Normal and pathological T-wave

T-wave definition

The T wave is the most labile wave in the ECG and represents the mid-latter part of ventricular repolarization, correspondent to phase 3 of the monophasic action potential (AP). In phase 2 the inward sodium and calcium current decays with time; the outward potassium current is activated allowing the cell to move to resting state. Phase 3 is caused by the closing of the long-lasting or the *L*-type *calcium* channel L Ca²⁺ Calcium voltage-gated calcium channels (VGCCs) and the opening of delayed rectifier K⁺ channels; I_{Ks}, I_{Kr} and I_{kur}. A sustained K⁺ efflux with a delay after membrane depolarization. Flows out of the cell due to its electrochemical gradient, to restore the resting membrane potential near -88 to -90 mV. The end of phase 3 is called Maximal Diastolic Potential (MDP).





Figure x. Normal profile of T wave with slow ascending ramp and faster descending ramp.

Representation of T vector of ventricular repolarization



Figure x. 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.

Items to be analyzed in the T wave

- 1. T-wave shape
- 2. Normal T-wave axis (SÂT) in the frontal plane (FP) and horizontal plane

(**HP**).

- 3. Normal Spatial QRS-T angle
- 4. Normal T-wave voltage or amplitude
- 5. Normal T-wave polarity in adults
- 6. T-wave duration

1. Normal T-wave shape or T wave aspect

The normal profile or shape of the T wave is asymmetric, with slow ascending ramp (upstroke) and faster descending ramp. a rapid down stroke. When the T wave has positive polarity, it normally rises slowly and then abruptly returns to the baseline. Consequently, this wave is normally slightly asymmetric since its downslope (second half) is steeper than its upslope. When the T-wave it is negative, it descends slowly and abruptly rises to the baseline. (Becker DE. Fundamentals of electrocardiography interpretation. Anesth Prog. 2006 Summer;53(2):53-63). In other words, normal profile of T wave has slow ascending ramp with a rounded peak and faster descending ramp. The exact point at which the ST segment ends and the T wave begins is somewhat arbitrary and usually impossible to pinpoint precisely. However, for clinical purposes accuracy within 40 msec (0.04 sec) is usually acceptable. Figure below the T-wave is relative asymmetry may vary because many females and elderly individuals, without identifiable cardiac disease, may have symmetric T waves. If the T wave appears symmetric, cardiac pathology such as ischemia may be present.



Figure. Normal profile of T wave with slow ascending ramp and faster descending ramp.

The end of the T wave is considered as the intercept between the isoelectric line with the tangent drawn through the maximum down slope (descendent ramp) of the T wave. When positive, it is characterized by being asymmetrical with its slow ascending ramp and with superior concavity and fast 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.

2. Normal T-wave axis or SÂT in the frontal plane

The normal adults the T-wave axis is between 0° to $+90^{\circ}$.

T wave axis or SÂT in the frontal plane: in adults



Figure x. Location in adults of normal T wave axis (SÂT) in the frontal plane (near the $+60^{\circ}$). The T wave is always positive in II; aVF and I; variable (biphasic or inverted) in aVL and III; and always negative in aVR.



Normal T-wave axis in adults or SÂT in the frontal plane.

Figure x. Extreme normal ranges of SAT in the frontal plane (-35° and +90°).

Kors et al considered a frontal normal T-axis between +15° and +75° as normal (Kors JA, de Bruyne MC, Hoes AW, van Herpen G, Hofman A, van Bemmel JH, et al. T-axis as an indicator of risk of cardiac events in elderly people. Lancet. 1998 Aug 22;352(9128):601-5. doi: 10.1016/S0140-6736(97)10190-8.)

Aro et al, considered the normal T-wave axis between 0° to +90°, These authors demonstrate that a wide frontal QRS-T angle measured from a standard 12-lead ECG increases the risk of arrhythmic death in excess of two-fold in both genders. The prognostic significance of a wide QRS-T angle was mainly due to an abnormal T-wave axis, while QRS-axis alone had no effect on prognosis. (Aapo L. Aro, Heikki V. Huikuri, Jani T. Tikkanen, M. Juhani Junttila, Harri A. Rissanen, Antti Reunanen, Olli Anttonen. QRS-T angle as a predictor of sudden cardiac death in a middle-aged general population EP Europace, Volume 14, Issue 6, June 2012, Pages 872–876, <u>https://doi.org/10.1093/europace/eur393</u>)

Normal T-wave axis (or SÂT) in the horizontal plane: polarity in normal adults



Figure. Location of SÂT in the horizontal plane in normal adults (around $+10^{\circ}$). SÂT is heading to the left and slightly to the front and is very close to V5-V6. Always positive from V3 to V6; generally positive in V2 and frequently negative in V1. 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 $+10^{\circ}$.

