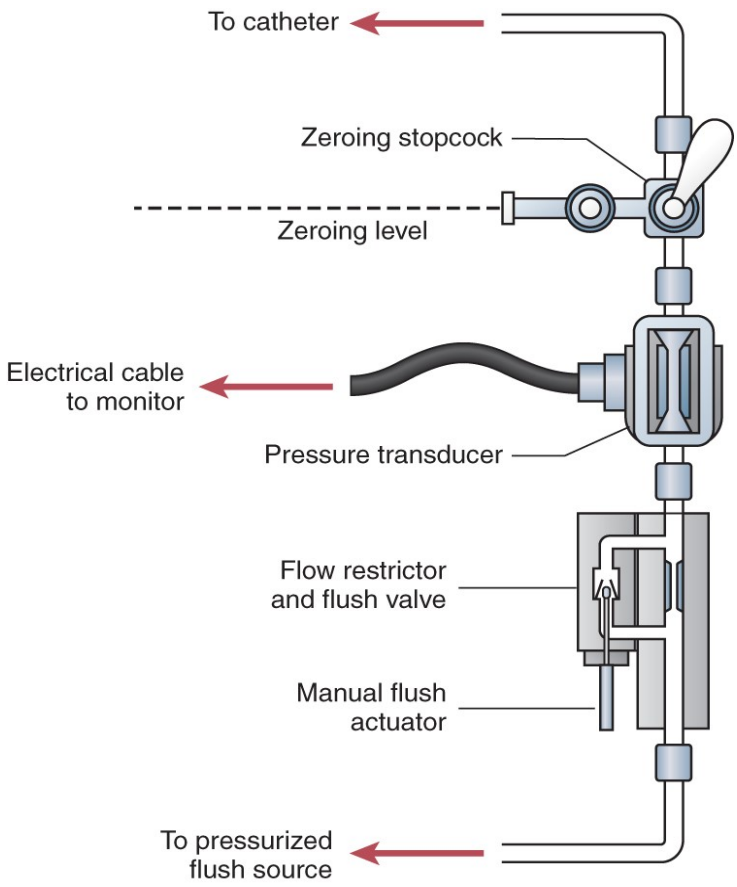

Figure 2.7 A schematic of a typical pressure transducer. From Kruse, J.A., Fink, M.P., Carlson, R.W. [Eds.]. [2003]. Saunders manual of critical care medicine. Philadelphia: Saunders. Used with permission (El Sevier, 2019)

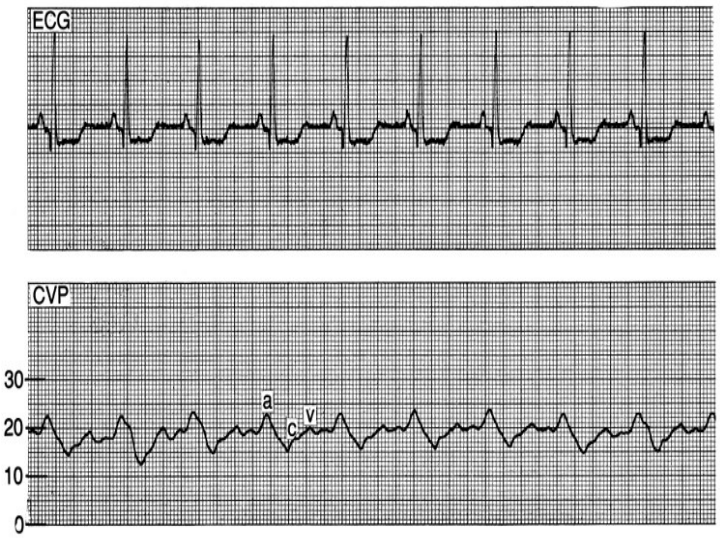

Figure 2.8 Central Venous Pressure Waveform. From Wiegand, D.L. [Ed.]. [2017]. AACN procedure manual for high acuity, progressive, and critical care [7th ed.]. St. Louis: Elsevier . Used with Permission (El Sevier, 2019)

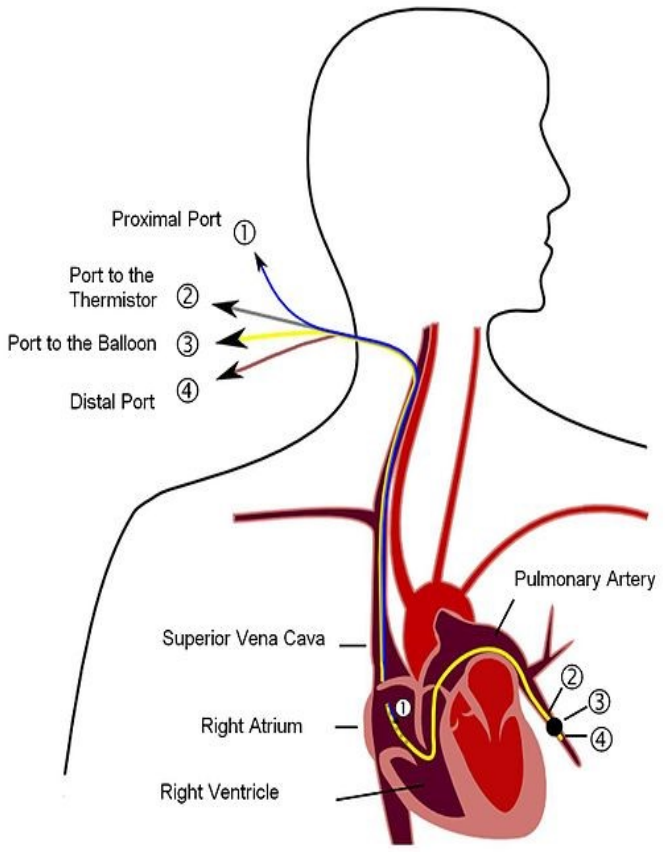

Figure 2.9 PA Catheter. Image by Chikumaya, Drawn with Inkscape 0.43 - Own work, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=817738

Pulmonary Artery Catheters

Pulmonary artery (PA) catheters are catheters inserted through the IJ and advanced so that the distal tip rests in the pulmonary artery. Various ports open along the trajectory of the PA catheter, allowing clinicians to transduce data from multiple places in the heart. PA catheters continuously transduce a CVP from the right atrium via a proximal port as well as pressures from the pulmonary artery via a distal port. Because the pulmonary arteries are in the pulmonary circulation, these pressures are much lower than the systemic pressures. Nonetheless, the pulmonary artery location is distal to the pulmonic valve, and pressures transduced within the pulmonary artery should exhibit an arterial waveform pattern with distinct systole, diastole and dicrotic notch. Normal PA systolic pressures should read 20-30 mmHg. Normal PA diastolic pressures should read 5 -10 mmHg. Elevated PA pressures may indicate essential pulmonary hypertension, embolus or other pathology increasing the vascular resistance in the lung beds.

PA catheters are "floated" into the pulmonary artery by inflating a balloon on the tip of the catheter that acts as a sail, pulling the catheter further into the heart. As the catheter is initially inserted, the nurse should notice a CVP waveform from the distal PA catheter port since the catheter is sitting within the right atrium (Figure 2.10, A). As the provider advances the catheter, the waveform will demonstrate a dynamic ventricular waveform (Figure 2.10 B). Finally, as the catheter advances past the pulmonic valve, the waveform assumes the characteristic arterial waveform and reflects the pressures of the pulmonary artery (Figure 2.10 C). On insertion, the provider will continue to advance the catheter until the balloon wedges in the pulmonary artery (Figure 2.10, D. Since there are no valves between the pulmonary artery and the left atrium, the wedge pressure is considered to be representative of left atrial filling pressures. Nurses should note that the wedge pressure assumes a waveform

PRECEPTOR PEARLS

It is important to assess the character of the PA waveform. If the PA catheter becomes wedged unintentionally, it could block blood flow within the heart and cause pulmonary infarction. A dampened or wedged waveforms from the PA catheter should be investigated immediately

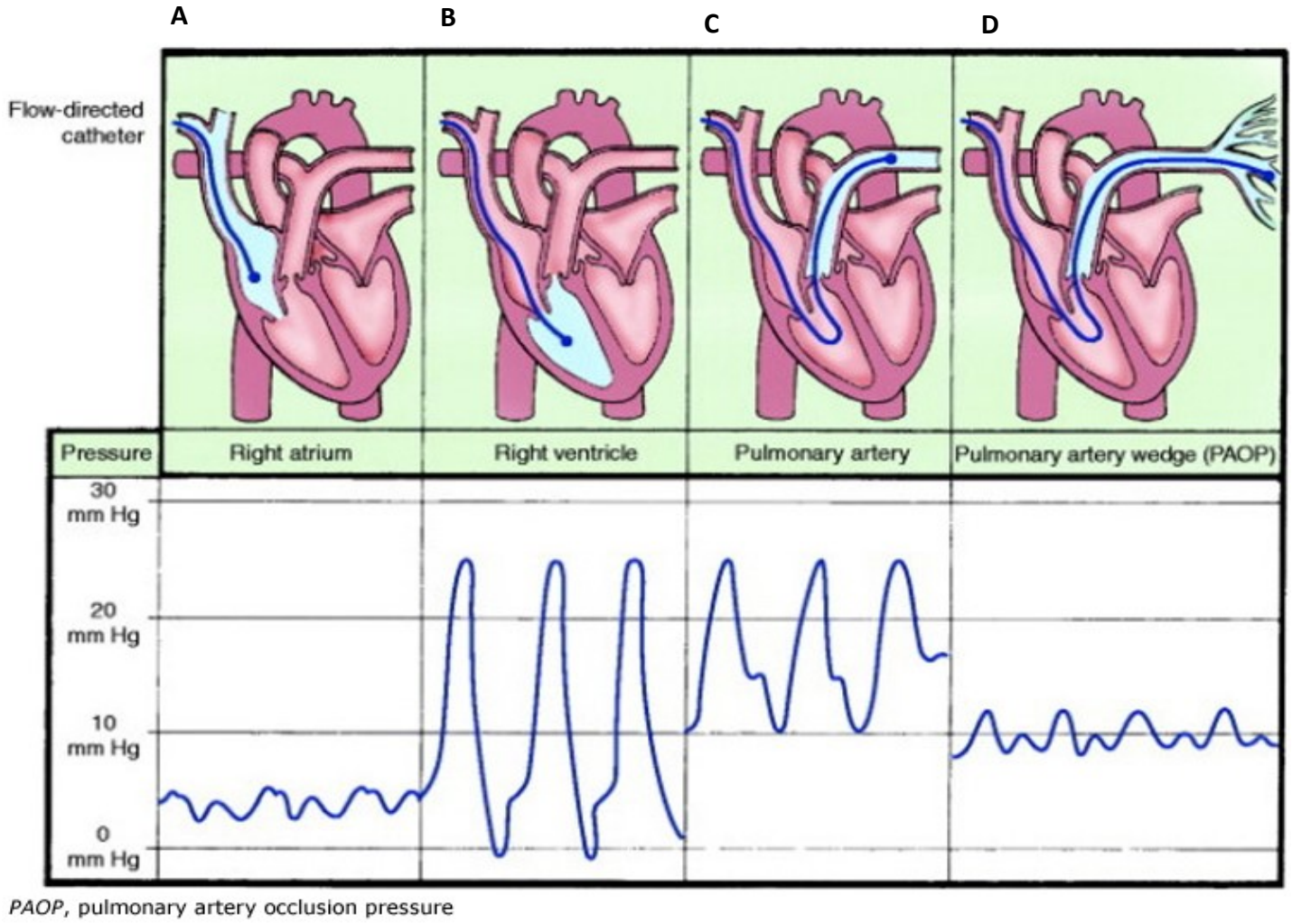

Figure 2.10 Normal Waveforms while floating a pulmonary artery catheter. From Urden, L.D., Stacy, K.M., Lough, M.E. [Eds.]. [2018]. Critical care nursing: Diagnosis and management [8th ed.]. Maryland Heights, MO: Elsevier.

similar to that of a CVP but with higher mean pressures. Average pulmonary capillary wedge pressure (PCWP) is 4-12 mmHg. Increased PCWP may indicate fluid overload, LV failure, mitral stenosis or mitral regurgitation. Low PCWP may indicate hypovolemia or the use of venodilators (Alspach, 2006). Wedge pressures are measured upon insertion and PRN by CCU fellows or cardiac surgery APRN

Cardiac Output and Index

PA catheters also allow for the direct measurement of cardiac output. While there are many methods to calculate cardiac output, CVICU most commonly uses a thermodilutional PA catheter. Using this method, 10 ml of room temperature normal saline is rapidly infused through the proximal (CVP) port of the catheter. The distal end of the catheter then measures the change in temperature, and uses this information to calculate the rate of blood flow through the heart in one minute. The average cardiac output ranges from 4-8 L/min, depending on body size and physiologic demand. To simplify the evaluation of this information, cardiac output can

be normalized for a patient's body surface area (BSA). This number, the cardiac index (CI), normally ranges from 2.5-4 L/min/m^2 . In the CVICU, a CI less than 2 L/min/m^2 is a reportable value to the provider team. A decrease in cardiac output is likely an indication of decreased preload (volume), increased afterload (resistance) or decreased contractility. Patients in moderate to severe septic shock may exhibit dramatic decreases in afterload that subsequently increase the total amount of cardiac output.

Systemic and Pulmonary Vascular Resistance

With the addition of a cardiac output, systemic and pulmonary vascular resistance can be calculated. Systemic vascular resistance (SVR) uses the mean arterial pressure (MAP), CVP, and cardiac output (CO) to estimate the total resistance to ejection during systole. Therefore, the SVR is a direct reflection of systemic afterload. Conversely, the pulmonary vascular resistance (PVR) can be calculated using the mean PA pressure, PCWP and cardiac output. This value uniquely reflects the afterload experienced by the right side of the heart during systole. Normal SVR ranges from 800-1400 dynes/sec/cm^{-5} and normal PVR ranges from 50-250 dynes/sec/cm^{-5}. Both SVR and PVR provide tremendous value in differentiating types of heart failure, types of shock and determining the course of treatment in mixed shock.

PRECEPTOR PEARLS

After PA catheter insertion, wedge pressures should only be obtained on medical cardiology patients with a physician order. Due to the risk of PA rupture and infrequency of use, our standards of care stipulate that only physicians or advanced practice providers wedge patients.

MECHANICAL VENTILATION

Patients may need to be mechanically ventilated for a variety of reasons, including but not limited to: inability to protect their airway, respiratory distress or arrest, sedation, or severe oxygenation issues. At Vanderbilt, the ICU fellow, attending or respiratory therapist (RT) are the only personnel allowed by policy to make ventilator changes. Nonetheless, nursing staff must maintain a basic knowledge of ventilator modalities, anticipate ventilator changes related to the patient's clinical picture, and troubleshoot common alarms.

In a healthy individual, respiratory rate and depth are determined by the autonomic nervous system. A negative intrathoracic pressure drives inhalation as the diaphragm lowers with each breath. In the mechanically ventilated patient, however, respiratory rate and depth care controlled manually. Respiration is instead driven by positive pressure via the ventilator. Ventilator modalities are named by the specific component of ventilation manipulated in each mode, classically volume control or pressure control modes. Ventilator parameters are reviewed in Box 2.1. Ventilator modes can be compared in Box 2.2.

Assist Control Ventilation

In assist control (AC) ventilation, a set number of breaths are delivered in a given amount of time. This modality can then be further specified as volume control (VC) or pressure control (PC). In volume control ventilation, the tidal volume of each respiration is set, but the pressure required to deliver this volume will vary with each breath. By contrast, pressure control ventilation sets the amount of positive pressure that will be used to deliver each breath, allowing the volume to vary. In both

modalities, respiratory rate, positive end expiratory pressure (PEEP), and amount of oxygen (FiO2) delivered are set. If the patient attempts to breath in between these breaths, the ventilator will assist the patient with these breaths to deliver the designated volume or pressure. This may unintentionally cause the patient to hyperventilate, and nurses should be careful to note the difference between the set number of ventilator breaths and the delivered number of ventilator breaths.

Synchronized Intermittent Mandatory Ventilation

In a synchronized intermittent mandatory ventilation (SIMV), the ventilator also delivers a set number of breaths during a given period of time using pressure or volume control. However, in this modality, the patient may breathe spontaneously between the ventilator-mandated breaths. Importantly, the ventilator will attempt to synchronize mandatory breaths with spontaneous patient effort, making this mode of ventilation more comfortable for the patient and decreasing the risk of hyperventilation (Parillo & Delinger, 2014). This mode of ventilation requires more patient effort than assist control ventilation. The Vanderbilt CVICU most commonly uses SIMV with a dual control mode of ventilation known as pressure-regulated volume control (PRVC). In PRVC, the volume for each respiration is set, but the pressure required to deliver this volume is limited.

Pressure Support

Pressure support (PS) mode of ventilation is considered to be a weaning mode of ventilation. In pressure support, the rate, tidal volume and inspiratory pressure are *not* set, requiring the patient to initiate their own breaths and maintain their own respiratory rate.

Alarms and Troubleshooting

Common ventilator alarms include: PAW high, High Respiratory Rate, and Regulation Pressure Limited. An airway pressure high (PAW High) alarm indicates increased airway pressure caused by blockage in the ventilator circuit. Common causes include: patient coughing or biting the endotracheal tube (ETT), patient needing to be suctioned, the Heat Moisture Exchanger (HME) needing to be changed, or crimped tubing. Nurses should assess the patient and the ventilator tubing to address this alarm, suctioning the patient as needed. If the HME appears saturated, nurses may change the HME on the ventilator circuit.

High respiratory rate alarms indicate the that respiratory rate has exceeded the alarm limit. This may be caused by pain or anxiety requiring increased sedation or by patient decompensation. Nurses should address the root cause of the increased respiratory rate as warranted by the patient picture. Nurses should also assess whether the respiratory rate alarm is appropriate for the patient and notify respiratory if the alarm limit should be reconsidered.

BOX 2.1

FiO2	Fraction of inspired oxygen. The amount of oxygen delivered to the patient.
PEEP	Positive End Expiratory Pressure. The amount of positive pressure continuously delivered to the patient to keep the alveoli open.
PIP	Peak Inspiratory Pressure. The maximum amount of pressure needed to inflate the desired volume of air. PEEP + PC settings = PIP
Respiratory Rate	The number of respirations in one minute.
Tidal Volume	The volume of air instilled with each respiration. Inspiratory volume should equal expiratory volume.
Minute Ventilation	Tidal volume x respiratory rate. The total amount of air ventilated in one minute. Measured in L/min.

BOX 2.2

Ventilator Modes

Mode	Definition	When Used	Key Clinical Points
SIMV **Synchronized Intermittent Mandatory Ventilation**	Delivers set number of breaths at a set volume per minute synchronized to go with patient's breathing pattern **Gives more control**	Most post-op patients are on this and PRVC, allows patient to do some of the work. Weaning mode.	Patient can take own breaths but the set tidal volume is not delivered with those breaths. Pressure support is used for those breaths
PRVC **Pressure Regulated Volume Control**	Control mode of ventilation with a set rate, set tidal volume, and set PEEP. The ventilator regulates how much pressure control needed by the set tidal volume. Does not aid spontaneous breaths.	Used in patients with less compliant lungs such as ARDs and post op patients. Combined with SIMV. Alternates modes. By itself, PRVC is not a weaning mode.	Control is taken back over by the ventilator, so patient cannot initiate own breaths as easily, thus can be anxiety producing mode.
CPAP and pressure support **Continuous Positive Airway Pressure**	Positive pressure exerted on lungs. Constant pressure through inspiratory and expiratory cycle. Pressure support added to help aid in keeping lungs open during inspiration	Used before extubation to see if patient can breath on own. Patient creates own tidal volume and rate.	Patient must be able to spontaneously breathe.
Volume Control **(also known as Assist control)**	Control ventilation with set rat and set tidal volume. Patient can initiate a breath and ventilator will give a breath	Used for patient with low spontaneous tidal volumes. Not weaning mode.	Can decrease cardiac output. Does not have pressure support.
Bi-Vent	Inverse expiratory and inspiratory rate. Prolongs inspiratory time with short expiratory time.	ARDS, decreased Sa02 and increased peak pressures. Unable to ventilate.	Very uncomfortable! Ensure adequate sedation.
VDR **Volumetric Diffusive Respirator**	High frequency ventilator. High rate with low tidal volumes. Oxygenation occurs through diffusion. Internal percussion.	ARDs, low Sa02 with increased PIP, unable to ventilate in other modes.	N/A

A regulation pressure limited alarm occurs in PRVC mode when the PIP becomes too high to allow the ventilator to deliver the set tidal volume. This may indicate that the patient's lung compliance is decompensating or that the patient is fighting the ventilator. Nurses should asses their patient to determine whether the patient appears to be in pain and ascertain whether increased sedation is warranted. If no discernable cause can be determined, the nurse should notify respiratory and the care team.

