Title
The Value of Continuous ST-Segment Monitoring in the Emergency Department

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The Practice Standards for Electrocardiographic Monitoring in Hospital Settings (Drew et al., 2004) recommend continuous ST-segment monitoring (C-STM) for 8 to 12 hours in combination with serum biomarker testing as a cost-effective strategy for determining the priority of treatment in patients who present to the emergency department (ED) with signs

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and/or symptoms of acute coronary syndrome (ACS). The ST-Segment Monitoring Practice Guideline International Working Group (Drew & Krucoff, 1999) also recommends C-STM for patients in the ED at risk for myocardial ischemia. In addition, C-STM has been assigned as a Class IIb recommendation by the American College of Cardiology (ACC)/American Heart Association (AHA) Task Force on Practice Guidelines for the Management of Patients with Non-ST-Elevation Acute Coronary Syndromes (Amsterdam et al., 2014) for patients in whom the initial electrocardiogram (ECG) is not diagnostic. The Task Force considered C-STM to be a reasonable alternative to serial 12-lead ECGs for ruling out myocardial ischemia in patients. A Class IIb recommendation signifies that although more studies on this technology are needed, the procedure may be considered, as its benefits far outweigh any risks involved in its use.

More than 8 million ED visits are associated with chest pain and other signs and symptoms of myocardial ischemia (Amsterdam et al., 2010). It is a chief manifestation of coronary heart disease, which caused one in every six deaths in the U.S. in 2009 (Mozaffarian et al., 2015). There are an estimated 635,000 new cases of myocardial infarction and 300,000 recurrent cases in the U.S. each year (Mozaffarian et al., 2015). In 2011, acute myocardial infarction was the fifth most expensive condition in U.S. hospitals, amounting to $11.5 billion in aggregate hospital costs and up to 612,000 hospital discharges (Torio & Andrews, 2013).

Emergency nurses are generally the first health care providers to evaluate patients presenting with signs and symptoms of ischemia or myocardial infarction. They are, therefore, largely responsible for determining the urgency with which patients receive medical attention, and for monitoring their condition while in the ED. Although bedside monitors are used in assessing ECG rhythm, vital signs, and other parameters in patients with clinical presentations suggestive of myocardial ischemia, surveys suggest that the C-STM feature is not commonly activated. In a survey among emergency nurses, Hart (2006) found that most (two-thirds) did not activate this feature on the bedside monitor. Anecdotal evidence suggests that nurses do not activate this feature because they do not perceive it as being easy to use. A survey conducted by Patton and Funk (2001) indicated that many nurses were not educated in its use and those who were complained that it generated unnecessary alarms. The results from another survey performed by Sandau, Sendelbach, Frederickson, and Doran (2010) suggested that many physicians lacked knowledge of published guidelines for its use.

Methods that identify ischemia at an early stage are essential for preserving myocardial tissue. ST-segment analysis software (introduced on bedside monitors in the 1980s) enables simultaneous assessment of ST-segment changes and patient presentation. C-STM, therefore, has the potential to affect health service delivery nationwide as it is widely available on bedside monitors in the U.S. (Drew & Krucoff, 1999; Sandau, Sendelbach, Frederickson, & Doran, 2010). Consequently, the study investigators hypothesized that implementation of 12-lead C-STM would reduce the time to diagnosis in the ED, which is the initial treatment point for most patients with signs and/or symptoms suggestive of myocardial ischemia, thereby, improving their outcomes. The purpose of this study was to evaluate the value of bedside 12-lead C-STM on the care and outcomes of ED patients with signs and/or symptoms suggestive of myocardial ischemia. The primary aim was to compare

the time to diagnosis of ACS, presumed ACS, or non-ACS, before and after implementation of C-STM. A secondary aim was to evaluate the diagnostic accuracy of C-STM in detecting ischemia and infarction (using stress tests and troponin I as reference tests). An exploratory aim was to compare the occurrence of 30-day adverse events (return to the ED with signs and/or symptoms of myocardial ischemia, all-cause hospital admission, acute myocardial infarction, use of reperfusion therapy, and all-cause mortality) before and after implementation of C-STM.

