Predicting the infarct-related artery in STEMI from the surface ECG: independent validation of proposed criteria

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Abstract

Aims: This study independently evaluated the diagnostic performance of electrocardiographic (ECG) criteria to predict the infarct-related artery (IRA) in patients with an acute ST-segment elevation myocardial infarction (STEMI). While a number of ECG criteria have been proposed to predict the IRA in STEMI, many of these "rules" came from modestly sized populations and did not undergo external validation. Therefore, we aimed to evaluate popular criteria from the literature in an independent cohort.

Methods and results: All acute STEMI cases over a 10-year period from a single hospital were retrospectively identified. We excluded patients with a missing pre-intervention ECG, irretrievable angiographic films, prior coronary artery bypass grafting, left bundle branch block, ventricular pacing, or not meeting strict STEMI criteria. After review of the angiograms for the IRA, cases with either no or multiple culprits were excluded. We included 480 subjects meeting STEMI criteria in inferior leads (192, 40%), anterior leads (184, 38%), both anterior and inferior leads (88, 18%), isolated lateral leads (nine, 2%), or a posterior pattern (seven, 1%). Notably, every pattern except isolated lateral STEMI included an IRA in both the right and left coronary arteries.

Conclusions: Existing ECG criteria to predict the IRA in STEMI have modest diagnostic performance when externally validated, and lower than in the original reports. Distinguishing the level of obstruction in the left anterior descending artery remains especially challenging. Hence, their use should be pragmatic when selecting an initial catheter for treating STEMI, since discordances will occur when compared to the actual angiogram.

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Abbreviations

D1	first diagonal branch
ECG	electrocardiogram
IRA	infarct-related artery
LAD	left anterior descending
LCA	left coronary artery
LCx	left circumflex
LMCA	left main coronary artery
RCA	right coronary artery
S1	first septal perforator
STD	ST-segment depression
STE	ST-segment elevation
STEMI	ST-segment elevation myocardial infarction

Introduction

The surface electrocardiogram (ECG) provides the cornerstone for diagnosing patients who present with acute chest pain. The subset with ST-segment elevation myocardial infarction (STEMI) proceeds immediately to intravenous thrombolytics or invasive angiography and revascularisation. The ECG in STEMI can denote a medical emergency and has traditionally been used to predict the anatomical location of coronary artery occlusion[1].

Based on the anticipated culprit vessel, operators often choose to perform an initial diagnostic angiogram of the contralateral artery. A survey has shown that the majority of interventional cardiologists begin with a diagnostic catheter for the presumed non-culprit artery, then proceed with a guide catheter (58% of responders) or diagnostic catheter (23%) for the culprit vessel[2]. However, retrospective, observational studies have shown that – logically – the "non-culprit first" approach increases time to reperfusion, albeit by an average of only 1[3], 4.5[4], 4 to 6[5], 6[6], 7[7], 8[8], or 9[9] minutes. Whether this small increase in ischaemic time translates into worse clinical outcomes has never been explored in a randomised trial, although observational studies suggest no significant impact[3-5,7-9]. Conversely, in some cases, knowledge of non-culprit anatomy may alter the approach to revascularisation.

Regardless of a "culprit first" or "non-culprit first" approach, a reliable method is required to predict the infarct-related artery (IRA). Several surface ECG criteria have been proposed in previous studies. However, these studies were limited by their modest size and often did not undergo external or extensive validation. Additionally, several of them developed rules only in specific angiographic subgroups, whereas in daily clinical practice an approach based purely on the ECG must be selected. Therefore, our study independently evaluated the performance of ECG criteria to predict the IRA in a large cohort of consecutive patients presenting with STEMI.

Methods

We assembled our cohort from all patients with acute STEMI who presented to a single, tertiary care hospital over a 10-year period (from January 2000 to December 2009) and underwent immediate invasive coronary angiography. The Northwestern Memorial Hospital institutional review board approved this retrospective study. A subset of the cohort examining ECG rules to distinguish anterior STEMI from Takotsubo cardiomyopathy has already been analysed and published[10].

Initially, we identified potential subjects by searching the electronic cardiac catheterisation laboratory database for acute infarctions. Each case was reviewed, including surface ECG, angiographic images, and clinical course. Subsequently, we excluded patients with an irretrievable angiogram or ECG, prior coronary artery bypass grafting, absent or multiple culprits, a left bundle branch block or paced ventricular rhythm, under 18 years of age, or those with non-STEMI or unstable angina. A single investigator reviewed the angiogram and clinical course to confirm a true myocardial infarction. In the small number of patients with multiple STEMIs during the decade, only the first episode was included. Hence each unique subject in the cohort had one clear IRA of a native vessel, and its location was recorded locally using the SYNTAX segment[11] but also summarised globally as right coronary artery (RCA) versus left coronary artery (LCA) or by the major epicardial vessel. The presence of a bystander total occlusion, felt to be chronic (CTO), was recorded when clear. For the left anterior descending (LAD) artery, we explicitly noted the order of the first septal perforator and first diagonal branch in relation to the occlusion, except in four cases where this information was not recorded. Coronary dominance based on review of angiographic images was recorded as right, left, or codominant, although in clinical practice this information would not be available before angiography. Demographic information and lab values within 24 hours of hospital presentation were extracted when available.