Ventilator Weaning

Spontaneous awakening trials (SATs) and spontaneous breathing trials (SBTs) should be conducted daily on ventilated patients in collaboration with the interdisciplinary care team. Prior to conducting an SAT, the nurse should complete the SAT safety screen (Box 2.3). To conduct an SAT, the nurse

should pause sedation and analgesia infusions until an adequate neurological exam can be obtained. If the patient is able to follow commands with sedation weaning, then the nurse should conduct an SBT safety screen and contact the care team for approval to begin an SBT. If the team approves to patient to participate in an SBT, the RT will place the ventilator into pressure support on minimal ventilator settings. However, if the patient experiences anxiety, agitation or pain that cannot be managed by prn medication, respiratory distress or any acute cardiac rhythm changes, the nurse should resume continuous sedation and contact the RT to place the patient back on full ventilator support. In the CVICU, it is our goal to extubate patients within six hours of cardiac surgery unless their clinical exam warrants otherwise. Once the patient achieves hemodynamic stability after surgery, the nurse should collaborate with the cardiac surgery team to determine when it will be appropriate to wean sedation for an SAT.

ARTERIAL BLOOD GAS MONITORING

Arterial blood gases (ABGs) measure the content of oxygen (PO2) and carbon dioxide (CO2) in dissolved in a patient's blood. Values generated through an ABG sample will offer insight into a patient's acid-base balance, determine the source of imbalances, and guide treatment changes in the critically ill patient. To maintain normal cellular metabolism, a narrow pH range of 7.35-7.45 must be maintained. A pH of less than 7.35 is considered acidotic while a pH greater than 7.45 is considered alkalotic. Under normal circumstances, the human body maintains homeostasis by regulating HCO3– through the kidneys and CO2 via the lungs (Figure 2.12). If the primary abnormality in the ABG is in the paCO2, then the disturbance is considered respiratory in nature. If the primary abnormality in the ABG is the HCO3-, then the disturbance is considered metabolic in nature. To interpret an ABG, the nurse should first determine whether the pH is normal, acidotic

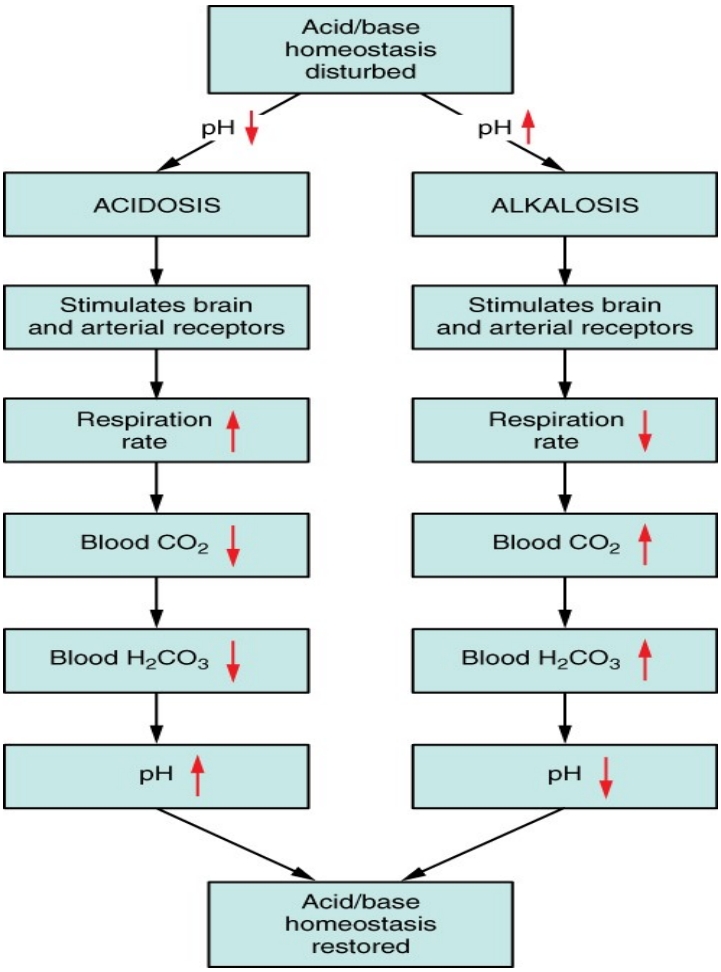

Figure 2.12 Acid-Base Imbalance. From Betts, J.G, Young, K.A., Wise, J.A., Johnson, B., Poe, B, Kruse, D.H....DeSaix, P. *Anatomy and Physiology.* Houston, Tx: OpenStaxx

BOX 2.3

SAT Safety Screen	
No active seizures	No agitation
No alcohol withdrawal	No paralytics
Normal Intracranial Pressure	
No myocardial ischemia	
SBT Safety Screen	
No agitation	Inspiratory Efforts
Oxygen Saturation >88%	
Fio2 < 50%	
PEEP < 7.5 cm H2O	
No vasopressor use	

or alkalotic. The nurse should then determine whether the paCO2 and HCO3– are within normal ranges (Box 2.4).

Respiratory Acidosis and Alkalosis

Respiratory acidosis is caused by any condition that induces hypoventilation, thus elevating a patient's CO2. This may include primary lung diseases, sedation impeding a patient's intrinsic respiratory drive, brain injuries, neuromuscular disorders, pneumothorax, and trauma impeding breathing mechanics, or incorrect ventilator settings (American Association of Critical Care Nurses, 2018). Conversely, respiratory alkalosis is caused by any condition that induces hyperventilation such as hypoxemia, sepsis, or even anxiety (American Association of Critical Care Nurses, 2018). In mechanically ventilated patients, nurses can expect providers to increase or decrease a patient's minute ventilation by manipulating tidal volume and respiratory rate to mitigate an acidosis or alkalosis of respiratory origin. An increase in minute ventilation will decrease pH. A decrease in minute ventilation will increase pH.

Metabolic Acidosis and Alkalosis

A decrease in HCO3– causes metabolic acidosis. Causes most commonly include diabetic ketoacidosis, lactic acidosis, and renal failure (American Association of Critical Care Nurses, 2018). Metabolic alkalosis is characterized by an increase in HCO3– levels and is most commonly caused by diuresis, vomiting, or potassium deficiency. Notably, massive transfusion may cause metabolic alkalosis due to citrate in the preserved blood products.

Compensation

Patients may partially or completely compensate for acid-base abnormalities. Patients who have compensated will demonstrate a normal or near-normal acid-base balance. To compensate for respiratory abnormalities, the kidneys will either excrete or reabsorb additional HCO3-. Compensation for respiratory acidosis, therefore, is characterized by a normal or near normal pH with abnormally increased HCO3 and CO2. Compensation for respiratory alkalosis is characterized by a normal or near-normal pH with decreased HCO3 and CO2. Renal compensation for respiratory abnormalities may take several days.

To compensate for metabolic abnormalities, a patient may regulate breathing to retain or remove CO2. For metabolic acidosis, a patient may increase their respiratory rate and depth to decrease the amount of CO2 dissolved in the blood. Conversely, a patient with metabolic alkalosis may decrease their respiratory rate and depth to retain CO2. Respiratory compensation occurs faster than metabolic compensation, taking only a few hours to compensate for abnormalities.•

BOX 2.4 NORMAL ABG VALUES

METRIC	NORMAL ABG RANGES	NORMAL VBG RANGES
pH	7.35-7.45	7.31-7.41
P02	80-100 mm Hg	35-40 mm Hg
SaO2	> 94%	70-75%
paCO2	35-45 mm Hg	41-51 mm Hg
HCO3-	22-26 mEq/L	22-26 mEq/L
Base Excess	-2 to +2	-2 to +2

APPLY YOUR KNOWLEDGE: CLINICAL CASE STUDY

Use the knowledge gained in this chapter and the following scenario to answer the questions below. When you are ready, check you answers on p. 69.

You are caring for a patient admitted to the CVICU yesterday for cardiogenic shock. The patient is intubated on the following ventilator settings: SIMV/PRVC; FiO2 60%; Rate 12; TV 450; PEEP 8. The patient has a PA catheter inserted to a depth of 48 cm with the following data populated on the Phillips monitor: CVP 18 mmHg; PA 45/25, Wedge pressure 24 mmHg.

1. Based on the data presented, you assess that the patient is:

A. Volume overloaded

B. Euvolemic

C. Volume depleted

2. After completing your 0800 thermodilutional cardiac output measurement, you note that the patient's cardiac index is 1.7 . Based on this information, you r *first* action is to:

A. Continue to monitor the patient . This index is expected for a patient in shock.

B. Notify the provider because the cardiac index is below the threshold for reportable values.

C. Review the patient's PVR and SVR to evaluate the type of heart failure the patient exhibits.

3. You send an ABG and note the following values: pH 7.30; PO2 96%; PaCO2 50; HCO3-24. Based on this information, you expect the provider to make what ventilator change?

A. Increase the ventilation rate to increase the minute volume

B. Decrease the ventilation rate to decrease the minute volume

C. Increase the ventilation rate to decrease the minute volume

3 CRITICAL CARE *Pharmacology*

Critical care nurses are responsible for understanding the therapeutic effects of the drugs that they administer as well as potential adverse reactions and key points of patient education. This chapter will review common classes of drugs utilized in the CVICU to care for the acutely ill cardiac patient. The list of drugs included in this chapter is not an exhaustive list of drugs administered in the critical care environment, however. Nurses should use appropriate clinical resources available at the bedside, such as Lexicomp, to inquire about additional ordered medications prior to administration.

VASOPRESSORS & INOTROPES

Vasopressors and inotropes agents of choice to manage shock states and hemodynamic instability in the post cardiac surgery patient. These agents augment intrinsic cardiac output by acting on the adrenergic receptors in the sympathetic nervous system to either constrict the peripheral vasculature or augment cardiac contractility and heart rate. Specific adrenergic receptors and their physiologic functionality can be found in Box 3.1. A comparison of hemodynamic consequences of select vasopressors and inotropes can be found in Box 3.2.

Dopamine

Dopamine augments cardiac output and MAP by increasing cardiac contractility, heart rate, and SVR. Doses typically range from 2 to 20 mcg/kg/min. The hemodynamic effects of dopamine are dose-dependent, with increased inotropy between 4 and 10 mcg/kg/min and increased alpha activity at doses greater than 10 mcg/kg/min (Hardin & Kaplow, 2020). This drug is most commonly titrated by provider order. In recent years, dopamine has fallen out of favor due to its increased risk of tachyarrhythmias at higher doses. Due to the risk of extravasation, dopamine should be administered through a central line.

Epinephrine

Epinephrine is a potent inotrope and vasopressor, with particularly vigorous inotropic properties at higher doses. Continuous infusions may be initiated at 1 mcg/min and titrated by 1 mcg/min every 2 minutes until desired effect or a maximum dose of 10 mcg/min is achieved. A provider order is required to titrate epinephrine. Due to risk of extravasation, epinephrine should be administered through a central line.

BOX 3.1 *ADRENERGIC RECEPTORS*

RECEPTOR	ANATOMICAL LOCATION	PHYSIOLOGIC FUNCTIONALITY
Beta 1	Heart	Contractility
Beta 2	Lungs/ Vasculature	Vasodilation
Alpha 1	Vasculature	Vasoconstriction
Vasopressin	Vasculature	Vasoconstriction

Norepinephrine

Norepinephrine is a both a vasopressor and an inotrope. The drug demonstrates much higher affinity for alpha receptors than beta 1 receptors, thus it is most commonly utilized for post-operative hypotension when a patient's SVR is low. Norepinephrine is also the initial vasopressor of choice in septic shock (Rhodes, 2017). Norepinephrine is titrated by nursing within the parameters outlined in the eStar order sets. The drug is initiated at 2-4 mcg/min and titrated by 1-4 mcg/minute every 2 minutes to achieve desired blood pressure goal. The maximum dose is 30 mcg/min, although providers should be notified for doses greater than 10 mcg/min. Due to the risk of extravasion, norepinephrine should be administered via central line.

Phenylephrine

Phenylephrine is a vasopressor with a rapid onset and a short half-life. It is used to treat post-operative hypotension in patients with decreased SVR and may be particularly useful in patients with tachyarrhythmias due to its lack of B1 stimulation. Continuous infusion should be started at 20 mcg/min and titrated by 20 mcg/min to desired dose. The maximum dose is 240 mcg/min. Phenylephrine may also be given as a 100-500 mcg bolus every 15 minutes and may be used as adjunctive treatment during rapid sequence intubation for propofol-related hypotension.

Vasopressin

Vasopressin is a peptide hormone that acts on vasopressin receptors to increase SVR. While vasopressin has a variety of clinical uses, it is most commonly used in CVICU to manage post-operative hypotension after reaching maximum doses of norepinephrine. The drug is initiated at 0.04 units/min and is not titrated. Rarely, providers may increase vasopressin dose to 0.06 units/min for persistent hypotension.

VASODILATORS & ANTIHYPERTENSIVES

Vasodilators are considered the preferred agents to treat postoperative hypertension following cardiac surgery, reducing blood pressure and preserving cardiac output by reducing afterload (Hardin & Kaplow, 2020). However, due to increased cost of commonly used vasodilators, providers will frequently order IV calcium channel blockers and, less commonly, beta blockers in place of IV vasodilators immediately following cardiac surgery. Beta blockers are the preferred agent following certain cardiothoracic procedures such as aneurism repairs due to their effect on decreasing heart rate, blood pressure and, cardiac output concurrently. Hemodynamic consequences of select vasodilatory agents can be found in Box 3.3.

Esmolol

Esmolol is a short-acting β1 selective beta blocker used for postoperative hypertension and tachyarrhythmias. Esmolol is also the drug of choice for vascular patients receiving IV beta blockers. Initial dosing begins at 50 mcg/kg/min and may be titrated by 50 mcg/kg/min every 2-10 minutes to a max dose 300 mcg/kg/min to achieve therapeutic goals. Side effects of esmolol include bradycardia and hypotension.

PRECEPTOR PEARLS

Although beta blockers are a cornerstone of heart failure management, beta blockers should avoided in patients demonstrating decompensated heart failure or cardiogenic shock. These patients rely on increased heart rate to maintain their cardiac output and may become hemodynamically unstable with beta blocker administration.

For these reasons, esmolol should not be used in patients in cardiogenic shock or in a second or third degree hear block (Hardin & Kaplow, 2020). Nurses should also exercise caution administering esmolol concurrently with a calcium channel blocker. Concurrent administration may exacerbate hypotension and bradycardia in some patients.

Labetalol

Labetalol is a non-selective beta-blocker that also demonstrates inhibitory effects on alpha receptors. For this reason, labetalol is generally considered to be the preferred IV beta blocker for blood pressure control and may be used as a tertiary agent following nicardipine and hydralazine for post-operative hypertension. Labetalol may be initiated as either a 10 or 20 mg IV push over 2 minutes, given at 10 minute intervals until therapeutic target is reached. Labetalol may also be given as a continuous infusion starting at 0.5 to 2 mg/minute with a maximum dose of 10 mg/minute. A total maximum dose of 300 mg/24 hours must be considered during administration. As with esmolol, labetalol should not be used in patients exhibiting decompensated heart failure or bradyarrhythmia (Hardin & Kaplow, 2020). Due to the non-selective beta activity of labetalol, patient may be at increased risk of bronchospasm and should be avoided in patients with bronchospastic disease.

Hydralazine

Hydralazine is an IV and PO vasodilator. The IV form of the drug is commonly used as a second line agent to manage post-operative hypertension not responsive to calcium channel blockers or other IV vasodilators. Hydralazine may be ordered post-operatively as a 10-20 mg IV push every four to six hours PRN. However, due to the risk of reflex tachycardia, low-dose hydralazine is recommended. Hydralazine PO may also be given to patients in heart failure with reduced ejection fraction (HFrEF) who cannot tolerate ACE or ARB therapy (Colucci, 2018).

Nicardipine

Nicardipine is an IV calcium channel blocker that causes arterial vasodilation in peripheral and coronary vasculature (Krakoff, 2017). The drug is commonly used as a first-line agent for postoperative hypertension due to the cost of other preferred vasodilators such as nitroprusside. Nicardipine IV may be initiated at 5 mg/hr and titrated by 2.5-5mg/hr every 5 minutes to a maximum dose of 15 mg/hr. Although nicardipine has minimal chronotropic effect, reflex tachycardia has been reported in the literature. Nicardipine demonstrates mild negative inotropic effects and should be used with caution in patients with heart failure with left ventricular dysfunction (Yancy et al., 2013).

Nitroglycerin

Nitroglycerin is an arterial and venous vasodilator used as an antihypertensive agent. Due to its effects on both the arterial and venous vasculature, nitroglycerin is effective at reducing preload, systemic afterload, and pulmonary arterial pressures. Importantly, nitroglycerin also has effect on dilating the coronary arteries, making it an excellent drug to treat myocardial ischemia and function as an antianginal agent. The drug is available in multiple dosage forms, including IV, oral tablet, sublingual solution, and transdermal patch or ointment. IV nitroglycerin is initiated at 5 mcg/min and titrated by 5 mcg/min every 5 minutes to desired effect. Maximum dosage of IV nitroglycerin is 400 mcg/min. Hypotension may occur in some patients, particularly those patients who are hypovolemic. Headache has also been reported in higher doses.

Nitroprusside

Nitroprusside is an powerful arterial and venous vasodilator used to reduce afterload with secondary effects decreasing wedge pressure and CVP.

Compared to nitroglycerin, nitroprusside has more arterial potency. Dosing is initiated at 0.1 mcg/kg/min and increased by 0.1-0.5 mcg/kg/min every 2-5 minutes until desired effect is achieved.

INODILATORS

Milrinone and dobutamine are two drugs that increase inotropy while minimizing vasoconstriction or promoting vasodilation. The combined inotropic and vasodilatory effect of milrinone and dobutamine make them exceptional drugs to treat end stage heart failure and cardiogenic shock.

Milrinone

Milrinone is a phosphodiesterase inhibitor used to treat low cardiac output states by increasing the amount of available calcium in myocardial ion channels, thus increasing myocardial contractility. By the same mechanism, milrinone decreases intracellular calcium concentrations in the peripheral vasculature, thus allowing for venous and arterial vasodilation (Hardin & Kaplow, 2020). Milrinone is started as a continuous infusion at 0.375 mcg/kg/min and titrated by provider order by increments of 0.125 mcg/kg/min. Notably, milrinone has a relatively long half-life of two to three hours, causing the drug's effect to linger for a prolonged period of time after it has been discontinued. The vasodilatory properties of the drug contribute to drug-related hypotension in some patients.

