# PREVALENCE OF SECONDARY ST-T WAVE ELECTROCARDIOGRAPHIC ABNORMALITIES CONFOUNDING THE DIAGNOSIS OF ACUTE MYOCARDIAL ISCHEMIA IN PATIENTS PRESENTING TO THE EMERGENCY DEPARTMENT WITH A CHIEF COMPLAINT OF CHEST PAIN

by

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Chest pain in patients presenting to the emergency department (ED) has a plethora of etiologies and electrocardiographic (ECG) manifestations. Admission to the hospital from the ED with chest pain will likely place the patient on a telemetry monitored unit for continued cardiac monitoring, specifically monitoring the ST-segment that can detect ischemia. The current guideline for in-hospital cardiac monitoring lists a few exclusions to ST-segment ischemia monitoring such as bundle-branch blocks, ventricular rhythms, and coarse atrial fibrillation or flutter (Drew et al., 2004). These conditions alter the ST-segment for reasons unrelated to acute myocardial ischemia, triggering ST-segment monitor alarms that can lead to alarm fatigue, misdiagnosis, or inappropriate treatment. The purpose of this study is to determine the prevalence and clinical significance of these non-ischemic ECG abnormalities that alter the ST-segment and affect the healthcare professionals' accurate assessment of myocardial ischemia in patients that present to the ED with a chief complaint of chest pain. This study includes a secondary analysis of the ongoing Electrocardiographic Methods for Prompt Identification of Coronary Events (EMPIRE) study data set, which aims to quantify ischemia-induced repolarization dispersion for early non-ST elevation myocardial infarction detection. The parent study has created a database of patients who arrive via ambulance to the ED with a chief complaint of chest pain (Al-Zaiti, Martin-Gill, Sejdic, Alrawashdeh, & Callaway, 2015). In this secondary analysis, the demographic, clinical,

and ECG data from 750 consecutively enrolled patients were assessed for acute coronary syndrome risk factors and ECG abnormalities, including secondary repolarization changes that interfere with ST-segment monitoring. 75% of patients were admitted and 16% of patients overall had confounders for ST-segment monitoring. Significant relationships between ST-segment monitoring confounders and important clinical variables such as age, coronary artery disease risk factors, and length of stay were found. Determination of the prevalence of ECG abnormalities that affect the ST-segment would provide valuable information on the clinical utility of ST-segment monitoring in chest-pain populations.

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# 1.0 INTRODUCTION

Chest pain is the second leading cause of emergency department visits in the United States; approximately six million Americans annually visit emergency departments with a chief complaint of chest pain (Hollander & Chase, 2016). Etiologies of chest pain may be life-threatening if involving the heart or lungs, such as acute coronary syndrome (ACS), pulmonary embolism, cardiac tamponade, and tension pneumothorax. Other non-cardiac causes that may cause chest pain are pneumonia, acid reflux, and musculoskeletal pain (Hollander & Chase, 2016). Ruling out life-threatening conditions, such as ACS or pulmonary embolism, is the priority during the evaluation of chest pain at the ED. ACS results from a ruptured plaque or thrombus that obstructs blood flow in a coronary artery and thus starves the cardiac tissue of oxygen, called myocardial ischemia, possibly leading to cell injury and death, called myocardial infarction. While the population presenting to the ED with chest pain is heterogeneous, the initial ED evaluation of these patients is consistent: history and physical examination, a 12-lead ECG, and evaluation of serum cardiac biomarkers of myocardial infarction, such as troponin level (Hollander & Chase, 2016).

The 12-lead ECG is the principle tool for evaluating chest-pain patients, regardless of etiology and risk factor assessment. The ST-segment portion of the electrocardiogram is normally isoelectric, so ST elevation or depression is clinically indicative of myocardial ischemia or infarction (Prutkin, 2016). A chest-pain patient with an ECG showing ST-segment elevation can be immediately diagnosed with an ST elevation myocardial infarction (STEMI) and would

immediately be sent for percutaneous coronary intervention (PCI) for revascularization. If the patient's ECG shows either ST-segment depression or minimal ST-segment deviation, it is inconclusive. In that case, cardiac biomarkers, such as troponins, are tested for with a simple blood draw lab test. Positive troponins in these patients would then indicate a non-ST elevation myocardial infarction (NSTEMI) and anticoagulation or PCI should be considered (Hollander & Chase, 2016). Unfortunately, troponin may take up to 24 hours to rise and reach a peak level and, in some patients, might remain negative despite critical coronary artery disease, such as in unstable angina (Troponin test, 2015). Therefore, the presence or absence of ACS (i.e., STEMI, NSTEMI, or unstable angina) frequently cannot be determined at the ED and might require admission for advanced diagnostics, such as nuclear perfusion imaging, and for telemetry monitoring to detect transient and evolving ECG changes.

Chest-pain patients who have negative initial assessments for ACS but also have risk factors such as a high body mass index (BMI), coronary artery disease history, tobacco use, and an age over 55, will likely be admitted to a telemetry unit for further evaluation and monitoring (Hollander & Chase, 2016). While on a telemetry unit, these undiagnosed chest-pain patients will likely be placed on ST-segment monitoring to detect ST amplitude changes that trigger an alarm. The American Heart Association (AHA) published a guideline for in-hospital telemetry monitoring in 2004 to establish practice standards for ST-segment monitoring (Drew et al., 2004).

Although these recommendations are widely adopted in clinical practice, there are still unmet needs in clinical settings. Specifically, there are some ECG confounders that preclude proper interpretation of the 12-lead ECG but are generally unrelated to the underlying cause of the chest pain (Rautaharju et al., 2009). These ECG confounders lead to secondary changes in the ST-T waveform that are not related to ischemia. For instance, while a chest-pain patient might present

with pneumonia, his or her ECG might show a bundle branch block. The latter is not caused by the pneumonia, but would lead to secondary ST-T changes, which would impede the assessment of myocardial ischemia in this patient. Identified ECG confounders include left bundle-branch block (LBBB), frequent intermittent right bundle-branch block (RBBB), ventricular pacing rhythm, coarse atrial fibrillation or flutter, and intermittent accelerated ventricular rhythm (Drew et al., 2004). Left ventricular hypertrophy (LVH) with strain pattern is also known to set off false ST-segment alarms (Rautaharju et al., 2009; Drew, Wung, Adams, & Pelter, 1998). Essentially, the computer ST measurement for these identified ECG abnormalities would indicate ST deviation for reasons other than ischemia.