ECG ANALYSIS

From our electronic database, for each subject we retrieved the ECG that was recorded before cardiac catheterisation. If multiple tracings existed, the one with the largest ST-segment deviation was selected. When available, the time of the ECG recording and start time of the coronary angiogram were noted. ECG recordings used 12 standard leads with 10 seconds of data sampled at 250 times/second and 10 mm/mV calibration. These tracings were imported with custom software into MATLAB® (MathWorks, Natick, MA, USA) for off-line analysis. Identification of QRS onset and J-point for each supraventricular beat was performed manually by a single investigator unaware of the angiographic findings. ST-segment deviation was computed as the average difference in millimetres for all marked beats in each lead between QRS onset and subsequent J-point. ST-segment deviations were measured exactly at the J-point, as recommended[12]. Positive deflections describe ST-segment elevation while negative deviations describe ST-segment depression. The total ST-segment deviation score summed the absolute depression or elevation in all 12 leads.

In accordance with the AHA/ACCF/HRS scientific statement on ECG interpretation in STEMI[12], we required that at least two contiguous ECG leads contained significant ST-segment elevation ($\geq$1 mm in all leads except for $\geq$2 mm in leads V1 to V3).

Additionally, we identified isolated posterior STEMI as significant ST depression ≥1 mm in leads V1 and V2. Because we used an average ST-segment deviation from many beats instead of the worst beat on the tracing, as often happens clinically to avoid missing a STEMI, we relaxed the criterion to ≥0.9 mm (or ≥1.9 mm in leads V1 to V3) to reflect real-world practice better. Subjects who did not meet these strict criteria were excluded. Additionally, we performed a sensitivity analysis using the more stringent ≥1 mm (or ≥2 mm in leads V1 to V3) threshold to determine how many subjects would be reclassified.

We divided our cohort into the classic ECG patterns that correlate with anatomical regions: anterior, inferior, posterior, and lateral[12] as follows.

– Anterior STEMI: significant ST-segment elevation in two contiguous ECG leads V1 to V6.
– Inferior STEMI: significant ST-segment elevation in two contiguous ECG leads III, aVF, and II.
– Posterior STEMI: no significant ST-segment elevation but significant ST-segment depression in ECG leads V1 and V2.
– Lateral STEMI: significant ST-segment elevation only in ECG leads I and aVL.

Because some ECGs fulfilled criteria for both anterior and inferior STEMI (termed "potentially overlapping"), we subdivided these two groups into five mutually exclusive patterns.

– Pure anterior STEMI: significant ST-segment elevation in two contiguous ECG leads V1 to V6 but no inferior STEMI.
– Pure inferior STEMI: significant ST-segment elevation in two contiguous ECG leads III, aVF, and II but no anterior STEMI.
– Inferoseptal STEMI: significant ST-segment elevation in two contiguous ECG leads V1 to V3 plus two contiguous ECG leads III, aVF, and II.
– Inferolateral STEMI: significant ST-segment elevation in two contiguous ECG leads V4 to V6 plus two contiguous ECG leads III, aVF, and II.
– Mixed anterior and inferior STEMI: not other patterns, but nevertheless significant ST-segment elevation in two contiguous ECG leads V1 to V6 plus two contiguous ECG leads III, aVF, and II.

ECG CRITERIA TO PREDICT STEMI CULPRIT

We began with a major review article describing ECG criteria to predict the IRA in STEMI[13]. Next, we sought out the original papers[14-16] and extracted basic information, including ECG criteria, sample size, sensitivity, and specificity. For some rules, we were able to find subsequent cohorts that had been evaluated independently[17,18]. We then applied these ECG criteria to our own cohort.

– Inferior STEMI: ST-segment elevation in lead III greater than lead II indicates an RCA culprit[14].

Inferior STEMI: ST-segment depression in lead I or aVL ≥1 mm indicates an RCA culprit[15].

Inferior STEMI: ST-segment depression in aVL greater than lead I indicates an RCA culprit[14].

– Anterior STEMI: ST-segment elevation in leads V2 and V3 ≥2 mm plus a variety of added criteria to indicate location

along the LAD[16] when proximal to the first septal perforator if ST-segment elevation >2.5 mm in lead V1; distal to the first septal perforator if no ST-segment depression in lead II; proximal to the first diagonal branch if ST-segment depression ≥1 mm in lead II; and distal to the first diagonal branch if no ST-segment depression in lead II.

