

Critical Thinking Tip Sheets

12 Lead EKG Review

Area of myocardium	Coronary artery involved	Leads affected
INFERIOR	RCA	II, III, AVF
SEPTAL	LAD	V1 & V2
ANTERIOR	LAD	V3 & V4
LATERAL	Circumflex	I, AVL, V5, V6

Normal EKG Depolarization

I	AVR	V1	V4
↑	↓	↓	Biphasic
II	AVL	V2	V5
↑	↑ or ↓	↓	↑
III	AVF	V3	V6
↑	↑	Biphasic	↑

Axis	Normal -30 to +90	Left -30 to - 90	Right +90 to ±180	Extreme -90 to ±180
Lead I	↑	↑	↓	↓
AVF	↑	↓	↑	↓

Hemiblocks	LAH	LPH
Lead I	↑	↓
Lead II	↓	↑
Lead III	↓	↑
Axis	Left	Right

LBBB = QRS \geq 0.12, Negative QRS in V1 (carrot)

RBBB = QRS \geq 0.12; Positive QRS in V1 (rabbit ears)

Advanced 12 Lead EKG

Left Atrial Enlargement P-mitrale	<ul style="list-style-type: none"> Notched p wave > 0.12 second in limb leads Causes prolonged conduction times required to travel through enlarged LA Produces a double hump (camel hump)
Right Atrial Enlargement P-pulmonale	<ul style="list-style-type: none"> Right Atrial Enlargement Peaked P wave taller than 2.5 mm in the limb leads P-pulmonale = teepee
Left Ventricular Hypertrophy	<ul style="list-style-type: none"> S in V1 or V2 + R in V5 or V6 \geq 35 mm. <p>Or</p> <ul style="list-style-type: none"> Any precordial lead is \geq 45 mm The R wave in AVL is \geq 11mm The R wave in Lead I is \geq 12 mm The R wave in lead AVF is \geq 20 mm
Right Ventricular Hypertrophy	<ul style="list-style-type: none"> R:S ratio is \geq 1 in leads V1 and/or V2 R is bigger than S
Wolff-Parkinson-White	<ul style="list-style-type: none"> Shortened PR interval < 0.12 sec with a normal p wave Wide QRS complex \geq 0.11 sec The presence of a delta wave ST-T wave changes or abnormalities Association with paroxysmal tachycardias – can be fatal
Pericarditis	<ul style="list-style-type: none"> Diffuse ST elevation Scooping upwardly concave ST segment elevation in almost all leads except AVR No reciprocal ST depression except in AVR PR depression
Early Repolarization	<ul style="list-style-type: none"> Elevated take-off of ST segment at the j point Concave upward ST elevation ending with a symmetrical upright T wave – often of large amplitude Gently upsloping and curving downward or sagging of the ST segment , producing the so called “smiley face” Contrasted with the junctional elevation and horizontal or straight ST segment & the curving upward of “sad face” of the STEMI examples No reciprocal ST segment depression
Pulmonary Embolus (not diagnostic ... may see these changes)	<ul style="list-style-type: none"> S1, Q3 or S1,Q3, T3 (inverted T) RBBB Inverted T waves secondary to RV strain may be seen in the right precordial leads and can last for months <p>or</p> <ul style="list-style-type: none"> R axis deviation noted by Lead I negative or S wave Lead I and AVR positive V1 has tall R wave Large p waves II, III, AVF Inverted T wave Lead III

Arterial Blood Gas

Normal pH 7.35 – 7.45

Normal pCO₂ 35 – 45

Normal pO₂ 80 - 90

Normal HCO₃ 23 – 27

Base Excess – 2 - + 2

Respiratory Acidosis

Cause: Hypoventilation

Guillain Barre

Spinal Cord Injury

Oversedation

Resp Acidosis pH ↓ pCO₂ ↑

Resp Alkalosis pH ↑ pCO₂ ↓

Metabolic Acidosis pH ↓ pCO₂ ↓

Metabolic Alkalosis pH ↑ pCO₂ ↑

Respiratory Alkalosis

Cause: Hyperventilation

Pain

Anxiety

Spinal Cord Injury

Metabolic Acidosis

Cause: Retention of Acid

Diarrhea

Renal Failure

DKA

Tissue Hypoxia

Hypoperfusion

Metabolic Alkalosis

Cause: Body Taking on Base

Eliminating Acid

Continuous NG Suction

Diuretic Use

	Uncompensated			Partially Compensated			Fully Compensated		
Fluid and Potassium Replacement	pH	pCO ₂	tCO ₂	pH	pCO ₂	tCO ₂	pH	pCO ₂	tCO ₂
Resp. Acidosis	↓	↑	-	↓	↑	↑	-	↑	↑
Resp. Alkalosis	↑	↓	-	↑	↓	↓	-	↓	↓
Met. Acidosis	↓	-	↓	↓	↓	↓	-	↓	↓
Met. Alkalosis	↑	-	↑	↑	↑	↑	-	↑	↑

$$CO = SV \times HR$$

SV = Preload, Afterload, Contractility

Inotropic: Effect on contractility

Chronotropic: Effect on Heart Rate

Dromotropic: Effect on Conductivity

Medication	Heart Rate	Preload	Afterload	Vasodilator	Vasopressor	Contractility
Dopamine Hydrochloride (Intropin)						
Epinephrine (Adrenalin)						
Norepinephrine bitartrate (Levophed)						
Phenylephrine (Neo-Synephrine)						
Vasopressin (Pitressin)						
Nitroprusside (Nipride)						
Nitroglycerin (Tridil)						
Dobutamine hydrochloride (Dobutrex)						
Digitalis (Digoxin, Lanoxin)						
Milrinone (Primacor)						
Calcium Chloride						
Amiodarone hydrochloride (Cordarone)						
Lidocaine (Xylocaine)						
Atropine sulfate						
ACE Inhibitors						
Beta Blockers						
Diltiazem (Cardizem)						
Nicardipine (Cardene)						

