American Journal of Emergency Medicine 48 (2021) 18–32

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Sharing and Teaching Electrocardiograms to Minimize Infarction (STEMI): reducing diagnostic time for acute coronary occlusion in the emergency department

Jesse T.T. McLaren, MD [a,b,*], Ahmed K. Taher, MD, MPH [a,c], Monika Kapoor, MD [a,b], Soojin L. Yi, MD [a,b], Lucas B. Chartier, MD CM, MPH [a,c]

[a] Emergency Department, University Health Network, Toronto, ON, Canada
[b] Department of Family and Community Medicine, University of Toronto, Toronto, ON, Canada
[c] Division of Emergency Medicine, Department of Medicine, University of Toronto, Toronto, ON, Canada

ARTICLE INFO

Article history:
Received 8 December 2020
Received in revised form 19 February 2021
Accepted 21 March 2021

Keywords:
ST elevation myocardial infarction
Electrocardiography
Quality improvement

ABSTRACT

Background: Limits to ST-Elevation Myocardial Infarction (STEMI) criteria may lead to prolonged diagnostic time for acute coronary occlusion. We aimed to reduce ECG-to-Activation (ETA) time through audit and feedback on STEMI-equivalents and subtle occlusions, without increasing Code STEMIs without culprit lesions.
Methods: This multi-centre, quality improvement initiative reviewed all Code STEMI patients from the emergency department (ED) over a one-year baseline and one-year intervention period. We measured ETA time, from the first ED ECG to the time a Code STEMI was activated. Our intervention strategy involved a grand rounds presentation and an internal website presenting weekly local challenging cases, along with literature on STEMI-equivalents and subtle occlusions. Our outcome measure was ETA time for culprit lesions, our process measure was website views/visits, and our balancing measure was the percentage of Code STEMIs without culprit lesions.
Results: There were 51 culprit lesions in the baseline period, and 64 in the intervention period. Median ETA declined from 28.0 min (95% confidence interval [CI] 15.0–45.0) to 8.0 min (95%CI 6.0–15.0). The website garnered 70.4 views/week and 27.7 visitors/week in a group of 80 physicians. There was no change in percentage of Code STEMIs without culprit lesions: 28.2% (95%CI 17.8–38.6) to 20.0% (95%CI 11.2–28.8%).
Conclusions
Our novel weekly web-based feedback to all emergency physicians was associated with a reduction in ETA time by 20 min, without increasing Code STEMIs without culprit lesions. Local ECG audit and feedback, guided by ETA as a quality metric for acute coronary occlusion, could be replicated in other settings to improve care.

1. Introduction

Quality Improvement (QI) initiatives for acute coronary occlusion have focused on setting quality benchmarks and developing methods to meet them [1]. However, prior work has not focused primarily on emergency physicians. According to the American Heart Association/American College of Cardiology (AHA/ACC) guidelines, "The D2B [door-to-balloon] time interval includes 3 key components: door-to-ECG time, ECG-to-catheterization laboratory time, and laboratory-to-device time." [2] While the first component provides a quality metric for nurses and the third component provides a quality metric for interventional cardiologists, the second component combines emergency physician diagnostic time with transportation from the emergency department (ED) to the cath lab (including potentially transferring patients from non-PCI centres to PCI centres). Door-to-activation time is a key driver of overall door-to-balloon times [3] and many quality improvement initiatives have focused on the first part of this process, door-to-ECG time. [4] But ECG-to-Activation (ETA) time, which reflects emergency physician clinical decision-making, has been neglected even within EDs. [5]

The diagnosis of acute coronary occlusion has been simplified into ST-Elevation Myocardial Infarction (STEMI) criteria, often automated by computer interpretation. But STEMI criteria miss a quarter of acute coronary occlusions [6-10] and are associated with delayed reperfusion. [11-13] Automated interpretation has high rates of error [14-16] and can bias physician judgement. [17] In response to this, STEMI-equivalent patterns [18-21] and rules for subtle occlusions [22-25] have been identified. This has led to questioning the current STEMI

* Corresponding author at: Toronto General Hospital, 200 Elizabeth Street, R. Fraser Elliott Building, Ground Floor, Room 480, Toronto, ON M5G 2C4, Canada.
E-mail addresses: jesse.mclaren@gmail.com (J.T.T. McLaren), ahmed.taher@mail.utoronto.ca (A.K. Taher), monika.kapoor@utoronto.ca (M. Kapoor), lucas.chartier@uhn.ca (L.B. Chartier).

https://doi.org/10.1016/j.ajem.2021.03.067

paradigm [26-28] and a proposed paradigm shift to Occlusion myocardial infarction (OMI). [29-31] However, there is also a concern about unnecessary cath lab activation for patients without culprit lesions. [32-36] Therefore, there needs to be a fine balance when translating new ECG insights to reduce diagnostic time for patients with acute coronary occlusion without increasing cath lab activation for patients without occlusion.

We have previously reported on a clinically significant but neglected time interval: ECG-to-Activation (ETA) time, i.e. the time between the first ED ECG and the time the cath lab is activated. [37] In our three-year retrospective review of Code STEMI activations from the ED with culprit lesions, approximately half of first ED ECGs were not labeled "STEMI" by automated interpretation and a third did not meet STEMI criteria.

But a further quarter had other diagnostic signs of occlusion including STEMI-equivalents and subtle occlusions, which were rarely recognized by automated interpretation and associated with significant diagnostic time as measured by ETA time. This suggested that education and feedback on the limits of automated interpretation and STEMI criteria, as well as on new ECG insights into acute coronary occlusion, could reduce ETA time.

In this study, we examined whether a QI initiative centered on weekly web-based feedback to all emergency physicians could reduce the ETA time (i.e. increasing sensitivity) without the unintended consequence of increasing Code STEMI activations without culprit lesions (i.e. decreasing specificity).

2. Methods

2.1. Study design, setting and population

This was a QI initiative on Code STEMI patients from the ED, and included a one-year baseline period and a one-year intervention period. It was part of an ongoing QI initiative aimed at improving the quality of care of patients with suspected acute coronary occlusion in the ED. We obtained Research Ethics Board exemption at our institution for this initiative (waiver #18–0261), and followed the SQUIRE guidelines.

We collected data at two urban, academic centres that collectively received 115,000 ED annual visits. There were 220 Code STEMIs per year, of which 80 were from activated from the ED and the rest from the field, other hospitals, or inpatient wards. The EDs were staffed by 80 attending emergency physicians, who directly activated Code STEMIs and also had the option of a STAT cardiology consultation for equivocal cases, after which cardiologists would activate the cath lab.

2.2. Intervention

The cath lab provided the database of all Code STEMI patients who underwent emergent catheterization from January 2018 to December 2019. Patients were categorized as with or without a culprit lesion requiring reperfusion, as determined by the interventional cardiologist. We excluded Code STEMI patients from inpatient wards and direct transfers from the field and other hospitals. We also excluded patients who died before catheterization results.

In January 2019, the lead author gave a grand rounds presentation to emergency physicians about unnecessary and delayed cath lab activations. The review of unnecessary cath lab activations included ECGs from six local de-identified cases: an ECG erroneously labeled "STEMI", LV aneurysm morphology, Brugada pattern, left ventricular hypertrophy with repolarization abnormalities, left bundle branch block without Sgarbossa/Smith criteria, and right bundle branch block without ischemic changes. The rounds also presented 20 de-identified cases and ECGs of our top 10 challenging decisions to activate the cath lab: patients presenting with angina equivalents, dynamic changes on ECG, subtle LAD occlusion, subtle inferior MI, posterior MI, first diagonal occlusion, diffuse ST depression with ST elevation in aVR, Q waves from acute MI, left bundle branch block with Sgarbossa/Smith criteria, right bundle branch block with occlusion MI, and left ventricular hypertrophy with occlusion MI. Each included one case with delayed cath lab activation and one case with rapid cath lab activation, along with insights from the literature that can help diagnosis. All cases were posted to an internal Wordpress blog, and we also distributed a review article on new ECG insights on acute coronary occlusion, by Miranda et al. [38]

Following this initial educational intervention, a weekly audit and collective feedback program ensued from January to November 2019. The lead author curated and distributed a weekly blogpost to all attending emergency physicians (Appendix). Each post contained five ECGs on average and focused on a different theme. Posts included examples of unnecessary or delayed activations and astute diagnoses from recent local de-identified cases, highlights from the literature on STEMI-equivalents and subtle occlusions, and take-home points. Because cases were shared with the entire emergency physician group, only the physicians who astutely diagnosed challenging cases (and not those who contributed delayed or unnecessary activations) were named and congratulated, in order to promote further engagement.

2.3. Measures

We used the ED triage sheet to obtain the age, sex, chief complaint, method of arrival, and triage time for all Code STEMIs presenting to the ED, and we reviewed charts to determine cardiac risk factors. We obtained the first ED ECG from the chart, determined whether or not it was labeled "STEMI" by the automated interpretation, and calculated the door-to-ECG time (from triage time to time stamp on the first ED ECG).

We reviewed charts of patients with culprit lesions to calculate the ETA time (from the time stamp on the first ED ECG to the time a Code STEMI was activated, as documented in the hospital call centre log). We used ETA time because it is a reliable, objective measurement and most closely reflects the diagnostic time of emergency physicians. We also reviewed cath lab reports of patients with culprit lesions to record the percentage of occlusion, which at our institution is more routinely recorded than pre-procedure TIMI flow.

Our outcome measure was ETA time for culprit lesions. Our process measure was website views/visits, as recorded by the Wordpress blog circulated to emergency physicians. Our balancing measure was the percentage of Code STEMIs without culprit lesion.

2.4. Data analysis

The analysis of demographics, cardiac risk factors, chief complaints, method of arrival, ECGs labeled "STEMI", and Code STEMIs used percentages, with baseline differences compared using Chi square. Median ETA times were calculated in minutes and Code STEMIs without culprit lesions used percentages, and confidence intervals were generated for both. A run chart and all statistics were completed with QI Macros© (Version 2018.04, KnowWare International Inc., Denver, CO, USA) and Microsoft Excel© (Microsoft Corporation, Redmond, WA, USA, Version 14.5.9).