STATISTICAL ANALYSIS

Analysis was performed using R, Version 3.3.1 (R Foundation for Statistical Computing, Vienna, Austria). We employed standard statistical techniques. To summarise diagnostic performance, we computed the Youden index, equal to the sum of sensitivity plus specificity minus 1[19]. A gold standard test has a Youden index of 1.0, while a useless test has a Youden index of zero. For each ECG pattern, we calculated the ratio of RCA to LCA culprits. A value >1 reflects a higher probability for an IRA in the RCA, while a value <1 reflects a higher probability for the LCA. Applicable tests were two-tailed, and $p<0.05$ was considered statistically significant.

Results

Over a 10-year period we examined 882 patients in our electronic database, of whom 480 met STEMI inclusion criteria as detailed in **Figure 1**. Demographic and clinical characteristics can be found in **Table 1** and reflect a typical infarct population. Total ST-segment deviations were always >5 mm except for two cases (4.6 and 4.8 mm), with a median value of 14 mm (interquartile

Table 1. Baseline characteristics.

		N=480
Age (years)		59±13
Male		74%
Race	White	68%
	Black	15%
	Hispanic	5%
Body mass index (kg/m²)		28±6
Heart rate (beats per minute)		78±20
Lab values	CK-MB peak (U/L)	249 (133-426)
	Total cholesterol (mg/dL)	196±54
	LDL (mg/dL)	126±48
	HDL (mg/dL)	41±15
	Triglycerides (mg/dL)	112 (79-203)
Coronary dominance	Right	95%
	Left	4%
	Codominant	1%
Level of LAD occlusion (n=184)	Proximal to S1 and D1	30%
	Distal to D1, proximal to S1	12%
	Proximal to D1, distal to S1	12%
	Distal to S1 and D1	45%

Values are shown as mean±SD, median (interquartile range), or percentage (if binary). Race shows only the three most common responses.
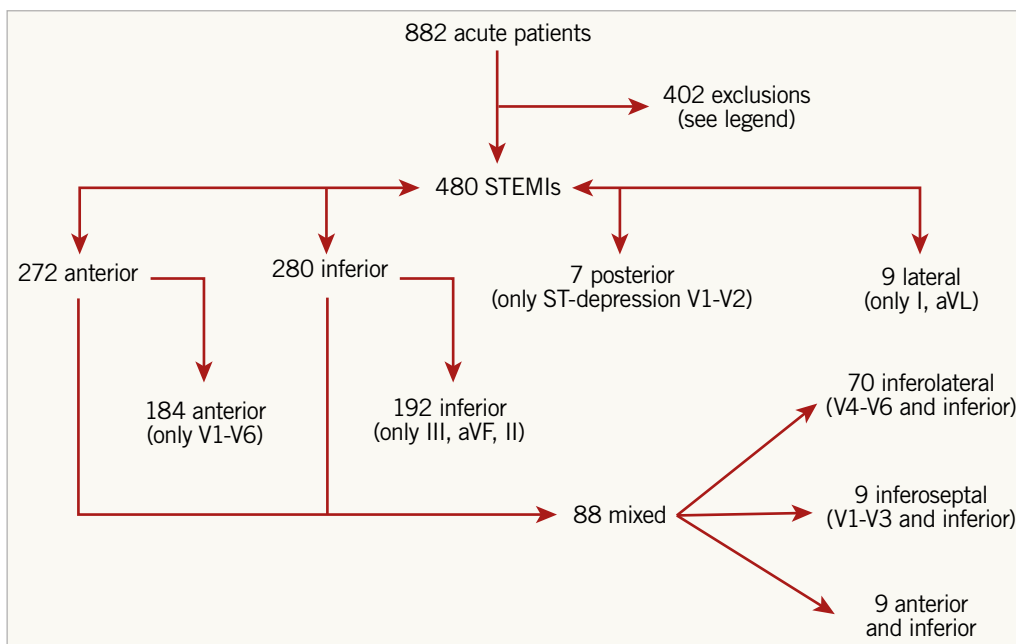
Figure 1. *Study flow chart. Starting with the total number of acute infarction cases over 10 years, we excluded patients who did not meet criteria for STEMI with an available ECG and angiogram. Exclusions: 247 did not meet STEMI criteria; 51 had prior CABG; 41 had multiple culprits or no culprit; 20 with duplicate event; 17 without retrievable ECG; 16 had LBBB or paced ventricular rhythm; 9 without retrievable angiogram; and 1 under the age of 18 years old. Included patients had various ECG patterns of ST-segment elevation, as further detailed in Table 2.*

range 10 to 21 mm). Valid time stamps for both ECG and angiogram were available in 392 (81.7%) cases, of which 132 (33.7%) were within 30 minutes of the angiogram and a further 96 (24.5%) within 60 minutes. A total of 23 subjects (5% of the included cohort) would not have been classified as STEMI using ≥1 mm (or ≥2 mm in leads V1 to V3) instead of 0.1 mm less, including 14 anterior (10 LAD culprits, two left circumflex [LCx], two RCA), eight inferior (six RCA culprits, one LCx, one LAD), and one lateral (LCx culprit) patterns.

