Hyperacute T Wave in the Early Diagnosis of Acute Myocardial Infarction

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Study objective: The diagnostic performance of T-wave amplitudes for the detection of myocardial infarction is largely unknown. We aimed to address this knowledge gap.

Methods: T-wave amplitudes were automatically measured in 12-lead ECGs of patients presenting with acute chest discomfort to the emergency department within a prospective diagnostic multicenter study. The final diagnosis was centrally adjudicated by 2 independent cardiologists. Patients with left ventricular hypertrophy, complete left bundle branch block, or paced ventricular depolarization were excluded. The performance for lead-specific 95th- percentile thresholds were reported as likelihood ratios (lr), specificity, and sensitivity.

Results: Myocardial infarction was the final diagnosis in 445 (18%) of 2457 patients. In most leads, T-wave amplitudes tended to be greater in patients without myocardial infarction than those with myocardial infarction, and T-wave amplitude exceeding the 95th percentile had positive and negative lr close to 1 or with confidence intervals (CIs) crossing 1. The exceptions were leads III, aVR, and V1, which had positive lrs of 3.8 (95% CI, 2.7 to 5.3), 4.3 (95% CI, 3.1 to 6.0) and 2.0 (95% CI, 1.4 to 2.9), respectively. These leads normally have inverted T waves, so T-wave amplitude exceeding the 95th percentile reflects upright rather than increased-amplitude hyperacute T waves.

Conclusion: Hyperacute T waves, when defined as increased T-wave amplitude exceeding the 95th percentile, did not provide useful information in diagnosing myocardial infarction in this sample. [Ann Emerg Med. 2022;■:1-9.]

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INTRODUCTION

Acute chest discomfort accounts for approximately 10% of all emergency department presentations. Rapid identification of myocardial infarction as a life-threatening disorder is important for the early initiation of highly effective evidence-based therapy, including early/immediate revascularization of the affected coronary artery.[1-4]

Substantial improvement in outcomes has been achieved in patients presenting with ST-segment elevation myocardial infarction (STEMI), who usually can be rapidly identified with a 12-lead ECG.[1-4] In contrast, improvements in patients presenting with non–ST-segment elevation myocardial infarction (NSTEMI) have been more subtle.[1-4] This difference may, at least in part, be explained by the substantial delay from ED presentation to diagnosis and then coronary revascularization in patients with NSTEMI.[1-4] Accordingly, the identification of additional ECG signatures associated with a high positive likelihood ratio (lr+) for acute myocardial infarction, which justifies early coronary angiography, is a major unmet clinical need.[5-8]

Based largely on experimental and anecdotal evidence, symmetrical increased-amplitude "hyperacute" T waves have been suggested as early markers for myocardial infarction and are recommended for clinical use in current clinical practice guidelines.[3,4,9-13] However, up to now, the diagnostic performance of hyperacute T waves has not been adequately scrutinized in a large diagnostic study.[10-20]

To address this unmet need, we performed a post hoc analysis of a large international multicenter diagnostic study to evaluate the diagnostic performance and clinical utility of

Editor's Capsule Summary

What is already known on this topic

Symmetrical increased-amplitude "hyperacute" T waves may be early ECG markers for myocardial infarction, but defining such is imprecise.

What question this study addressed

What is the diagnostic accuracy for myocardial infarction of a hyperacute T waves defined as amplitude above the study population's lead-specific 95th percentile threshold?

What this study adds to our knowledge

This post hoc analysis of data from a multicenter study found this diagnostic threshold did not provide useful diagnostic information other than in leads that normally have inverted T waves.

How this is relevant to clinical practice

Hyperacute T-wave definitions and reliable utility remain impaired when trying to diagnose early myocardial infarction.

hyperacute T waves in patients presenting to the ED with acute chest discomfort.

Goals of This Investigation

The primary objective was to analyze the diagnostic performance of hyperacute T waves for the diagnosis of myocardial infarction according to the study population's lead-specific 95th-percentile T-wave amplitude thresholds.

MATERIALS AND METHODS

Study Design and Population

This is a post hoc analysis of the prospective international multicenter Advantageous Predictors of Acute Coronary Syndrome Evaluation (ClinicalTrials.gov registry, number NCT00470587) study enrolling adult patients presenting to the ED with acute chest discomfort and aiming to improve the early diagnosis of myocardial infarction.[21-24]

Patients recruited in centers that recorded digital 12-lead ECG data at ED presentation allowing automated quantification of T waves, including large tertiary and community hospitals, were eligible for this analysis.

