CVICU Reference Guide

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Quick Reference

ADULT PERIOPERATIVE & CRITICAL CARE REFERENCE
BY RISHI KUMAR, MD - HTTPS://RK.MD

Abbreviations: alkaline phosphatase (**ALP**), blood pressure (**BP**), calcium channel blocker (**CCB**), dihydropyridine (**DHP**), dopamine (**DA**), fraction of inspired oxygen (**FiO2**), pralidoxime (**PAM**), pulmonary artery occlusion pressure (PAOP, ie "wedge"), pulmonary vascular resistance (**PVR**), seratonin (**5-HT**), systemic vascular resistance (**SVR**)

SCAN TO SHARE

	MEDICATION	BOLUS DOSE	INFUSION DOSE (mcg/kg/min)	RECEPTOR(S)	HR	BP	CO	SVR	PVR
Pressor	Angiotensin II	-	0.01 - 0.04	A-II type 1	↓	↑↑↑	varies	↑↑↑	↑↑
	Phenylephrine	50 - 100 mcg	0.1 - 0.7	α_1	↓	↑↑↑	varies	↑↑↑	↑↑
	Vasopressin	-	0.01-0.10 units/min	$V_1 > V_2$	↓	↑↑↑	↔/↓	↑↑↑	varies
Inopressor	Calcium Chloride	250 - 1000 mg	-	-	↔	↑	↑	↑	↑
	Dopamine	-	1 - 4	DA_1	↔	↓	↑	↓	
			5 - 10	β	↑	varies	↑↑	varies	
			10 - 20	α	↑↑	↑↑↑	↑	↑↑	↑
	Ephedrine	5 - 10 mg	-	$\alpha_1 > \beta_1$	↑	↑	↑	↑	
	Epinephrine	5 - 10 mcg	0.01 - 0.20	α and β	↑↑	↑↑	↑↑	varies	
	Norepinephrine	5 - 10 mcg	0.01 - 0.20	$\alpha_1 > \beta_1$	↑	↑↑	↔/↑	↑↑↑	↔
Ino-dilator	Dobutamine	-	2 - 20	β_1 and β_2	↑↑	varies	↑↑↑	↓	↓
	Isoproterenol	-	1 - 20 mcg/min	β_1 and β_2	↑↑↑	varies	↑↑↑	↓	
	Milrinone	25 - 50 mcg/kg	0.125 – 0.75	↑ cAMP	↑↑	varies	↑↑↑	↓↓	↓↓
β - Blocker	Esmolol	10 - 50 mg	10 - 50	β_1	↓↓	↓	↓	↔	
	Labetalol	5 - 20 mg	-	β_1, β_2 ,and α_1	↓	↓↓	↓↓	↓	
	Metoprolol	5 - 20 mg	-	β_1	↓↓	↓	↓	↔	
CCB	Clevidipine	-	1 - 16 mg/hr	DHP-CCB	↔/↑	↓↓	↔/↑	↓↓↓	
	Nicardipine	-	1 - 15 mg/hr	DHP-CCB	↔/↑	↓↓	↔/↑	↓↓↓	
	Diltiazem	5 - 20 mg	5 - 20 mg/hr	non-DHP-CCB	↔/↓	↓	↓		
	Verapamil	2.5 - 10 mg	-	non-DHP-CCB	↓	↓	↓		
Dilator	Hydralazine	5 - 20 mg	direct arterial vasodilator		↑	↓↓	↑	↓↓	↓
	Nitroglycerin	50 - 200 mcg	0.5 - 10	venodilation > vasodilation					
	Nitroprusside	50 - 200 mcg	0.5 - 10	vaso = venodilation, cyanide toxicity					
Hypnotic	Dexmedetomidine	0.5 - 1.0 mcg/kg (over 10 min)	0.2 - 1.4 mcg/kg/hr	$\alpha_2 >> \alpha_1$	anxiolytic, weak analgesic, preserves respiratory drive, bradycardia/hypoTN				
	Etomidate	0.2 - 0.3 mg/kg	-	$GABA_A$	↑	↓/↔	↔	↓/↔	
	Ketamine	1 - 2 mg/kg	1 - 5	NMDA	↑↑	↑↑	↑↑	↔/↑	
	Midazolam	0.1 - 0.3 mg/kg	0.2 - 1.5	$GABA_A$	↔/↑	↓	↔	↓	
	Propofol	1 - 2.5 mg/kg	25 - 200	$GABA_A$	↑	↓↓	↓/↔	↓↓	
Paralytic	Cisatracurium	0.15 mg/kg	1 - 5	nAChR	metabolized in plasma				
	Rocuronium	0.6 - 1.2 mg/kg	5 - 10	nAChR	sugammadex reversal (2, 4, 16 mg/kg)				
	Succinylcholine	1 mg/kg	-	nAChR	MH trigger: avoid in acute burns, denervation injury, prolonged immobility, severe infection				
Opioid [rel. potency]	Fentanyl [100x]	12.5 - 100 mcg	25 - 200 mcg/hr	long context-sensitive half-life					
	Hydromorphone [5-10x]	0.2 - 1 mg	0.5 - 4 mg/hr	H3G metabolite may cause neuroexcitation					
	Meperidine [0.1x]	10 - 25 mg	↓ shivering (kappa-opioid receptor activation)						
	Methadone [4x]	0.2 - 0.3 mg/kg	$t_{1/2}$ ~ 1-2 days, NMDA effects, ↑ QT_c						
	Morphine [1x]	2 - 5 mg	2 - 10 mg/hr	active M6G metabolite ↑ in renal dz					
	Remifentanil [100-300x]	1 mcg/kg	0.05 - 2	metabolized in plasma					
	Sufentanil [1000x]	0.25 - 2 mcg/kg	0.1 - 1 mcg/kg/hr						
Anti-emetic	Dexamethasone	4 mg	steroidal						
	Diphenhydramine	12.5 - 50 mg	H_1 receptor blockade, sedating						
	Haloperidol	1 - 5 mg	anti-DA, QT prolongation						
	Lorazepam	0.5 - 2.0 mg	GABA-ergic						
	Metoclopramide	5 - 10 mg	anti-DA, tardive dyskinesia						
	Olanzapine	2.5 - 10 mg PO	anti-DA						
	Ondansetron	4 - 8 mg	anti-5HT						
	Promethazine	6.25 - 25 mg	anti-histamine, sedating						
	Scopolamine	1 patch	anti-muscarinic, not MRI compatible						

Adrenergic receptors: **α1** (vasoconstriction), **α2** (presynaptic negative feedback ↓ sympathetic outflow), **β1** (↑ HR and contractility, ↑ renin), **β2** (bronchodilation, vasodilation in skeletal muscle, ↑ insulin secretion, glycogenolysis/gluconeogenesis)

	PARAMETER	EQUATION	NORMAL VALUES
Hemodynamic	Heart Rate (HR)	-	60-100
	Cardiac Output (CO)	HR x SV	4 - 8 L/min
	Cardiac Index (CI)	CO / BSA	2.5 - 4.0 L/min/m^2
	Stroke Volume (SV)	(CO / HR) x 1000	50 - 100 cc/beat
	Stroke Index (SI)	SV / BSA	40 - 60 cc/beat/m^2
	Shock Index	HR / SBP	0.5 - 0.7
Pressure	Systolic BP (SBP)	-	90 - 140 mmHg
	Diastolic BP (DBP)	-	60 - 90 mmHg
	Pulse Pressure (PP)	SBP - DBP	20 - 70 mmHg
	Mean Arterial (MAP)	(2/3 x DBP) + (1/3 x SBP)	60 - 100 mmHg
	Right Atrium (RAP) / CVP	-	2 - 6 mmHg
	Right Ventricle	-	15 - 25 mmHg / 0 - 8 mmHg
	Pulmonary Artery	-	15 - 25 mmHg / 8 - 15 mmHg
	PA Occlusion ("wedge")	-	6 - 12 mmHg
	Left Atrium	-	6 - 12 mmHg
	Coronary Perfusion Pressure (CPP)	DBP - LVEDP (to LV) or DBP - RVEDP (to RV)	60 - 80 mmHg
Resistance	SVR	[(MAP - CVP) / CO] x 80	750 - 1500 dynes-sec/cm^5
	SVRI	[(MAP - CVP) / CI] x 80	1500 - 2400 dynes-sec/cm^5
	PVR	[(mPAP - PAOP) / CO] x 80	50 - 200 dynes-sec/cm^5
	PVRI	[(mPAP - PAOP) / CI] x 80	50 - 225 dynes-sec/cm^5
Gas Exchange And Delivery	FiO2	fraction of inspired O_2 = 0.21 - 1.00 (often given as fraction, 0.21 = room air)	
	P_b	barometric pressure = 0 - 760 mmHg (760 mmHg at sea level)	
	Respiratory Quotient (RQ)	0.7 (only lipids), 0.8 (balanced diet), 1.0 (only carbs)	
	Alveolar Gas Equation	$P_AO_2 = F_IO_2 (P_b - P_{H2O}) - P_aCO_2 / RQ$ P_{H2O} ~47 mmHg at sea level	~100 mmHg at sea level
	P_ACO_2 Equation	$P_ACO_2 = (VCO_2 \times 0.863) / V_A$	
	P/F Ratio	P_aO_2 / F_IO_2	> 400
	A-a Gradient	$P_AO_2 - P_aO_2$	7 mmHg (young) 15 mmHg (elderly)
	Respiratory Index	RI = (P_AO_2 - PaO2) / P_aO_2	< 0.4
	Arterial O_2 Content	$C_aO_2 = (1.34 \times [Hb] \times [S_aO_2]) + (0.003 \times P_aO_2)$	17 - 20 cc O_2/100 cc
	Mixed Venous O_2 Content	$C_vO_2 = (1.34 \times [Hb] \times [S_vO_2]) + (0.003 \times P_vO_2)$	12 - 15 cc O_2/100 cc
	Arteriovenous Difference	$C_aO_2 - C_vO_2$	4 - 5 cc O_2/100 cc
	Fick O_2 Consumption	$VO_2 = 10 \times CO \times (C_aO_2 - C_vO_2]$	3.5 cc O_2/kg/min
	Fick Cardiac Output	$CO = VO_2 / [10 \times (C_aO_2 - C_vO_2)]$	4 - 8 L/min

Antidote	
Anticholinergic	Physostigmine 0.5 - 2 mg IV
Acetylcholinesterase	Atropine 0.5 mg q3m PRN, PAM 1-2 grams IV over 30 minutes
Benzodiazepine	Flumazenil 0.2 mg IV q1m PRN (seizure risk)
Beta-Blocker	Glucagon 5 mg IV then 2-5 mg/hr, insulin+glucose, pacing
CCB	Calcium repletion
Carbon Monoxide	O_2 therapy (hyperbaric O2 if AMS, MI, > 55 y/o, pregnant)
Cyanide	Cyanokit 5 g IV or 4-DMAP/nitrates + sodium thiosulfate
Digoxin	DigiFab 400 mg IV
Local Anesthetic	Intralipid 1.5 cc/kg then 15 cc/kg/hr, use 1 mcg/kg epi for ACLS
Malignant Hyperthermia	Stop succinylcholine/volatile anesthetic, dantrolene 2.5 mg/kg (up to 10 mg/kg), hyperventilate, ↑ oxygen, cool patient
Methemoglobinemia	Methylene blue 1-2 mg/kg IV (not in G6PD deficiency) or VitC
Opioids	Naloxone 40 mcg IV (to effect)

Poison Control: 1-800-222-1222; **MH Hotline**: 800-644-9737

Contact me with errors/suggestions at rishi@rk.md.

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Sources: *Miller's Anesthesia* (8th Edition), Vincent's *Textbook Of Critical Care* (7th Edition), Owen's *The Ventilator Book* (1st Edition), *Stoelting's Anesthesia And Co-Existing Disease* (7th Edition)

Please contact me at rishi@rk.md to report any errata or provide recommendations!

SCAN TO SHARE

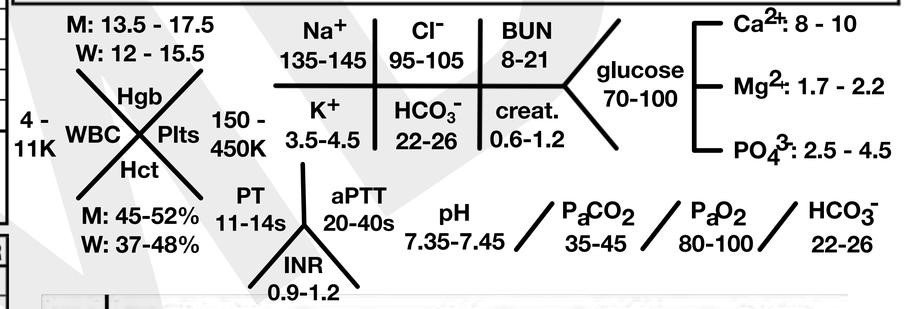
1° DISORDER	PH	P_aCO_2	$[HCO_3^-]$	COMPENSATION
AG/non-AG Metabolic Acidosis	↓	↓ (2°)	↓ (1°)	**$P_aCO_{2,\ expect} = 1.5\ [HCO_3^-] + 8 \pm 2$** If $P_aCO_{2,\ actual} < P_aCO_{2,expect}$ also **1° respiratory alkalosis** If $P_aCO_{2,\ actual} > P_aCO_{2,expect}$ also **1° respiratory acidosis**
AG Acidosis "Delta/Delta"	For AG metabolic acidosis, calculate **$\Delta AG\ /\ \Delta[HCO_3^-] = (AG - 12)\ /\ (24 - [HCO_3^-])$** if **< 0.8, non-AG acidosis**; if **> 2, metabolic alkalosis**			
Metabolic Alkalosis	↑	↑ (2°)	↑ (1°)	**$P_aCO_2 = 0.7 \times [HCO_3^-] + 20 \pm 5$** If $P_aCO_{2,\ actual} < P_aCO_{2,expect}$ also **1° respiratory alkalosis** If $P_aCO_{2,\ actual} > P_aCO_{2,expect}$ also **1° respiratory acidosis**
Respiratory Acidosis	↓	↑ (1°)	↑ (2°)	**For each ↑ 10 mmHg in P_aCO_2** Acute: ↑ $[HCO_3^-]$ 1 mmol/L and ↓ pH 0.08 Chronic: ↑ $[HCO_3^-]$ 4 mmol/L and ↓ pH 0.03
Respiratory Alkalosis	↑	↓ (1°)	↓ (2°)	**For each ↓ 10 mmHg in P_aCO_2** Acute: ↓ $[HCO_3^-]$ 2 mmol/L and ↑ pH 0.08 Chronic: ↓ $[HCO_3^-]$ 5 mmol/L and ↑ pH 0.03
Primary disorder (1°), compensation (2°); arrows relative to baseline P_aCO_2 ~ 40 mmHg and $[HCO_3^-]$ ~ 24 mEq/L				

Response	Action	Score
Eye ("four eyes")	Spontaneous opening	4
	Open to command	3
	Open to pain	2
	No opening	1
Motor ("V6 engine motor")	Follows commands	6
	Purposely movement to pain	5
	Withdraws from pain	4
	Abnormal flexion, decorticate	3
	Abnormal extension, decerebrate	2
	No motor response	1
Verbal ("the Jackson 5")	Oriented	5
	Confused but answers questions	4
	Inappropriate, words discernible	3
	Incomprehensible	2
	No verbal response	1

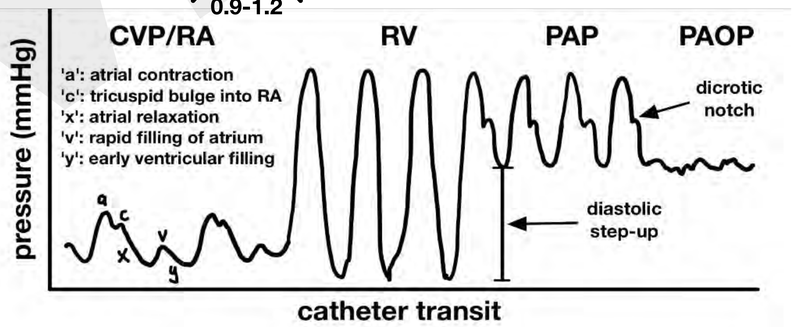
GCS score ranges from 3 to 15. If **intubated**, cannot perform verbal section. 'T' is affixed to score (ie, GCS 8T).
Brain injuries: mild (GCS 13-15), moderate (GCS 9-12), severe (GCS 3-8)

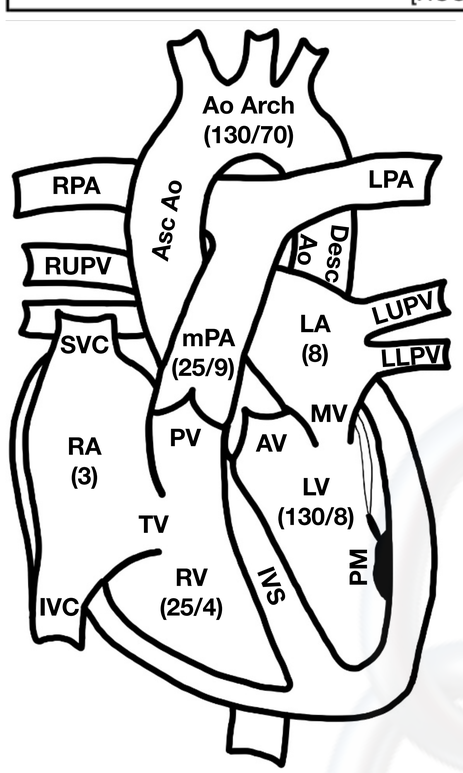
Score	Term	Description
+ 4	combative	violent, danger to self/staff
+ 3	very agitated	pulls tubes/catheters, aggressive
+ 2	agitated	frequent, non-purposeful movement
+ 1	restless	anxious but not aggressive
0	calm and alert	
- 1	drowsy	sustained awakening (> 10 sec) to voice
- 2	light sedation	briefly (< 10 sec) awakens to voice
- 3	moderate sedation	any movement (but no eye contact) to voice
- 4	deep sedation	no response to voice but responds to physical stimuli
- 5	unarousable	no response to verbal/physical stimuli

RASS goal typically -2 to 0. RASS > +2 should be assessed for delirium, anxiety, and/or pain.



	SBP	SVR	CVP	CO	PAP	PAOP	PVR
Cardiogenic Shock	↓	↑	↑	↓	↑	↑	↑
Cardiac Tamponade	↓	↑	↑	↓	↑	↑	↔
Hypovolemic Shock	↓	↑	↓	↓	↓	↓	↑
Neurogenic Shock	↓	↓	↓	↓	↓	↓	↓
Pulmonary Embolism	↓	↑	↑	↓	↑	↔/↓	↑
Septic Shock	↓	↓	↓	↑	↓	↓	↓

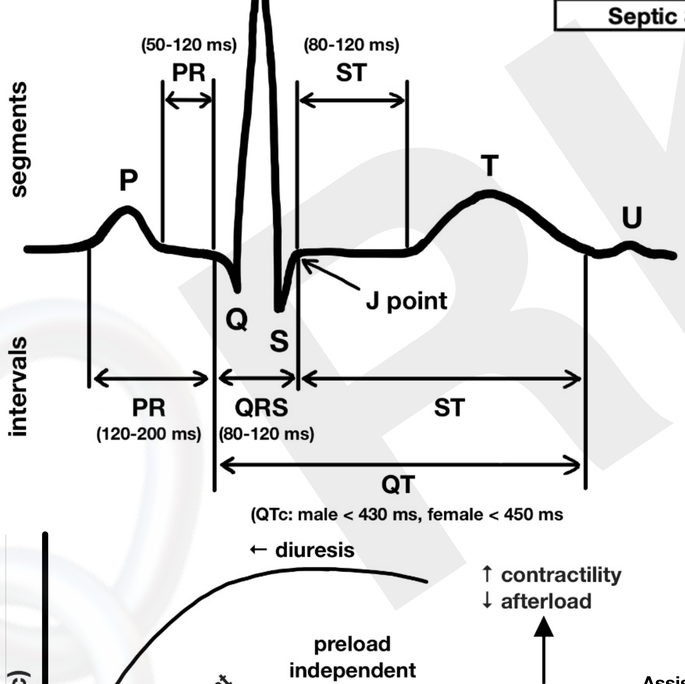


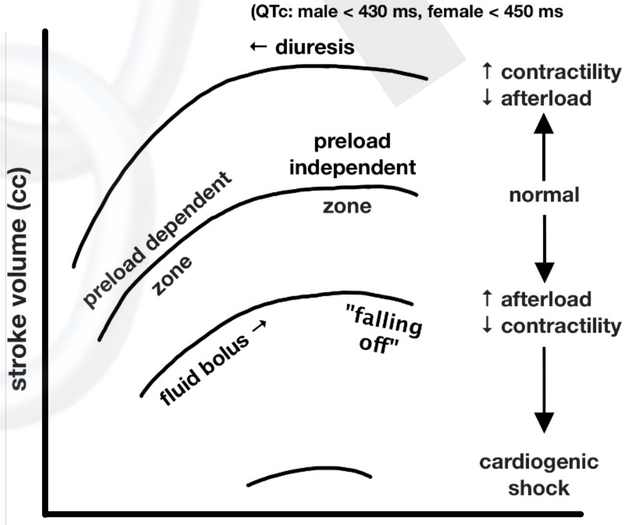


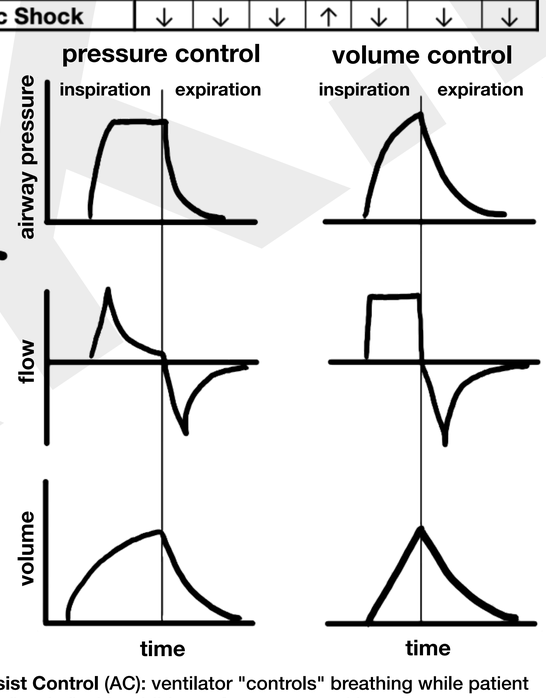
Chambers/Vessels (normal mean chamber pressure in mmHg)

Blood Flow: SVC/IVC → RA → RV → mPA → LA → LV → Asc Ao

Abbreviations: superior/inferior vena cava (**SVC/IVC**), right/left atrium (**RA/LA**), right/left ventricle (**RV/LV**), main/right/left pulmonary artery (**mPA/RPA/LPA**), right upper/left upper/left lower pulmonary vein (**RUPV/LUPV/LLPV**), tricuspid/pulmonic/mitral/aortic valve (**TV/PV/MV/AV**), interventricular septum (**IVS**), papillary muscle (**PM**), aorta (**Ao**)







Assist Control (AC): ventilator "controls" breathing while patient can "assist" with additional, fully supported breaths
1.) Volume Control (AC-VC): AC mode with set tidal volume (V_T). Driving pressure will depend on compliance. PEEP, F_iO_2, RR also set (mode guarantees a minute ventilation).
2.) Pressure Control (AC-PC): AC mode with set inspiratory pressure. V_T based on compliance. PEEP, F_iO_2, RR adjustable.
3.) Synchronized Intermittent Mandatory Ventilation (SIMV): spontaneous breaths are NOT fully supported (they get what they can pull). Therefore, V_T can vary
4.) Pressure Support (PS): spontaneous breaths receive an inspiratory pressure support. PEEP and F_iO_2 can be adjusted.
5.) Spontaneous Breathing Trial (SBT): typically performed on "minimals" (5 mmHg of inspiratory pressure on top of 5 mmHg PEEP) with minimal oxygen (F_iO_2 < 0.4) to assess pulmonary mechanics, ABG, etc. to determine feasibility for extubation.

	CAUSES	SYMPTOMS	TREATMENT
↑ Na (> 145)	dehydration, ↓ thirst, DI, GI losses, osmotic diuresis, Na overload	AMS, weakness, lethargy, **seizures**, **coma**	correct free H_2O deficit (FWD) (~10 mEq/24 hrs if chronic, 1-2 mEq/hr if acute)
↓ Na (< 136)	CHF, cirrhosis, nephrosis, SIADH, diuretics, salt wasting, polydipsia	headache, lethargy, nausea, **seizures**, **coma**	H_2O restriction, If CNS symptoms: hypertonic saline (↑ Na < 10-12 mEq/24 hrs)
↑ K (> 5)	K-sparing diuretics, acidosis, ↓ Na, trauma, hemolysis, rhabdo, tumor lysis, ↓ aldosterone	abdominal pain, confusion, paralysis, **arrhythmias**, peaked T wave, prolonged PR	calcium, dextrose/insulin, bicarbonate, diuretics, Kayexalate, Lokelma, dialysis
↓ K (< 3.5)	↓ intake, diuretics, GI losses (vomiting, diarrhea, gastric suctioning)	lethargy, weakness, **arrhythmias**, ileus, U wave, ↑ QT	oral/IV K repletion
↑ Ca (> 10.4)	↑ PTH, granulomatous dz, ↑ Vit D, malignancy	renal stones, abdominal pain, AMS, ↓ QT	fluids, loop diuretics, bisphosphonates
↓ Ca (< 8.8)	poor intake, ↓ Vit D, ↓ PTH, ↑ Phos, citrate, pancreatitis	tetany, seiures, ↑ QT, Chvostek/Trousseau signs	oral/IV Ca repletion
↑ Mg (> 2.3)	↑ Mg intake (antacids/laxatives), CKD, Addison's dz	↓ DTRs, sedation, paralysis, weakness, arrhythmias, coma	stop Mg repletion, fluids, loop diuretics, dialysis
↓ Mg (< 1.6)	GI losses (pancreatitis, diarrhea, PPIs) and renal losses (diuretics, fluids, uncontrolled DM, alcoholism, ↑ Ca, resolved ATN)	weakness, hyperreflexia, arrhythmias, refractory hypokalemia	oral/IV Mg repletion
↑ Phos (> 4.5)	AKI/CKD, tumor lysis, hemolysis, rhabdo, bisphosphonates, laxatives	paresthesias, **arrhythmias**, muscle cramps, perioral tingling, ↓ Ca, renal stones	limit Phos intake, calcium acetate, sevelamer, fluids/diuretics, dialysis
↓ Phos (< 2.5)	malnutrition, refeeding, insulin, fluids/diuretics, DKA, GI losses (vomiting, diarrhea, gastric suctioning), hyperparathyroidism	muscular **weakness**, respiratory failure, hemolysis, ↓ inotropy, AMS	oral/IV Phos repletion

Cardiac Meds

CARDIAC MEDICATIONS

A NURSE DOSE CHEAT SHEET

RECEPTORS

- There are many different receptors that influence different systems throughout the body.
- When activated (with agonists) or blocked (with antagonists) these receptors cause different things to happen throughout the body.
- Our main focus, the heart, is home to Beta1 receptors.
- When activated these receptors cause increase heart rate (chronotropy) and increased squeeze (inotropy).
- When blocked, the inverse happens and a decrease in rate and squeeze are observed.

BUT WHAT ABOUT ALPHA RECEPTORS?

- While Alpha receptors do not directly affect the heart they can indirectly affect cardiac function!
- Alpha1 receptors are located within the vasculature (among other places) and cause smooth muscle contraction when stimulated, i.e VASOCONSTRICTION.
- With increased vasoconstriction comes increased BP and increased afterload that the heart must work against to push blood forward.

MEDICATION CATEGORIES

INOPRESSORS

- Inopressors are medications that cause increased vasoconstriction through alpha 1 stimulation while also causing increased heart rate and squeeze via beta 1 stimulation.
- The three main medications we use with this property are Levophed, Epinephrine, and Dopamine.

PRESSORS

Are very similar to inopressors. The main difference is there is no effect on beta receptors and no direct effect on the function of the heart.

INODILATORS

- Inodilators are medications that increase inotropy (cardiac squeeze) while also causing arterial vasodilation.
- This allows for a harder squeeze against less resistance causing increased cardiac output.
- These are commonly refered to as chemical balloon pumps as they achieve the same therapeutic affects as a balloon pump.
- These medications are used to treat heart failure and some patients even have them infused at home.

CONTINUED

BETA BLOCKERS

- Beta blockers block the effects of Beta receptors by blocking receptor binding.
- They can cause decrease in blood pressure and heart rate.
- There are different classes of beta blockers that range from working systemically to being cardiac specific.

CALCIUM CHANNEL BLOCKER

- Calcium channel blockers disrupt the movement of calcium via calcium channels.
- They are used to control blood pressure and heart rate.
- Different classes affect different systems.
- Cardiac vs Systemic Vasculature
- You should always monitor for hypotension and bradycardia with these drugs.

AFTERLOAD REDUCERS

- Afterload reducers reduce the pressure needed to push blood from the heart through the aortic valve and into the aorta.
- There are many different classes of afterload reducers and many drugs we have already talked about reduce afterload in some way.
- Reduce in afterload comes with reduction in Systemic vasculature resistance and subsequently BP (usually).
- Decreasing afterload decreases stress on a sick heart.
- Need to monitor for hypotension and rebound tachycardia.

