Occlusion MI and the revival of hyperacute T waves and others T wave abnormalities

Occlusion MI and the revival of hyperacute T-waves

T waves represent ventricular repolarization. They are normally upright (except for aVR, V_1 and sometimes III), with a concave and asymmetric morphology (gradual rise and quick descent), and a height proportional to their QRS complex (with T amplitude usually <5mm in limb leads and <10mm in precordial leads).

There are many causes of tall T-waves [**Somers MP, Brady WJ, Perron AD, et al. The prominent T wave: electrocardiographic differential diagnosis. Am J Emerg Med 2002 May;20(3):243-51**] but the most common are:

I. Normal variant, including early repolarization pattern

II. Secondary to abnormal depolarization

A. LBBB: deep/wide anterior S waves followed by ST elevation and tall T wave
B. LVH: deep/narrow anterior S waves followed by ST elevation and tall T wave

III. Primary repolarization abnormality

A. *Hyperkalemia:* "peaked T waves" are diffuse, look pinched (narrow base and sharp symmetric peak), and are associated with other signs of hyperkalemia (eg bradycardia, junctional rhythm, long PR, wide QRS)

B. *Occlusion MI:* "hyperacute T waves" are regional, look inflated (broad base, rounded peak, and large

relative to QRS), and are associated with other signs of occlusion MI (loss of R wave, new Q wave, ST elevation, reciprocal ST depression)

The hallmarks of hyperacute T waves have been described for a century:

1. **First sign of occlusion**: in 1918, the first experimental study on ECGs in ACO noted that, "immediately following the ligation the T-waves became more prominent…in some instances it exceeded the height of the R wave.[**Smith F. The ligation of coronary arteries with electrocardiographic study. 1918 Ann Noninvas Electrocardiol 2004 Jan;9(1):80-93**]
2. **Precede ST elevation, but can also be associated with ST depression**: In 1947, a series of case reports noted that, "high T-waves were the outstanding feature in the earliest stage of AMI, when elevation of S-T and significant changes of QRS were absent," including, "in one instance huge upright T waves were associated with marked depression of S-T.**[Dressler W, Roesler H. High T waves in the earliest stage of myocardial infarction. Am Heart J 1947 Nov;34(5):627-45]**
3. **Defined relative to QRS and associated with reciprocal change**: in 1982 the term "hyperacute T waves" was defined as a "relative increase in T-wave positivity due to transmural myocardial ischemia"

3. and it was noted that “the T waves may be only relatively, and not absolutely increased in amplitude. Occasionally with acute infarction, the reciprocal ST segment depressions may actually exceed the amplitude of primary, hyperacute changes. Only careful inspection will disclose the primary locus of injury in such cases.”[**Ary Goldberger. Hyperacute T waves revisited. Am Heart J 1982;104(4): 888-890**]

Take home points on hyperacute T-waves and occlusion MI

1. Tall T-waves can be a normal variants (including early repolarization), secondary to abnormal depolarization (LBBB, LVH), or a primary repolarization abnormality (hyperkalemia, occlusion MI)
2. “Peaked T-waves” from hyperkalemia are diffuse, look pinched (narrow base, sharp peak), and are associated with other signs of hyperkalemia (brady, junctional rhythm, long PR, wide QRS)
3. “Hyperacute T-waves” from OMI are regional, look inflated (wide base, round peak, large relative to the QRS), and are associated with other signs of occlusion like acute Q/loss of R waves, STE or reciprocal STD
4. Variants of hyperacute T-waves include: upsloping STD + hyperacute T wave (deWinter T wave), and anterior QS waves with hyperacute T-waves (T/QRS>0.36, which differentiates from LV aneurysm)

The early STEMI guidelines incorporated this knowledge, explaining that “in the very early phase of acute infarction, giant, hyperacute T-waves may be present with no ST-segment elevation,” and that “it is appropriate to administer thrombolytic therapy. Thus, it should be clear that certain cases require experienced interpretation of the ECG before withholding reperfusion therapy.”[**Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). JACC Nov 1 1996;28(5):1328-428**] But as the STEMI paradigm took hold it, its exclusive focus on ST elevation criteria displaced other findings of acute coronary occlusion including hyperacute T-waves. The only mention in the current American STEMI guidelines is that “rarely, hyperacute T-wave changes may be observed in the very early phase of STEMI, before the development of ST elevation”[**O’Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. JACC January 29, 2013;61(4):378-140**]. This implies hyperacute T-waves are both infrequent and inconsequential,

