ECG's:

Take a second look at what you may be missing

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Trifascicular block:

Refers to a block in the Right Bundle, Left Anterior and Left Posterior Fascicles.

Ususally seen as an incomplete block with delayed conduction; i.e. 1st OR 2nd degree block in addition to a Bifasicular block. A bifascicular block is usually seen as RBBB with LAFB (or sometimes LPFB); LBBB is bifascicular. Also seen in 1st deg HB and LBBB.

Left Anterior Fascicular Block is defined as: small R waves inferiorly, small Q waves laterally, left axis deviation (large R wave lateral and deep S wave inferior), also have high voltage in limb leads.

Left Posterior Fascicular Block is defined as: Right axis deviation, small initial R and deep S wave in lateral, and small q and tall R in inferior.

Right Bundle Branch Block is defined as: long QRS, rSR' in V1, wide S laterally

Trifascicular block can occasionally be seen as a complete 3rd degree AV block (bifasicular block+3rd degree AV block).

If patient is symptomatic, require permanent pacemaker.

There are various causes including ischemia, valve disease, primary conducting system disease, congenital disease, from hyperkalemia or digoxin

Therefore look for **RBBB pattern, Left Axis Deviation and Long PR.**

Tall T waves (hyperkalemia, hyperacute)

The normal T wave is upright in all leads except AVR and V1, it is usually <5mm in limb leads and <15 mm in precordial leads.

Hyperacute T waves are broad based and asymmetrically (blunt) peaked, usually seen in the anterior leads and they are seen in early STEMI or with Prinzmetal angina. Hyperacute T waves are generally associated with low R wave voltage (i.e. they "ToweR" over R).

One study of computer modelling found that T to QRS amplitude >0.75 and a longer QTc was associated with MI.

Peaked T waves are seen in hyperkalemia, these are tall and narrow and symmetric (tented). Thus the morphology of T wave is important, and it is important to remember to look at the initial upslope of the T wave to see if it is straightening (indication of ischemia)

An upright T wave in V1, especially if it is larger than the T wave in V6 signifies a loss of precordial balance and is considered a hyperacute T wave. It can continue onto V2, and should be considered abnormal if this is a new finding.

Although an upright T in V1 can also be seen in LBBB/LVH/high voltage.

In one study, 84% of patients in a group of 218 with upright T in V1 had severe atherosclerotic disease to LCx or RCA.

Beware the **Tall T wave in V1.**

Horizontal ST segment that forms a sharp angle with the ascending limb of the T wave

Normally, the ST segment curves into the early T wave and has a mild concave shape, with no sharp angle. However, a convex elevation of the ST segment is typical of MI.

A horizontal ST segment is abnormal. An early sign of pending STEMI is a sharp angled ST-T junction, and thus the ST segment appears horizontal. This produces a flattened segment with an obvious acute angle to the T wave. This finding on ECG is more specific than downsloping of the ST segment.

Recent retrospective trial in Korea showed patients with sudden cardiac death arrest with BER pattern versus controls had horizontal ST segment (<0.1 mV within 100 ms after J point)

Look at the **Morphology of ST segment and angle to T wave**

Reciprocal changes: (can occur first before ST elevates – T wave should be concave up)

Reciprocal changes occur in STEMI due to lead placement, this can make diagnosing a STEMI easier. Occasionally reciprocal changes can be recognized before the ST elevation can be appreciated on ECG. These changes include T wave inversion, downsloping of ST segment, then ST depression. However, these changes can sometimes also be seen in blocks and hypertrophy.

Inferior STEMI (II, III, AVF) shows reciprocal changes in lateral leads, usually AVL and occasionally I and V5/V6. Furthermore, inverted T wave in AVL maybe the only early sign of a pending inferior STEMI. 40% of these have concomitant RV infarct, and can have hypotension. 20% get bradycardia (blocks) and some get posterior involvement. These are usually from RCA occlusion, however sometimes LCx is involved. Generally III STE is greater than II STE. There was a paper from Oman published in 2010 showing that isolated AVL T wave inversion portends an odds ratio of 2.93 in predicting coronary artery stenosis.

Anterior STEMI (usually from LAD) shows reciprocal changes inferiorly. Anterior MI have increased mortality, rates of CHF, and arrhythmia. Included in this group is LMCA and Wellens occlusions. If distribution is anterolateral, there are more ST depressions inferiorly. LMCA is diagnosed by seeing widespread ST depression with ST elevation in AVR>V1. Wellen's presents as deep precordial T wave inversions or biphasic T waves in V2-3 – this is synonymous with Proximal LAD occlusion.

Lateral STEMI usually is seen in association with other territories, however can sometimes be seen alone in occlusion of D1 from LAD, or OM of LCx – reciprocal changes occur in inferior leads III and AVF, and anterior leads V2 and V3.

Posterior infarcts present with tall broad R waves, upright T waves, dominant R waves (R/S ratio >1 in V2), and horizontal ST depression.

Right Ventricle infarcts show ST elevation in V1, III>II, and ST depression V2

Beware the **Inverted T wave AVL**

Inverted U waves: (Riera et al. Cardiology Journal 2008 Vol 15, no. 5, pp408-421)

U waves are best seen in V2, V3, it is the small deflection after T wave usually in same direction

Originate as delayed repolarization of purkinje fibers OR mid-myocardial cells, or after potentials from mechanical forces in ventricle wall

U wave is normally concordant with T wave, and gets larger with slower heart rates (<65), it is usually <33% of T wave amplitude and maximally 1-2mm

A prominent upright U wave is seen in: bradycardia, severe hypokalemia, hypocalcemia, hypomagnesemia, hypothermia, LVH, HOCM, raised ICP, digoxin, phenothiazines, Class Ia and Class III (Procainamide/amiodarone/sotalol)

An inverted U wave is highly specific for heart disease: CAD, HTN, valve disease, congenital disease, and cardiomyopathy

In the setting of chest pain, it is very specific for MI, it is also an early marker of unstable angina/prinzmetal angina or evolving MI, it can predict >75% stenosis to LAD and LMCA and LV dysfunction

Effort induced u wave inversion is 93% specific for proximal LAD stenosis (poorly sensitive)

If you are lucky enough to see an **Inverted U wave** think ischemia.