3. Results

The final analysis included 71 cath lab activations with 51 culprit lesions in the baseline period, and 80 cath lab activations with 64 culprit lesions in the post-intervention period. Fig. 1 presents the flow diagram of exclusion and inclusion criteria.

Table 1 compares the characteristics for all ED Code STEMI patients in the baseline and intervention period. There were no differences in age, cardiac risk factors, chief complaint, arrival by ambulance, Door-to-ECG time, or percentage of first ED ECGs labeled "STEMI". There were relatively fewer men in the intervention period.

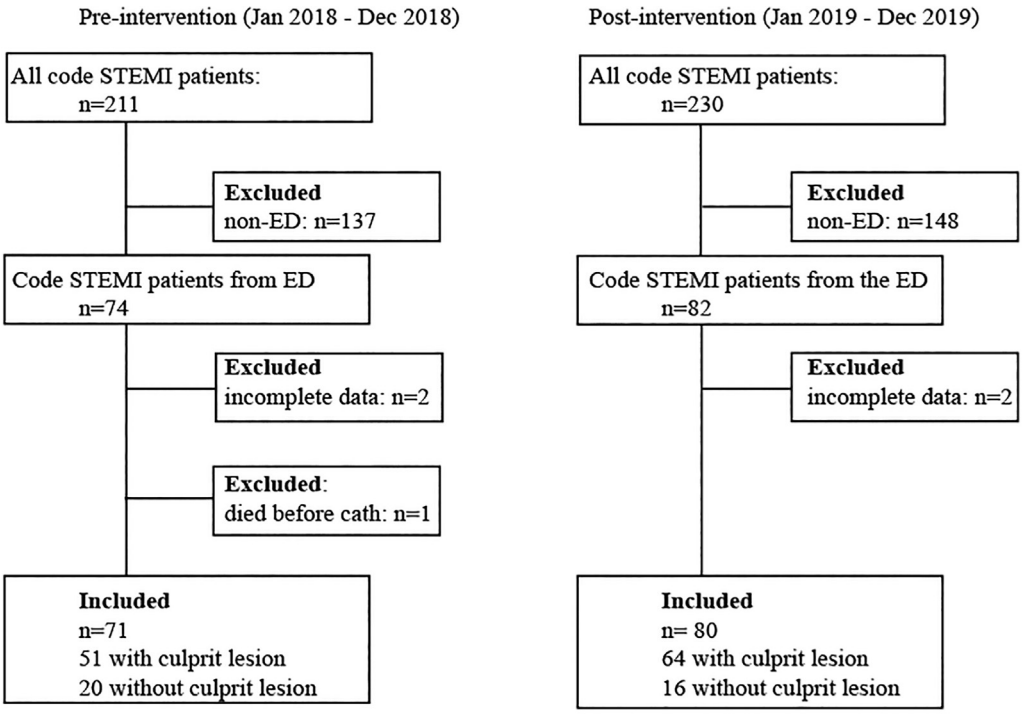


Fig. 1. Flow diagram of excluded and included patients.

Our outcome measure, median ETA time for Code STEMI patients with culprit lesions, decreased by 20 min—from 28.0 (95% confidence interval [CI] 15.0–45.0) to 8.0 (95%CI 6.0–15.0). Table 2 presents the comparison of culprit lesions between the baseline and intervention periods, with no differences found.

Fig. 2 presents the run chart of median monthly ETA time. It demonstrates that the reduction in ETA time coincided with the start of the intervention and achieved a lower median ETA time. A process change was further noted by the change in the centerline.

The website was launched with 15 posts based on the grand rounds presentation. This was followed by 40 weekly posts from January to November. Regarding our process measures, the website garnered 2816 views (70.4/week) and 1107 unique visitors (27.7/week). This was in a group of 80 physicians, i.e. approximately a third of the group each week. Fig. 3 demonstrates the website views and unique visitors. The most common reasons for improved ECG diagnosis of acute coronary occlusion was subtle LAD occlusion (e.g. borderline anterior ST elevation with convex ST segments, hyperacute T waves or reciprocal changes), subtle inferior MI (minor inferior ST elevation with reciprocal ST depression in aVL), and posterior MI (anterior ST depression with minor posterior ST elevation). End each of these was reinforced in multiple blog posts.

For our balancing measure, there was no change in the percentage of Code STEMIs without culprit lesions: baseline of 28.2% (95% confidence interval [CI] 17.8 to 38.6) and 20.0% (95%CI 11.2 to 28.8) during the intervention period.

Table 1
Baseline data for emergency department Code STEMI patients.

	Baseline, $n = 71$ (%)	Intervention, $n = 80$ (%)	*p*-value
Age, median (IQR)	64.0 (53.0–72.0)	64.0 (54.5–73.0)	
Sex	61 (85.9%) male	61 (76.3%) male	0.01
Risk factors			
- prior CAD	15 (21.1%)	12 (15.0%)	0.18
- diabetes	18 (25.4%)	25 (31.3%)	0.23
- hypertension	33 (46.5%)	40 (50%)	0.53
- dyslipidemia	21 (29.6%)	26 (32.5%)	0.57
- smoking	10 (14.1%)	15 (18.8%)	0.23
Chief complaint			
- Cardiac arrest	5 (7.0%)	10 (12.5%)	0.06
- Chest pain	55 (77.7%)	55 (68.8%)	0.06
- Anginal equivalent	11 (15.5%)	15 (18.8%)	0.42
Arrival by ambulance	29 (40.8%)	36 (45.0%)	0.50
ECG-to-Activation time, median (IQR)	9.0 (0–21.0)	10.0 (0–21.5)	
First ED ECG labeled "STEMI"	35 (49.3%)	39 (48.8%)	0.92

STEMI, ST-Elevation Myocardial Infarction; IQR, inter-quartile range; CAD, coronary artery disease; ED, emergency department.

4. Discussion

Our strategy of using education and weekly web-based feedback to all emergency physicians was associated with a reduction in ETA time by 20 min, which is a clinically significant value. [39-43] This was accomplished without increasing the percentage of Code STEMIs without culprit lesions. This demonstrates how ETA time can be used as a metric to guide quality improvement specific to emergency physicians, and

Table 2
Comparison of culprit lesions

Degree of occlusion (%)	Baseline (51 patients) n (%)	Intervention (64 patients) n (%)	*p*-value
100	28 (54.9%)	32 (50%)	0.43
99	8 (15.7%)	8 (12.5%)	0.48
95	7 (13.7%)	11 (17.2%)	0.42
90	6 (11.8%)	9 (14.1%)	0.57
<90	2 (3.9%)	4 (6.3%)	0.34

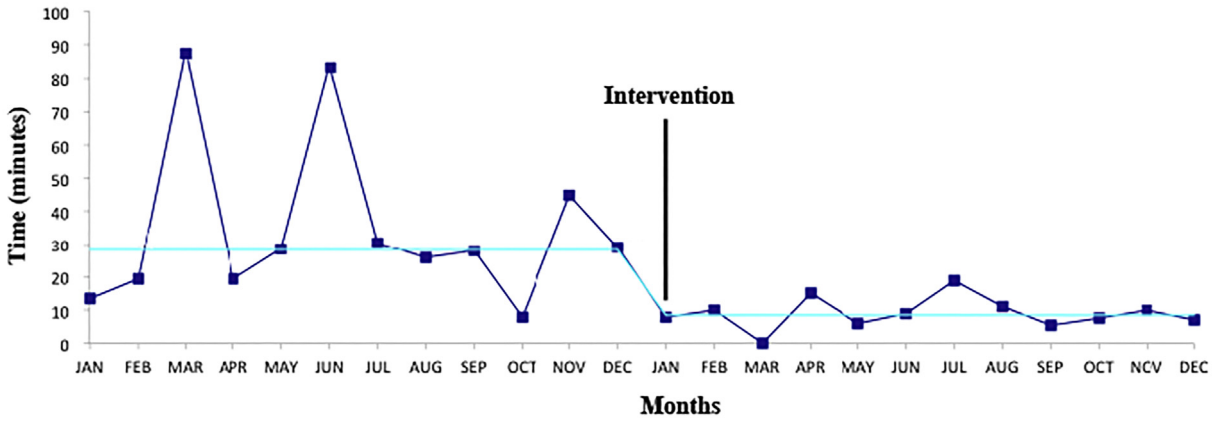


Fig. 2. ECG-to-Activation time.

how audit and feedback on new ECG insights can be implemented locally to reduce reperfusion delay without increasing unnecessary cath lab activation.

In the baseline group 54.9% of culprit lesions had 100% occlusion, corresponding to TIMI 0 flow, which is similar to other studies [44-46]. There was no difference in the intervention group in terms of the percentage of patients with total or subtotal (99%) occlusions. This suggests that the intervention did not broaden cath lab activation to "NSTEMI" with non-occlusive culprit lesions (Non-Occlusive MI), but maintained a focus on those with occlusive MI. In other words, cath lab activation was both faster and accurate, which we posit is related to improved ECG interpretation based on our intervention.

Audit and feedback has been recognized as a strategy to reduce delays to reperfusion [47,48] and added to AHA/ACC STEMI guidelines. [2] But audit and feedback initiatives, either as part of multiple strategies [49-54] or in isolation [55-58] have restricted themselves to classic STEMI criteria, provided feedback to the entire healthcare team, and focused on overall door-to-balloon times. Our study was novel in that it directed feedback exclusively to emergency physicians as a group, provided education beyond classic STEMI criteria to include newer ECG insights into acute coronary occlusion, and assessed ETA time as the quality metric.