Normal SÂT in the horizontal plane



Figure x. Location in adults of normal T wave axis (SAT) in the frontal plane (near the +60°). Location in adults of normal T wave axis (SAT) in the frontal plane (near the +60°). Normal SÂT in newborn babies in the HP



Figure x. Normal location in newborn babies of T wave axis (SÂT) in the horizontal plane (near the $+75^{\circ}$ pointing towards V3). In newborn babies, SÂT is heading towards the V3 lead, i.e. around $+75^{\circ}$.

Normal SÂT between 1h to 6h of life in the Horizontal Plane



Figure. Normal location of the T wave axis in the horizontal plane between 1h and 6h of life: SÂT is to the right, near the $+100^{\circ}$. In these few initial hours, T wave polarity is negative in V6, because the axis of the T wave is located in the negative hemifield of this lead. (SÂT in $+100^{\circ}$). Between 1h and 6h of life: SÂT moves to the right, near $+100^{\circ}$. In these few initial hours, T wave polarity is negative in V6 because SÂT is located in the negative hemifield of this negative hemifield of this lead (SAT in $+100^{\circ}$).





Figure x Normal location of T wave axis (SÂT). After 72h of life, SÂT moves backwards, located near the -45° . This explains why T wave is negative in the precordial leads V1 to V3 in children. After 72h of life, SÂT moves backwards, being near -45° .

3. Normal Spatial QRS-T angle

The QRS-T angle is defined as the angle between the directions of ventricular depolarization and repolarization. In normal adults, the normal Spatial QRS-T angle is between 0° to +90°, QRS-axis -30 to +90°. Therefore, a wide QRS-T angle reflects either structural abnormalities affecting the depolarization or regional pathophysiological changes in ionic channels altering the sequence of repolarization. In an elderly general population cohort from Rotterdam, the Netherlands, the spatial QRS-T angle was found to be a strong predictor of cardiac mortality. (Kardys I, Kors JA, van der Meer IM, Hofman A, van der Kuip DA, Witteman JC. Spatial QRS-T angle predicts cardiac death in a general population, Eur Heart J, 2003, vol. 24 (pg. 1357-64)) The spatial QRS-T angle is a significant and independent predictor of cardiovascular death. It provides more information than any other established ECG characteristics and can easily be calculated as part of computerized ECG analysis. The standard ECG is widely available, playing a critical role in evaluation of possible and known cardiac disease. Spatial or vectorcardiography assessment of the angle between the QRS and T-wave vectors offers unique information not obtained from conventional markers derived from standard ECG. It can be used to further stratify patients with standard ECG abnormalities. The prognostic value of spatial QRS-T angle provide clinicians with cut points for its clinical application. The physician can apply these cut points to assist decision making regarding further cardiac testing, the intensity of risk factor management, and/or referral to a cardiologist. (Takuya Yamazaki 1, Victor F Froelicher, Jonathan Myers, Sung Chun, Paul Wang; Spatial QRS-T angle predicts cardiac death in a clinical population. Heart Rhythm. 2005 Jan;2(1):73-8. doi: 10.1016/j.hrthm.2004.10.040.)

4. Normal T-wave voltage or amplitude

T-wave voltage is less than 5 mm in limb leads, less than 10 mm in precordial leads. (Wei Qin, Lin; Swee, Guan Teo; Kian Keong, Poh (2013). "Electrocardiographic T wave abnormalities" (PDF). Singapore Medical Journal. 54 (11): 606–610. doi:10.11622/smedj.2013218.). The T-wave amplitude is highest in V2-V3. The amplitude diminishes with increase age. A decrease in T wave amplitude or T wave inversion in leads I, II, and V_2 – V_4 occurs frequently within 30 minutes after a meal of about 1200 calories. Postprandial T wave abnormalities occurred in 3.9 percent of 2000 young, healthy airmen. These abnormalities may arise from lowering of the plasma potassium concentration, tachycardia, and possibly sympathetic stimulation. It has been

shown that various nonspecific T wave abnormalities may disappear when the ECG is recorded after fasting.