Medication	Receptors	Dose	Considerations
INOPRESSORS			
Levophed	alpha, beta-1	0.1 - 1 mcg/kg/min	More alpha than beta receptor activation. Has the potential to cause dysthymias at high doses.
Epinephrine	alpha, beta-1	0.1 - 0.5 mcg/kg/min	More beta than alpha receptor activation. Used in bradycardia or decreased cardiac output.
Dopamine	Dopamine, alpha, beta-1	• 1 - 5 mcg/kg/min • 5 - 10 mcg/kg/min • >10 mcg/kg/min	• (1-5)Dopamine receptors cause inotropy • (5-10)Beta receptors cause more inotropy and increase in HR • (>10)alpha stim cause increase vasoconstriction. Moderate increase in HR.
PRESSORS			
Vasopressin	V1, V2	0.02 - 0.04 units/min	• V1 receptors cause smooth muscle contraction in the vasculature. • V2 receptors in the kidney cause water retention when stimulated
Phenylephrine	alpha	0.5 - 1.5 mcg/kg/min	Pure alpha agonist. Cirrhosis patients may require increased dose.
INODILATORS			
Dobutamine	Beta-1, Beta-2	1 - 20 mcg/kg/min	Beta 2 stimulation can cause vasodilation and decrease in afterload (hypotension). Causes an increase in myocardial demand and O2 consumption.
Milrinone	Phosphodiasterase inhibitor	0.25 - 0.5 mcg/kg/min Titrated in 0.125 increments	Great for patients on beta blockers since beta receptors are not required for effect. Watch for hypotension

BETA BLOCKERS

Metoprolol	Cardioselective Beta-1 Blocker	IV push dose of 5mg slow. Can repeat for a total of 15mg	• Used for tachycardia and sometimes afib • Can cause hypotension
Labetalol	Beta-1,2 and Alpha Blocker	IV Push up to 20mg slow	• Used primarily for hypertension. • Contraindicated for patients with asthma.
Esmolol	Cardioselective Beta-1 Blocker	Continuous drip between 50 - 200 mcg/kg/min May require initial bolus	• Used to treat stable SVT and hypertension. • Monitor for hypotension and bradycardia.

CALCIUM CHANNEL BLOCKERS

Diltiazem	Calcium Channel Blocker	Drip starts at 5 mg/hr and can be titrated to a max of 15 mg/hr	• Monitor for hypotension and bradycardia. • Somewhat cardioselective.
Nicardipine	Calcium Channel Blocker	Drip starts at 5 mg/hr and can be titrated to a max of 15 mg/hr	• Monitor for hypotension and bradycardia. • Mostly systemic.
Clevidipine	Calcium Channel Blocker	Drip starts at 1-2 mg/hr and is doubled every 2 min until BP approaches target. Max around 21 mg/hr	• Monitor for hypotension and bradycardia. • Has white lipid look like propofol

AFTERLOAD REDUCERS

Hydralazine	vasodilator in arterioles	10 - 20 mg IV	• Effects are often unpredictable and can be prolonged. • Works mainly from dilation of arterioles
Nitroglycerin (gtt)	venous vasodilator	5 to 100 mcg/min as IV infusion	• Can decrease preload as well as afterload. • Used in congestive HF, hypertensive emergency and MI.
Nitroprusside	systemic vasodilator	0.3 mcg/kg/min; evaluate BP for at least 5 minutes before titrating to a higher or lower dose to achieve desired BP	• Can cause cyanide toxicity. • Infusion duration should be as short as possible and generally not exceed 2 mcg/kg per minute.

Other Antiarrhythmic Drugs

Amiodarone	Potassium Channel Blocker	150mg loading dose followed by infusion of 1mg/min for 6 hours and 0.5 mg/min for 18 hours	• Used to treat VT, VF, and afib. • Monitor for hypotension
Adenosine	Opens Potassium Channels	1st dose 6mg IVP 2nd dose 12 mg	• Used to treat narrow complex SVT. • Must be pushed RAPIDLY
Digoxin	Inhibits Na/K+ Pumps	IV dose: 0.1 - 0.4 mg/day	• Used to treat a-flutter and a-fib. • Can cause heart block. • Has a lot of potential side effects.

Heart Failure Primer

Heart Failure Essentials

NURSE DOSE

Definition and Classification

- **Definition:** The basic definition of heart failure is a chronic or acute condition where the heart is unable to pump enough blood to meet the body's needs. This directly translates to a decrease in cardiac output and eventually a decrease in organ perfusion.
- **Classification Systems**: The New York Heart Association categorizes heart failure based on symptoms and their severity as well as the patient's limitations. The staging system developed by the American College of Cardiology and the American Heart Association categorizes HF based on the structural changes and the presence of symptoms.

	ACC/AHA		NYHA
A	At high risk for HF but without structural heart disease or symptoms of HF	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF
B	Structural heart disease but without signs or symptoms of HF	II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, dyspnea
c	Structural heart disease with prior or current symptoms of HF	III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, dyspnea
D	Refractory HF requiring specialized interventions	IV	Unable to carry on any physical activity without discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases

Etiologies and Risk Factors

- **Etiologies:** Etiologies refer to the underlying causes or origins of heart failure. These are the specific diseases, conditions, or structural abnormalities that directly lead to the development of heart failure. Etiologies of heart failure include conditions such as **ischemic heart disease (coronary artery disease)**, **hypertensive heart disease**, **cardiomyopathies** (e.g., dilated cardiomyopathy, hypertrophic cardiomyopathy), and **valvular heart disease** (e.g., aortic stenosis, mitral regurgitation).

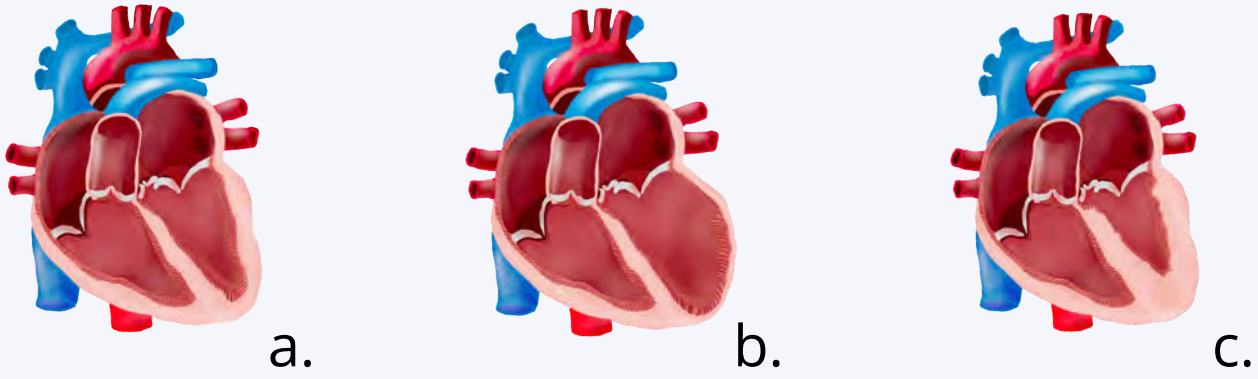
- **Risk Factors:** Risk factors are characteristics, conditions, or behaviors that increase an individual's likelihood of developing heart failure. These factors may not directly cause heart failure but contribute to its development by predisposing individuals to the underlying conditions that lead to heart failure. Common risk factors for heart failure include **hypertension**, **diabetes mellitus**, **obesity**, **advancing age**, **family history of heart failure**, **smoking**, **excessive alcohol consumption**, **illicit drug use** (e.g., cocaine), and **certain medications** (e.g. certain chemotherapeutic agents).

Pathophysiology

- The pathophysiology of heart failure involves a cascade of interconnected processes, including impaired contractility, increased afterload, volume overload, and neurohormonal activation. These processes contribute to progressive myocardial dysfunction, structural remodeling of the heart, and ultimately, the clinical manifestations of heart failure.

- There are two categories that heart dysfunction can be placed in: Systolic Dysfunction and Diastolic Dysfunction.
  - **Systolic Dysfunction**: this refers to the impaired ability of the ventricle to contract effectively during systole. The ventricles often appear dilated and have thinning of the ventricular walls. In systolic dysfunction the ejection fraction is reduced (HFrEF) leading to decreased stroke volume and, ultimately, cardiac output.

  - **Diastolic Dysfunction**: this refers to impaired relaxation or compliance of the ventricle. This dysfunction leads to decreased ventricular filling. Due to the decreased filling of the ventricles, stroke volume will also decrease. However, ejection fraction can be "preserved" (HFpEF) or normal as the heart is still ejecting a good percentage of blood, the ventricle chamber just doesn't allow for adequate filling.



(a.) Represents a normal heart with normal ventricle chamber size and wall thickness. (b.) represents how a heart with left ventricle systolic failure may look. The chamber is dilated and the ventricle wall is thinned. (c.) represents how a heart with diastolic failure may appear. The left ventricle chamber size is reduced and the ventricle wall size is increased.

	Ejection Fraction	**Cardiomyopathy**	**Blood Pressure**	**Extra Heart Sound**
Systolic Failure	<40%	Dilated	usually Low	S3
Diastolic Failure	Usually Normal EF	Hypertrophic	usually High	S4

Heart Sounds in Heart Failure

- **Systolic Failure:**
  - S3 Heart Sound: This heart sound is heard after the normal "lub-dub" of the S1, S2 heart sounds. This sound is usually caused by rapid filling and rapid distention of a pliable ventricle during early diastole. It is important to note that this sound can sometimes be heard in children, young adults, and pregnant women where it may be considered normal.

ken - tuc - KY

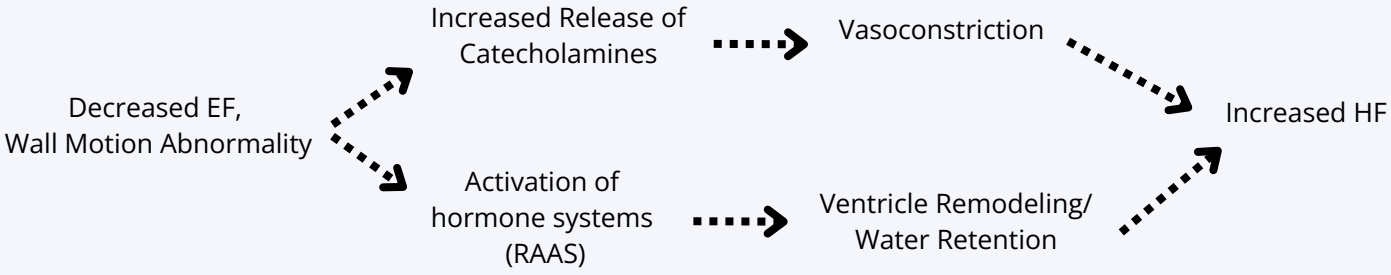
S1 - S2 - S3

- **Diastolic Failure:**
  - S4 Heart Sound: This heart sound is heard BEFORE the normal "lub-dub" of the S1, S2 heart sounds. This sound occurs when the atria contract forcefully against a stiffened ventricle during late diastole. This typically happens when the ventricle is enlarged and/or thickened which causes resistance to filling.

TE - nuh - see

S4 - S1 - S2

How Heart Failure Progresses



- Heart Failure can be further divided in right-sided, left-sided, or biventricular, with each having their own distinct clinical presentations.
  - **Left-sided Heart Failure**: This most common form of heart failure affects the left ventricle, impairing its ability to pump oxygen-rich blood through the body's systemic circulation. Left-sided failure can also force blood to back-up into the pulmonary vascular causing an array of symptoms and issues. If bad enough, left-sided heart failure could lead to right-ventricular strain and eventually failure, cause biventricular failure.
  - **Right-sided Heart Failure**: This form of heart failure affects the right ventricle, impairing its ability to pump blood to the lungs for oxygenation. Right-sided failure can also cause blood to back up within the systemic circulation, causing numerous symptoms throughout the body.

Systolic Failure Causes

- **Coronary Artery Disease (Myocardial Infarction)**
  - Coronary Artery Disease involves the reduction of oxygen-rich blood supply to areas of the heart muscle. This leads to ischemia which can damage the heart muscle over time, leading to cardiomyopathy, where the ventricles become weakened and cannot pump effectively.
  - Myocardial Infarctions can cause significant damage to the heart muscle and further contribute to systolic heart failure.

- **Valve Dysfunction (regurgitation)**
  - Valve dysfunction, specifically valve regurgitation, can cause or exacerbate systolic heart failure. In aortic regurgitation, the failing valve allows for blood to flow back into the left ventricle from the aorta during diastole. This results in increased pressure and volume in the ventricle which can eventually lead to systolic heart failure. With Mitral or Tricuspid Regurgitation, stroke volume from the ventricles is reduced as blood is allowed to flow back into the atria and reducing pumping efficiency.

- **Cardiomyopathy**
  - Dilated Cardiomyopathy: Dilation of the ventricle with thinning of the ventricle walls is characteristic of dilated cardiomyopathy. This can be caused by activation of hormone systems like the RAAS system which can lead to direct ventricle remodeling (dilation or hypertrophy). Dilation can also be caused from increased diastolic volume and pressures. Finally, Dilated Cardiomyopathy can also be caused by genetic factors, viral infections, and certain drugs/toxins.
  - Ischemic Cardiomyopathy: This cardiomyopathy is caused by CAD and myocardial infarctions. The lack of blood supply to the heart muscle can cause damage and scarring of the heart leading to cardiomyopathy and decreased systolic efficiency.

Diastolic Failure Causes

- **Chronic Hypertension:**
  - Hypertension causes the heart to work harder to pump blood against elevated pressure in the aorta. Over time, this increased workload can lead to hypertrophy or thickening of the heart muscle, specifically in the left ventricle. This thickened and stiff muscle impairs the ability of the ventricle to relax properly, leading to diastolic failure via decreased filling.

- **Valve Dysfunction (Stenosis):**
  - Much like with hypertension, valve stenosis cause the heart to pump harder against a stiffened and non-compliant valve. This increased workload can lead to hypertrophy or thickening of the heart muscle. A thickened and stiff heart does not allow for adequate filling. Valve stenosis can also lead directly to impaired ventricular filling.

- **Hypertrophic Cardiomyopathy:**
  - As stated above, increased workload on the heart as well as RAAS activation can cause the heart muscle/ventricle to thicken and stiffen leading to Hypertrophic Cardiomyopathy. Other factors that could lead to Hypertrophic Cardiomyopathy include genetics/family history and age.

Clinical Presentation

For clinical presentation, we are going to focus on the presentations of left-sided heart failure versus right-sided heart failure:

- **Left-Sided Heart Failure:**
  - **Tachycardia:** Elevated heart rate, reflecting the body's compensatory response to decreased cardiac output and impaired tissue perfusion.
  - **Dyspnea:** Shortness of breath, particularly on exertion or when lying flat (orthopnea), due to pulmonary congestion and inadequate oxygenation.
  - **Fatigue:** Generalized weakness and fatigue, often exacerbated by decreased cardiac output and tissue perfusion.
  - **Orthopnea:** Difficulty breathing while lying flat, prompting patients to prop themselves up with pillows to relieve symptoms.
  - **Pulmonary Congestion:** Manifested by cough, wheezing, frothy sputum, and crackles on auscultation due to fluid accumulation in the lungs. This is also evident by elevated PA pressures and wedge pressure.
  - **Confusion:** Cognitive impairment or disorientation, particularly in elderly patients, due to inadequate cerebral perfusion and hypoxemia.

- **Right-Sided Heart Failure:**
  - **Peripheral Edema:** Swelling of the lower extremities, ankles, and feet, resulting from fluid retention and venous congestion.
  - **Jugular Venous Distention:** Visible distention of the jugular veins in the neck, indicative of increased central venous pressure (CVP).
  - **Hepatomegaly:** Enlargement of the liver due to congestion and impaired hepatic blood flow, often associated with elevated liver enzymes.
  - **Abdominal Pain:** Reduced appetite and gastrointestinal symptoms due to hepatic congestion and impaired nutrient absorption.

Diagnostic Evaluations

The Diagnostic Evaluations that we use to diagnose heart failure include the following:

- **Echocardiogram (Echo):** A non-invasive ultrasound imaging test that provides detailed information about the structure and function of the heart. Echocardiography can assess cardiac chamber size, wall thickness, ejection fraction, valvular function, and the presence of any structural abnormalities or signs of heart failure.
- **B-type Natriuretic Peptide (BNP) Test:** BNP is a hormone produced by the heart in response to increased ventricular stretch and pressure, such as in heart failure. Measuring BNP levels in the blood can help diagnose and assess the severity of heart failure, with higher levels correlating with more advanced disease.
- **Chest X-ray (CXR):** A commonly used imaging modality that can reveal signs of heart failure, such as cardiomegaly (enlargement of the heart), pulmonary congestion, and vascular redistribution.
- **Electrocardiogram (ECG/EKG):** ECG findings in heart failure may include evidence of left ventricular hypertrophy, atrial fibrillation, conduction abnormalities, or signs of myocardial ischemia or infarction.
- **Cardiac MRI (Magnetic Resonance Imaging):** An advanced imaging technique that provides detailed images of the heart's structure and function, including assessment of myocardial viability, scar tissue, and chamber volumes. Cardiac MRI can be particularly useful for evaluating complex cases or assessing for underlying etiologies of heart failure.
- **Cardiac Catheterization:** An invasive procedure used to evaluate coronary artery disease and assess hemodynamic parameters such as pressures within the heart chambers and cardiac output. It may be indicated in select cases to guide treatment decisions or assess for reversible causes of heart failure.

Treatment

- **Medications:**
  - **Angiotensin-Converting Enzyme (ACE) Inhibitors or Angiotensin II Receptor Blockers (ARBs):** These medications help dilate blood vessels, reduce blood pressure, and decrease the workload on the heart by interrupting the Renin-Angiotensin-Aldosterone System. These medications can also help decrease cardiac remodeling. **Example: ACE Inhibitor - Lisinopril, ARB - Valsartan**
  - **Aldosterone Antagonists:** These medications help reduce fluid retention and improve survival in patients with heart failure by blocking the effects of aldosterone, a hormone that promotes sodium and water retention. Chronic exposure to high aldosterone levels can lead to cardiac fibrosis and remodeling. **Example: Spironolactone**
  - **Beta-Blockers (chronic failure only!):** These medications slow the heart rate (decrease oxygen demand), reduce blood pressure (decrease workload of the heart), and improve heart function by blocking the effects of catecholamines on the heart. Beta-blockers can also aid in upregulating beta receptors in the heart leading to increased inotropic effects over time. It should be noted that beta-blockers are not indicated for acute heart failure leading to cardiogenic shock. **Example: Metoprolol succinate**
  - **Diuretics (Mainly systolic failure):** Diuretics help reduce fluid retention by increasing urine output, thus alleviating symptoms of congestion such as edema and dyspnea. Diuretics should be given with caution to someone with diastolic failure as the issue is reduced filling. For diastolic failure, euvolemia should be goal, dehydration could lead to increased failure and symptoms. **Example: Lasix**
  - **Inotropes:** Inotropes, such as **Dobutamine and Milrinone**, are medications that increase the strength of the heart's contractions (positive inotropy), helping to improve cardiac output and tissue perfusion. Dobutamine and Milrinone also have the added effect of decreasing afterload. They are often used in cases of acute decompensated heart failure or severe systolic dysfunction refractory to standard medical therapy. Inotropes are not generally indicated for diastolic failure as issue is with filling rather than pumping.
  - **Vasodilators:** Vasodilator medications, such as hydralazine and nitrates, help dilate blood vessels, reducing blood pressure and easing the workload on the heart.

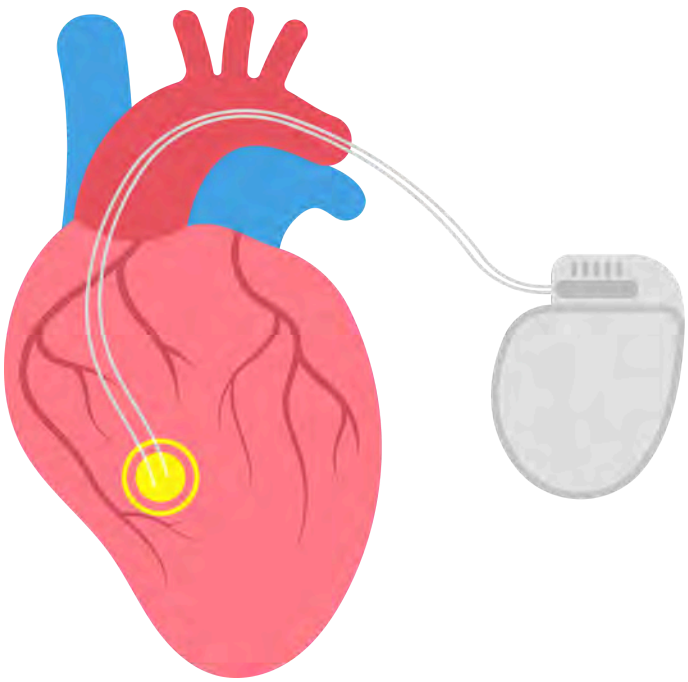
- **Device Therapy:**
  - **Automatic Implantable Cardioverter-defibrillator (AICD):** this device is implanted in patients with heart failure primarily to prevent sudden cardiac death caused by life-threatening ventricular arrhythmias such as ventricular tachycardia or fibrillation which are more common in patients with heart failure. It continuously monitors the heart's rhythm and delivers an electrical shock to restore normal heart rhythm if a dangerous arrhythmia occurs.
  - **Intra-aortic Balloon Pump (IABP):** The IABP can provide hemodynamic support for patients with heart failure by augmenting coronary perfusion, reducing afterload, and improving cardiac output. The IABP consists of a balloon catheter inserted into the aorta, which inflates during diastole and deflates during systole, synchronized with the cardiac cycle. This inflation and deflation pattern enhances coronary blood flow during diastole, improving myocardial oxygen supply, while reducing afterload during systole, easing the workload on the heart.
  - **Ventricular Assist Device (VAD):** A VAD provides mechanical circulatory support for patients with advanced heart failure by assisting or replacing the function of a failing heart. This implantable device helps pump blood from the left ventricle (LVAD) to the systemic circulation, thereby improving cardiac output and organ perfusion. VADs are typically used in patients with severe heart failure who are refractory to medical therapy, awaiting cardiac transplantation (bridge to transplant), or as destination therapy for those who are not transplant candidates.
  - **Impella:** The Impella is a form of ventricular assist device that is placed percutaneously to provide temporary mechanical circulatory support for patients with severe heart failure or cardiogenic shock. It works by inserting a catheter-mounted axial flow pump into the left ventricle, which actively pumps blood from the ventricle into the ascending aorta, thereby increasing cardiac output and perfusion to vital organs. The Impella device can unload the left ventricle, reduce myocardial oxygen demand, and improve coronary perfusion, making it a valuable tool in managing acute decompensated heart failure. Impellas can also now be used in right heart failure as well.

Treatment Continued

- **Cardiac Resynchronization Therapy (CRT):** Cardiac resynchronization therapy (CRT) is a treatment for heart failure that involves implanting a special type of pacemaker known as a biventricular pacemaker. CRT works by coordinating the contractions of the heart's ventricles to improve cardiac function and alleviate symptoms in patients with heart failure and electrical dyssynchrony. CRT is typically indicated in patients with moderate to severe heart failure symptoms despite optimal medical therapy, reduced left ventricular ejection fraction (HFrEF), and evidence of ventricular dyssynchrony on electrocardiogram or echocardiogram.

- **Surgical Interventions:**
  - **Coronary Artery Bypass Graft (CABG):** CABG improves heart failure by restoring blood flow to areas of the heart affected by coronary artery disease. By bypassing blocked or narrowed coronary arteries, CABG improves myocardial perfusion, reduces ischemia, and enhances cardiac function, thereby alleviating heart failure symptoms and promoting better outcomes.
  - **Valve Repair or Replacement:** Repair involves surgical techniques to restore the function of a damaged valve, while replacement involves replacing the valve with a mechanical or biological prosthetic valve. By restoring proper valve function, these procedures improve hemodynamics, reduce regurgitation or stenosis, and alleviate symptoms such as dyspnea, fatigue, and fluid retention associated with heart failure. Valve interventions can improve overall cardiac performance and quality of life in heart failure patients.

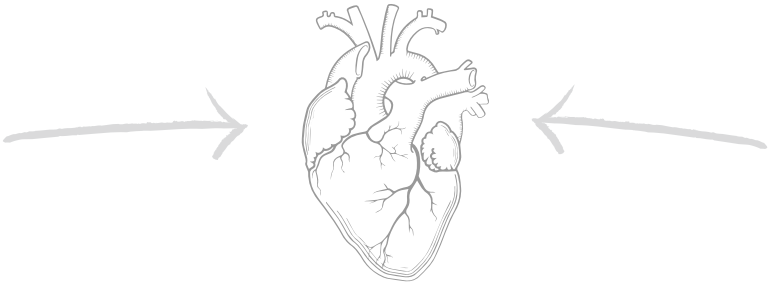
- **Heart Transplantation**
  - Heart transplantation is the ultimate treatment for end-stage heart failure, offering the potential for a cure by replacing a failing heart with a healthy donor heart. It is reserved for patients with advanced heart failure who have exhausted all other medical and surgical options and have a limited life expectancy without transplantation. Heart transplantation can significantly improve quality of life, functional capacity, and long-term survival in carefully selected patients. However, it requires lifelong immunosuppressive therapy to prevent rejection of the transplanted heart, and the availability of suitable donor organs remains limited, necessitating stringent criteria for candidate selection.



Hemodynamics Primer

Hemodynamics:

The study of the movement of blood through the circulatory system and the forces that affect it.



Most of hemodynamics can be condensed into one formula...

Cardiac Output = Stroke Volume x Heart Rate

Cardiac Output (Normal Value 4 - 8L/min) is the amount of blood (usually in liters) that is pumped by the heart in one minute. CO is a core measurement of hemodynamics as it quite literally describes the movement of blood through the body.

However, there is an even better value that we look at more often: **Cardiac Index**.

CO: 4

CO: 8

Refer to the image above. Just because the girl's CO is 4 while the bodybuilders is 8 does not mean that the girl's heart is performing any worse than the guys. This is why body surface area (BSA) is so important. Cardiac index takes this into account and gives us a more accurate understanding of how the heart is performing in relation to total body size.

Normal Range for Cardiac Index is:

2.5 - 4 L/min/m^2

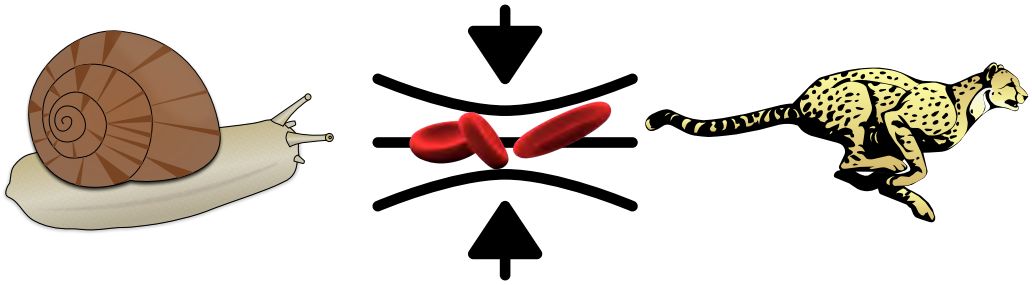
This value is extremely helpful as it allows us to compare all patients to one range, regardless of body size.

Now lets take a closer look at the other components of the equation.

Stroke Volume is the amount of blood pumped by the left ventricle with each beat. Normal stroke volume is 60 - 100 ml/beat but stroke volume index is important to consider due to body surface area, much like cardiac index. A normal stroke volume index is 33-47.

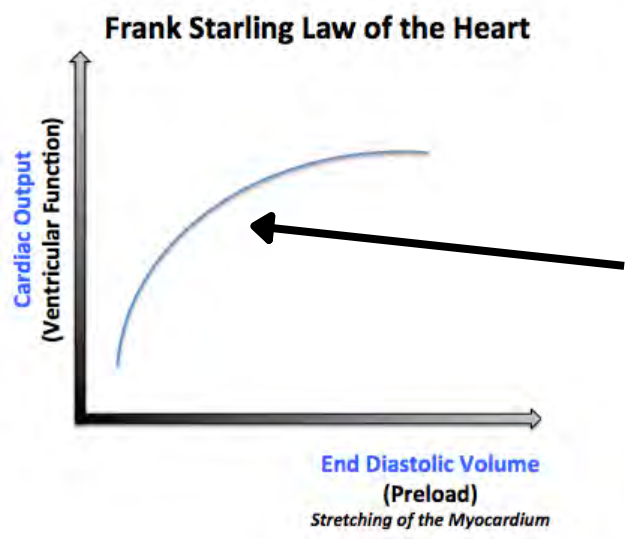
"Will my patient's SV be low if they need fluid?"

Not necessarily. The body has so many different mechanisms to compensate for low fluid status that if stroke volume is low you are way behind in resuscitation.



Increasing squeeze of the vasculature or 'vasoconstriction' can increase the return of blood to the heart.

This return on blood causes the heart to 'stretch' more. This stretch, called 'Preload', can lead to better contraction and cardiac output... up to a point.



You can see from this graph of Sterling's Law of the Heart that once you get past a certain point, more preload does not give you as much return on cardiac output.

Think of preload this way: Imagine you put about 10ml of water in your mouth and you spit it out. Not much force behind it right?

Now pretend you fill your entire mouth up with water to the point that your cheeks stretch. When you go to spit that water out you have much more contraction and force behind it. That is the effect of preload.

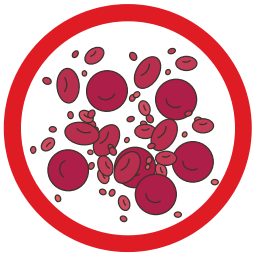
Can you also imagine how a failing heart might not be able to deal with increased preoload?

