CLINICAL INVESTIGATIONS

Electrocardiographic ST-segment Elevation: The Diagnosis of Acute Myocardial Infarction by Morphologic Analysis of the ST Segment

WILLIAM J. BRADY, MD, SCOTT A. SYVERUD, MD, CHARLOTTE BEAGLE, RN, MSN, ANDREW D. PERRON, MD, EDWARD A. ULLMAN, MD, CHRISTOPHER HOLSTEGE, MD, RALPH J. RIVIELLO, MD, ANNE RIPLEY, RN, CHRIS A. GHAEMMAGHAMI, MD

Abstract. Acute myocardial infarction (AMI) is one of many causes of ST-segment elevation (STE) in emergency department (ED) chest pain (CP) patients. The morphology of STE may assist in the correct determination of its cause, with concave patterns in non-AMI syndromes and non-concave waveforms in AMI. **Objectives:** To determine the impact of STE morphologic analysis on AMI diagnosis and the ability of this technique to separate AMI from non-infarction causes of STE. **Methods:** The electrocardiograms (ECGs) of consecutive ED adult CP patients (with three serial troponin I determinations) were interpreted in two-step fashion by six attending emergency physicians (EPs): 1) the determination of STE by three EPs followed by 2) STE morphologic analysis (either concave or non-concave) in those patients with STE. The impact of STE morphology analysis was investigated in the identification of AMI and non-AMI causes of STE. Acute myocardial infarction was diagnosed by abnormal serum troponin I values (>0.1 mg/dL) followed by a rise and fall of the serum marker; STE diagnoses of non-AMI causes were determined by medical record review. Interobserver reliability concerning STE morphology was determined. Study inclusion criteria included at least three troponin values performed in serial fashion no more frequently than every three hours, initial ED ECG, ED diagnosis, and final hospital diagnosis. **Results:** Five hundred ninety-nine CP patients were entered in the study, with 171 (29%) individuals having STE on their ECGs. Of the 171 patients who had STE, 56 had AMI, 50 had unstable angina pectoris (USAP), and 65 had non-coronary final diagnoses. Forty-nine patients had non-concave STE, 46 with AMI and three with USAP; no patient with a non-coronary diagnosis had a non-concave STE morphology. The sensitivity and specificity of the non-concave STE morphology for AMI diagnoses were 77% and 97%, respectively; the positive and negative predictive values for non-concave morphology in AMI diagnoses were 94% and 88%, respectively. Interobserver reliability in the STE morphology determination revealed a kappa coefficient of 0.87. **Conclusions:** A non-concave STE morphology is frequently encountered in AMI patients. While the sensitivity of this pattern for AMI diagnosis is not particularly helpful, the presence of this finding in adult ED chest pain patients with STE strongly suggests AMI. This technique produces consistent results among these EPs. **Key words:** electrocardiogram; ST-segment elevation; acute myocardial infarction. ACADEMIC EMERGENCY MEDICINE 2001; 8: 961–967

CHEST pain patients presenting to the emergency department (ED) are evaluated with the history, physical examination, and other selected diagnostic studies. One of these diagnostic studies, the electrocardiogram (ECG), is a time-honored tool used by the emergency physician (EP) not only to establish diagnoses but also to make therapeutic decisions, to predict risk of cardiovascular complication and death, and to choose appropriate inpatient disposition locations. As is obvious from this statement, numerous important clinical decisions rely on the EP's ability to interpret the ECG. The ability of the EP to correctly interpret the ECG in such patients directly and immediately impacts on management decisions as well as influ-

From the Department of Emergency Medicine, University of Virginia School of Medicine (WJB, SAS, CB, ADP, EAU, CH, RJR, AR, CAG), Charlottesville, VA.

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Address for correspondence and reprints: William Brady, MD, 3020 Cove Lane, Charlottesville, VA 22911. Fax: 804-982-4118; e-mail. wb4z@virginia.edu

ences patient outcome.[1–4] For example, the widely recognized benefits of rapid reperfusion therapy of acute myocardial infarction (AMI) rely heavily on this mastery of the ECG.