Selection of Participants

Patients receiving hemodialysis for chronic kidney failure were excluded; otherwise, patients were included regardless of kidney function. Patients with an unknown final diagnosis after the final adjudication and with at least one elevated high-sensitivity cardiac troponin (hs-cTn) concentration possibly indicating myocardial infarction, as well as patients with a time to chest pain onset and peak more than 12 hours, were excluded.

In addition, we excluded patients with pacemaker rhythm, left bundle branch block, or left ventricular hypertrophy defined as a Sokolow-Lyon Index $S_{V1}+RMax._{V5/V6}>3.5$ mV or Cornell voltage $R_{aVL}+S_{V3}>2.8$ mV (♂) or >2.0 mV (♀) from this analysis because these entities can be associated with altered repolarization and varied T-wave morphology.[25] Left ventricular hypertrophy and left bundle branch block were detected automatically using the ETM V01.12.09.00 ECG analysis software (Schiller AG) and then verified by 2 independent physicians. Mismatches were adjudicated by 2 additional physicians.

Role of the Funding Source

The study was conducted according to the principles of the Declaration of Helsinki and approved by the local ethics committees. Written informed consent was obtained from all patients. The authors designed the study, gathered, and analyzed the data according to the STARD guidelines, vouched for the data and analysis, wrote the paper, and decided to submit it for publication. The authors are solely responsible for the design and conduct of this study and its final contents. Patients or the public were not involved in the design, conduct, reporting, or dissemination plans of our research.

Adjudicated Final Diagnosis

Adjudication of the final diagnosis was performed centrally by 2 independent board-certified cardiologists or cardiology fellows at the core laboratory (University Hospital Basel) applying the universal definition of myocardial infarction using 2 sets of data: first, all available medical records obtained during clinical care including history, physical examination, results of laboratory testing, including serial clinical (hs)-cTn levels, radiologic testing, ECG, echocardiography, cardiac exercise test, lesion severity and morphology in coronary angiography–pertaining to the patient from the time of ED presentation to 90-day follow-up; second, study-specific assessments including detailed chest pain characteristics using 34 predefined criteria, serial hs-cTnT blood concentrations obtained from study samples, and clinical follow-up by telephone and/or mail.[21,22,24,26,27] In situations of disagreement about the diagnosis, cases were reviewed and adjudicated in conjunction with a third cardiologist.

Myocardial infarction was defined, and (hs-)cTn was interpreted as recommended in current guidelines.[4,21,22,24] In brief, myocardial infarction was diagnosed when, in combination with symptoms of myocardial infarction, a significant rise or fall of hs-cTn with at least one concentration above the 99th-percentile was measured.

The criteria used to define a rise or fall in hs-cTnT are described in detail in the Method section of the online material (Appendix E1, available at http://www.annemergmed.com). Myocardial infarction could be diagnosed without evidence of ECG abnormalities. All other patients were classified in the categories of unstable angina, noncardiac chest pain, cardiac but noncoronary disease (eg, tachyarrhythmias, perimyocarditis), and symptoms of unknown origin with normal levels of hs-cTnT.

Recording of Digital ECGs and Automated Analysis

Ten-second 12-lead resting ECGs were acquired during the standard clinical assessment of patients in the ED (AT-110 ECG device, Schiller AG; Page Writer TC30 ECG device, Philips Healthcare). The digital ECG raw data has a sampling frequency of 500 Hz, a resolution of 2.5 uV/bit, and a bandwidth of 0.05 Hz to 150 Hz. The maximum within the beat averaged T-wave amplitudes, as well as the confounder types, were automatically measured using ETM V01.12.09.00 ECG analysis software (Schiller AG). If the T-wave amplitude maximum was negative, it was set to 0. Therefore, T-wave amplitude refers to the maximum positive amplitude within a T-wave (Figure 1). If not otherwise stated, amplitude denotes the maximum amplitude of the respective segment.