Although these patients with ST confounders are potentially high risk patients with cardiopulmonary compromise, current guidelines recommend against using ST-segment monitoring when these ECG confounders exist. They are not excluded because they are at minimal risk for ischemia, but rather because computer measurement of ST amplitude cannot be trusted. Unfortunately, there are no alternative methods available for monitoring ischemia in these patients. Nurses are the primary observers of these patients, but they lack proper training in ST-segment monitoring and the understanding of ST confounders. Consequently, nurses frequently turn off ST-segment monitoring alarms in these patients, limiting the value of telemetry monitoring for which these patients were admitted in the first place.

An example scenario is an overweight, older adult male who has smoked for 30 years and has hypertension who comes to the hospital with chest pain. His initial troponin may be negative and his presenting ECG may show what looks like a STEMI, but it is evaluated further and is a previously-documented LBBB. This patient is at high risk not only for ACS but also for heart failure related to left ventricular dyssynchrony (Sauer, 2014.) This patient will likely be admitted

and placed on telemetry monitoring, but will constantly trigger the ST-segment alarm, thus deeming the feature not useful. The patient should still be monitored, but if not with ST-segment monitoring, then how?

The question then becomes: what is the frequency of chest-pain patients with secondary repolarization changes that induce non-ischemic ST changes, making them ineligible for ST-segment monitoring? It is important to identify patients that are at high risk for ACS and cardiopulmonary instability, such as patients with LBBB. Additionally, these high-risk patients may experience longer hospital stays because reaching a conclusive diagnosis may be hindered by confounding ECG abnormalities. Evidence suggests that healthcare professionals should not only be relying on telemetry monitoring for observation, but should also implement other measures to closely observe these patients. Quantifying the magnitude of this problem would be the first step to inform future development of targeted interventions. The primary purpose of this thesis is to define the prevalence and clinical significance of these non-ischemic ST-T changes in patients presenting to the ED with a chief complaint of chest pain.

## 2.0 BACKGROUND

The electrocardiogram illustrates the electrical activity of the cardiac conduction system. A normal ECG waveform (Figure 1A) starts with the P wave as the first positive deflection representing depolarization of the atria, followed by the QRS complex, which signifies ventricular depolarization. The T wave denotes the repolarization of the ventricles, and the isoelectric ST-segment shows an electrocardiographic resting period between ventricular depolarization and repolarization (Prutkin, 2016). Figure 1B illustrates ST elevation that is specific to acute myocardial ischemic injury, while figure 1C illustrates ST depression that may be seen during myocardial ischemia in some patients but may in fact be indicative of other pathologies. Figure 1D illustrates another manifestation of ST elevation that is caused by secondary repolarization changes, which preclude proper interpretation of ST-segment and render ST-segment monitoring useless.

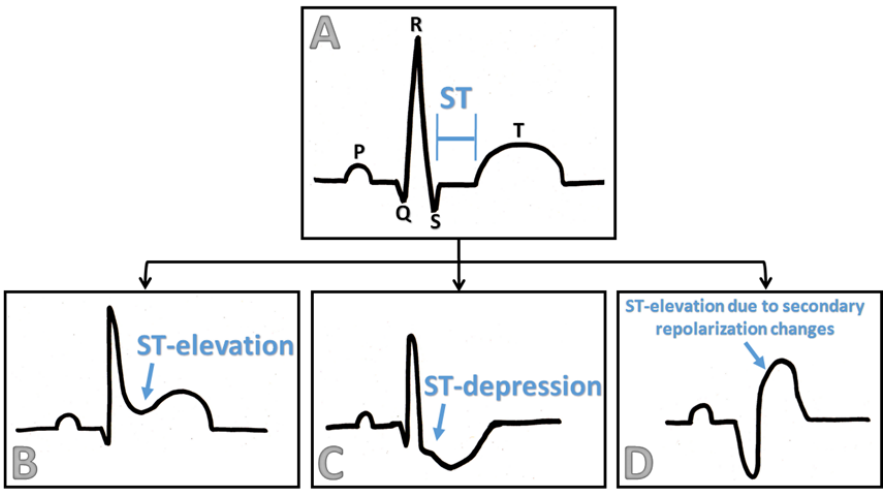


**Figure 1.** Normal and Abnormal Electrocardiographic Waveform Patterns

In 2004, the American Heart Association published the currently used practice standards for ECG monitoring in-hospital settings. In this guideline, best practices are presented based on expert opinion on what types of patients should be monitored and what monitoring settings should be applied (Drew et al., 2004). Specifically, ST-segment ischemia monitoring is addressed and categorizes patients into classes of clinical situations in which some patients should have ST-monitoring and others do not need it. ST-monitoring is utilized because ST-segment amplitude changes, such as elevation or depression, usually indicate myocardial ischemia or infarct. Class III is defined as the category of patients who are so low of a risk that no therapeutic benefits are gained from cardiac monitoring. Class III for ST-monitoring includes patients with left bundle-branch block (LBBB), frequent intermittent right bundle-branch block (RBBB), ventricular pacing rhythm, coarse atrial fibrillation or flutter, and intermittent accelerated ventricular rhythm (such as ventricular tachycardia) (Drew et al., 2004). However, within the context of chest pain, patients with these ECG abnormalities are in fact at the highest risk for dire outcomes (Anderson et al., 2013). Thus, the recommendation of no ST-segment monitoring is based solely on the inability to accurately interpret ST-segment changes in patients with ECG confounders rather than on their low risk for an adverse outcome.