Standardization and automation are higher on the hierarchy of intervention effectiveness than feedback and education. [59] But given that standard STEMI criteria miss a quarter of acute coronary occlusions [8] and automated interpretation has been shown to be inaccurate, [16] education and feedback must assume greater importance. A recent meta-analysis of physician ECG interpretation found suboptimal accuracy across all practice levels, and suggested novel education strategies. [60] This is especially important for the time-sensitive diagnosis of acute coronary occlusion. Given our context of a diverse group of 80 ED physicians who only activate the cath lab approximately 80 times a year, individual feedback may only happen on an annual basis. This is too infrequent to be meaningful. Instead, this project shared all challenging cases with all ED physicians to learn from each other's cases, including rapid and delayed diagnosis in addition to unnecessary cath lab activation.

Despite the existence of well-known websites for ECG education which inspired this project (ECG Weekly: https://ecgweekly.com; Dr. Smith's ECG Blog: http://hqmeded-ecg.blogspot.com), interest in our

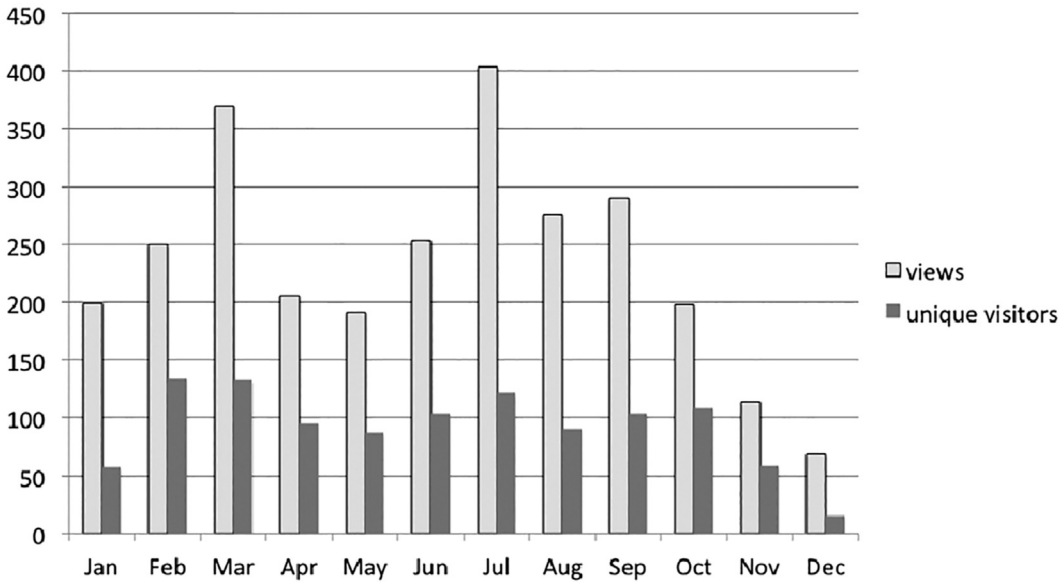


Fig. 3. Website views and unique visitors over time.

own website was maintained throughout the intervention period in part because it used local cases, and it coincided with reduced ETA times. These ECG cases are now being shared on an emergency medicine website (ECG Cases: https://emergencymedicinecases.com/blogs/ecg-cases/) as Free Open Access Medical Education.

4.1. Study limitations

There may have been unmeasured clinical variables that contributed to the reduction in ETA time. Additionally, ETA time improvement may also have been affected by the Hawthorne effect, as passive observation can reduce door-to-balloon time [64]. However, the sustained engagement with the website and the improved speed and accuracy of cath lab activation, without increasing unnecessary cath lab activations, suggests the intervention played a positive role.

Our intervention may have been associated with a change in the type of culprit lesion, as we analyzed percentage of occlusion rather than flow. Occlusion MI is defined as occlusion or near occlusion of a major coronary artery with insufficient collateral circulation, and it is measured as TIMI 0–2 flow [31]. As this metric is not routinely recorded in our institution's cath lab reports, it is possible that our intervention was associated with a shift to more non-occlusive culprit lesions. However, there was no difference in the percentage of culprit lesions with 100% occlusion (corresponding to TIMI 0 flow) and no difference in the percentage with subtotal occlusion, the two groups which constituted the majority of cases and which are the culprit lesions of greatest concern.

Potential limitations to generalizability include the time and effort to review cath lab activations and curate collective feedback along with literature, or interest in new ECG insights outside of academic centres. On the whole, however, this project made use of resources readily available to any ED.

5. Conclusions

Our QI initiative using weekly ECG audit and feedback was associated with a reduction in ETA time of 20 min for Code STEMI patients with culprit lesions, without increasing the percentage of Code STEMIs without culprit lesions. Reviewing local ETA times and providing group feedback on ECG interpretation including STEMI-equivalents and subtle occlusions could be expanded to other settings and may help set new standards of care.

Author contributions

JTTM developed the study concept/design, data acquisition and analysis, and contributed to data interpretation, drafting of the manuscript and critical revisions.

AKT, MK and SLY contributed to data interpretation and critical revisions.

LBC contributed to data interpretation, manuscript drafting and revisions.

Prior presentations

Best abstract in Quality Improvement and Patient Safety, Canadian Association of Emergency Physicians annual conference 2020.

Funding sources/disclosures

All authors report no conflict of interest.

Declaration of Competing Interest

The authors declare that they have no conflict of interest.

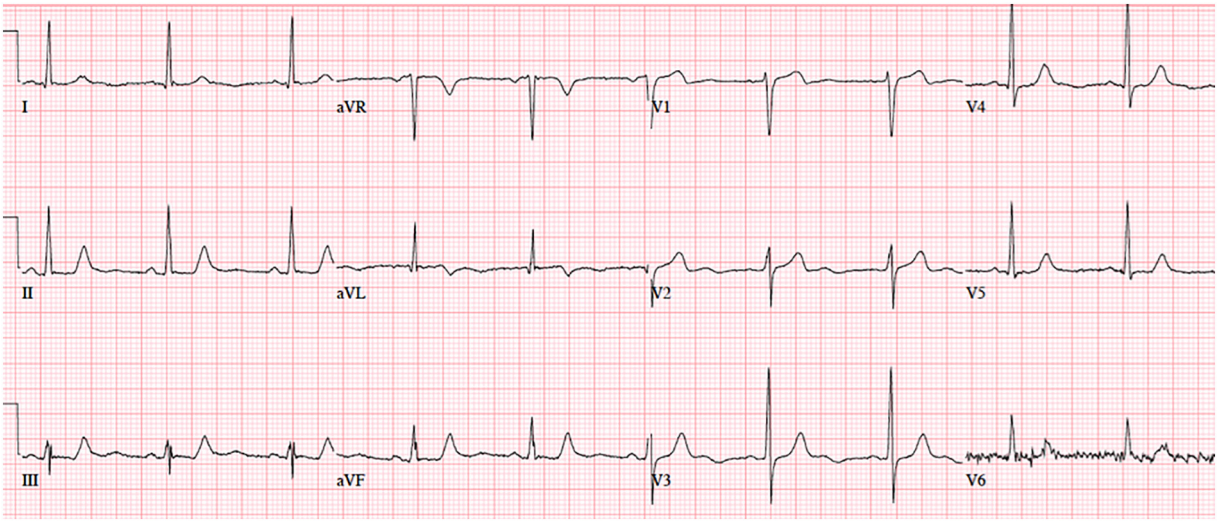
Acknowledgments

We would like to thank Stephen Chan for supporting the data acquisition for this project; and Dr. Olivia Ostrow and Dr. Brian Wong for supervising the Excellence in Quality Improvement Certificate Program (EQUIP), through which this project originated.

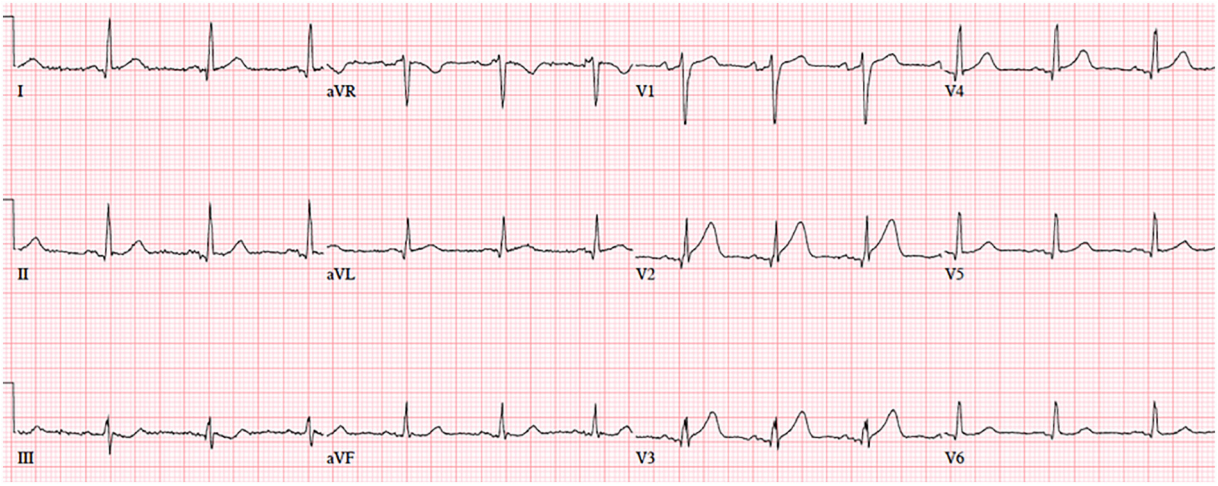
Appendix A. Example of weekly blog post shared with emergency physicians