Now, Lets get back to talking about vasoconstriction

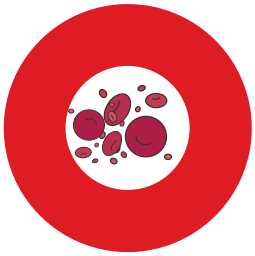
Vasoconstriction and dilation can actually be measured through a value known as SVR or 'Systemic Vascular Resistance'. This value also has an index value that correlates to body surface area so we will focus on that. SVR can also be referred to as 'afterload'

SVRI - Systemic Vascular Resistance Index

Low	High



Normal Value:
1700 - 2400



- Distributive Shock
- Vasoplegia
- Hyperthermia
- Euvolemia

- Hypovolemia
- Sympathetic Stim
- Hypothermia
- Cardiogenic Shock

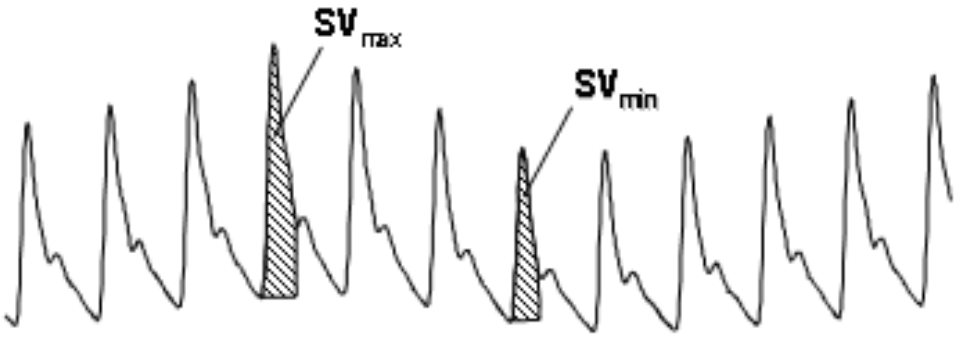
While it seems like a good thing to vasoconstrict to get more blood back to the heart, Afterload could also hinder a sick heart. Afterload is a force that the heart must overcome to push blood out of the aortic and pulmonic valve.

Question!

What kind of Cardiac output do you expect to see on someone with a very low SVRI and normal heart function? Hint: Decreased Afterload = less force to overcome to eject blood from the ventricles.

Since we have the basics of hemodynamics down now lets start talking about some more advanced topics.

Stroke Volume Variation (SVV)



$$SVV = \frac{SV_{max} - SV_{min}}{(SV_{max} + SV_{min})/2}$$

Stroke Volume Variation (SVV) is a hemodynamic parameter used to assess the variability in the stroke volume of the heart during each cardiac cycle. It measures the changes in the volume of blood ejected by the heart with each heartbeat.

SVV is typically expressed as a percentage and is derived from the analysis of arterial pressure waveforms or other monitoring techniques like the Vigileo and EV1000.

The main purpose of measuring SVV is to evaluate fluid responsiveness in patients. A high SVV value suggests that the patient's stroke volume is likely to increase with fluid administration, indicating that they may be fluid-responsive. Conversely, a low SVV value indicates that the patient's stroke volume is less likely to improve with further fluid administration, suggesting that they may be fluid non-responsive.

Stroke Volume Variation Normal Value

< 13%

"High is DRY"

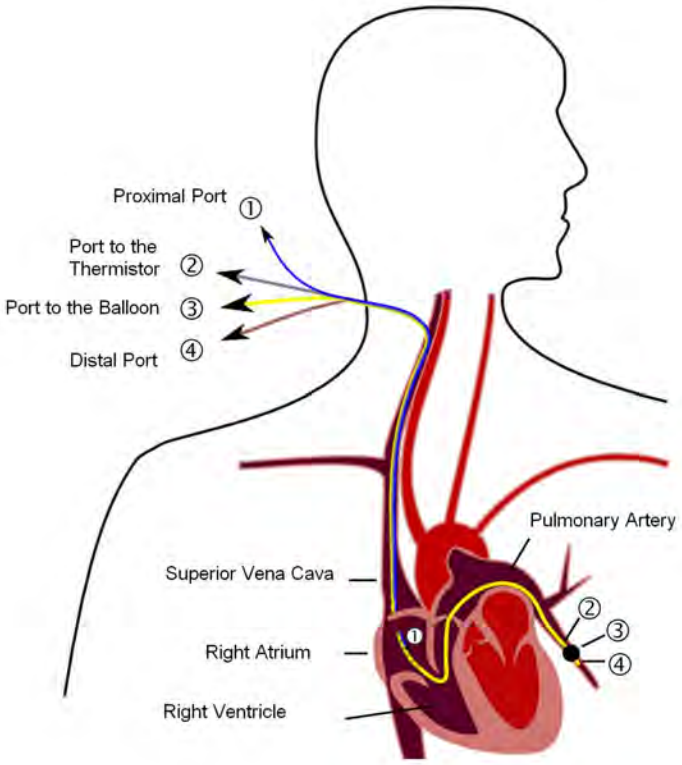
Pulmonary Artery Pressures (PA Pressures)

Pulmonary artery pressures are typically measured during right heart catheterization or with a Swan Ganz catheter that is placed at the bedside, and understanding the normal and abnormal values is important for interpretation. Here are the normal values and their significance:

Pulmonary artery systolic pressure (PASP): Normal range is typically around 15-30 mmHg. Elevated PASP may indicate conditions such as pulmonary hypertension, left heart failure, or lung diseases.

Pulmonary artery diastolic pressure (PADP): Normal range is approximately 5-15 mmHg. Higher PADP values can suggest impaired left ventricular function or increased pulmonary vascular resistance.

Mean pulmonary artery pressure (MPAP): Normal range is approximately 10-20 mmHg. MPAP above 25 mmHg at rest or 30 mmHg during exercise is considered elevated and may indicate pulmonary hypertension.



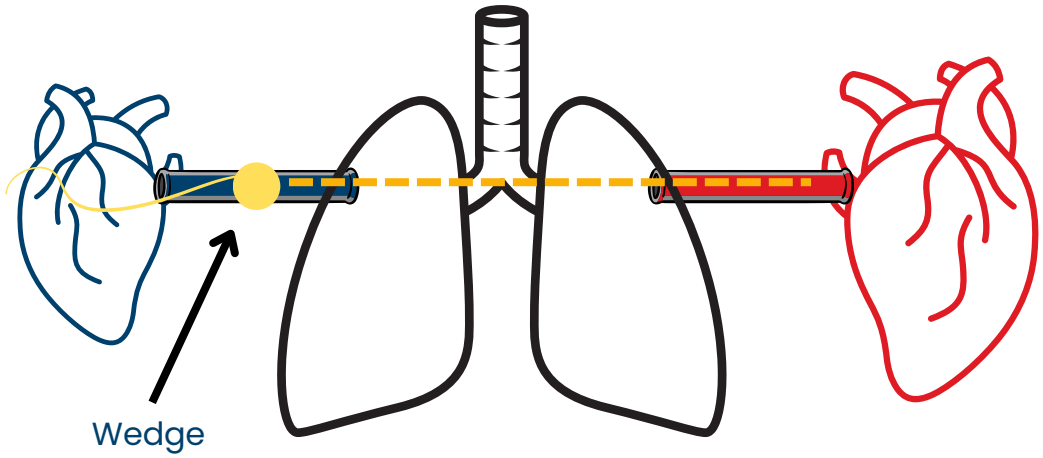
In addition to pulmonary artery pressures, another important measurement obtained during right heart catheterization is the pulmonary capillary wedge pressure (PCWP).

Pulmonary capillary wedge pressure (PCWP): PCWP represents the pressure in the left atrium and left ventricle at end-diastole. It is estimated by wedging the catheter in a small branch of the pulmonary artery, temporarily blocking the blood flow and allowing the catheter to capture the pressure. PCWP serves as an indirect measure of left atrial pressure and left ventricular end-diastolic pressure.

Normal PCWP values: Normal PCWP values typically range from 6-12 mmHg. This pressure reflects the left heart's ability to fill with blood during diastole and provides information about left ventricular preload.

Elevated PCWP: Increased PCWP values above the normal range may suggest left ventricular dysfunction, such as left heart failure or myocardial infarction. It can also indicate fluid overload, as in cases of volume overload or congestive heart failure. Elevated PCWP may result in pulmonary congestion and symptoms like dyspnea, cough, and edema.

Low PCWP: Decreased PCWP values may indicate hypovolemia (low blood volume) or reduced left ventricular filling due to conditions like hypovolemic shock, severe dehydration, or aortic stenosis.



Imagining the two halves of the heart separated as illustrated above can help conceptualize how wedging the PA artery can help us measure pressure in the left atrium.

What is an ICU Nurse's favorite value?...

Mean Arterial Pressures (MAP)

Mean arterial pressure (MAP) is a measure of the average pressure within the arteries during a cardiac cycle. It represents the force exerted by the blood against the arterial walls, ensuring adequate perfusion of organs and tissues. MAP is a critical parameter in assessing overall cardiovascular health and organ perfusion.

MAP is calculated using the following formula:

MAP = (systolic blood pressure + 2 * diastolic blood pressure) / 3

Normal MAP range 70 - 100 mmhg

While systolic pressure is important in assessing cardiac function and determining the workload on the heart, MAP offers a more comprehensive picture of arterial pressure, organ perfusion, and overall cardiovascular health. By considering both systolic and diastolic pressures, MAP provides a valuable parameter for monitoring and managing blood pressure to support optimal organ function and tissue perfusion.

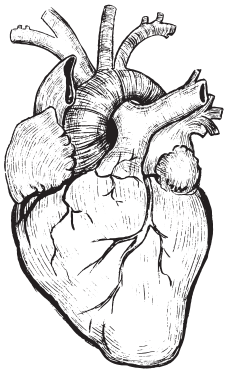
Central Venous Pressures (CVP)

Central venous pressure (CVP) refers to the pressure within the large veins that return deoxygenated blood to the right atrium of the heart. It reflects the blood volume and the ability of the heart to pump blood effectively. Measuring CVP provides important information about the fluid status, cardiac function, and intravascular volume of a patient.

CVP is important for several reasons. First, it serves as an indicator of intravascular volume and fluid status. Elevated CVP may suggest volume overload or impaired fluid balance, while low CVP values may indicate hypovolemia or inadequate fluid resuscitation. Second, CVP provides information about cardiac function and right heart filling pressures. It helps assess the effectiveness of the heart's pumping action and guides treatment decisions, such as fluid administration, diuretic therapy, or vasopressor support.

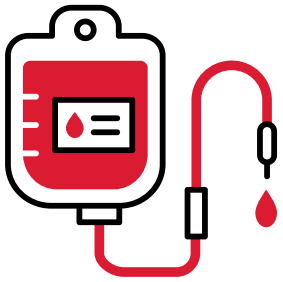
Types of SHOCK

Cardiogenic shock is a type of shock that occurs when the heart is unable to pump enough blood to meet the body's demands. It is usually caused by a severe heart attack (myocardial infarction) or other conditions that result in significant damage to the heart muscle. It is characterized by decreased contractility, increased afterload, and increased filling pressures.



Distributive shock, also known as vasodilatory shock, is a type of shock that occurs due to widespread dilation of blood vessels throughout the body. This dilation leads to a decrease in systemic vascular resistance (SVR), which is the resistance encountered by blood flow in the blood vessels. As a result, distributive shock is characterized by a decrease in blood pressure and impaired tissue perfusion despite the presence of an adequate cardiac output.

Hypovolemic shock is a type of shock that occurs when there is a significant loss of blood volume or fluid from the body, leading to a decrease in circulating blood volume. This can be caused by severe bleeding, excessive fluid loss (e.g., dehydration), or fluid shifts (e.g., burns). Characterized by decreased preload, increased systemic vascular resistance and increased heart rate.



Hemodynamics Chart

	MAP	CI	SVV	SVRI	CVP
Euvolemic (Normal)	N	N	N	N	N
Hypervolemic	N ↑	N ↑	N ↓	N ↓	↑
Hypovolemic	N ↓	N ↓	↑	↑	N ↓
Cardiogenic Shock	↓	↓	N ↓	↑	↑
Distributive Shock	↓	↑	↑	↓	N ↓

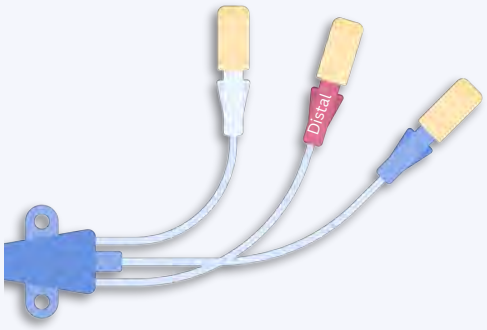
CVP Primer

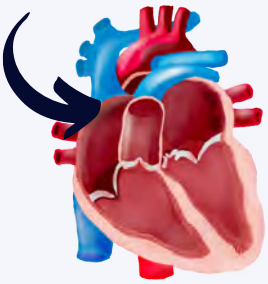
Central Venous Pressure (CVP)

NURSE DOSE

Setup and Placement

- Central venous pressure (CVP) is the pressure measured in the right atrium or superior vena cava (IVC for femoral lines), providing insight into the filling pressure on the right side of the heart.
- It can be measured via a central venous catheter through the lumen that terminates closest to the superior vena cava (SVC) or right atrium.

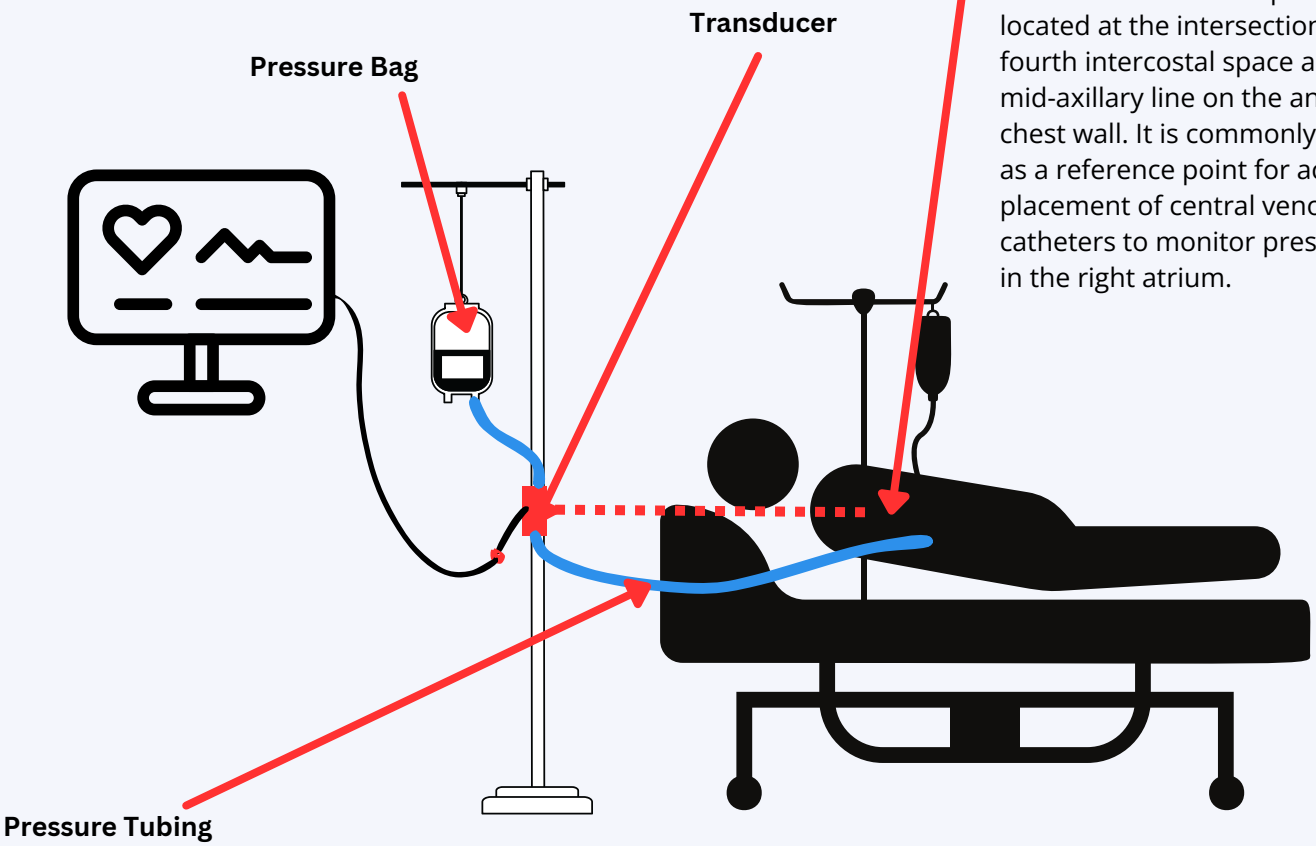




- In most CVCs, the distal port lumen terminates in the SVC and is ideal for CVP monitoring. However, in a pulmonary artery catheter the blue port usually terminates in the right atrium as the distal port terminates in the pulmonary artery.
- Just like any other directly measured pressure value, the CVP line must be zeroed to atmospheric pressure at least once a shift (depending on hospital policy) or after any transportation/ repositioning. Be sure the transducer is at the level of the phlebostatic axis when zeroing.

Phlebostatic Axis

The phlebostatic axis is an anatomical reference point located at the intersection of the fourth intercostal space and the mid-axillary line on the anterior chest wall. It is commonly used as a reference point for accurate placement of central venous catheters to monitor pressures in the right atrium.



CVP Values

Normal Central Venous Pressure (CVP) values typically fall within the range of 2 to 6 mmHg (millimeters of mercury).

Implications of Low CVP:

Hypovolemia: A CVP below the normal range may indicate hypovolemia, which means there's a decreased volume of blood in the circulatory system. This can be caused by conditions such as severe dehydration, hemorrhage, or excessive fluid loss.

NOTE: CVP is a poor indicator of fluid responsiveness.

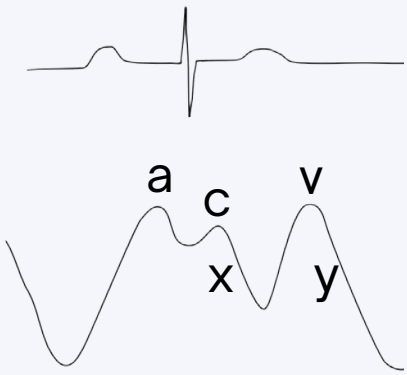
Implications of High CVP:

Fluid Overload: An elevated CVP suggests that there's an increased volume of blood within the right atrium, often indicating fluid overload or an excess of intravascular fluid. This can result from conditions like heart failure, renal failure, or excessive intravenous fluid administration.

Pulmonary Hypertension: In some cases, high CVP may be a sign of pulmonary hypertension, which can place additional pressure on the right side of the heart.

Right Heart Dysfunction: Any condition that impairs right heart function can lead to an increase in CVP. This includes conditions like right ventricular failure or pulmonary embolism.

Normal CVP Waveforms

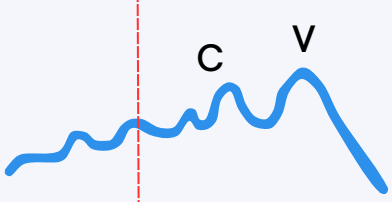


- a = Atrium Contraction
- c = Closing of tricuspid valve
- x = Relaxation of the ventricle
- v = Filling of the atrium
- y = opening of tricuspid valve and ventricular filling

- **Atrial Wave (a-wave):** This is the first positive deflection on the CVP waveform. It is caused by atrial contraction, which occurs during ventricular diastole. The a-wave represents an increase in right atrial pressure due to atrial contraction, and it's normally seen as a small, distinct peak.
- **Ventricular Wave (v-wave):** The v-wave follows the a-wave and represents an increase in pressure as the tricuspid valve closes and blood is forced into the right atrium during ventricular systole. This is another small, distinct peak.
- **X Descent:** After the a-wave, there's a gradual decline in CVP during ventricular systole when the atrium is relaxed. This is known as the X descent and is typically a descending limb without any prominent peaks.
- **Y Descent:** The Y descent follows the V-wave and is the continuous decline in CVP during ventricular diastole. It represents the passive filling of the right ventricle.

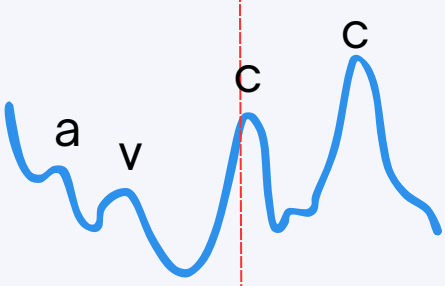
Abnormal CVP Waveforms

Atrial Fib/Fluttler



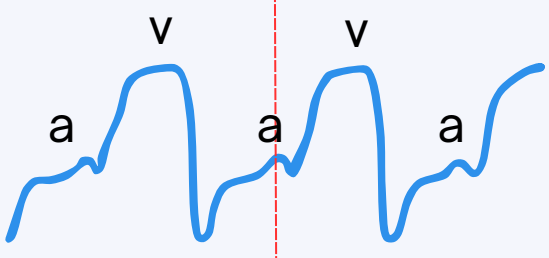
- A-fib is characterized by absent a-waves and small indistinct waves that represent the fib/flutter of the atrium.

Third Degree Heart Block



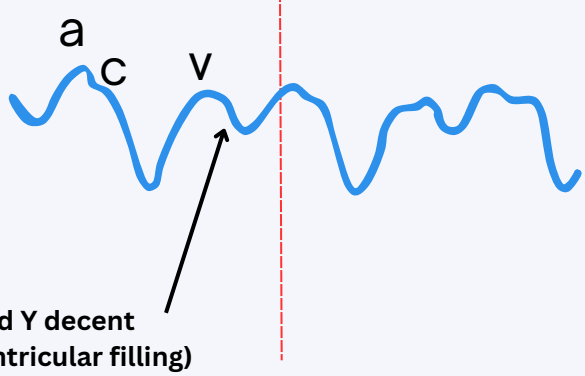
- Third degree heart block or AV dissociation is characterized by “cannon a-waves”, when the ventricle and atrium have a simultaneous contraction.

Tricuspid Regurgitation



- With tricuspid regurgitation there is a early and large v-wave due to the atrium filling from back flow of the ventricle during systole.

Tamponade



- The defining feature of tamponade in CVP waveforms is the diminished Y-decent which indicates decreased ventricular filling. This is due to the increased global pressure put on the heart from the tamponade.

PA Catheter Primer

Pulmonary Artery Catheter Cheat Sheet

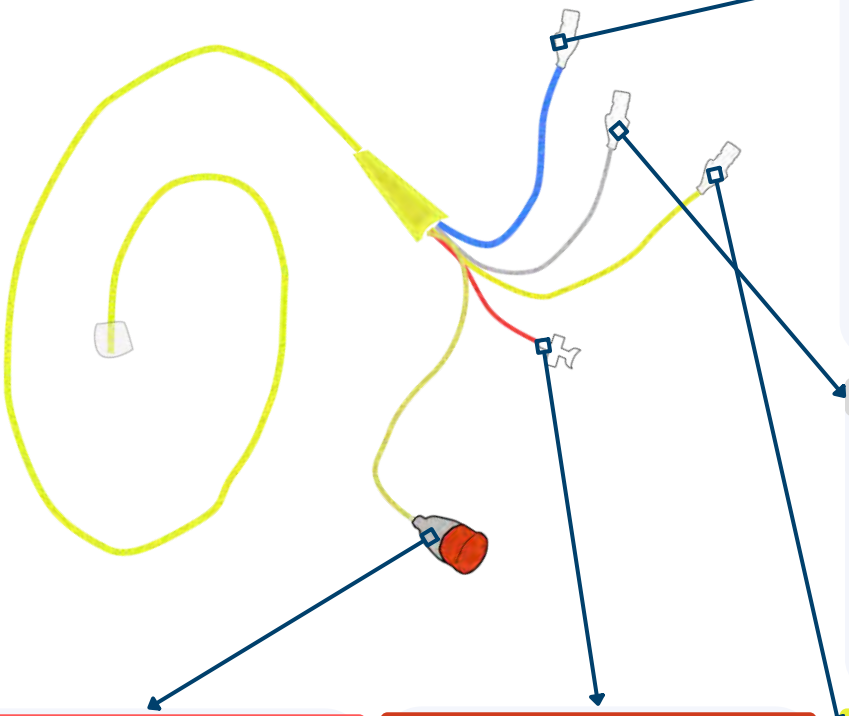
NURSE DOSE

What is a Pulmonary Artery Catheter?

A Pulmonary Artery (PA) catheter, commonly known as a Swan-Ganz catheter, is a tool used in critical care settings to assess and monitor a patient's cardiovascular status. By threading a thin, flexible catheter into the pulmonary artery through a large vein, healthcare providers gain real-time insights into key hemodynamic parameters. These parameters include central venous pressure (CVP), pulmonary artery pressure (PAP), pulmonary artery occlusion pressure (PAOP), cardiac output (CO), and mixed venous oxygen saturation (SvO2). The information obtained from a PA catheter aids in guiding treatment decisions and optimizing patient care in complex and critically ill situations.

Normal Values

CVP: 2-8 mmHg
PAP: Systolic 20-30 mmHg, Diastolic 8-15 mmHg, Mean 10-20 mmHg
PAOP: 6-12 mmHg
CO: 4-8 L/min
SvO2: 60-75%



Blue Port

The blue port, situated proximally for injectate, is a vascular passage that terminates 30 cm from the catheter's tip.

Proper positioning places it in the right atrium. This port is utilized for administering fluid boluses to calculating cardiac output.

It also connects to continuous right atrial pressure monitoring with waveform visualization. While intermittent IV therapy can be administered through this port, vasoactive drugs should not be used.

White Port

The white port, designed for proximal infusion, concludes its path 31 cm from the catheter's tip.

It ends in the right atrium, positioned above and on the opposing side of the catheter in relation to the blue port.

This port can be employed for administering IV fluids and medications

Thermistor

The thermistor, identifiable by its square white connector with a red cap, is linked to the cardiac output trunk cable.

It gauges the internal blood temperature within the pulmonary artery, detecting alterations after introducing room temperature or cold IV fluid.

This assessment is employed in the calculation of cardiac output.

Balloon Port

The balloon port, indicated by an attached syringe, enables balloon inflation at the catheter's tip.

Balloon inflation is utilized during placement of the catheter to "float" the tip into the pulmonary artery. The balloon is also inflated when measuring a pulmonary artery occlusive pressure.

Its maximum inflation volume is 1.5 cc of air.

Yellow Port

The yellow port, extending to the catheter's distal tip along the full 110 cm length, serves as a vascular conduit.

Connected to a pressure transducer, it displays the pressure at the tip of the catheter. Pressures measured here include PA systolic, PA diastolic, and wedge pressures when indicated.

Typically, the catheter's tip should reside in the pulmonary artery, except during insertion, repositioning, or wedging procedures.

Special Considerations

When not wedging or positioning catheter the balloon should have no air. The syringe should be attached with no air present and the port should be in the open position to confirm that no air resides in the balloon.

When performing a wedge procedure, the maximum amount to be instilled into the balloon is 1.5ml. If a wedge is not achieved, the balloon must be deflated and catheter repositioned. The syringe should not be taken off in an attempt to instill more than 1.5ml into the balloon. Over-inflation of the balloon could cause rupture of the pulmonary artery. PA diastolic numbers can be used to estimate PAOP. The PAOP will never be above the diastolic PA pressure.

The position of the catheter should be noted in cm at least every shift and whenever patient transport or repositioning has occurred.

If the patient is exhibiting arrhythmias, the catheter may be in the incorrect position. The provider should be notified and x-ray should be used to determine correct placement.

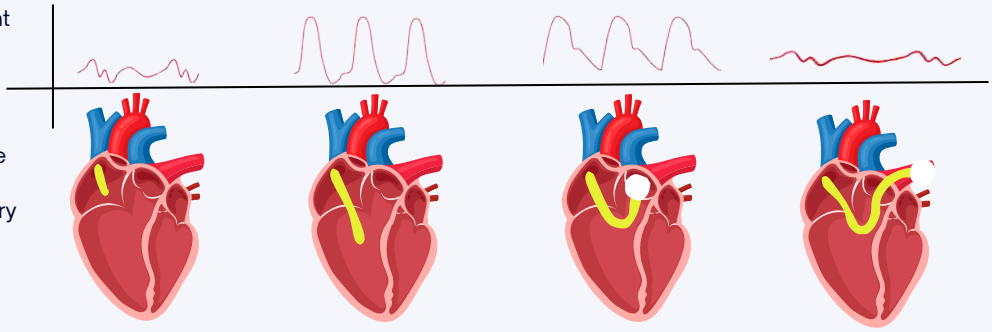
Pulmonary Artery Catheter Cheat Sheet

NURSE DOSE

Insertion and Placement

When inserting the Pulmonary Artery Catheter it is important that the line is being transduced so that the appropriate waveforms are visualized as the tip makes its way through the heart.

When the tip enters the right atrium you can expect a CVP waveform around 2-8 mmHg. In the right ventricle around 25mmHg. The balloon is then inflated and passed into the pulmonary artery where a dicrotic notch will be observed from pulmonic valve. Eventually the waveform will resemble a wedge around 8-10 mmHg



Calculating CO using Thermodilution

1. **Introduction of Indicator Solution**: A known volume of cool or room-temperature saline solution (indicator) is injected into the patient's circulation through the proximal injectate port (usually Blue Port) of the PA catheter. The solution is typically injected rapidly to create a temperature change in the blood.
2. **Indicator Mixing:** As the indicator solution mixes with the warmer blood in the right atrium, the temperature of the mixed blood decreases temporarily. This change in temperature is detected by the thermistor located on the PA catheter, which measures the temperature of the blood passing by.
3. **Temperature Detection and Analysis:** The thermistor measures the temperature changes caused by the indicator solution. The rate of temperature change is then analyzed to determine the area under the curve of the temperature-time graph. This area corresponds to the cardiac output.
4. **Calculation of Cardiac Output:** The computerized monitoring system connected to the PA catheter uses the indicator dilution curve to calculate the cardiac output. It compares the area under the curve of the indicator dilution with a pre-established calibration curve. This curve is generated using known CO values and their corresponding indicator dilution curves.
5. **Output Display:** Once the calculation is performed, the cardiac output value is displayed on the monitoring system in real-time. This value reflects the patient's current cardiac output based on the thermodilution method.