5 Normal T-wave polarity in adults

Normal T-wave polarity is upright in all leads I, II, and V3-V6, and always inverted or negative in aVR, III and V1 or V1-V2. Generally, the T-waves are negative in leads aVR, V₁ and III. The T-wave in V₁ may be inverted normally at any age and in V₂ it is sometimes normally negative. (Marriott HJL. Practical electrocardiography. 7th ed. Baltimore/London: Williams & Wilkins; 1983.) The other leads are variable depending on the direction of the QRS and the age of the patient. T wave inversions from V1 to V4 leads are frequently found and normal in children. In normal adults, T wave inversions are less commonly found, but can be normal from V1 to V3. (Wei Qin, Lin; Swee, Guan Teo; Kian Keong, Poh (2013). "Electrocardiographic T wave abnormalities" (PDF). Singapore Medical Journal. 54 (11): 606–610. doi:10.11622/smedj.2013218). (Salah AM Said, Rene Bloo, Ramon de Nooijer, and Andries Slootweg Cardiac and non-cardiac causes of T-wave inversion in the precordial leads in adult subjects: A Dutch case series and review of the literature Clinical causes of T-wave inversionWorld J Cardiol. 2015 Feb 26; 7(2): 86–100.Published online 2015 Feb 26. doi: 10.4330/wjc.v7.i2.86)

7.Normal T-wave inversion in the anterior chest wall leads in children and adolescents. It is relatively common(Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009;53:982–991.)

8. Athletes engaged in high intensity dynamic endurance sport activities

(Fagard R. Athlete's heart. Heart. 2003;89:1455–1461.)

Causes of pathologic T-wave inversion

- Heart block,
- Ischemic heart disease,
- o Bradycardia,
- Right ventricular hypertrophy subsequent to alterations of sequence of ventricular activation.
- Right bundle branch block: subsequent to alterations of sequence of ventricular activation.
- Metabolic disturbances,
- Changes during diagnostic coronary angiography
- Changes during cerebral disturbance., (Jacobson D, Schrire V. Giant T wave inversion. Br Heart J. 1966;28:768–775.)
- o LV anterior wall ischemia,
- o Acute central nervous system disorders,
- Acute adrenergic stress (Takotsubo cardiomyopathy "TTC") \cap (Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, Yoshiyama M, Miyazaki S, Haze K, Ogawa H, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. J Am Coll Cardiol. 2001;38:11-18.)(Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC. Neurohumoral features of myocardial sudden emotional stress. N stunning due to Engl J Med. 2005;352:539-548)
- Pulmonary edema (Littmann L. Large T wave inversion and QT prolongation associated with pulmonary edema: a report of nine cases. J Cardiol. 1999;34:1106-1110.)(Pascale Am Coll P, Quartenoud B, Stauffer JC. Isolated large inverted T wave in edema due to hypertensive pulmonary crisis: a novel electrocardiographic phenomenon mimicking ischemia? Clin Res Cardiol. 2007;96:288-294.)

- Antiarrhythmic drug effects (Said SA, Somer ST, Oude Luttikhuis HA. Flecainide-induced JT prolongation, T wave inversion and ventricular tachycardia during treatment for symptomatic atrial fibrillation. Int J Cardiol. 1994;44:285–287.)
- Pulmonary embolism, (Pillarisetti J, Gupta K. Giant Inverted T waves in the emergency department: case report and review of differential diagnoses. J Electrocardiol. 2010;43:40–42.)
- Cardiac memory secondary consequence of:
 - Transient tachycardia (Hanna EB, Glancy DL. ST-segment depression and T-wave inversion: classification, differential diagnosis, and caveats. Cleve Clin J Med. 2011;78:404–414.)
 - Post-ventricular pacing states, (Littmann L, Fertman AF. Large T-wave inversion in a patient with a pacemaker. Arch Intern Med. 2011;171:1314; discussion 1315–1316.)
 - Idiopathic (Corbella F, Dragonetti L, Rivas C, Eyheremendy E, Acunzo R. Giant negative T waves of indeterminate origin. Rev Argent Cardiol. 2009;2:131–134.)
 - Relation to cocaine use (Dhawan SS. Pseudo-Wellens' syndrome after crack cocaine use. Can J Cardiol. 2008;24:404.)(Zimmerman FH, Gustafson GM, Kemp HG. Recurrent myocardial infarction associated with cocaine abuse in a young man with normal coronary arteries: evidence for coronary artery spasm culminating in thrombosis. J Am Coll Cardiol. 1987;9:964–968.)
- Congenital coronary artery-ventricular multiple micro-fistulas (MMFs).(
 Said SA, Schiphorst RH, Derksen R, Wagenaar L. Coronary-cameral fistulas in adults (first of two parts) World J Cardiol. 2013;5:329–336.)

