12 Lead ECG Review Sheet

Lead Placement

V1 – 4th intercostal space to right of sternum
Investigates anterior wall of LV

Septum

V2 – 4th intercostal space to left of sternum
Investigates anterior wall of LV

Septum

V3 – No true anatomical placement
Located midway between V2 and V4
Investigates anterior wall of LV

V4 – 5th intercostal space mid-clavicular line
Often directly under left nipple
Investigates anterior wall of LV

V5 – 5th intercostal space anterior axillary line
Investigates lateral wall of LV

V6 – 5th intercostal space mid-axillary line
Investigates lateral wall of LV

Leads V1 – V4 investigate anterior wall of LV
Leads V5 – V6 investigate lateral wall of LV
ECG deflection

V1-V2 negative
V3 equiphasic
V4-V6 positive

Ischemia

ST segment depression with or without T wave inversion

Subendocardial

1mm or more ST segment depression with or without T wave inversion in two or more anatomically contiguous leads

Injury

ST segment elevation with or without a hyperacute T wave

Transmural

1mm or more ST segment elevation in two or more anatomically contiguous leads

Infarction

Pathological (enlarged) Q waves

Pathological Q waves

Transmural MI

Full thickness MI

Extends through all tissue layers

Appreciable ECG changes

ST elevation

Q wave infarction
Subendocardial MI

Partial thickness MI
Isolated to endocardial surfaces
ST segment depression
Non Q wave MI
Indicative of myocardial tissue necrosis
Scars of infarction

Large Q waves
> 25% of height of R wave in same lead
> 0.04 seconds of time

Electrode “looks” through dead tissue and records electricity on opposite wall
Provide exclusive oxygenated blood supply to the heart muscle and electrical conduction system

RCA
RA, RV, Inferior wall of LV (IWMI)
SA node 60% pop
AV node 90% pop

LCA
Left anterior descending (AWMI)
Circumflex (LWMI)
Anterior Wall MI

Etiology

Occlusion of LAD artery

Reflecting leads

Precordial leads V1-V4

V1-V2 view anterior septum

V3-V4 view anterior surface of LV

Reciprocal changes

ST depression in inferior leads

II, III, aVF

Loss of R wave progression

Complications of AWMI

Typically associated with large infarctions

Cardiogenic shock

Conduction abnormalities

Type II second degree AV block (Mobitz II)

Complete (3rd degree) block

Pacing versus atropine therapy
Inferior Wall MI

Etiology

RCA occlusion

Reflecting leads

II, III, aVF

Reciprocal changes

ST depression

Lateral leads

I and aVL

Complications of IWMI

RCA supplies AV node in 90% of population

AV nodal heart blocks

Associated with excessive vagal tone

Characterized by bradycardia and hypotension

Responsive to atropine and fluid therapy

Concomitant right ventricular infarction

Lateral Wall MI

Etiology

Occlusion of the circumflex

Reflecting leads

I, aVL, V5, V6

Often associated with anterior and inferior MI

Anterolateral MI

Inferolateral MI
Anterolateral MI

Etiology

Proximal occlusion of left main CA or combination occlusion to the LAD and circumflex

Reflecting leads

V1-V4 (anterior)

I, aVL, V5, V6 (lateral)

ST segment elevation in leads I, aVL, V5, V6 (lateral)

ST segment elevation in V1-V4

Loss of R-wave progression

Inferolateral MI

Etiology

Occlusion to the RCA and circumflex branch

Reflecting leads

II, III, aVF (inferior)

I, aVL, V5, V6 (lateral)

ST segment elevation in leads V5, V6 (lateral)

ST segment elevation in leads II, III, aVF
**Posterior MI**

**Etiology**

RCA occlusion

Most often occurs as extension of inferior or lateral infarcts

**Reflecting leads**

No direct view of posterior wall of LV with standard 12-lead ECG

Leads V8 and V9

Reciprocal (mirror image) changes are evaluated in V1 and V2 (anterior leads)

**Reciprocal changes**

ST elevation of posterior MI will present as:

ST depression in V1 and V2

Q waves as an enlargement of typical small “r” wave in V1 and V2

12-lead ECG turned upside down and observed in mirror

ST elevation and Q waves would be present

ST depression and large R waves are produced because depolarization of the posterior myocardium proceeds opposite the direction of depolarization of anterior myocardium
Right Ventricular Infarction

Pathophysiology

RCA supplies inferior wall of LV and RV

30% of IWMI also involve the RV

RVI is due to proximal RCA occlusion and must always be suspected in the presence of IWMI

Recognition of RVI is important because patients are at risk for:

- Hypotension
- Decreased cardiac output

ECG Evidence of RVI

Obtained with the use of right sided chest leads

V3R-V6R

V3R-V6R are applied to the right side of the chest in the same anatomical position as their left sided counterparts

V4R is the most sensitive and specific indicator of RVI

V4R “looks” directly at the RV

Infarction is present V4R will demonstrate ST segment elevation

Note ST segment elevation in leads II, III, aVF (inferior leads)

ST elevation in V4R

Lead V4R

Whenever inferior wall infarction is suspected (II, III, aVF) V4R should be obtained

To obtain V4R

Detach lead wire from V4 (left side of chest) and reconnect it to an additional electrode applied to the right side of chest

5th intercostal space midclavicular line

Under right nipple

ECG is again recorded and the right ventricle is now observed
Clinical Evidence of RVI

Triad of clinical (physical exam) findings

Hypotension

Jugular venous distention

Clear dry lungs

Pathophysiology

RV infarcts it does not effectively pump blood to and through pulmonary vasculature

Right atrial pressure rises and venous return exceeds ventricular output

Blood “backs up” into systemic vessels causing JVD

Hydrostatic pressure exceeds osmotic pressure

Lungs remain dry because blood is not pumped into pulmonary system from RV

Cardiac output is decreased and hypotension occurs

Amount of blood entering the lungs and LV is reduced

Remember the LV can only pump as much blood as it receives

LV receives less blood

Both stroke volume and CO are reduced

Analogy: Fire truck pump cavitating

Treatment of RVI

Elevation of RV filling pressure to maintain CO is goal of therapy

Judicious fluid therapy

Preload dependent

Fluid boluses in increments (200cc-300cc)

May require as much as 2-3 liters

Evaluate BP and breath sounds

PE develops an inotrope is necessary
Inotropic agents (dopamine, dobutamine)

Caution: Sensitivity to preload reducing agents

Nitrates and MS

Use with caution or avoid vasodilator therapy

Reduce venous return and exacerbate hypotension

Sublingual versus IV nitrates

Clinical Manifestations

Ischemic heart disease and its complications cause the greatest number of deaths each year in U.S.

Lethal dysrhythmias account for the overwhelming majority (V-Fib)

Many process produce imbalance between myocardial oxygen supply and demand that precipitate ischemia

Most common cause by far is atherosclerosis of coronary arteries

Commonly termed CAD