Cardiac Output calculation using equipment like the HemoSphere by Edward Lifesciences utilize an "energy pulse" and thermistor on a compatible Swan-Ganz catheter to produce the temperature differential and allow for a "continuous" cardiac output measurement.

It's important to note that while the thermodilution method provides a reliable estimate of cardiac output, it involves certain assumptions and limitations. Variations in body temperature, injection technique, and catheter positioning can impact the accuracy of the measurement. Therefore, multiple measurements are often taken and averaged to improve accuracy.

Cause of Abnormal Pulmonary Artery Pressures

HIGH

1. **Primary Pulmonary Hypertension (Idiopathic Pulmonary Arterial Hypertension)**: A rare condition characterized by elevated PA pressures without a clear underlying cause.
2. **Secondary Pulmonary Hypertension**: Elevated PA pressures due to an underlying condition, such as:
   - Chronic obstructive pulmonary disease (COPD)
   - Interstitial lung disease
   - Pulmonary embolism (blood clot in the lung)
   - Left heart failure (left ventricular dysfunction)
   - Connective tissue disorders (e.g., scleroderma)
   - Sleep apnea
   - Congenital heart diseases affecting the pulmonary vasculature

LOW

1. **Hypovolemia**: Low blood volume, often due to dehydration or excessive fluid loss, can lead to decreased PA pressures.
2. **Sepsis**: Severe infections can cause widespread vasodilation and low blood pressure, which may result in decreased PA pressures.
3. **Valvular Regurgitation**: Conditions like aortic regurgitation can cause a backflow of blood from the aorta into the left ventricle, leading to decreased resistance in the pulmonary circulation and lower PA pressures.
4. **Advanced Heart Failure**: In advanced heart failure, the heart's ability to pump blood effectively is compromised, which can lead to decreased pressures in both the systemic and pulmonary circulation.
5. **Massive Blood Loss**: Acute blood loss can reduce blood volume and lead to decreased PA pressures.
6. **Certain Medications**: Some medications, such as vasodilators, can reduce blood pressure and subsequently lead to decreased PA pressures.

SCAI Shock Classification

SCAI Shock Classification Checklist

SCAI Shock Stage (+/- Arrest Modifier): _____

Patient: _____ **Date:** _____ **Time:** _____

SCAI SHOCK STAGE	DESCRIPTION	PHYSICAL EXAM	BIOCHEMICAL MARKERS	HEMODYNAMICS
A At risk	Not currently experiencing signs or symptoms of CS, but at risk for its development	❒ Normal JVP ❒ Warm and well-perfused ❒ Strong distal pulses ❒ Normal mentation ❒ Clear lung sounds	❒ Normal lactate ❒ Normal (or at baseline) renal function	❒ SBP >100mmHg (or at baseline) ❒ CI ≥2.5 (if acute) ❒ CVP ≤10 ❒ PCWP ≤15 ❒ PA Sat ≥65%
B Beginning CS	Clinical evidence of hemodynamic instability without hypoperfusion	❒ Elevated JVP ❒ Warm and well-perfused ❒ Strong distal pulses ❒ Normal mentation ❒ Rales in lung fields	❒ Normal lactate ❒ Minimal acute renal function impairment ❒ Elevated BNP	❒ SBP <90mmHg, or MAP <60mmHg, or > 30mm drop from baseline ❒ HR ≥100bpm
C Classic CS	Hypoperfusion requiring intervention (pharmacologic or mechanical) beyond volume resuscitation	❒ Volume overload ❒ Altered mental status ❒ Cold and clammy ❒ Extensive rales ❒ Urine output <30mL/h	❒ Lactate ≥2 ❒ CR 1.5 x baseline or >50% drop in GFR ❒ Increased LFTs ❒ Elevated BNP	❒ CI <2.2 (if invasive hemodynamics performed [strongly recommended]) ❒ PCWP >15
D Deterio-rating	Failure of initial support strategy to restore perfusion	❒ Any of stage C and worsening (or not improving) signs/sx of hypoperfusion despite initial therapy	❒ Any of stage C and lactate rising and persistently ≥2 ❒ Deteriorating renal function ❒ Worsening LFTs ❒ Rising BNP	❒ Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of MCS device to maintain perfusion
E Extremis	Acute or impending circulatory collapse	❒ Typically unconscious ❒ Near pulselessness ❒ Cardiac collapse ❒ Multiple defibrillations	❒ Lactate ≥8 ❒ CPR ❒ Severe acidosis (pH <7.2)	❒ Profound hypotension despite maximal hemodynamic support ❒ Need for bolus doses of vasopressors
A (Arrest) Modifier	Cardiac arrest with concern for anoxic brain injury			

*Refer to 2022 SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies–Journal of the Society for Cardiovascular Angiography & Interventions (jscai.org) for further details

Endorsing societies:

American College of Cardiology (ACC)
American College of Emergency Physicians (ACEP)
American Heart Association (AHA)
Association for Acute Cardiovascular Care (ACVC)
European Society of Cardiology (ESC)
International Society for Heart and Lung Transplantation (ISHLT)
Society of Critical Care Medicine (SCCM)
Society of Thoracic Surgeons (STS)

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Approach to Identifying Early SCAI SHOCK

When to check:

- Cardiac arrest
- AMI and ADHF
- Clinical instability
- ICU admission

What to do:	Indications of Shock:
1. Monitor Vitals	SBP <90 mmHg MAP <60 mmHg HR >100 bpm
2. Assess Labs/Data	Elevated LFTs Elevated creatinine Urine output <30 ml/hr
3. Lactate and RHC	Lactate >2.0 mmol/L Cardiac index <2.2 L/min/m^2 CPO <0.6 Watts
4. Exclude Deterioration	Rising lactate Rising pressor requirements Rising MCS requirement Recurrent CPR

Early SCAI Shock are considered here as Stages B and C. AMI = acute myocardial infarction, ADHF = acute decompensated heart failure, ICU = intensive care unit, RHC = right heart catheterization, SBP = systolic blood pressure, MAP = mean arterial pressure, HR = heart rate, LFT = liver function test, CPO = cardiac power output, MCS = mechanical circulatory support, CPR = cardiopulmonary resuscitation. CPO = MAP*cardiac output/451.

Reference: Naidu S et al. SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies. JSCAI 2022. DOI: https://doi.org/10.1016/j.jscai.2021.100008.

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Simple Steps for SCAI SHOCK Staging

			*Observed In-Hospital Mortality No Cardiac Arrest	*Observed In-Hospital Mortality Cardiac Arrest
Step 1: Any Extremis?	□ Serum pH <7.2 □ High lactate >5-10 □ Recurrent CPR or pressor boluses	Any factor	Stage E **EXTREMIS**	55%	77%
Step 2: Any Deterioration?	□ Lactate ≥2.0 and rising □ Escalating pressors/MCS □ Worsening LFTs, creatinine, or UOP	Any factor	Stage D **DETERIORATING**	32%	54%
Step 3: Any Hypoperfusion?	□ Lactate ≥2.0 □ Any pressor/MCS support □ CI <2.2 or CPO <0.6, with hypoperfusion: altered mentation, cool extremities, ↑LFTs or creatinine, ↓UOP <30 ml/hr	Any factor	Stage C **CLASSIC / HYPOPERFUSION**	8%	40%
Step 4: Any Hypotension?	□ SBP <90 mmHg □ MAP <60 mmHg □ HR >100 bpm	Any factor	Stage B **BEGINNING / HYPOTENSION**	5%	26%

CPR = cardiopulmonary resuscitation, MCS = mechanical circulatory support, LFT = liver function test, UOP = urine output, CI = cardiac index, CPO = cardiac power output, SBP = systolic blood pressure, MAP = mean arterial pressure, HR = heart rate. CPO = MAP*cardiac output/451. Reference: Naidu S et al. SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies. JSCAI 2022. DOI: https://doi.org/10.1016/j.jscai.2021.100008. *Mortality rates reported from Jentzer JC et al. Cardiogenic shock classification to predict mortality in CICU. J Am Coll Cardiol 2019;74:2117-28.

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Optimal Interdisciplinary Shock Communication

Part 1: Initial Evaluation

History and Physical
Age, primary diagnosis, comorbidities, meds, allergies
Vitals, BMI, supplemental O_2, exam, SCAI SHOCK class

Hemodynamic Support
Current lines, drips and doses
Mechanical circulatory support, settings, complications

Laboratories
CBC, BMP+LFTs, troponin, BNP, procalcitonin
Lactate, ABG, SaO2, SvO2

Social History
Frailty, baseline function, social support, adherence

Code Status / Goals of Care

Part 2: Advanced Evaluation

Echocardiogram
BiV size/function, valvular and structural abnormalities, effusion, CO/CI, LVOT VTI, other pertinent details

Right Heart Cath
RA, RV, PA (systolic, diastolic, mean), PCW pressures
Calculated CO, CI, CPO, PAPi, PVR

Coronary Angiogram
Anatomy ± PCI, complications, antiplatelet therapy

Mechanical Circulatory Support
Settings, anticoagulation, limb perfusion, LV venting

Advanced Therapies Candidacy

BMI = body mass index, O_2 = oxygen, CBC = complete blood count, BMP = basic metabolic panel, LFT = liver function test, BNP = brain natriuretic peptide, ABG = arterial blood gas, SaO2 = arterial oxygen saturation, SvO2 = mixed venous oxygen saturation, BiV = biventricular, CO = cardiac output, CI = cardiac index, LVOT = left ventricular outflow tract, VTI = velocity time integral, RA = right atrium, RV = right ventricle, PA = pulmonary artery, PCW = pulmonary capillary wedge, CPO = cardiac power output, PAPi = pulmonary artery pulsatility index, PVR = pulmonary vascular resistance, PCI = percutaneous coronary intervention, LV = left ventricle. CPO = MAP*cardiac output/451. PAPi = (PA systole – PA diastole)/RA. Reference: Tehrani BN et al. A standardized and comprehensive approach to the management of cardiogenic shock. JACC Heart Fail 2020;8:879-91.

CABG Primer

CABG Crash Course

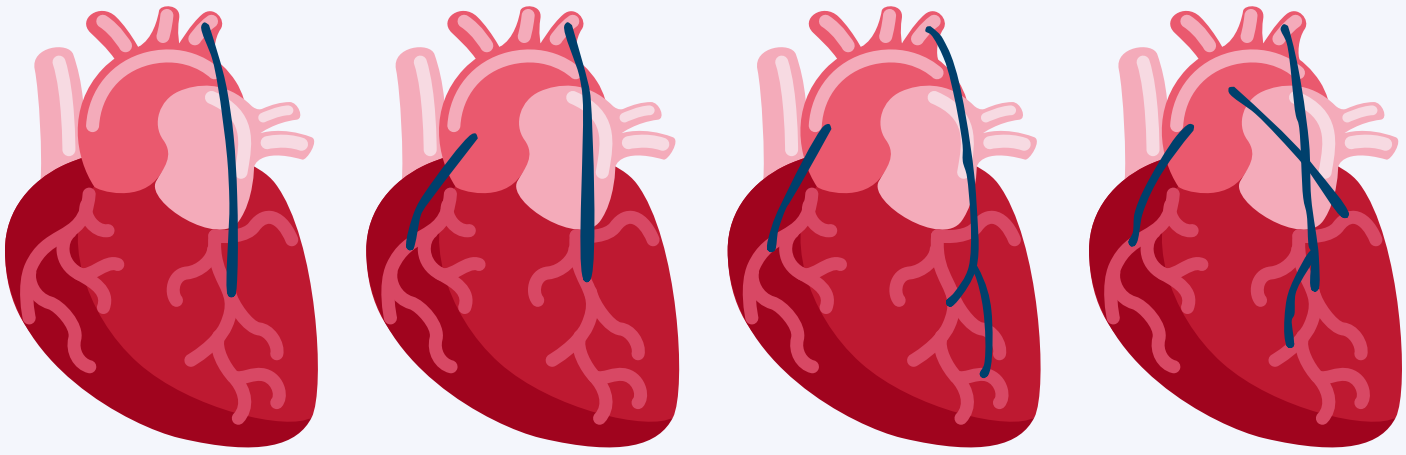
NURSE DOSE

Definition and Indication

- **Definition:** Coronary Artery Bypass Grafting (CABG): A surgical procedure used to improve blood flow to the heart in patients with coronary artery disease (CAD). During CABG, a surgeon uses a graft—often the internal mammary artery, a vein from the leg, or even an artery from the arm—to bypass blocked or narrowed coronary arteries, creating a new route for oxygen-rich blood to reach heart muscle tissue. CABG can be performed on-pump (with a heart-lung machine) or off-pump (heart beating), depending on the patient's needs.

- **Indications for CABG Surgery**:
  - **Severe Coronary Artery Disease (CAD):** When one or more of the major coronary arteries are significantly narrowed or blocked, especially in cases where:
    - The left main coronary artery, which supplies a large portion of blood to the heart, is narrowed by more than 50%.
    - Multiple coronary arteries are affected, especially if left ventricular function is impaired. ("Triple Vessel Disease")
  - **Persistent Angina:** When angina (chest pain) is unresponsive to medical therapy and lifestyle modifications, or if symptoms severely limit daily activities.
  - **Failed Percutaneous Coronary Intervention (PCI):** For patients whose stents or angioplasty did not relieve symptoms or failed over time.
  - **High-Risk CAD with Comorbidities:** CABG may be recommended for patients with diabetes, extensive coronary blockages, or those who have had a previous heart attack with reduced heart function (ejection fraction) to prevent future cardiac events.
  - **Emergent CABG:** In acute cases, such as after a heart attack, CABG may be performed emergently if PCI is not an option or if complications arise during an interventional procedure.

Bypass Terminology



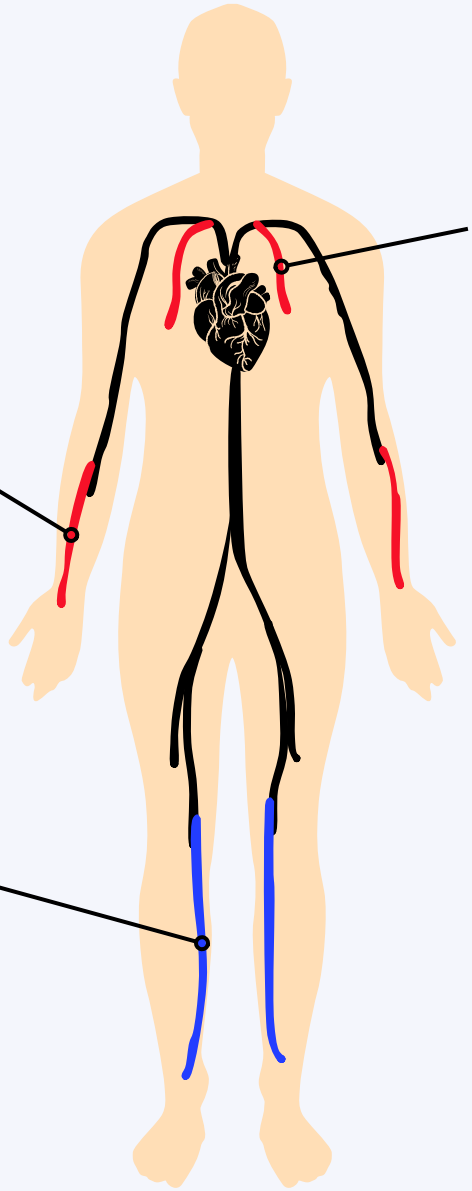
Single Double Triple Quadruple

In CABG, terms like single, double, triple, and quadruple bypass refer to the number of coronary arteries being bypassed due to blockages. For example, a double bypass means two arteries are blocked and each requires a separate graft to restore blood flow, while a quadruple bypass involves four arteries, indicating more extensive coronary disease.

Graft Sites

In Coronary Artery Bypass Grafting, choosing the right graft site is essential for restoring blood flow to blocked coronary arteries. Surgeons select grafts from various vessels around the body, each with unique advantages in terms of durability, patency, and accessibility. The most common grafts include arteries from the chest and arms or veins from the leg, but alternative sites are also available when additional or specific grafting needs arise. Each graft site option is chosen based on the patient's anatomy, the severity of their disease, and the best long-term outcomes for heart function.

The **radial artery**, taken from the arm, is a popular arterial graft for CABG because of its durability and resistance to plaque buildup, making it a good option when an additional artery is needed beyond the internal mammary artery. However, it has a higher risk of spasm compared to other grafts, so patients are often given medications like calcium channel blockers to help keep it open post-op.



The **internal mammary artery (IMA)** is the preferred graft in CABG, especially for bypassing the left anterior descending artery, due to its excellent long-term patency. Unlike other grafts, it typically remains attached to its original blood supply at the subclavian artery, reducing the need for complete harvesting and providing a consistent, durable blood flow.

The **saphenous vein** is harvested from the leg, often using a minimally invasive technique called endoscopic vein harvesting (EVH). This approach involves small incisions and a camera-guided tool to retrieve the vein, reducing scarring, infection risk, and recovery time for the patient. While versatile and easily accessible, the saphenous vein has a higher tendency to develop blockages over time compared to arterial grafts, making it less durable for long-term patency.

Other, less commonly used graft options in CABG include the **gastroepiploic artery** from the stomach lining and the **inferior epigastric artery** from the lower abdomen. These arteries are used when additional arterial grafts are needed and other primary options are unsuitable, though they are technically challenging due to their location and smaller size. In rare cases, the **ulnar artery** may also be used as an alternative to the radial artery if needed. Each of these grafts can be beneficial for specific cases, but they are typically reserved for patients who require additional grafting and have limited vessel options.

Surgical Procedure Overview for CABG

- **Incision and Sternotomy**
  - The surgeon performs a median sternotomy (opening of the chest) to access the heart.

- **Harvesting Grafts**
  - Common grafts include:
    - Internal Mammary Artery: Often preferred due to its long-term patency and durability. This artery remains connected at its origin.
    - Saphenous Vein: Taken from the leg, this vein graft is commonly used for bypassing multiple coronary vessels.
    - Radial Artery: Harvested from the arm, it is favored for its muscular structure and durability compared to veins, though it requires careful selection to ensure good flow to the hand.

- **Cardioplegia and Pump Initiation (On-Pump CABG)**
  - Initiating Cardiopulmonary Bypass: For on-pump procedures, the heart is connected to a cardiopulmonary bypass machine that takes over heart and lung functions.
  - Administering Cardioplegia: Cardioplegia (a cold potassium solution) is infused to stop the heart, allowing the surgeon to work on a still heart, minimizing oxygen demand and preserving myocardial tissue.
  - Off-Pump Consideration: In off-pump CABG, the heart remains beating, with stabilizers securing areas of the heart as the surgeon performs the bypasses without using the pump.

- **Coronary Vessel Exposure and Evaluation**
  - The target coronary arteries are identified, with graft sites chosen based on the location of blockages.

- **Anastomosis (Connecting Graft to Coronary Artery)**
  - Distal Anastomosis: Grafts are sewn to the coronary arteries beyond the blockage. Both vein and arterial grafts (such as the radial artery) are used based on durability and suitability for the specific artery needing bypass.
  - Proximal Anastomosis: If using saphenous vein grafts or radial artery grafts that aren't directly attached at their origin, they are connected to the aorta to establish blood flow.

- **Weaning from the Pump (On-Pump CABG)**
  - After confirming graft placement, the heart is gradually warmed, cardioplegia is stopped, and the heart resumes beating as the patient is weaned from bypass.

- **Hemostasis and Closure**
  - The surgical team ensures all grafts are functioning and secures hemostasis. Chest tubes are placed for drainage, and pacing wires may be added as needed.

On-Pump vs Off-Pump CABG

In CABG surgery, the surgeon may choose an on-pump or off-pump approach, based on the patient's condition and procedure complexity. Understanding each method helps ICU nurses anticipate specific post-op needs.

- **On-Pump CABG**
  - On-pump CABG uses a cardiopulmonary bypass machine to take over heart and lung functions, allowing the surgeon to work on a still heart. Cardioplegia stops the heart temporarily, which provides a clear and stable field for grafting, especially beneficial for complex cases.

- **Off-Pump CABG**
  - In off-pump CABG, the heart continues beating during surgery. Stabilizers keep parts of the heart steady for graft placement, eliminating the need for a bypass machine and potentially reducing pump-related complications. This method is often preferred for high-risk patients, like those with kidney issues.

Factor	On-Pump CABG	Off-Pump CABG
Heart Function	Stopped with cardioplegia	Beating throughout
Cardiopulmonary Bypass	Required; machine supports heart/lung functions	Not required; stabilizers used instead
Technical Difficulty	Easier for precise grafting on a still heart	More challenging due to beating heart
Risk of Stroke	Slightly higher due to pump use	Lower as pump is avoided
Recovery	May have more inflammation post-op	Generally faster recovery, less inflammation
Patient Suitability	Good for complex cases	Ideal for high-risk patients (e.g., kidney disease)
Common Complications	Stroke, bleeding, systemic inflammation	Patency issues, higher technical demands

Post-Op Management Priorities

After CABG surgery, patients require close, comprehensive monitoring and management to ensure a smooth recovery and to minimize the risk of complications. Here are key post-op priorities to focus on, each critical to supporting the patient's stability and long-term outcomes.

- **Hemodynamic Stability**
  - **Why It's Important:** CABG patients can experience rapid fluctuations in blood pressure and cardiac output post-op. This can result from the surgery itself, effects of anesthesia, or issues like vasoplegia.
  - **What to Monitor:** Close monitoring of MAP (mean arterial pressure), heart rate, and cardiac output helps ensure adequate perfusion to vital organs. Hypotension may require fluid resuscitation, inotropes, or vasopressors if caused by low systemic vascular resistance, especially in cases of vasoplegia.
  - **Management Tip:** Adjust medications based on invasive monitoring (like central venous pressure or arterial line) and aim for patient-specific MAP targets to avoid organ hypoperfusion.
    - **Commonly Used Medications for Hemodynamic Support**:
      - **Vasopressors** like norepinephrine, epinephrine, and vasopressin are often employed to maintain adequate blood pressure when hypotension is refractory to fluid resuscitation.
      - **Inotropes** such as milrinone may be used if the patient has low cardiac output due to poor myocardial function. These agents help improve contractility, especially important in cases where the heart muscle has weakened post-surgery.
    - **Choosing the Right Pressor:**
      - **Norepinephrine (Levophed)** is often the first-line vasopressor, as it provides strong alpha-adrenergic effects to increase systemic vascular resistance (SVR) with minimal impact on heart rate. It's commonly used in vasoplegia and general post-op hypotension.
      - **Epinephrine** can be used if there's a need to improve both blood pressure and cardiac output, as it has both inotropic and vasoconstrictive properties. It's useful for patients with low cardiac output and hypotension that isn't responding to norepinephrine alone.
      - **Vasopressin** is frequently used alongside norepinephrine to treat vasoplegia, especially in cases where hypotension is primarily due to low SVR. Vasopressin acts on different receptors than catecholamines, so it can be synergistic and effective in patients with refractory hypotension.
      - **Phenylephrine** is less commonly used but may be appropriate for patients who need purely alpha-agonist support without inotropic effects, like those with tachyarrhythmias.

- **Advanced Hemodynamic Monitoring**
  - **Purpose and Benefits:** Advanced monitoring, such as with pulmonary artery catheters (PAC) or continuous cardiac output (CCO) monitoring devices, provides detailed insights into parameters like cardiac output (CO), pulmonary artery pressures (PAP), systemic vascular resistance (SVR), mixed venous oxygen saturation (SVO2), etc. This level of monitoring helps clinicians precisely manage fluids, inotropes, and vasopressors, especially in patients with compromised cardiac function or those at risk for conditions like low cardiac output syndrome.
  - **When It's Used:** PACs or CCO devices are typically used in high-risk CABG patients—such as those with low ejection fraction, significant comorbidities, or a history of heart failure—where conventional monitoring may not provide sufficient information for optimal management.
  - **Management Tip:** Use data from advanced hemodynamic monitoring to guide fluid and medication adjustments, aiming for targeted perfusion goals. For example, PAC data can help differentiate causes of hypotension, such as hypovolemia versus vasodilation, allowing for more tailored treatment.

Post-Op Management Priorities Cont.

- **Respiratory Management**
  - **Why It's Important:** CABG patients are at risk for atelectasis and pneumonia post-extubation due to factors like limited mobility, pain, and the effect of anesthesia.
  - **What to Monitor:** Patients should be extubated as soon as possible when stable, then encouraged to use an incentive spirometer and perform deep breathing exercises. Close observation of oxygen saturation, respiratory rate, and lung sounds is important to identify any respiratory compromise early.
  - **Management Tip:** Encourage early mobilization, elevate the head of the bed, and ensure adequate pain control to support effective breathing.

- **Fluid Resuscitation**
  - **Why It's Important:** After CABG, patients often experience fluid shifts due to surgical stress, cardiopulmonary bypass effects, and possible blood loss. Effective fluid resuscitation ensures that blood volume is adequate to maintain cardiac output and blood pressure, which are essential for organ perfusion and graft function.
  - **What to Monitor:** Monitor CVP (central venous pressure), MAP (mean arterial pressure), and urine output as indicators of fluid status. Low urine output or hypotension may indicate the need for additional fluids. It's essential to balance fluid resuscitation with the risk of fluid overload, which can lead to pulmonary edema, particularly in patients with compromised cardiac function.
  - **Types of Fluids:** Usually start with crystalloids (such as plasmalyte) for initial resuscitation. In cases of significant blood loss or low hemoglobin, colloids or blood products (like packed RBCs) may be required to restore blood volume and oxygen-carrying capacity.
  - **Management Tip:** Use a conservative approach, avoiding excessive fluid loading, as CABG patients are often fluid-sensitive. Assess hemodynamic response to fluid boluses and consider using diuretics if signs of fluid overload develop.

- **Chest Tube and Drain Management**
  - **Why It's Important:** Chest tubes help prevent tamponade and other issues by draining excess blood or fluid from around the heart and lungs. Accumulated fluid or clots can increase pressure on the heart, leading to life-threatening complications.
  - **What to Monitor:**
    - Check drainage regularly—expect 100–200 mL in the first hour but watch for sudden increases or decreases. Excessive bleeding may require blood products or surgical intervention, while minimal drainage could indicate tube occlusion.
    - Monitor drainage amount, color, and consistency.
    - Check for an air leak in the water seal chamber; a continuous bubbling indicates an air leak that needs assessment.
  - **Management Tips:**
    - Gently milk the chest tube if there are signs of blockage, but avoid stripping, as this can increase pressure in the thoracic cavity.
    - Ensure secure connections to avoid air leaks and the chest tube is not kinked or obstructed.
    - Keep the collection device below chest level to promote drainage and prevent backflow.
    - Chest tubes are typically removed once drainage is minimal (usually less than 50-100 mL over 8 hours) and there are no signs of air leaks.
      - Apply a petroleum gauze and airtight dressing to the site post-removal to prevent air from entering the pleural space.

Post-Op Management Priorities Cont.

- **Arrhythmia and EKG Monitoring**
  - **Why It's Important:** Up to 30% of CABG patients experience post-op arrhythmias, with atrial fibrillation (AFib) being the most common. AFib can lead to hemodynamic instability, thromboembolism, or increased length of stay.
    - In addition to arrhythmia detection, EKG monitoring post-CABG is crucial for **graft surveillance**, as new changes in the EKG may indicate issues like graft occlusion or ischemia. Continuous EKG allows for early identification of ST-segment changes, which could signal compromised blood flow in the grafted vessels.
  - **What to Monitor:** Continuous ECG monitoring is essential. Watch for AFib or other arrhythmias like ventricular tachycardia. If AFib occurs, prompt rate or rhythm control (with beta-blockers, amiodarone, or, if needed, cardioversion) is crucial.
  - Know which arteries were bypassed so you know which leads to monitor for ST changes. (*Check out the STEMI Location Cheat Sheet for more in-depth education*)
    - For example: if the LAD was stented, you would want to be sure that the anterior leads were being monitored.
  - **Management Tip:** Early identification and treatment can minimize complications. Preemptive use of beta-blockers is common unless contraindicated, as it can reduce arrhythmia risk. Make sure your monitor is set so that the correct primary leads are selected for monitoring. Turn on ST monitoring if your monitor supports it.

- **Glycemic Control**
  - **Why It's Important:** Tight glycemic control in post-op CABG patients, even non-diabetics, has been shown to reduce infection rates and support better wound healing.
  - **What to Monitor:** Check glucose levels frequently, aiming to keep them within a target range (often 140–180 mg/dL in critically ill patients). Insulin drips or sliding-scale insulin may be used as part of post-op protocols.
  - **Management Tip:** Avoid hypoglycemia by closely adjusting insulin doses based on blood sugar trends, especially with changing dietary intake as patients transition from IV fluids to solids.

- **Early Mobility**
  - **Why It's Important**: Early mobilization is essential for preventing complications such as atelectasis, deep vein thrombosis (DVT), and post-op delirium. Mobilizing as soon as possible also supports overall recovery and improves circulation.
  - **What to Monitor**: Assess the patient's stability before mobilizing and ensure pain is well-controlled to facilitate movement. Start with sitting up and gradually progress to short walks under supervision.
  - **Management Tip**: Collaborate with physical therapy to establish a tailored mobility plan, aiming to have the patient out of bed as soon as tolerated.