A.1. Acute coronary occlusion with "normal" ECG

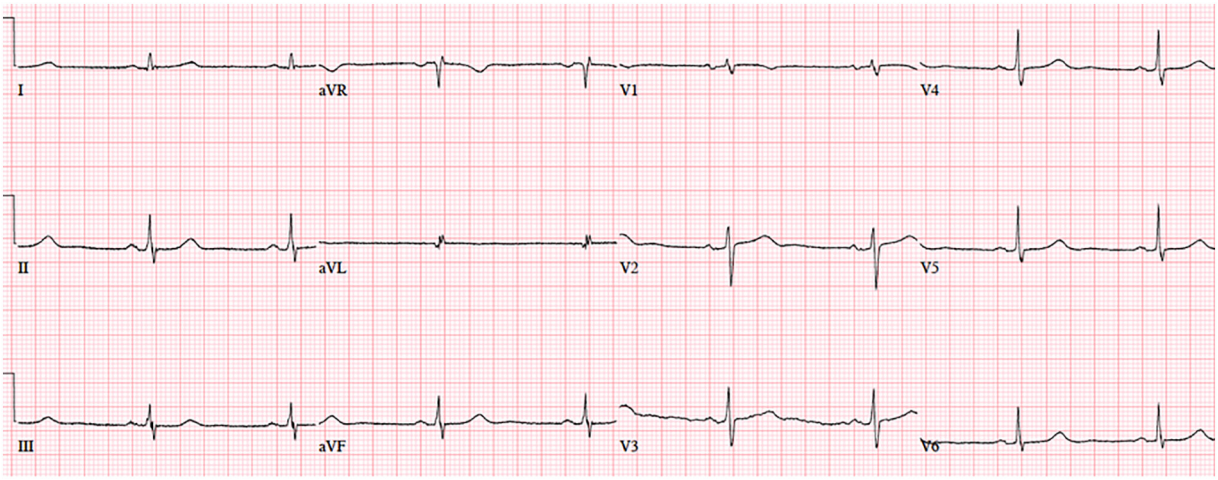
The following five patients presented with ischemic symptoms and an ECG labeled "normal" by automated interpretation.
Case 1: 50yo with 3 h of mid-sternal squeezing chest pain and diaphoresis

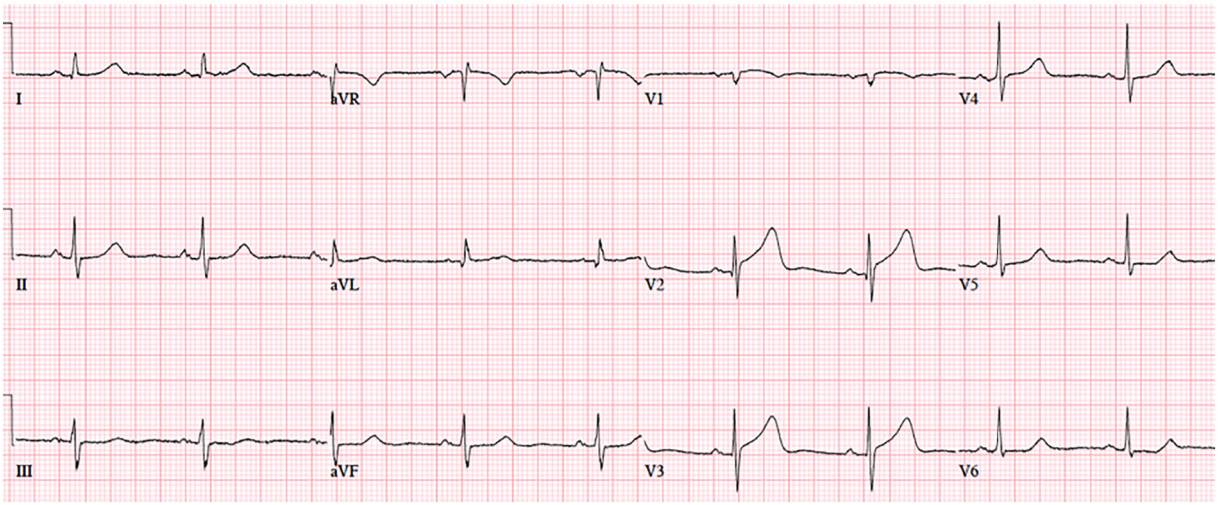


Case 2: 70yo with 90 min of exertional chest pain and nausea

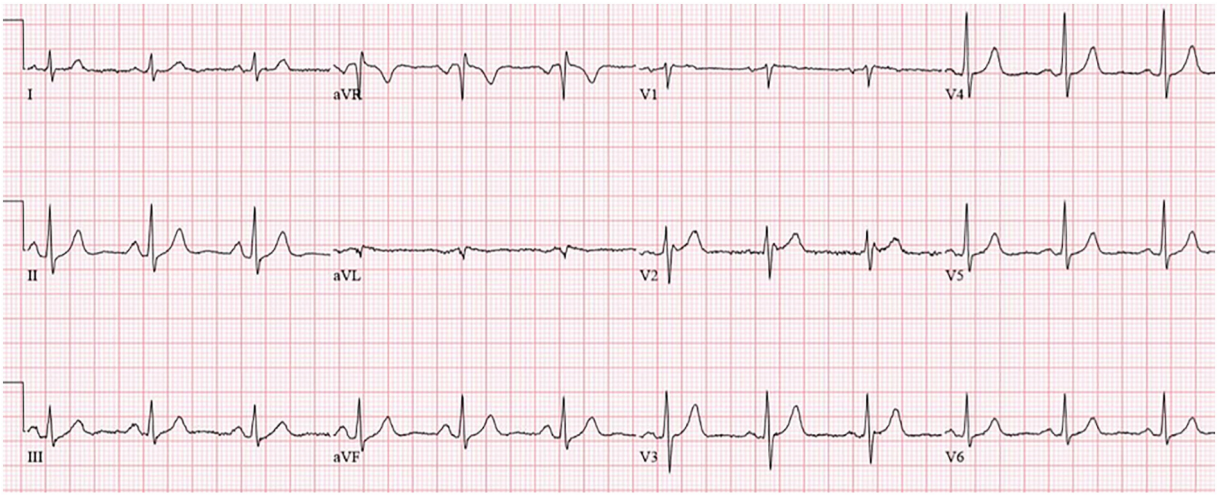


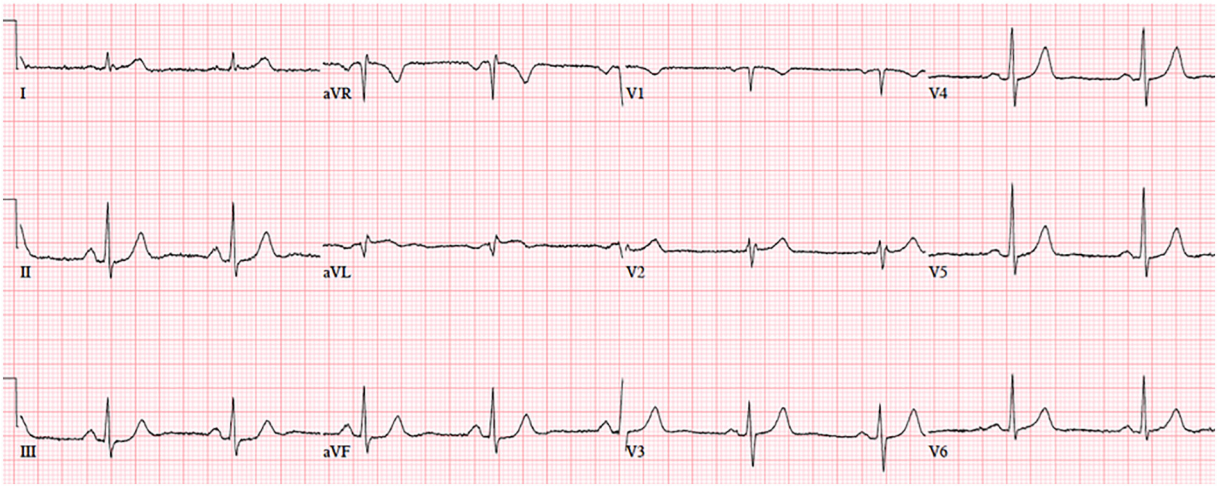
Case 3: 70yo with 6 h on/off chest tightness, now constant. Old then new ECG