According to AHA recommendations for interpretation of an ECG (Rautaharju et al., 2009), secondary repolarization changes are defined as “abnormalities in the ST-segment and T wave that occur as a direct result of changes in the sequence and/or duration of ventricular depolarization, manifested electrocardiographically as changes in QRS shape and/or duration.” The previously described ECG abnormalities that alter ST-segment are considered examples of secondary repolarization changes.

Patients with these ECG abnormalities render ST-monitoring ineffective because they have secondary repolarization changes or unstable ST-amplitudes from “chaotic atrial activity”, unrelated to myocardial ischemia or infarction, that would cause an ST-monitor to be constantly alarming, leading to alarm fatigue in telemetry floor nurses (Drew et al., 2004). This raises the question of what decisions clinicians should make regarding a patient who has an ECG abnormality that renders ST-monitoring useless, such as LBBB, who is admitted for telemetry monitoring to rule out acute coronary syndrome. Healthcare providers would elect to admit patients at high risk for ACS for further observation, but how nurses should monitor and observe patients with ST-segment monitoring confounders remains open for discussion.

A recent study found that almost 50 percent of patients who presented with chest pain over a five-year period to a hospital system were admitted (Weinstock et al., 2015). With healthcare costs rising, admitting every other patient of the 6 million Americans visiting ED for chest pain adds a tremendous burden on healthcare systems and aggravates nursing shortages, especially in acute care settings. To what extent secondary repolarization changes are prevalent in these admitted patients remains unclear, which complicates the problem of chest pain even further. The role of ST-segment monitoring in these patients is limited and only adds to the burden of alarm fatigue among critical care nurses. The first step to address this issue would be to quantify the magnitude of the problem; what is the prevalence of secondary repolarization changes in chest-pain patients and how does this interfere with telemetry monitoring decisions?

Based on an existing data set of ECGs collected on patients that were enrolled during hospital transport via ambulance for a chief complaint of chest pain, prevalence of ECG abnormalities that are defined in literature as having non-ischemic ST changes was investigated, specifically among admitted patients. This investigation on practice recommendations will provide

insight into the need for developing new approaches to observing chest-pain patients who are admitted for close observation for ischemia but who have an uninterpretable ECG for ST-segment changes.

# 3.0 PURPOSE

The purpose of this secondary analysis is to define the frequency of chest-pain patients with ST confounders and evaluate the clinical significance of these ECG abnormalities. Using an existing data set collected via observational study of a cohort of consecutive chest-pain patients at the ED, this thesis will investigate the following specific aims:

**Specific Aim 1: Determine the magnitude of the problem among patients evaluated in the ED for chest pain:**

Aim 1(a). What is the distribution of ischemic vs. non-ischemic causes of chest pain?

Aim 1(b). What percentage of patients admitted to a telemetry unit to rule out ACS had a final diagnosis of non-ischemic chest pain?

Aim 1(c). What is the prevalence of ECG abnormalities that lead to secondary non-ischemic ST changes or interfere with proper ST measurement?

**Specific Aim 2: Investigate the relationship between the presence of non-ischemic ST confounders and other important clinical variables:**

Aim 2(a). Is there a relationship between the presence of non-ischemic ST confounders and demographic and clinical characteristics of patients?

Aim 2(b). Is there a relationship between the presence of non-ischemic ST confounders and chest pain etiology?

Aim 2(c). Is there a relationship between the presence of non-ischemic ST confounders and course of hospitalization?

# 4.0 METHODS

## 4.1 DESIGN

This thesis is a secondary dataset analysis of Electrocardiographic Methods for Prompt Identification of Coronary Events (EMPIRE) (Al-Zaiti, Martin-Gill, Sejdic, Alrawashdeh, & Callaway, 2015). EMPIRE is an ongoing, prospective, observational cohort study for building a database of chest-pain patients in Pittsburgh for expediting identification and treatment of NSTEMI. EMPIRE enrolled consecutive patients that called 9-1-1 for a chief complaint of chest pain and were transported via ambulance to one of three UPMC-affiliated hospitals in Pittsburgh, PA. Inclusion criteria were age 18 or older, chief complaint of non-traumatic chest pain or equivalent (e.g., shortness of breath, palpitation, etc.), arrival to the hospital via ambulance, and acquisition of a 12-lead ECG during prehospital transport. Exclusion criteria were traumatic chest-pain patients and absence of pre-hospital ECG.

The EMPIRE study was approved by the University of Pittsburgh IRB under a waiver of informed consent to facilitate the creation of a representative, unbiased cohort of consecutive chest pain cases. The current secondary analysis was approved by Dr. Salah Al-Zaiti. The parent study was minimal risk as it collected routine care data and there was no patient contact; data were extracted from electronic medical records by reviewers blinded to study outcomes. All extracted data were de-identified before storage and a linkage list was kept separate from the data; both measures were taken to reduce the risk of breach of confidentiality.

## 4.2 DEMOGRAPHIC AND CLINICAL DATA

Relevant demographic and clinical data to be studied in this secondary analysis were identified. Given that age, sex, and race are well-known considerations in the interpretation of electrocardiographic data, they will be included as key demographic data in the current secondary analysis (Macfarlane, McLaughlin, Devine, & Yang, 1994). Other clinically-relevant variables that will be included as key clinical variables for the current investigation include obesity class, smoking history, and presence of the following diseases: hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease (CAD), and angina. Past cardiac history of MI, congestive heart failure (CHF), or coronary artery bypass graft (CABG) or PCI will also be included in the analysis. Variables regarding clinical presentation (e.g., symptomatology, diagnostic tests, and course of hospitalization) will also be assessed. For example, ischemic changes on the presenting ECG will be evaluated using the Universal Definition criteria for the identification of MI (Thygesen et al., 2012).

The demographic and clinical data described in this section will be used as independent variables in relation to study outcomes and predictors. Age will be defined as a continuous variable, and sex and race as dichotomous variables. Obesity class will be defined according to WHO as: normal weight is 18.5-24.9kg/$m^2$, overweight is 25-29.9kg/$m^2$, and obesity is over 30 kg/$m^2$ (BMI classification, 2017). Other key clinical variables (i.e., past medical history and clinical presentation) will be defined as dichotomous. Length of stay will be defined as the number of days and hours a patient is in the hospital (e.g. 1.6 days).