- **Pain Control**
  - **Why It's Important:** Effective pain management is crucial for promoting deep breathing, coughing, and early mobility, which help prevent complications like atelectasis and DVTs.
  - **What to Monitor**: Regularly assess pain using pain scales, particularly in the first 48 hours post-op, and observe for signs of discomfort, like shallow breathing or guarded movements.
  - **Management Tip**: Encourage splinting by having the patient hold a pillow or folded blanket firmly against their chest when coughing, moving, or changing positions. This technique provides support and reduces pain during movement. Combine splinting with scheduled pain medications and non-pharmacological methods like repositioning, heat or cold applications, and relaxation techniques. Educate patients on the importance of taking pain relief proactively, especially before activities, to stay ahead of pain.

Miscellaneous CABG Management Insights

- **Sternal Precautions**
  - CABG patients are often given sternal precautions to protect the healing sternum, such as avoiding pushing, pulling, or lifting heavy objects. Educate patients to use the “heart hug” technique (holding a pillow against their chest) when coughing or moving.

- **Potential for Renal Dysfunction**
  - Many CABG patients develop acute kidney injury post-op due to hypoperfusion, inflammation, or effects of the heart-lung machine. Monitor creatinine and urine output closely, especially in patients with pre-existing kidney issues.

- **Postoperative Delirium and Cognitive Issues**
  - Post-op delirium and cognition issues are common, especially in elderly patients, due to factors like post-bypass pump, anesthesia, the stress response, and the ICU environment. Reduce delirium risk by orienting patients, minimizing sedatives, and encouraging early mobilization.

- **Medication-Centered Treatment for Post-Op Bleeding**
  - Antifibrinolytics (e.g., Tranexamic Acid or Aminocaproic Acid): These medications help stabilize clots by inhibiting fibrinolysis, the process that breaks down blood clots. They are often given prophylactically or administered post-op if bleeding is excessive.
  - Reversal Agents (e.g., Protamine for Heparin): Protamine is used to neutralize heparin, which may still be present in the patient’s system post-op. The dose is titrated based on the amount of heparin given during surgery.

- **Risk of Postoperative Pericarditis**
  - Post-op pericarditis can develop in CABG patients due to inflammation around the heart, typically presenting as chest pain that worsens with inspiration or when lying flat. Look for signs such as a pericardial friction rub and diffuse, concave ST-segment elevation across multiple EKG leads, often with PR-segment depression, which differentiates it from ischemic changes. Treatment generally includes anti-inflammatory medications, such as NSAIDs.

Emergent Resternotomy

Emergent Resternotomy

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What is an Emergent Resternotomy?

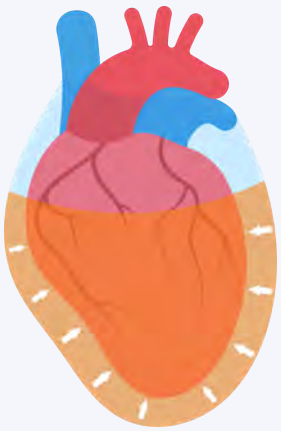
- **Definition**: An emergent resternotomy is the rapid reopening of a sternotomy incision (usually after some kind of cardiac surgery) at the bedside in order to manage life-threatening complications such as tamponade, increased internal bleeding, or even cardiac arrest.

- **Goal**: The primary goal of an emergent resternotomy is to rapidly address life-threatening complications by reopening the chest post-cardiac surgery. Doing so enables direct visualization and control of bleeding sources, evacuation of fluid compressing the heart, and stabilization of cardiac output. Ultimately, the goal is to quickly correct the mechanical or surgical issue causing the patient's instability, creating a stable physiological environment for recovery and further management.

Indications and Early Recognition

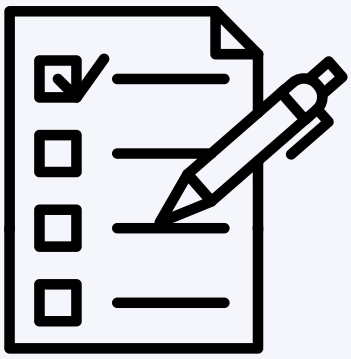
- **Potential Indications**
  - Sudden cardiovascular collapse or severe hemodynamic instability (e.g., refractory hypotension, acute low cardiac output). This includes cardiac arrest.
  - Evidence of tamponade (elevated CVP, pulsus paradoxus, diminished heart sounds).
  - Massive postoperative bleeding or significant drainage from chest tubes with hemodynamic compromise.

- **Early Warning Signs**
  - Vital Signs: Sudden drop in BP, narrowing pulse pressure, tachycardia.
  - Respiratory Changes: Increasing difficulty ventilating, decreased O2 saturation.
  - Clinical: Distended neck veins, acute mental status changes, weak peripheral pulses.



Preparation and Resources

Emergent resternotomy calls for rapid, organized action—and that starts with having the right supplies, staff, and environment ready at a moment's notice. Clear labeling of resternotomy kits, pre-positioning of essential medications and fluids, and well-defined role assignments are key to reducing response time and ensuring patient safety. This preparation not only streamlines the procedure itself but also gives the entire care team confidence that they can quickly address life-threatening complications when they arise.



Three areas in which we can be prepared.

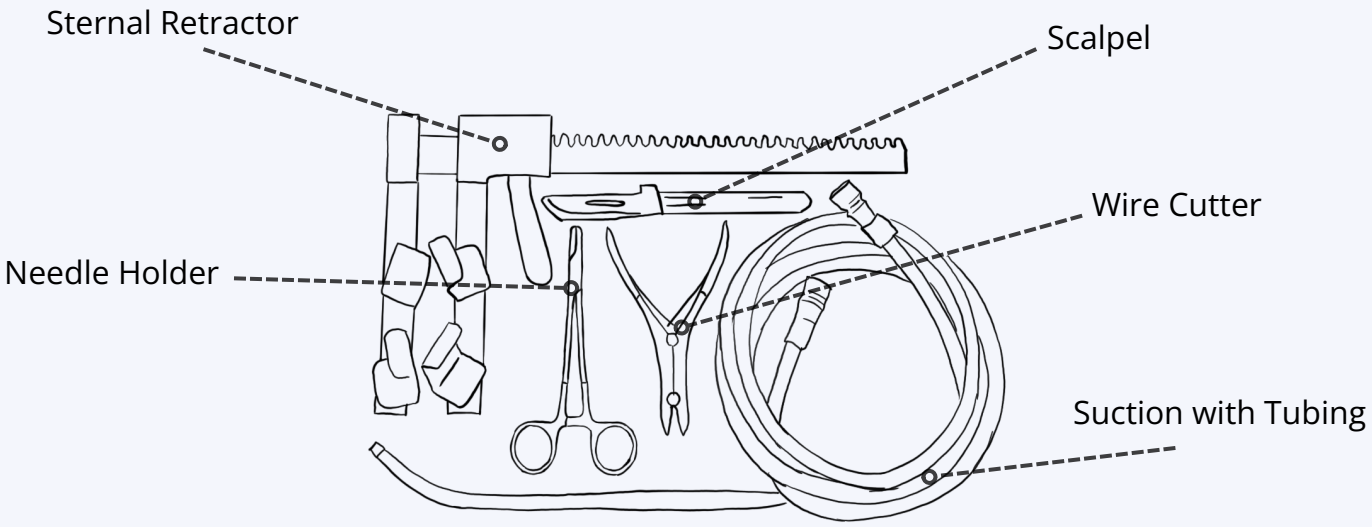
- Location of resternotomy equipment and how to access.
- Prepared medications and supplies.
- Staff role assignments.

Preparation and Resources cont.

Location of Equipment

Supplies and equipment necessary for an emergent resternotomy should be stored together and easily accessible. Many units often have all of this equipment stored together in an equipment cart, much like what you would see with a crash cart. This cart should have a designated spot on the unit and should not be moved unless needed. If a cart is not available for storage of the equipment then the equipment tray must be stored in a location that is easy for all staff members to access. It is important to note, many of these carts and trays have a lock on them so that equipment is not taken for procedures other than an emergent resternotomy. You may need to ask the charge or unit manager for the key/combination.

Prepared Supplies



There are 5 essential items that are needed for a resternotomy, however, much more might be in the cart. The 5 essential items are a wire cutter, scalpel, needle holder, sternal retractor, and suction with tubing.

If an emergent resternotomy cart is being utilized it may contain much more equipment. However, the tray of essential items is usually stored on top of the cart and secured closed.

The other items that may be included in the cart include (but are not limited to):

- PPE
- Gauze
- Irrigation/Suction Equipment
- Sutures/Staples/Clips
- Scalpels
- Defibrillation/Pacing equipment

It is very important that every drawer in the cart is labeled correctly and kept organized so that supplies can be retrieved rapidly.

Staff Role Assignments

Clearly defining staff roles in an emergent resternotomy scenario ensures a rapid and coordinated response. Below are five key nursing roles and their primary duties:

1. **Chest Cart Nurse**
   - **Primary Responsibility**: Manages the chest cart or resternotomy kit.
   - **Key Duties**:
     - Opens the cart and ensures availability of essential supplies.
     - Maintains organization of the cart and quickly hands off items as needed.
     - Keeps track of used or missing supplies for prompt restocking post-procedure.
2. **Circulating Nurse**
   - **Primary Responsibility:** Oversees the room setup and coordinates the overall workflow.
   - **Key Duties:**
     - Prepares the environment (adjusts bed height, lighting, etc.), ensures patient positioning and safety.
     - Maintains clear communication with the surgical team and other supporting staff.
     - Monitors the timeline, documents critical events, and facilitates any additional support needed.
3. **Scrub Nurse**
   - **Primary Responsibility:** Maintains a sterile field and assists the surgeon directly.
   - **Key Duties:**
     - "Scrubs in" to pass instruments, sutures, and supplies to the surgeon.
     - Monitors and manages the sterile environment.
     - Anticipates surgical needs and ensures smooth instrument exchange to minimize delays.
4. **Medication Nurse**
   - **Primary Responsibility:** Prepares and administers medications, including blood products.
   - **Key Duties:**
     - Draws up, labels, and provides medications as needed.
     - Collaborates with pharmacy and the medical team to ensure timely availability of drugs.
     - Documents all administered medications.
5. **Runner Nurse**
   - Primary Responsibility: Obtains additional supplies and manages external communication.
   - Key Duties:
     - Retrieves any equipment or resources not immediately available in the room.
     - Coordinates with other departments (e.g., lab, blood bank) to expedite lab results or blood products.
     - Acts as a liaison between the rest of the unit or hospital resources and the emergent resternotomy team.

Role assignments should be established at the start of each shift or as soon as the team anticipates a potential need for emergent resternotomy. By assigning these distinct roles in advance, the team ensures clarity, prevents duplication of tasks, and fosters swift decision-making. This organization streamlines communication and ultimately improves patient safety and outcomes.

Overview of Possible Step-by-Step Protocol

Emergent resternotomy procedures benefit greatly from having a clear, step-by-step outline to guide the entire care team. However, it's important to recognize that every hospital or unit may have its own specific protocols based on available resources, staffing, and institutional policies. The following steps offer a general framework.

1 Recognize the Need

- **Identify Clinical Signs:**
  - Hypotension, tachycardia, narrowed pulse pressure, elevated CVP, distended neck veins, altered mental status, etc.
  - Excessive chest tube output or no output with clinical deterioration (suggesting tamponade).
  - Rapidly expanding hematoma in the chest or around the sternum.
- **Immediate Assessment:**
  - Confirm alarms and hemodynamic readings (art line, CVP/PA catheter if present).
  - Perform quick bedside evaluation for tamponade (muffled heart sounds, pulsus paradoxus, JVD).

2 Mobilize the Team

- **Notify Key Personnel:**
  - Surgeon on-call.
  - Anesthesia, perfusionists (if needed), and any advanced practice providers.
  - Charge nurse or resource nurse.
- **Overhead Page or Dedicated Code:**
  - Per facility guidelines if applicable.
- **Mobilize Nursing Team:**
  - Assign roles (Chest Cart Nurse, Circulating Nurse, Scrub Nurse, Medication Nurse, Runner Nurse).

3 Prepare

- **Bed & Patient Setup:**
  - Move the bed away from the wall, ensure 360° access if possible.
  - Remove or reposition bed attachments, drapes, etc., for rapid access to the sternum.
  - Ensure adequate lighting and room temperature (patients can rapidly become hypothermic).
- **Gather Equipment:**
  - Chest Tray: Wire cutter, scalpel, needle holder, sternal retractor, suction with tubing.
  - Surgical Instruments: Additional clamps, retractors, suture material.
  - Medication & Resuscitation Supplies: IV fluids, blood products, pressors, analgesics, and sedatives.
  - Monitoring: Ensure arterial line and central line transducers are functioning.
  - Have defibrillator on standby.

4 Ensure Patient Stability & Sedation

- **Airway Management:**
  - Confirm endotracheal tube is secure or prepare for intubation if not already intubated.
  - Increase FiO2 to 100% or per protocol.
- **Hemodynamic Support:**
  - Administer fluid boluses or blood products if indicated (guided by protocols or MD orders).
  - Start or titrate vasopressors/inotropes to maintain adequate perfusion.
- **Sedation & Pain Control:**
  - Administer IV sedation (e.g., propofol, benzodiazepines, or other agents) per anesthesia or MD orders.

5 Sterile Preparation & Surgical Opening

- **Staff Scrub & Gown:**
  - Scrub Nurse and Surgeon (or advanced practitioner) don sterile attire.
  - Chest Cart Nurse remains "clean" to manage resternotomy cart if not scrubbing in.
- **Incision & Sternum Entry (Performed by Surgeon or Authorized Provider):**
  - Remove dressings to fully expose the surgical site.
  - Open the incision with a scalpel along the existing sternal incision line.
  - Use wire cutters to cut through sternal wires (be mindful of wire ends and disposal).
  - Carefully separate the sternum (manual spread or sternal retractor).
  - Evacuate any hematoma or clots to relieve pressure on the heart.

6 Control Bleeding & Inspect Grafts

- **Identify Source of Bleeding (Performed by Surgeon or Authorized Provider):**
  - Check anastomoses, graft sites, suture lines, or the heart itself for obvious bleeds.
  - Suction and sponging to keep the field clear.
- **Achieve Hemostasis:**
  - Clamp or tie off bleeding vessels.
  - Apply pressure with laparotomy sponges or hemostatic agents as needed.
  - Use electrocautery (if available) or suture repair to definitively control bleeding sites.

7 Ongoing Hemodynamic & Procedural Support

- **Monitor Vitals Continuously:**
  - Reassess BP, arterial line waveforms, central venous pressures, and ECG.
  - Adjust vasopressors/inotropes based on real-time measurements and MD orders.
- **Hand & Document Blood Products:**
  - Transfuse PRBCs, FFP, platelets, or cryoprecipitate as needed to correct coagulopathy.
  - Keep accurate input-output (I/O) documentation, including chest tube output.
- **Maintain Communication:**
  - Coordinate with anesthesia, perfusion (if involved), and other team members.
  - Update the team on the patient's status and anticipated next steps.

8 Closure

- **Assess Whether to Close or Leave Sternum Open:**
  - If bleeding is controlled and the heart is stable, surgeon may re-approximate the sternum with wires or place a temporary closure device.
  - If instability persists (e.g., significant edema or ongoing bleeding risk), the chest may be left open with vacuum-assisted closure or packing.
- **Place Drainage Tubes:**
  - Position chest tubes to facilitate ongoing drainage of blood or fluid.
  - Secure and label all drainage lines.

9 Post-Procedure Stabilization

- **Vital Signs & Hemodynamic Monitoring:**
  - Continue close observation of arterial lines, central venous pressure, and potential signs of re-bleeding or tamponade.
- **Medications & Labs:**
  - Check coagulation studies (PT/INR, aPTT, fibrinogen), CBC, electrolytes.
  - Adjust sedation, analgesia, and vasopressors per MD orders and unit protocols.
- **Document Thoroughly:**
  - Times of critical events (incision start, chest open, closure, total blood product used).
  - All medications, interventions, and patient responses.

10 Ongoing Communication & Handoff

- **Multidisciplinary Collaboration:**
  - Communicate patient status and changes to ICU team.
  - Involve family communication as soon as feasible to provide updates.
- **Handoff Procedures:**
  - Ensure the next shift or covering team receives a concise overview of interventions, blood product usage, wound status, and any pending labs or follow-ups.

Cardiac Arrest & Cardiac Massage

Cardiac arrest in a postoperative cardiac surgery patient is a time-critical emergency where conventional advanced life support measures may be less effective or contraindicated. External chest compressions can disrupt newly placed grafts, damage suture lines, or worsen bleeding, making emergent resternotomy the preferred intervention in many cases. Once the chest is opened, direct cardiac massage can be performed, providing more effective circulation than external compressions. This approach allows the surgical team to address underlying causes such as tamponade, massive hemorrhage, or graft failure. Additionally, internal defibrillation using sterile internal paddles can deliver lower-energy shocks directly to the myocardium, reducing the risk of myocardial injury while improving the success of rhythm conversion. This hands-on approach not only restores circulation but also enables immediate surgical correction of life-threatening complications.

Key Reminders and Proactive Measures

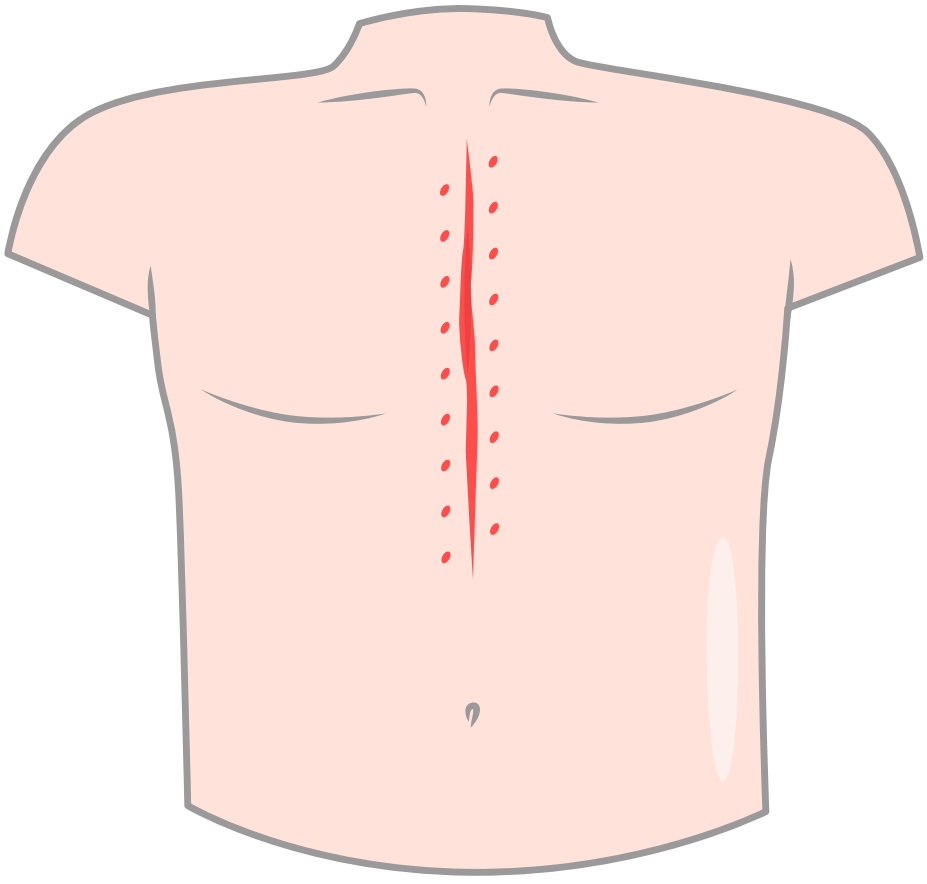
Staying ahead of potential complications and remaining prepared for emergent situations are crucial aspects of successful resternotomy care.

- **Identify High-Risk Patients**
  - Patients with known coagulopathies, platelet dysfunction, low ejection fraction, or multiple prior sternotomies are at greater risk for needing an emergent resternotomy. Recognizing these risk factors early allows the care team to monitor more closely, maintain a lower threshold for intervention, and prepare equipment or resources preemptively.

- **Verify Equipment Readiness**
  - Regularly check that the resternotomy kit, crash cart, and all associated supplies are fully stocked, functioning, and within expiration dates. A quick equipment review at the start of each shift ensures you won't be scrambling for essentials when seconds matter.

- **Engage in Practice Drills**
  - Scheduled simulations help refine technical skills and reinforce each team member's role during an emergent resternotomy. By rehearsing communication protocols and equipment management in a controlled setting, staff can reduce errors and improve response times when confronted with real-life emergencies.

- **Know Your Team & Resources**
  - Maintain an up-to-date roster of key personnel, including contact numbers and escalation pathways (charge nurse, perfusionist, surgeon on-call). Familiarity with who to call and how to mobilize resources quickly enhances coordination and streamlines patient care during critical moments.



CRRT Primer

CRRT CHEAT SHEET

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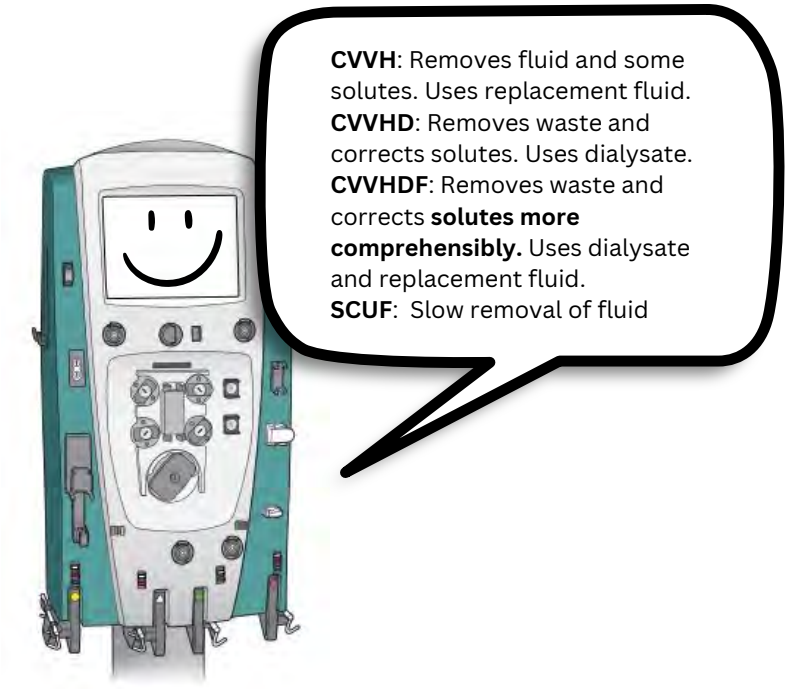
What is CRRT?

- Continuous Renal Replacement Therapy (CRRT) is used for critically ill patients with acute kidney injury/severe kidney dysfunction among other reasons.
- It involves the continuous removal of waste products, excess fluids, and electrolytes from the blood.
- CRRT operates continuously, providing gradual and precise fluid and solute balance adjustments.
- It is particularly beneficial for hemodynamically unstable patients as it offers slower fluid removal and more tolerable renal support in the intensive care setting.



Indications for CRRT

General Indications for CRRT

- Volume Overload
- Drug/Toxin overdose
- Poor Kidney Function
- Acid-Base Imbalances

More Specific Indications for CRRT

- Low urine output
- Increased BUN/Cr
- Acute Kidney Injury
- Increase Potassium: Hyperkalemia
- Hypo/Hypernatremia
- Pulmonary Edema
- Third Spacing
- Acidemia when pH is less than 7.1
- Pericarditis (Uremic especially)
- Encephalopathy

Principles of CRRT

1. Diffusion: Diffusion operates based on the principle of concentration gradients. As blood flows through a semipermeable membrane in the dialysis filter, solutes like urea and creatinine move from areas of higher concentration in the blood to areas of lower concentration in the dialysate fluid. This movement is driven by the difference in solute concentrations across the membrane, facilitating the removal of waste products and helping to restore proper electrolyte balance in patients with acute kidney injury.
2. Ultrafiltration: Ultrafiltration is another key principle alongside diffusion. It involves the removal of fluid from the bloodstream through the semipermeable membrane of the dialysis filter. Pressure differentials across the membrane drive the movement of water molecules, allowing excess fluid to pass from the blood into the dialysate or replacement fluid. Ultrafiltration helps in controlling fluid overload, a common issue in critically ill patients with kidney dysfunction
3. Convection: Convection involves the movement of solutes across a semipermeable membrane along with fluid flow, driven by pressure differentials. As blood passes through the dialysis filter, solutes are carried across the membrane with the ultrafiltrate, aiding in their removal from the bloodstream. Solute drag refers to the dragging force exerted on solutes as they are carried along with the ultrafiltrate during convection

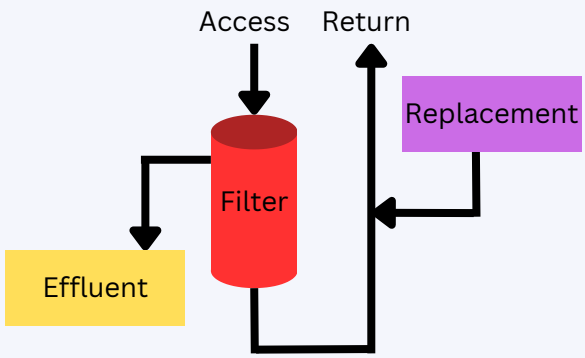
CRRT MODES

NURSE DOSE

Continuous Veno-Venous Hemofiltration (CVVH)

Description: blood is pumped from a vein, passed through a hemofilter to filter waste products and remove excess fluids, and then the filtrate is replaced with a balanced solution. CVVH provides gentle fluid removal as well as some solute removal and is suitable for hemodynamically unstable patients.

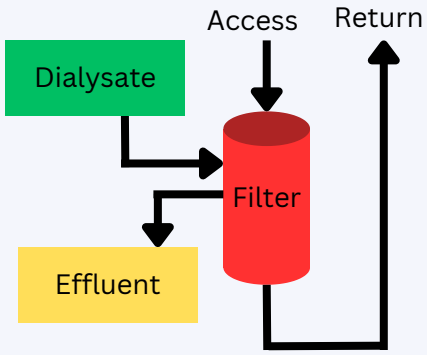
Principles Used: Ultrafiltration and convection. No dialysate. Requires replacement fluid either pre- or post filter.



Continuous Veno-Venous Hemodialysis (CVVHD)

Description: blood is pumped from a vein and passed through a hemofilter. In CVVHD, the hemofilter also allows for additional solute removal through diffusion via dialysate, making it effective for patients with specific electrolyte imbalances

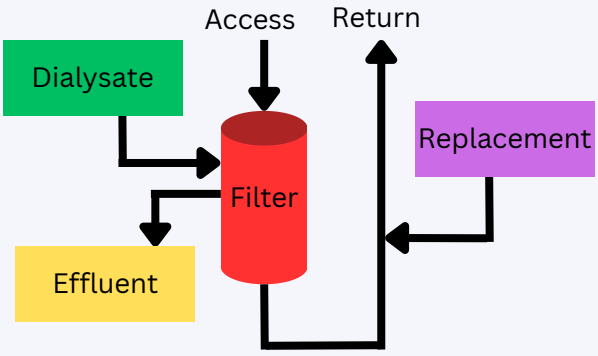
Principles Used: Diffusion, some ultrafiltration. No replacement fluid necessary.



Continuous Veno-Venous Hemodiafiltration (CVVHDF)

Description: This mode combines both hemofiltration and hemodialysis, allowing for a more comprehensive removal of waste products and solutes. It is suitable for patients with severe kidney dysfunction and significant solute imbalances.

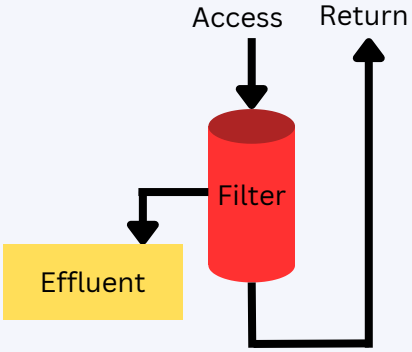
Principles Used: Ultrafiltration, Convection, Diffusion. Both dialysate and replacement fluid are utilized.



Slow Continuous Ultrafiltration (SCUF)

Description: In SCUF, the emphasis is primarily on fluid removal rather than solute clearance. It involves the slow and continuous removal of excess fluids without significant solute removal. SCUF is useful for patients with fluid overload but stable electrolyte levels. It provides a more gradual approach to fluid management, which can be beneficial in certain clinical scenarios.

Principles Used: Ultrafiltration, very little convection



CRRT TMP/PRESSURE DROP & ORDERS

NURSE DOSE

Pressure Drop and Transmembrane Pressure(TMP)

1. Pressure Drop: In CRRT, pressure drop refers to the decrease in pressure that occurs as blood moves through the circuit. This drop in pressure is influenced by various factors such as the diameter and length of the tubing, the resistance within the filter, the viscosity of the blood, and the flow rate of the blood. A pressure drop is essential to drive the movement of blood through the circuit and facilitate the filtration or dialysis process. However, excessive pressure drop can indicate issues such as clotting within the filter, kinks or obstructions in the tubing, or inadequate blood flow rates. Monitoring pressure drop helps ensure proper blood flow and effective therapy during CRRT.
2. Transmembrane Pressure (TMP):TMP refers to the pressure gradient across the semipermeable membrane of the filter used in CRRT. It represents the difference in pressure between the blood side and the filtrate (or dialysate) side of the filter membrane. TMP is a critical parameter in CRRT because it drives the movement of fluid and solutes across the membrane during filtration or dialysis. A sufficient TMP is necessary to achieve adequate ultrafiltration rates and clear toxins and excess fluid from the patient's blood. However, excessively high TMP can indicate issues such as filter clogging/clotting, membrane fouling, or excessive resistance within the circuit. Healthcare providers monitor TMP closely during CRRT to ensure it remains within the optimal range for effective therapy while minimizing the risk of complications such as filter clotting or hemolysis (rupture of red blood cells).