and that they'll rapidly and inevitably give way to diagnostic ST elevation. But STEMI criteria lacks sensitivity for ACO, a dynamic process that can fluctuate between total occlusion and partial reperfusion. In this context, hyperacute T-waves can help identify subtle occlusions that don't meet STEMI criteria, including those with dynamic occlusion. As a recent review explained, "STE might be preceded by (or if immediately after reperfusion and resolution of chest pain, followed by) large 'hyperacute' T-waves, which might thus be a subtle finding of early acute thrombotic occlusion."[**Miranda DF, Lobo AS, Walsh B, et al. New insights into the use of the 12-lead electrocardiogram for diagnosing acute myocardial infarction in the emergency department. Can J Cardiol 2018;34:132-145**].

The newer European STEMI guidelines consider hyperacute T waves in the absence of ST elevation as an indication for PCI [**Ibanez M, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39:119-177**], and deWinter has revived

the pattern of upsloping ST depression and hyperacute T-waves first described by Dressler in 1947. [**Littman L. The Dressler – de Winter sign of acute proximal LAD occlusion. J of Electrocardiol 2018;51:138-139**]

Hyperacute T-waves and reciprocal STD can help identify subtle inferior or lateral OMI that do not meet STEMI criteria, differentiate LAD occlusion from early repolarization, and differentiate old LV aneurysm (T/QRS<0.36) from anterior QS waves with new OMI (T/QRS>0.36).[**Smith SW. T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction. Am J of Emerg Med 2005;23:279-287**] Signs of OMI can double the sensitivity of STEMI criteria for ACO, with preserved specificity, and can identify STEMI(-)OMI patients who experience preventable delays to reperfusion. [**Meyers HP, Bracey A, Lee D, et al. Accuracy of OMI ECG findings versus STEMI criteria for diagnosis of acute coronary occlusion myocardial infarction. IJC Heart & Vasc 2021 Apr;33:100767**]

T wave abnormalities

1. Peaked T waves: Tall, narrow, symmetrically peaked T-waves are characteristically seen in hyperkalaemia
2. Hyperacute T waves: Broad, asymmetrically peaked or '*hyperacute*' T-waves (HATW) are seen in the early stages of ST-elevation MI (STEMI), and often precede the appearance of ST elevation and Q waves. Particular attention should be paid to their size in relation to the preceding QRS complex, as HATW may appear 'normal' in size if the preceding QRS complex is of a small amplitude.
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4. Biphasic T waves: Biphasic T waves in leads V2-V3 in anterior chest derivations of ECG (24% of WS), and symmetrical and deeply inverted T wave in same derivations (76% of Wellens Syndrom), are the two types into which WS are divided. The timely recognition of WS is important both for diagnosis and treatment
5. 'Camel Hump' T waves. ***Camel hump'*** T waves is a term used by Amal Mattu to describe T-waves that have a double

peak. There are two causes for camel hump T waves:**Prominent U waves** fused to the end of the T wave, as seen in severe hypokalaemia **Hidden P waves** embedded in the T wave, as seen in sinus tachycardia and various types of heart block

6. **Inverted T waves are seen in the following conditions:** Normal finding in children(Inverted T-waves in the right precordial leads (V1-3) are a normal finding in children, 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 ischaemia or infarction occur in contiguous leads based on the anatomical location of the area of ischaemia/infarction: Inferior = II, III, aVF, Lateral = I, aVL, V5-6 Anterior = V2-6 (including Wellens Syndrome), ***Dynamic*** T-wave inversions are seen with acute myocardial ischaemia and ***Fixed*** T-wave inversions are seen following infarction, usually in association with pathological Q waves, In bundle branch block, T-wave inversion is an expected finding, even in the absence of ischaemia: *Appropriate discordance* refers to the fact

6. that abnormal depolarization (such as in bundle branch block) should be followed by abnormal repolarization, which appears discordant to the preceding QRS complex in the form of ST-depression and T-wave inversion, Left bundle branch block produces T-wave inversion in the lateral leads I, aVL
7. Inverted T wave in V5-6 Ventricular hypertrophy ('strain' patterns), Left ventricular hypertrophy (LVH) 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 bundle branch block produces T-wave inversion in the right precordial leads V1-3, 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, Hypertrophic cardiomyopathy is associated with deep T wave inversions in all the precordial lead and Raised intracranial pressure **(ICP)** Events causing a sudden rise in intracranial pressure (e.g. subarachnoid haemorrhage) produce widespread deep T-wave inversions with a bizarre morphology