## 4.3 CLINICAL OUTCOMES

Etiology of chest pain. Chest pain can be induced by multiple causes. In this thesis, etiology is defined as (1) ACS (i.e., STEMI, NSTEMI, or unstable angina), (2) non-ischemic cardiopulmonary (e.g., stable angina, pulmonary embolism, pneumonia, cardiac tamponade, tension pneumothorax, valvular heart disease, and heart failure), (3) other non-cardiac causes (e.g., gastrointestinal related, substance related, etc.), and (4) undifferentiated chest pain (e.g., musculoskeletal, unknown, etc.).

Admission status. The course of hospitalization will be defined as if the patient was (1) discharged from the emergency department (length of stay <12 hours), (2) admitted to telemetry for overnight observation (length of stay ≤36 hours), or (3) admitted to the hospital for treatment of underlying pathology (length of stay ≥36 hours).

ST-segment monitoring confounders. These ECG confounders will be defined as either secondary ST changes induced by non-ischemic causes or causes that interfere with proper ST measurement. ST changes induced by non-ischemic causes include left bundle branch block (LBBB), left ventricular hypertrophy (LVH) with strain pattern, ventricular pacing, and ventricular rhythm. Causes that interfere with computerized ST measurement include coarse atrial fibrillation or flutter (AFib) and frequent intermittent right bundle branch block (RBBB). Each 12 lead ECG will be assessed for the presence of any of the listed abnormalities using the standardized criteria defined in Table 1.

**Table 1.** ECG Diagnostic Criteria Used for Interpretation

| | ECG Abnormality | Description | Coding |
|---|---|---|---|
| **Ischemic Changes** | ST Elevation | New ST elevation at J point in 2 contiguous leads with cut-points: ≥0.1 mV in all leads other than V2-V3; ≥0.2 mV men less than 40, ≥0.25 mV men over 40, ≥0.15 mV in women (Thygesen et al., 2012). | Y/N |
| | ST Depression | New horizontal or down-sloping ST depression ≥0.05 mV in two contiguous leads (Thygesen et al., 2012). | Y/N |
| **Secondary ST Changes Induced by Non-Ischemic Causes** | Left Bundle Branch Block | QRS area >1/4 of (QRS duration × maximum R amplitude) in lead I or V6, QRS >120 ms, QRS balance negative in lead V1 and V2 (Tan, Sungar, Myers, Sandri, & Froelicher, 2009). | Y/N |
| | Left ventricular hypertrophy with strain | S in V1 + R in V5 > 35 mm, with ST depression and T wave inversion (Hancock et al., 2009). | Y/N |
| | Ventricular Pacing | Prolonged QRS complexes with discordant T waves during the presence of ventricular pacemaker spikes (Zehender et al., 1992). | Y/N |
| | Ventricular Rhythm | Prolonged QRS complexes due to non-sinus activity below the AV node (e.g., ventricular tachycardia or third degree heart block) (Goldberger et al., 2008). | Y/N |
| **Causes that Interfere with Proper ST-segment Measurement** | Coarse Atrial fibrillation/flutter | R-R interval variability with lack of discernible P wave and visible atrial waveforms causing artifact; fluctuating ST amplitudes from chaotic atrial activity (Tan, Sungar, Myers, Sandri, & Froelicher, 2009; Drew et al., 2004) | Y/N |
| | Right Bundle Branch Block | QRS duration>120 ms, QRS area in lead 1 positive, no terminal S wave in lead V1, S amplitude + ST junction <100 mV and <R amplitude in lead V1 (Tan, Sungar, Myers, Sandri, & Froelicher, 2009). | Y/N |

## 4.4 DATA COLLECTION AND CODING

ECG Data. The EMPIRE dataset obtained the presenting 12-lead ECG at the ED in a standard PDF format. All ECGs were de-identified and saved to a research drive protected by UPMC firewall. First, a reviewer (ED medical resident), blinded to clinical data, interpreted each 12-lead ECG using study definitions (Table 1) and logged his annotations in an excel sheet. Second, a manufacturer-specific automated algorithm interpreted the same ECG confounders and logged in its annotation on the same excel sheet. Finally, a third reviewer (research scientist) adjudicated any disagreement between the blinded reviewer and the automated algorithm.

Clinical Outcomes. A reviewer blinded from all ECG analyses reviewed the entire hospital record of the indexed visit for each patient and determined the etiology of chest pain as either ACS, non-ischemic cardiopulmonary disease, non-cardiac related disease, or undifferentiated cause as previously defined. The reviewer also classified the course of hospitalization as either patient discharged from ED, patient admitted for overnight observation/further testing, or patient admitted for treatment of serious underlying pathology.

## 4.5 STATISTICAL ANALYSIS

The data were analyzed using IBM's SPSS 22 software and all variables were reviewed for normal distribution, and descriptive analyses were reported as mean ± standard deviation or median (inter-quartile range) for continuous variables and n (%) for categorical variables. Age and length of stay were continuous, and all other key demographic and clinical variables are categorical as previously described. Clinical outcomes were also defined as categorical: etiology (4 mutually exclusive

groups); course of hospitalization (3 mutually exclusive groups); presence of non-ischemic ST confounders (yes/no). Comparisons between groups were done using independent samples t-test for continuous variables and chi-square for categorical variables. Length of stay was compared between groups using Mann-Whitney U test. Significance level was set as $p < 0.05$ two tailed for hypothesis testing.

# 5.0 RESULTS

## 5.1 BASELINE CHARACTERISTICS

As shown in table 2, the sample size was 750 patients with a mean age of 59±17 years old. 433 (58%) of patients were males, and 301 (40%) of the patients were African Americans. The sample had 445 (59%) patients that were overweight or obese, and 436 (58%) of the patients were former or current smokers. Hypertension was the most prevalent disease (73%), then hyperlipidemia (35%), coronary artery disease (34%), and lastly diabetes mellitus (28%). 31% of the sample population had a history of a previous myocardial infarction, 23% had a past percutaneous coronary intervention and 9% had a previous coronary artery bypass graft. 18% had a history of congestive heart failure.