T-wave inversion in right precordial leads is relatively rare (0.5%) in the general population and not associated with adverse outcome. Aro AL, Anttonen O, Tikkanen JT, Junttila MJ, Kerola T, Rissanen HA, Reunanen A, Huikuri HV. Prevalence and prognostic significance of T-wave inversions in right precordial leads of a 12-lead electrocardiogram in the middle-aged subjects. Circulation. 2012;125:2572–2577.

T-wave inversion in the anterior precordial leads

- 1. With corrected QT prolongation >450ms using Bazett formula (. Bazett HC. An analysiss of time relations of electrocardiograms. Heart. 1920;7:353–370.)
- 2. Without corrected QT prolongation <450ms

T-wave inversion classification according to its depth

- Giant negative T-wave is defined as ≥ 10 mm in any of the leads. (Eriksson MJ, Sonnenberg B, Woo A, Rakowski P, Parker TG, Wigle ED, Rakowski H. Long-term outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol. 2002;39:638–645.) (Hanna EB, Glancy DL. ST-segment depression and T-wave inversion: classification, differential diagnosis, and caveats. Cleve Clin J Med. 2011;78:404–414.) (Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009; 53:982–991.)
- 2. Deep T-wave negative ≥ 5 mm (Hanna EB, Glancy DL. ST-segment depression and T-wave inversion: classification, differential diagnosis, and caveats. Cleve Clin J Med. 2011;78:404–414.)
- 3. Mild T-negative 1-3mm

6. Normal T-wave Duration

 T_{peak} - T_{end} interval: is a subinterval of the T-wave from peak of the T-wave until the end of the T-wave T_{peak} - T_{end} interval (Haarmark C, Graff C, Andersen MP, et al. (2010). "Reference values of electrocardiogram repolarization variables in a healthy population". Journal of Electrocardiology. 43 (1): 31–39. doi:10.1016/j.jelectrocard.2009.08.001) 7.Voltage or amplitude of the T wave



Figure x. The values of normal maximal voltage are described in the frontal and horizontal planes. Characteristics of T wave in subepicardial ischemia: inverted, symmetrical and with a wide base: T wave in seagull wing.

The maximal normal limit of T wave in the FP is 5 mm or 6 mm and for the HP 10 mm to 12 mm.

About precordial leads, V2 and V3 have a greater voltage and the left ones, (V5 and V6)

have lower voltage.

The increase in voltage may be:

- Physiological: parasympathetically.
- Pathological: ischemic T wave.
- Subepicardial ischemia: negative polarity, wide base, symmetrical branches and acute nadir: T in "seagull wings".

Main causes of deep negative T waves

- Strokes: great negative t waves in precordial leads.
- Strain pattern of right ventricular enlargement: in V1 and V2;
- After Adams-stokes Episode;
- In Hypertrophic Apical Cardiomyopathy;
- After removing artificial pacemaker.
- Acute adrenal crisis (Iga, Hori et al. 1992)
- Wellens Syndrome (Perez Riera, Ferreira et al. 2008) (Riera, Ferreira et al. 2008)
- Stress-induced cardiomyopathy (Simoes, Marin-Neto et al. 2007)
- Tako-tsubo cardiomyopathy (Athanasiadis, Vogelsberg et al. 2006)
- Sodium azide poisonings
- 1. Induced by cardiac compression from a retrosternal gastric tube used to reconstruct the esophagus after tumor resection (Takato, Ashida et al. 2006)

T-wave inversion classification according to its depth

- A. Giant negative T-wave is defined as \geq 10 mm in any of the leads. (Eriksson MJ, Sonnenberg B, Woo A, Rakowski P, Parker TG, Wigle ED, Rakowski H. Longterm outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol. 2002;39:638–645.)(Hanna EB, Glancy DL. ST-segment depression and T-wave inversion: classification, differential diagnosis, and caveats. Cleve Clin J Med. 2011;78:404-414.) (Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009; 53:982–991.)/(Aro, AL 1, Huikuri HV, Tikkanen JT, Junttila MJ Rissanen HA, Reunanen A, Anttonen O. QRS-T angle as a predictor of sudden cardiac death in a middle-aged general population Europace. 2012 Jun;14(6):872-6. doi: 10.1093/europace/eur393.)
- B. Deep negative T-wave \geq 5 mm
- C. Mild T-wave inversion 1-3mm