Manual Analysis of Standard 12-Lead ECGs

All 12-lead resting ECGs were also interpreted in the ECG core laboratory at the University Hospital Basel by cardiologists blinded to the clinical and biochemical details of the patients. ECG manifestations indicative of myocardial infarction in the absence of ST-segment elevations, ie, ST-segment depression, T-wave inversion, and left bundle branch block were defined as recommended in current guidelines.[4]

Statistical Analysis

Continuous variables are presented as median (interquartile range [IQR]); categorical variables as numbers and percentages. The performance for lead-specific 95th-percentile thresholds (within the study population) was reported as positive likelihood ratios (lr+), negative likelihood ratios (lr−), specificity, and sensitivity. All statistical analyses were performed using R 4.2.1 with packages tableone and DescTools.[28-30] Following additional analysis are part of the online supplement: first, a subgroup analysis was performed for early presenters presenting within 1 hour after chest pain onset to the ED as based on previous experimental studies and case reports, we hypothesized that rule-in performance might be highest in these patients.[9-13] Second, we performed an additional subgroup analysis in patients with an available prior ECG recording within 3 years. This period was chosen to balance the likelihood of having a prior ECG available with the likelihood that it still represented a valid reference for possible acute changes. A possible association between changes in T-wave amplitude in the current ECG versus the last available prior ECG was evaluated.

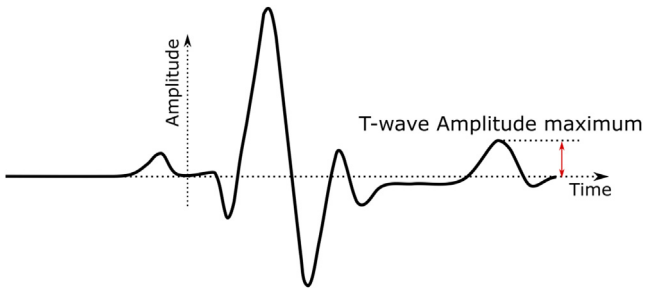

Figure 1. Measurement of T-wave amplitude

RESULTS

Study Cohort and Characteristics of the Patients

From April 2006 to August 2015, 4323 patients were prospectively enrolled. Of these, 2457 patients were eligible for the analysis of ECG characteristics (Figure 2). Table 1 shows the characteristics of the study population. Median (IQR) time from chest pain onset to ED presentation was 5 (IQR [2.5, 12.0]) hours.

Adjudicated Final Diagnosis

Myocardial infarction was the adjudicated final diagnosis in 445 patients (18%), 82 (3.3%) of whom had a STEMI, and 363 (15%) had NSTEMI. The other adjudicated final diagnoses were unstable angina in 238 (10%); cardiac symptoms of an origin other than coronary artery diseases, such as tachyarrhythmia, Tako-Tsubo cardiomyopathy, heart failure, or myocarditis in 309 (13%); noncardiac symptoms in 1375 (56%); and unknown in 90 patients (4%).

T-Wave Characteristics

Table 2 compares the median and IQR for T-wave amplitude between patients with and without myocardial infarction. Patients with myocardial infarction tended to have smaller T-wave amplitudes than patients with other causes of chest pain. The exceptions were leads III, aVR,