Methods

Study Design and Setting

This was a prospective quasi-experimental separate sample pre-post study (Campbell & Stanley, 1963) with 163 participants (pre-C-STM phase: n=78; C-STM phase: n=85). A sample size of 78 participants per study phase was targeted based on a 5% significance level and 80% power to detect a post-intervention reduction of 45 minutes in time to diagnosis. The study was conducted at a 338-bed teaching hospital (36 ED beds) in the Northeastern United States. General Electric (GE) (Solar ® 8000i) bedside monitors are used in the ED.

Participants

The principal investigator enrolled patients aged 18 years or older presenting with signs and/or symptoms of myocardial ischemia (e.g., chest pain; pain radiating to the jaw, back, or left arm; diaphoresis; and shortness of breath). Patients were excluded if they had:

- Initial ECG criteria for immediate reperfusion therapy—ECG evidence of acute myocardial infarction described as:

  - New or presumed new significant ST-T changes or new left bundle branch block

  - Development of pathological Q waves (Thygesen et al., 2012)

- Conditions that made it difficult to assess ST-segment deviations accurately, such as agitation, tachyarrhythmia (ventricular tachycardia, supraventricular tachycardia or rapid atrial fibrillation), and implanted pacemakers

- Inability to communicate in English or Spanish

- End-stage renal disease with persistently elevated troponin levels

Study Procedures

In both study phases, consecutive patients entering the ED with signs and/or symptoms suggestive of ACS when the principal investigator (LRB) was available were invited to participate. The principal investigator enrolled participants during weekdays, weekends, and on all shifts; and monitored participants (along with their assigned health care providers) until they were admitted to the hospital or discharged from the ED. She collected data on participants' demographic characteristics, chief complaints, comorbidities (e.g., diabetes and hypertension), and clinical factors from the participant or proxy, or from the electronic medical record (see Table 1). Care of the participants was directed by emergency physicians.

Pre-C-STM phase—Approval to conduct the study was obtained from governing institutional review boards to ensure compliance with applicable regulations, including informed consent. The first phase of the study started after these approvals were obtained. During this phase, repeat 12-lead ECGs were obtained by the principal investigator whenever a change in the participants' symptoms occurred. Repeat ECGs were compared with the initial ECG taken on the participants' arrival to the ED. Those that were different in any regard were handed to an ED attending physician. Per hospital protocol, another copy was transmitted to a cardiologist who was blinded to the presentation of the participants (for an unbiased review of the ECGs).

C-STM phase—The second study phase started immediately after the first. During this phase, the principal investigator activated the C-STM feature on the bedside monitor as it was not automatically activated when a patient was placed on the bedside monitor. The bedside monitors were programmed to measure ST deviations at J+60 ms and alarm at 1mm above and below the participant's baseline ST-segment. The Mason-Likar configuration (limb electrodes attached to the participant's torso) was used for C-STM monitoring but standard 12-lead ECGs (limb lead wires attached to the limbs) were obtained on evaluation of ST-alarms and whenever a change in participants' symptoms occurred. Repeat ECGs were processed similarly to the pre-C-STM study phase.

Thirty-day follow-up—The principal investigator and a co-author (VJ) contacted participants after 30 days from the day of the index ED visit in both study phases. They collected data on 30-day adverse events by medical record review, e-mail, telephone, or regular mail from participants or their friends or family listed as contacts.

Measures

Time to diagnosis—The starting time used for the computation of time to diagnosis was the time that the participant arrived in the ED. The ending time was the time the disposition decision was recorded in the medical record. The disposition decision could be indicated by several events, including the time an admission order was placed, the conversion of the participant's status to discharge or admission in the medical record, the time a hospital bed was requested, or the time a nursing note indicated the disposition status of the participant. Whichever occurred first was used. There was no interaction with clinicians about the time to disposition and only the electronic medical record was used to obtain this information.

The HEART score—Participants were categorized according to risk of 30-day adverse events using the History, ECG, Age, Risk factors, and Troponin (HEART) score criteria (Backus et al., 2013; Six, Backus, & Kelder, 2008). The HEART score is a risk score for ACS that was specifically designed for the ED patient population. A score of 0–3 points carries a low risk of 2.5% and supports ED discharge; a score of 4–6 points carries an intermediate risk of 20.3% and indicates hospital admission for observation; and a score of 7 carries a high risk of 72.7%, supporting early invasive treatment.