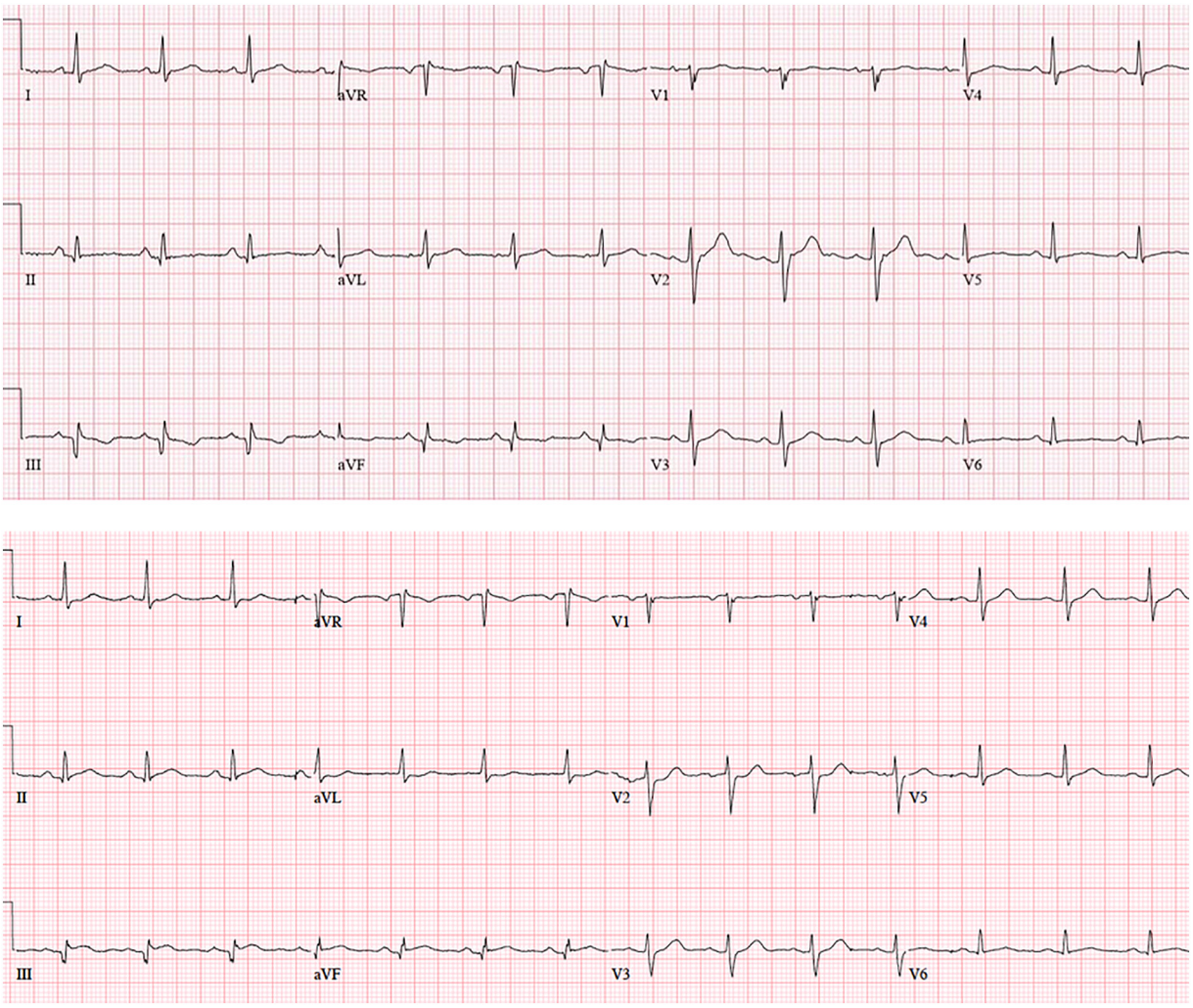


Case 4: 75yo with one day chest pain radiating to bilateral shoulders. Old then new ECG





Case 5: 55yo prior RCA stent with 30 min chest pain. Old then new ECG



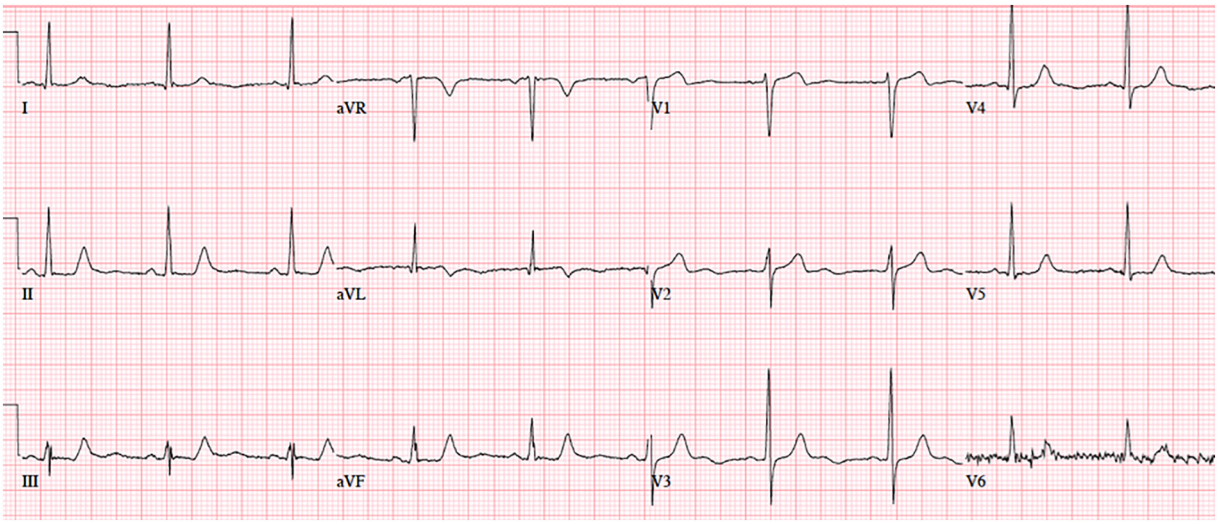
A.2. ECGs labeled "normal" by automated interpretation

Automated interpretation has high rates of error for detecting ischemia, up to 42% for STEMI [61]. But what about those ECGs read as "normal"? A 2017 study concluded that "triage ECGs identified by the computer as normal are unlikely to have clinical significance that would change triage care. Eliminating physician review of triage ECGs with a computer interpretation of normal may be a safe way to improve patient care by decreasing physician interruptions" [62]. But this was based on only 4 months of triage ECGs collected at a single centre with a low incidence of STEMI.

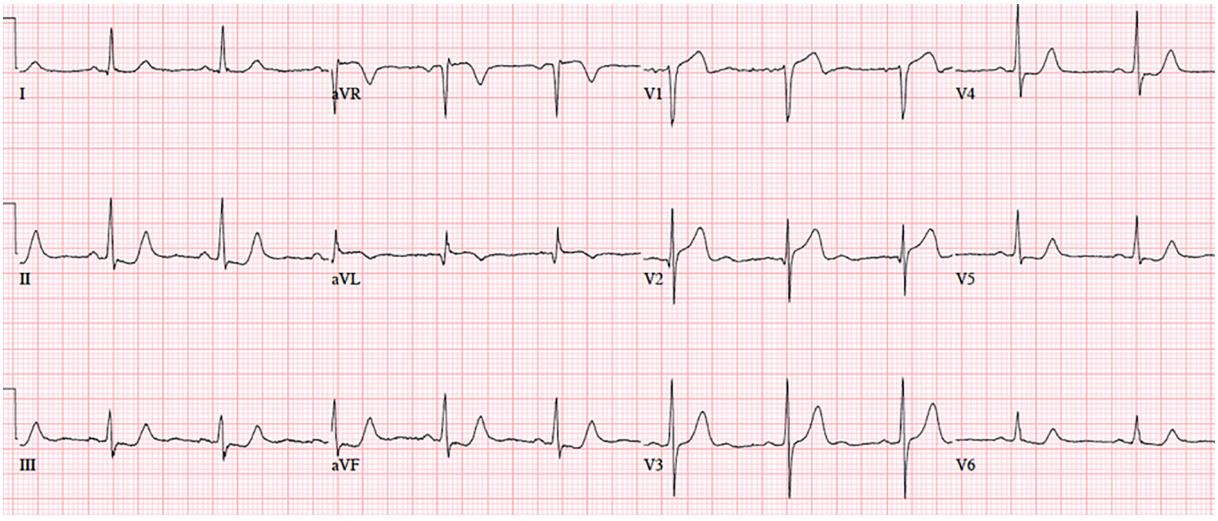
A critical response explained both the shortcomings of the study, and a different approach to "normal" ECGs: "Using non-blinded expert review rather than outcome as a reference standard precludes any meaningful conclusion about triage ECGs…A sample size of 855 has no chance of generating a meaningful conclusion about the reliability of computer 'Normal.'…Physicians should take steps to develop their skills in detecting subtle signs of myocardial ischemia, and computerized interpretation algorithms should state 'No abnormalities detected' rather than 'Normal ECG'" [63].

A.3. Back to the cases

Case 1: ischemic morphology

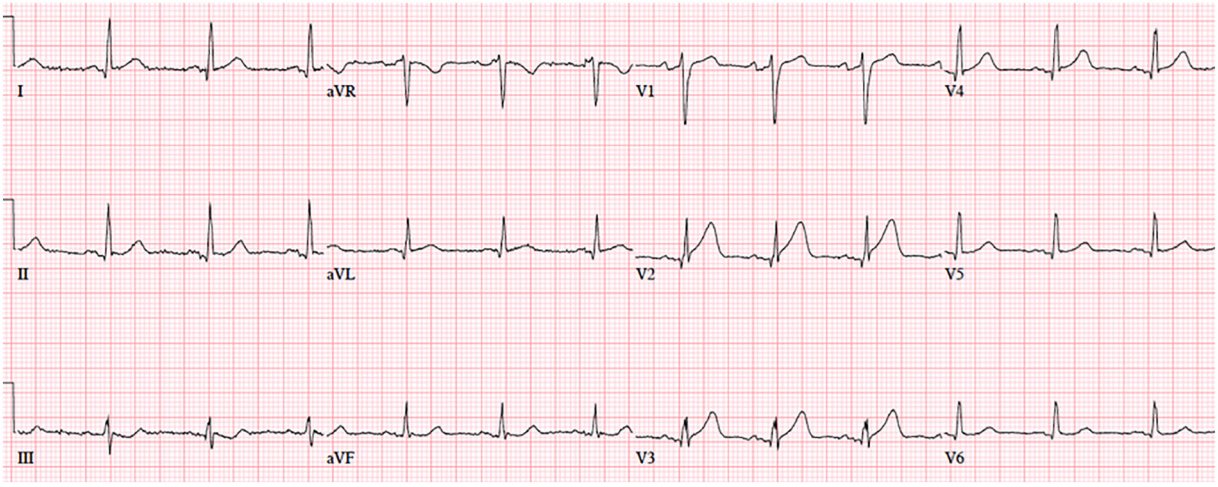


NSR, normal conduction/axis/R wave, no hypertrophy. No ST segment elevation or depression, but there is terminal T-wave inversion in aVL and reciprocal down-up T waves in III/III/aVF, and inverted U wave in V2–3. The physician was concerned about these and asked for a repeat ECG:

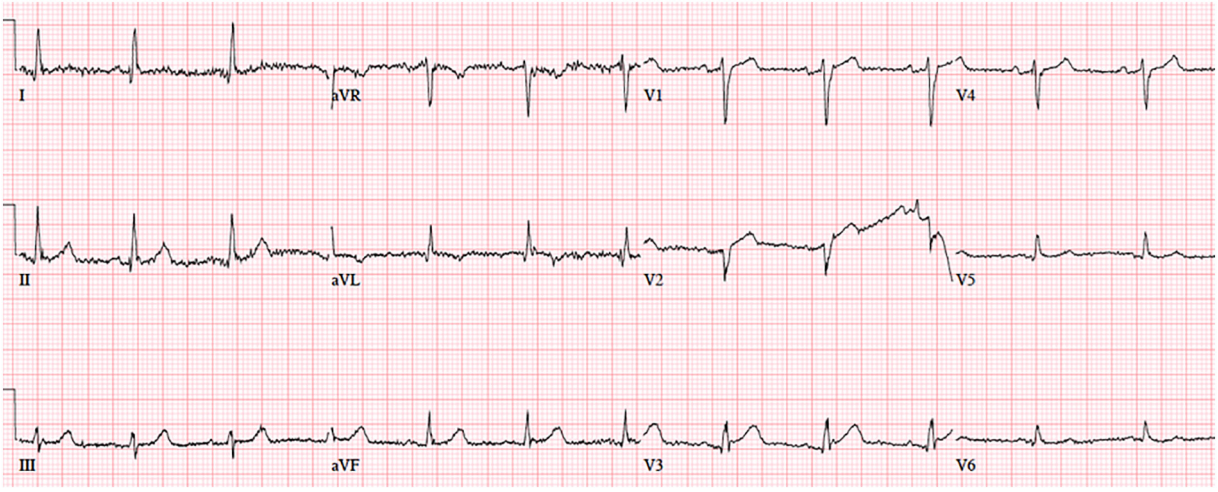


Now there is ST elevation in V1–2 and aVL, a deeper Q wave in aVL and new Q wave in V2, and ST depression inferolaterally. Code STEMI called: 95% proximal LAD occlusion, peak troponin $I = 12{,}000$. ETA 60 min.