Dobutamine

Dobutamine is a β1 agonist that, unlike other β1 agonists, has minimal effect on α1, minimizing vasoconstrictive properties and decreasing compensatory vasoconstriction as cardiac output improves. Dobutamine is initiated at 2.5-5 mcg/kg/min and titrated by provider order to achieve desired effect. Maximum dose is 20 mcg/kg/min, although higher doses have anecdotally been reported. Similar to other β1 agonists, dobutamine demonstrates an increased risk of tachyarrhythmias but at a lower incidence than dopamine. Hypovolemia may exacerbate drug-related hypotension and patients should be adequately fluid resuscitated prior to or concurrent with administration.

ANTI ARRHTHYMICS & HEART RATE CONTROL

Amiodarone

Amiodarone is an antiarrhythmic given in the setting of atrial fibrillation , ventricular fibrillation, or unstable ventricular tachycardia. The drug increases CO and decreases MAP, HR, SVR, wedge, CVP, and PVR. It Patients will almost always receive a loading dose of 150 mg mixed in D5W given over 10 minutes. The infusion will start at 1 mg/min for 6 hours. If the initial rhythm has resolved, the nurse should anticipate that the provider will drop the infusion rate to 0.5 mg/min after 6 hours, maintaining the drip at 0.5 mg/min for the subsequent 18 hours prior to transitioning to oral Amiodarone. Amiodarone may also be used for patients who arrest with a VF/pulseless VT etiology and are unresponsive to defibrillation (American Heart Association, 2018).

Diltiazem

Diltiazem is a calcium channel blocker used for heart rate control in the setting of sinus tachycar-

PRECEPTOR PEARLS

When giving nitroprusside, it is important to evaluate the color of the drug. When nitroprusside turns blue, it has decomposed into cyanide and should not be given to the patient.

BOX 3.2 *ACTIONS OF SELECT VASOPRESSORS AND INOTROPES*

	Norepinephrine (Levophed)	Phenylephrine (Neo-synephrine)	Epinephrine	Dopamine	Vasopressin
CO	↑	↓	↑	↑	↓
PAOP	↑	↑	↑	↑	↑
SVR	↑	↑	↑	↑	↑
MAP	↑	↑	↑	↑	↑
HR	↔	↓	↑	↑	↓
CVP	↑	↑	↑	↑	↑
PVR	↑	↑	↑	↑	↑
Receptor Agonist/ MOA	Primarily alpha, some beta agonist properties	alpha	Alpha, Beta1, Beta 2. More Beta at higher doses	Alpha, Beta1, Beta2	Vasopressin 1A
Dose/ Titration	2-20 mcg/min Titrate by 1-2 mcg/ min	20-200 mcg/min Titrate by 20 mcg/ min	1-10 mcg/min Titrate by 1 mcg/min with provider order	1-20 mcg/kg/min Titrate by 5 mcg/ kg/min with provider order	0.04 units/min May see 0.06 units/min; do not titrate without order
Concentration	8mg/250ml 16mg/250ml D5W D5WNS	30mg/250ml 120mg/250ml D5W NS	4mg/250ml 8mg/250ml D5W NS	400mg/250ml 1600mg/250ml D5W NS	60un/100ml 100un/100ml D5W
Access	Central	Central	Central	Central	Central
Uses	Increase HR and contractility to increase systemic blood pressure	Alpha agonist that increases arterial vasoconstriction	Increase heart rate and contractility. Alpha, B1,B2 agonist	Positive inotrope increases contractility. B1 agonist (low dose) Alpha agonist (high dose)	V1 agonist that works on vascular smooth muscle increase BP
Key Clinical Points	Notify team for levophed doses greater than 10 mcg/min or for rapidly increasing levophed requirements	Can cause bradycardia, not a first line drug in post-cardiac surgery patients	May cause hyperglycemia—trend blood glucose carefully. May increase lactate production.	Can cause tachyarrhythmias.	Consider Use on high dose levophed, not to be used as monotherapy.

BOX 3.3 *ACTIONS OF SELECT VASODILATORS AND INODILATORS*

	Dobutamine	Milrinone	Nitroprusside	Nitroglycerine
CO	↑	↑	↑	↑
PAOP	↓	↓	↓	↓
SVR	↓	↓	↓	↓
MAP	↑	↔	↓	↓
HR	↑	↑	↑	↑
CVP	↓	↓	↓	↓
PVR	↓	↓	↓	↓
Receptor Agonist/ MOA	B1 agonist	Phosphodiesterase Inhibitor, increases cAMP which increases Ca++ in heart		
Dose/ Titration	1-30 mcg/kg/min Titrate with order by 1-2 mcg	0.375-0.75 mcg/kg/min (can go lower too). MD order to titrate.	0.2-10 mcg/kg/ min titrate for target BP by 0.1-.0.2 mcg.	25 mcg-400 mcg. Titrate by 10 to 25 mcg to lower BP and decrease chest pain.
Concentration	250mg/250ml 1000mg/250ml D5W NS	40mg/200ml 80mg/200ml .45%NaCl, NS, D5W	50mg/250ml 100mg/250 ml D5W	25mg/250ml 100mg/250ml D5W NS
Access	PIV or Central	PIV or central	Central	Central or PIV
Uses	Positive Inotrope, stimulate B1 adrenergic receptors to increase contractility and reduce left ventricular filling pressures in heart failure, post open heart, pulmonary congestion	Phospho diasterase-inhibitor, non-adrenergic . Increases contractility, decrease preload, decrease afterload. Use in low CI, heart failure patients	Potent-direct vasodilator. Increases CO by decreasing afterload. Used initially in the post -op period to decrease BP	In MI patients due to the reduction in preload thus reducing cardiac oxygen demand. Vasodilates primarily veins. Reduces BP and chest pain.
Key Clinical Points	Can Cause tachy- arrhythmias esp after 72hrs, ensure patient if fluid volume resuscitated.	Monitor for thrombocytopenia. When weaning watch for changes in CI in 3-6 hours	Protect from light, monitor for low SaO2 due to shunting and acidosis. Team aware if on >1mcg	Can cause headaches, interacts with Viagra. Prescribed sometimes to help decrease spasms after an intervention.

tachycardia be given IV push over 2 minutes prior to initiating the drip. Diltiazem may be titrated between 5-15mg every 5 minutes until desired heart rate response is achieved. Nurses should monitor the patient for hypotension, particularly during bolus administration.

SEDATION

Sedation infusions are commonly used in critical care for procedures and to manage anxiety related to mechanical ventilation. When managing sedation, it is important to assess and treat alternative sources of agitation such as pain prior to initiating sedation therapy (Devlin et al., 2018). Analgesic medications to manage post-operative pain can be found in Chapter 3. Due to their association with ICU delirium, routine use of benzodiazepines should be avoided. Nurses should assess patient sedation depth using the validated Richmond-Agitation Sedation Scale (RASS) at minimum every two hours for patients receiving continuous sedation, adjusting the rate of infusion to meet sedation targets (Box 3.5). Actions of select sedation agents can be found in Box 3.4.

Dexmedetomidine

Dexmedetomidine is a selective alpha2-agonist used sedation. The drug does not have analgesic properties. Patients receiving this drug are arousable to stimulation and have lower rates of delirium compared to other agents such as benzodiazepines. Due to the minimal effect on respiratory suppression, dexmedetomidine is the only sedative agent approved for use in extubated patients. Dexmedetomidine is started at 0.2 to 0.5 mcg/kg/hour and may be titrated by 0.1 to 0.3 mcg/kg/hr to a maximum dose of 1.5 mcg/kg/hr. Due to the risk of hypotension and bradycardia, loading doses and boluses are not routinely administered in the ICU. Nurses should monitor for dose-dependent hypotension and bradyarrhythmia throughout the duration of therapy.

Ketamine

Ketamine NMDA receptor antagonist that inhibits the action of glutamate, an excitatory neurotransmitter. The drug has sedative, analgesic, and amnestic properties. Although ketamine may be given through a variety of routes, it is most commonly used in the ICU for procedural sedation or as a continuous infusion for sedation or analgesia to reduce opioid use. Ketamine may be continuously infused at 1-2 mcg/kg/min to maintain desired parameters. If used for procedural sedation, a bolus of 1 to 4.5 mg/kg may be ordered. Nurses should monitor for tachycardia and hypertension with ketamine infusion and report an increased heart rate greater than 110 bpm or an increase in systolic blood pressure greater than 25 mmHg from baseline. Due to its increase on myocardial oxygen demand, ketamine should be used with caution in patients with known coronary artery disease. Ketamine may also induce hallucinations or nightmares in some patients.

BOX 3.5 *RICHMOND AGITSTION-SEDATION (RASS) SCALE*

SCORE	DESCRIPTION
+4	Combative: violent with staff or a threat to self
+3	Very Agitated: Attempts to remove tubes or lines; aggressive with staff
+2	Agitated: Frequent, non purposeful movement; fights ventilator
+1	Restless: Visibly anxious or apprehensive
0	Alert and Calm
-1	Drowsy: Not fully alert but sustains eye contact for greater than 10 seconds to voice
-2	Light Sedation: Briefly (less than 10 seconds) awakens to voice
-3	Deep sedation: movement to physical stimulation only
-4	Unarousable to voice or physical stimulation

BOX 3.4 *ACTIONS OF SELECT SEDATION AGENTS*

	Dosage	Onset	Peak	Duration	Side Effects
Precedex	0.2-0.7 mcg/kg/hr Max dose 1.5 mcg/kg/hr	5-10 minutes	15-30 minutes	1-2 hours	Bradycardia, hypotension, oversedation
Propofol	10-50 mcg/kg/min	<40 seconds	Unknown	10-15 min	Bradycardia, hypotension, respiratory depression
Ketamine	1-2 mcg/kg/min	30-40 seconds	Unknown	10-15 min	Tachycardia, hypertension, hallucinations, respiratory depression

Propofol

Propofol is a GABA agonist that has both sedative and amnestic properties. Propofol may be titrated from an initial dose of 5 mcg/kg/min by 5 to 10 mcg/kg/min every 5-10 minutes until desired effect is achieved. The maximum dose of propofol for continuous sedation in the ICU is 50 mcg/kg/min. Propofol may cause dose-related bradycardia and hypotension, particularly in patients with underlying cardiovascular disease. For this reason, propofol infusions are never bolused in CVICU patient populations.

NEUROMUSCULAR BLOCKADE

Neuromuscular blocking agents, or paralytics, are used in conjunction with sedation during rapid sequence intubation (RSI) to facilitate intubation and during targeted temperature management to prevent shivering. When used for RSI, nurses may be asked to give a one-time push of a paralytic agent under physician supervision. The following agents are used most commonly in RSI: vecuronium, rocuronium, and succinylcholine. Of these, succinylcholine has the most rapid onset, between 30-60 seconds, and the shortest duration of 6-10 minutes. These characteristics make it an ideal choice in RSI. However, the drug may cause a precipitous rise in potassium and should be avoided in hyperkalemic patients or those with extensive tissue trauma. The remaining drugs, rocuronium and vecuronium, have longer durations of approximately 30 minutes, making them appropriate choices for procedures such as bedside trach placement.

Cisatracurium

Cisatracurium is a nondepolarizing paralytic with a quick onset of 2-3 minutes and a longer duration of 35-45 minutes. It is the only paralytic routinely given as a continuous infusion and is part of the targeted temperature management order set to prevent shivering in patients cooled after cardiac arrest. For patients receiving continuous paralytic, nurses should monitor the extent of paralysis with a Train of Four (TOF). Cisatracurium should be initiated at a rate of 1-2 mcg/kg/minute and titrated every hour to achieve and maintain a TOF of 1-2 twitches. More information regarding TOF management can be found in Chapter 5. •

PRECEPTOR PEARLS

A patient should be adequately sedated prior to giving a paralytic. A Bispectral Index Range (BIS) monitor is used to gauge the level of alertness in paralyzed patients. BIS scores range from 1-100, with higher numbers correlating with more alertness. Sedation should be titrated to achieve a BIS score of 40-60.

APPLY YOUR KNOWLEDGE: CLINICAL CASE STUDY

Use the knowledge gained in this chapter and the following scenario to answer the questions below. When you are ready, check you answers on *p. 69.*

You are caring for a patient who is admitted for decompensated heart failure, awaiting a heart transplant. The CCU team places a PA catheter, obtaining the following data: PA pressure 48/32, CVP 12, SVR 2250, CI 1.7. The team starts the patient on milrionone 0.375 mcg/kg/min.

1. The team asks you to shoot another set of numbers one hour after the drip is initiated. Which set of numbers represents the anticipated response to the initiation of milrinone?

    A. PA pressure 55/42, CVP 14, SVR 2500, CI 1.5

    B. PA pressure 35/24, CVP 12, SVR 1800, CI 2.0

    C. PA pressure 45/35, CVP 12, SVR 2400, CI 1.9

2. The following day, the patient receives a heart transplant. The patient is persistently hypotensive in the OR and arrives to the unit on epinephrine 3 mcg/min and norepinephrine 8 mcg/min. Which of the following represents the expected hemodynamic response to levophed?

    A. Decrease MAP by decreasing SVR

    B. Increase MAP by decreasing SVR

    C. Increase MAP by increasing SVR

3. Overnight, the team weaned the levophed. This morning the team asks you to turn the epinephrine down from 3 mcg/min to 2 mcg/min. When shooting your next set of numbers, you expect:

    A. The cardiac index to decrease from 2.4 to 2.2

    B. The cardiac index to increase from 2.3 to 2.5

    C. The cardiac index to remain unchanged

4 CARDIAC SURGERY

Nursing Care of the Patient Undergoing Cardiac Surgery

Cardiovascular intensive care nurses can expect to care for patients undergoing a wide variety of cardiac procedures as well as vascular procedures that require a thoracic approach. This chapter will provide an introduction to the most common procedures received in the CVICU, highlighting standard preoperative and immediate postoperative care. Postoperative complications will also be reviewed.

PREOPERATIVE NURSING CARE

Preoperative patient assessment includes a baseline assessment of heart function and comorbidities to evaluate surgical risk. Standard preoperative labs include: CBC, CMP, HgA1c, type & screen, and an urinalysis. Patients taking coumadin at home or patients receiving heparin preoperatively will also receive coagulation studies. Comorbidities such as COPD, diabetes, renal insufficiency, peripheral vascular disease, and heart failure with reduced ejection fraction are associated with increased perioperative risk (Nadim et al., 2018). Additional diagnostic studies such as 12-lead EKG; echocardiography; CT or cardiac MRI; cardiac catheterization; and pulmonary function tests may also be performed preoperatively in non-emergent cases. Reviewing preoperative results may inform post-operative nursing assessment, priorities, and plan of care.

Patients admitted to the ICU prior to surgery require specific nursing interventions. Intranasal mupirocin, used to prevent MRSA infection, will be ordered every 12 hours starting the night before surgery and will continue until the patient receives 10 postoperative doses. Preoperative antibiotics will be ordered to go to the OR with the patient and will be given by the anesthesia team within one hour of the first incision. Unless the patient has an allergy, standard antibiotics include cefazolin and vancomycin.

Patients should be allowed to shower or receive a soap and water bath the night before surgery. After the patients have received a soap and water bath, nurses should provide two preoperative CHG scrubs, one the night before surgery and one the morning of surgery as close to the operative time as possible. Patients should have hair clipped immediately prior to the second CHG scrub. Patients should be clipped at minimum four inches wider than any possible incision. Special attention should be paid to clipping groin areas in the event that the surgical team will need to perform a cut down to the femoral artery. For urgent or emergent cases, patients should receive a minimum of one preoperative CHG scrub immediately following patient clipping.

All CVICU patients will be transported to the operative suite with defibrillator pads in place. In lieu of the standard placement, pads will be placed laterally on either side of the chest, immediately inferior to the axilla with the wires tracing up between the should blades. This placement allows the pads to be easily accessed during surgery while remaining free of the surgical field for a median sternotomy approach. Patients who will receive mini thoracotomy approaches should have pads placed in the standard fashion.

CORONARY ARTERY BYPASS

Coronary artery bypass (CAB) is indicated for patients with multivessel coronary disease or sever single vessel disease that cannot be treated with percutaneous coronary intervention (PCI). To perform a CAB, the surgeon most commonly accesses the thoracic cavity via a median sternotomy incision. Vessel grafts, harvested from the left internal mammary artery, saphenous veins, or radial artery, are used as conduits to bypass diseased vessels and restore blood flow to the myocardium. Grafts are sewn into the proximal aorta and routed into the affected coronary artery just distal to the occluded portion of the vessel.

CAB is usually performed using cardiopulmonary bypass, commonly referred to as 'on pump' (Figure 4.1). In this procedure, the patient is cooled between 30-34° Celsius, the heart is stopped using cardioplegia, and the aorta is cross clamped. Blood is drained from the right atrium, oxygenated in the bypass machine, and returned to the body via a cannula placed distally to the cross clamped section of the aorta. Heparin is used to reduce the risk of clot formation in the circuit. Less frequently, CAB will be performed without use of the bypass machine, referred to as an off-pump CAB (OPCAB). In this procedure, the surgeon sews grafts into the myocardium while the heart is still beating. Although OPCAB may avoid certain risks associated with cardiopulmonary bypass, it is associated with higher rates of graft occlusion than traditional CAB (Hardin & Kaplow, 2020). Additionally, a patient may need to be converted to an on-pump procedure if the coronary arteries are determined to be particularly small, the left ventricular function is poorer than expected, or uncontrollable arrhythmias develop.

Nursing Considerations

Anesthesia providers will bring patients to the ICU immediately after the chest is closed. The use of cardiopulmonary bypass simultaneously increases the risk of bleeding and clot formation. Bleeding may occur from inadequate heparin reversal; hemodilution and hemolysis during cardiopulmonary bypass; and bleeding from anastomosis sites. Hypothermia

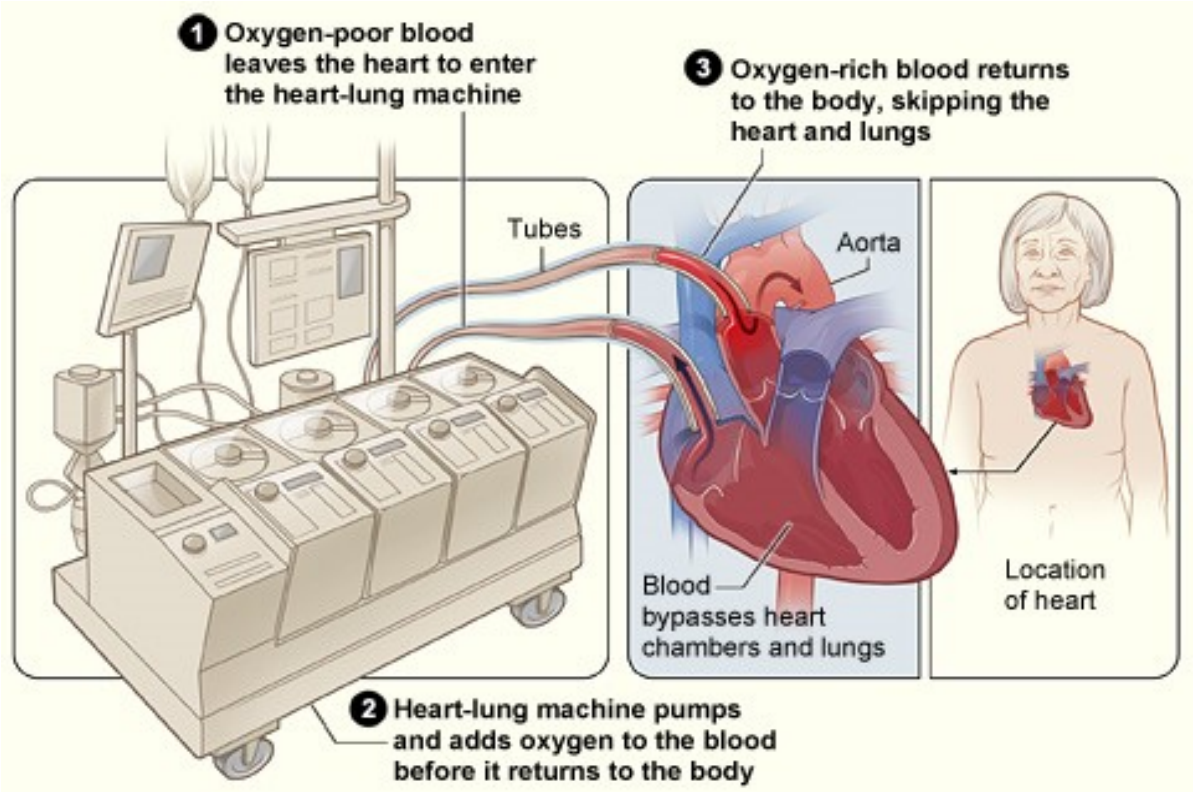

Figure 4.1. Cardiopulmonary Bypass. By National Heart Lung and Blood Institute (NIH) - National Heart Lung and Blood Institute (NIH), Public Domain, https://commons.wikimedia.org/w/index.php?curid=29588210

exacerbates the risk of bleeding due to the inactivation of the clotting cascade at low temperatures. The risk for clot formation is largely due to the activation of platelets and inflammatory mediators when blood interacts with the bypass circuit. Nurses must remain vigilant for signs and symptoms of bleeding, closely monitoring chest tube output (including the presence of clots), maintaining chest tube patency, and monitoring patient labs.