Anatomy of a CRRT Order

1. CRRT Mode: Specify the chosen CRRT mode. CVVH, CVVHD, CVVHDF, SCUF
2. Flow Rates: The prescription should indicate the desired flow rates for blood (QB) and effluent (QF). Blood flow rate determines the amount of blood passing through the CRRT circuit per unit of time, and effluent flow rate corresponds to the rate at which waste products and excess fluids are removed from the patient.
3. Replacement Fluid Rate (Qr): If the patient requires additional fluids, the Qr is prescribed to maintain fluid balance.
4. Anticoagulation Protocol: Specify the anticoagulant agent, dosage, and mode of administration (e.g., continuous infusion) to prevent clotting in the CRRT circuit and filter.
5. Filter Type and Size: Specify the type and size of the hemofilter being used for the CRRT treatment.
6. Prescribed Effluent Composition: If using CVVHDF, the prescription may include the desired composition of the effluent, including the concentration of bicarbonate, calcium, and other electrolytes.
7. Ultrafiltration Rate: Set the desired rate of fluid removal (ultrafiltration) based on the patient's fluid balance requirements.
8. Monitoring Parameters: Indicate the target ranges for critical lab values (e.g., BUN, creatinine, potassium, sodium, pH) to guide adjustments in the CRRT prescription.

CRRT COMMON ALARMS

NURSE DOSE

"Access Extremely Negative"

Description:

This alarm occurs when the pressure required to pull blood from the patient becomes more negative than set limit.

Causes:

- Patient coughing/moving
- Access Line is kinked
- Catheter is clotted

Actions:

- Fix any kinks in the tubing
- Flush or reposition catheter per hospital protocol
- Lower blood flow rate
- Wait for patient to stop moving/coughing.

"Return Extremely Positive"

Description:

This alarm occurs when the pressure required to return blood to the patient becomes more positive than set limit.

Causes:

- Patient coughing/moving
- Return Line is kinked/clamped
- Catheter is clotted

Actions:

- Fix any kinks in the tubing
- Flush or reposition catheter per hospital protocol
- Lower blood flow rate
- Wait for patient to stop moving/coughing.

"TMP excessive"

Description:

Transmembrane Pressure exceeds membrane pressure limit.

Causes:

- Ultrafiltration rate is too high.
- Inadequate anticoagulation in the circuit.

Actions:

- Decrease PBP, replacement fluid and/or UF rate.
- Increase blood flow rate.
- Adjust anticoagulation
- Change circuit if filter clotted.

External Pacing Primer

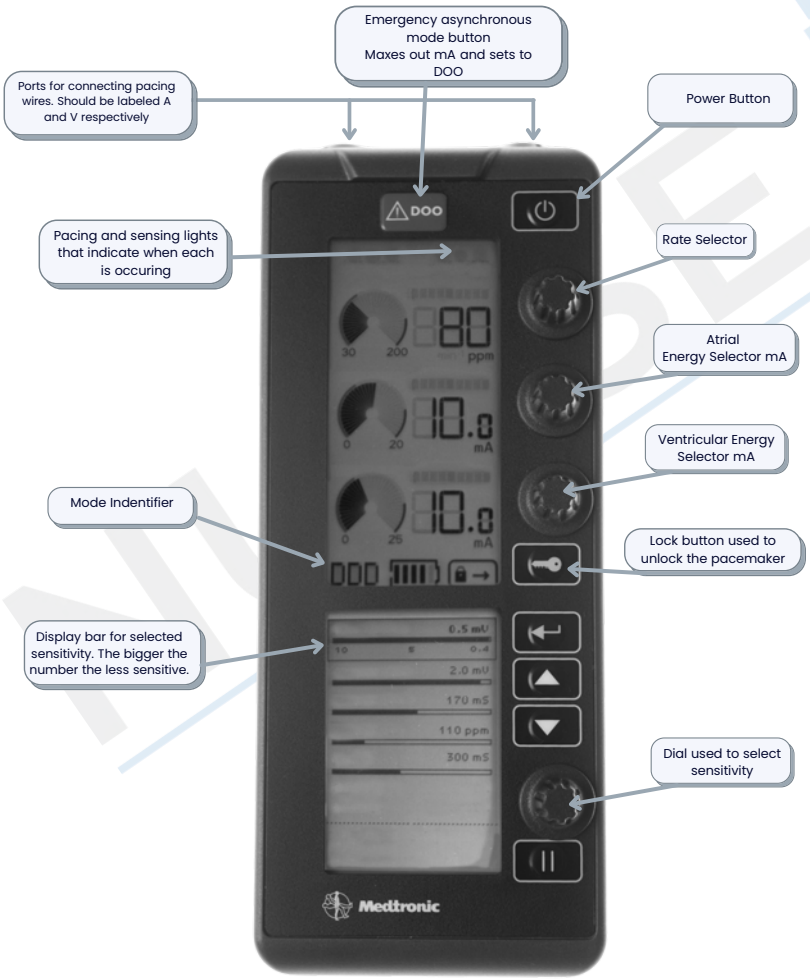
External Pacer Cheat Sheet

NURSE DOSE

PACER SETTINGS		
CHAMBERS PACED	CHAMBERS SENSED	RESPONSE
O = NONE	O = NONE	O = NONE
A = ATRIUM	A = ATRIUM	I = INHIBIT
v = VENTRICLE	v = VENTRICLE	T = TRIGGERED
D = DUAL	D = DUAL	D = DUAL

VVI

COMMON PACER MODES	
DDD	Paces and senses both chambers. Great for patients with both SA and AV node dysfunction
AAI	Paces the atrium as well as senses it. Inhibits pacing if atrium is sensed. For patients with SA node dysfunction
VVI	Paces and senses the ventricles. Inhibits pacing if ventricle is sensed. Great for a-fib without RVR.
DOO	Paces both the atrium and ventricles without sensing. Used mostly in emergent situation.



USES

The primary indications for pacemaker initiation are **heart block** and **bradycardia**.

Pacemakers are particularly common in patients that have recently undergone cardiac surgery, especially valve replacements/repair. This is due to the heart block that can occur due to inflammation caused by certain procedures that disrupt the SA and AV node.

Pacemakers may also be used by providers to overdrive pace arrhythmias. This is done by selecting a rate that is faster than the arrhythmia to override it then decreasing rate once the arrhythmia is disrupted. This should always be done with a provider present and an appropriate order.

Special Considerations

The rate programmed into the pacemaker should only be changed per provider order.

The pacemaker battery should be checked Qshift and changed per unit protocol.

Underlying rate can be analyzed by using the pause button on the pacemaker. Pacer cables should never be taken out of the pacemaker.

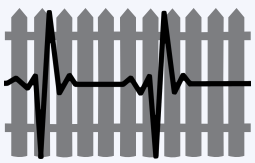
Energy output (mA) should be set to an appropriate level. Thresholds should be done per unit protocol. Keeping the output at too high of a level could cause scar tissue to develop and decrease efficiency of the pacemaker.

Sensitivity

Sensitivity determines how much current must be detected before the pacemaker identifies an depolarization event.

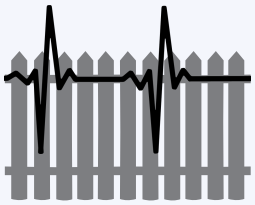
Sensitivity must be adjusted so that the proper events are detected and to avoid artifacts from interfering with rhythm detection.

You can think of sensitivity as a fence. The higher the number (mV) the higher the fence and the less sensitivity.



mV 3

While on the other hand, if the mV number is low (lowered fence) the sensitivity is greater.



mV 1

External Pacer Troubleshooting

NURSE DOSE

Failure to Pace

-Switch out battery or pacemaker unit

-Check pacer wire placement for dislodgement.

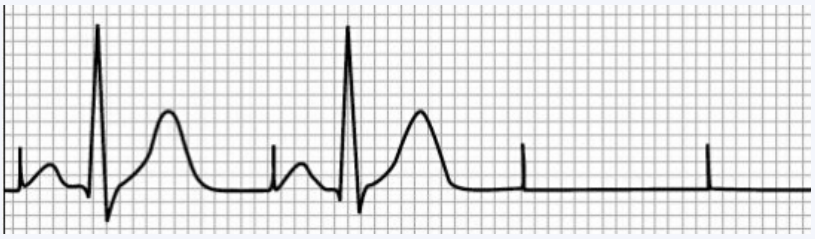
-Check cable connection.

Failure to Capture

-Switch out battery or pacemaker unit

-Increase power output (mA)

-Check pacer wires are in correct position.

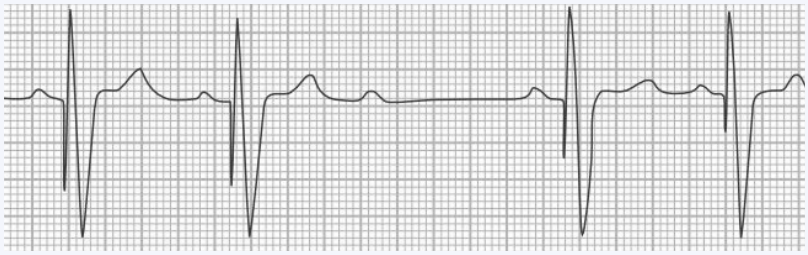


Oversensing

Oversensing occurs when the pacemaker mistakes artifact or other electrical waves for the QRS and inhibits pulse generation.

-Decrease sensitivity, increase mV

-Check connections



Undersensing

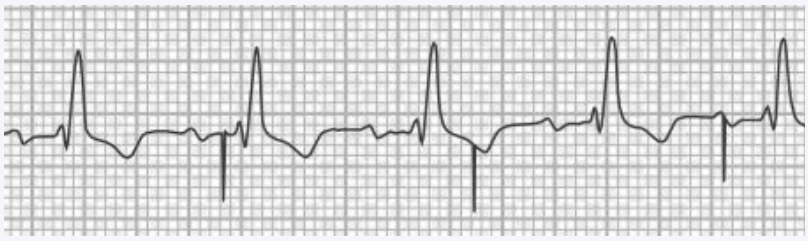
Undersensing occurs when the pacemaker does not detect the intrinsic rhythm and generates pulses inappropriately.

-Check battery

-Check connections

-Increase sensitivity, decrease mV

Warning: This could cause a dysrhythmia to develop. Undersensing must be fixed asap.

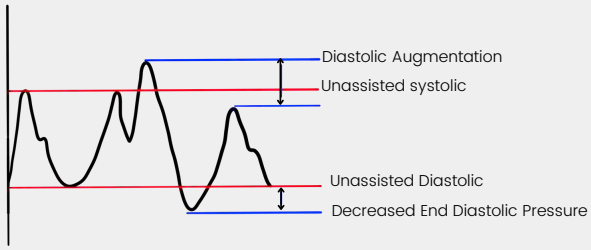


IABP Primer

Balloon Pump Cheat Sheet

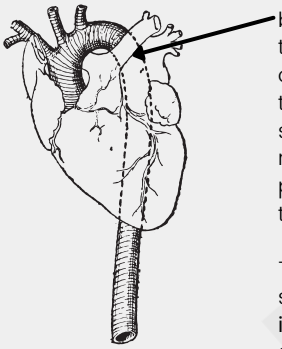
NURSE DOSE

WAVEFORM BASICS



The purpose of the balloon pump is to increase aortic diastolic pressure (Diastolic Augmentation) and decrease left ventricular end diastolic pressure in order to increase coronary perfusion pressure. **CPP = DBP - LVEDP**

CORRECT PLACEMENT



The balloon should be placed adjacent to the inferior aspect of the aortic arch so that the left subclavian artery is not occluded and to prevent occlusion of the renal arteries.

The radiopaque tip should lie in the **2nd intercostal space** on a chest X-ray.

TYPES OF TRIGGERS

ECG Trigger

The balloon will deflate at the peak of the R wave. Inflation will be triggered in the middle of the T wave.

Pressure Trigger

The arterial waveform is used to trigger the balloon pump. Inflation is triggered at the dicrotic notch.

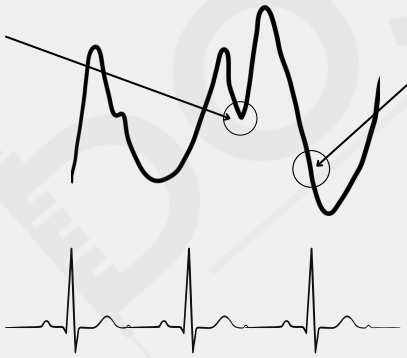
Internal Trigger

This is an asynchronous mode that is set during cardiac arrest or bypass. Usually set at 80 bpm.

PROPER TRIGGERING

Proper Inflation

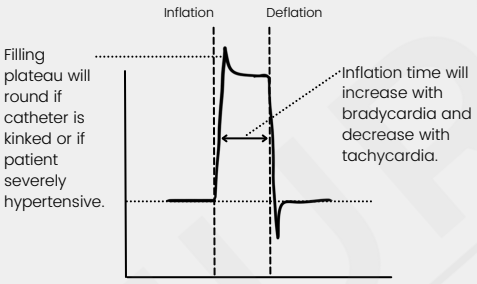
The proper point of inflation is at the diuretic notch. Should correlate approximately with the T wave.



Proper Deflation

The proper point of deflation is at the end of diastole and should correlate with the peak of the R wave.

BALLOON WAVEFORM



A sudden decrease in the baseline pressure of the balloon could indicate a balloon rupture, triggering an alarm.

MODES OF OPERATION

Automatic

The computer picks the most reliable trigger source and sets the inflation and deflation time.

Semi-Automatic

The operator chooses the source and sets the initial inflation and deflation times. The computer will then determine subsequent intervals.

RATIO OF AUGMENTATION

1:1
Augmentation will occur with every beat.

1:2
Augmentation will occur with every other beat.

1:3
Augmentation will occur with every third beat.

Weaning is often done by gradually decreasing the rate of augmentation from 1:1 to 1:3

ASSESSMENT

-Assess MAP from IABP waveform and titrate drips accordingly.

-Monitor pedal and radial pulses on the same side that the balloon pump is inserted. Absent pulses could indicate migration and blockage of arteries.

-Monitor urine output. A decrease in urine output could indicate balloon migration and occlusion of the renal arteries.

-Monitor insertion site for any signs of bleeding or hematoma.

-Monitor helium tubing for any signs of leak or blood. Could indicate balloon rupture.

-Make sure a chest X-ray is done at least every 24 hours to confirm placement.

Miscellaneous

Augmentation alarm should be set 10mmHg below the augmented diastolic pressure. Decreased augmentation could indicate need for vasopressor support.

Check helium tank levels QShift.

Never power flush or draw from the IABP arterial line. If needed, get a provider order to do so.

Patients with a pacer should still be on ECG trigger unless there is not a pronounced R wave.

Patients with afib should still use ECG trigger but pressure trigger will work as well.

Impella Primer

Impella Cheat Sheet

NURSE DOSE

General Information and Indications

The Impella device is a small ventricular assist device that helps pump blood from the ventricle systemically. Aids in maintaining systemic circulation between 2.5 and 5.0 L/min depending on the specific device used.

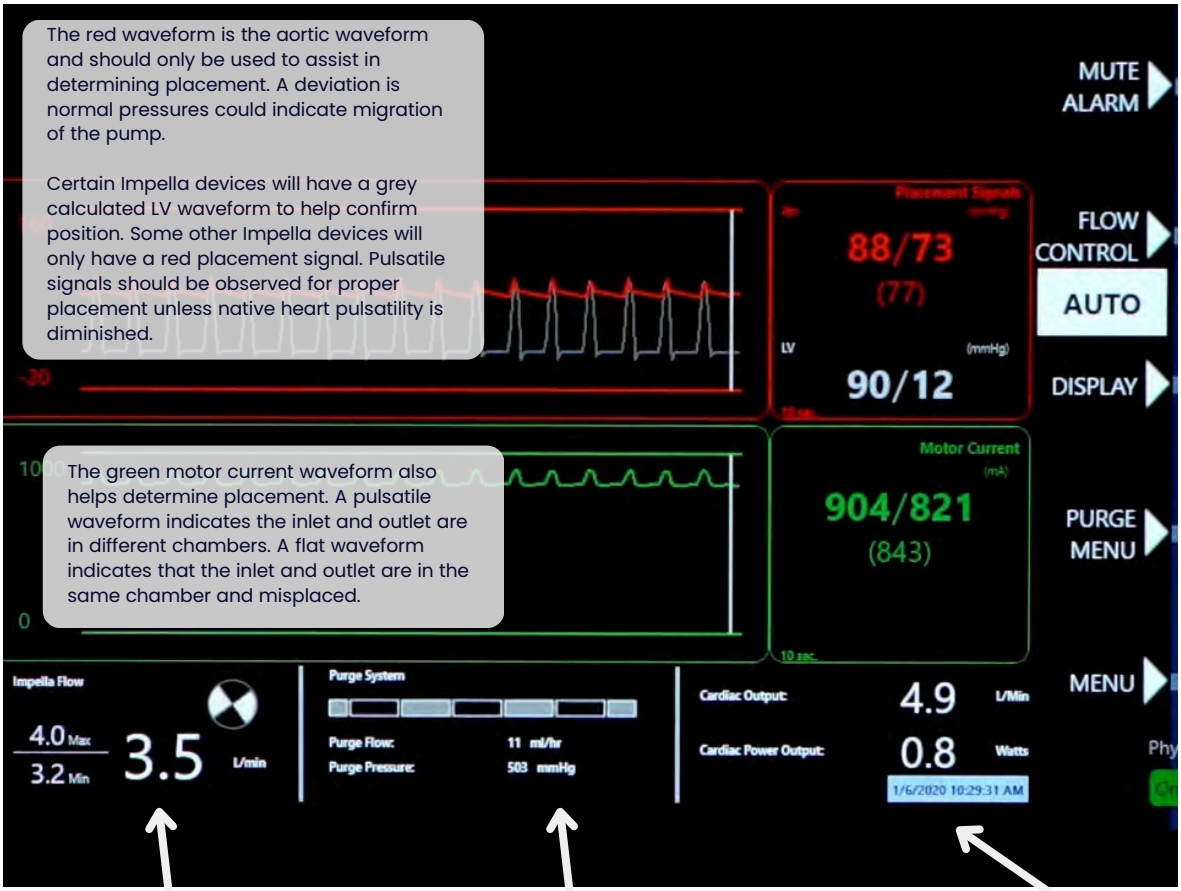
Indications

- Treatment of MI with Cardiogenic shock
- Aid in "High Risk" PCI
- Treatment of acute heart failure decompensation
- Off pump coronary bypass surgery

Placement

- Impella 2.5 and CP: Femoral artery approach
- Impella 5.5: Axillary approach, requires arteriotomy
- Impella RP: Femoral vein approach

All left sided Impella devices will cross aortic valve and have inlet (distal) in ventricle with outflow in ascending aorta.
RP device bypasses RV with inlet in IVC and outflow in pulmonary artery.



The red waveform is the aortic waveform and should only be used to assist in determining placement. A deviation is normal pressures could indicate migration of the pump.

Certain Impella devices will have a grey calculated LV waveform to help confirm position. Some other Impella devices will only have a red placement signal. Pulsatile signals should be observed for proper placement unless native heart pulsatility is diminished.

The green motor current waveform also helps determine placement. A pulsatile waveform indicates the inlet and outlet are in different chambers. A flat waveform indicates that the inlet and outlet are in the same chamber and misplaced.

White Alarm: Notification

Yellow Alarm: Advisory

Red Alarm: Immediate Action necessary

P-level and weaning

The P-Level determines the RPM of the impella motor which in turn determines the flow rate of the device. P-0 is the lowest level and correlates with the motor not running. P-9 is the highest P-level and correlates to the highest RPM and flow rate the device can produce.

Per Abiomed, to initiate weaning the MD should reduce P-level in 2 level increments as hemodynamics tolerate.

When removing impella, P-Level should be reduced to P-0 after patient stable at P-2.

The Impella Flow screen shows the minimum (diastole) and maximum (systole) flow along with the average flow in L/min.

The purge solution prevents debris buildup in the Impella pump. Abiomed now recommends using 25 mEq of sodium bicarbonate in D5W, especially for patients intolerant to heparin. Monitoring purge pressure is vital: low pressure may indicate a leak, while high pressure can signal a kink or obstruction.

Cardiac output and calculated Cardiac Power output. Cardiac power output represents heart pumping ability. CPO of less than 0.6 is associated with hemodynamic compromise and increased mortality.

ASSESSMENT

- Proper impella placement should be checked by the provider after transportation from cath lab to the unit.
- Thereafter, careful monitoring of both the placement signal and the motor current waveform should be done.
- Assess entry site periodically for bleeding and possible migration.
- Assess urine color to check for any hemolysis. Pink tinged urine could indicate hemolysis is occurring.
- To reduce chance of suction against ventricle walls, the patient's volume status should always been maintained.
- All alarms should be given immediate attention. Troubleshooting advice will accompany the onscreen alarm.

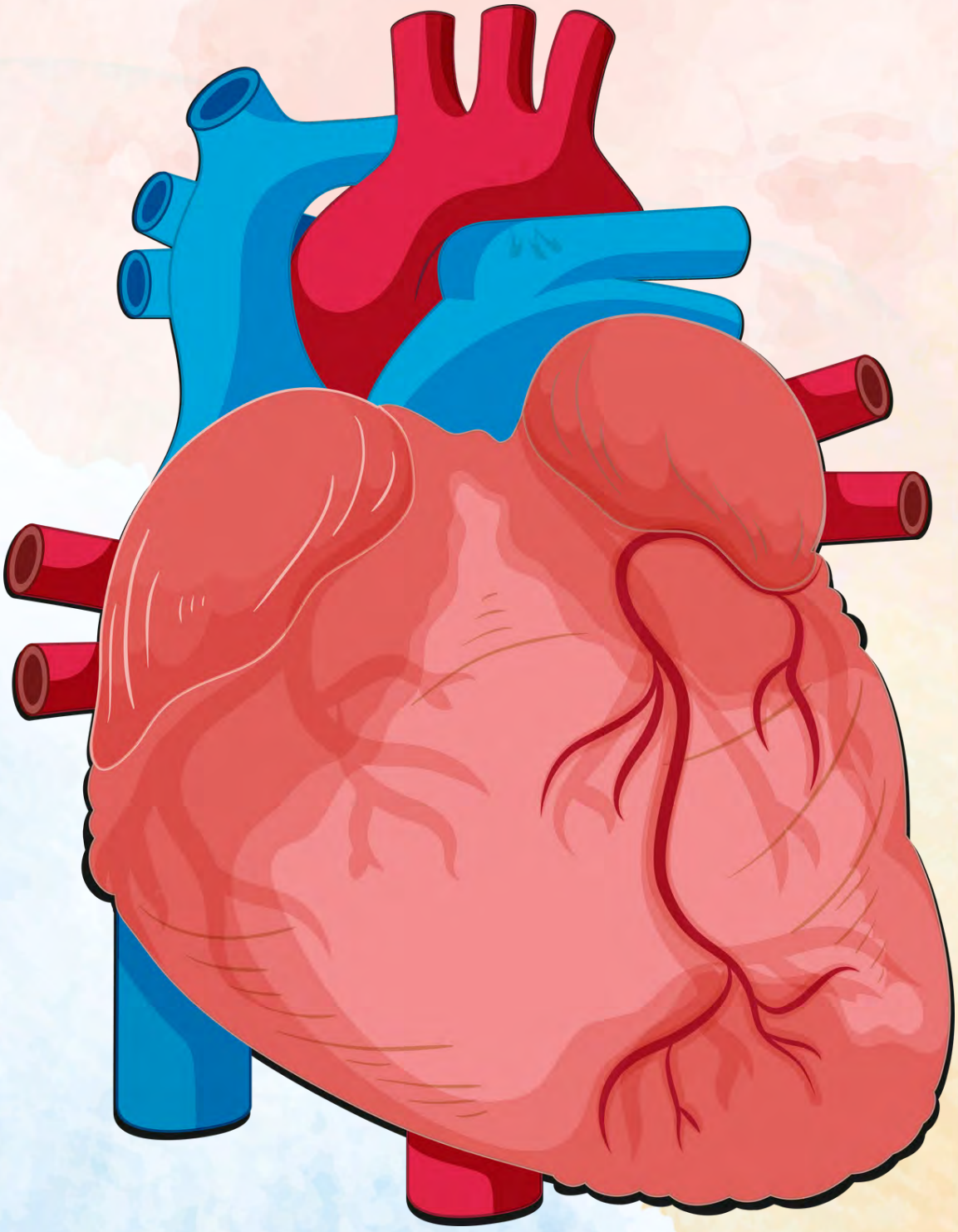
Miscellaneous

- If hemolysis is suspected and positioning is correct, aim to increase volume if CVP and/or wedge pressure is <10 mmhg.
- If the pump position is wrong, have provider reposition under echo guidance in P-2.
- For high purge pressure alarms check to see if any cabling is kinked.
- If unable to resolve purge pressure alarms evaluate the motor current waveform. An increase in motor current could indicate failure of impella pump in likely.
- If CPR is initiated while on impella therapy, per Abiomed, the p-level should be reduced to P-2 until cardiac function is reestablished.

Rhythm & EKG Primer

HEART RHYTHM & EKG BUNDLE

STUDY GUIDE FOR NURSING STUDENTS



FAMZ NURSING NOTES

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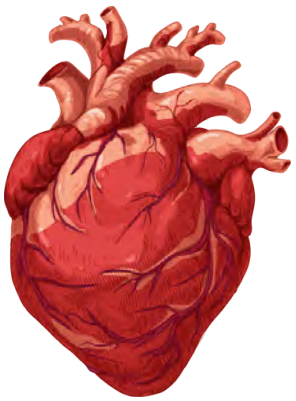
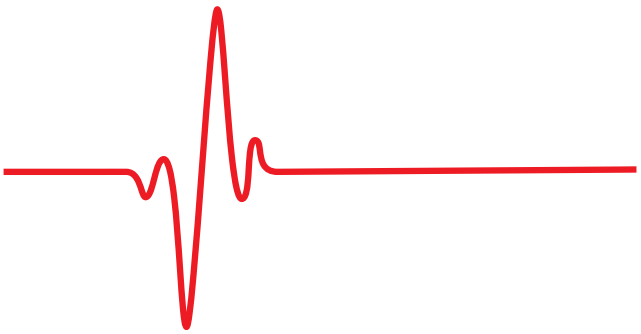
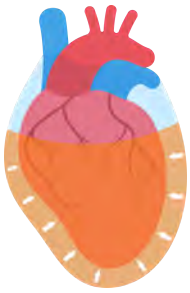




HEART RHYTHM & EKG

INTRODUCTION

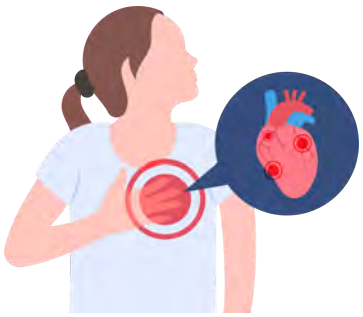


Heart rhythm and interpreting electrocardiograms (EKGs) is a crucial skill for nursing students, especially those planning to work in cardiology, critical care, or any field where cardiac monitoring is essential.

1. Anatomy of the Heart

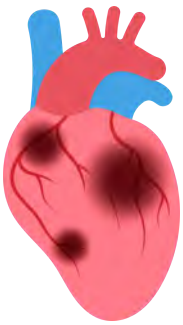
- The heart is a muscular organ divided into four chambers: two atria and two ventricles.
- The heart's primary function is to pump blood throughout the body, delivering oxygen and nutrients while removing waste products.

2. Electrical Conduction System of the Heart



- The heart has its electrical system that controls the rhythmic contractions.
- Key components include the sinoatrial (SA) node, atria, atrioventricular (AV) node, bundle of His, and Purkinje fibers.

3. Normal Sinus Rhythm (NSR)

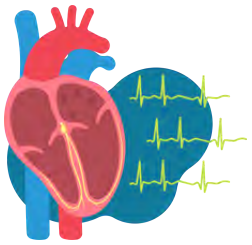


- NSR is the standard electrical pattern of the heart, originating in the SA node.
- It consists of a P wave, QRS complex, and T wave.
- The normal heart rate ranges from 60 to 100 beats per minute.

4. EKG Basics

- Electrocardiogram (EKG or ECG) is a graphical representation of the heart's electrical activity.
- Leads, or electrodes, are placed on the patient's skin to record electrical signals from different angles.
- The standard EKG includes 12 leads.

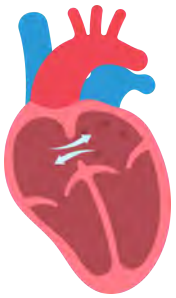
5. Key EKG Waves and Intervals



- **P wave:** Represents atrial depolarization.
- **QRS complex:** Depicts ventricular depolarization.
- **T wave:** Reflects ventricular repolarization.
- **PR interval:** Measures the time from atrial depolarization to ventricular depolarization.
- **QT interval:** Represents ventricular depolarization and repolarization.

6. Common Rhythm Disturbances

- **Atrial Fibrillation (AFib):** Irregular atrial contractions with no distinct P waves.
- **Ventricular Fibrillation (VFib):** Chaotic, uncoordinated ventricular contractions.
- **Bradycardia:** Heart rate below 60 bpm.
- **Tachycardia:** Heart rate above 100 bpm.



7. Interpretation

- Analyze the rhythm strip systematically: rate, rhythm, P waves, PR interval, QRS complex, and T wave.
- Assess for abnormalities and identify the type of rhythm disturbance.

