

Posterior reperfusion T-waves: Wellens' syndrome of the posterior wall

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ABSTRACT

Background Reperfusion after coronary occlusion (myocardial infarction, MI), as in Wellens' syndrome, is often represented on ECG as T-wave inversion in the leads overlying the affected myocardial wall(s). As an extension of this logic, reperfusion of the posterior wall should manifest on right precordial leads (which are opposite the posterior wall) as enlarged T-waves.

Objective We sought to determine whether T-wave amplitude (TWa) in leads V2 and V3 after reperfusion in posterior MI (PMI) is greater than in patients without PMI.

Methods Review of ECGs from patients with ST elevation MI of the left circumflex or right coronary artery with post-procedure thrombolysis in MI (TIMI) flow >0 between 2007 and 2009. Blinded experts reviewed admission ECGs to determine the presence of PMI and measure TWa before and after reperfusion. Maximum TWa in V2 and V3 and the difference between maximum and admission V2 and V3 TWa were compared between those with and without PMI.

Results Of 72 patients, 48 had PMI. Values expressed are medians and IQRs. Maximum TWa after reperfusion was greater in PMI than in non-PMI in V2 (5.00 mm (3.5 to 8.25) vs 3.9 mm (2.75 to 5.5), $p=0.04$), but not in V3 (4.0 mm (2 to 5.5) vs 3.0 mm (1.75 to 4), $p=0.09$). The increase in TWa in V2 and V3 after reperfusion was greater in PMI compared with non-PMI: (V2, 3.4 mm (2 to 5.25) vs 1.25 mm (−0.25 to 2), $p=0.0005$; V3, 2 mm (−0.5 to 3.25) vs 0.25 mm (−1 to 1.75), $p=0.03$).

Conclusions Reperfusion of the posterior wall results in higher right precordial TWa, and an even greater increase in TWa, as measured in leads V2 and V3. This observation has important implications for emergency physicians to accurately identify recent posterior infarction in patients who may be symptom free on presentation but at risk of reocclusion.

INTRODUCTION

Wellens' syndrome was first described in 1982 as T-wave inversion in the right precordial leads in patients with unstable angina who were symptom free (figure 1). These patients were found to have critical stenosis of the left anterior descending (LAD) coronary artery and were at high risk of subsequent anterior wall myocardial infarction (MI).¹ This T-wave inversion, which also occurs in inferior and lateral MI,^{2,3} is now known to represent spontaneous reperfusion and an open artery, but one which has active thrombus and is at risk for reocclusion.² Because of this risk, emergency physicians must recognise the T-waves that accompany spontaneous reperfusion in order to provide early aggressive treatment.

The T-wave evolution associated with reperfusion of MI of the posterior wall has not been previously described. For echocardiographic classification of Q-wave MI, the term posterior MI (PMI) has been replaced with 'basal inferior'.^{4,5} This new terminology fails to differentiate the ECG findings of acute MI, (ST segment depression in right precordial leads) from those which manifest ST elevation in inferior (II, III and aVF) or lateral (V5 and V6) leads. Therefore, for the purposes of this study, we continue to use the term 'PMI' for MI which manifests right precordial ST depression, with or without concomitant inferior or lateral ST elevation, even if it is in echocardiographic 'inferior basal' or 'inferior lateral' walls. Because there are no leads overlying this 'posterior' myocardium on the standard 12-lead ECG, posterior wall T-wave inversion cannot be recorded. We had observed anecdotally that, after posterior wall reperfusion, the right precordial leads V1–V3 record an increase in T-wave amplitude (TWa); therefore, we hypothesised that posterior T-wave 'inversion' registers as additive positive voltage (upright) in the ('anterior') right precordial leads. Thus, we sought to study whether TWa in leads V2 and V3 after cardiac catheterisation laboratory reperfusion in PMI is greater than without PMI. If so, it would aid in the recognition of posterior 'Wellens' syndrome and, similar to the anterior Wellens' waves, serve to alert the clinician of a high-risk coronary lesion that has spontaneously reperused, which would prompt aggressive treatment and consideration of cardiac intervention earlier in the ED course.