Surgical stress and hypothermia increase the risk of postoperative hyperglycemia, which has also been shown to increase postoperative morbidity. Insulin protocol will be initiated on all postoperative patients to maintain blood sugars within a normal range and the patient should be rewarmed as quickly as possible. Following cardiac surgery, patients are at increased risk of renal dysfunction, hemorrhagic stroke, and ischemic stroke. It is important, therefore, that nurses perform a comprehensive patient assessment as quickly as possible and at routine intervals. A comprehensive postoperative workflow can be found in *Box 4.1*. Provider notification parameters can be found in *Box 4.2* on the following page.

VALVE REPLACEMENT

Valve surgery is performed to either repair or replace diseased cardiac valves. Valvular disease is described by the affected valve and the functional malformation, either stenosis or regurgitation. Stenosis describes a valve with anatomic narrowing that impedes forward blood flow within the heart, most commonly caused by calcifications on the valve leaflets, congenital abnormalities, or rheumatic heart disease. Regurgitation describes a valve that no longer closes appropriately, allowing regurgitant and inefficient blood flow through the heart. Surgical intervention is indicated when patients become symptomatic or when ventricular ejection becomes severely impeded.

Open valve replacement is typically conducted via

BOX 4.1 *Open Heart Admission Workflow*

Upon Arrival	Hook up arterial line to Phillips monitor*
	Hook up EKG, SPO2, CVP & PA to Phillips monitor*
	Hook up chest tubes to suction, marking initial level
	Document initial urine output
	Shoot cardiac outputs and print rhythm strip*
	Trace IV lines from bag to pump to patient
	Check OG tube placement and hook up to LIWS*
	Send CBC, coagulation studies (if bleeding), and ABG
	Apply Bair hugger if patient <36°
	Change NS carrier to D5NS per insulin Protocol; Slowly decrease rate to 30 ml/hr
	Administer 4 mg Magnesium Sulfate per provider order
Postoperative Phase	Record vital signs, UOP, and CT output q 15 minutes x4 or until CT output < 30 ml every within a 15 minute interval
	Shoot cardiac outputs Q1 hour x4 then Q4 hour
	Trend labs, particularly electrolytes, and replace per protocol or order as needed
	Titrate vasoactive medications to SBP & MAP goals

*Items typically delegated to Help All

PRECEPTOR PEARLS

Keeping tight control of blood pressure is important post surgery. MAP must be high enough to perfuse end organs, but Increased SBP puts increased pressure on fresh anastomoses., increasing risk of bleeding.

a median sternotomy, however, mitral valve and aortic valve replacement may be performed through a thoracotomy incision. Both median sternotomy and thoracotomy approaches require the use of cardiopulmonary bypass. Replacement valves are either mechanical or bioprosthetic. Mechanical valves are more durable but also carry a higher risk of thromboembolic events, requiring patients to remain on anticoagulation after surgery. While bioprosthetic valves avoid the need for anticoagulation, these valves do not last as long as their mechanical counterparts (Hardin & Kaplow, 2020). The decision to utilize a bioprosthetic or mechanical valve is highly patient specific and discussed during the preoperative patient evaluation.

In recent years, the advent of transcatheter aortic valve replacement (TAVR) has revolutionized the treatment of aortic stenosis in patients whose surgical risk would otherwise be prohibitive to open replacement. The success of these procedures has prompted subsequent clinical trials in lower risk surgical patients, increasing excitement that minimally invasive valve replacement may be available to more patients in the near future. TAVR is traditionally performed by accessing the femoral artery and deploying a specially crafted mechanical valve atop the existing aortic valve. The is conducted in a hybrid OR with a cardiac surgeon and bypass machine available if the patient's clinical condition deteriorates. Increasingly, TAVRs are performed under moderate sedation and patients are frequently transferred to cardiac stepdown immediately following sheath removal.

Nursing Considerations

Open valve replacements carry the same risk profile of CAB procedures and patients should be monitored accordingly. An open heart admission workflow can be found in *Box 4.1* on the previous page and provider notification parameters can be found in *Box 4.2.* In addition to the standard risks of cardiopulmonary bypass, patients undergoing valve replacement are at increased risk of thromboembolic events, infective endocarditis, paravalvular leak, atrial fibrillation, and AV block post procedure (Gaasch & Zoghbi, 2017). To mitigate the risk of bradyarrhythmia postoperatively, pacing wires are usually kept in place for at least 48 hours after surgery. Importantly, the acute hemodynamic changes incurred from replacing a chronically dysfunctional valve may prompt hemodynamic instability in the immediate postoperative phase. This requires careful volume management; close hemodynamic monitoring; and either inotropic or vasodilatory support (Hardin & Kaplow, 2020). In spite of these risks, patients are usually extubated within 6 hours of arrival to the unit and discharged to stepdown POD 1 or POD 2.

BOX 4.2 *Provider Notification Parameters*

Signs & Symptoms of CVA/TIA	CI<2.0 or significant decrease
SVO2<60	SBP<90 or >150
HR >100 or <55 (w/rhythm strips)	Loss/Change of extremity pulse, temp, movement
Starting vasoactive drips not initiated prior to arrival in CVICU	Levo>10 mcg/min or significant increase
Nipride >2 mcg/kg/min	Propofol >50 mcg/kg/min
Chest Tube output >100cc/hr or >25cc/15 min	UOP<30 cc/hr
Oxygen Requirement >6LNC after extubation	Sat<92%
FiO2 >60% while ventilated	pH<7.25
pCO2 >55	pO2<60
PCV <25 or Hgb <7	K<3 or >5.5

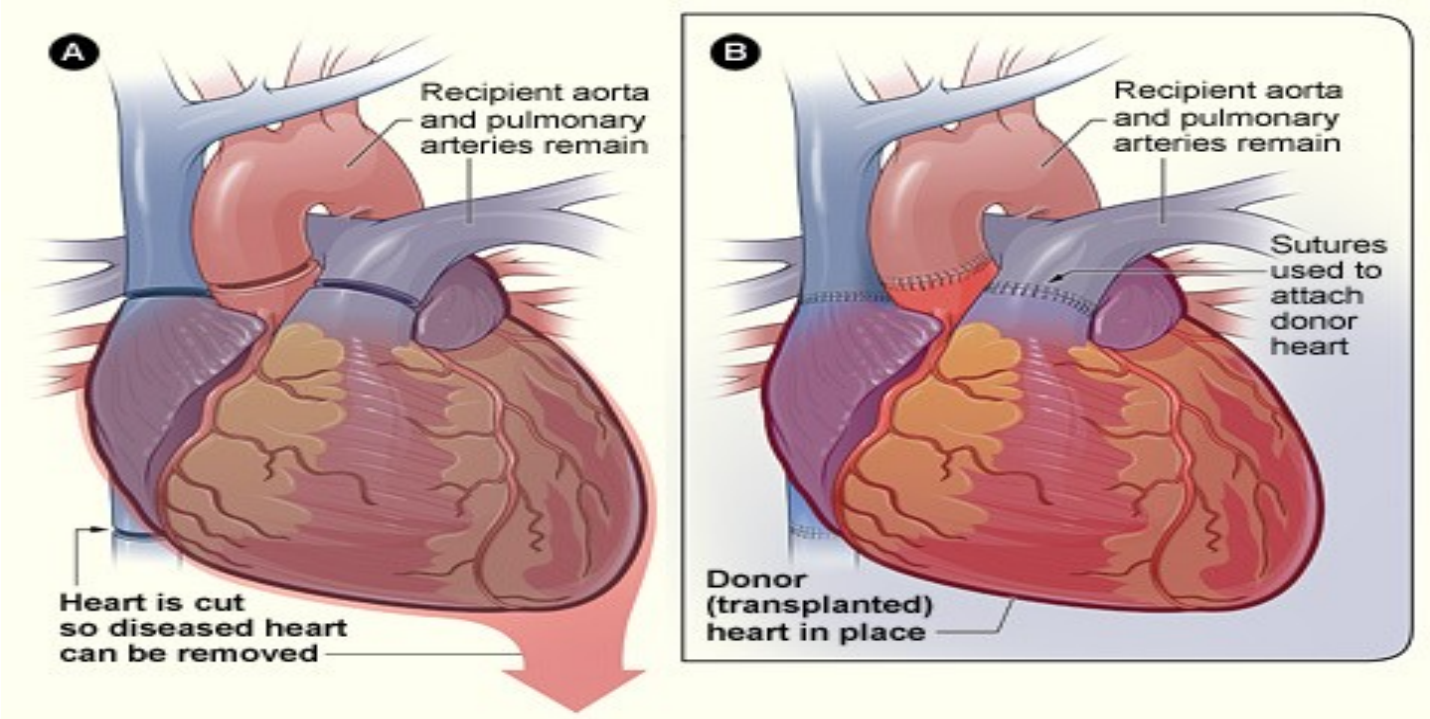

Figure 4.2. A) Pictorial diagram showing where the diseased heart is removed for transplant. B) Anastomoses of the transplanted heart after implantation. By National Heart Lung and Blood Institute (NIH) - National Heart Lung and Blood Institute (NIH), Public Domain, https://commons.wikimedia.org/w/index.php?curid=29588222

TAVR carries a relatively high risk of stroke and post-operative dysrhythmias. Neurological checks should be every hour in the immediate postoperative phase and routinely thereafter. Any neurological changes should be reported immediately to the provider team and a STAT CT should be ordered. Additionally, nurses should carefully monitor TAVR access sites for signs of bleeding, including signs of retroperitoneal bleeding, due to the large sheath sizes utilized for the procedure.

HEART TRANSPLANTATION

Heart transplantation is indicated for patients who have failed medical treatment of their heart failure and continue to exhibit significant symptoms and reduction in quality of life, defined as New York Heart Failure Association (NYHA) class III or IV. Further information about heart failure classification and medical management of heart failure can be found in Chapter 5. Heart transplantation may also be indicated for patient who exhibit recurrent arrhythmias refractory to treatment, cardiomyopathy, or congenital heart disease in which traditional repair techniques are not applicable (Hardin & Kaplow, 2020). Patients undergo extensive testing to determine their eligibility for heart transplantation, including but not limited to: evaluation of comorbidities such as pulmonary hypertension, diabetes, and renal dysfunction; immunization status; psychological evaluation as well as an evaluation of social and financial support. Once approved, a transplant coordinator will work with the Organ Procurement and Transplantation Network (OPTN) to place the patient on the national waitlist. Patients are listed according to the severity of their illness, ranked as status 1-6 by the OPTN. Status 1 patients represent the most acutely ill patients, such as patients who are admitted to the ICU for non-dischargeable biventricular support. A complete list of organ allocation criteria for medical urgency can be found in Box 4.3 on the following page. Organs are allocated by

geographic region, patient size, antibody matching, and patient acuity. Once an organ has been matched, a member of the surgical team will travel to the donor to conduct a final evaluation of the organ for transplantation and the transplant coordinator will contact the patient to alert them that an organ has become available.

Nursing Considerations

Heart transplantation is a 6-8 hour operation. Patients will arrive to the ICU prior to transplant for preoperative care. In addition to the usual preoperative nursing care, ICU nurses can expect to send additional patient-specific antibody labs and assist with line insertion prior to the patient going to the OR. If the patient is admitted with a PICC line for IV inotropy, this line should be removed prior to the patient going to the OR suite or PROMPTLY upon return.

BOX 4.3 *OPTN Adult Heart Allocation Criteria, 2018 revision*

Status 1	VA ECMO Non-dischargeable biventricular support MCS with life-threatening arrhythmia
Status 2	Non-dischargable LVAD IABP or other percutaneous MCS Life-threatening arrhythmias w/o MCS TAH, BiVAD, or RVAD, VAD for single ventricle patients
Status 3	Multiple inotropes or single inotrope with continuous hemodynamic monitoring VA ECMO after 7 days; Non-dischargeable LVAD after 14 days, IABP after 14 days MCS device with clinical complication
Status 4	Dischargeable LVAD Inotropes without hemodynamic monitoring Re-transplant, congenital heart disease, amyloidosis, certain cardiomyopathies Ishemic heart disease with angina
Status 5	Those waitlisted for multiple organs at a single facility
Status 6	All remaining candidates

The postoperative course of heart transplants patients is variable and dependent upon recipient, donor, and operative factors. Recipients with preoperative pulmonary hypertension, renal dysfunction, or mechanical circulatory support (MCS) are more likely to have postoperative complications. Older donor age, longer ischemic times, and recipient-donor mismatch may also influence postoperative outcomes (Hardin & Kaplow, 2020). Bleeding, hypovolemia, and right ventricular dysfunction are common complications following heart transplantation. To protect the right ventricle, patients may arrive to the ICU on inhaled vasodilators such as Flolan or nitric oxide. Volume status must also be delicately managed in the post-heart transplant patient. Hypovolemia may decrease coronary artery perfusion to a stunned myocardium, however, hypervolemia may overwhelm a stunned right ventricle (Hardin & Kaplow, 2020). Lastly, nurses can expect that the post-heart transplant patient will arrive to the ICU with epicardial pacing between 100-110 bpm. Pacing at a higher rate not only augments cardiac output postoperatively but also stimulates the return of sinoatrial node function after the vagus nerve has been severed during transplant.

In addition to standard monitoring of the post-cardiotomy patient, ICU nurses must closely monitor for signs of rejection. All patients will receive immunosuppressive agents immediately prior to transplant (induction) and for the duration of their life thereafter. Anti-rejection agents of choice include calcineuron inhibitors (CNI), antiproliferative agents, and steroids. A list of select anti-rejection agents, including administration and specialized considerations, can be found in Box 4.4. Hyperacute rejection, caused by allograft mismatch, is rare and occurs within minutes to hours of transplantation. Acute cellular rejection is more common and occurs most frequently within the first 6 months of transplant (Eisen, 2017). For this reason, cardiac biopsies are

BOX 4.4 *Select Immunosuppressive Agents*

Medication	Class	Administration	Special Considerations
Cyclosporine	CNI	PO or IV Time sensitive: Q12 hours, dosed at 0600 and 1800	Draw daily lab troughs at 5 am Use gloves when handling medication; do not crush; must order suspension for per tube
Tacrolimus	CNI	PO or Sublingual Time sensitive: Q12 hours, dosed at 0600 and 1800	Draw daily lab troughs at 5 am Use gloves when handling; do not crush, must open capsule and deliver contents sublingual if unable to swallow pill (wear globes & N95 to do so).
Mycophenolate	Anti-proliferative	PO or IV (slowly over 2 hours) Time sensitive: Q12 hours, dosed at 0600 and 1800	Use care when discarding. Put IV tubing and bag in yellow containers. Use gloves when handling; do not crush
Methylprednisolone	Steroid	PO or IV (may be given IVP or piggback depending on dose Ordered daily	Closely monitor blood glucose Dose weaned by team based on cardiac biopsies May be increased if s/s of rejection
THYMOglobulin	Polyclonal Antibody	IV (1st dose over 6 hrs, remaining doses over 4-6 hrs.)	Used for induction or steroid-resistant rejection MUST use Central access and 0.22 micron filter Premedicate with Tylenol, Benadryl, steroids. Anaphylaxis kit at bedside.
Basiliximab (Simulect)	Monoclonal Antibody	IV infusion over 30 minutes	Ordered as induction ONLY No premeds needed

obtained at routine intervals for the first 6 months after transplant. Antibody-mediated rejection is less common, but may occur at any time after transplantation, necessitating lifelong immunosuppression. It is important to note that mild rejection is often asymptomatic and only discovered by routine surveillance. Symptomatic or more severe rejection may present with new or worsening heart failure symptoms from primary graft dysfunction.

Transplant recipients receive extensive education preoperatively. Nonetheless, ICU nurses must be prepared to reinforce this education during the immediate postoperative course. Since transplant patients are immunosuppressed, nursing staff, patients, and family members must practice meticulous infection prevention measures. In addition to antirejection medication, patients will receive prophylactic antibiotics and antiviral medication immediately following transplant.. Patients will also need to avoid undercooked meats and wash all fruits and vegetables prior to consuming them. Raw foods should be avoided immediately after transplant. Finally, cardiac rehab is an important part of postoperative recovery. All Vanderbilt transplant patients stay locally after discharge for frequent appointments with the outpatient heart transplant team and to attend cardiac rehab at the Dayani Center.

LUNG TRANSPLANTATION

Lung transplantation is indicated for patients who have end –stage lung disease that is no longer responsive to medical management.. This may include patients with COPD, cystic fibrosis, primary pulmonary hypertension, or sarcoidosis. The pre-transplant evaluation for lung transplantation is similarly rigorous to that for heart transplantation. As with heart transplantation, patients are listed according to the acuity of their clinical status and matched according to geographical region, organ size, and antibody screening. Upon arrival to the unit, patients should receive routine pre-operative care, including the additional pre-transplant labs. The lung transplant team prefers their patients to be clipped in the OR immediately prior to the first incision.

Nursing Considerations

Lung transplants are performed using a clamshell incision, which is particularly painful postoperatively. For this reason, all lung transplant patients receive an epidural prior to extubation. The Vanderbilt Pain Management service will insert and set up the epidural in the ICU. Nursing staff are responsible for monitoring the epidural site at routine frequency, documenting epidural infusion volumes, and changing epidural medication bags. An epidural quick guide can be found in *Box 4.5.*

Much of the postoperative care of lung transplant patients is geared toward avoiding reperfusion injury to the allograft. Reperfusion injury presents as severe pulmonary edema of non-cardiac etiology with reduced lung compliance and impaired gas exchange. To this end, patients will arrive to the unit on pulmonary vasodilators and will likely be extubated with these agents in place. Lung protective ventilation strategies and early extubation also prevent unnecessary barotrauma to the new organ. Conservative fluid resuscitation will also prevent reperfusion injury and nurses should expect to keep lung patients drier than other populations, sometimes at the expense of increased pressor utilization in the short term. Patients should not be wedged to avoid unnecessary pressure on the anastomosis site at the pulmonary artery.

After extubation, patients will be kept strictly NPO until a swallow evaluation can be completed to decrease the risk of aspiration. As with other transplants, meticulous infection prevention strategies

BOX 4.5 *Epidural Quick Guide*

To Unlock the Keypad:	-Press STOP -Enter Code 016
To View the Current Program Settings:	-Press OPTIONS -Press 1 for REVIEW PROGRAM
To Change the Program:	-The pump must be stopped and the keypad unlocked before the program can be changed -Press CHANGE -Press #3 for CHANGE PROGRAM -Press ENTER at each screen for variables you want to change -Use the down arrow to review the revised program -Press ENTER
To Change the Empty Container:	-The pump must be stopped and the keypad unlocked -Press CHANGE -Press #1 for NEW CONTAINER -Press START -The pump will resume the previously programmed delivery at the previously selected container size
To Review History/Event Log:	-Press OPTIONS -Press #2 HISTORIES -Press #1 HISTORY, scroll through the event log by pressing the

should be employed. Patients will receive a similar immunosuppression regimen to that previously described in this chapter and should be educated regarding their medications. Lung transplant patients will also need to follow a transplant-approved diet, including fully cooked meats and stringent washing of fresh fruits and vegetables.