Ischemia on C-STM—The criteria for new/evolving ischemia on C-STM were new ST-segment changes at J+60 ms that lasted at least 1 minute:

- ST-segment elevation greater than 1mm (0.1mV) in two or more contiguous leads (leads over neighboring parts of the heart), and/or 2mm (0.2mV) in at least one lead

- Horizontal or downsloping ST-segment depression greater than 1mm (0.1mV) in two or more contiguous leads

Data Analysis

Each variable within the sample was described using frequencies or measures of central tendency and dispersion. Characteristics of participants in the two study phases were compared using chi-squared, Fisher's exact, and Mann-Whitney U tests, as appropriate. Because the time to diagnosis data were not normally distributed, the non-parametric analog to the independent t-test (Mann-Whitney U test) was used to compare the time to diagnosis before and after the implementation of C-STM.

The frequency and characteristics (elevation and depression) of ST-segment changes detected by C-STM were described. Descriptive statistics (frequencies and measures of central tendency and dispersion) were used to describe new episodes of myocardial ischemia on C-STM.

The ability of C-STM to detect myocardial ischemia or infarction during the assessment of participants in the ED was evaluated by calculating its sensitivity and specificity. The reference standard for determining unstable angina (UA) was documented reversible ischemia on a stress test. For myocardial infarction, the reference standard was elevation above 0.120 ng/mL in the troponin I cardiac biomarker (VITROS® ECi/ECiQ Immunodiagnostic System). Likelihood ratios (LRs), which combine the sensitivity and specificity of a test in one number, were also reported for C-STM. The LR for a positive test result (+LR) = sensitivity/(1-specificity), while the LR for a negative result (−LR) = (1-sensitivity)/specificity.

Chi-squared and Fisher's exact tests were conducted to determine any associations between C-STM and each adverse event and the composite variable of 30-day adverse events. Additionally, the data were further categorized and examined for associations within HEART score risk categories.

All analyses were performed using SAS® (Version 9.3). All tests of statistical significance were 2-tailed. An alpha level of 0.05 was the criterion for statistical significance.

Results

Participants in both phases of the study were enrolled over a 4-month period. They were monitored for a minimum of 1 hour in the ED and the median duration of monitoring was 6 hours. Overall, 18 (11.0%) had a discharge diagnosis of ACS and 115 (70.6%) of participants were discharged home from the ED. Participant characteristics are presented in Table 1. The groups were similar, with the exception of a significantly higher proportion of Caucasian participants in the pre-intervention group (63% vs. 45%, p = 0.01).

The time to diagnosis ranged from 1.4 to 13.6 hours pre-intervention and 1.3 to 18.0 hours during the intervention phase. There was no statistically significant difference between the median time to diagnosis before and after the implementation of C-STM (5.55 vs. 5.98 hours; p = 0.43). There was a wide variability in the time to diagnosis both before and after implementation of C-STM, which could be due to multiple factors, including hospital organizational systems and policies. As shown in Table 2, time to diagnosis was not significantly different before and after C-STM implementation in any the three HEART score risk categories.

The frequency and characteristics (elevation and depression) of the ST-segment changes detected by C-STM in the intervention phase are shown in Table 3. Only three (3.5%) of the 85 participants had new/evolving ischemia detected on C-STM. Sixteen (18.8%) of the 85 participants in the intervention phase of the study had a stress test. Of these 16, three had their stress test prior to hospital discharge. Only one of the 16 participants had a positive stress test result. This participant subsequently had a negative coronary angiogram. One participant with a negative stress test had a positive coronary angiogram.

All participants received at least one troponin test during the ED evaluation. The results of the diagnostic accuracy calculations for C-STM are presented in Table 4. By convention, likelihood ratios are thought to be diagnostic at >10 for +LR and <0.1 for –LR. Although the +LR of 24 for ischemia and 13.7 for infarction could be considered diagnostic, confidence intervals were wide.