The vast majority of the cohort had some type of anterior or inferior STEMI (96%), while less than 5% had an isolated lateral or posterior STEMI. **Table 2** provides the distribution of culprit vessels for each ECG pattern among the left main coronary artery (LMCA), LAD, LCx, and RCA. As in **Figure 1**, patterns in **Table 2** are separated between those that potentially overlap versus the mutually exclusive grouping. Notably, all patterns except lateral STEMI could result from either an LCA or RCA culprit, making an absolute rule based solely on ST-segment elevation

Table 2. Culprit vessels in STEMI by ECG pattern.

Potentially overlapping patterns	RCA	LAD	LCx	LMCA	RCA versus LCA*
Inferior (II, III, aVF)	209	23	48	0	2.94
Anterior (V1 to V6)	56	183	30	3	0.26
Posterior (ST depression V1-V2)	1	0	6	0	0.17
Lateral (only I and aVL)	0	6	2	1	0.00
Mutually exclusive patterns					
Inferior (II, III, aVF and not anterior)	159	5	28	0	4.82
Inferolateral (V4-V6 and inferior)	45	5	20	0	1.80
Anterior+inferior (V1-V6 and inferior)	3	6	0	0	0.50
Inferoseptal (V1-V3 and inferior)	2	7	0	0	0.29
Posterior (ST depression V1-V2)	1	0	6	0	0.17
Anterior (V1 to V6 and not inferior)	6	165	10	3	0.03
Lateral (only I and aVL)	0	6	2	1	0.00
*Ratio of RCA culprits to LCA culprits. Values >1 reflect a higher probability for an IRA in the RCA, while values <1 reflect a higher probability for the LCA.					

impossible. For the 12 cases with a clear, chronic total occlusion, only one case of an acute LAD culprit produced an isolated, inferior STEMI (no significant anterior ST-segment elevation) with chronic RCA occlusion; all other chronic total occlusions displayed agreement between acute culprit vessel and ECG pattern (anterior with LAD culprit; inferior or posterior with RCA or LCx culprit). As specific examples of discordance, **Figure 2** shows an inferior STEMI due to an LCx culprit (despite ST-segment elevation in lead III larger than lead II combined with ST-segment depression in leads I and aVL); **Figure 3** shows an anterior STEMI due to an RCA culprit.

PREDICTING THE CULPRIT FOR INFERIOR STEMI

A total of 280 subjects had an inferior STEMI, with an almost 3-to-1 dominance of RCA culprits **(Table 2)**. If only the inferior leads were involved, then the ratio climbed to almost 5-to-1. Proposed rules for separating RCA from LCA culprits had modest diagnostic performance, as summarised in **Table 3**, and always

worse in validation cohorts than the initial derivation cohort. However, the Youden index improved when moving from isolated inferior STEMI to our entire cohort of inferior STEMI to inferolateral STEMI, probably due to the decreasing likelihood of an RCA culprit, as seen in **Table 2** by the RCA/LCA ratio.

PREDICTING THE CULPRIT FOR ANTERIOR STEMI

A total of 272 subjects had an anterior STEMI, with an approximate 5-to-1 dominance of LCA culprits **(Table 2)**. If only the anterior leads were involved, then the ratio climbed to over 30-to-1. In either case, culprits were found in the RCA, implying that anterior STEMI does not always arise from the LCA. As summarised in **Table 4**, proposed rules for distinguishing the site of LAD occlusion in isolated anterior STEMI performed very poorly in our validation cohort and uniformly worse than in the derivation cohort. For predicting an occlusion proximal to the first septal perforator, the Youden index was near zero, implying an almost worthless diagnostic test.
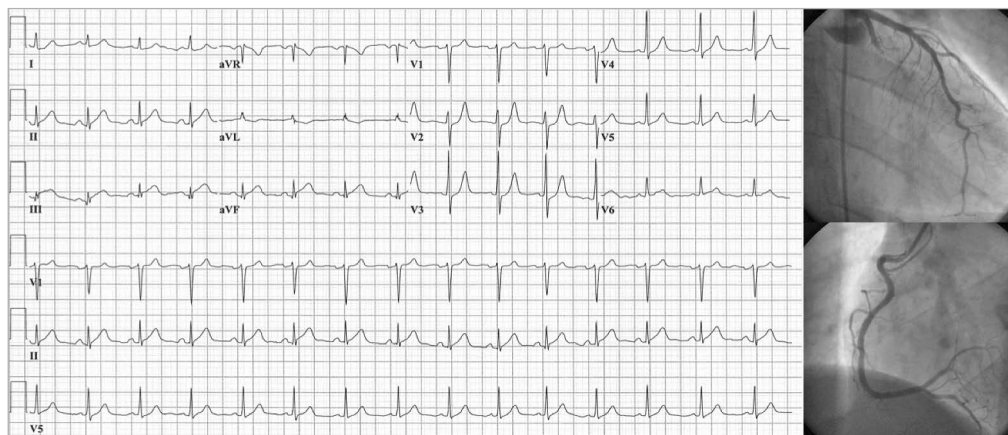