Figure x. Clinical diagnosis: Subarachnoid bleeding. ECG diagnosis: long QT interval, largely wide and inverted T waves: "giant T waves". ECG that shows inverted T waves, with great width and wide base with prolonged QT interval in a patient with subarachnoid bleeding.

Strain pattern of Right Ventricular Enlargement: inV_1 and V_2 .



Figure 1x. Repolarization pattern and QRS in right precordial leads (V3R-V1 and V2) in congenital heart disease with suprasystem right intraventricular pressure: QRS: qR or qRs pattern, ST depression upwardly convex and inverted T wave with branches that show a tendency to be symmetrical. The classical example of strain pattern is found in severe pulmonary stenosis.

Strain pattern of RVH severe pulmonary stenosis



Figure x Supra-systemic right intraventricular pressure. V2 and V3 continue showing QRS predominantly positive. Inverted T wave and with a tendency to be symmetrical (primary). 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.



Figure x. ECG strip that shows total AV block in a patient that suffered a recent episode of Stokes-Adams: 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. Negative T wave after Adams-Stokes episode in complete AV block.

Name: SFS.Age: 15y.Weight: 70Kg.Height: 1.72m.Medication in use: beta-blocker.

Sex: M. Number: 718. Race: W. Date: 03/31/98



Figure x Typical ECG of obstructive form of hypertrophic cardiomyopathy in a 15-yearold teenager. Left chamber enlargement, important depression of ST segment upwardly convex and followed by wide-based and deeply inverted T waves: alteration secondary to ventricular repolarization in antero-lateral and inferior wall. HCM obstructive form. Apical area of the septum with 32 mm of diastolic thickness. LAE. Systolic pattern of LVH by important alteration secondary to ventricular repolarization in antero-lateral and inferior wall.

Hyperacute phase of anterior STEMI

Hyperacute T-waves

Immediately after coronary artery occlusion, the ECG undergoes predictable temporal changes. Classically, coronary vessel occlusion leads to elevation of the ST-segments (producing STEMI). However, the earliest findings on an ECG are subtle changes in the T-wave shape and size. When a coronary artery is occluded, within the first 30 minutes, the T-wave amplitude increases. (**Dressler, W and Roesler, H. High T waves in the earliest stage of myocardial infarction.** <u>Am Heart J.</u> 1947 Nov;34(5):627-45.). The next changes are ST-segment elevation and loss of the R-wave amplitude. If the vessel remains occluded, Q-waves develop. Without intervention, the ECG will then begin to exhibit T-wave inversions and eventually, the ST-segments will normalize. (Nable, JV and Brady, W. The evolution of electrocardiographic changes in ST-segment

elevation myocardial infarction. <u>Am J Emerg Med.</u> 2009 Jul;27(6):734-46.) Persistent ST-segment elevation suggests aneurysm formation. Early in the course of AMI, biochemical markers may not be elevated, although this may be changing in the era of highly sensitive troponin assays. Regardless, the **development of T-wave changes is** the first sign that we can see on the ECG and the ECG is fast, cheap, noninvasive, and readily available in the ED. In the early stages of MI, prior to the development of necrosis, the myocardium is suffering from ischemia. Timely revascularization may actually prevent complete infarction and death of the affected portion of the myocardium. Therefore, recognizing ACS early is beneficial because patients have improved outcome the timelier revascularization occurs (Nable, JV and Brady, W. The evolution of electrocardiographic changes in ST-segment elevation myocardial infarction. Am J **Emerg Med. 2009 Jul;27(6):734-46**.), and delay to reperfusion causes larger infarction size and worse functional outcomes. (Keeley, EC et al. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet. 2003 Jan 4;361(9351):13-20.). It is well known that new ST-segment elevation represents complete vessel occlusion and transmural infarct. However, the STEMI criteria have limited sensitivity in diagnosing coronary artery occlusion. (Zarling, EJ et al. Failure to diagnose acute myocardial infarction: The clinicopathologic experience at a large community hospital. JAMA. Sep 1983;250(9):1177-81.)(Scott, PJ et al. Optimization of the precordial leads of the 12lead electrocardiogram may improve detection of ST-segment elevation myocardial infarction. J Electrocardiol. Jul-Aug 2011;44(4):425-31.)(Thygesen, K et al; Third universal definition of myocardial infarction. Circulation. Oct 2012;126(16):2020-**2035.**). This means that some patients ultimately diagnosed with NSTEMI will also have complete coronary artery occlusion.