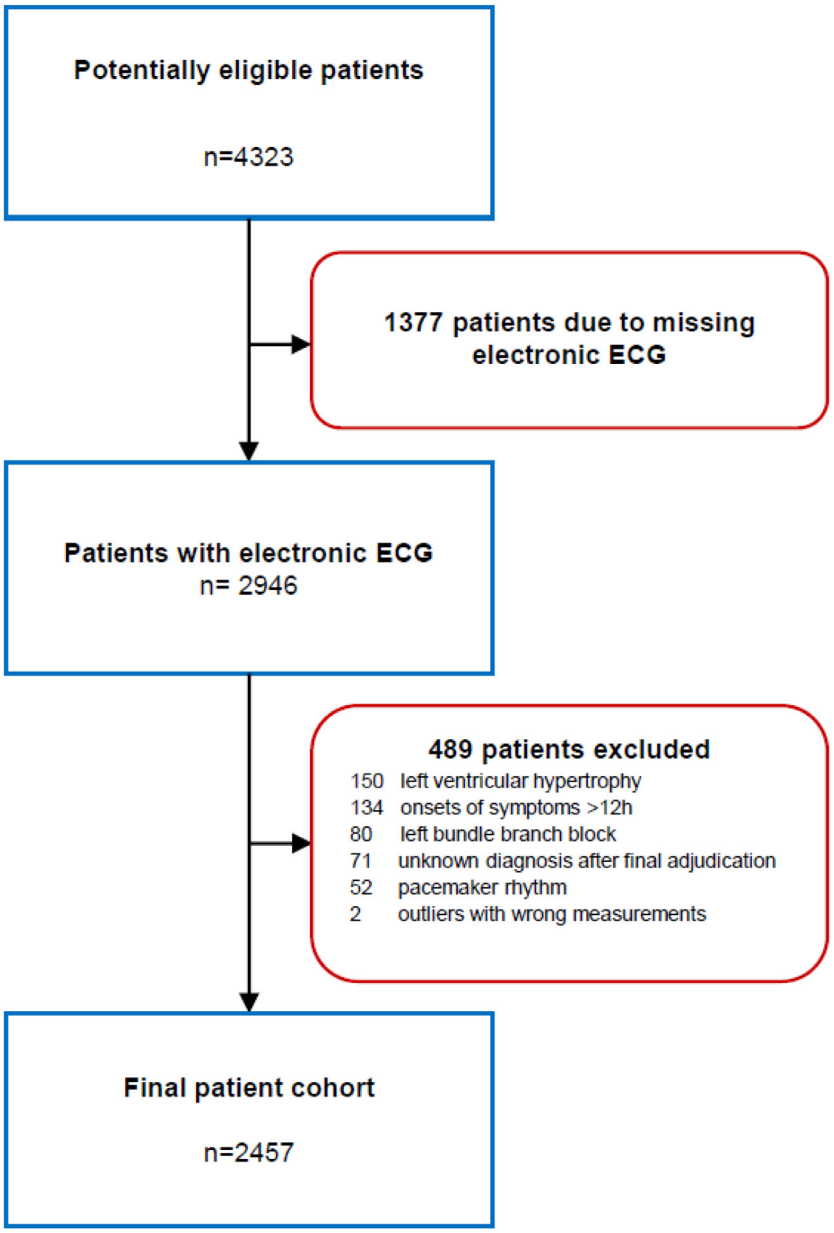

Figure 2. Patient flow

and V1. These leads normally have inverted T-waves, so the greater amplitude in patients with myocardial infarction reflects an upright T-wave that would normally be inverted rather than a hyperacute T-wave. Table 3 shows the proportion of myocardial infarction in each quartile of T-wave amplitude from quartile 1 (lowest amplitude) to quartile 4 (greatest amplitude). In most leads, the proportion with myocardial infarction was greatest in the quartile with the lowest T-wave amplitude. The exceptions were leads III, aVR, aVF and V1, where the proportion with myocardial infarction was greatest in the quartile with the greatest T-wave amplitude. The STEMI patients had significantly higher T-wave amplitudes in leads II, III, and aVF than NSTEMI patients (Table E1, available at http://www.annemergmed.com).

Performance of changes in T-wave amplitude. Table 4 shows for each lead the diagnostic accuracy of a T-wave amplitude exceeding the 95th percentile threshold. Most leads had positive and negative lrs close to 1 or with a confidence interval (CI) overlapping 1, indicating no useful diagnostic information. The exceptions were lead III (lr+ 3.8 [95% CI, 2.7 to 5.3]; lr− 0.90 [95% CI,0.87 to 0.94]); aVF (lr+ 2.0 [95% CI, 1.4 to 2.9]; lr− 0.95 [95% CI, 0.93 to 0.98]); aVR (lr+ 4.3 [95% CI, 3.1 to 6.0]; lr− 0.89 [95% CI, 0.86 to 0.93]); and V1 (lr+ 2.0 [95% CI,1.4 to 2.9]; lr− 0.96 [95%

Table 1. Baseline characteristics of the patient.