Figure 2. *Example of inferior STEMI. ECG and corresponding coronary angiogram depict an inferior STEMI with ST-segment elevation in lead III >lead II and <1 mm ST-segment depression in leads I and aVL, where the infarct-related artery was nevertheless the proximal left circumflex.*
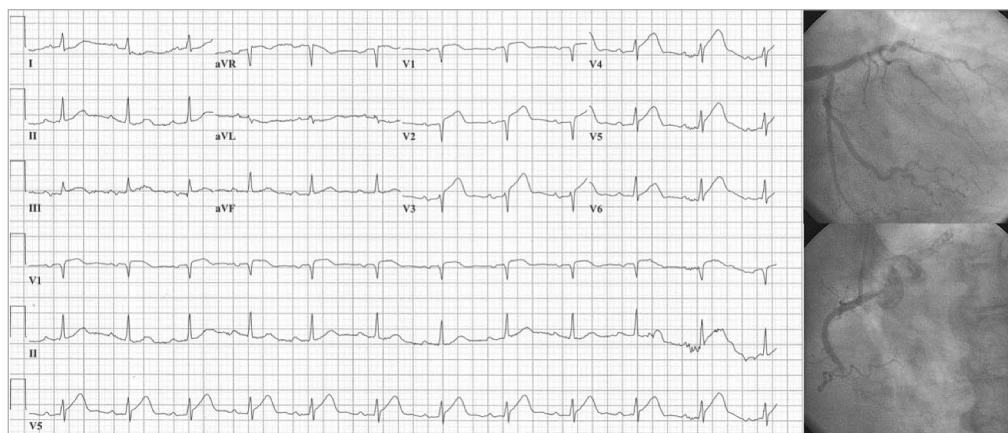

Figure 3. *Example of anterior STEMI. ECG and corresponding coronary angiogram depict an anterior STEMI where the infarct-related artery was the mid RCA despite a complete lack of significant inferior ST-segment deviation.*

Table 3. Inferior STEMI rules to predict an RCA culprit.

Criterion	Sensitivity	Specificity	Youden index	# of patients	Source
STE III > II	84%	56%	0.40	1,131	Verouden 2009[18]
	95%	48%	0.43	280	Inferior
	94%	33%	0.28	192	Inferior (only II, III, aVF)
	98%	60%	0.58	70	Inferolateral (V1-V3 plus inferior)
	88%	94%	0.82	83	Herz 1997[14]
	73%	100%	0.73	69	Zimetbaum 1998[17]
STD I or aVL	82%	56%	0.38	280	Inferior
	77%	48%	0.26	192	Inferior (only II, III, aVF)
	100%	56%	0.56	70	Inferolateral (V1-V3 plus inferior)
	83%	67%	0.50	41	Bairey 1987[15]
STD aVL > I	82%	22%	0.04	1,131	Verouden 2009[18]
	95%	52%	0.47	280	Inferior
	95%	33%	0.28	192	Inferior (only II, III, aVF)
	98%	64%	0.62	70	Inferolateral (V1-V3 plus inferior)
	80%	94%	0.74	83	Herz 1997[14]
STD: ST-segment depression; STE: ST-segment elevation					

Discussion

Predicting the culprit vessel in STEMI carries clinical relevance for several reasons. Most importantly, interventional cardiologists would like to know the IRA in advance of the procedure to choose either a "culprit first" or a "non-culprit first" approach[2]. Although neither strategy has been tested in a randomised trial of outcomes, the correct choice can clearly make a difference for select patients who present in extremis such as with cardiogenic shock, complete heart block, or electrical storm. Additionally, understanding the culprit artery allows anticipation of clinical scenarios such as conduction disturbances and mechanical complications.