Thirty-day follow-up for the exploratory aim on adverse events was completed for all 78 (100%) of the participants in the pre-intervention study phase and for 81 of the 85 (95.3%) participants in the intervention phase. None of the participants who were lost to follow-up had been diagnosed with myocardial ischemia during the index visit. Table 5 shows the 30-day event rate of the total sample and bivariate analyses by study phase. There was no significant difference in the occurrence of any adverse events or for the composite of all events for participants who were monitored by C-STM compared with those who were not.

Of the 15 participants (3 low risk [20.0%], 11 intermediate risk [73.3%], 1 high risk [6.7%]) in the combined study phases who returned to the ED within 30 days of the index visit with signs and/or symptoms of ischemia, 8 (all of whom were of intermediate risk) were admitted to the hospital. Two additional participants returned to the ED without signs and/or symptoms of myocardial ischemia and were admitted to the hospital for other medical conditions. Of the 10 who were admitted to the hospital, 7 had a non-cardiovascular diagnosis, 2 had a UA diagnosis, and 1 had a diagnosis of stable angina. None of the participants in the follow-up phase of the study had a positive troponin test or had reperfusion therapy. One participant in the pre-intervention phase of the study was diagnosed with Stanford type A aortic dissection during the ED evaluation and opted for non-surgical treatment. This participant died after hospital discharge.

Discussion

The findings of this study are consistent with other studies reporting that C-STM has limited value in the evaluation of low- to intermediate-risk patients in the ED (Decker et al., 2003; Fesmire, 2000). In this study, 11% of participants who presented to the ED with chest pain were diagnosed with ACS. This was comparable to the Centers for Disease Control and Prevention (CDC) estimate of 13.0% (CDC, 2014). The percentage would have been higher had patients with ECG evidence of ST-elevation myocardial infarction been included. Findings of the current study are consistent with results from Scheuermeyer et al. (2012) who reported a similar incidence of ACS. They conducted a prospective cohort study of ED patients in Vancouver, Canada, in which acute myocardial infarction was diagnosed in 39 of 1,116 patients (3.5%) and UA was diagnosed in 60 (5.4%) patients at the index hospital visit.

A low percentage of ACS diagnoses in patients presenting with signs and/or symptoms suggestive of myocardial ischemia will consequently lead to a low rate of monitor-detected events. This implies that C-STM may not be beneficial for the majority of these patients. The small number of episodes of ischemia (n=3) detected by C-STM in our study limits the interpretation of its diagnostic accuracy. This is reflected in the wide confidence intervals for sensitivity and +LR for both myocardial ischemia and infarction.

The diagnostic accuracy of C-STM in the current study was found to be comparable to others (Decker et al., 2003; Fesmire, 2000; Fesmire et al., 1998). Fesmire (2000) reported a sensitivity of 41.7% (95% CI 27.6 to 56.8) and specificity of 98.1% (95% CI 96.7 to 99.0), of C-STM in detecting acute myocardial infarction. The corresponding +LR and the −LR were 21.9 and 0.59, respectively. Albeit, C-STM was used in conjunction with serial ECGs in the Fesmire study. Similar to our study, the specificity of C-STM in detecting ACS was higher than its sensitivity. In our study, both the +LR of C-STM for myocardial ischemia (24.0) and myocardial infarction (13.7) were greater than 10, which is regarded as diagnostic of the disease (Jaeschke, Guyatt, & Sacket, 1994; Straus, Richardson, Glasziou, & Haynes, 2011). A −LR of 0.2 to 0.5 is useful and less than 0.1 is regarded as diagnostic (Jaeschke, Guyatt, & Sacket, 1994). The −LR of C-STM for myocardial ischemia (0.26) indicated that it was useful in ruling out ischemia, while the −LR for infarction (0.68) indicated that it was not very useful in ruling out infarction. Although the study findings indicated that C-STM may discriminate well in ruling in myocardial ischemia or infarction they should be interpreted cautiously in light of limitations in the reference tests and the limited number of episodes of ischemia detected by C-STM.

