

About T waves - 2014

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The T wave is a positive deflection after each QRS complex. It represents ventricular repolarization. The T wave represents the unconcealed potential differences of ventricular repolarization.

Characteristics of the normal T wave

- Upright in all leads except aVR and V1
- Amplitude < 5 mm in limb leads, < 15 mm in precordial leads
- Normal profile of T wave with slow ascending ramp and faster descending ramp. When positive, T wave is characterized by being asymmetrical with its ascending slope being slow and of superior concavity, and fast descending slope.
- Representation of depolarization vectors (QRS) and ventricular repolarization (T wave). Both phenomena present similar directions, because in normal conditions, repolarization begins in the epicardium, while depolarization does it in the endocardium. As both phenomena are opposite, the polarities of the waves they represent are similar.

T-wave axis

- Frontal plane: SAT in the FP is between $+15^{\circ}$ & $+80^{\circ}$. T wave polarity nearly always positive in II; nearly always positive in aVF and I; variable (biphasic or inverted) in aVL and III; and always negative in aVR. Location in adults of normal T wave axis (SAT) in the frontal plane (near the $+60^{\circ}$). Extreme normal ranges of SAT in the frontal plane are -35° and $+90^{\circ}$. T wave inversion in lead III is a normal variant. New T-wave inversion (compared with prior ECGs) is always abnormal. Pathological T wave inversion is usually symmetrical and deep ($>3\text{mm}$).
- Horizontal plane SAT is heading to the left and slightly **IS HEADING TO THE LEFT AND SLIGHTLY** to the front and is very close to $V_6(0^{\circ})$. T wave is always positive from V_3 to V_6 ; generally positive in V_2 and frequently negative in V_1 . In normal adults, invariably the ventricular repolarization vector (T vector) is heading to the left and down, and usually discretely to the front near the $+10^{\circ}$. Always positive from V_3 to V_6 ; Generally positive in V_2 and frequently negative in V_1 . In adults older than 30 years old, negative T wave can only be found in V_1 being always positive from V_2 to V_6 . In newborn babies, SAT is heading towards the V_3 lead, i.e. around $+75^{\circ}$. Normal location in newborn babies of T wave axis (SAT) in the horizontal plane (near the $+75^{\circ}$ pointing towards V_3). Between 1h and 6h of life: SAT moves to the right, near $+100^{\circ}$. In these few initial hours, T wave polarity is negative in V_6 because SAT is located in the negative hemifield of this lead (SAT in $+100^{\circ}$). Normal location of the axis of the T wave in the horizontal plane between 1h and 6h of life: SAT is to the right, near the $+100$ degrees. In these few initial hours, T wave polarity is negative in V_6 , because the axis of the T wave is located in the negative hemifield of this lead. (SAT in $+100$ degrees). After 72h of life, SAT moves backwards, located near the -45 degrees. This explains why T wave is negative in the precordial leads V_1 to V_3 in children.

Duration

100ms to 250ms (up to five times more than ventricular depolarization).

T wave abnormalities

- Hyper acute T waves
- Inverted T waves
- Biphasic T waves
- “Camel Hump” T waves
- Flattened T waves

Peaked T waves

Tall, narrow base, symmetrically peaked T-waves are characteristically seen in hyperkalemia. It is observed when the rate of potassium reaches 5.5 mEq/l. The sensitivity is just 22% of cases. It is visible and may be confused with the T wave observed in bradycardia, diastolic LVE, subendocardial ischemia, schizophrenia and stroke.

And in Congenital Short QT syndrome. Positive polarity, wide base, symmetrical branches and acute apex. SUBENDOCARDIAL ISCHEMIA

Hyperacute T waves

Broad, asymmetrically peaked or ‘hyperacute’ T-waves are seen in the early stages of ST-elevation MI (STEMI) and often precede the appearance of ST elevation and Q waves. They are also seen with Prinzmetal angina.

T-WAVE ALTERNANS

The alternation of T wave polarity is a characteristic of patients carriers of long QT syndrome (LQTS).

Isolated T wave alternans is not related to tachycardia or extra-systole, and it usually indicates advanced heart disease or severe electrolytic disorder.

CAUSES OF ISOLATED T-WAVE ALTERNANS

- Tachycardia.
- Sudden changes in cycle length or HR cycle.
- Severe hyperpotassemia of uremia.
- Experimentally, in hypocalcemia in dogs.
- Severe myocardial impairment: cardiomyopathy.
- Acute myocardial ischemia, particularly in variant angina.
- Post-resuscitation.
- Acute pulmonary embolism.
- After administration of amiodarone or quinidine (rare).
- Congenital long QT syndromes of the Romano-Ward or Jervell-Lange Nielsen types.
- Brugada syndrome.

Loss of precordial T-wave balance

Loss of precordial T-wave imbalance occurs when the upright T wave is larger than that in V6. This is a type of hyperacute T wave.