	MI (n=445)	No MI (n=2012)	All patients (n=2457)
Age–y	70.0 [59.0, 80.0]	58.0 [46.0, 70.0]	60.0 [48.0, 73.0]
Female sex–no. (%)	98 (22)	658 (33)	756 (31)
Time from chest pain onset to presentation–hours	5.8 [2.5, 13.0]	5.0 [2.5, 12.0]	5.0 [2.5, 12.0]
Presentation within 1 hour after chest pain onset–no. (%)	32 (7)	194 (10)	226 (9)
Vital signs, median (IQR)			
Heart rate–beats/min	76.0 [65.0, 87.0]	77.0 [67.0, 89.0]	76.0 [66.0, 89.0]
Systolic blood pressure–mmHg	142.0 [128.0, 161.0]	140.0 [126.0, 155.0]	140.0 [126.0, 156.0]
Diastolic blood pressure–mmHg	81.0 [71.0, 91.8]	82.0 [72.0, 91.0]	82.0 [72.0, 91.0]
Oxygen saturation–%	98.0 [97.0, 99.0]	98.0 [97.0, 100.0]	98.0 [97.0, 100.0]
Risk factors–no. (%)			
Hypertension	338 (76)	1112 (55)	1450 (59)
Hypercholesterolemia	275 (62)	865 (43)	1140 (46)
Diabetes	110 (25)	288 (14)	398 (16)
Current smoking	112 (25)	554 (28)	666 (27)
History of smoking	191 (43)	741 (37)	932 (38)
History–no. (%)			
Coronary artery disease	203 (46)	618 (31)	821 (33)
Previous myocardial infarction	156 (35)	438 (22)	594 (24)
Previous stroke	30 (7)	94 (5)	124 (5)
Positive family history	184 (44)	828 (43)	1012 (43)
Previous pulmonary embolism	18 (4)	52 (3)	70 (3)
Peripheral artery disease	47 (11)	83 (4)	130 (5)
Body mass index–kg/m^2	26.3 [24.1, 28.9]	26.3 [23.6, 29.4]	26.3 [23.8, 29.4]
Chronic medication at presentation–no. (%)			
Aspirin	215 (48)	671 (33)	886 (36)
Vitamin K antagonist	45 (10)	175 (9)	220 (9)
β-Blocker	176 (40)	635 (32)	811 (33)
Calcium antagonist	73 (16)	278 (14)	351 (14)
Nitrate	66 (15)	147 (7)	213 (9)
Statin	187 (42)	640 (32)	827 (34)
ACEI/ARB	221 (50)	693 (34)	914 (37)
Biochemistry			
Creatinine clearance–mL/min/m^2	77.1 [60.8, 96.8]	88.6 [73.9, 103.6]	86.9 [71.3, 102.5]

Numbers are presented as median (IQR), or numbers (%)
ACEI, Angiotensin-converting enzyme inhibitor; *ARB*, angiotensin receptor blocker; *MI*, myocardial infarction.

CI,0.93 to 0.99]). These leads often or normally have inverted T waves.

Subgroup analyses in patients presenting within 1 hour of chest pain onset and patients with prior ECG are presented in the Appendix (Tables E2 and E3, available at http://www.annemergmed.com). These showed similar findings to the main analysis.

LIMITATIONS

Some limitations merit consideration when interpreting these findings. First, this study was conducted in patients in the ED with symptoms suggestive of acute myocardial infarction. Further studies are required to quantify the utility of rule-out and rule-in strategies in patients with either a higher pretest probability (eg, in a coronary care unit setting) or in patients with a lower pretest probability (eg, in a general practitioner setting) for myocardial infarction, as well as in patients with even less time between chest pain onset and ECG examination, eg, patients evaluated in the ambulance. Second, although we used the most stringent methodology to adjudicate the presence or absence of acute myocardial infarction, including central

Table 2. T-wave amplitudes in patients with and without MI.

Lead	MI (n=445)	No MI (n=2012)	All patients (n=2457)
I, mV	0.137 [0.070, 0.211]	0.199 [0.133, 0.269]	0.186 [0.121, 0.262]
II, mV	0.177 [0.100, 0.248]	0.208 [0.141, 0.279]	0.203 [0.132, 0.274]
III, mV	0.068 [0.020, 0.150]	0.037 [0.000, 0.088]	0.042 [0.000, 0.098]
aVF, mV	0.109 [0.058, 0.187]	0.112 [0.062, 0.171]	0.112 [0.061, 0.174]
aVL, mV	0.066 [0.000, 0.135]	0.095 [0.044, 0.151]	0.090 [0.037, 0.149]
aVR, mV	0.000 [0.000, 0.029]	0.000 [0.000, 0.000]	0.000 [0.000, 0.000]
V1, mV	0.055 [0.022, 0.133]	0.038 [0.014, 0.085]	0.040 [0.015, 0.092]
V2, mV	0.300 [0.142, 0.461]	0.309 [0.173, 0.461]	0.306 [0.167, 0.461]
V3, mV	0.312 [0.161, 0.466]	0.362 [0.222, 0.519]	0.354 [0.209, 0.507]
V4, mV	0.243 [0.130, 0.381]	0.320 [0.198, 0.461]	0.308 [0.186, 0.447]
V5, mV	0.194 [0.097, 0.313]	0.270 [0.171, 0.378]	0.257 [0.157, 0.370]
V6, mV	0.151 [0.077, 0.250]	0.211 [0.139, 0.296]	0.201 [0.127, 0.289]