Our cohort provides a large, independent validation of commonly used criteria for predicting the IRA in STEMI. We found that either the RCA or LCA can produce every ECG pattern with the exception of isolated lateral STEMI. Therefore, using only the location of ST-segment elevation cannot serve as a foolproof rule. Furthermore, many proposed criteria have poor diagnostic performance when validated outside their derivation cohorts, suggesting a lack of generalisability. As such, their successful application to clinical practice will remain limited. Indeed, the pre-test probabilities in our **Table 2** probably provide a more realistic guide for the IRA.

A review of prior work highlights an important difference with our study. Some previous cohorts were first selected on the basis of angiographic criteria before studying the ECG patterns. For example, one study began by searching for RCA or LCx culprits in STEMI, then applying inferior ECG criteria, thereby explicitly excluding LAD culprits[18]. By design, such a search strategy differs

Table 4. Anterior STEMI rules to predict the level of LAD occlusion.

Criterion	Sensitivity	Specificity	Youden index	# of patients	Source
STE V2-V3 and STE V1 >2.5 (proximal to S1 in LAD)	4%	98%	0.02	272	Anterior
	4%	97%	0.01	184	Anterior (only V1-V6)
	12%	100%	0.12	100	Engelen 1999[16]
STE V2-V3 and STD II ≥1.0 (proximal to D1 in LAD)	25%	97%	0.22	270	Anterior
	27%	95%	0.21	182	Anterior (only V1-V6)
	34%	98%	0.32	100	Engelen 1999[16]
STE V2-V3 and no STD II (distal to D1 in LAD)	45%	83%	0.28	270	Anterior
	43%	76%	0.19	182	Anterior (only V1-V6)
	66%	73%	0.39	100	Engelen 1999[16]
STE V2-V3 and no STD II (distal to S1 in LAD)	43%	83%	0.26	272	Anterior
	43%	77%	0.19	184	Anterior (only V1-V6)
	67%	74%	0.41	100	Engelen 1999[16]
D1: first diagonal branch; STD: ST-segment depression; STE: ST-segment elevation; S1: first septal perforator					

fundamentally from clinical practice where the ECG comes first and must predict the invasive IRA. Our **Table 2** demonstrates that 8% of inferior STEMI arise from the LAD.

For inferior or inferolateral STEMI, prediction rules cannot anticipate coronary dominance. As shown in **Table 2**, an almost 2-to-1 split exists between RCA and LCA culprits when significant ST-segment elevations occur in both inferior leads and V4 to V6 (an inferolateral STEMI). This ratio approximates the population prevalence of right- versus left-dominant circulations, typically 80% pure right dominant[20], although the dominant vessel may not always be the culprit. Thus, while reasonable in general, we cannot expect an ECG to provide us with information regarding dominance at the level of a single patient. Similarly, the presence of unanticipated chronic total occlusions or "wrap around" LAD or posterior descending arteries cannot be known from the ECG, but can affect the resulting ECG pattern. Therefore, the modest and imperfect predictive ability of rules for STEMI culprits should not be surprising when considering anatomic variations.

For the level of LAD occlusion in anterior STEMI, our results show that proposed rules have very little diagnostic value. While disappointing, the practical consequences for identifying the LCA as the culprit remain minimal, as the vast majority of (but not all) anterior STEMI arises from the LCA and therefore will not affect the choice of vessel order during an invasive procedure.

The modest diagnostic performances in **Table 3** and **Table 4** serve as an additional warning regarding overenthusiastic adoption of results without independent confirmation. Our sample size for validation was larger than any of the original derivation cohorts, again providing thoughtful restraint and shaping realistic expectations for results from small populations. For this reason, we did not seek to derive new rules in our cohort, but instead focused on the more important task of independently examining existing criteria.

Logical future work that builds on our results would be a randomised trial comparing "culprit first" to "non-culprit first" interventional approaches in STEMI. While a topic of frequent debate in the literature[21-23], the practical consequence for ordering the sequence of angiography and intervention in STEMI has not been definitively established. Both methods are used routinely in practice, STEMI occurs frequently, and the general idea falls under the umbrella of comparative effectiveness research wherein informed consent may be simplified greatly[24]. In the protocol of such a trial, our **Table 2** could serve to develop rules for how to select the culprit, with full awareness of real-world variation and hence modest diagnostic performance regardless of the chosen strategy.