Below are the AHA criteria that define STEMI(**Thygesen**, **K** et al; **Third universal** definition of myocardial infarction. <u>Circulation</u>. Oct 2012;126(16):2020-2035.)

ECG Manifestations of Acute Myocardial Ischemia

ST elevation

New ST elevation at the J point in two adjacent leads with the cut-points \geq 0.1 mV in all leads other V2-V3 where the following cut points apply \geq 0.2mV in men \geq 40mm year or \geq 0.15mV in women.

ST depression and T wave changes

New horizontal or down-sloping WT depression ≥ 0.05 mV in two contiguous leads and /or T inversion ≥ 0.1 mV in two contiguous leads with prominent R wave or R/S ratio>1.

It is important to recognize an obvious STEMI, but patients may present initially with only subtle ECG changes and minimal ST-segment elevation they may not meet the official criteria. These "subtle STEMI" patients have higher rates of inappropriate ED discharge and significant delays to reperfusion. (Pope, JH et al. Missed diagnoses of acute cardiac ischemia in the emergency department. <u>N Engl J Med</u>. Apr 2000;20;342(16):1163-70.)(Sharkey, SW et al. Impact of the electrocardiogram on the delivery of thrombolytic therapy for acute myocardial infarction. <u>Am J Cardiol.</u> Mar 1994;15;73(8):550-3.)(Martí, D et al. Incidence, angiographic features and outcomes of patients presenting with subtle ST-elevation myocardial infarction. <u>Am Heart J.</u> Dec 2014;168(6):884-90.)

It is true that more ST elevation indicates a larger area of infarcted myocardium, however patients with subtle ST elevation MI experience similar functional outcomes and mortality rates as those with obvious STEMI. (Martí, D et al. Incidence, angiographic features and outcomes of patients presenting with subtle ST-elevation myocardial infarction. <u>Am Heart J.</u> Dec 2014;168(6):884-90.) Furthermore, approximately 25% of patients who do not meet the STEMI criteria, and are diagnosed with NSTEMI, have a completely occluded artery on angiography. (Wang, TY et al. Incidence, distribution, and prognostic impact of occluded culprit arteries among patients with non-ST-elevation acute coronary syndromes undergoing diagnostic angiography. <u>Am Heart J.</u> Apr 2009;157(4):716-23.). Some experts would

argue that patients with subtle findings of coronary vessel occlusion should be treated as expeditiously as patients with obvious STEMI. (Martí, D et al. Incidence, angiographic features and outcomes of patients presenting with subtle ST-elevation myocardial infarction. <u>Am HeartJ.</u> Dec 2014;168(6):884-90.).

The T-wave is often the first deflection on the ECG to change in acute vessel occlusion. **Initial changes to the T-wave are straightening of the ST-segment and enlargement of the T-wave height and width.** The T-wave becomes disproportionately large when compared to the QRS. The prominent T-waves seen early in coronary vessel occlusion are called hyperacute T-waves. They were first described in 1947 as an early marker of coronary artery occlusion (Dressler, W and Roesler, H. High T waves in the earliest stage of myocardial infarction. <u>Am Heart J.</u> 1947 Nov;34(5):627-45.)

Hyperacute T-waves are often bulky, and wide at the base and are localized to an anatomic area of infarct. The widening of the T-wave may also lengthen the QT interval. It must be emphasized that hyperacute T-waves are not necessarily always tall, they **may only be relatively large when compared to the R-wave.** This means that even a small T-wave can still be hyperacute if paired with a low-voltage QRS. It is important to note that there is no acceptable universal definition of hyperacute T-waves, but there can be other clues on the ECG. During the development of hyperacute T-waves, there can be associated ST-segment depression in the reciprocal leads.



Typical example of an ECG with hyperacute T-waves localized to the anterior wall.