Similarly to other studies, a low (1.3%) incidence of ACS in 30-day follow-up in patients evaluated for signs and/or symptoms of ACS in the ED was found in our study. In the study conducted by Decker et al. (2003), patients who met the Agency for Health Care Policy and Research criteria for intermediate risk had a 30-day adverse event rate of 1.6%. Farkouh et al. (2009), reported that 30-day major adverse cardiovascular and cerebrovascular events occurred in 11.5% of a high risk group, 6.2% in an intermediate risk group, and 2.5% in a low risk group. Than et al. (2012) reported a 15% incidence of 30-day major adverse cardiac events (acute myocardial infarction, death, cardiac arrest, cardiogenic shock, emergency

revascularization, and ventricular dysrhythmia) in a study conducted in two hospitals in Australia and New Zealand. The limited detection of ischemia on C-STM in our study implies that it may not be beneficial in reducing 30-day adverse events.

Strengths and Limitations

A strength of this study is its prospective design, which allows for more control in how the data were collected than with a retrospective design. Another strength was that the study provided an appreciation of the impact and effectiveness of this technology in a real world clinical setting. This is important for development of future clinical studies using this technology and for providing input to manufacturers of bedside ECG monitoring software. Of note, several participants verbalized peace of mind about being continuously monitored for ST-segment changes on the bedside monitor during the ED visit.

A limitation of this study is that episodes of ischemia may have been missed when participants were disconnected from the monitor for procedures, such as x-rays and computerized tomography scans. The majority of participants had a chest x-ray. Efforts were made to ensure that C-STM was resumed and participants reassessed immediately after a necessary disruption.

Ideally, the patient's condition should not change appreciably in the time interval between the implementation of C-STM and the reference tests. Sufficient time for a change in the participants' condition could occur before the administration of the stress test, which was frequently performed during an outpatient follow-up visit.

While the reference tests were interpreted by individuals who were blinded to the results of C-STM, the results of C-STM were interpreted by the principal investigator who was not blinded to the results of the reference tests. It is possible that there could be bias in interpretation of the results of C-STM. However, the ECGs that were printed as a result of C-STM changes were also interpreted by blinded evaluators, which should offset this possible bias. Another limitation was that the software used had limited storage capacity for ST-segment deviation data. The GE (Solar ® 8000i) bedside monitors that were used in this study were programmed to store up to 10 ST events, necessitating that the central monitor be continuously checked for possible missed events.

This was a single center study, which could influence the generalizability of study findings. Since participants enrolled in this study were not randomized to C-STM, a larger, multicenter, randomized controlled trial could offer additional evidence on which to base current practice and establish possible benefit. An additional limitation is that the study was not designed to quantify the cost of implementing C-STM. This additional information could potentially increase the significance of the study.

Conclusions and Implications

Most patients presenting with signs and/or symptoms of myocardial ischemia in the ED are not ultimately diagnosed with ACS, and of those who are diagnosed with ACS, use of C-STM does not significantly improve time to diagnosis or 30-day outcomes. Although the

diagnostic accuracy of C-STM was comparable to other ED studies and could be regarded as useful, only a small number of episodes of ischemia were detected.

In this sample of patients in the ED, the majority of whom were categorized as being of intermediate risk for ACS, use of C-STM did not provide much added benefit in detecting myocardial ischemia or infarction. These findings did not support use of C-STM in the ED. However, these study findings need to be validated at other centers and also with other brands of monitors.

Acknowledgments

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Table 1

Comparison of Demographic and Clinical Characteristics of Study Patients Before and After C-STM Implementation (N=163)