Majority of the sample (86%) presented with chest pain, only 13% had an initial positive troponin, and majority of the patients (82%) had normal serum potassium levels. According to their initial ECGs, majority of the patients (86%) were initially in sinus rhythm, only 73 (10%) were in atrial fibrillation. 223 (30%) of the patients had T wave inversion, 153 (20%) had ST-segment depression, and 55 (7%) had ST-segment elevation. Nearly 9% were treated by PCI, and 1% underwent CABG.

**Table 2.** Baseline Sample Characteristics

| **n=750** | |
|---|---|
| ***Demographics*** | |
| **Age** | Mean 59±17 years (19-100) |
| **Sex (Male)** | 433 (58%) |
| **Race (Black)** | 301 (40%) |

| | |
|---|---|
| ***CAD Risk Factors*** | |
| **Obesity class** | |
| Normal | 303(41%) |
| Overweight | 160 (21%) |
| Obese | 285 (38%) |
| **Smoking History** | |
| Never | 307 (41%) |
| Quit | 273 (37%) |
| Current | 163 (22%) |
| **Hypertension** | 547 (73%) |
| **Diabetes Mellitus** | 210 (28%) |
| **Hyperlipidemia** | 262 (35%) |
| **Coronary Artery Disease (CAD)** | 256 (34%) |
| **Angina** | 146 (20%) |
| **History of MI** | 231 (31%) |
| **History of CHF** | 135 (18%) |
| **Past PCI** | 172 (23%) |
| **Past CABG** | 70 (9%) |
| ***Clinical Presentation*** | |
| **Chest Pain** | 645 (86%) |
| **Shortness of Breath** | 215 (29%) |
| **Positive Initial Troponin** | 96 (13%) |
| **Serum Potassium Level** | |
| Hypokalemia | 113 (15%) |
| Normal | 614 (82%) |
| Hyperkalemia | 23 (3%) |
| **Rhythm** | |
| Sinus | 646 (86%) |
| Ventricular Rhythm | 8 (1%) |
| Atrial fibrillation/flutter | 73 (10%) |
| Pacing | 23 (3%) |
| **Ischemic Changes** | |
| ST-segment Elevation | 55 (7%) |
| ST-segment Depression | 153 (20%) |
| T Wave Inversion | 223 (30%) |
| ***Course of Hospitalization*** | |
| **Admitted** | |
| Discharged from ED | 193 (26%) |
| Admitted overnight | 234 (31%) |
| Admitted for treatment | 323 (43%) |
| **Length of Stay (mean±SD)** | 2.3±3.6 days (0-51) |
| **Final Impression** | |
| ACS | 130 (17%) |
| Non-ischemic cardiopulmonary | 237 (32%) |
| Non-cardiac | 69 (9%) |
| Undifferentiated CP | 314 (42%) |
| **PCI Done** | 65 (9%) |
| **CABG Done** | 9 (1%) |

## 5.2 SPECIFIC AIM 1(A)

As shown in Figure 2, nearly 1 in 5 patients had an ischemic cause of chest pain. The most prevalent final diagnosis for chest pain was undifferentiated (42%), followed by non-ischemic cardiopulmonary (32%), ACS (17%), and non-cardiac related (9%).

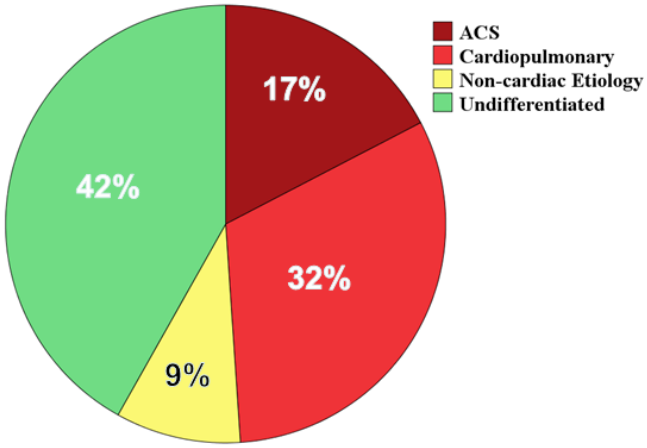


**Figure 2.** Chest Pain Etiologies

## 5.3 SPECIFIC AIM 1(B)

As shown in figure 3, more than one quarter of patients were discharged from the ED and nearly one third were admitted to telemetry for overnight observation. Of note, more than 50% of those admitted for observation had an undifferentiated etiology for chest pain. Among those admitted for treatment, majority were non-ischemic cardiopulmonary etiology followed by ACS patients.