Do not be distracted by the first-degree AV block or by the PVCs. This ECG shows very prominent, broad-based T-waves in the anterior leads (V2-6). Notice also the loss of R-wave height throughout the precordium and the how the T-waves are massive in comparison to the QRS complexes. This ECG is concerning for LAD occlusion.



The patient underwent repeat ECG 40 minutes later, which showed obvious anterior ST elevation. Now there is obvious ST elevation in the anterior leads (V2 and V3), as well as ST elevation in the lateral leads (I, aVL, V5 and V6) with reciprocal depression in lead III. Also, the Q-waves are deepening in the leads V2 and V3.



Here is another example of hyperacute T-waves, this time in the inferior leads. This is the ECG of a 75 year-old woman presenting with chest pain: *Notice the large T waves in the inferior leads. The total height of the T-waves is not all that impressive, but when compared to the QRS complexes, especially in aVF, the T-wave is massive. The ST-segments are straighter than normal and there is subtle ST elevation in lead III, aVF, V5-V6. Notice the subtle reciprocal ST depression and T-wave inversion in aVL. The machine read this ECG as Early Repolarization. Her Troponin I came back slightly elevated (0.07 ng/mL). She was found to have complete occlusion of the RCA on angiogram and was diagnosed with "NSTEMI".*

De Winter T-waves

An interesting variant of hyperacute T-waves are those paired with J-point depression. This causes a T-wave takeoff point that is below the isoelectric line. This "depressed T-wave takeoff" pattern was first described in 2009 by Verouden and colleagues and was found to represent complete LAD occlusion (a STEMI-equivalent) (Verouden, NJ et al. Persistent precordial "hyperacute" T-waves signify proximal left anterior descending artery occlusion. <u>Heart.</u> 2009 Oct;95(20):1701-6.) This pattern of up sloping ST-segment depression paired with a tall, prominent T-wave is present in about 2% of patients with LAD occlusion. It was initially postulated that these findings are not dynamic, but rather that they remain static throughout coronary vessel occlusion until the time of reperfusion (Verouden, NJ et al. Persistent precordial "hyperacute" T-waves

signify proximal left anterior descending artery occlusion. <u>Heart.</u> 2009 Oct;95(20):1701-6.) (de Winter, RW et al. Precordial junctional ST-segment depression with tall symmetric T-waves signifying proximal LAD occlusion, case reports of STEMI equivalence. <u>J Electrocardiol.</u> 2015 Oct 13.). However, some experts have documented de Winter T-waves developing during anterior STEMI and would argue that these findings may represent subtotal occlusion of the LAD. Regardless, these patients require immediate reperfusion.



This ECG initially appeared at: http://lifeinthefastlane.com/ecg-library/de-winters-twaves/Notice the up sloping ST depression seen in leads V2-V6 followed by very tall and symmetric T-waves. Notice also the subtle reciprocal depression in the inferior leads (II, III, and aVF).

For an interesting case of the de Winter T-wave pattern occurring in a patient who initially presented with an obvious anterior STEMI, read this case on Dr. Smith's ECG blog: <u>http://hqmeded-ecg.blogspot.com/2014/07/is-lad-really-completely-occluded-when.html</u>.

Pseudonormalization

Another interesting phenomenon of the T-waves is the pseudonormalization in AMI. This occurs when a patient with baseline T-wave inversions presents with acute coronary occlusion. Hyperacute T-waves in these patients manifest as upright T-waves, which may be confused for a normal ECG. This finding highlights the fact that it is not solely the height, but rather the increase in positive amplitude, that signifies a hyperacute T-wave. (Dressler, W and Roesler, H. High T waves in the earliest stage of myocardial infarction. <u>Am Heart J.</u> 1947 Nov;34(5):627-45.). The other scenario for pseudonormalization is a patient who presents with re-occlusion of a recently reperfused artery, also known as Wellens' Syndrome.

Here is the classic appearance of Wellens' syndrome, type A (left) and type B (right):



These images originally appeared at: <u>http://lifeinthefastlane.com/ecg-library/wellens-</u> syndrome/

It is important to note that this pattern appears when the patient is asymptomatic because this represents a reperfusion pattern on ECG. Type A, the biphasic T-waves, are seen immediately upon reperfusion. As the artery remains open, the T-waves evolve to be more deeply inverted, a Type B pattern. When the patient becomes symptomatic it is because the vessel re-occludes. When that happens the T-waves become upright (pseudonormalization) and if it remains occluded, ST-segment elevation will appear. These lesions are unstable because the vessel can re-occlude at any time and the patient requires revascularization.