Case 2: subtle LAD occlusion

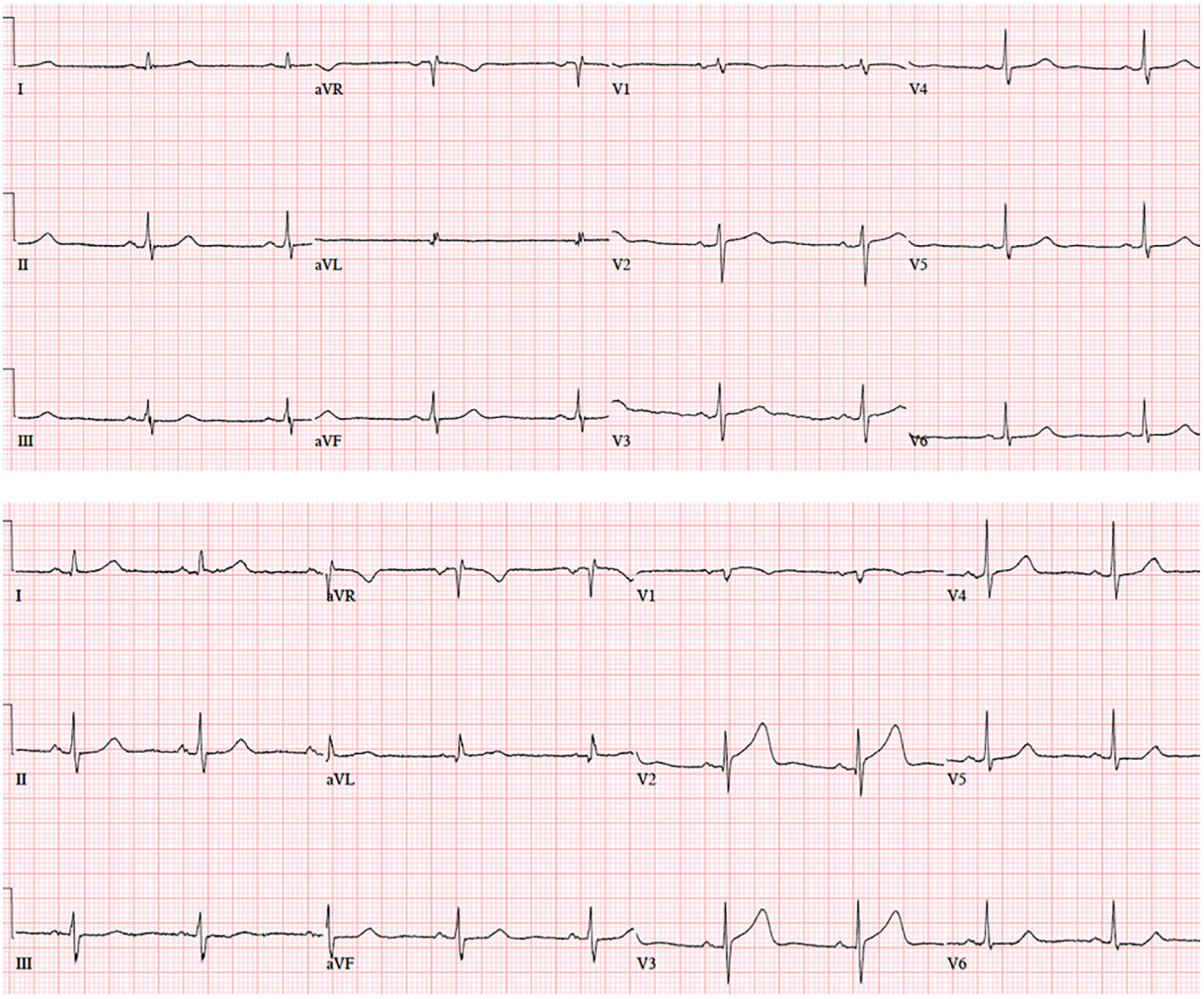


NSR, normal conduction/axis, no hypertrophy. There is 1–2 mm concave ST elevation in V2–3 which could be normal, but there are a number ischemic changes: reverse R wave progression from V2 to V3, Q wave in V2, terminal QRS distortion (no S wave or J wave) in V2, hyperacute T waves (relative to their preceding QRS) in V2–3, and inferior reciprocal changes. When Trop I came back at 100, a repeat ECG was done:



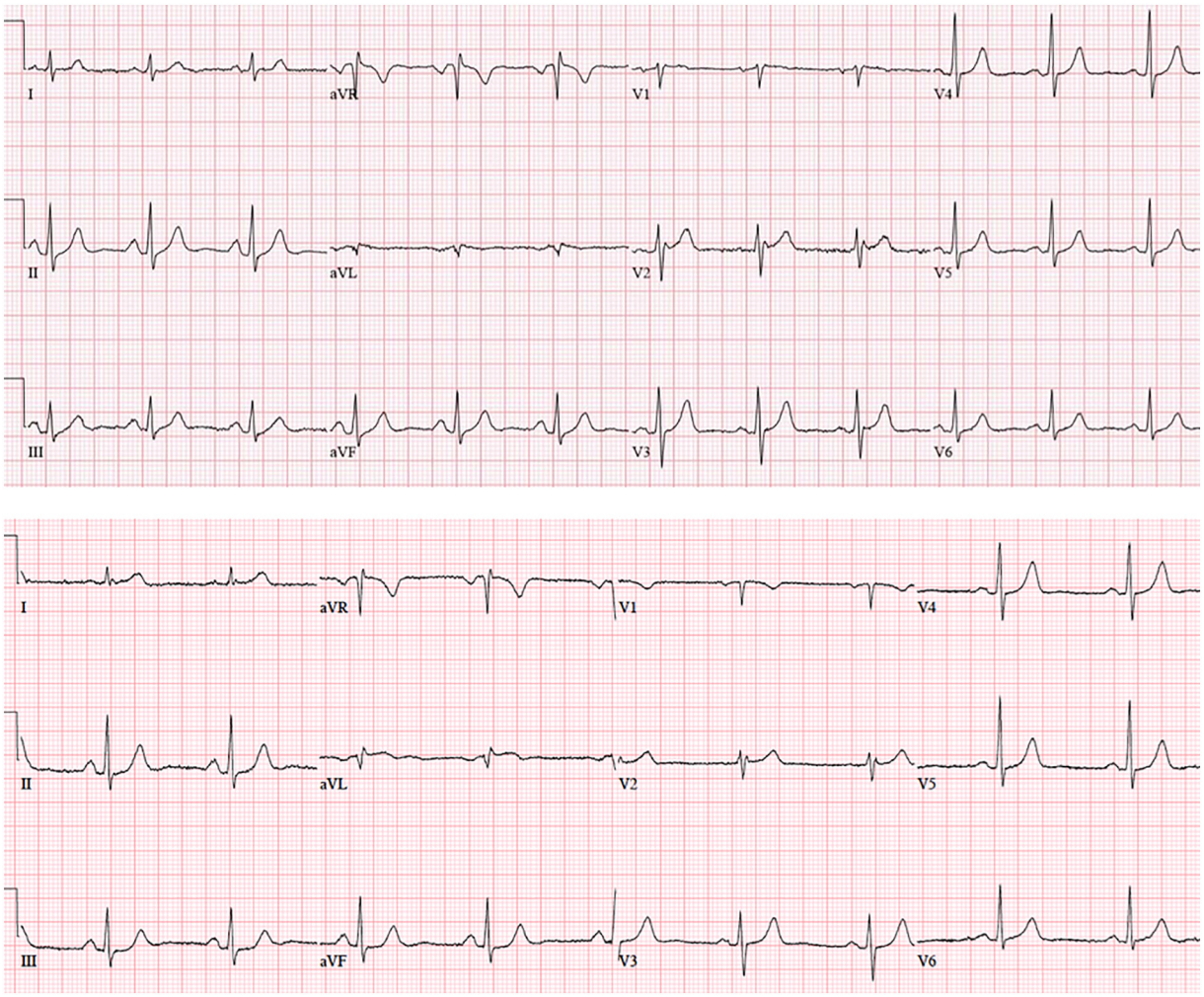
Now there is a full QS wave in V2. Code STEMI called: 100% mid-LAD occlusion. Peak trop $I = 8000$. ETA 116 min.