Numbers are presented as median (IQR)
T-wave amplitude stratified by cause of chest pain.
The median amplitudes of leads III, aVR, and V1, which tend to have inverted T waves, were increased for patients with MI compared with patients with another cause of chest pain.

adjudication by board-certified cardiologists or cardiology fellows, we may still have misclassified a small number of patients. Third, we cannot generalize our findings to patients with terminal kidney failure requiring dialysis because they were excluded from this study. Fourth, no specific sample size calculation was performed. Although this post hoc analysis from the ongoing multicenter study is one of the largest ever performed, it may still have been underpowered for some comparisons. Also, digital ECG data were not available for all patients. This applies even more to the analysis incorporating prior ECGs. Therefore, detailed analyses, such as eg, the effect of time-span between the prior ECG and the ECG at presentation were not possible. Fifth, the final adjudication of the diagnoses was based on all available medical records obtained during clinical care and study-specific assessments, including detailed chest pain characteristics, serial hs-cTnT blood concentrations, ECG, and clinical follow-up. The ECG's availability during adjudication could lead to an incorporation bias. Sixth, based on the study design, we only included patients with chest discomfort and hypothetically missing patients with an atypical presentation for myocardial infarction. Furthermore, serial ECGs were not available. Seventh, a clear definition of what a T-wave constitutes is still missing. We defined hyperacute T wave as a tall positive T-wave amplitude above the lead-specific 95th-percentile. Furthermore, in this study, each lead was evaluated individually.

Table 3. Percentage of MI stratified by T-wave amplitude quartiles*.

	T-wave amplitude quartiles			
	1 N=615	2 N=614	3 N=614	4 N=614
I, n (%)	192 (31.2)	111 (18.1)	78 (12.7)	64 (10.4)
II, n (%)	165 (26.8)	102 (16.6)	90 (14.7)	88 (14.3)
III, n (%)	79 (12.8)	83 (13.5)	105 (17.1)	178 (29.0)
aVF, n (%)	121 (19.7)	108 (17.6)	89 (14.5)	127 (20.7)
aVR, n (%)	108 (17.6)	81 (13.2)	78 (12.7)	178 (29.0)
aVL, n (%)	173 (28.1)	94 (15.3)	85 (13.8)	93 (15.1)
V1, n (%)	94 (15.3)	80 (13.0)	110 (17.9)	161 (26.2)
V2, n (%)	130 (21.1)	102 (16.6)	102 (16.6)	111 (18.1)
V3, n (%)	149 (24.2)	99 (16.1)	112 (18.2)	85 (13.8)
V4, n (%)	164 (26.7)	109 (17.8)	93 (15.1)	79 (12.9)
V5, n (%)	178 (28.9)	113 (18.4)	70 (11.4)	84 (13.7)
V6, n (%)	182 (29.6)	99 (16.1)	89 (14.5)	75 (12.2)

*Quartile 1 is the quartile with the lowest T-wave amplitudes; Quartile 4 is the quartile with the highest T-wave amplitudes.

DISCUSSION

This large diagnostic study was performed to investigate the clinical utility of hyperacute T waves as a universally available inexpensive ECG signature in the early diagnosis

Table 4. All patients. Performance of all patients 95%-percentile T-Wave threshold.