METHODS

We conducted a retrospective observational analysis in a single academic county hospital from January 2007 to December 2009. This study was approved by the local institutional review board. Patients were included if: (1) they had ST elevation MI (STEMI), as coded for the National Cardiovascular Data Registry (determined from clinical, ECG and angiographic data); (2) the infarct-related artery was the left circumflex or right coronary artery; and (3) if that artery underwent intervention with post-procedure thrombolysis in MI (TIMI) flow ≥ 1 . Those with bundle-branch block or paced rhythm on any ECG were excluded, as were patients who did not have both a pre-reperfusion and post-reperfusion ECG. The goal of this study was to describe an ECG finding that may occur in spontaneous reperfusion of a PMI. Because spontaneous reperfusion of a PMI can be difficult to detect on clinical grounds alone (without this ECG finding), we examined ECGs before and after known reperfusion from percutaneous coronary intervention (PCI).



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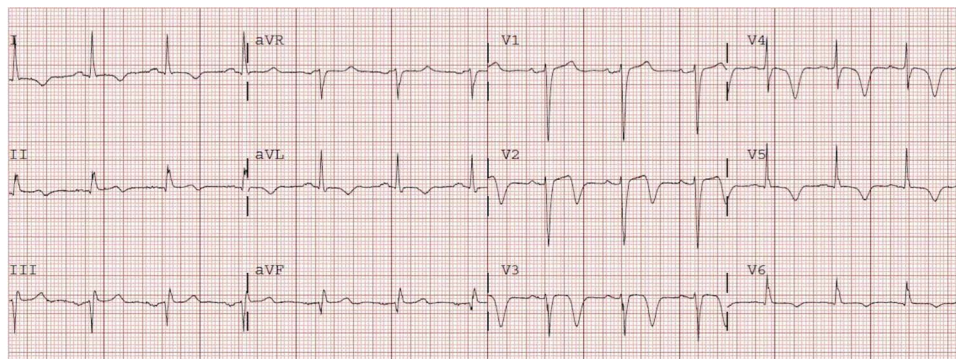


Figure 1 An example of Wellens waves, pattern B.

A blinded expert reviewed all admission ECGs (pre-perfusion) to determine if acute PMI was present. Presence of right precordial ST depression, maximal in leads V2 and/or V3, not explained by QRS abnormalities, was considered diagnostic of PMI.⁶ A second trained blinded reviewer examined ECGs for the first 72 hours of hospitalisation. The positive TWa in leads V2 and V3 before and after reperfusion, measured in millimetres relative to the PQ junction, was recorded. Although the Recommendations for Standardization of the ECG recommend measuring ST elevation at the J-point relative to the PQ junction, there are no standards for measuring TWa; therefore, as is recommended with ST elevation, we measured TWa relative to the PQ junction.⁷

Maximum TWa in V2 and V3 after reperfusion and the difference between the maximum and admission V2 and V3 TWa were compared between those with and without acute PMI. Median and IQR values are presented. Non-parametric testing was used because the data were not normally distributed. Maximum TWa values were compared using the Mann-Whitney U test; the difference between maximum and admission TWa were compared using repeated measures analysis of variance. Test performance characteristics, including sensitivity, specificity and positive and negative likelihood ratios (LR), are presented for thresholds that attempt to maximise specificity. Statistical evaluations were performed using STATA V.12 (Stata, College Station, Texas, USA).

RESULTS

Eighty-five patients had an STEMI involving the left circumflex or right coronary artery during the study timeframe. Thirteen patients were excluded (five each had right and left bundle-branch block; three had a missing ECG), leaving 72 patients for analysis. Forty-eight (66%) had PMI; 12 (17%) had isolated PMI. See [table 1](#) for baseline characteristics.

Maximal TWa in V2 after reperfusion ($p=0.04$) and the increase in TWa after reperfusion in leads V2 ($p=0.0005$) and V3 ($p=0.03$) were greater in patients with PMI compared with those without PMI. Though maximal TWa in V3 after reperfusion was greater in patients with PMI compared with those without PMI, this difference did not reach statistical significance ($p=0.09$). See [table 2](#) for ECG measurements.

Post-reperfusion V2 TWa ≥ 7 mm was 92% specific (95% CI 73% to 99%) for PMI, though only 32% sensitive (95% CI 20% to 47%). A total of 16/48 (33%) patients with PMI, and 2/24 (8%) patients without PMI, had post-reperfusion TWa in V2 ≥ 7 mm. Positive and negative LR at this cut-off were 3.8 and 0.7, respectively. An increase in TWa in V2 ≥ 3.25 mm between admission and post-reperfusion ECG was 96% specific

Table 1 Baseline characteristics of patients presenting with STEMI involving the right or left circumflex coronary arteries

	Meeting criteria for posterior STEMI n=48	Not meeting criteria for posterior STEMI n=24
Age, mean (SD)	57 (10.8)	58.5 (16.2)
Male gender (%)	35 (73)	14 (58)
Infarct-related artery		
Left circumflex (%)	13 (27)	4 (17)
Right coronary (%)	35 (73)	20 (83)
Pre-PCI TIMI flow of zero (%)	15 (31)	7 (29)

PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction.