ST-segment elevation (STE) is perhaps the "most demanding" of the electrocardiographic features seen in the chest pain patient; it is "demanding" in that its presence must be explained and, if the etiology involves AMI, urgent therapeutic decisions must be made. Conversely, in the instance of the chest pain patient whose ECG demonstrates STE resulting from a non-infarction syndrome, the correct diagnosis must be made not only to offer appropriate management for that particular illness but also to avoid incorrect, potentially dangerous therapies. While STE is a not uncommon finding on the ECG of the chest pain patient, its cause does not always involve AMI. In fact, AMI is a less-than-frequent cause of electrocardiographic STE in the chest pain patient.[5–8]

In the setting of the chest pain patient with electrocardiographic STE, the EP can arrive at the correct diagnosis using any number of general or advanced electrocardiographic interpretative tools. General electrocardiographic interpretative skills are used; in many instances, this approach suffices. In other cases, advanced interpretation methods are necessary. In the patient with equivocal or questionable STE, serial ECGs or ST-segment trend monitoring may be used to demonstrate either rapid evolution of the abnormality as seen in AMI or a lack of electrocardiographic change as encountered in the non-infarction syndrome.[9–15] The ECG of the patient with confounding electrocardiographic patterns such as left bundle branch block (LBBB) or ventricular paced rhythm (VPR) is best approached with both a sound knowledge of the appropriate ST-segment/T-wave morphologies and a familiarity with the clinical decision guides formulated to assist in these complicated scenarios.[16,17] It has also been suggested that ST-segment depression—termed either reciprocal ST-segment depression or reciprocal change—in the patient with electrocardiographic coincident STE may assist in establishing a diagnosis of AMI.[6,18]

Another electrocardiographic tool potentially useful in the patient with chest pain and STE is waveform analysis of the elevated ST segment. This technique involves a morphologic examination of the initial upsloping portion of the ST segment in the setting of electrocardiographic STE, assuming that AMI and non-AMI syndromes will manifest different configurations of this important portion of the electrical cardiac signal. Acute myocardial infarction is suggested to manifest as convex or obliquely straight (i.e., non-concave) ST-segment configurations, while non-AMI causes of STE manifest as concave morphologies (Fig. 1). This technique has been suggested in the past[18–20] though, to the best of our knowledge, has never been explored in the clinical setting. We undertook the following study to investigate the use of the ST-segment morphologic analysis in adult chest pain patients with STE.

METHODS

Study Design. A retrospective study investigating the use of STE waveform analysis as an adjunct in

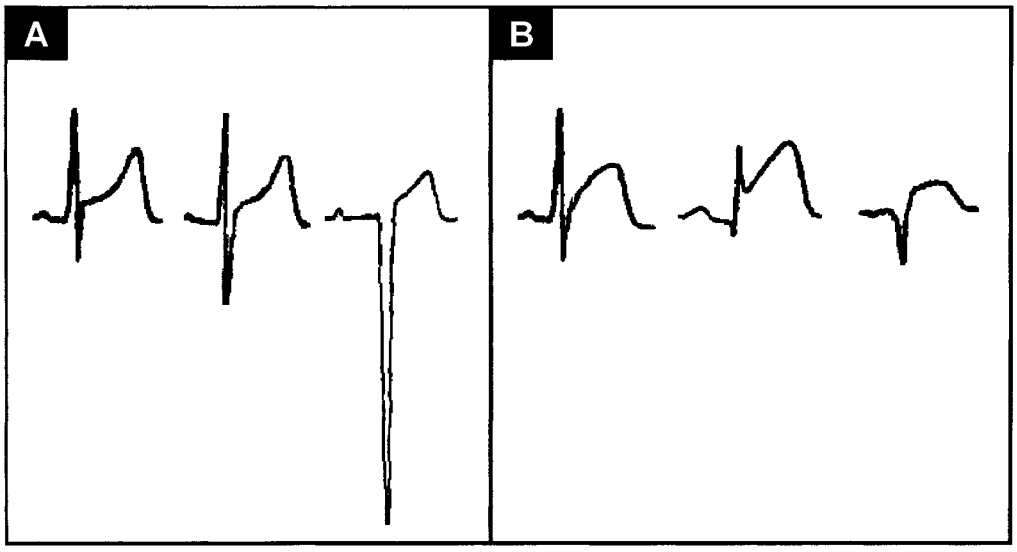

Figure 1. The different ST-segment elevation morphologies in chest pain patients. **A.** Concave morphology—consistent most often with non–acute myocardial infarction (non-AMI) causes of ST-segment elevation, such as benign early repolarization, acute pericarditis, and left ventricular hypertrophy pattern, respectively, in this figure. **B.** Non-concave morphology—most often consistent with AMI.