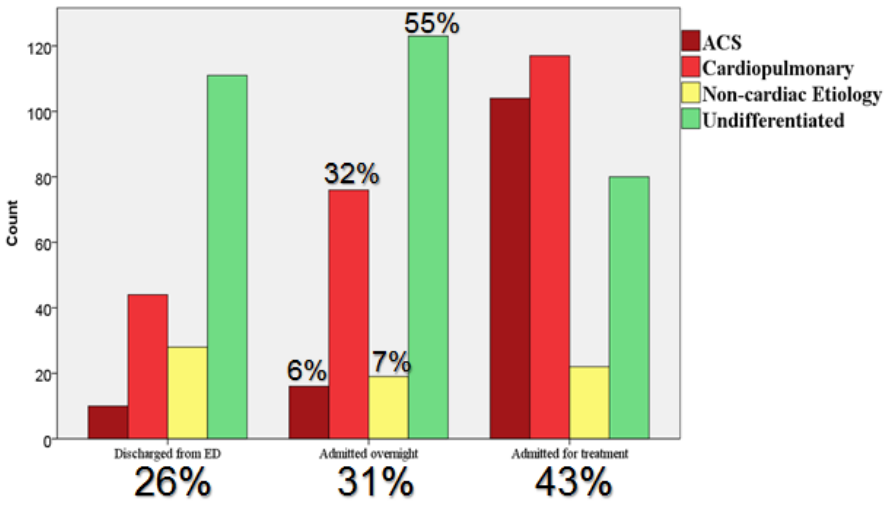


**Figure 3.** Chest Pain Etiology and Outcomes

## 5.4 SPECIFIC AIM 1(C)

As shown in Table 3, (1) 80 patients (10.7%) had at least one cause that leads to secondary non-ischemic ST changes, (2) 49 patients (6.5%) had a cause that interferes with proper ST measurement. Specifically, LVH with strain was the most common (3.6%) cause for secondary non-ischemic ST changed, followed by pacing and LBBB. The presence of intermittent RBBB (4.3%) was the most common cause that interferes with proper ST measurement, followed by coarse AFIB (2.4%). Overall, about 1 in 6 patients evaluated at ED for chest pain have confounders to ST-segment monitoring (Figure 4).

**Table 3.** Prevalence of Non-Ischemic ST Confounders

| ***Causes that lead to Secondary Non-Ischemic ST Changes*** | |
|---|---|
| LBBB | 22 (2.9%) |
| LVH with strain | 27 (3.6%) |
| Pacing | 23 (3.1%) |
| Ventricular Rhythm | 8 (1.1%) |
| ***TOTAL*** | **80 (10.7%)** |

| ***Causes that Interfere with Proper ST Measurement*** | |
|---|---|
| Coarse AFIB | 18 (2.4%) |
| Intermittent RBBB | 32 (4.3%) |
| ***TOTAL*** | **49 (6.5%)** |
| ***Any ST Confounder*** | **123 (16.4%)** |

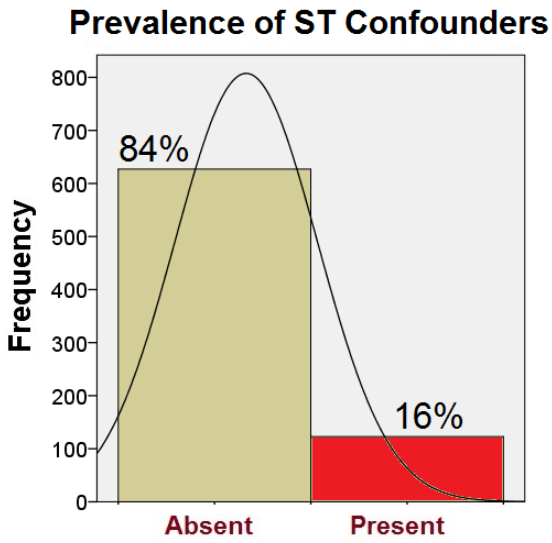


**Figure 4.** Prevalence of Non-Ischemic ST Confounders

## 5.5 SPECIFIC AIM 2(A)

As shown in table 4, older age and the presence of hypertension, coronary artery disease, angina, history of CHF, and past CABG are univariately associated with the presence of ST confounders ($p<0.001$). ST confounders are also related to diabetes mellitus ($p=0.001$), hyperlipidemia ($p=0.007$), history of MI ($p=0.018$), and past PCI ($p=0.002$). Length of stay, as calculated by the Mann-Whitney U Test, was also related to presence of confounders ($p= <0.001$).

**Table 4.** Baseline Characteristics in relation to presence of ST Confounders

| Variables | | Confounder Present (n=123) | Confounder Absent (n=627) | P Value |
|---|---|---|---|---|
| ***Demographics*** | | | | |
| **Age (years)** | Mean 59±17 | **68 ± 16** | 57 ± 16 | **<0.001** |
| **Sex (Male)** | 433 (58%) | 75 (61%) | 358 (57%) | 0.43 |
| **Race (Black)** | 301 (40%) | 50 (41%) | 251 (40%) | 0.69 |
| ***CAD Risk Factors*** | | | | |
| **Obesity class** | | | | 0.37 |
| Normal | 303(41%) | 55 (45%) | 248 (40%) | |
| Overweight | 160 (21%) | 28 (23%) | 132 (21%) | |
| Obese | 285 (38%) | 40 (32%) | 245 (39%) | |
| **Smoking History** | | | | 0.41 |
| Never | 307 (41%) | 56 (46%) | 251 (40%) | |
| Quit | 273 (37%) | 44 (36%) | 229 (37%) | |
| Current | 163 (22%) | 22 (18%) | 141 (23%) | |
| **Hypertension** | 547 (73%) | 108 (88%) | 439 (70%) | **<0.001** |
| **Diabetes Mellitus** | 210 (28%) | 49 (40%) | 161 (26%) | **0.001** |
| **Hyperlipidemia** | 262 (35%) | 56 (46%) | 206 (33%) | **0.007** |
| **CAD** | 256 (34%) | 71 (58%) | 185 (30%) | **<0.001** |
| **Angina** | 146 (20%) | 40 (33%) | 106 (17%) | **<0.001** |
| **History of MI** | 231 (31%) | 49 (40%) | 182 (29%) | **0.018** |
| **History of CHF** | 135 (18%) | 44 (36%) | 91 (15%) | **<0.001** |
| **Past PCI** | 172 (23%) | 43 (36%) | 129 (21%) | **0.002** |
| **Past CABG** | 70 (9%) | 29 (24%) | 41 (7%) | **<0.001** |
| ***Clinical Presentation*** | | | | |
| **Chest Pain** | 645 (86%) | 105 (85%) | 540 (86%) | 0.83 |
| **Shortness of Breath** | 215 (29%) | 42 (34%) | 173 (28%) | 0.15 |
| **Positive Initial Troponin** | 96 (13%) | 21 (17%) | 75 (12%) | 0.122 |
| **Serum $K^+$ Level** | | | | 0.28 |
| Hypokalemia | 113 (15%) | 14 (12%) | 99 (17%) | |
| Normal | 614 (82%) | 101 (84%) | 451 (79%) | |
| Hyperkalemia | 23 (3%) | 5 (4%) | 18 (3%) | |
| **LOS (median [IQR])** | 1.0 (0.5-3.0) | **2.0 (1.0-3.0)** | 1.0 (0.2-3.0) | **<0.001** |
| **PCI Done** | 65 (9%) | 13 (11%) | 52 (8%) | 0.41 |
| **CABG Done** | 9 (1%) | 3 (2%) | 6 (1%) | 0.17 |

## 5.6 SPECIFIC AIM 2(B)

As shown in figure 5, ST confounders are prevalent in all patients with chest pain irrespective to the etiology of chest pain. For example, 12% of patients with undifferentiated chest pain has at least 1 ST confounder; compared to 20% in those with ACS. Compared to patients with non-cardiac or undifferentiated causes of chest pain, those with cardiac-related cause of chest pain (ACS or non-ischemic cardiopulmonary disease) were more likely to have ST confounders on their ECG (chi-square =18.9, p<0.001).