Limitations

As our data were collected retrospectively, our results share all the well-known limitations of this design. We did not examine lead V4R due to its inconsistent recording, although that lead might be useful in distinguishing between the RCA and LCx in inferior STEMI[1]. We excluded patients with prior bypass grafting as their more complex anatomy can produce unusual ECG patterns during STEMI. However, post-bypass patients remain a small minority of STEMI populations[25] and their history is almost always available before beginning cardiac catheterisation. Following current recommendations[12], we measured ST-segment deviation exactly at the J-point, in contrast to some – but not all[16] – earlier publications that added an 80-millisecond delay[14,15,17], leading to small differences in definitions. While our strict STEMI criteria come from the guidelines[12], in practice a borderline ECG in the correct clinical setting will prompt immediate angiography. Although we excluded these tracings, apart from a small 5% minority with elevations within 0.1 mm of the criteria, probably the results would have been even less predictive for the IRA due to smaller ST-segment deviations. The vast majority, but not all, of the cohort had a total ST-segment deviation >5 mm, previously found to be highly predictive of the culprit artery in contrast to smaller amounts of ST-segment deviation[26]. The time between the ECG and angiogram was not uniformly recorded or standardised; a 12-lead ECG at the time of angiography would have provided simultaneous assessment of anatomy and electrocardiography, although not reflective of routine clinical practice. We did not systematically record if a "wrap around" LAD supplied the inferior region, which might explain some cases of inferior STEMI due to a LAD culprit, although clinically this information would not be available prior to angiography. Finally, we applied single rules in isolation and did not combine existing rules; potentially a combination might improve diagnostic performance if the incorrect results from one rule were corrected by a second rule.

Conclusions

Existing ECG criteria to predict the IRA in STEMI have modest diagnostic performance when externally validated, and lower overall than in the original reports. Distinguishing the level of obstruction in the LAD artery remains especially challenging. Hence, use of ECG prediction rules should be pragmatic when selecting an initial catheter for treating STEMI, since discordances will occur when compared to the actual anatomy.

Impact on daily practice

Existing electrocardiographic (ECG) criteria to predict the infarct-related artery in ST-segment elevation myocardial infarction (STEMI) have modest diagnostic performance when externally validated, and lower overall than in the original reports. Distinguishing the level of obstruction in the LAD artery remains especially challenging. Hence use of ECG prediction rules should be pragmatic when selecting an initial catheter for treating STEMI, since discordances will occur when compared to the actual anatomy.

Conflict of interest statement

J. Flaherty has received investigator and proctor fees from Direct Flow Medical (SALUS, NCT01932099) for a device study in aortic stenosis. L. Dekker has received consultancy fees from

References

1. Wellens H, Conover M. The ECG in emergency decision making. Second edition. St. Louis, MO, USA: Saunders Elsevier; 2006.

2. Chiang A, Gada H, Kodali SK, Lee MS, Jeremias A, Pinto DS, Bangalore S, Yeh RW, Henry TD, Lopez-Cruz G, Mehran R, Kirtane AJ. Procedural variation in the performance of primary percutaneous coronary intervention for ST-elevation myocardial infarction: a SCAI-based survey study of US interventional cardiologists. *Catheter Cardiovasc Interv.* 2014;83:721-6.

3. Jurado-Román A, García-Tejada J, Hernández-Hernández F, Granda-Nistal C, Rubio-Alonso B, Agudo-Quílez P, Velázquez-Martín M, Albarrán-González-Trevilla A, Tascón-Pérez J. Does reducing ischemia time justify to catheterize firstly the culprit artery in every primary PCI? *Heart Vessels.* 2016;31:1022-9.

4. Lachance P, Déry JP, Beaudoin J, Barbeau GE, Noël B, Bertrand OF, Rodés-Cabau J, Nguyen CM, Proulx G, Gleeton O, Larose E, Roy L, Delarochelliére R. ECG-guided immediate intervention at the time of primary PCI to reduce door-to-balloon time in ST-elevation myocardial infarction patients. *J Invasive Cardiol.* 2008;20:623-6.

5. Couture EL, Bérubé S, Dalery K, Gervais A, Harvey R, Nguyen M, Parenteau É, Daneault B. Culprit Vessel Revascularization Prior to Diagnostic Angiography as a Strategy to Reduce Delays in Primary Percutaneous Coronary Intervention: A Propensity-Matched Analysis. *Circ Cardiovasc Interv.* 2016;9:e003510.

6. Ahn J, Cha KS, Choi JC, Kim JH, Park JS, Lee HW, Oh JH, Choi JH, Lee HC, Hong TJ. The impact of electrocardiogram-guided, direct-targeting intervention of the infarct-related artery on the door-to-balloon time: a pilot study. *Int J Cardiol.* 2013;168:4530-1.

7. Applegate RJ, Graham SH, Gandhi SK, Kutcher MA, Sacrinty MT, Santos RM, Little WC. Culprit vessel PCI versus traditional cath and PCI for STEMI. *J Invasive Cardiol.* 2008;20:224-8.