8. Nursing Interventions

- Administer medications as prescribed (e.g., antiarrhythmics).
- Monitor vital signs, oxygen saturation, and symptoms.
- Prepare for cardioversion or defibrillation in emergencies.
- Educate patients about their heart condition and medications.

9. Documentation

- Document EKG findings accurately.
- Note any changes in rhythm, heart rate, or patient symptoms.
- Maintain clear and organized records.

10. Continuous Learning

- Stay updated with the latest guidelines and research in cardiology.
- Practice EKG interpretation regularly to improve your skills.

EKG BASICS

WHAT IS AN EKG/ECG?

- An electrocardiogram (EKG or ECG) is a graphical representation of the electrical activity of the heart.
- It records the electrical impulses generated by the heart as it contracts and relaxes, creating a visual representation of the heart's rhythm and electrical conduction.

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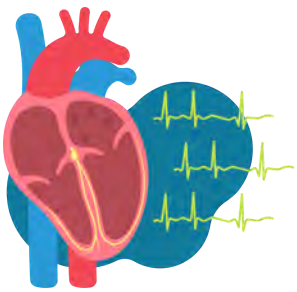
- It records the electrical impulses generated by the heart as it contracts and relaxes, creating a visual representation of the heart's rhythm and electrical conduction.

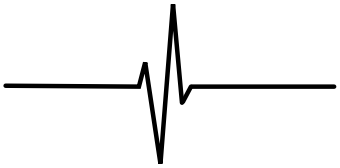
ELECTRODES AND LEADS

- Electrodes (also called leads) are placed on the patient's skin to detect electrical signals from different angles.
- There are 12 standard leads used in a 12-lead EKG, each providing a unique view of the heart's electrical activity.

EKG WAVES AND INTERVALS

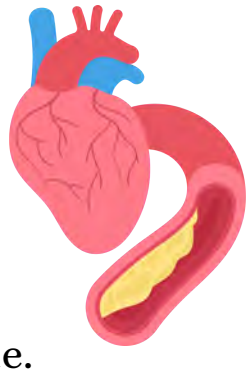
- **P wave:** Represents atrial depolarization (contraction).
- **QRS complex:** Depicts ventricular depolarization (contraction).
- **T wave:** Reflects ventricular repolarization (relaxation).
- **PR interval:** Measures the time from atrial depolarization to ventricular depolarization.
- **QT interval:** Represents ventricular depolarization and repolarization.





LEAD PLACEMENT

- The standard limb leads (I, II, III, aVR, aVL, aVF) monitor electrical activity in the frontal plane.
- The precordial leads (V1 to V6) monitor electrical activity in the transverse plane.
- Proper lead placement is crucial for accurate EKG readings.



EKG WAVES AND INTERVALS

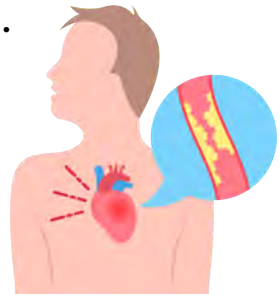
- NSR is the standard electrical pattern of a healthy heart.
- The normal heart rate ranges from 60 to 100 beats per minute.
- NSR includes a P wave followed by a QRS complex and a T wave.

INTERPRETING EKGS

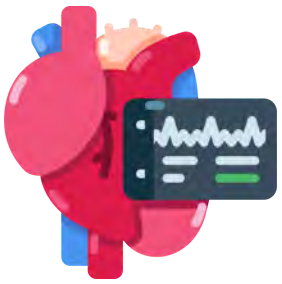
- Begin by assessing the rhythm (regular or irregular).
- Analyze the rate (count the number of QRS complexes in a 6-second strip and multiply by 10 to get the beats per minute).
- Examine the P waves, QRS complexes, and T waves for abnormalities.
- Measure the PR interval and QT interval, noting any deviations from normal.

COMMON EKG FINDINGS

- **Atrial Fibrillation (AFib):** Absence of distinct P waves with irregularly spaced QRS complexes.
- **Bradycardia:** Heart rate below 60 bpm.
- **Tachycardia:** Heart rate above 100 bpm.
- **ST-segment elevation or depression:** May indicate myocardial infarction (heart attack).
- **Bundle Branch Blocks**: Delayed conduction in the ventricles.



NURSING IMPLICATIONS



- Monitor patients during EKG recording.
- Educate patients about the procedure and the need for proper lead placement.
- Report any abnormal findings promptly to healthcare providers.
- Administer medications or interventions as ordered based on EKG results.

EKG MONITORING

- Continuous EKG monitoring is essential in critical care units and for patients at risk of arrhythmias.
- Telemetry units allow for continuous cardiac monitoring outside of critical care settings.

CONTINUOUS LEARNING

- Keep learning and practicing EKG interpretation to improve your skills.
- Seek mentorship and guidance from experienced nurses and cardiology specialists.

EKG Parameter	Normal Range
PR Interval:	0.12-0.20 sec
QRS Interval:	0.06-0.10 sec
ST Segment:	0.08 sec
QT Interval:	0.32-0.50 sec

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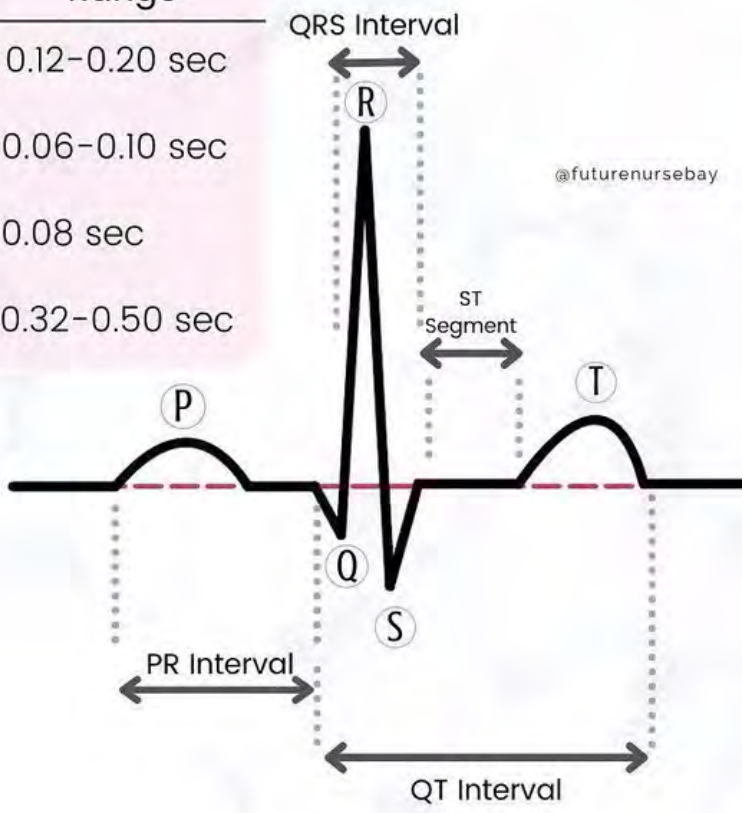
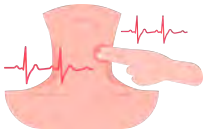


CARDIAC PHYSIOLOGY

Cardiac physiology is the study of how the heart functions to pump blood and supply oxygen and nutrients to the body's tissues.

HEART ANATOMY

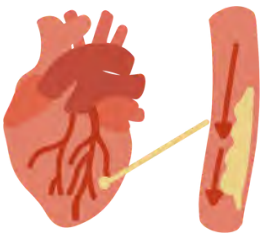


- The heart is a muscular organ located in the chest cavity.
- It has four chambers: two atria (right and left) and two ventricles (right and left).
- Valves, such as the atrioventricular (AV) valves (tricuspid and mitral) and semilunar valves (pulmonary and aortic), ensure one-way blood flow.

BLOOD FLOW THROUGH THE HEART

- Deoxygenated blood returns to the right atrium from the body via the superior and inferior vena cava.
- The right atrium contracts, pushing blood through the tricuspid valve into the right ventricle.
- The right ventricle contracts, sending blood to the lungs for oxygenation through the pulmonary valve and pulmonary arteries.
- Oxygenated blood returns to the left atrium via the pulmonary veins.
- The left atrium contracts, forcing blood through the mitral valve into the left ventricle.
- The left ventricle contracts, pumping oxygenated blood throughout the body via the aortic valve and aorta.

CARDIAC CYCLE



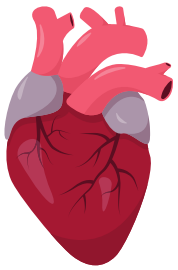
- The cardiac cycle consists of systole (contraction) and diastole (relaxation) phases.
- During systole, the ventricles contract, and blood is ejected into the pulmonary and systemic circulation.
- During diastole, the ventricles relax and fill with blood from the atria.

ELECTRICAL CONDUCTION SYSTEM

- The heart has its electrical system that controls heartbeat.
- The sinoatrial (SA) node generates electrical impulses, initiating each heartbeat.
- The impulses travel through the atria, causing atrial contraction.
- The atrioventricular (AV) node delays the impulse briefly to allow the ventricles to fill.
- The impulse then travels along the bundle of His and Purkinje fibers, causing ventricular contraction.

CARDIAC OUTPUT

- Cardiac output (CO) is the amount of blood the heart pumps per minute.
- It is calculated as CO = Heart Rate (HR) x Stroke Volume (SV).
- Normal adult CO is around 4-8 liters per minute.

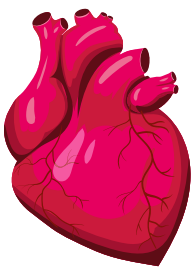


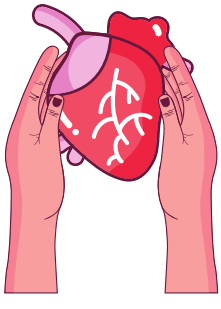
BLOOD PRESSURE

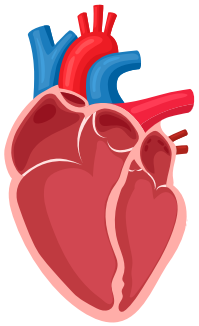
- Blood pressure (BP) is the force exerted by blood against the walls of arteries.
- BP is measured in millimeters of mercury (mm Hg) and consists of systolic (ventricular contraction) and diastolic (ventricular relaxation) pressures.

HEART SOUNDS

- The "lub-dub" sounds of the heart are produced by heart valves closing during the cardiac cycle.
- S1 (lub) is the closure of the AV valves (tricuspid and mitral) at the start of systole.
- S2 (dub) is the closure of the semilunar valves (aortic and pulmonary) at the start of diastole.

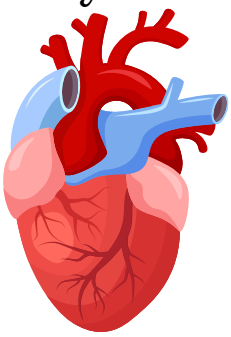






REGULATION OF CARDIAC OUTPUT

- The autonomic nervous system, hormones (e.g., epinephrine, norepinephrine), and factors like preload, afterload, and contractility influence cardiac output.

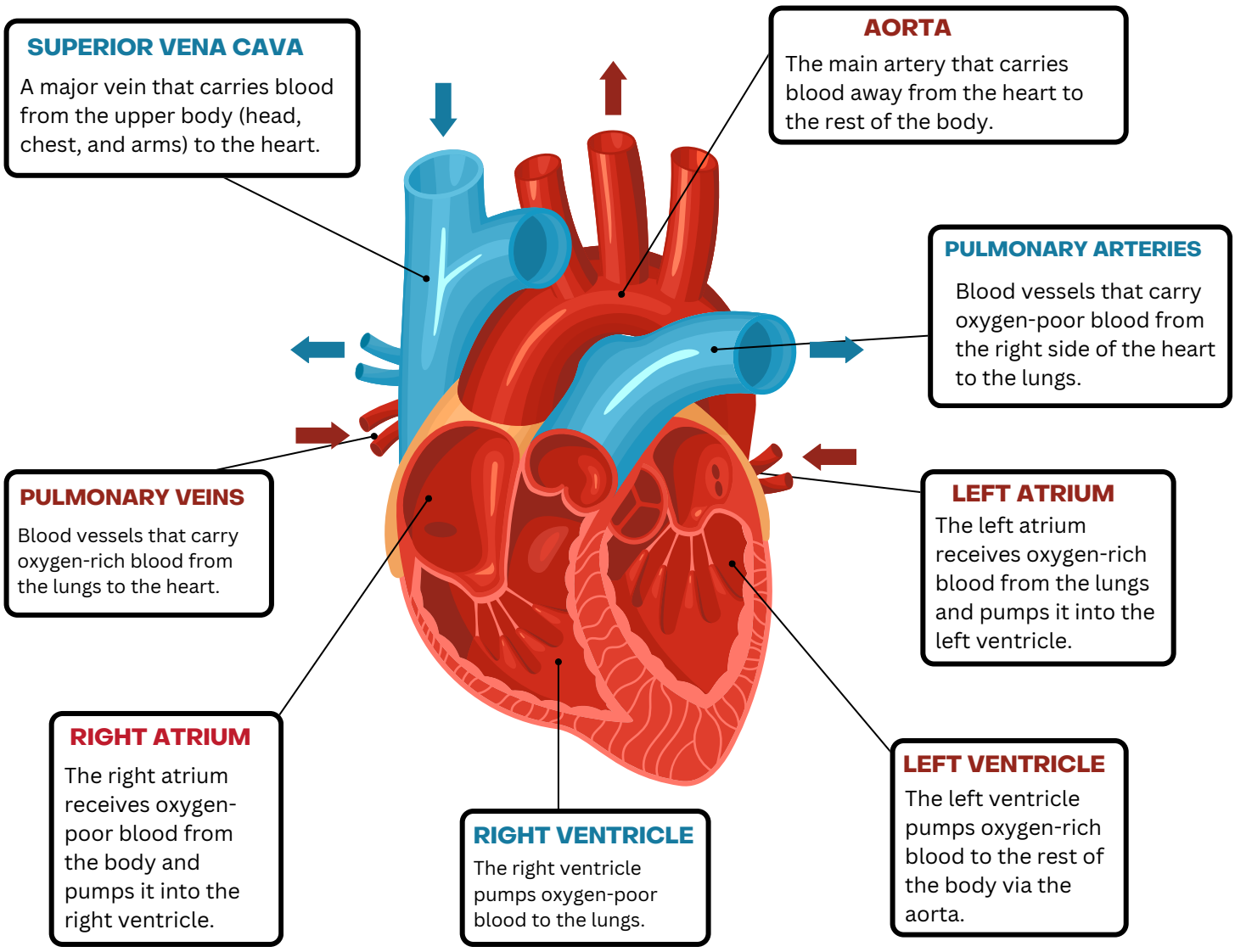


NURSING IMPLICATIONS

- Assessing vital signs, including heart rate and blood pressure.
- Monitoring for signs and symptoms of cardiac issues.
- Administering medications as prescribed to manage heart conditions.
- Educating patients on heart-healthy lifestyles and medication adherence.

PATHOPHYSIOLOGY

- Nursing students should also learn about common cardiac disorders like heart failure, coronary artery disease, arrhythmias, and hypertension.



CARDIAC CONDUCTION SYSTEM

- The heart's electrical impulses follow a specific pathway: SA node → atria → AV node → bundle of His → bundle branches → Purkinje fibers.
- The SA node sets the heart's rhythm, but other parts of the conduction system can take over if needed.

CARDIAC CYCLE PHASES:

- **Preload:** The amount of blood in the ventricles at the end of diastole, determining the stretch of cardiac muscle fibers.
- **Afterload:** The resistance the heart must overcome to eject blood into the systemic circulation.1
- **Contractility:** The force generated by the myocardium to eject blood.

FRANK-STARLING LAW

- This law describes the relationship between preload and stroke volume: as preload increases, stroke volume increases up to a certain point.
- It explains how the heart can adjust its output based on the volume of blood returning to it.

AUTONOMIC NERVOUS SYSTEM (ANS) CONTROL

- The ANS regulates heart rate and contractility.
- The sympathetic nervous system increases heart rate and contractility (fight-or-flight response), while the parasympathetic nervous system (vagus nerve) decreases heart rate (rest-and-digest response).

HORMONAL REGULATION

- Hormones like epinephrine and norepinephrine from the adrenal glands can increase heart rate and contractility in response to stress.
- The renin-angiotensin-aldosterone system (RAAS) regulates blood pressure and fluid balance.

16. CARDIAC OUTPUT (CO) DETERMINANTS

- **Heart Rate (HR):** Controlled by the SA node and ANS.
- **Stroke Volume (SV):** Affected by preload, afterload, and contractility.

Increasing HR or SV can increase CO.

OXYGEN SUPPLY AND DEMAND

- Myocardial oxygen demand increases during periods of increased workload (e.g., exercise), and supply is affected by coronary artery blood flow.
- Coronary arteries supply oxygen to the heart muscle.

EJECTION FRACTION (EF)

- EF is the percentage of blood pumped out of the left ventricle with each contraction.
- A normal EF is typically 50-70%.
- It's an important measure in assessing cardiac function, especially in heart failure patients.

CARDIAC ASSESSMENT SKILLS

- Nursing students should learn to perform a thorough cardiac assessment, including inspection, palpation, percussion, and auscultation.
- Auscultation involves listening for heart sounds, murmurs, and other abnormal sounds using a stethoscope.

MEDICATIONS AND CARDIAC CARE

- Nursing students should understand the actions and side effects of common cardiac medications such as beta-blockers, ACE inhibitors, diuretics, and antiarrhythmics.
- Administering medications and monitoring for adverse reactions is a critical part of nursing care.

EKG INTERPRETATION

EKG (Electrocardiogram) interpretation is a fundamental skill for healthcare professionals, especially nurses. EKGs provide valuable information about the electrical activity of the heart and help diagnose various cardiac conditions.

STANDARD EKG LEADS

- There are 12 standard leads, divided into limb leads (I, II, III, aVR, aVL, aVF) and precordial leads (V1 to V6).
- These leads provide different views of the heart's electrical activity.

PAPER SPEED AND VOLTAGE

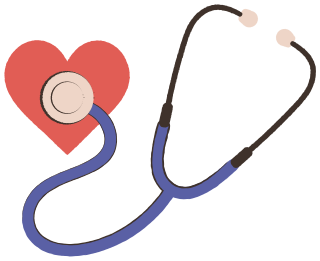
- EKG paper typically runs at 25 mm/s (standard) or 50 mm/s (for high-speed recordings).
- Each small box on the paper represents 0.04 seconds (40 ms), and each large box represents 0.20 seconds (200 ms).
- Voltage: 1 mV (millivolt) is represented by 10 mm (1 cm) on the vertical axis.

BASIC EKG WAVES AND INTERVALS

- **P wave:** Represents atrial depolarization (contraction).
- **QRS complex:** Depicts ventricular depolarization (contraction).
- **T wave:** Reflects ventricular repolarization (relaxation).
- **PR interval:** Measures the time from atrial depolarization to ventricular depolarization.
- **QT interval:** Represents ventricular depolarization and repolarization.

ASSESSING EKG RHYTHM

- Begin by assessing the rhythm: Is it regular or irregular?
- Identify the underlying rhythm (e.g., sinus rhythm, atrial
- fibrillation, ventricular tachycardia).



HEART RATE CALCULATION

- The heart rate can be calculated by counting the number of QRS complexes in a 6-second strip and multiplying by 10. Or, for regular rhythms, you can count QRS complexes in a 30-second strip and multiply by 2.
- For irregular rhythms, you may need to count QRS complexes for a full minute.

NORMAL SINUS RHYTHM (NSR)

- NSR is the standard electrical pattern of a healthy heart.
- Normal heart rate is typically 60 to 100 beats per minute.
- NSR includes a P wave followed by a QRS complex and a T wave.

COMMON RHYTHM DISTURBANCES

- **Atrial Fibrillation (AFib):** Irregularly irregular rhythm with no distinct P waves.
- **Ventricular Fibrillation (VFib):** Chaotic, uncoordinated ventricular activity.
- **Atrial Flutter:** Regular, sawtooth-shaped P waves (atrial activity) at a rapid rate.
- **Ventricular Tachycardia (VTach):** Wide QRS complexes at a rapid rate.
- **Bradycardia:** Heart rate below 60 bpm.
- **Tachycardia:** Heart rate above 100 bpm.

AXIS DETERMINATION

- The electrical axis represents the overall direction of electrical depolarization in the heart.
- Axis deviation can indicate various cardiac conditions.

ISCHEMIA, INJURY, AND INFARCTION

- EKG can help diagnose myocardial ischemia, injury, and infarction (heart attack) by observing ST-segment changes.
- ST-segment elevation may indicate an acute myocardial infarction.

ARTIFACT RECOGNITION

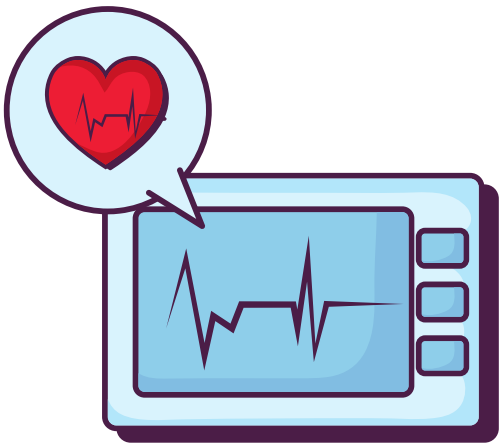
- Recognize and differentiate EKG artifacts (e.g., muscle tremors, patient movement) from true EKG abnormalities.

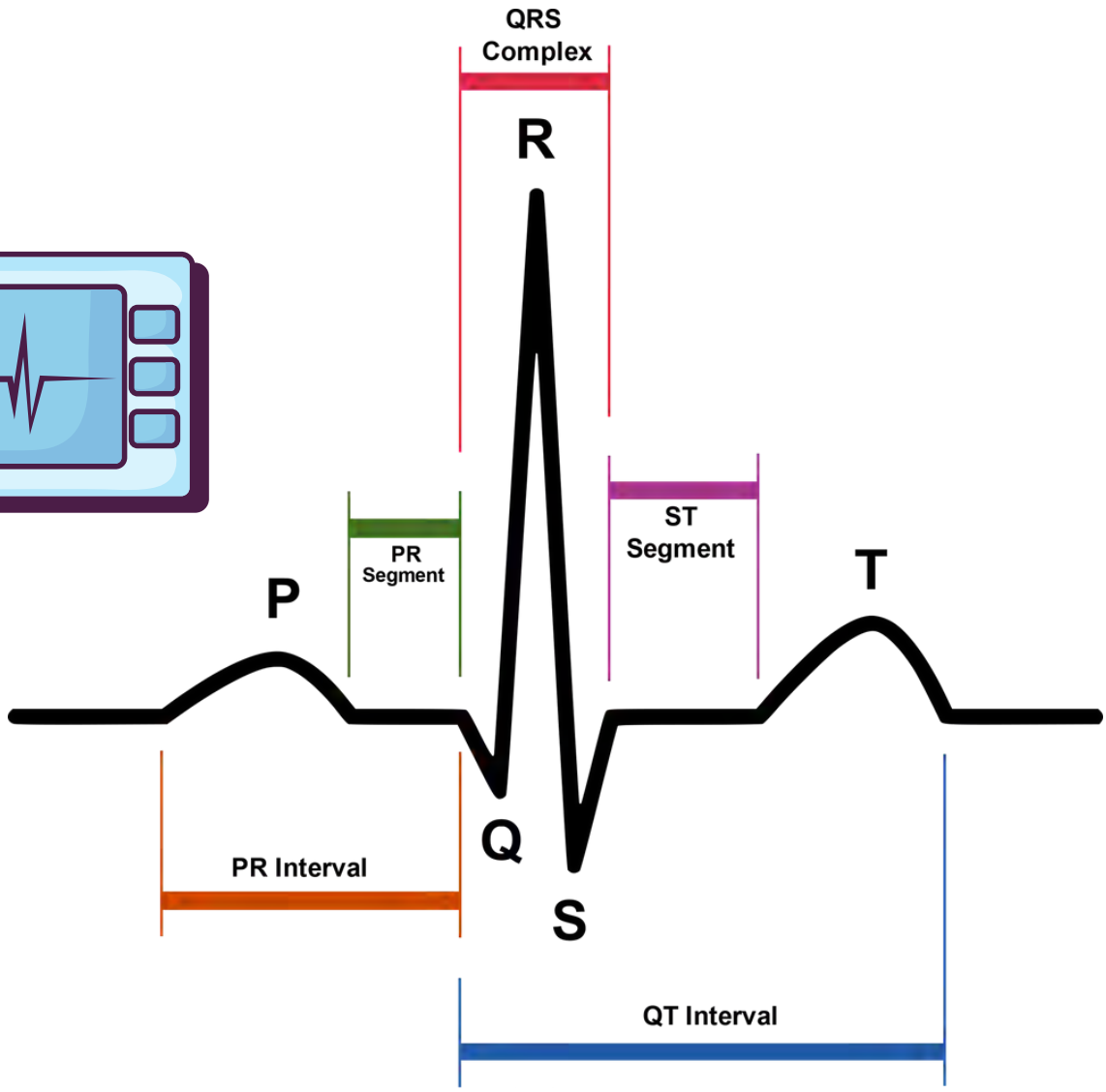
CLINICAL IMPLICATIONS AND NURSING CARE

- EKG interpretation guides clinical decision-making, including the administration of medications, interventions (e.g., defibrillation, cardioversion), and patient monitoring.
- Nurses should understand the significance of EKG findings in the context of the patient's clinical condition.

CONTINUOUS LEARNING

- EKG interpretation is a skill that improves with practice and experience.
- Keep learning and stay updated with the latest guidelines and research in cardiology.





WAVEFORM MORPHOLOGY

- Understanding the shape and appearance of EKG waveforms is crucial.
- Variations in waveforms can indicate different cardiac conditions.

PROLONGED QT INTERVAL

- A prolonged QT interval can predispose the patient to life-threatening arrhythmias.
- It may be congenital or acquired due to medications or electrolyte imbalances.

BUNDLE BRANCH BLOCKS (BBB)

- Bundle branch blocks are conduction delays in the electrical pathway.
- Right bundle branch block (RBBB) and left bundle branch block (LBBB) have distinct EKG patterns.
- They can indicate underlying heart disease.

HYPERTROPHY PATTERNS

- EKG can detect cardiac hypertrophy, which occurs in response to chronic pressure or volume overload.
- Left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH) have specific EKG criteria.

ST-SEGMENT CHANGES

- ST-segment elevation can be a sign of myocardial infarction (MI).
- ST-segment depression can indicate myocardial ischemia.

T-WAVE ABNORMALITIES

- Inverted T waves or T-wave flattening can signify various cardiac and non-cardiac conditions, including electrolyte imbalances.

ARTIFACT TROUBLESHOOTING

- Recognize common artifacts such as wandering baseline, muscle tremors, and 60-cycle interference.
- Troubleshoot and eliminate artifacts to obtain accurate EKG readings.

ADVANCED RHYTHM ANALYSIS

- Master interpreting complex rhythms, including atrial and ventricular arrhythmias, heart blocks, and junctional rhythms.

ST-SEGMENT MONITORING

- Continuous ST-segment monitoring is crucial in critical care units for early detection of ischemic changes.
- Nursing students should understand how to set up and interpret continuous ST-segment monitoring.

PEDIATRIC EKG

- Pediatric patients have unique EKG patterns and normal values that differ from adults.
- Learn about age-specific EKG considerations when caring for children.

DRUG EFFECTS ON EKG

- Many medications can affect the EKG, including antiarrhythmics, beta-blockers, and calcium channel blockers.
- Understand how these drugs influence EKG findings.

12-LEAD EKG

- The 12-lead EKG provides a comprehensive assessment of cardiac function.
- Learn how to place electrodes correctly to capture specific cardiac views.

EKG LEAD PLACEMENT

EKG lead placement is a critical aspect of performing an electrocardiogram (EKG or ECG) accurately. Proper placement ensures that the electrical activity of the heart is recorded correctly, allowing for an accurate interpretation

LIMB LEADS

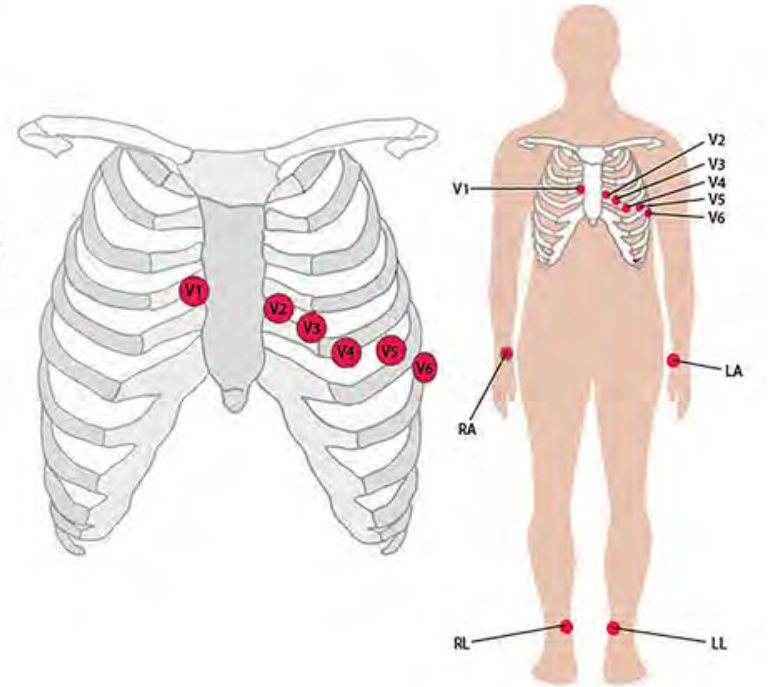
Limb leads, also known as standard leads, provide a frontal plane view of the heart's electrical activity.
They are labeled I, II, III, aVR, aVL, and aVF.
These leads use the arms and legs as electrodes.