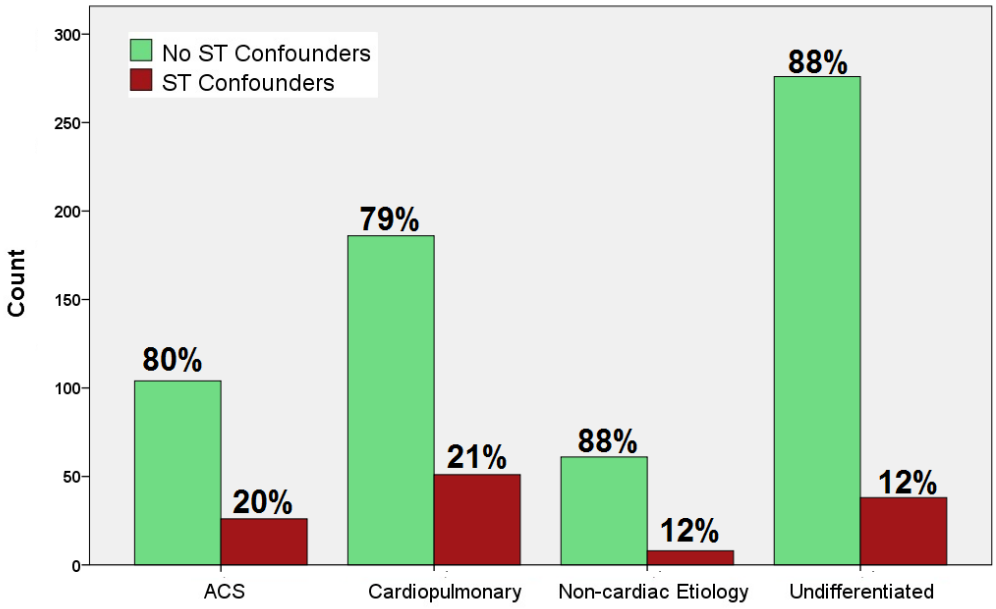


**Figure 5.** Relationship between presence of ST Confounders and Chest Pain Etiology

## 5.7 SPECIFIC AIM 2(C)

Finally, as shown in figure 6, the presence of ST confounders was not only prevalent among patients admitted for treatment but also among those discharged from ED or admitted to telemetry

for overnight observation. Among patients that were admitted for treatment, 22% of them had ST confounders. This means that about 1 in 5 patients that were admitted to hospital, ST-segment monitoring was of limited value. The presence of ST confounders was associated with a median of 1 day longer length of stay (Figure 7, p<0.001).