Variable	Total Sample		Pre-C-STM (n=78)		C-STM (n=85)		t	p
	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range		
Age (years)	58 (14)	24–98	59 (15)	24–98	57 (14)	35–89	0.83	0.41
	N (%)		N (%)		N (%)		χ^2	
Male gender	72 (44)		35 (45)		37 (43)		0.03	0.86
White race	87 (53)		49 (63)		38 (45)		10.32	**0.01**[*]
Hispanic ethnicity	41 (25)		21 (27)		20 (23)		0.25	0.62
Coronary artery disease	54 (33)		26 (33)		28 (33)		0.00	0.96
Diabetes	65 (40)		27 (35)		38 (45)		1.73	0.19
Never smoked	75 (46)		38 (49)		37 (43)		0.78	0.87
Hypertension	131 (80)		63 (81)		68 (80)		0.01	0.90
Hyperlipidemia	109 (67)		51 (65)		58 (68)		0.15	0.70
Overweight/obese	145 (89)		69 (88)		76 (89)		0.04	0.85
Family history of CAD	90 (55)		43 (55)		47 (55)		0.93	1.00
Discharged home from the ED	115 (70)		58 (74)		57 (67)		1.04	0.31
Admitted to a cardiac unit	43 (26)		18 (23)		25 (29)		0.84	0.36
Troponin value indicative of MI	6 (4)		3 (4)		3 (3)		0.01	1.00
Positive stress test results	2 (1)		1 (1)		1 (1)		3.41	0.34
ACS Diagnosis:							3.62	0.32
Probable ACS	3 (2)		2 (3)		1 (1)			
STEMI	0 (0)		0 (0)		0 (0)			
NSTEMI	6 (4)		4 (5)		2 (2)			
UA	6 (4)		1 (1)		5 (6)			
Reperfusion therapy:							0.01	1.00
PCI	3 (2)		1 (1)		2 (2)			
CABG	1 (1)		1 (1)		0 (0)			

Note. ACS = Acute coronary syndrome; AMI = Acute myocardial infarction; BMI = Body mass index; CABG = Coronary artery bypass graft; CAD = Coronary artery disease; ED = Emergency department; C-STM = Continuous ST-segment monitoring; MI = Myocardial infarction; NSTEMI = Non-ST-segment elevation myocardial infarction; SD = Standard deviation; STEMI = ST-segment elevation myocardial infarction; Probable ACS = Patients with signs and/or symptoms of ACS but with non-diagnostic test results; UA = Unstable angina; PCI = Percutaneous coronary intervention

*
$p < .05$

Table 2

Time to Diagnosis of Study Participants

Participants	Median Time to Diagnosis (Hours)		p
	Pre-C-STM	**C-STM**	
All (N=163)	5.55	5.98	0.42
*HEART Score Risk Categories:			
Low (N=51)	5.90	6.28	0.52
Intermediate (N=100)	5.15	5.82	0.38
High (N=12)	5.80	4.90	0.32

Note. The HEART is composed of 5 components: History, ECG, Age, Risk factors, and Troponin (Backus et al., 2013; Six, Backus, & Kelder, 2008)

Table 3

Description of New/Evolving Ischemia on Continuous ST-Segment Monitoring (N=85)

Participant Gender	Type of ST-Segment Deviation	Maximum Deviation from the Isoelectric Line (mm)	Duration of ST-Segment Deviation (minutes)
Female	Elevation	1.7	6
Female	Depression	3.3	13
Male	Depression	2.4	1

Note. ST-segment deviation was measured at the J point + 60 msec. Alarms were set at 1mm above and below the patient's baseline.

Table 4

Diagnostic Accuracy of Continuous ST-Segment Monitoring (N=85)

Measure	Ischemia	Infarction
Sensitivity	75.0%	33.3%
Specificity	96.9%	97.6%
+Likelihood Ratio	24.0 (95% CI 1.4–412.0)	13.7 (95% CI 1.7–112.3)
–Likelihood Ratio	0.3 (95% CI 0.02–2.9)	0.7 (95% CI 0.3–1.5)

Table 5

Events Occurring within 30-days of Discharge Following the Index Event (N=159)

Description	Total Sample	Pre-intervention (N=78)	Intervention (N=81)	χ^2	p
	N (%)	N (%)	N (%)		
Return to the ED with signs and/or symptoms of myocardial ischemia	15 (9)	7 (9)	8 (10)	0.04	0.85
All-cause admission to the hospital within 30 days of the index hospital visit	10 (6)	6 (8)	4 (5)	0.63	0.52
Biomarker indication of MI	0 (0)	0 (0)	0 (0)	-	-
Reperfusion therapy	0 (0)	0 (0)	0 (0)	-	-
All-cause mortality	1 (1)	1 (1)	0 (0)	1.10	0.48
Composite of 30-day events	18 (11)	9 (12)	9 (11)	0.01	0.93

Note. CABG = Coronary artery bypass graft; C-STM = Continuous ST-segment monitoring; ED = Emergency department; MI = Myocardial infarction; PCI = Percutaneous coronary intervention.