VASCULAR SURGERY

A number of vascular diseases, most commonly aortic aneurysm and dissections, are also cared for in the CVICU. An aortic aneurysm is defined as a dilation of the aorta at least 50% of its normal size (Sidebotham, Mckee, Gillham, & Levy, 2007). Aneurysms are described by their anatomical location. Ascending aortic and aortic arch aneurysms arise between the aortic valve and the left subclavian artery. Descending thoracic and thoracoabdominal aneurisms occur distal to the left subclavian artery.

By contrast, an aortic dissection is caused by a tear in the intimal layer of the aortic wall that develops a false lumen. As with aneurysms, dissections are classified by anatomical location. Type A dissections affect the ascending aorta and Type B dissections affect the descending aorta. Both diseases are multifactorial in origin. Patients may have genetic predisposition to the disease, congenital malformation, or family history of vascular disease. Comorbidities such as obstructive lung disease and a number of modifiable risk factors such as hypertension and smoking also impact disease prevalence. In the early stages, aortic malformations involving the descending thoracic and abdominal aorta may be treated medically by managing hypertension and monitoring the malformation with routine CT scanning. Malformations involving the ascending aorta or large malformations in any part of the descending aorta require surgical repair.

Nursing Considerations

Patients receiving open thoracic and thoracoabdominal repair will experience significant fluid shifts and potential blood loss, requiring generous volume resuscitation. Neurovascular checks should be performed every hour during the immediate postoperative phase due to the high risk of ischemia following aortic cross clamp. Up to 40% of patients undergoing open repair will experience acute kidney injury (Becker, 2016). Nurses should closely monitor intake , output, BUN, and creatinine in these patients. Patients will remain strictly NPO until they are passing gas. Nurses should educate patients regarding pulmonary toilet and facilitate ambulation TID beginning POD 2.

Thoracic endovascular aortic repair (TEVAR) involves the insertion of a vascular graft into the affected region of the aorta via the femoral artery. Endovascular repair demonstrates decreased surgical risk across all categories, decreased risk of blood loss, and is an option for many descending aortic malformations. Patients will arrive to the ICU extubated and transfer to the floor POD 1. As with open repair, nurses should conduct frequent neurovascular checks postoperatively.

All thoracic aortic repairs, including TEVAR, will arrive to the unit with a lumbar drain in place due to the risk of spinal chord ischemia during aortic cross clamp or graft placement. The lumbar drain will remain in place for 24 hours after the procedure. Nurses should monitor output and CSF pressure hourly while the drain is open, reporting any output greater than 50 ml in 4 hours to the vascular team. Unless complications arise, the vascular team will clamp the lumbar drain POD 1. Nurses should continue neurovascular checks at routine frequency while the drain is clamped and monitor CSF pressure continuously until the drain is removed. If any neurovascular changes are noted, the nurse should increase the frequency of the neurovascular checks to Q 15 minutes and notify the vascular team immediately.•

APPLY YOUR KNOWLEDGE: CLINICAL CASE STUDY

Use the knowledge gained in this chapter and the following scenario to answer the questions below. When you are ready, check you answers on *p. 69.*

You land a patient from the OR following coronary artery bypass and mitral valve replacement. Your Help All places the patient on the monitor, applies suction to your chest tubes and OG tube, and sends a loaded ABG and CBC to the lab.

1. You expect to perform all of the following nursing interventions within the first hour EXCEPT:

    A. Assess chest tube output every 15 minutes and report output greater than 100 ml/hr

    B. Extubate the patient if the STAT ABG is within normal limits

    C. Shoot cardiac indices every hour and notify the provider if the index is less than 2

2. As you prepare to assess your patient, you reflect that patients receiving valve replacement surgery are at particularly high risk for:

    A. Acute Kidney Injury

    B. Hyperglycemia

    C. Ischemic Stroke

3. Your patient's ABG results with the following values: pH 7.36, PO2 96%; PaCO2 42; HCO3-24; K 2.9 PCV 28. Based on these values, you anticipate:

    A. Notifying the provider and using the ventilator protocol to increase the respiratory rate

    B. Notifying the provider and using the nursing algorithm to administer a unit of blood

    C. Notifying the provider and using the electrolyte replacement protocol to replete potatassium

5 MEDICAL CARDIOLOGY

Nursing Care of the Medical Cardiology Patient

Vanderbilt University Hospital CVICU cares for both cardiovascular surgery and medical cardiology service lines. This chapter will provide an introduction to common patient diagnoses cared for in the medical cardiology service line, including patients admitted for acute coronary syndrome, cardiogenic shock, and post-arrest patients. Current practices in heart failure management and cardiomyopathy will also be reviewed.

ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) is an umbrella term that covers a wide variety of clinical diagnoses, ranging from unstable angina to acute myocardial infarction. Patients with acute coronary syndromes present with signs and symptoms of myocardial ischemia such as chest pain; pain down one or both arms; shortness of breath; fatigue; nausea; vomiting; or anxiety. Women are more likely to present with atypical symptoms such as epigastric, back, or jaw pain. Notably, those with diabetes, postoperative patient, and the elderly may not present with typical signs and symptoms of ischemia and should be treated with a lower threshold for additional testing. Patients presenting with these symptoms should receive a STAT EKG and cardiac markers upon arrival to the facility or upon presentation of symptoms if already admitted.

Unstable Angina

Stable angina is defined by chest pain that follows a predictable pattern such as pain during exertion or times of stress. Stable angina may be relieved by rest or nitroglycerin. By contrast, unstable angina is characterized by chest pain at rest or unpredictable chest pain that may or may not be relieved with nitroglycerin. Patients with unstable angina may present with new or worsening chest pain symptoms, however upon further investigation, do not demonstrate increased troponin. Patients with unstable angina may or may not demonstrate transient EKG changes. Once the diagnosis of unstable angina has been determined, management focuses on resolving the acute ischemic pain and assessing whether the patient demonstrates acute hemodynamic compromise. Supplemental oxygen should be supplied to patients with oxygen saturation less than 94%. Unless contraindicated, patients will receive a beta blocker within 24 hours to decrease myocardial oxygen consumption. Patients unresponsive to sublingual nitroglycerin or patients

PRECEPTOR PEARLS

Troponin, indicative of myocardial injury, may not rise for 2-3 hours after the onset of myocardial infarction. A patient who presents soon after onset of chest pain may not yet demonstrate a troponin rise and should be monitored closely.

who are persistently hypertensive will receive a nitroglycerin drip. Following resolution of the acute incident, patients will remain in observation for at least 12 hours to trend cardiac markers and monitor for symptom recurrence.

Non-ST Elevation MI

Non-ST Elevation MI (NSTEMI) is characterized by new or worsening, unrelenting chest pain with positive biomarkers and either ST depression or T wave inversion on EKG. NSTEMI is most commonly caused by a non-occlusive plaque rupture that significantly alters blood flow to a portion of the myocardium. From a nursing standpoint, the care of the NSTEMI patient is much the same as the care of a patient with unstable angina. Patients who demonstrate hemodynamic compromise will likely undergo emergent coronary angiography for potential percutaneous coronary intervention (PCI). Patients who are hemodynamically stable will be admitted to an inpatient unit for medical management and undergo coronary angiography within 24 hours.

ST-Elevated MI

ST elevated MI is characterized by unrelenting chest pain with positive biomarkers and elevation of ST segment in two or more contiguous leads in a 12-lead EKG. An ST-elevated infarction, commonly known as a STEMI, is indicative of complete loss of blood flow distal to a ruptured coronary plaque. If blood flow is not restored, the portion of the myocardium supplied by the affected coronary artery will die. For this reason, the team will activate a STEMI alert and the cardiac cath lab immediately if a STEMI is suspected. The national standard for reperfusion, known as door-to-balloon time is 90 minutes. Depending on the affected vessel and the time to reperfusion, STEMI patients are more likely to become unstable than other ACS patients. For this reason, all STEMI patients post PCI will be admitted to the CVICU for close patient monitoring following intervention.

Care of the Post-Intervention Patient

When a diagnostic catheterization is warranted, the

BOX 5.1 *ACS Standards and Rationales*

Standard	Rationale
Acute Myocardial Infarction (AMI)	
Aspirin within 24 hours of arrival	Decreases clotting by inhibiting platelet aggregation, decreases vasoconstriction, and decreases the risk of death during AMI by 70%
Beta-Blocker within 24 hours of arrival	Given early (IV) in the course of AMI, decreases size of infarct, decreases the incidence of arrhythmias, and decreases the risk of cardiac rupture
PCI for ST elevation within 90 minutes of arrival	Early reperfusion decreases the size of the infarct
Aspirin prescribed at discharge	Decreases clotting by inhibiting platelet aggregation
Beta-Blocker prescribed at discharge	Decreases myocardial oxygen demand by decreasing heart rate and contractility, protects against sudden death by decreasing electrical impulse conduction, and decreases the risk of a recurrent MI
ACE Inhibitor/Angiotensin Receptor Blocker (ARB) prescribed for LV dysfunction (EF < 40%)	Preserves LV function by decreasing remodeling (formation of scar tissue)
Smoking cessation counseling provided for current smokers and those that have quit within 12 months of admission	Risk factor modification – nicotine promotes clotting, vasoconstriction, and increased myocardial oxygen demand

interventional cardiologist and the cardiac catheterization team will choose either a femoral or radial approach based on the patient's anatomy, catheterization urgency, and anticipated intervention. Radial artery catheterization has a lower risk profile post-catheterization and allows the patient to ambulate faster upon arrival to the unit.

Radial catheterizations will have a TR band (Figure 5.1) applied to the sheath exit site prior to arrival to the unit to maintain hemostasis. Upon arrival to the unit, the cardiac cath team will report the time that the TR band was applied as well as the time that the TR band may be deflated. At the appropriate time, the nurse should deflate air from the TR band at regular intervals until air is completely removed from the device. During this phase, nurses should monitor for signs of bleeding and re-inflate air as needed to maintain hemostasis. For more detailed information regarding TR band management, nurses should refer to the associated order set and TR band policy, available in PolicyTech.

Interventional cardiologists may utilize a variety closure devices following femoral artery sheath removal. Angioseal, an absorbable anchor and collagen plug, is the most common closure device utilized. Nurses may also see other brands of collagen plugs such as Mynx or Starclose. All collagen plus dissolve within 30-60 days following deployment, once the vessel has healed. For larger sheath sites, cardiologists may choose to use a suture-style closure device such as Perclose. Regardless of closure device, all patients following femoral sheath removal should remain flat for a minimum of two hours following diagnostic angiography and 6 hours following PCI. During this time, nurses should closely monitor for signs of oozing or hematoma formation. If bleeding or hematoma is noticed, nurses should apply pressure 1-2 cm above the puncture site and notify the provider team immediately. Less commonly, femoral sheath removal may result in a retroperitoneal

Figure 5.1 TR Band.

bleed, characterized by flank pain and hypotension unresponsive to fluid bolus. If a retroperitoneal bleed is suspected, notify the provider team immediately.

In addition to monitoring sheath removal sites, CVICU nurses can expect to administer and provide education about standard post-intervention medications. A complete list of ACS standards and their rationales can be found in Table 5.1. Patients who have received contrast in the lab will receive a bolus of 1 Liter of crystalloid following intervention to prevent contrast-induced nephropathy.

TARGETED TEMPERATURE MANAGEMENT

Targeted temperature management (TTM) has been investigated experimentally and used clinically for over 100 years to treat patients who do not respond neurologically following cardiopulmonary arrest. Recent literature has initiated changes to the protocol.

During protocol implementation, It is strongly recommended that a copy of the complete protocol be printed for refer-ence prior to initiation of TTM. A complete copy of the TTM protocol can be found on the CVICU web-site.

Initiation and Cooling

Based on a large body of evidence, VUMC has in-cluded specific inclusion and exclusion criteria to initiate TTM. A complete list of inclusion and exclu-sion criteria can be found in Box 5.2. Those patients who qualify for protocol initiation will be cooled with the Artic Sun Targeted Temperature Manage-ment System. Patients will be cooled to 33 degrees for 24 hours from their initial downtime.

During the cooling phase, patients will experience a number of physiologic changes. As patients cool to 33 degrees, they often become bradycardic. This may or may not result in hemodynamic compromise for the patient. In this population, hemodynamic instability is defined as hypotension requiring nore-pinephrine greater than 15 mcg/min or hypotension that requires the addition of a second inotrope or vasopressor. If hemodynamic instability presents in these patients, the protocol prompts providers to consider increasing the targeted temperature from 33 degrees to 36 degrees.

All TTM patients are paralyzed and sedated through-out the cooling phase of treatment. This is, in part, to prevent shivering that increases the patient's metabolic rate while the patient is hypothermic. All patients have continuous BIS monitoring to measure level of sedation, titrating sedation to achieve a BIS of 40-60. Train of Four (TOF), a peripheral nerve stimulator, is utilized to monitor the degree neuro-muscular blockade throughout the duration of the cooling phase. The TOF may be applied to the ulnar nerve (Figure 5.2) to elicit a thumb twitch or the fa-cial nerve to elicit an eyelid twitch (Figure 5.3). TOF is monitored every hour while a patient is receiving paralytics, titrating paralytics to achieve one to two twitches for every four stimuli given. The number of MA's required to elicit a response should be noted in the patient's chart. Paralytics and sedation should never be discontinued while a patient is cold.

Lastly, it is important to recognize that hypothermia causes electrolytes to shift across the cellular mem-brane, requiring judicious use of electrolyte replace-ment protocols. Potassium is not replaced unless the serum level falls below 2.8 mEq/L and should be checked every 6 hours for the first 24 hours.

Rewarming

The patient will be rewarmed at 0.25 degrees per hour 24 hours after the time of their arrest. During the rewarming phase of treatment, patients are like-ly to experience rebound hyperthermia. For this rea-

BOX 5.2 *Inclusion and Exclusion Criteria for TTM*

Inclusion Criteria	Exclusion Criteria
Arrest with primary cardiac etiology	Arrest from non-cardiac etiology
Ability to initiate TTM within 6-12 hours of ROSC	Patients with known terminal illness, bleeding issues, recent head trauma or a traumatic arrest
18 years or older	Pregnancy
Unresponsive and not following commands after ROSC	Awakens spontaneously with purposeful movement and abil-ity to follow commands
Estimated time from arrest to ROSC <60 minutes	Unwitnessed arrest with suspected prolonged downtime AND initial rhythm unshockable
	Initial temperature less than 34C

-son, the Artic Sun pads are kept on the patient for up to 72 hours after rewarming and acetaminophen Is scheduled to control for fever. Paralytic infusion will be discontinued when the patient reaches 36 degrees and sedation will be weaned when the TOF returns to four twitches. During any phase of treatment, shivering or suspicion of shivering may be treated by counter warming extremities with the Bair Hugger or with demerol.

In the event that a patient needs to be rewarmed outside of the protocol, the Withdrawal Guidelines When Discontinuation Determinations Are Made After Therapy Initiation algorithm can be found at the back of the protocol. Neuroprognotication is withheld until 72 hours after rewarming to allow adequate time for paralytics and sedation to be metabolized.

Management of the Heart Failure Patient

Heart failure is defined as the inability of the heart to provide adequate cardiac output to meet the metabolic demands of end organs. Heart failure is classified anatomically, physiologically, and by patient symptomology.

Heart Failure Classifications

Anatomically, heart failure is categorized as either left ventricular failure, right ventricular failure, or bi-ventricular failure. Patients with left ventricular failure classically present with pulmonary symptoms such as dyspnea on exertion, orthopnea, or pulmonary edema. Conversely, patients with right ventricular failure classically present with systemic symptoms such as ascites or peripheral edema. Patients with bi-ventricular failure will present with symptoms from both categories.

Physiologically, heart failure is categorized as systolic or diastolic hear failure based on the affected portion of the cardiac cycle. Systolic hear failure represents a problem with the heart's ability to pump and

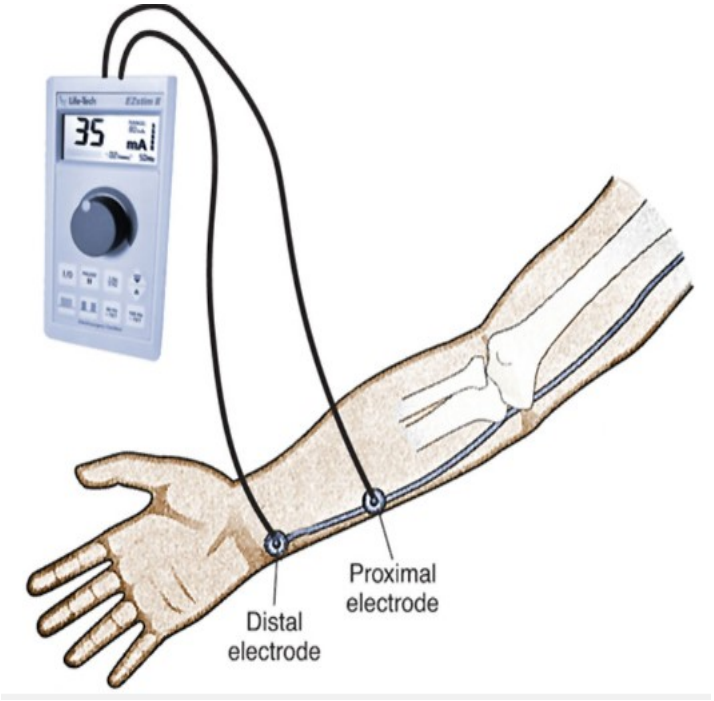

Figure 5.2 Placement of TOF electrodes along the ulnar nerve. From Wiegand, D.L. [Ed.]. [2017]. AACN procedure manual for high acuity, progressive, and critical care [7th ed.]. St. Louis: Elsevier.

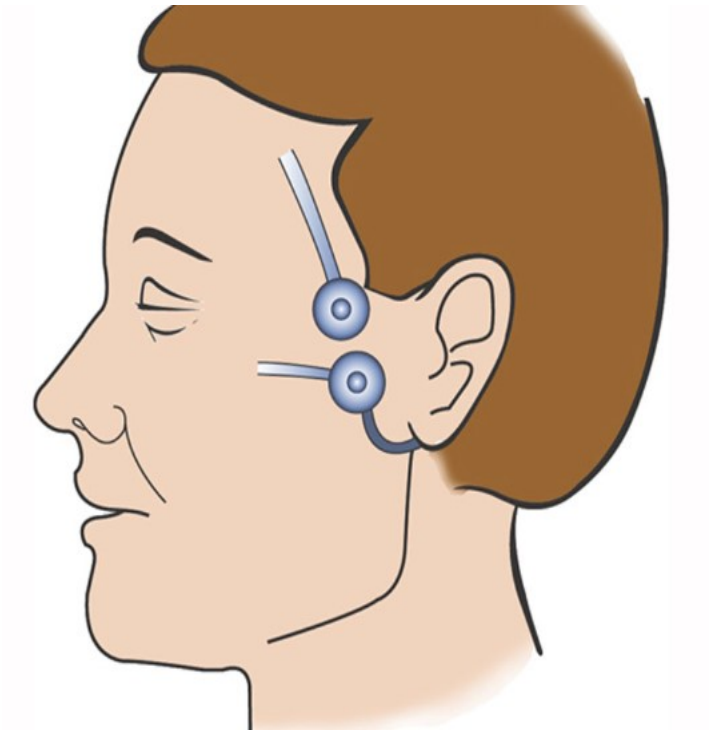

Figure 5.3 Placement of TOF electrodes along the facial nerve. From Wiegand, D.L. [Ed.]. [2017]. AACN procedure manual for high acuity, progressive, and critical care [7th ed.]. St. Louis: Elsevier.

may be caused by a variety of factors including but not limited to ischemic heart disease, valvular disease, or poorly controlled hypertension. Systolic heart failure is also referred to as heart failure with reduced ejection fraction (HFrEF) and is characterized by an ejection fraction (EF) less than or equal to 40%.. By contrast, diastolic heart failure represents a problem with the heart's ability to fill. Diastolic heart failure may be caused by a variety of factors, including but not limited to restrictive cardiomyopathies or valvular diseases. Diastolic heart failure is also referred to as heart failure with preserved ejection fraction (HFpEF) and is characterized by an EF greater than or equal to 50%.