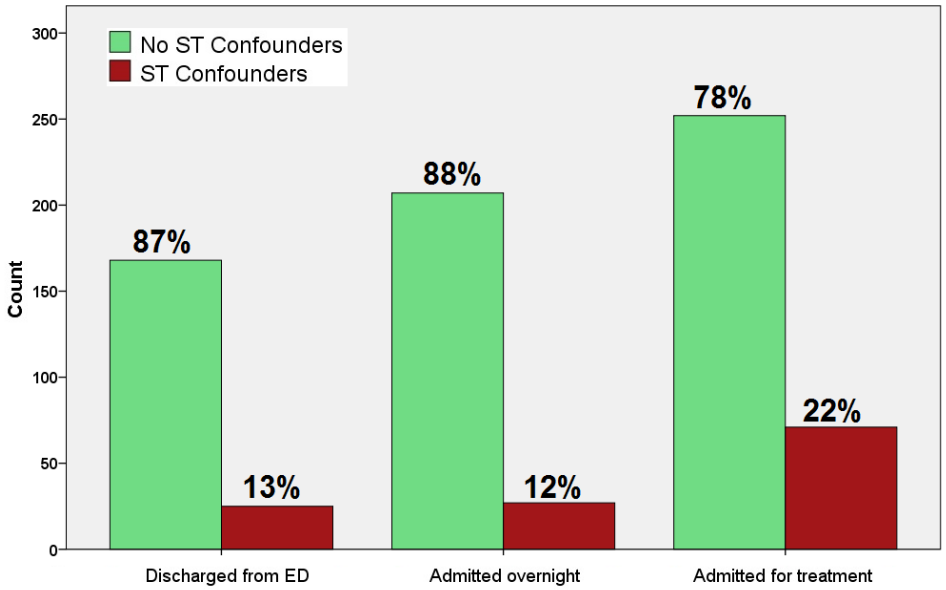


**Figure 6.** Relationship between presence of ST Confounders and Admission Status

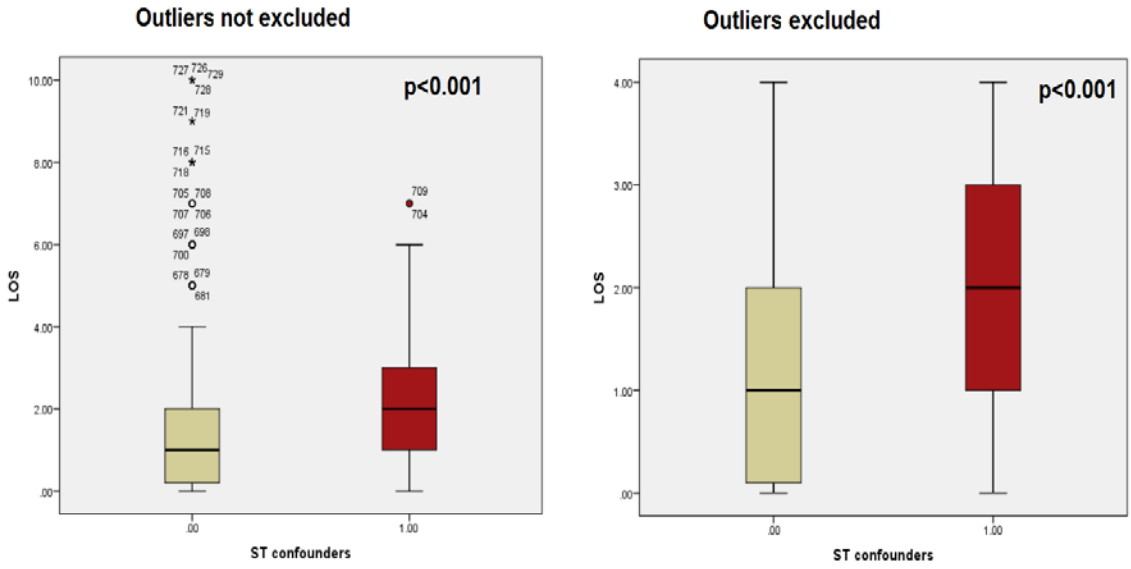


**Figure 7.** Relationship between presence of ST Confounders and LOS

# 6.0 DISCUSSION

## 6.1 OVERVIEW

This study was conducted to define the frequency of chest-pain patients with ST confounders and evaluate the clinical significance of these ECG abnormalities. In this secondary analysis of an existing data set, 750 patients that presented to the emergency department with chest pain were studied; majority of the patients were male and 40% were identified racially as black. The most prevalent final diagnosis for chest pain was undifferentiated chest pain, followed by non-ischemic cardiopulmonary, ACS, and non-cardiac related. Nearly 75% of patients were either admitted for treatment or overnight observation, with vast majority having a final diagnosis of non-ischemic chest pain. In comparison to Weinstock's study that found almost 50% of patients being admitted, this is a relatively large amount of admissions (Weinstock et al., 2015). About 1 in 6 patients overall had non-ischemic ST-segment changes. Older age, hypertension, coronary artery disease, angina, diabetes mellitus, hyperlipidemia, history of MI and CHF, past CABG and PCI were all related to the presence of ST confounders. Non-ischemic cardiopulmonary patients had the highest prevalence of ST confounders with about 1 in 5 having a confounder, but the ST confounders could be found among all etiological groups. 1 in 5 patients admitted for treatment had an ST confounder, which was significantly associated with increased length of stay.

## 6.2 SAMPLE CHARACTERISTICS AND MAGNITUDE OF PROBLEM

Some striking CAD risk factor data were that normal weight and obesity were almost equally prevalent and that a clear majority of the patients had hypertension, exemplifying that obesity class and hypertension are key components in CAD risk factor assessment. Additionally, over half of the patients that were admitted overnight had undifferentiated chest pain diagnoses. Although these patients pose a threat to healthcare economics related to the use of time and resources spent on finding a diagnosis, it can be seen in a positive light that people take chest pain seriously and seek medical attention. Results showed that about 16% of the sample size had an ST-segment monitoring confounder; this means that about 1 in 6 patients that come in to the ED with chest pain have an ECG abnormality that hinders cardiac telemetry monitoring from accurately detecting an acute myocardial ischemia.

## 6.3 RELATIONSHIPS BETWEEN ST CONFOUNDERS AND CLINICAL VARIABLES

Sex and race were not determinants of ST confounders presence, but age and CAD risk factors had significant relationships with the presence of confounders. Clinical presentation also did not have significant relationships to presence of ST confounders. The presence of confounders also had statistically significant relationships with chest pain etiologies; cardiac related etiology (ACS or non-ischemic cardiopulmonary) had the highest prevalence of ST confounders. Many etiologies in this category are chronic in nature, such as heart failure, as well as many ST confounders are also chronic as they are conduction related issues. Of the patients that were admitted for treatment,

1 in 5 had an ST confounder. If a nurse working on a telemetry monitored floor had a five-patient assignment, one of her patients may have an ST confounder that causes the ST-segment monitor to be constantly alarming, leading to alarm fatigue for the nurse. Clinicians may be misled by the alarms into thinking the patient is experiencing acute myocardial ischemia, which could result in unnecessary diagnostic testing, a longer hospital stay, and/or increased cost of care (Drew, Wung, Adams, & Pelter, 1998). Our data supports this notion and demonstrates that the presence of confounders is associated with a length of stay that is 1 day longer, which increases healthcare costs and potentially avoidable diagnostic testing.

## 6.4 LIMITATIONS

The parent study did not collect data on false alarms from monitors in telemetry units, which could have provided more in depth analysis of the relationship between the presence of confounders and the frequency of false alarms leading to alarm fatigue. Another future area for improvement is examining the relationship between the presence of ST confounders and important clinical outcomes, for instance rate of 30 day readmissions, re-infarctions, or mortality. These outcome data are being collected in the ongoing EMPIRE study, but they were not available for this secondary analysis.

## 6.5 CONCLUSION

This study opens the door to future research by quantifying the magnitude of the problem nurses face with patients that present with chest pain and have ST confounders. Although computerized telemetry cardiac monitoring is clinically valuable for observation in patients that present with chest pain, nurses need to understand their limitations for observing high-risk patients with non-ischemic ECG confounders. Future research should explore alternative methods of monitoring patients with ST confounders. Perhaps, more frequent vital signs should be taken on these patients or more frequent observation of serum electrolyte levels and cardiac biomarkers. Future research can examine the frequency of unnecessary treatment in ST-segment monitoring false alarms. Additionally, future research can investigate methods to enhance existing computerized algorithms specifically for patients that have ST-segment monitoring confounders.

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