Table 2 ECG measurements

	Posterior MI n=48	Non-posterior MI n=24	p Value
Admission lead V2			
ST depression	1 (0.5, 2)	0 (-0.75, 0)	
TWa	1.5 (0.75, 2.5)	2.75 (2, 4.25)	
Admission lead V3			
ST depression	1 (0, 1.5)	0 (-1, 0)	
TWa	2 (1, 3.5)	3 (2, 4)	
Post-reperfusion			
V2 TWa	5 (3.5, 8.25)	3.9 (2.75, 5.5)	0.04
V3 TWa	4 (2, 5.5)	3 (1.75, 4)	0.09
Increase in V2 TWa after reperfusion	3.4 (2, 5.25)	1.25 (-0.25, 2)	0.0005
Increase in V3 TWa after reperfusion	2 (-0.5, 3.25)	0.25 (-1, 1.75)	0.03

All values are medians and IQRs. All units are millimetres, except QTc (ms). MI, myocardial infarction; TWa, T-wave amplitude.

(95% CI 79% to 100%) and 50% sensitive (95% CI 36% to 65%) for PMI, respectively. The positive LR at this cut-off was favourable at 12; negative LR was 0.5.

DISCUSSION

ECG findings in PMI were first described in 1964 by Perloff as increased R wave amplitude, R/S ratios >1 and an increase in TWa in leads V1–V3.⁸ In 1977, Lewis described the hyperacute phase of PMI, with ECG findings of ST depression and wide and deep T waves in leads V1–V4, while in the ‘fully evolved

Figure 2 Prehospital ECG with inferoposterior ST elevation myocardial infarction.

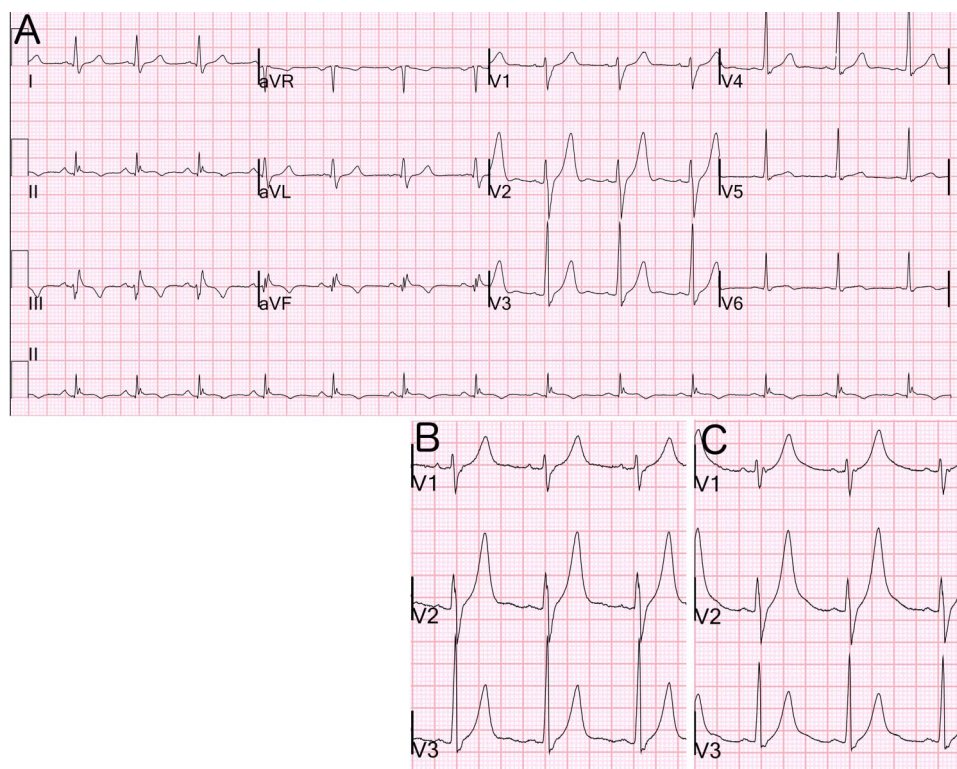
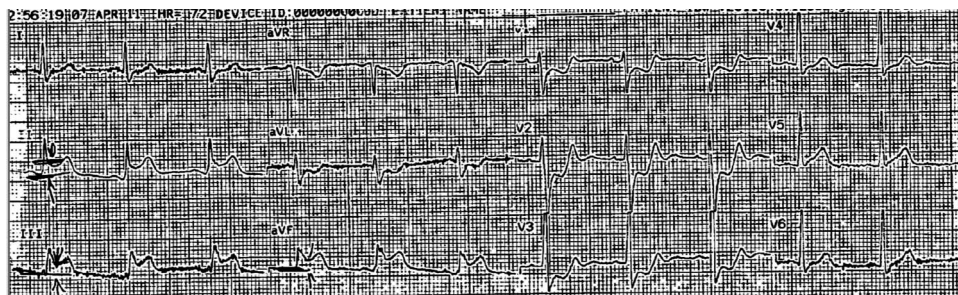