8. Plourde G, Abdelaal E, Bataille Y, MacHaalany J, Déry JP, Déry U, Larose É, De Larochellière R, Gleeton O, Barbeau G, Roy L, Costerousse O, Bertrand OF. Effect on door-to-balloon time of immediate transradial percutaneous coronary intervention on culprit lesion in ST-elevation myocardial infarction compared to diagnostic angiography followed by primary percutaneous coronary intervention. *Am J Cardiol.* 2013;111:836-40.

9. Dib C, Hanna EB, Chaudhry MA, Hennebry TA, Stavrakis S, Abu-Fadel MS. Culprit-vessel percutaneous coronary intervention followed by contralateral angiography versus complete angiography in patients with ST-elevation myocardial infarction. *Tex Heart Inst J.* 2012;39:359-64.

10. Johnson NP, Chavez JF, Mosley WJ 2nd, Flaherty JD, Fox JM. Performance of electrocardiographic criteria to differentiate Takotsubo cardiomyopathy from acute anterior ST elevation myocardial infarction. *Int J Cardiol.* 2013;164:345-8.

11. Sianos G, Morel MA, Kappetein AP, Morice MC, Colombo A, Dawkins K, van den Brand M, Van Dyck N, Russell ME, Mohr FW, Serruys PW. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. *EuroIntervention.* 2005;1:219-27.

12. Wagner GS, Macfarlane P, Wellens H, Josephson M, Gorgels A, Mirvis DM, Pahlm O, Surawicz B, Kligfield P, Childers R, Gettes LS, Bailey JJ, Deal BJ, Gorgels A, Hancock EW, Kors JA, Mason JW, Okin P, Rautaharju PM, van Herpen G; American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: 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;53:1003-11.

13. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med.* 2003;348:933-40.

14. Herz I, Assali AR, Adler Y, Solodky A, Sclarovsky S. New electrocardiographic criteria for predicting either the right or left circumflex artery as the culprit coronary artery in inferior wall acute myocardial infarction. *Am J Cardiol.* 1997;80:1343-5.

15. Bairey CN, Shah PK, Lew AS, Hulse S. Electrocardiographic differentiation of occlusion of the left circumflex versus the right coronary artery as a cause of inferior acute myocardial infarction. *Am J Cardiol.* 1987;60:456-9.

16. Engelen DJ, Gorgels AP, Cheriex EC, De Muinck ED, Ophuis AJ, Dassen WR, Vainer J, van Ommen VG, Wellens HJ. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol.* 1999;34:389-95.

17. Zimetbaum PJ, Krishnan S, Gold A, Carrozza JP 2nd, Josephson ME. Usefulness of ST-segment elevation in lead III exceeding that of lead II for identifying the location of the totally occluded coronary artery in inferior wall myocardial infarction. *Am J Cardiol.* 1998;81:918-9.

18. Verouden NJ, Barwari K, Koch KT, Henriques JP, Baan J, van der Schaaf RJ, Vis MM, van den Brink RB, Piek JJ, Tijssen JG, de Winter RJ. Distinguishing the right coronary artery from the left circumflex coronary artery as the infarct-related artery in patients

undergoing primary percutaneous coronary intervention for acute inferior myocardial infarction. *Europace.* 2009;11:1517-21.

19. Youden WJ. Index for rating diagnostic tests. *Cancer.* 1950;3:32-5.

20. Knaapen M, Koch AH, Koch C, Koch KT, Li X, van Rooij PC, Tijssen JG, Peters RJ, van der Wal AC, Damman P, de Winter RJ. Prevalence of left and balanced coronary arterial dominance decreases with increasing age of patients at autopsy. A postmortem coronary angiograms study. *Cardiovasc Pathol.* 2013;22:49-53.

21. Chhatriwalla AK, Bhatt DL. Breaking with tradition: is diagnostic angiography really necessary before primary PCI? *J Invasive Cardiol.* 2008;20:229-30.

22. Fernandes MR, Silva GV, Perin EC. Contralateral angiography in acute myocardial infarction patients: is less really more? *Tex Heart Inst J.* 2012;39:365-6.

23. Blankenship JC, Scott TD. What matters and what does not: variations in STEMI PCI techniques. *Catheter Cardiovasc Interv.* 2014;83:727-8.

24. Kim SY, Miller FG. Informed consent for pragmatic trials--the integrated consent model. *N Engl J Med.* 2014;370:769-72.

25. Welsh RC, Granger CB, Westerhout CM, Blankenship JC, Holmes DR Jr, O'Neill WW, Hamm CW, Van de Werf F, Armstrong PW; APEX AMI Investigators. Prior coronary artery bypass graft patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. *JACC Cardiovasc Interv.* 2010;3:343-51.

26. Meissner A, Trappe HJ, de Boer MJ, Gorgels AP, Wellens HJ. The value of the ECG for decision-making at first medical contact in the patient with acute chest pain. *Neth Heart J.* 2010;18: 301-6.