Hyperkalemia

Perhaps the most well known cause of prominent T-waves is the peaked T-waves seen with hyperkalemia, and they can be confused with the hyperacute T-waves of ACS. There is no exact correlation between serum potassium and onset of ECG changes but about 80% of patients begin to exhibit ECG changes at 6.8-7.0mEq/L. The typical progression of ECG changes in hyperkalemia is first the development of peaked T-waves, followed by decreased P-wave amplitude, widening of the QRS complex and finally development of a sine wave. But, as we know, hyperkalemia can cause a myriad of ECG changes including AV and bundle branch blocks, bradycardias, and even a STEMI mimic. Here are the typical changes with hyperkalemia.



This image originally appeared at http://lifeinthefastlane.com/hyperkalemia/Although the T-waves of early hyperkalemia are very tall and prominent, the key differentiator from hyperacute T-waves is the shape of the T-wave. Hyperacute T-waves are fat and wide with a more blunted peak. The T-waves of hyperkalemia are very pointy, peaked or "tented" with a narrow base, they have sharp apex and tend to be extraordinarily symmetric.(Somers, MP et al. The prominent T wave: electrocardiographic differential diagnosis. Am J Emerg Med. 2002 May;20(3):243-51.)

The hyperkalemic T waves



Figure x. Tent-shaped T wave of hyperpotassemia: tall and narrow-based. This wave is observed with slightly increased serum potassium levels. It is present only in 22% of the cases of hyperpotassemia. Not too sensitive but quite specific. This type of wave may be observed in familial short QT syndrome, a new entity recently discovered. Narrow-based, symmetrical, pointy T wave, of increased voltage, in "desert tent". QRS complex widens and p wave is flattened or disappears.

Typical hyperkalemic T wave



Figure. 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 LVH, subendocardial ischemia, schizophrenia and stroke. Once again, T wave of hyperpotassemia. It is observed when the rate of potassium reaches 5.5 mEq/l. Sensitivity is just 22% of the cases. It may be confused with T wave observed in bradycardias, in LVH of the diastolic type, subendocardial ischemia, schizophrenia and stroke.

Hyperpotassemia associated to hypocalcemia



Figure x. Characteristics of repolarization in uremia: prolonged ST (hypocalcemia) followed by T wave of great voltage (hyperpotassemia).



Here is the ECG of a patient with a history of type I diabetes who presented with nausea and vomiting. EMS reported that the patient was in sinus tachycardia with a rate of 300. ECG Courtesy of Dr. Morgan Wilbanks, University of Wisconsin. *Notice the very tall, pointy T waves, which have a narrow base and are extremely symmetric. This patient was found to be in severe DKA, with a pH of 7.17 and a potassium of 7.1 mmol/L. The tall T-waves were likely being mistaken for QRS complexes and cardiac monitor misread the rate to be 300, when in fact it is about 150.*

Conclusion

The earliest changes on an ECG after acute vessel occlusion are hyperacute T-waves and patients with coronary vessel occlusion can present with only subtle ECG changes. The ECG is an important tool, but should not always be used in isolation (unless clearly diagnostic). The **clinical picture and adjunctive information should always be considered**. If suspicious for vessel occlusion by the appearance of the ECG, it is important to look carefully for reciprocal changes and **get serial ECGs every 15 minutes** (not in an hour or two) and look for evolving changes because **ACS is a dynamic process**. Other adjuncts that may help diagnose ACS in patients with subtle ECG changes include continuous ST-segment monitoring, echocardiogram to evaluate for wall motion abnormality, and cardiac biomarkers.

Take home points

- Hyperacute T-waves are often the first manifestation of complete vessel occlusion; they are wide, bulky and prominent.
- Hyperacute T-waves are not necessarily tall, and small T-waves can still be hyperacute when paired with a low-amplitude QRS complex.
- De Winter T-waves represent LAD occlusion (a STEMI equivalent) requiring immediate revascularization.
- Previously inverted T-waves can appear normal and upright in the setting of acute vessel occlusion. This is known as pseudonormalization.
- The tall T-waves associated with hyperkalemia are **sharp**, **pointy**, **symmetric**, **and have a narrow base**.
- When in doubt, get serial ECGs (every 15 minutes) and use adjunctive information.