PRECORDIAL LEADS

Precordial leads, also called chest leads, provide a transverse plane view of the heart's electrical activity.
They are labeled V1, V2, V3, V4, V5, and V6.
These leads use the chest as the electrode placement area.

ELECTRODE PLACEMENT

Clean the skin at each electrode placement site to ensure good skin-electrode contact.
Use alcohol pads or skin-prep solutions to remove oils and dead skin cells.
Properly position the electrodes to minimize artifact and ensure accurate readings.

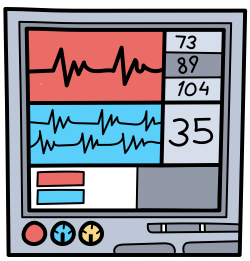


LIMB LEAD PLACEMENT

- **Lead I:** Place the positive electrode (red) on the left arm (LA) and the negative electrode (black) on the right arm (RA).
- **Lead II:** Place the positive electrode (red) on the left leg (LL) and the negative electrode (black) on the right arm (RA).
- **Lead III:** Place the positive electrode (red) on the left leg (LL) and the negative electrode (black) on the left arm (LA).
- **aVR (Augmented Voltage Right):** Place the positive electrode (red) on the right arm (RA) and the negative electrode (black) on the midpoint between the left arm (LA) and left leg (LL).
- **aVL (Augmented Voltage Left):** Place the positive electrode (red) on the left arm (LA) and the negative electrode (black) on the midpoint between the right arm (RA) and left leg (LL).
- **aVF (Augmented Voltage Foot):** Place the positive electrode (red) on the left leg (LL) and the negative electrode (black) on the midpoint between the right arm (RA) and left arm (LA).

PRECORDIAL LEAD PLACEMENT

- Locate the landmarks for V1 to V6:
- **V1:** Fourth intercostal space at the right sternal border.
- **V2:** Fourth intercostal space at the left sternal border.
- **V4:** Fifth intercostal space at the midclavicular line.
- **V3:** Midway between V2 and V4.
- **V5:** Fifth intercostal space at the anterior axillary line.
- **V6:** Fifth intercostal space at the midaxillary line.



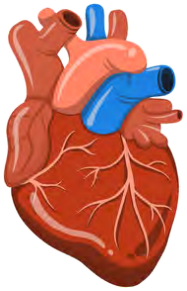
- Apply each precordial electrode directly over the corresponding landmark.
- Ensure that the leads are evenly spaced along the chest to maintain a balanced view of the heart.

LEAD COLOR CODING

- Electrodes and lead cables are color-coded for ease of placement.
- Red (or white) is typically the positive electrode.
- Black (or green) is the negative electrode.
- Green (or black) or brown is the ground electrode.

LEAD MISPLACEMENT

- Incorrect lead placement can result in distorted EKG tracings and misinterpretation.
- Always double-check lead placement to ensure accuracy.



SPECIAL LEADS

- Occasionally, additional leads, such as right-sided precordial leads (V3R, V4R, V5R) or posterior leads (V7, V8, V9), may be used in specific clinical situations to obtain additional information.

CONTINUOUS MONITORING

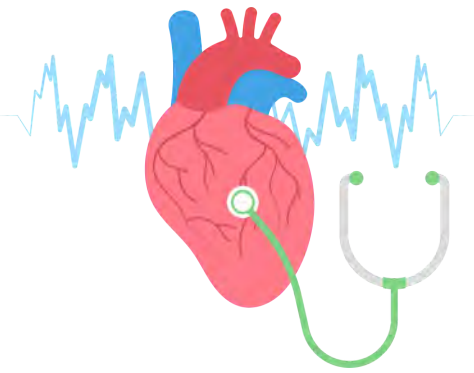
- For continuous EKG monitoring, chest electrodes may need to be repositioned or replaced periodically to maintain good contact and prevent skin irritation.

12-LEAD EKG CONFIGURATION

- A standard 12-lead EKG involves the combination of limb and precordial leads.
- This configuration provides a comprehensive view of the heart from different angles.

LIMB LEAD PLACEMENT REVIEW

- Confirm the proper placement of limb leads:
- **Lead I:** LA (-) and RA (+)
- **Lead II:** LL (-) and RA (+)
- **Lead III:** LL (-) and LA (+)
- **aVR:** RA (-) and midpoint between LA and LL (+)
- **aVL:** LA (-) and midpoint between RA and LL (+)
- **aVF:** LL (-) and midpoint between RA and LA (+)

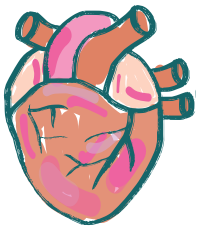


CHEST LEAD PLACEMENT TIPS

- Ensure that the precordial leads are placed accurately for reliable EKG tracings.
- Check for correct landmarks and palpate bony landmarks to locate the intercostal spaces.
- Proper alignment of precordial leads is crucial to prevent artifact and ensure accurate recordings.

RIGHT-SIDED LEADS (V3R, V4R, V5R)

- Right-sided precordial leads (V3R, V4R, V5R) may be used to assess the right ventricle in specific clinical scenarios.
- These leads are placed in the same intercostal spaces as their left-sided counterparts but on the right side of the sternum.



POSTERIOR LEADS (V7, V8, V9)

- Posterior leads provide a view of the posterior wall of the heart.
- They are placed on the patient's back, corresponding to the same horizontal level as V4, V5, and V6 on the front.

HOLTER MONITOR LEAD PLACEMENT

- Holter monitors record continuous EKG data over an extended period (e.g., 24-48 hours).
- Electrodes are attached to the chest, and the leads are connected to a portable monitor worn by the patient.
- Proper skin preparation and secure lead placement are essential to ensure accurate monitoring.

TELEMETRY LEAD PLACEMENT

- Telemetry leads are used for continuous EKG monitoring in hospital settings.
- These leads are typically placed on the chest and connected to bedside monitors.

CONTINUOUS TRAINING

- Healthcare professionals should undergo regular training and competency assessments in EKG lead placement to maintain accuracy and consistency in recordings.

TROUBLESHOOTING LEAD PLACEMENT ISSUES

- Be prepared to troubleshoot issues such as poor skin-electrode contact, electrode detachment, or patient discomfort during EKG recordings.

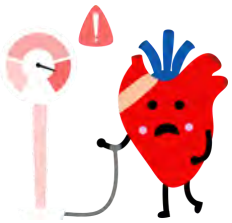
SKIN PREPARATION

- Proper skin preparation is essential for optimal electrode adherence and accurate EKG recordings.
- Clean the skin thoroughly with soap and water, and dry it completely before attaching electrodes.
- Avoid applying lotions, oils, or creams to the skin where electrodes will be placed.

ELECTRODE APPLICATION

- Apply the electrodes firmly to the skin, ensuring good contact without excessive pressure.
- Use electrode gel or paste if needed to improve conductivity.
- Place the electrodes flat and securely to minimize movement artifacts.

LEAD WIRE CONNECTION



- Ensure that lead wires are securely connected to the electrodes.
- Check for loose connections or frayed wires, which can lead to signal loss or artifacts.

TOP TESTED EKG STRIPS

EKG (electrocardiogram) strips, also known as ECG (electrocardiogram) strips, are graphical representations of the electrical activity of the heart over a period of time. They are essential tools in diagnosing and monitoring various heart conditions. When you refer to "top tested EKG strips

COMMON EKG PATTERNS

- **Normal Sinus Rhythm:** This is considered the standard, healthy heart rhythm. The P-wave, QRS complex, and T-wave are all present and follow a specific pattern.
- **Atrial Fibrillation (AFib):** AFib is an irregular heart rhythm characterized by the absence of a distinct P-wave and an irregular ventricular rate.
- **Atrial Flutter:** This is a regular, fast atrial rhythm, often with a "sawtooth" appearance on the EKG.
- **Ventricular Tachycardia (VT):** VT is a fast, regular rhythm originating in the ventricles. It can be life-threatening.
- **Ventricular Fibrillation (VFib):** VFib is a chaotic, disorganized rhythm that can lead to cardiac arrest.

SPECIFIC EKG FINDINGS

- ST-Segment Elevation: Elevated ST segments can indicate myocardial infarction (heart attack).
- ST-Segment Depression: Depressed ST segments can be a sign of myocardial ischemia (insufficient blood supply to the heart).
- QT Interval Prolongation: A prolonged QT interval can lead to a type of arrhythmia called Torsades de Pointes.
- Wolff-Parkinson-White Syndrome (WPW): This EKG pattern shows an accessory pathway between the atria and ventricles, leading to abnormal conduction.

EKG INTERPRETATION

- EKG interpretation requires an understanding of waveforms, intervals, and segments. Healthcare professionals, such as doctors, nurses, and paramedics, undergo training to accurately interpret EKGs.
- Automated EKG machines are commonly used to capture EKG strips and can help identify abnormalities for further review by a healthcare provider.

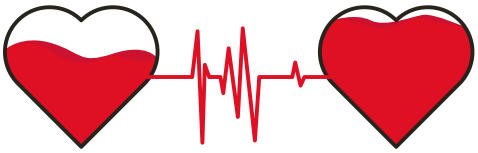
CLINICAL USE

- EKGs are used in various clinical settings, including hospitals, clinics, and emergency rooms.
- They help diagnose heart conditions, assess cardiac function, monitor the effects of medications, and guide treatment decisions.

EKG EQUIPMENT AND PAPER

EKG machines record the electrical signals from the heart, producing EKG strips on special paper.
Modern EKG machines often include digital displays, making it easier to review and interpret EKGs.

12-LEAD EKG

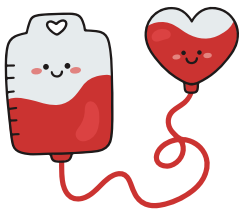


A standard EKG typically records three to six leads, providing specific views of the heart's electrical activity. However, a 12-lead EKG offers a more comprehensive assessment by recording electrical signals from 12 different angles or "views." This is crucial in diagnosing and localizing heart conditions.

HOLTER MONITOR

In addition to a standard EKG, a Holter monitor is a portable device that continuously records the heart's electrical activity over a 24 to 48-hour period. It helps diagnose intermittent arrhythmias and assess how the heart responds to daily activities.

TELEMETRY



In hospitals, telemetry units use wireless EKG monitoring to continuously track a patient's heart rhythm. It allows healthcare providers to monitor multiple patients simultaneously and receive alerts if there are any irregularities.

PACEMAKERS AND IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS (ICDS)

- These devices use EKG technology to monitor and regulate the heart's electrical activity. They can also deliver electrical shocks or pacing impulses when needed to treat arrhythmias or heart failure.

TREADMILL STRESS TEST

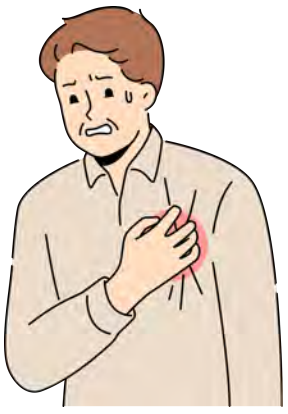
- During a stress test, EKG is recorded while a patient exercises on a treadmill or stationary bike. This helps evaluate the heart's response to physical stress and can uncover exercise-induced arrhythmias or ischemia.

PHASES OF THE CARDIAC CYCLE

- EKG strips represent different phases of the cardiac cycle. The P-wave corresponds to atrial depolarization, the QRS complex to ventricular depolarization, and the T-wave to ventricular repolarization.

ARTIFACT AND NOISE

- EKGs can be affected by various artifacts and noise, such as muscle tremors, patient movement, and electrical interference. Healthcare professionals must differentiate these from true EKG abnormalities.



PEDIATRIC EKG

- EKG interpretation in children differs from that in adults due to age-related variations in normal heart rate, size, and electrical patterns. Pediatric cardiologists specialize in interpreting EKGs for children.

COMPUTER-AIDED EKG ANALYSIS

- Computer algorithms can assist healthcare professionals in interpreting EKGs by flagging potential abnormalities. However, human expertise is still crucial for accurate diagnosis.

LONG-TERM EKG MONITORING

- In addition to Holter monitors, event monitors are used for long-term EKG monitoring. Patients can activate them when they experience symptoms, allowing healthcare providers to capture arrhythmias during specific events.

T-WAVE ABNORMALITIES

- Changes in T-wave morphology (T-wave inversion or flattening) can indicate various cardiac conditions, including ischemia, electrolyte imbalances, and certain medications' side effects.

EKG EDUCATION

- Many healthcare professionals, including nurses, paramedics, and technicians, receive training in EKG interpretation to ensure accurate diagnosis and timely intervention.

AMBULATORY EKG MONITORING

- Ambulatory EKG monitoring, such as the Holter monitor and event monitor, is used to capture irregular heart rhythms that may occur infrequently. It's particularly helpful in diagnosing conditions like paroxysmal arrhythmias.

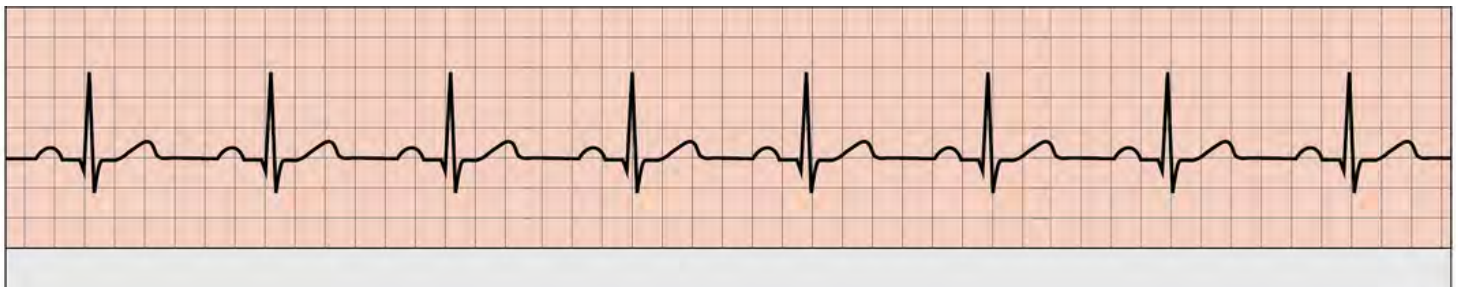
EKG LEADS

- EKG machines use various leads to record electrical signals from different angles. The standard limb leads (I, II, III, aVR, aVL, aVF) and precordial leads (V1-V6) provide specific perspectives on the heart's electrical activity.

VECTORCARDIOGRAPHY

- Vectorcardiography is a more advanced technique that utilizes multiple leads to represent the heart's electrical activity in three dimensions. It offers a comprehensive view of cardiac conduction and is especially valuable in research and complex cases.

EKG WAVEFORM CHARACTERISTICS



- The P-wave represents atrial depolarization (contraction).
- The QRS complex reflects ventricular depolarization (contraction).
- The T-wave signifies ventricular repolarization (relaxation).
- The PR interval measures the time from atrial depolarization to ventricular depolarization.
- The QT interval measures the total time for ventricular depolarization and repolarization.

ARRHYTHMIAS TREATMENT

Arrhythmias are irregular heart rhythms that can be either too fast (tachycardia) or too slow (bradycardia). The treatment of arrhythmias depends on the type of arrhythmia, its underlying cause, the severity of symptoms, and the patient's overall health.

LIFESTYLE MODIFICATIONS

- In some cases, lifestyle changes can help manage arrhythmias. These changes may include reducing caffeine and alcohol intake, quitting smoking, managing stress, and getting regular exercise.

MEDICATIONS

- Antiarrhythmic Medications: These drugs are used to control and stabilize heart rhythm. Examples include amiodarone, flecainide, and propafenone.
- Beta-Blockers: These drugs slow the heart rate and can be used to control tachycardias. Examples include metoprolol and propranolol.
- Calcium Channel Blockers: These drugs help regulate heart rhythm by affecting the movement of calcium in the heart cells. Examples include verapamil and diltiazem.
- Blood Thinners (Anticoagulants): Patients with certain arrhythmias, like atrial fibrillation, may be prescribed anticoagulants like warfarin or newer agents such as apixaban or rivaroxaban to reduce the risk of stroke.

CARDIOVERSION

- Electrical Cardioversion: This procedure involves delivering an electric shock to the heart to restore normal rhythm. It's often used for atrial fibrillation or atrial flutter.

CATHETER ABLATION

In this minimally invasive procedure, a catheter is used to deliver energy (usually radiofrequency or cryoenergy) to the specific area of the heart causing the arrhythmia. It's commonly used for supraventricular tachycardias (SVTs) and some atrial and ventricular arrhythmias.

PACEMAKERS

Pacemakers are implanted devices that help regulate slow heart rhythms (bradycardias). They send electrical impulses to the heart to maintain a regular heartbeat.

IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS (ICDS)

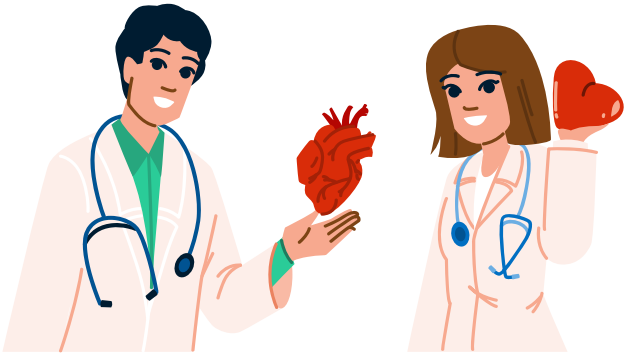
ICDs are implanted devices designed to treat life-threatening ventricular arrhythmias. They can deliver electrical shocks to restore normal rhythm or provide pacing when needed.

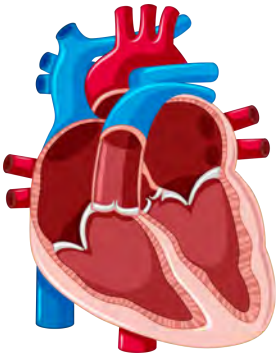
LIFESTYLE AND DIETARY CHANGES

Patients with arrhythmias, especially atrial fibrillation, may be advised to manage their diet to maintain a healthy weight, reduce salt intake, and avoid excessive alcohol and caffeine consumption.

SURGERY

In some cases, surgical procedures may be necessary to treat arrhythmias. For instance, the Maze procedure is a surgical technique used to treat atrial fibrillation by creating scar tissue in the atria to block irregular electrical signals.





REMOTE MONITORING

- Some patients with arrhythmias may benefit from remote monitoring devices that allow healthcare providers to track their heart rhythm remotely, making it easier to detect and address arrhythmias.

MANAGEMENT OF UNDERLYING CONDITIONS

- Treating underlying conditions that contribute to arrhythmias, such as hypertension or heart disease, is crucial in managing and preventing recurrent arrhythmias.

LIFESTYLE CHANGES

- **Dietary Modifications:** Reducing sodium (salt) intake can help manage high blood pressure, which is a common contributor to arrhythmias. A heart-healthy diet rich in fruits, vegetables, and whole grains is recommended.
- **Alcohol and Caffeine:** Limiting or avoiding alcohol and caffeine intake can help prevent or manage certain arrhythmias, particularly atrial fibrillation.
- **Stress Management:** Stress can trigger or exacerbate arrhythmias. Relaxation techniques, meditation, and regular exercise can help manage stress levels.

ANTIARRHYTHMIC MEDICATIONS

- There are various classes of antiarrhythmic drugs, each targeting specific types of arrhythmias. The choice of medication depends on the type of arrhythmia, its underlying cause, and the patient's medical history.

ELECTRICAL CARDIOVERSION

- Besides atrial fibrillation, electrical cardioversion may be used for other arrhythmias, such as atrial flutter or certain ventricular tachycardias. It can quickly restore a normal rhythm.

ABLATION PROCEDURES

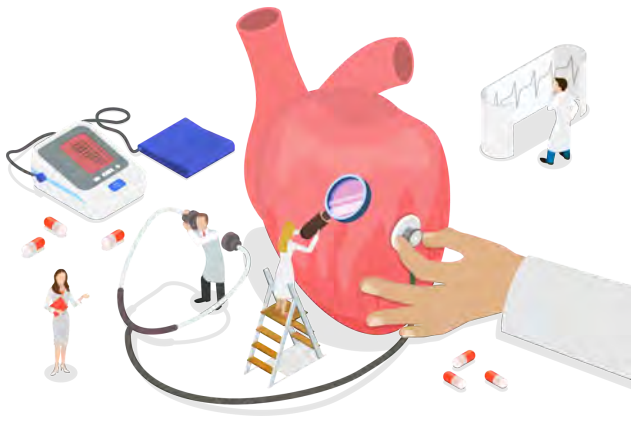
- **Catheter Ablation:** In addition to treating SVTs, catheter ablation can be used for atrial fibrillation, atrial flutter, and some ventricular arrhythmias. It aims to eliminate or isolate the abnormal electrical pathways causing the arrhythmia.
- **Surgical Ablation:** In some cases, open-heart surgery may be required to perform ablation procedures. This is more invasive and typically reserved for complex cases.

IMPLANTABLE DEVICES

- **Cardiac Resynchronization Therapy (CRT):** CRT devices are used for heart failure patients with arrhythmias. They synchronize the contractions of the heart's ventricles to improve pumping efficiency.
- **Leadless Pacemakers:** These small, self-contained devices are implanted directly into the heart, eliminating the need for leads (wires). They are used for certain bradycardias.
- **Subcutaneous ICDs:** These devices are implanted under the skin and are used to treat ventricular arrhythmias without leads in the heart.

LIFESTYLE MONITORING

- Some arrhythmias, particularly those associated with syncope (fainting), may be managed with lifestyle monitoring techniques like tilt-table testing to evaluate how specific activities trigger arrhythmias.



LIFESTYLE EDUCATION

- Patients with arrhythmias often receive education on recognizing and managing symptoms, understanding medication management, and knowing when to seek medical attention.

GENETIC COUNSELING

- For certain arrhythmias with a genetic component, genetic counseling may be recommended to assess the risk to family members and provide guidance on genetic testing.

LIFESTYLE REHABILITATION

- Cardiac rehabilitation programs can help individuals with arrhythmias improve their overall cardiovascular health through structured exercise, education, and support.

COMPLEMENTARY THERAPIES

- Some patients explore complementary therapies like acupuncture or yoga for stress management and symptom relief, but these should be discussed with healthcare providers.

FOLLOW-UP CARE

- Regular follow-up appointments with a cardiologist or electrophysiologist are essential to monitor the effectiveness of treatment, make necessary adjustments to medications or therapies, and address any new developments in the condition.

STEMI Leads & Locations

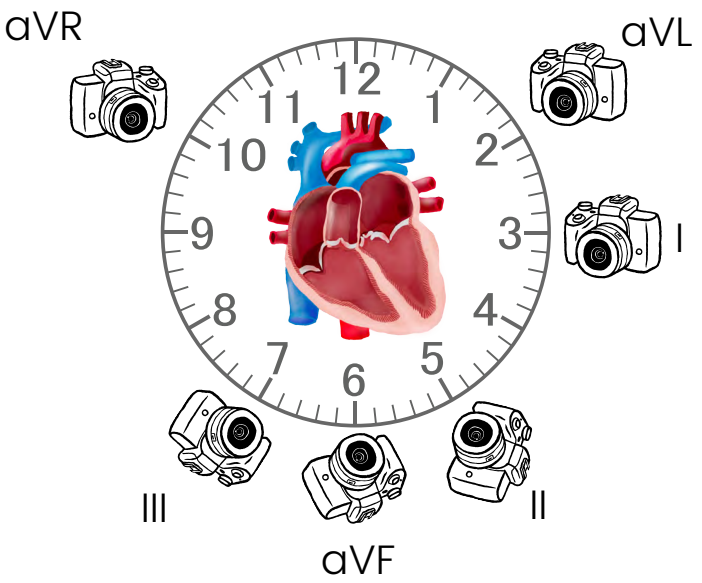
STEMI EKG Leads and Locations

NURSE DOSE

What is a lead?

A "lead" refers to the specific combination of two electrodes that are used to measure electrical activity in the heart. An EKG measures the electrical impulses generated by the heart during each heartbeat and represents them as waves on a graph.

A lead can be thought of us a camera looking at the heart from a specific angle.



Limb Leads

Limb leads in an EKG measure the electrical activity of the heart by recording the voltage differences between two of the limb electrodes (right arm, left arm, and left leg), providing information about the heart's electrical axis and overall cardiac function on a 2D plane.

- Lead I: Records the electrical potential difference between the right arm (RA) and left arm (LA). Looks at the lateral side.
- Lead II: Records the electrical potential difference between the right arm (RA) and left leg (LL). Looks at the inferior side.
- Lead III: Records the electrical potential difference between the left arm (LA) and left leg (LL). Looks at the inferior side.

Where We are Looking

- aVL and I look at the heart from the lateral side.
- II, III, and aVF look at the heart from the inferior aspect.
- aVR looks at the heart from the right side.

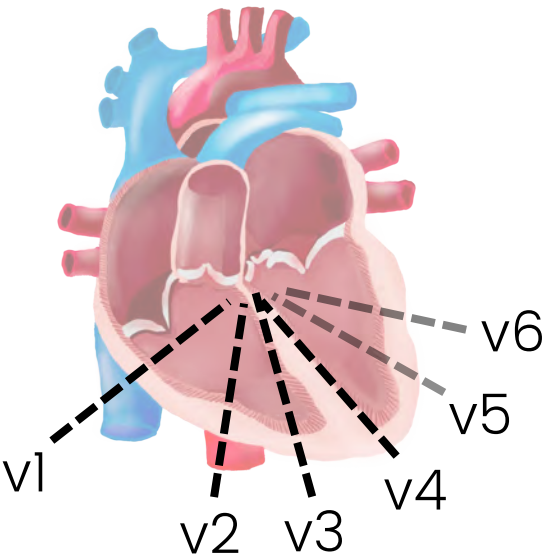
Augmented Leads

Augmented leads are another set of leads used in electrocardiography (ECG or EKG) to provide additional perspectives on the heart's electrical activity. Unlike the standard limb and precordial leads, augmented leads are derived from a combination of two limb electrodes and are labeled as aVR, aVL, and aVF

Chest (precordial) Leads

The chest leads in an electrocardiogram (ECG or EKG) provide a three-dimensional perspective of the heart's electrical activity. Each lead is placed on specific locations on the chest to capture electrical signals from different angles. Here's a simplified representation of how the chest leads (V1 to V6) "look at" the heart in 3D:

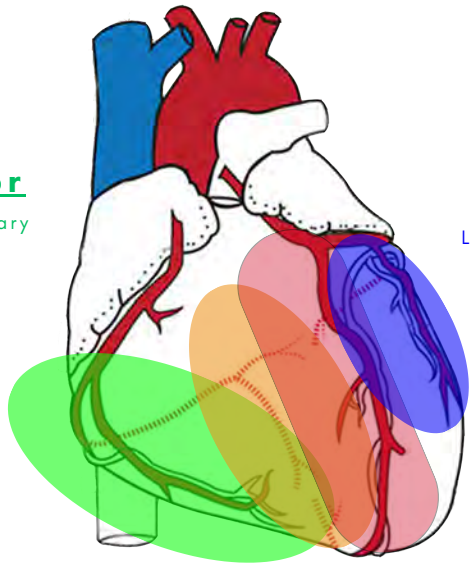
- V1 and V2 (Septal Leads):
  - These leads are positioned on the right and left sides of the sternum at the fourth intercostal space.
  - They focus on the septum, the wall that divides the right and left sides of the heart.
- V3 and V4 (Anterior Leads):
  - Placed in the middle of the chest, between V2 and V6.
  - These leads look at the front (anterior) part of the heart, including the interventricular septum.
- V5 and V6 (Lateral Leads):
  - Positioned on the left side of the chest.
  - These leads provide a lateral view, looking at the side of the left ventricle.



How to Determine STEMI Location with Leads

Limb leads and chest leads work together to determine the location of a ST-segment elevation myocardial infarction (STEMI) on the heart. Limb leads help monitor a general area of the heart in a 2D plane while chest leads help us zone in on particular regions of the heart in a more three-dimensional plane.

If you can understand and remember the locations of the 12 leads and the regions that they monitor you can easily deduce where in the heart ischemia is occurring. Actually understanding how leads work and where they monitor will allow you to determine STEMI types more accurately and efficiently as opposed to those hard to memorize tables.



Inferior - II, III, aVF

- The inferior portion of the heart is monitored by leads II, III, and aVF.
- An inferior STEMI will have ST elevation to these leads and reciprocal depression to anterior and lateral leads.
- The right coronary artery is the main coronary artery to supply the inferior region of the heart.
- Right sided MIs can be confirmed with a right sided EKG.

Lateral - aVL, V5, V6

- The lateral portion of the heart is monitored by leads I, aVL, V5, and V6.
- A lateral STEMI will have ST elevation to these leads and can have reciprocal depression to inferior leads.
- The left circumflex artery is the main coronary artery to supply the lateral region of the heart.
- Lateral MIs usually involve the lateral aspect of the left ventricle.

Septal - V1, V2

- The septal portion of the heart (where the septum divides the left and right ventricle) is monitored by leads V1 and V2.
- A septal STEMI will have ST elevation to these leads and can have reciprocal depression to lateral leads.
- The left anterior descending (LAD) artery is the main coronary artery to supply the septal region of the heart. However, the right coronary artery also supplies a portion of this region as well..

Anterior - V3, V4

- The anterior portion of the heart is monitored by leads V3 and V4.
- An anterior STEMI will have ST elevation to these leads and can have reciprocal depression to inferior leads.
- The left anterior descending (LAD) artery is the main coronary artery to supply the lateral region of the heart.
- Anterior MIs usually involve the anterior aspect of the left ventricle.