Case 3: ST/T changes compared to prior



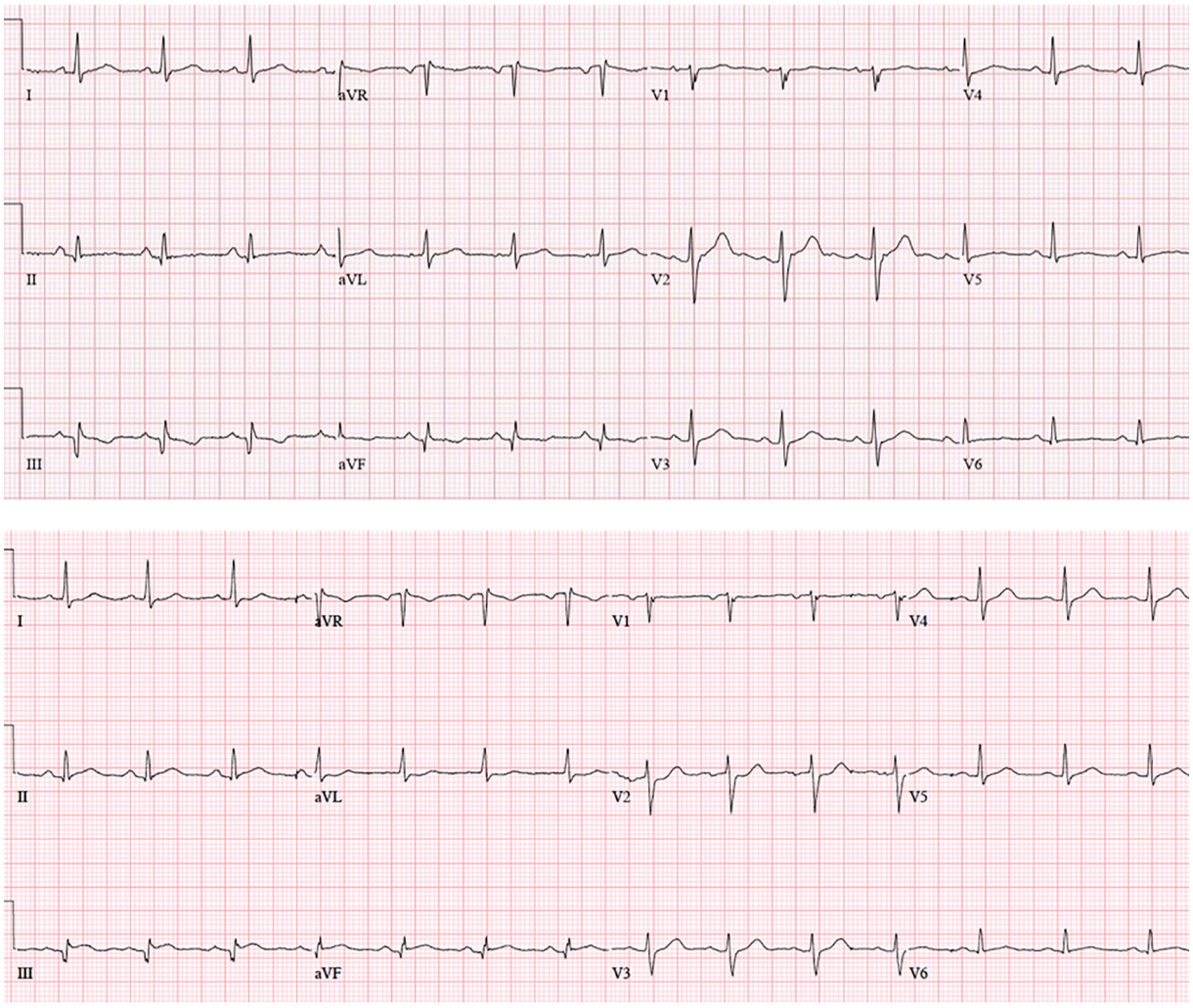
There's mild concave ST elevation in lead V2–3 which doesn't meet STEMI criteria. But this is new compared to previous, along with hyperacute T waves in V2–3 and mild ST depression laterally. When troponin I came back at 600, the cath lab was activated: 100% distal LAD occlusion, peak trop 50,000. ETA 78 min.

Patient 4: ST elevation relative to the QRS



There's barely 1 mm of ST elevation in aVL only (no STEMI criteria), but this is large compared to its small QRS complex, and there's mild reciprocal ST depression inferiorly, and pseudonormalized ST segment in V3. Concerned about the patient's symptoms and the new ischemic changes, the physician activated the cath lab: 99% occlusion of first obtuse marginal branch of circumflex. First troponin I negative, peak 13,000. ETA 26 min.

Case 5: pseudonormalization



NSR, normal conduction/axis, R wave, no hypertrophy. There's an old Q wave from the prior MI. But there is now mild ST elevation in III/AVF with reciprocal ST depression in aVL (which is very sensitive for inferior MI), the inverted T waves in III/aVF are now upright (pseudonormalization), and there is relative ST depression (pseudonormalization) in V2—all concerning for inferoposterior MI. Cath lab activated: 100% RCA stent occlusion, peak Troponi$n = 8000$. ETA 15 min.

Take-home points

Never trust the ECG computer interpretation, even if it says “normal,” because:

1. ischemic morphology: the computer focuses on ST segment elevation, and can miss ischemic ST-T wave morphology—including straight or convex ST segments, terminal T wave inversion, down-up T waves, hyperacute T waves, and inverted U waves
2. dynamic change: the computer interprets each ECG in isolation, and can't compare to prior or repeat ECGs (which is critical in a dynamic process like coronary occlusion)—so it can miss subtle changes, including pseudonormalization of ST segments or T waves
3. STEMI criteria: computer interpretation is based on STEMI criteria, which has limited sensitivity for identifying acute coronary occlusion—so it can miss subtle ST elevation (which may be significant in small amplitude QRS complexes), ST depression in aVL (which is very sensitive for inferior MI), and subtle signs of LAD occlusion like terminal QRS distortion.

References

[1] Bradley EH, Nallamothu BK, Herrin J, et al. National efforts to improve door-to-balloon time: results from the door-to-balloon alliance. J Amer Coll Cardiol. 2009 Dec;54(25):2423–9.

[2] 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. Circulation. 2013 Jan;127(4):e362–425.

[3] McCabe JM, Armstrong EJ, Hoffmayer KS, et al. Impact of door-to-activation time in primary percutaneous coronary intervention for ST-segment elevation myocardial infarctions: a report from the Activate-SF registry. Circ Cardiovasc Qual Outcomes. 2012 Sept 1;5(5):672–9.

[4] Chhabra S, Eagles D, Kwok ESH, et al. Interventions to reduce emergency department door-to-electrocardiogram times: a systematic review. CJEM. 2019;21(5):607–17.

[5] Tsai CL, Magid DJ, Sullivan AF, et al. Quality of care for acute myocardial infarction in 58 U.S. emergency departments. Acad Emerg Med. 2010 Sep;17(9):940–50.

[6] Dixon WC, Wang TY, Dai D, et al. Anatomic distribution of the culprit lesion in patients with non-ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention: findings from the National Cardiovascular Data Registry. J Am Coll Cardiol. 2008;52(16):1347–8.

[7] Wang TY, Zhang M, Fu Y, et al. Incidence, distribution, and prognostic impact of occluded culprit arteries among patients with non-ST-elevation acute coronary syndromes undergoing diagnostic angiography. Am Heart J. 2009;157(4):716–23.

[8] Khan AR, Golwala H, Tripathi A, et al. Impact of total occlusion of culprit artery in acute non-ST elevation myocardial infarction: a systematic review and meta-analysis. Eur Heart J. 2017 Nov 1;38(41):3082–9.

[9] Hung CS, Chen YH, Huang CC. Prevalence and outcome of patients with non-ST segment elevation myocardial infarction with occluded 'culprit' artery—a systematic review and meta-analysis. Crit Care. 2018;22:34.

[10] Hillinger P, Strebel I, Abächerli R, et al. Prospective validation of current quantitative electrocardiographic criteria for ST-elevation myocardial infarction. Int J Cardiol. 2019;292:1–12.

[11] From AM, Best PJ, Lennon RJ, et al. Acute myocardial infarction due to left circumflex artery occlusion and significance of ST-segment elevation. Am J Cardiol. 2010;106(8):1081–5.

[12] Waldo SW, Brenner DA, Li S, et al. Reperfusion times and in-hospital outcomes among patients with an isolated posterior myocardial infarction: insights from the National Cardiovascular Data Registry (NCDR). Am Heart J. 2014;167(3):350–4.

[13] Marti D, Mestre JL, Salido L, et al. Incidence, angiographic features and outcomes of patients presenting with subtle ST elevation myocardial infarction. Am Heart J. 2014 Dec;168(6):884–90.

[14] Garvey JL, Zegre-Hemseu J, Gregg R, et al. Electrocardiographic diagnosis of ST segment elevation myocardial infarction: an evaluation of three automated interpretation algorithms. J Electrocardiol. 2016;49(5):728–32.

[15] Mawri S, Michaels A, Gibbs J, et al. The comparison of physician to computer interpreted electrocardiograms on ST-elevation myocardial infarction door-to-balloon times. Crit Pathw Cardiol. 2016;15(1):22–5.

[16] Schlapfer J, Wellens HJ. Computer-interpreted electrocardiogram: benefits and limitations. J Am Coll Cardiol. 2017 Aug 29;70(9):1183–92.

[17] Martinez-Losas P, Higueras J, Gomez-polo JC, et al. The influence of computerized interpretation of an electrocardiogram reading. Am J Emerg Med. 2016;34(10):2031–2.

[18] Rokos IC, French W, Mattu A, et al. Appropriate cardiac cath lab activation: optimizing electrocardiogram interpretation and clinical decision-making for acute ST-elevation myocardial infarction. Am Heart J. 2010 Dec;160(6):995–1003.

[19] Lawner BJ, Nable JS, Mattu A. Novel patterns of ischemia and STEMI equivalents. Cardiol Clin. 2012 Nov;30(4):591–9.

[20] Macias M, Peachey J, Mattu A, et al. The electrocardiogram in the ACS patient: high-risk electrocardiographic presentations lacking anatomically oriented ST-segment elevation. Am J Emerg Med. 2016;34(3):611–7.