Cardiac Medications & Effect on Cardiac Output						
Medication	Heart Rate	Preload	Afterload	Vasodilator	Vasopressor	Contractility
Dopamine Hydrochloride (Intropin)	↑	↑	↑	---	✓	↑
Epinephrine (Adrenalin)	↑	↑	↑	---	✓	↑
Norepinephrine bitartrate (Levophed)	↑	↑	↑	---	✓	↑
Phenylephrine (Neo-Synephrine)	Slight ↑	↑	↑	---	✓	↑
Vasopressin (Pitressin)	Slight ↑	↑	↑	---	✓	---
Nitroprusside (Nipride)	---	↓	↓	✓	---	---
Nitroglycerin (Tridil)	---	↓	↓	✓	---	---
Dobutamine hydrochloride (Dobutrex)	Slight ↑	---	---	Slight	Slight	↑
Digitalis (Digoxin, Lanoxin)	↓	---	---	---	---	↑
Milrinone (Primacor)	Slight ↑	Slight ↓	Slight ↓	Slight	---	↑
Calcium Chloride	---	---	---	---	---	↑
Amiodarone hydrochloride (Cordarone)	↓	---	---	---	---	---
Lidocaine (Xylocaine)	---	---	---	---	---	↓
Atropine sulfate	↑	---	---	---	---	---
ACE Inhibitors	----	↓	↓	✓	---	----
Beta Blockers	↓	↓ then ↑	↓	✓	---	↓
Diltiazem (Cardizem)	↓	↑ when HR ↓	---	---	---	↓
Nicardipine (Cardene)	---	↓ then ↑	↓	✓	---	↓

Herrmann 15

Hemodynamics

Parameter	Normal Values
Cardiac Output (CO)	4 - 8 l/min
Cardiac Index (CI)	2.5 – 4.2 l/min/m ²
Right atrial pressure (CVP)	0 – 8 mmHg
Pulmonary artery pressure (PAS/PAD)	15 - 30/6 -12 mmHg
Pulmonary artery occlusive pressure	4 – 12 mmHg
Systemic vascular resistance (SVR)	770 – 1500 dyne/sec/cm ⁵
Pulmonary vascular resistance (PVR)	20 – 120 dyne/sec/cm ⁵
Stroke Volume (SV)	60 -130 mL/beat
Stroke Volume Index (SVI)	30 – 65 mL/beat/m ²
Arterial oxygenation saturation	95 – 100 %

	Hypovolemia	Fluid Overload	LV failure	RV failure	RV & LV failure	Sepsis
CO/CI						
CVP						
PAD						
SV/SVI						
SVR/SVRI						
PVR/PVRI						

	Hypovolemia	Fluid Overload	LV failure	RV failure	RV & LV failure	Sepsis
CO/CI	↓	Nx or ↓	↓	↓	↓	↑
CVP	↓	↑	Normal	↑	↑	↓
PAD	↓	↑	↑	Normal	↑	↓
SV/SVI	↓	↑	↓	↓	↓	↓
SVR/SVRI	Normal	Normal	↑	Normal	↑	↓
PVR/PVRI	Normal	Normal	Normal	↑	↑	↓

LOW	CARDIAC OUTPUT Treatment Options	HIGH
Volume	PRELOAD CVP, PAD, PAOP	Diuretics Venous Vasodilation
Vasopressors	AFTERLOAD SVR,PVR	Vasodilators Calcium Channel Blockers IABP Valve Surgery
Optimize preload Inotropes Calcium Ventricular Assist Devices	CONTRACTILITY CO/CI indirect measurement	-----
Pacemaker Atropine Isuprel Dopamine	RATE/RHYTHM	Beta Blockers Calcium Channel Blockers

Cardiac Output Treatment Options

$$CO = SV \times HR$$

Preload, Afterload, and Contractility determine Stroke Volume

Determinants of Cardiac Output	How to measure	If Determinant is LOW , how to treat.	If Determinant is HIGH how to treat.
Preload	LEFT VENTRICLE = LVEDP PAOP: 8-12 mm Hg PAD: 8-15 mm Hg RIGHT VENTRICLE = RAP RAP/CVP: 2-5 mm Hg	<u>Volume</u>	Diuretics Venous Vasodilators
Afterload	Systemic Vascular Resistance (SVR) reflects LV afterload 800-1400 dynes/sec/cm ⁻⁵ Pulmonary Vascular Resistance (PVR) reflects RV afterload 40-220 dynes/sec/cm ⁻⁵	Vasopressors Dopamine, Epinephrine, Neosynephrine, Norepinephrine	Vasodilators Nipride, Tridil, Beta Blockers IABP
Contractility	CO/ CI - indirect measurement CO: Normal 4-8 l/min CI: Normal 2.5 - 4 l/min/m ² Subclinical: 2.2 - 2.7 l/min/m ² Low perfusion: 1.8 - 2.2 l/min/m ² Shock << 1.8 l/min/m ²	Positive inotropes Dopamine, Epinephrine, Neosynephrine, Norepinephrine, Dobutamine, Milirone, Calcium	Negative inotropes
Heart Rate	Monitor/Pulse	Atropine Pacemaker	Treat Cause Beta Blockers Calcium Channel Blockers

Cheryl Herrmann, APN, CCNS Methodist, Peoria, IL 2005

	MS	MR	AS	AR/AI
Heart Sounds				
Symptoms				
Atrial size				
Ventricular Size				

	Myocarditis	Endocarditis	Pericarditis
Definition			
Heart Sounds			
Symptoms			