Figure 3 These are post-reperfusion ECGs obtained for the patient whose prehospital ECG is shown in [figure 1](#). (A) 174 min after arrival: at coronary angiography, the left circumflex coronary artery was found to be occluded and percutaneous coronary intervention was performed. Note the strikingly tall T-waves in V2 and V3. (B) V1–V3 406 min after arrival. (C) V1–V3 36 hours after arrival. Both (B and C) demonstrate a persistent increase in T-wave amplitude in V2 and V3, due to reperfusion of the posterior wall.

phase' tall R waves and tall and widened T waves were present in the same leads.⁹ What Perloff and Lewis noted as a phase of PMI, we believe was actually a finding of spontaneous reperfusion of the posterior wall manifested as an increase in TWa in the right precordial leads, analogous to a mirror image of the well-known ECG findings of Wellens' syndrome of anterior wall reperfusion.

To the best of our knowledge, the ECG findings of posterior reperfusion have not been described previously. This study demonstrates that patients with PMI on arrival, compared with patients without PMI, have greater post-reperfusion maximal TWa in V2 and a greater increase in TWa in leads V2 and V3 after reperfusion. [Figures 2](#) and [3](#) clearly demonstrate these findings. Although the patient's baseline ECG is not available, the TWa is so large as to be obviously abnormal.

Reperfusion (whether spontaneous or after PCI) of the infarct-related artery supplying a myocardial territory results in T-wave inversion in leads that formerly had ST elevation during occlusion.^{10 11} This is the physiological substrate for Wellens' syndrome of the anterior wall.² Unless posterior leads are

recorded, such T-wave inversion will not manifest on the standard ECG. However, such a posterior directed T-wave vector will record as a positive deflection on anterior (right precordial, V2) leads, and add to the already positive vector of upright T-waves from anterior wall repolarisation, leading to increased voltage in these leads. Infarct with subsequent reperfusion, which is not directed 180° opposite the right precordial leads because the infarct is in a more inferior or lateral location, will manifest less increase in V2 voltage and presumably be less immediately recognisable.

This finding is of particular importance to emergency physicians who routinely evaluate patients for acute coronary syndrome. TWa in an unselected population of ED patients with chest pain is variable and dependent on many factors, especially the presence of early repolarisation, which is known to manifest large T-waves. Thus, the sensitivity and specificity of TWa in such an unselected population is difficult to establish. However, if the T-wave in lead V2 appears abnormally tall, upright and broad in a patient with resolved ischaemic chest pain, one should consider that this may be a