[21] Wall J, White LD, Lee A. Novel ECG changes in acute coronary syndromes. Would improvement in the recognition of 'STEMI-equivalents' affect time until reperfusion? Intern Emerg Med. 2018;13(2):243–9.

[22] Klein LR, Schroff GT, Beeman W, et al. Electrocardiographic criteria to differentiate acute anterior ST-elevation myocardial infarction from left ventricular aneurysm. Am J Emerg Med. 2015;33:786–90.

[23] Bishof JE, Worrall C, Thompson P, et al. ST depression in lead aVL differentiates inferior ST-elevation myocardial infarction from pericarditis. Am J Emerg Med. 2016 Feb;34(2):149–54.

[24] Driver BE, Khalil A, Henry T, et al. A new 4-variable formula to differentiate normal variant ST segment elevation in V2-V4 (early repolarization) from subtle left anterior descending coronary occlusion – adding QRS amplitude of V2 improves the model. J Electrocardiol. 2017;50(5):561–9.

[25] Meyers HP, Limkakeng Jr AT, Jaffa EJ, et al. Validation of the modified Sgarbossa criteria for acute coronary occlusion in the setting of left bundle branch block: a retrospective case-control study. Am Heart J. 2015 Dec;170(6):1255–64.

[26] Phibbs B, Nelson W. Differential classification of acute myocardial infarction into ST- and non-ST segment elevation is not valid or rational. Ann Noninvasive Electrocardiol. 2010;15(3):191–9.

[27] Birnbaum I, Birnbaum Y. High risk ECG patterns in ACS—need for guideline revision. J Electrocardiol. 2013 Nov-Dec;46(6):535–9.

[28] Gorgels APM. ST-elevation and non-ST-elevation acute coronary syndromes: should the guidelines be changed? J Electrocardiol. 2013;46:318–23.

[29] Meyers HP, Smith SW. Prospective, real-world evidence showing the gap between ST elevation myocardial infarction (STEMI) and occlusion MI (OMI). Int J Cardiol. 2019;293:48–9.

[30] Aslanger EK, Yıldırımtürk Ö, Şimşek B, et al. DIagnostic accuracy oF electrocardiogram for acute coronary OCClUsion resuLTing in myocardial infarction (DIFOCCULT Study). Int J Cardiol Heart Vasc. 2020 Jul 30;30:100603.

[31] Meyers HP, Bracey A, Lee D, et al. Comparison of the ST-Elevation Myocardial Infarction (STEMI) vs. NSTEMI and Occlusion MI (OMI) paradigms of acute MI. J Emerg Med. 2021 Mar;60(3):273–84 [Online first].

[32] Nfor T, Kostopoulos L, Hashim H, et al. Identifying false-positive ST-elevation myocardial infarction in emergency department patients. J Emerg Med. 2012 Oct;43(4):561–7.

[33] McCabe JM, Armstrong EJ, Kulkarni A, et al. Prevalence and factors associated with false-positive ST-segment elevation myocardial infarction diagnoses at primary percutaneous coronary intervention–capable centers: a report from the Activate-SF registry. Arch Intern Med. 2012 Jun 11;172(11):864–71.

[34] Chung SL, Lei M-H, Chen C-C, et al. Characteristics and prognosis in patients with false-positive ST-elevation myocardial infarction in the ED. Am J Emerg Med. 2013 May;31(5):825–9.

[35] Wong CK. Minimizing false activation of cath lab for STEMI—a realistic goal? Int J Cardiol. 2014;172(1):e91–3.

[36] Lange DC, Conte S, Pappas-Block E, et al. Cancellation of the cardiac catheterization lab after activation for ST-segment-elevation myocardial infarction. Circ Cardiovasc Qual Outcomes. 2018 Aug;11(8):e004464.

[37] McLaren JTT, Kapoor M, Yi SL, et al. Using ECG-to-activation time to assess emergency physician diagnostic time for acute coronary occlusion. J Emerg Med. 2020;S0736–4679(20):30964-1.

[38] 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 Feb;34(2):132–45.

[39] De Luca G, Suryapranata H, Ottervanger JP, et al. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. Circulation. 2004 Mar 16;109(10):1223–5.

[40] Rathore SS, Curtis JP, Chen J, et al. Association of door-to-balloon time and mortality in patients admitted to hospital with ST elevation myocardial infarction: national cohort study. BMJ. 2009 May 19;338:b1807.

[41] Scholz KH, Maier SKG, Maier LS, et al. Impact of treatment delay on mortality in ST-segment elevation myocardial infarction (STEMI) patients presenting with and without haemodynamic instability: results from the German prospective, multicenter FITT-STEMI trial. Eur Heart J. 2018 Apr 1;39(13):1065–74.

[42] Mentias A, Raza MQ, Barakat AF, et al. Effect of shorter door-to-balloon times over 20 years on outcomes of patients with anterior ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Am J Cardiol. 2017 Oct 15;120(8):1254–9.

[43] Park J, Choi KH, Lee JM, et al. Prognostic implications of door-to-balloon time and onset-to-door time on mortality in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. J Am Heart Assoc. 2019 May 1;8(9):e012188.

[44] Joost A, Stiermaier T, Eitel C, et al. Impact of initial culprit vessel flow on infarct size, microvascular obstruction, and myocardial salvage in acute reperfused ST-elevation myocardial infarction. Am J Cardiol. 2016 Nov 1;118(9):1316–22.

[45] Bailleul C, Aissaoui N, Cayla G, et al. Prognostic impact of percutaneous coronary intervention TIMI flow in patients with ST-segment and non-ST-segment elevation myocardial infarction: results from the FAST-MI 2010 registry. Arch Cardiovasc Dis. 2018 Feb;111(2):101–8.

[46] Hashimoto T, Ako J, Nakao K, et al. Pre-procedural thrombolysis in myocardial infarction flow in patients with ST-segment elevation myocardial infarction. Int Heart J. 2018 Sep 26;59(5):920–5.

[47] Bradley EH, Nallamothu BK, Curts JP, et al. Summary of evidence regarding hospital strategies to reduce door-to-balloon times for patients with ST-segment elevation. Crit Pathw Cardiol. 2007 Sep;6(3):91–7.

[48] Bahiru E, Agarwal A, Berendsen MA, et al. Hospital-based quality improvement interventions for patients with acute coronary syndrome: a systematic review. Circ Cardiovasc Qual Outcomes. 2019 Sep;12(9):e005513.

[49] Huang RL, Donelli A, Byrd J, et al. Using quality improvement methods to improve door-to-balloon time at an academic medical center. J Invasive Cardiol. 2008 Feb;20(2):46–52.

[50] Nestler DM, Noheria A, Haro LH, et al. Sustaining improvement in door-to-balloon tmie over 4 years: the Mayo Clinic ST-elevation myocardial infraction protocol. Circ Cardiovasc Qual Outcomes. 2009 Sept 1;2:508–13.

[51] Kelly EW, Kelly JD, Hiestand B, et al. Six sigma process utilization in reducing door-to-balloon time at a single academic tertiary care center. Prog Cardiovasc Dis. 2010 Nov-Dec;53(3):219–26.

[52] Camp-Rogers T, Kurz MC, Brady WJ. Hospital based strategies contributing to percutaneous coronary intervention time reduction in the patient with ST-segment elevation myocardial infarction: a review of "system-of-care" approach. Am J Emerg Med. 2012 Mar;30(3):491–8.

[53] Tödt T, Thylén I, Alfredsson J, et al. Strategies TO reduce time delays in patients with AcuTe coronary heart disease treated with primary PCI—the STOP WATCH study: a multistage action research project. BMJ Open. 2013;3(9):e003493.

[54] Pan MW, Chen WY, Chen CC, et al. Implementation of multiple strategies for improved door-to-balloon time in patients with ST-segment elevation myocardial infarction. Heart Vessel. 2014;29:142–8.

[55] Scholz KH, Hilgers R, Ahlersmann D, et al. Contact to balloon time and door to balloon time after initiation of a formalized data feedback in patients with acute ST-elevation myocardial infarction. Am J Cardiol. 2008 Jan 1;101(1):46–52.

[56] Lai C-L, Fan C-M, Liao P-C, et al. Impact of an audit program and other factors on door-to-balloon times in acute ST-elevation myocardial infarction patients destined for primary coronary intervention. Acad Emerg Med. 2009 Apr;16(4):333–42.
[57] Lin J-F, Hsu S-Y, Wu S, et al. Data feedback reduces door-to-balloon time in patients with st-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Heart Vessel. 2011 Jan;26(1):25–30.
[58] Scholz KH, Maier SK, Jung J, et al. Reduction in treatment times through formalized data feedback: results from a prospective multicenter study of ST-segment elevation myocardial infarction. JACC Cardiovasc Interv. 2012 Aug;5(8):848–57.
[59] Cafazzo J, St-Cyr O. From discovery to design: the evolution of human factors in healthcare. Healthc Q. 2012:24–9 15 Spec No.
[60] Cook DA, Oh S-Y, Pusic MV. Accuracy of physicians' electrocardiogram interpretations: a systematic review and meta-analysis. JAMA Intern Med. 2020 Sep;28: e203989.
[61] Shry EA, Eckart RE, Winslow JB, et al. Effect of monitoring of physician performance on door-to-balloon time for primary angioplasty in acute myocardial infarction. Am J Cardiol. 2003 Apr 1;91(7):867–9.
[62] Schläpfer J, Wellens HJ. Computer-interpreted electrocardiograms: benefits and limitations. J Am Coll Cardiol. 2017;70(9):1183–92.
[63] Hughes KE, Lewis SM, Katz L, Jones J. Safety of computer interpretation of normal triage electrocardiograms. Acad Emerg Med. 2017;24(1):120–4.
[64] Litell JM, Meyers HP, Smith SW. Emergency physicians should be shown all triage ECGs, even those with a computer interpretation of "Normal". J Electrocardiol. 2019;54:79–81.