Lastly, heart failure is described by the extent to which it affects the patient's functionality. One common scale used to quantify heart failure symptoms is the New York Heart Failure Association (NYHA) scale. This scale ranks patient symptomology from Class I (least severe) to Class IV (most severe). The American Heart Association prioritizes certain diagnostic tests and treatments based partly on a patient's NYHA scale. Providers may reference these recommendations in addition to the patient's complete clinical picture to escalate a treatment plan for a patient admitted with a diagnosis of heart failure.

Diagnostic Criteria

A diagnosis of heart failure is made based on comprehensive clinical exam and diagnostic evaluation. Serial weights and jugular vein distention may be used to establish and trend volume status in addition to a patient's subjective symptoms. A variety of tests and procedures may be ordered to evaluate heart function. Biomarkers such as B-type natriuretic peptide (BNP) as well as other natriuretic peptides have diagnostic value to establish the initial presence and degree of heart failure. In the acute setting, BNP will be drawn at minimum on admission and prior to discharge. Recent data also suggests that BNP may have value as a primary screening mechanism for heart failure (Yancy et al., 2017). Elevated troponin levels may indicate cardiac strain causing ischemia or necrosis (Yancy et al., 2017). In addition to biomarker testing, transthoracic echocardiography and chest x-ray as well as left and right heart catheterizations have diagnostic and prognostic value.

Treating Heart Failure

ACE inhibitors and beta blockers remain a mainstay of heart failure management and have been shown to decrease both morbidity and mortality in this vulnerable patient population (Yancy et. al., 2013). ACE inhibitors work to counteract the activation of the renin-angiotensin-aldosterone system (RAAS) triggered by pathophysiologic changes in heart failure. Beta blockers slow heart rate and decrease blood pressure, reducing strain on the heart and increasing filling time in patients with reduced ejection fraction. Patients who cannot tolerate ACE inhibitors may alternatively be prescribed Angiotensin Receptor Blockers (ARB) or a combination of hydralazine and isosorbide dinitrate.

As heart failure progresses, the addition of aldosterone antagonists and diuretics may be used to further counteract fluid retention in heart failure. ICD placement is recommended for patients with non-ischemic dilated cardiomyopathy and patients post MI with left bundle branch pattern and EF less than 35% to reduce the risk of sudden cardiac death (Yancy et. al., 2013). Patients with heart failure refractory to typical medical treatments may be candidates for IV inotropy, mechanical circulatory support, or heart transplantation.

CARIODMYOPATHY

Cardiomyopathy is a disease process in which the structure of the heart is changed in the absence of ischemic disease, congenital disease, or hypertensive remodeling (Cooper, Mckenna, and Yeon, 2019).

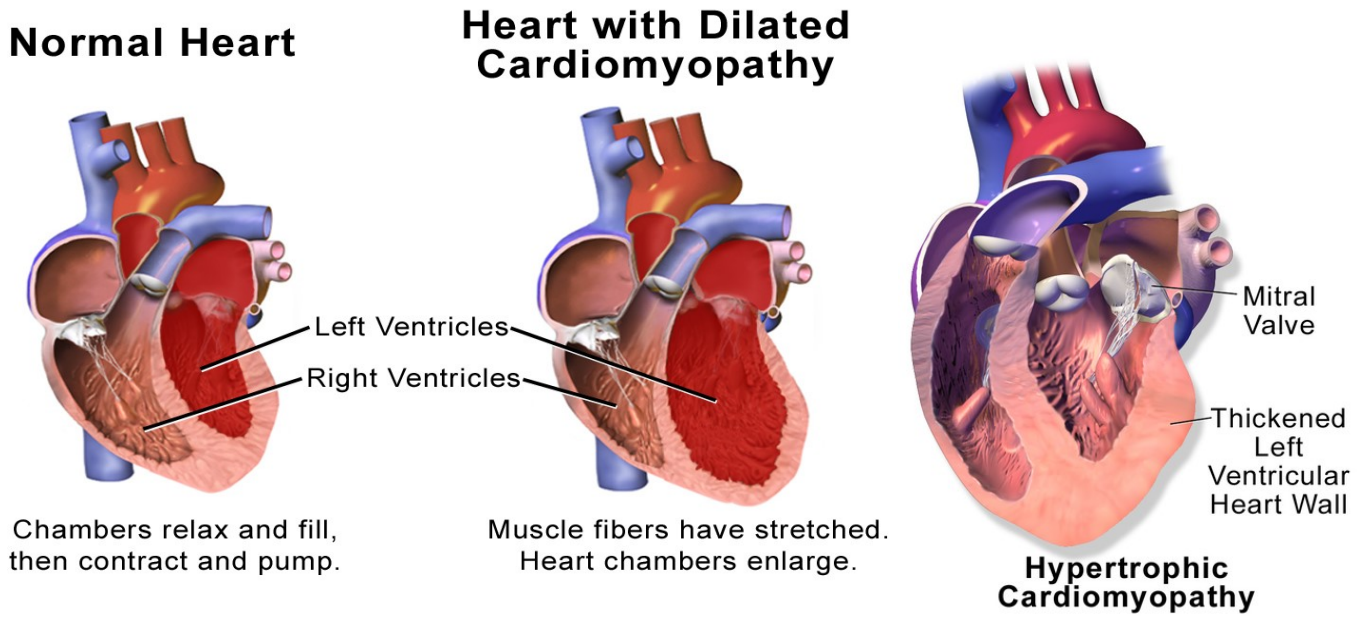

Figure 5.4 Cardiomyopathy. From: *Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2). DOI:10.15347/wjm/2014.010. ISSN 2002-4436. [CC BY 3.0 (https://creativecommons.org/licenses/by/3.0)]*

Cardiomyopathy is categorized as hypertrophic, dilated or restricted. Dilated cardiomyopathy demonstrates marked dilation, decreased wall thickness, and impaired contractility of the myocardium. By contrast, hypertrophic cardiomyopathy demonstrates thickening of the left ventricular wall that impedes diastolic filling. Hypertrophic cardiomyopathy may assume an obstructive morphology known as HOCM in which the ventricular septum obstructs the aortic outflow tract with contraction. Lastly, restrictive cardiomyopathy is characterized by impaired ventricular filling in the absence of hypertrophy or dilation. The treatment of cardiomyopathy addresses the type of dysfunction created and, if known, treat the root cause of the myopathy.

CARDIOGENIC SHOCK

Cardiogenic shock is a shock condition in which cardiac output is acutely unable to meet the oxygen demands of the end organs. Cardiogenic shock is characterized by high preload (CVP) as fluid congests the heart. As a compensatory mechanism, the peripheral vasculature constricts in response to low cardiac output, increasing SVR. Importantly, this compensatory mechanism increases oxygen demand on the heart and precludes a vicious cycle that further diminishes cardiac output. Patients may be admitted for cardiogenic shock following an acute event such as a myocardial infarction or, alternatively, as an acute exacerbation of chronic heart failure. Unlike patients with stable, chronic heart failure, however, the patient in cardiogenic shock experiences rapid changes to which the body is unaccustomed to adapting. For this reason, patients in cardiogenic shock may be particularly tenuous to manage. Patients suffering from cardiogenic shock appear hypotensive, tachypneic, tachycardic, and often cool to the extremities from hypoperfusion. During the acute phase of shock, patients may require vasopressors and inotropes to support blood pressure and cardiac output. Patients who do not respond adequately to these treatments may need temporary mechanical support to augment cardiac output. Treatable causes of cardiogenic shock such as acute valve abnormalities, infarction, or tamponade should be ruled out immediately.•

APPLY YOUR KNOWLEDGE: CLINICAL CASE STUDY

Use the knowledge gained in this chapter and the following scenario to answer the questions below. When you are ready, check you answers on *p. 69.*

You are caring for a patient admitted from the cath lab following a bare metal stent placement to the RCA. The patient has a dressing on his left groin access site following sheath removal.

1. The patient complains of back pain and wants to know when he can sit up in the chair. Based on his intervention, you inform him that he must lay flat for:

    A. 2 hours after sheath removal

    B. 4 hours after sheath removal

    C. 6 hours after sheath removal

2. Based on the area of his infarct, you anticipate that the patient will receive all of the following medications EXCEPT:

    A. ACE Inhibitors

    B. Aspirin

    C. Beta Blockers

3. Over the course of their admission, the patient develops increased fatigue and significant peripheral edema. Based on the clinical history, you suspect these changes may be due to:

    A. Bi-ventricular failure

    B. Left Ventricular failure

    C. Right ventricular failure

6 Advanced Therapies and Devices

Mechanical Support for Cardiac Compromise

Patients experiencing cardiac compromise may need mechanical support to augment their cardiac output. This chapter reviews a variety of mechanical assist devices utilized in the CVICU to improve cardiac output, either temporarily or as permanent cardiac support. An introduction to device mechanics, nursing sensitive actions, and basic troubleshooting will be reviewed with each section.

TEMPORARY PACEMAKERS

Temporary pacemakers are utilized to increase heart rate and may be inserted for conduction disorders such as heart blocks, rate disorders such as symptomatic bradycardia, or prophylaxis for post-surgical augmentation of cardiac output. In the CVICU, temporary pacing is executed most frequently through the use of epicardial wires placed directly on the myocardium during surgery. Alternatively, a patient may have pacing wires placed transvenously, threading either a temp-perm pacing wire or a pacing-capable PA catheter through the right atrium. In an emergency, transcutaneous pacing may be utilized.

Nursing Considerations

When assuming care of a patient with a pacemaker, nurses should note the mechanism of pacing (transvenous or epicardial), the part of the heart being paced (atrial or ventricular), and the mode of pacing (demand or asynchronous). In a demand pacing mode, the pacemaker will only pace if the patient's rhythm drops below a set threshold. Asynchronous pacing ignores the patient's underlying rhythm and should never be used on a patient with an viable intrinsic rhythm. Doing so may result in an R-on-T phenomenon that causing lethal arrhythmia.

All temporary pacing wires will be connected to a temporary pacing box. The replacement battery is taped to pacer at all times. Battery changes are done with shift change at 0700 every day and, for patients that are pacer dependent, two nurses must be present. The location of the back-up pacemaker should also be confirmed at shift change. If a patient is asystolic underneath their paced rhythm, a back up pacemaker box must be at the bedside with the same programmed settings as the primary pacemaker box.

Underlying rhythm should be checked daily by lowering the heart rate setting on the temporary pacemaker until the underlying rhythm is revealed. If, at any point, the patient does not tolerate lowering the rate setting, the nurse should reset the rate to the original setting and notify the provider team.. Underlying rhythm check is done only with a provider at the bedside for patients with life-threatening underlying rhythms or severe bradycardia. Due to the risk of malposition, patients with transvenous pacers who are pacer dependent are on strict bedrest unless a specific order is present to allow patient to chair or bedside commode.

Troubleshooting

The most common complications from temporary

BOX 6.1 *Pacemaker Malfunctions*

MALFUNCTION	CAUSES	INTERVENTIONS
Loss of Capture	Increased pacing thresholds due to: • Fluid status changes • Pericardial effusion • Electrolyte or metabolic disturbances • Tissue fibrosis Interruption in pacing system Dislodged/fractured lead Low Battery	Ensure connections secure Recheck pacing threshold; may need to increase output (mA) Turn patient Replace battery Replace pacemaker
Undersensing	Inadequate QRS signal MI Fibrosis Electrolyte disturbance Bundle Branch Block Fusion Beat Battery depletion Inappropriate sensitivity setting	Increase sensitivity (decrease mV) – allows pacemaker to more readily see intrinsic cardiac activity Correct underlying problem
Oversensing	Electromagnetic interference Mypotential inhibition T waves outside of the refractory period Dislodged or fractured lead Inappropriate sensitivity setting	Check pacemaker connections Decrease sensitivity (increase mV) – pacemaker is seeing too much

pacing generally fall into two categories: loss of capture and inappropriate pacemaker sensitivity. Loss of capture occurs when the electrical stimuli delivered by the pacemaker does not result in depolarization of the atria or the ventricle. Failure to capture is noted on EKG by the presence of pacemaker spikes without corresponding P-waves or QRS complex, depending on the area of the heart being paced. To fix this problem, the nurse must increase the electrical output from the pacemaker box.

By contrast, sensitivity describes the pacemaker's ability to detect intrinsic cardiac electrical activity. Therefore, inappropriate pacemaker sensitivity occurs when the pacemaker does not sense the heart's intrinsic electrical activity correctly. This appears on EKG as inappropriate pacemaker spikes. If the pacemaker is too sensitive (oversensing), the pacemaker will inhibit itself inappropriately and underpace the patient. If the pacemaker is not sensitive enough (undersensing), the pacemaker will overpace the patient. Both over and under sensing can be dangerous and require immediate intervention. Sensitivity is

adjusted by manipulating the millivolts (mV) on the pacemaker box. Increasing the millivolts will decrease sensitivity and decreasing the millivolts will increase sensitivity. A complete list of causes and interventions for loss of capture and inappropriate sensitivity can be found in Box 6.1.

INTRA AORTIC BALLOON PUMP

An intra-aortic balloon pump (IABP) is a device in-serted through a sheath in the femoral artery, or cutdown in the subclavian artery, that augments cardiac output, decreases afterload, and improves myocardial oxygenation. The balloon is situated in the aorta just distal to the left subclavian artery and proximal to the renal arteries. The balloon inflates during diastole to displace blood into the supply. The balloon deflates just prior to the next systole. By deflating immediately prior to the next systolic contraction, the balloon does not allow the aorta an opportunity to recoil, effectively decreasing afterload and myocardial oxygen demand. The mechanics of an IABP will provide approximately 1.5 L/min of additional cardiac output.

Nursing Considerations

As with any large sheath, nurses should closely monitor the site for bleeding and hematoma formation. Patients with a femorally-inserted IABP must remain on bedrest with the head of their bed elevated no more than 30 degrees. Nurses should monitor urine output every hour to ensure that the device has not migrated distally and obstructed the renal arteries.

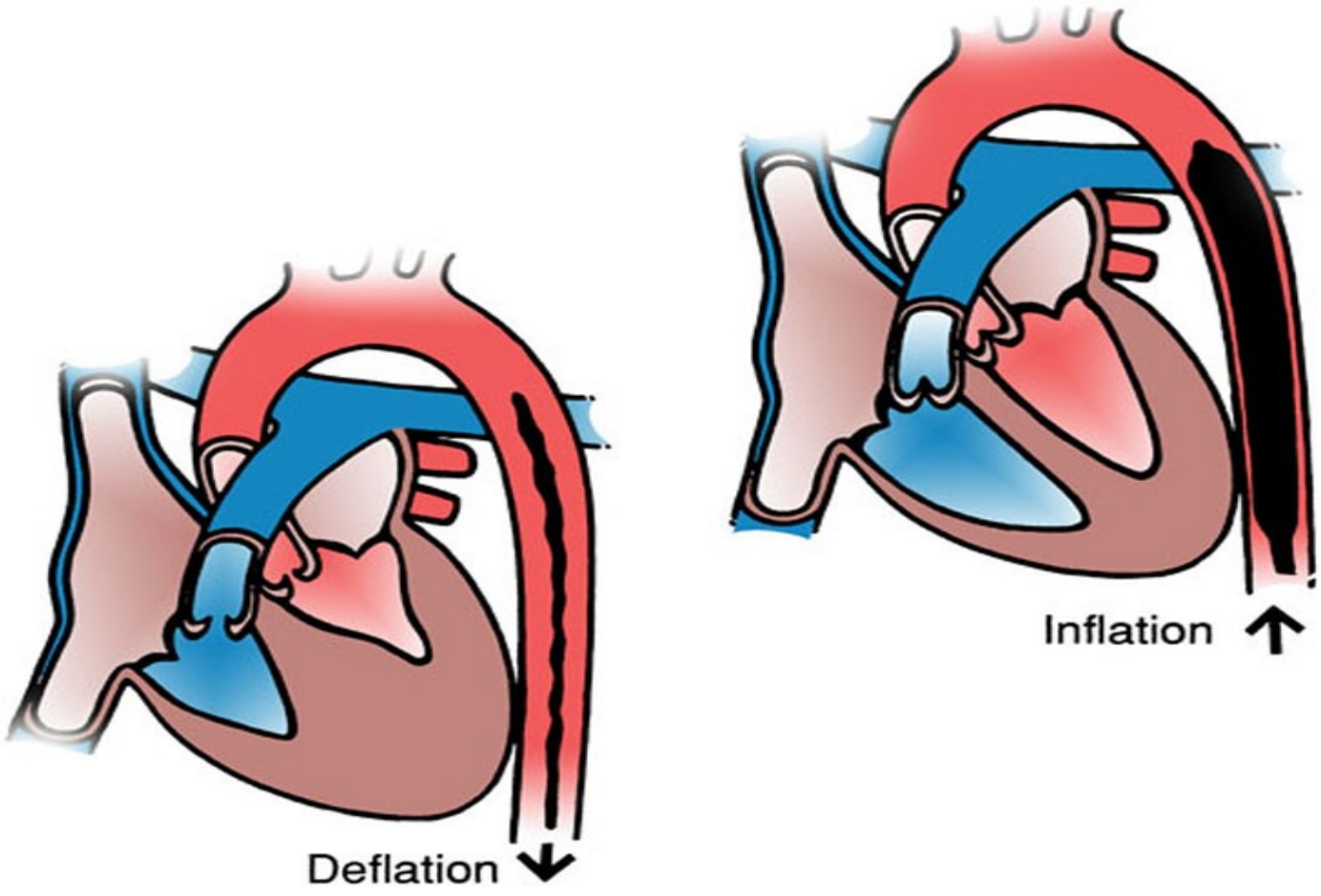

Figure 6.1 Intra Aortic Balloon Pump Inflation and Deflation. Counterpulsation. (Courtesy of MAQUET Cardiovascular, LLC, Wayne, NJ.) Used with permission (El Sevier, 2019).

Peripheral pulses, including upper extremity pulses, should be monitored at minimum every two hours if a NIRS device is in, without the frequency is hourly pulse checks. Loss of upper extremity pulses may indicate that the device has migrated too far into the aorta, obstructing the subclavian arteries. Loss of pedal pulses may indicate distal clot showering or obstruction of peripheral perfusion by the femoral sheath.

Regarding the balloon and console, nurses should assess the IABP waveforms every hour to ensure that the balloon is inflating and deflating appropriately within the cardiac cycle. When functioning ap-propriately, the balloon should begin augmenting diastole at the level of the dicrotic notch and deflate such that the assisted diastole is lower than the un-assisted diastole (Figure 6.2). Assisted and unassist-ed numbers are documented in eStar every two hours.

Nurses also should monitor the tubing that supplies helium to the balloon every hour. If blood is noted in the tubing, the nurse should immediately turn off the balloon pump and notify the provider team that the balloon has lost its integrity. Once a balloon con-sole is turned off, the balloon must be removed by a provider within 30 minutes to reduce the risk of clot formation, which may become dislodged during re-moval. The character of the helium lumen waveform may also provide additional information regarding balloon function and integrity.