- The normal T wave in V1 is inverted. An upright T wave in V1 is considered abnormal – especially if it is tall (TTV1), and especially if it is new (NTTV).
- This finding indicates a high likelihood of Coronary artery disease, and when new implies acute ischemia

Inverted T waves

Inverted T waves are seen in the following conditions:

- Normal finding in children
- Persistent Juvenile T wave pattern
- Myocardial ischemia and infarction: Subepicardial ischemia: negative polarity, wide base, symmetrical branches and acute nadir: T in “seagull wings”.
- Bundle branch Block
- Ventricular pre-excitation
- Ventricular hypertrophy (“strain pattern”) LVH in V5-6 Strain pattern of right ventricular enlargement: in V₁ and V₂; Suprasystemic right intraventricular pressure. V2 and V3 continue showing QRS predominantly positive. Repolarization pattern and QRS in right precordial leads (V3R-V1 and V2) in congenital heart disease with suprasystemic right intraventricular pressure (severe pulmonary stenosis): QRS: qR pattern, ST and inverted T wave with branches that show a tendency to be symmetrical.
- Inverted T wave and with a tendency to be symmetrical (primary).
- Hypertrophic cardiomyopathy In Hypertrophic Apical Cardiomyopathy;
- Raised intracranial pressure: Strokes: great a inegative t waves in precordial leads.
- After Adams-stokes Episode: giant T waves, deeply inverted and with prolonged QT interval. This situation causes a tendency to appearance of polymorphic ventricular tachycardia of the torsade des pointes (TdP) type.
- After removing artificial pacemaker

Pediatric T waves

Inverted T-waves in the right precordial leads (V1-3) are a normal finding in children, and adolescents representing the dominance of right ventricular forces.

Persistent Juvenile T-wave Pattern

T-wave inversions in the right precordial leads may persist into adulthood and are most commonly seen in young Afro-Caribbean women. Persistent juvenile T-waves are asymmetric, shallow (<3mm) and usually limited to leads V1-3.

Myocardial Ischemia and Infarction

T-wave inversions due to myocardial ischemia or infarction occur in contiguous leads based on the anatomical location of the area of ischemia/infarction:

- Inferior = II, III, aVF
- Lateral I, aVL, V5-6
- Anterior = V2-6

Dynamic T-wave inversion are seen with acute myocardial ischemia

Fixed T-wave inversions are seen following infarction, usually in association with pathological Q waves.

Left Bundle Branch Block produces T-wave inversion in the lateral leads I, aVL, and V5-6.

Right bundle branch block produces T-wave inversion in the right precordial leads V1-3.

Left ventricular hypertrophy produces T-wave inversion in the lateral leads I, aVL, V5-6 (left ventricular ‘strain’ pattern), with a similar morphology to that seen in LBBB.

Right ventricular hypertrophy produces T-wave inversion in the right precordial leads V1-3 (right ventricular ‘strain’ pattern) and also the inferior leads (II, III, aVF).

Pulmonary Embolism

Acute right heart strain (e.g. secondary to massive pulmonary embolism) produces a similar pattern to RVH, with T-wave inversions in the right precordial (V1-3) and inferior (II, III, aVF) leads.

Pulmonary embolism may also produce T-wave inversion in lead III as part of the $S_I Q_{III} T_{III}$ pattern (S wave in lead I, Q wave in lead III, T-wave inversion in lead III)

Hypertrophic Cardiomyopathy (HOCM)

HOCM is associated with deep T wave inversions in all the precordial leads.

Raised intracranial pressure

Events causing a sudden rise in ICP (e.g. subarachnoid hemorrhage) produce widespread deep T-wave inversions with a bizarre morphology

Biphasic T waves

There are three main causes of biphasic T waves:

- Myocardial ischemia
- Hypokalemia
- LQT2.

The two waves go in opposite directions:

Ischemic T waves go up then down

Hypokalemic T waves go up down then up.

Wellens' Syndrome

Wellens' syndrome is a pattern of inverted or biphasic T waves in V2-3 (in patients presenting with ischemic chest pain) that is highly specific for critical stenosis of the left anterior descending artery.

There are two patterns of T-wave abnormality in Wellens' syndrome:

- Type 1 Wellens' T-waves are deeply and symmetrically inverted
- Type 2 Wellens' T-waves are biphasic, with the initial deflection positive and the terminal deflection negative.

'Camel hump' T waves

- This is a term used by the great ECG lecturer and Emergency Physician Amal Mattu to describe T-waves that have a double peak or 'camel hump' appearance.

- There are two causes for camel hump T waves:

Prominent U waves fused to the end of the T wave, as seen in severe hypokalemia

Hidden P waves embedded in the T wave, as seen in sinus tachycardia and various types of heart block.

Flattened T waves

Flattened T waves are a non-specific finding, but may represent ischemia (if dynamic or in contiguous leads) or electrolyte abnormality, e.g. hypokalemia (if generalized).

DECREASE OF T WAVE VOLTAGE

- Sympathotonia.
- Chronic coronary insufficiency, (it reaches several leads).
- Digitalis effect.
- Hypopotassemia, (associated to ST depression and appearance of prominent U).
- Hypothyroidism (usually they reverse in weeks or months with specific treatment).

Ischaemia

Dynamic T-wave flattening due to anterior ischaemia . T waves return to normal once the ischaemia resolves.

Hypokalaemia

Generalized T-wave flattening with prominent U waves in the anterior leads (V2 and V3).

The normal value of T_{peak}/T_{end} interval (T_{pe}) is 94 ms in men and 92 in women when measured in the V₅ lead. T_{pe} prolongation to values ≥ 120 ms is associated to a greater number of events in patients carriers of BrS Interval elapsed from the apex to the end of T wave (T_{peak}-T_{end} interval or T_{pe}). T_{pe} may correspond to transmural dispersion of repolarization and consequently, the amplification of this interval is associated to malignant ventricular arrhythmias.

References

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