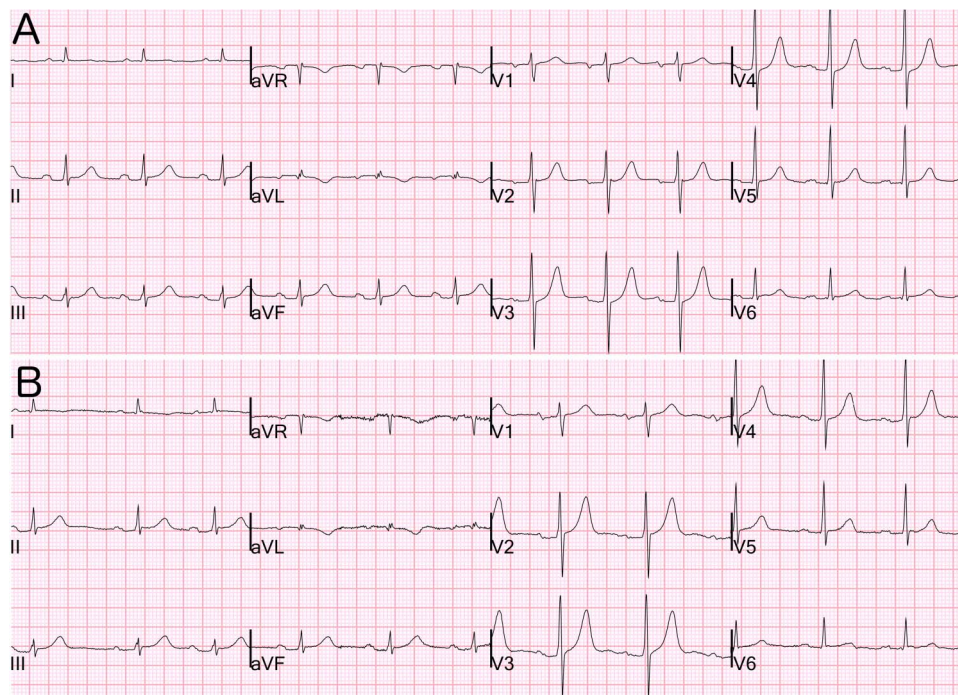


Figure 4 ECGs of a patient with proven reperfused isolated posterior myocardial infarction during pain and then 2 hours later, after resolution of pain. (A) Obtained while the patient had chest pain. The ST segments in right precordial leads are isoelectric and there is no definite evidence of acute infarction. (B) Two hours after resolution of pain. Relative to the presenting ECG, the ST segments are relatively slightly elevated and the T-wave has enlarged significantly. This was not recognised by the treating physicians.

spontaneously reperfused PMI. It should prompt scrutiny for ST depression on any previous ECG which may have been recorded during pain (such as on a prehospital ECG) and scrutiny for corresponding reperfusion T-waves in inferior and/or lateral leads.

These T-waves can mimic hyperacute T-waves of anterior MI. Hyperacute T-waves in the right precordial leads due to LAD occlusion are generally present shortly after the onset of chest discomfort, resolve after reperfusion of the LAD and continue to diminish in size after reperfusion. In contrast, posterior reperfusion T-waves will appear *after* the chest pain abates, and *increase* in size as time passes after resolution of chest discomfort (figure 4). Thus, the finding of a large upright T-wave in lead V2 or an increase in V2 TWa after resolution of pain may prompt more aggressive early diagnostic investigation and therapeutic management, vigilant monitoring and likely early invasive therapy. Furthermore, it can be conceived that, without the knowledge of posterior reperfusion T-waves, a clinician could be falsely reassured by absence of an anterior wall motion abnormality on a bedside echocardiogram, whereas the wall motion abnormality would in fact be expected to involve the posterior wall.

Because PMI can present in isolation,¹² and is associated with increased in-hospital complications, dysrhythmias, worse left ventricular ejection fraction and larger infarct size,^{12 13} prompt recognition and early treatment are important. Moreover, these patients who may have the pathophysiology of ischaemia and spontaneous reperfusion may be at risk of reocclusion even though they may be deceptively free of symptoms on presentation to the ED.

LIMITATIONS

This study occurred at a single institution, involving a small patient population. This somewhat limits the generalisability of

these results though ECG findings should vary less between institutions than other patient treatments and outcomes. This study examined patients between 2007 and 2009; although the diagnostic criteria for PMI have not changed, angiographic assessment of lesion characteristics has evolved since then, including intravascular ultrasound and fractional flow reserve assessment.

CONCLUSION

This investigation demonstrated the ECG findings of posterior wall reperfusion as a significant increase in T wave amplitude in leads V2 and V3. Maximal T wave amplitude was greater in V2 after reperfusion in patients with PMI. The increase in TWa in leads V2 and V3 after reperfusion was greater in PMI compared with non-PMI. It is well known that ECG findings of Wellens' syndrome, a state of spontaneous reperfusion of a previously occluded LAD, are important for rapid recognition of high-risk LAD acute coronary syndrome (ACS). We believe we have described an analogous ECG finding of PMI.

Contributors BED, GRS and SWS conceived the investigation. BED performed chart review. BED and SWS performed ECG measurements. GRS performed echocardiogram review. BED drafted the initial manuscript, and all authors contributed substantially to the final draft.

Competing interests None declared.

Ethics approval Minneapolis Medical Research Foundation Human Subjects Committee.

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