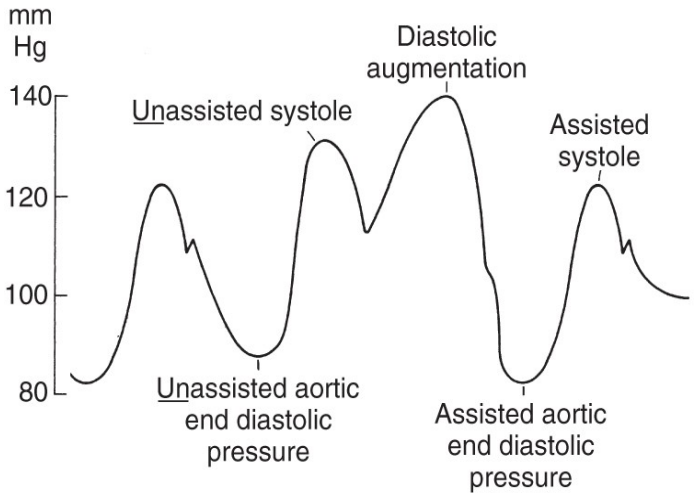

Figure 6.2 Correct IABP Timing. (Courtesy of MAQUET Cardiovascular, LLC, Wayne, NJ.) Used with permission (El Sevier 2019)

Troubleshooting

The most common and easily remedied complication from IABP therapy is improper timing. Nurses may adjust inflation and deflation intervals as needed to achieve correct timing of the IABP. Because an IABP uses predictive algorithms to time balloon inflation and deflation, balloons may have difficulty achieving correct timing in irregular rhythms. If a patient is in an irregular rhythm, the nurse should place the balloon in the arrhythmia tracking mode, denoted as "R -Track" on some consoles. This mode will automatically deflate the balloon in response to a premature QRS complex, ensuring that the balloon is deflated during systole.

In the event of an emergency, the console should be

BOX 6.2 *Common Maquet Alarms and Interventions*

ALARM	INTERVENTION
IABP Disconnected	Reconnect IABP tubing and hit "restart".
Autofill Failure	Check helium tank/tubing connections; ensure tank is open; check helium volume.
Augmentation Below Set Limit	Treat patient's hemodynamics as needed; If alarm limit inappropriate for patient, adjust limit. VUMC does not titrate therapy to the augmentation level.
Rapid Gas Loss	Observe for blood in tubing; If blood not observed, verify tubing connections and hit "restart".
Check IABP Catheter	Check catheter for kink; verify catheter placement in sheath to ensure balloon is not in sheath; Consider manually inflating and deflating the balloon.

placed in pressure trigger mode so that the IABP will automatically inflate and deflate in response to chest compressions. On Maquet consoles, nurses can change the trigger in semi-auto mode by adjusting the trigger menu on the console. If an Arrow console is in autopilot mode, the console algorithm will move directly into pressure trigger if the EKG cable is pulled from the console. Alternatively, nurses may place the Arrow console in Operator mode and change the trigger using the trigger menu.

The Arrow IABP console uses a tiered alarm system in which lower priority alarms result in a message-only display and higher priority alarms result in an audible alarm. The Arrow console will provide troubleshooting messages related to each alarm and the alarm will automatically reset once the inciting incident has been resolved. Gas loss and trigger loss alarms will default the console to "off" and should be investigated immediately.

The Maquet console uses only audible alarms. Each alarm will display an error message on the software screen. A summary of common Maquet alarms and associated interventions can be found in Box 6.2.

IMPELLA

An Impella is a temporary mechanical support device inserted through a tear-away sheath in the femoral artery. The distal end of the device is situated in the left ventricle, pulling blood from the left ventricle and ejecting it across the aortic valve into the systemic circulation (Figure 6.3). Depending on the specific Impella inserted, the device may provide 2.5L/min, 3.0L/min, or 5.0L/min of additional cardiac output. Less commonly, an Impella RP may be inserted through the femoral vein, sitting across the pulmonic valve with the distal tip in the pulmonary artery to provide right ventricular support.

Nursing Considerations

As with an IABP, the sheath, insertion site, and pulses distal to the insertion site should be monitored at routine intervals.

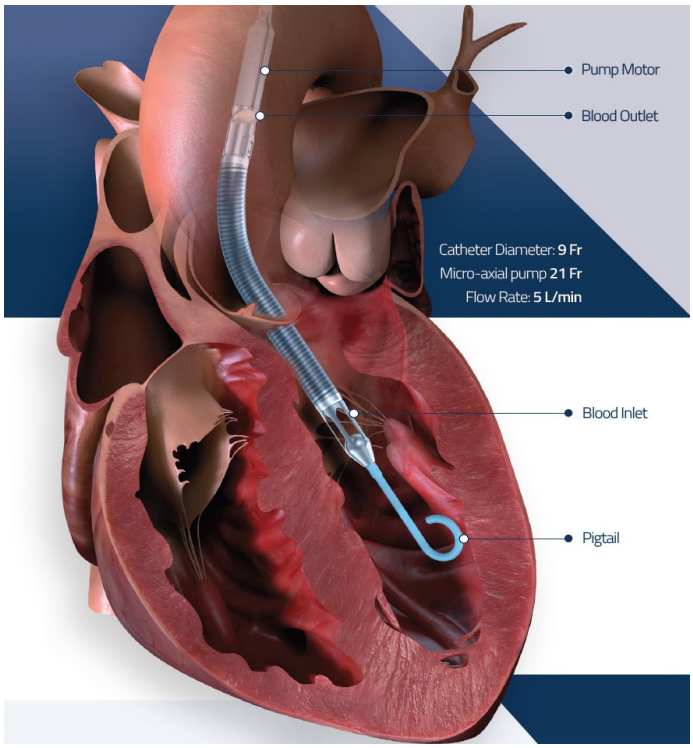

Figure 6.3 Impella for Left Ventricular Support

(From Abiomed®, Inc. [2018]. Impella 5.0® heart pump: For patients with AMI cardiogenic shock [product brochure]. ABIOMED, Inc.: Danvers, MA.)

An Impella requires a purge fluid of dextrose with heparin running through the device to prevent blood from entering the motor housing. For a left ventricular device, the standard purge fluid concentration is 12.5 units/ml. For a right ventricular device, the standard purge fluid concentration is 50 units/ml. Nurses should monitor and document the purge fluid flow rate and pressure hourly for the duration of support.

Nurses should also monitor the quality of the motor current every hour. Motor current is a measure of the energy intake of the Impella catheter motor. The energy intake varies with motor speed and the pressure difference between the inlet and outlet areas of the cannula. Monitoring motor current provides information about the catheter position rela-

tive to the aortic valve. When positioned correctly, with the inlet area in the ventricle and the outlet area in the aorta, the motor current is pulsatile because the pressure difference between the inlet and outlet areas changes with the cardiac cycle. When the inlet and outlet areas are on the same side of the aortic valve, the motor current will be dampened or flat because there is little or no pressure difference between the inlet and outlet areas. Impella RP may demonstrate a sporadic and dampened motor current since it crosses two valves on the venous side of the heart. This should be noted as a normal variation for right-sided support.

Lastly, nurses should document device flow rate and power level, commonly called P-level, hourly. The greater the P-level, the more support the device is providing. Impella P-levels range from P-2 to P-8.

TroubleshooWing

If a patient with an Impella CP codes, nurses should perform CPR and defibrillation as needed. AbioMed recommends turning down flow to P-2 during CPR. For the Impella 5.5 that enters a shock able rhythm, validate hemodynamics prior to initiating CPR. Nurses should note, however, that placement moni-toring and flow calculations will not be accurate dur-ing CPR. Once ROSC is achieved, device placement should be checked immediately with trans-thoracic echo.

The most common Impella alarm is a suction alarm. A suction alarm is most commonly caused by hypovolemia. If a suction alarm is noted, the nurse should turn the P-level down until the suction is re-leased and notify the team. If the CVP is below 10 mmHg, the nurse should also request an order for a bolus to fluid resuscitate the patient. However, if the patient is adequately fluid resuscitated, the right ventricle should be investigated via transthoracic echo.

In the event that an Impella position alarm sounds, nurses should confirm the position by assessing the motor current waveform against the Impella placement signal waveform as well as the placement markers on the Tuohy-Borst valve where the Impella exits the patient's groin. If the placement waveform or centimeter markings are noted to be different from the patient's baseline, the provider team should be notified immediately and Impella placement should be re-evaluated with a transthoracic echo. Placement alarms will not sound with an Impella RP device and placement should instead be verified with chest x-ray.

CENTRIMAG

Centrimag is a short-term centrifugal support device for patients in acute heart failure. The device decreases ventricular workload by bypassing the affected area of the heart, improving hemodynamic conditions to optimize myocardial recovery. Centrimag may be utilized to support left ventricular, right ventricular, or bi-ventricular failure. A centrimag can provide up to 9 L/min of support.

To support the left ventricle, a drainage cannula will be placed in the left atrium or ventricle with a return to the aorta. Right-sided support is used less frequently and is most commonly utilized to support right ventricular failure following transplant, transient pulmonary hypertension, or right-sided infarction. To support the right ventricle, a drainage cannula will be placed in the right atrium with a return cannula to the pulmonary artery.

Nursing Considerations

Nurses should monitor the cannulas to ensure that there are no kinks and that the cannulas have not moved. Centrimag cannulas are almost exclusively placed in the OR via direct cardiotomy and should be treated with extreme care. Patients with Centrimag support should be repositioned every two hours but perfusionist and/or provider must be present for any additional mobilization.

Device flow is controlled by the RPMs set by the provider as well as the patient's intrinsic preload

and afterload. Centrimag RPMs are typically set between 3,000-4,000 to achieve a flow of 3-4 L/min, depending on patient size and ventricular function. Pump flow and speed should be documented every hour for duration of therapy. In the instance of biventricular failure, nurses should closely monitor that right ventricular flow does not exceed left ventricular flow, which could cause pulmonary edema and worsen the patient's clinical condition.

Hypertension may decrease Centrimag flow rate at a set RPM. Nurses should titrate pressors to achieve a systemic pressure of approximately 90/70 with mean of 70-80 mmHg. Note that the arterial line in patients with a Centrimag will often appear flat due to non-pulsatile flow from the device.

Patients receiving Centrimag support are also at risk for clotting and hemolysis. Nurses should monitor coagulation labs, including PTT or ACT, at the discretion of the provider team. Nurses should also observe device tubing for fibrin strands or clot every hour, paying close attention to angles and connection points since these areas are particularly at risk for clot formation. To prevent clot formation around the flow probe, the flow probe should be moved at least once per shift. Any new or changed clot should be reported immediately to the provider team.

Troubleshooting

Because the Centrimag pump is preload-dependent, nurses should closely monitor CVP and work with their provider teams to maintain a CVP of 10-15 mmHg. If the pump does not have enough preload, the nurse may observe passive swinging of the drainage cannula, commonly referred to as chugging or chatter. This should be reported to providers immediately since the provider may decrease the device speed temporarily to resolve the issue until the patient's volume status can be addressed.

If the primary pump ceases to function, the nurse will need to press the staff assist button and switch the pump head to the back up controller immediately. When switching to a back up controller, the nurse should always clamp the outflow tubing first and then press and hold stop button on the primary controller. Stopping the pump without clamping will allow retrograde flow. Once the pump head is established in the back-up pump motor housing, the device should be turned on and RPMs increased to 10000 prior to unclamping the outflow tubing and returning the device to the set speed.

Although air entrainment is uncommon in a Centrimag device, it is always an emergency. If air is noted in the device, perfusion and the provider team should be notified immediately.

Extra-corporeal Membrane Oxygenation (ECMO)

Extra-Corporeal Membrane Oxygenation (ECMO) is a treatment platform used to support patients in the setting of severe heart and/or lung dysfunction. ECMO is used to support patients whose organ systems should recover such as those suffering from pneumonia, ARDS, Takotsubo cardiomyopathy, or acute rejection following heart or lung transplant. It can also be used as a bridge therapy for those in end-stage heart or lung disease to either durable mechanical support (VAD) or transplant. ECMO is not a curative therapy. It does, however, allow critically ill

PRECEPTOR PEARLS

At least three specialized tubing clamps should be hanging on the Centrimag and ECMO consoles at all times. In the rare instance that a nurse needs to clamp out the circuit to switch to the back-up controller or hand crank, these clamps will not harm the tubing integrity.

patients and their provider team time to explore their underlying disease differential to create a treatment plan.

There are two types of ECMO support. Veno-Arterial (VA) ECMO supports a patient's hemodynamics and oxygenation by bypassing both the cardiac and pulmonary circulation. By contrast, Veno-Venous (VV) ECMO supports a patient's oxygenation only. To cannulate a patient for ECMO, a large bore cannula is placed in a primary vein to drain blood into the ECMO circuit. A centrifugal pump pulls blood from the venous system using negative suction. The blood comes out of the centrifugal pump in a positive pressure that then pushes blood through an artificial lung (oxygenator). Inside the oxygenator, oxygen is picked up and CO2 is cleared just like in a patient's native lungs. Blood is then pushed back to the patient and reinfused through a cannula either into their venous system (VV ECMO) or to the arterial system (VA ECMO). ECMO always drains blood from a vein – where the blood is returned determines what kind of ECMO the patient is on and which organ system is being supported or bypassed.

Nursing Considerations

Well-supported patients on ECMO may seem very stable; however, they are in fact among the sickest patients in the hospital. As such, it's important to remember than an ECMO-trained nurse must be available to watch the patient at all times, even if the patient's primary nurses only anticipates being away from the bedside for a short period of time. Nurses should be very cautious with the cannulas, circuity and pump, encouraging families and other staff to avoid close proximity with pump components in order to protect the circuit.

Cannula site dressing changes are done with the same frequency and manner as central line dressing changes. It is important not to use alcohol-based products because these products will break down the polymers in the ECMO circuitry and cause it to become brittle. Central cannulation dressing changes are done per the provider's discretion.

All ECMO patients, including open chest central cannulation, should be repositioned at minimum every two hours to redistribute pressure and promote skin

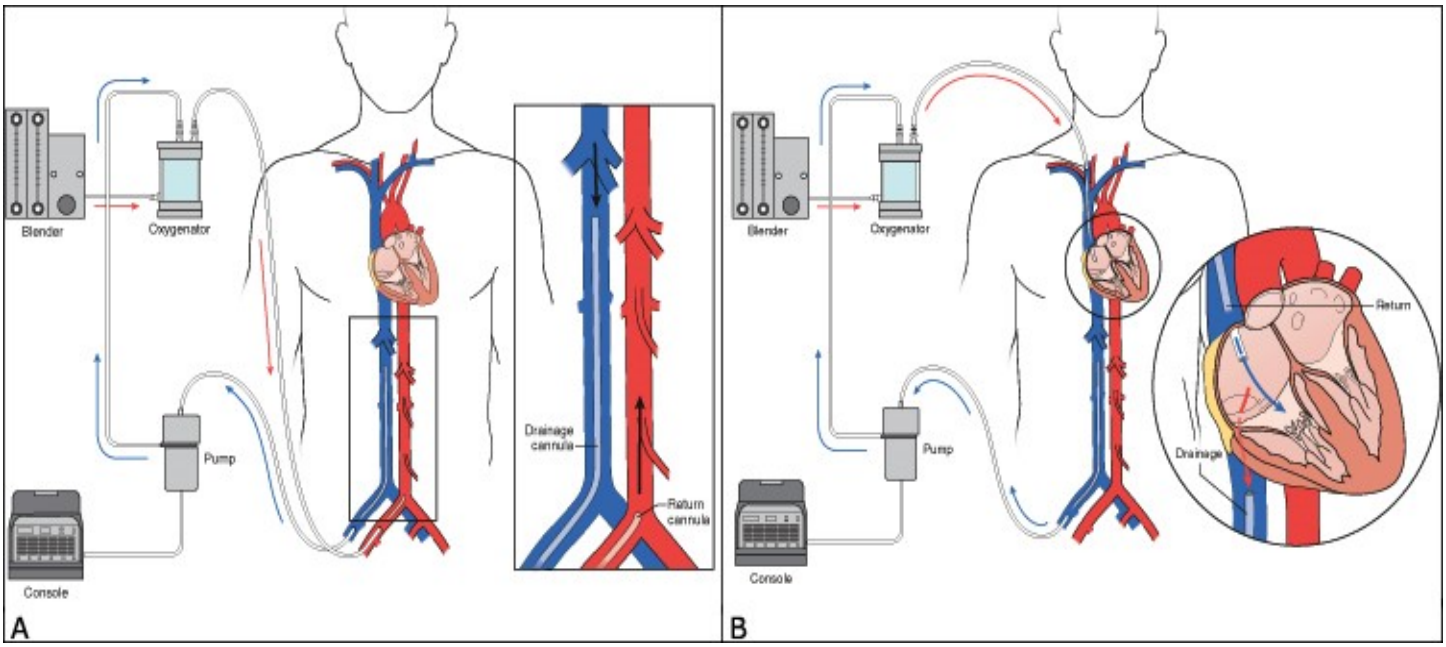

Figure 6.4 A, Peripheral venoarterial extracorporeal membrane oxygenation [ECMO] standard cannulation and circuit. B, Peripheral bicaval cannulation venovenous ECMO standard cannulation and circuit. (From Kaplan, J.A. and others [Eds.]. [2017]. Kaplan's cardiac anesthesia for cardiac and noncardiac surgery [7th ed.]. Philadelphia: Elsevier.)

integrity. As with Centrimag, additional mobility of ECMO patients should be conducted only with a perfusionist present. On rare occasions, a patient may be deemed too unstable for repositioning. In this case, and attending physician must enter a "Do Not Turn" order. This order must be renewed every 12 hours.

Troubleshooting

Air entrainment will cause the ECMO pump to clamp and forego supporting the patient. As little as 0.3 ml of air may cause the pump to clamp. To prevent air entrainment, it is important that any venous access lines including central lines, manifolds, peripheral IVs, and vascaths have a clave. Extreme caution should be used when flushing to ensure that there is no air in the syringe or IV lines. Due to the high risk of air entrainment, free flow tubing should be avoided on ECMO patients.

If something happens to cause the ECMO pump to stop, an ECMO patient will likely become unstable very quickly. The nurse should press the staff assist alarm and notify perfusion using the perfusion pager immediately, supporting the patient's hemodynamics and oxygenation status until additional help arrives. In the event that the ECMO pump has lost power, the nurse should clamp the circuit and transition pump head to hand crank housing and crank to desired hemodynamic or oxygenation goal. Any additional troubleshooting will be executed by the provider and perfusion team upon their arrival.

Left Ventricular Assist Device (LVAD)

Left ventricular assist devices (LVADs) provide durable mechanical support to the left ventricle that, unlike many other devices, allow patients to be discharged from the hospital. Vanderbilt supports three types of LVADs: Heartmate II, Heartmate III, and Heartware. All devices are approved as a bridge to heart transplant or as destination therapy.

The Heartmate II pump is an axial flow pump anastamosed to the apex of the left ventricle and the ascending aorta. Hearmate II speed ranges from 6,000-15000 rpms (average 9,600 rpms) to provide 3-10 L/min of support. The HMII operates in a fixed speed mode only, which maintains a continuous, constant flow. The initial speed is set in the operating room at approximately 6,000 rpm and gradually increased in increments of 200 rpm with TEE observation until the desired flow is achieved. Adjustments can be made by members of the VAD team using the System Monitor as needed, depending on the patient's clinical status. When the pump is functioning appropriately, an increase in speed (rpms) will increase flow while a decrease in speed (rpms) will decrease flow. As speed increases, device power will also increase.