Lead	Amplitude (mV)	STEMI n (%)	NSTEMI n (%)	Specificity (%)	Sensitivity (%)	lr+	lr−
I	0.37	4 (3.3)	11 (9.1)	94.7 [93.7, 95.6]	3.4 [2.1, 5.5]	0.6 [0.4, 1.1]	1.02 [1.00, 1.04]
II	0.395	9 (7.3)	10 (8.1)	94.8 [93.8, 95.7]	4.3 [2.8, 6.6]	0.8 [0.5, 1.3]	1.01 [0.99, 1.03]
III	0.231	24 (20)	32 (26)	96.7 [95.8, 97.4]	12.6 [9.8, 16.0]	3.8 [2.7, 5.3]	0.90 [0.87, 0.94]
aVF	0.28	21 (17)	17 (14)	95.8 [94.8, 96.6]	8.5 [6.3, 11.5]	2.0 [1.4, 2.9]	0.95 [0.93, 0.98]
aVR	0.05	9 (7.3)	51 (41)	96.9 [96.0, 97.5]	13.5 [10.6, 17.0]	4.3 [3.1, 6.0]	0.89 [0.86, 0.93]
aVL	0.246	10 (8.2)	19 (16)	95.4 [94.4, 96.2]	6.5 [4.6, 9.2]	1.4 [0.9, 2.1]	0.98 [0.95, 1.01]
V1	0.226	11 (9.1)	26 (21)	95.8 [94.9, 96.6]	8.3 [6.1, 11.3]	2.0 [1.4, 2.9]	0.96 [0.93, 0.99]
V2	0.722	8 (6.6)	24 (20)	95.5 [94.5, 96.3]	7.2 [5.1, 10.0]	1.6 [1.1, 2.4]	0.97 [0.95, 1.00]
V3	0.8	6 (5.0)	17 (14)	95.1 [94.1, 96.0]	5.2 [3.5, 7.6]	1.1 [0.7, 1.7]	1.00 [0.97, 1.02]
V4	0.717	7 (5.7)	11 (8.9)	94.8 [93.7, 95.7]	4.0 [2.6, 6.3]	0.8 [0.5, 1.3]	1.01 [0.99, 1.03]
V5	0.566	5 (4.1)	10 (8.2)	94.7 [93.6, 95.6]	3.4 [2.1, 5.5]	0.6 [0.4, 1.1]	1.02 [1.00, 1.04]
V6	0.439	5 (4.1)	10 (8.1)	94.6 [93.6,95.5]	3.4 [2.1, 5.5]	0.6 [0.4, 1.1]	1.02 [1.00, 1.04]

Sensitivity, Specificity, lr+/− refer to MI (STEMI + NSTEMI).
Percentages of STEMI and NSTEMI refer to the number of ECGs over the respective threshold.

of myocardial infarction in patients presenting to the ED with acute chest discomfort. We found that in most leads, T-wave amplitudes were greater in patients without myocardial infarction than those with myocardial infarction, and T-wave amplitude greater than the 95th percentile had no useful diagnostic value. The exceptions were leads III, aVR, V1, which had greater T-wave amplitudes in those with myocardial infarction, and a T-wave amplitude greater than the 95th percentile had some diagnostic value in ruling in myocardial infarction. However, these leads usually have inverted T waves, so T-wave amplitudes above the 95th percentile represent upright rather than hyperacute T-waves. Therefore, we found no evidence that hyperacute T-waves assist in establishing the diagnosis of myocardial infarction.

These findings run counter to previous work on novel ECG signatures in the early detection of myocardial infarction, including numerous case reports suggesting hyperacute T waves as an early ECG signature of myocardial infarction and pilot studies from the out-of-hospital field triage on tall symmetrical T waves in conjunction with junctional ST-depression.[8,10-13] The latter was observed in 0.2% of total transmitted ECGs and in 1.6% of anterior myocardial infarctions, estimates that are fully in line with the low lr+ observed in our cohort, including the subgroup of early presenters.

It remains speculative if the incidence of hyperacute T-waves is small in general or if it would be higher in an even more acute setting. The lack of a common definition of hyperacute T-waves makes the analysis and evaluation of hyperacute T-waves challenging. It is possible that our definition of hyperacute T-waves (amplitude greater than the 95th percentile) does not reflect the way hyperacute T-waves are interpreted by clinicians. Furthermore, other T-waves changes (particularly inversion) may mean that average measures of T-wave amplitude do not reflect the potential diagnostic value of hyperacute T-waves.

In conclusion, we found no evidence that hyperacute T-wave changes, defined as T-wave amplitude exceeding the 95th percentile, provide useful information in diagnosing myocardial infarction.

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