By contrast, Heartmate III and HeartWare devices are centrifugal pumps anastamosed to the left ventricle and aorta. The Heartmate III speed ranges from 3,000-9,000 rpms (average between 4800 and 5800) to achieve a flow of 3-10 L/min. Unlike Heartmate II, Heartmate III has generates an artificial pulse every two seconds by dropping the speed 2000 rpms above and below the set speed. These speed changes will not be visible on the system monitor, but may be assessed on the patient's arterial waveform. HeartWare devices are fixed flow devices that deliver a constant speed between 1800

PRECEPTOR PEARLS

Strict air precautions should be utilized for Centrimag and ECMO devices. This means that no free-flow tubing is hung , claves on every access port, and even small bubbles of air must be meticulously removed from any medication prior to being given.

and 4,000 rpms (average 2400-3200 rpms) to achieve the desired flow.

Nursing Considerations

Because LVADs are continuous flow devices, narrow pulse pressures are a normal variation in this patient population. Peripheral pulses may not be palpable and non-invasive blood pressures will be assessed using a doppler. Vasoactive drips will be titrated to achieve mean arterial pressure (MAP) since systolic blood pressures are unreliable in VAD patients. With continuous flow devices, a pulse pressure greater than 30 mmHg may imply inadequate LV unloading, and the LVAD speed may need to be increased. If a pulse pressure greater than 30 mmHg is assessed, the provider team should be notified. All speed changes require an MD order. Device parameters may only be adjusted by an attending physician, heart failure attending, or surgeon.

All LVAD devices are preload dependent and afterload sensitive. As such, nurses should ensure that patients are adequately fluid resuscitated, maintaining a CVP between 10 and 15 mmHg. CVP greater than 20 should be reported to the provider team. To manage afterload, nurses should maintain a MAP of 60-80 mmHg. Doppled MAPs are correlated with the patient's arterial line at minimum every shift. When the patient's arterial line is removed, doppled MAPs are recorded every two hours. MAPs greater than 90 should be reported to the provider team since increased MAP may decrease device flows.

Pump flow, speed, power, and pulsatility index are assessed every hour and recorded in eStar. Pulsatility index (PI) is the magnitude of flow pulses through the pump. During left ventricular systole, the increase in ventricular pressure causes an increase in pump flow. Higher values indicate more ventricular filling and higher native pulsatility, indicating that the pump is providing less support to the ventricle. Lower values indicate less ventricular filling and lower native pulsatility, indicating that the pump is providing greater support to the left ventricle. In Heartmate devices, PI will be provided numerically on the device screen. In HeartWare devices, the PI is calculated by subtracting the trough of the pulsatility waveform from the peak of the pulsatility waveform on the device monitor.

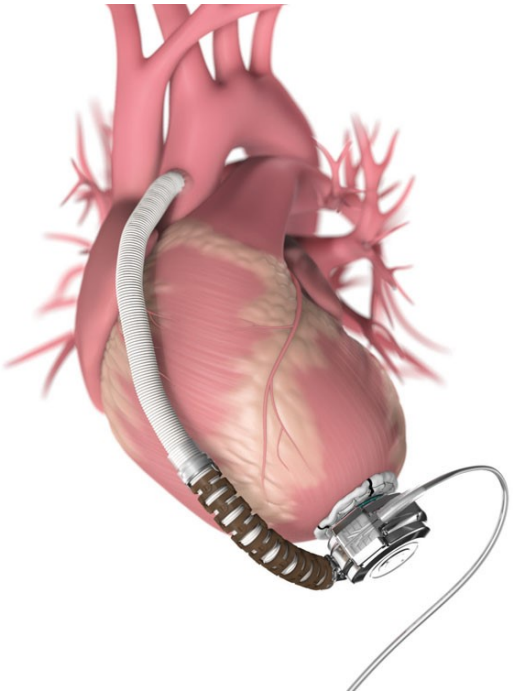

Figure 6.5 Implanted HeartWare® Ventricular Assist Device pump. (Courtesy of HeartWare, Inc., Framingham, MA.) (El Sevier, 2019)

Driveline infection is a major cause of morbidity and mortality in VAD patients. For this reason, meticulous driveline care is a nursing priority. As a standard, operative dressings remain in place for the first 72 hours. Gauze dressings are applied and changed daily on post-operative days three through seven. If site drainage is minimal and conversion is approved by a VAD Coordinator, Centurion dressings begin on POD 8. Centurion dressings are changed every three days and PRN on the implant admission. Nurses should note that Centurion dressing change frequency during readmissions may vary depending on the stage of healing, site appearance, and/or home dressing change schedules previously established by

the patients and their VAD Coordinators.. Centurion dressings are changed on any day that the patient showers irrespective of standard change schedules.

Troubleshooting

In the event of a cardiopulmonary arrest, chest compressions will not be performed without an order from an attending physician to decrease the risk of device dislodgement causing a surgical emergency. In the event of a cardiac arrest:, the nurse should first confirm that the power supply and system controller are operational. Next the nurse should defibrillate and use medications per ACLS protocol.

A suction event occurs when flows drop below 2.5 L/min and may be due to ventricular collapse or inflow occlusion. Ventricular collapse occurs when a continuous flow VAD attempts to pump more blood from the left ventricle than is available, resulting in considerable reduction in ventricular volume. Left ventricular collapse can be the result of clinical events affecting left ventricular preload, including hypovolemia (bleeding), right heart failure, arrhythmia or pulmonary embolus. An inflow occlusion occurs when the inflow cannula is obstructed, causing a suction condition. Temporary inflow obstruction may also occur as a result of surgical positioning, patient position or during straining (valsalva). If a patient experiences a suction event, the nurse should immediately notify the provider team, support the patient's hemodynamics, and anticipate treatment of the root cause of the event based on the patient's clinical picture. To provide the nurse and audible alert of a suction event, the nurse should ensure that the suction alarm is on at the beginning of each shift.

Other LVAD complications may include: PI events, pump thrombus, right ventricular failure, and arrhythmias. A PI event is characterized by a PI less than 2 with a correlative drop in device flows and may indicate that more cardiac support is indicated. Pump thrombus is characterized by decreased flows with a correlative increase in pump power and may indicate the need for further evaluation and thrombolytic therapy. Right ventricular failure in the LVAD patient presents similarly to right ventricular failure in the patient without mechanical circulatory support. However, if severe, right ventricular failure may result in decreased LVAD flow due to inadequate left ventricular preload. Arrhythmias are common in the LVAD patient population and should be treated using the same algorithms utilized in patients without mechanical circulatory support. Importantly, due to the nature of continuous flow devices, LVAD patients generally tolerate arrhythmias better than patients without a continuous flow device. Treatment type and urgency are determined by assessing patient clinical status. All new arrhythmias should be reported immediately to the provider team, regardless of the patient's clinical status.

TOTAL ARTIFICIAL HEART

Total artificial heart is a durable mechanical support device approved as a bridge to transplant for patients with biventricular failure, left ventricular thrombus, or malignant arrhythmias that preclude LVAD implantation. To implant the device, all four native valves and both ventricles are removed and replaced with two mechanical ventricles and four mechanical valves. Blood is moved through the de-

PRECEPTOR PEARLS

LVAD patients generally tolerate arrhythmias better than patients without continuous flow devices. Patient assessment is paramount to determine what intervention is most appropriate and the degree of urgency that this intervention requires.

-vice by inflating and deflating diaphragms within the ventricles via pneumatic drivelines that connect to the device console.

Syncardia recommends adjusting device parameters to achieve partial device fill and full ejection of blood from the device. Partial fill is defined as biventricular fill volumes of 50-60 ml for a 70 ml device, allowing the device to function effectively with normal variations in venous return to the heart. Full ejection is determined by observing a secondary rise in pressure, known as the full eject flag, on the pressure waveform (Figure 6.7, A). Full ejection ensures that clot does not form within the ventricles due to blood stasis. Providers may adjust the device beat rate and percent systole as well pneumatic drive and vacuum pressures to achieve these goals. An increase in beat rate, or decrease in vacuum pressure will decrease ventricular fill volume. An increase in pneumatic drive pressure will increase the force by which blood is ejected from the ventricles. Adjustment of device parameters from baseline should always be viewed as a temporizing measure and the patient's clinical picture must always be taken into account. As with LVADs, only attending physicians, heart failure attendings, or a surgeon may adjust device parameters.

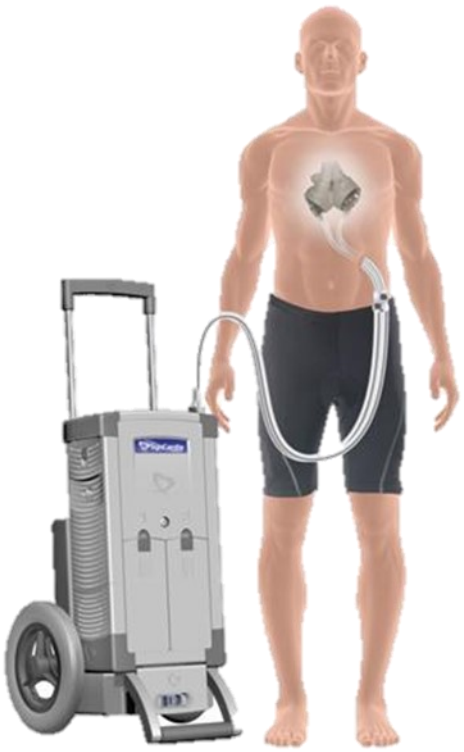

Figure 6.6 Total Artificial Heart. Used with permission. Syncardia, 2019.

Nursing Considerations

Because device implantation requires removal of the native ventricles, the patient will not have an QRS complex on EKG. For this reason, EKG is not monitored on patients with a Total Artificial Heart. Unlike LVADs and other continuous flow devices, the Total Artificial Heart is pulsatile and will produce a blood pressure with a pulse pressure within the normal range. For this reason, automatic blood pressure cuffs will produce a reliable blood pressure and doppled pressures are not required.

Nurses should assess device output and fill for both ventricles every at least every hour and PRN as patient condition warrants. Device beat rate; percent systole; left and right drive pressures; and left and right vacuum pressures are documented every hour. In addition to these parameters, nurses should monitor the pressure, flow, and average cardiac output waveforms hourly (Figure 6.7).

The pressure waveform measures the pressure of air in the pneumatic driveline required to generate systole. A normal pressure waveform will demonstrate a secondary rise in pressure, known as the full eject, flag (Figure 6.7, A). The flow waveform demonstrates the flow of air in L/min moving out of the pneumatic drivelines, deflating the ventricular diaphragms. A normal flow waveform should demonstrate consistent flow of air from the ventricles throughout diastole, depicted as bilateral parallel lines following the opening of the inflow valves into the heart (Figure 6.7, B). Any change from baseline should be reported to the provider team.

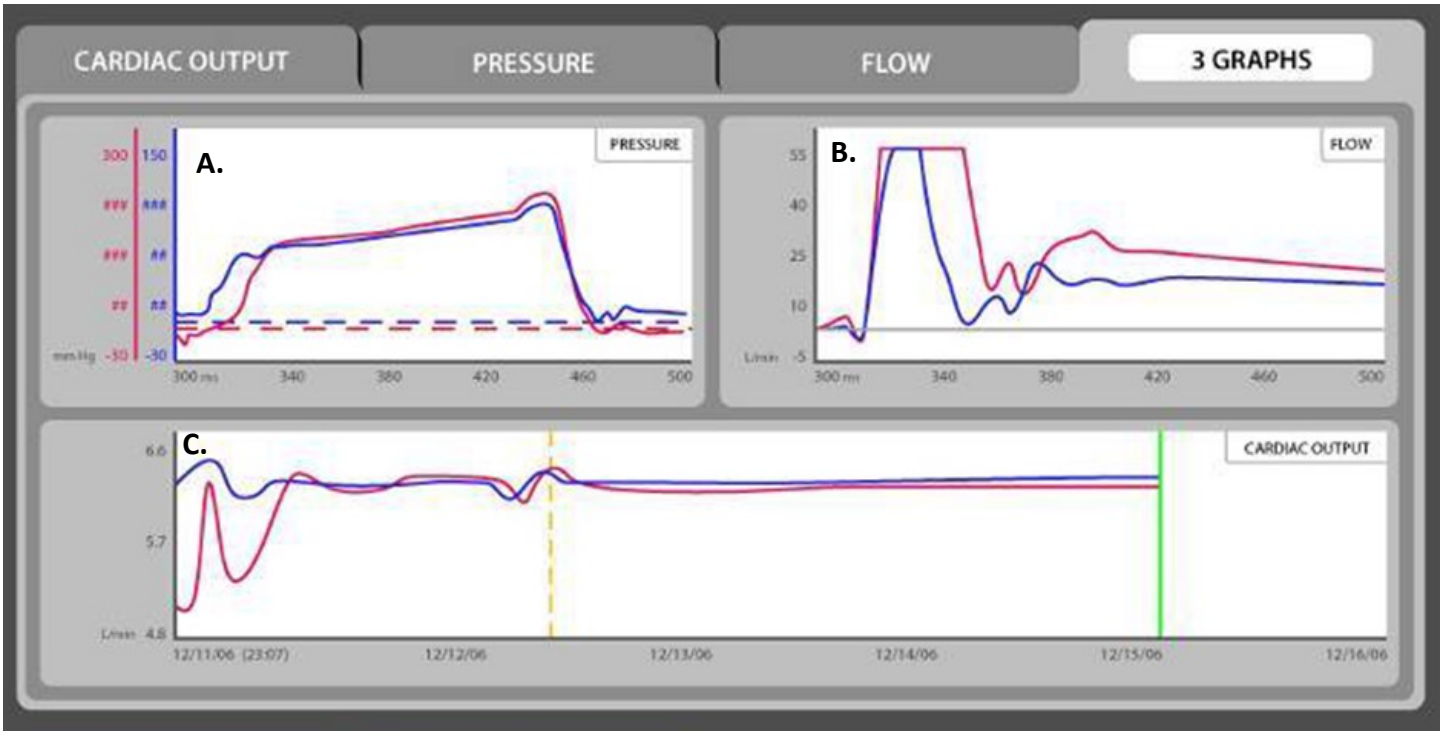

Figure 6.7 Expected Waveforms for Total Artificial Heart. Used with permission. Syncardia, 2019. A. The pressure waveform depicts the pressure within the pneumatic drivelines during systole. B. The flow waveform depicts the flow of air out of the pneumatic drivelines during diastole. C. The cardiac output graph depicts the average cardiac output over time.

Troubleshooting

In the event of an emergency, CPR, defibrillation, and ACLS protocols should not be utilized. If a patient with a Total Artificial Heart is found without a pulse, the nurse should first assess that the driveline is intact and plugged into the console. If this does not resolve the issue, the nurse should ensure that the console is on and functioning appropriately, switching to the back up console if required. If this does not resolve the issue, the nurse should switch to the hand pump, assist with respirations, and prepare to administer a 1L normal saline bolus. If a patient with a Total Artificial Heart is found unresponsive with a pulse, the nurse should support respirations and assess for non-cardiac etiologies of the change in level of consciousness such as hypoglycemia, stroke, or hypoxia.

As with other mechanical circulatory devices, the Total Artificial Heart is afterload sensitive. Systemic or pulmonary hypertension may prohibit the device from ejecting effectively. If a full eject flag is lost on the left pressure waveform, the nurse should first assess the patient's blood pressure and titrate medications to maintain a systolic blood pressure between 90 and 140 mmHg. The provider may choose to increase the pneumatic drive pressure to improve ejection until normotension is achieved. If a full eject flag is lost on the right pressure waveform, pulmonary vasodilators may be considered.

Cardiac tamponade is a major complication in the immediate postoperative phase following implantation. Cardiac tamponade is characterized by declining cardiac output on the cardiac output graph (Figure 6.7, C) that coincides with decreased fill volumes and diastolic flow. If the obstruction is in the inflow tract, the patient will present with a characteristic rise in CVP. If the obstruction is in the outflow tract, a loss of full eject may be observed. Suspicion of tamponade is a surgical emergency and should be reported immediately to the provider team. •

APPLY YOUR KNOWLEDGE: CLINICAL CASE STUDY

Use the knowledge gained in this chapter and the following scenario to answer the questions below. When you are ready, check you answers on *p. 70.*

You assume care of a patient with a left-sided Impella CP placed through the left femoral artery. The plan is to send this patient for a HeartMate III later in the shift. The patient has a PA cath displaying the following information: PA pressure 35/22 and CVP 5 mmHg.

1. While performing your morning assessment, the Impella sounds a suction alarm. Your next action is to:

    A. Turn off the console to prevent hemolysis

    B. Turn down the P-Level until the suction alarm resolves

    C. Press the staff assist button and wait for help to arrive

2. The suction alarm resolves. In response to this event, you anticipate the team will:

    A. Order a 500 ml fluid bolus

    B. Pull the Impella within 30 minutes of the console being turned off

    C. Increase epinephrine to achieve hemodynamic targets

3. Following implantation of the patient's HeartMate III, the patient enters in to V-Tach. The patient's MAPs drop from 65 to 40. Your next action is to:

    A. Defibrillate the patient at 200 J

    B. Initiate chest compressions at 120 bpm

    C. Push 1 mg epinephrine and obtain an order for lidocaine

Clinical Case Studies: Answers and Rationales

Chapter 2

1. A. Rationale: The normal CVP is 2-8 mmHg. A CVP of 18 is elevated and thus represents volume overload.
2. B. Rationale: Although an index of 1.7 may be normal for a patient in cardiogenic shock, this should be immediately reported to the provider because it is below the threshold for reportable values and requires treatment. While you may evaluate the patient's PVR and SVR, this is not the most urgent action.
3. A. Rationale: The patient is exhibiting respiratory acidosis. To relieve this condition, the minute ventilation should be increased by increasing the respiratory rate.

Chapter 3

1. B. Rationale: Milrionone will decrease afterload (SVR) and improve cardiac contractility, thus improving the cardiac index.
2. C. Rationale: Levophed is an alpha agonist and will improve blood pressure by increasing SVR.
3. A. Rationale: Epinephrine is an inotrope. Decreasing an inotrope will decrease the patient's cardiac index. An index below 2 should be reported to the provider team.

Chapter 4

1. B. Rationale: The goal to extubation for post-cardiotomy patients is 6 hours. The other answers reflect appropriate interventions for the first hour after surgery.
2. C. Rationale: All post-cardiotomy patients are at increased risk of hyperglycemia and acute kidney injury, however, valve replacement is associated with a higher risk of embolic, ischemic stroke.
3. C. Rationale: A potassium less than three requires notification of the provider team and treatment using the nurse-driven replacement protocol. The ABG does not require ventilatory intervention. Nurses must contact a provider to obtain an order for blood.

Chapter 5

1. C. Rationale: A patient must remain flat for 6 hours after sheath removal if an intervention is performed in the cath lab. If the patient does not receive intervention in the cath lab, they must remain flat for two hours after sheath removal

.

2. A. Rationale: Although the patient may have other indication to receive an ACE inhibitor, ACE inhibitors are indicated for left sided infarctions only in order to prevent ventricular remodeling. Aspirin and beta blockers are indicated for both right and left-sided infarctions.

3. C. Rationale: The symptoms described are indicative of right ventricular failure, which is consistent with the patient's presenting infarction.

Chapter 6

1. B. Rationale: If a suction alarm sounds, the nurse should immediately turn down the P-level to relieve the suction and support the patients hemodynamics as needed. The console should not be turned off unless therapy is about to be discontinued. While the nurse may choose to hit the staff assist button, immediate measures must be taken while the nurse is in the patient room.
2. A. Rationale: Suction alarms are most commonly caused by hypovolemia. The nurse should anticipate a fluid bolus is needed since the patient's CVP is less than 10 mmHg. Increased inotropic support would only be indicated if right ventricular failure was suspected. Turning off the console is not indicated for a suction alarm.
3. A. Rationale: Defibrillation is the appropriate intervention for V-Tach. CPR is contraindicated in patient with an LVAD, although all other components of ACLS protocol are followed. Per ACLS protocol, defibrillation is the priority intervention for V-Tach. Epinephrine is only indicated after the second shock in pulseless V-Tach.

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