the diagnosis of AMI was performed. Prospectively, consecutive ED chest pain patients who underwent the rule-out myocardial infarction (R/O MI) evaluation were entered in the study. For the purposes of this study, the review of the ECG and the medical records in final diagnosis occurred retrospectively. The study was reviewed by the institution's internal review board and considered exempt from informed consent due to its retrospective nature.

Study Setting and Population. The setting of the study was a university hospital ED with an annual patient volume of 60,000 serving a primarily suburban and rural area with an urban section of approximately 40,000 persons; the general population of the area is approximately 120,000. The chest pain center (CPC) manages an annual volume of 4,000 patients who are ED patients, representing approximately 7% of the general ED annual census. Emergency department patients with a chief or secondary complaint of chest pain are initially evaluated in the CPC; ED triage criteria for initial CPC bed assignment includes age more than 30 years with a nontraumatic etiology of the chest pain. The ED is staffed by emergency medicine resident- and attending-level physicians 24 hours a day. The CPC is located within the ED; patients in the CPC are under the direct supervision of the ED attending physician.

The study population consisted of consecutive adult chest pain patients presenting to a university hospital ED with a CPC who underwent the R/O MI evaluation. The R/O MI evaluation included serial tropinin I determinations (for a minimum of three determinations) and ECGs. The initial ECG performed in the ED-based CPC was used as the study ECG; subsequent ECGs were not reviewed. The R/O MI evaluation either occurred in the CPC or was initiated in the CPC with completion on inpatient wards. Study inclusion criteria included at least three troponin values performed in serial fashion no more frequently than every three hours, initial ED ECG, ED diagnosis, and final hospital diagnosis. Creatinine phosphokinase values were not obtained for the study patients.

Measurements. The ECGs of the study patients were interpreted in a two-step fashion by six attending EPs: 1) the determination of STE by three EPs followed by 2) STE morphologic analysis (either concave or non-concave) in those patients with STE. In the initial review, the presence or absence of STE was noted. Three attending EPs—who knew only the patient's age, gender, and complaint of chest pain—reviewed the ECGs using the following criteria for STE in at least two anatomically contiguous leads: 1) at least 1 millimeter (mm) of STE in leads I, II, III, aVl, aVf, V5, and/or V6; and 2) at least 2 mm of STE in leads V1, V2, V3, and/or V4. Majority opinion prevailed in terms of determining the presence or absence of STE—i.e., at least two attending EPs must have indicated the presence of STE for the ECG to be classified as "ECG with ST segment elevation." The anatomic distribution of the STE was noted on the ECG.

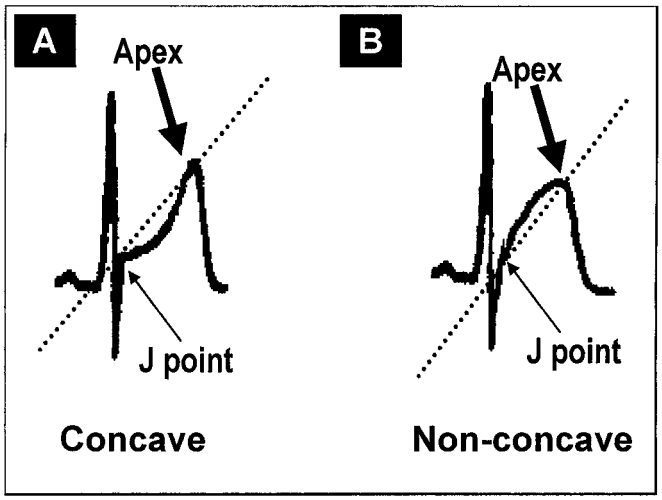

Figure 2. Determination of ST-segment morphology was made by noting two points on the initial, upsloping portion of the elevated ST segment: the J point (the point at which the QRS complex ends and the ST segment begins) and the apex of the ST-segment/T-wave complex. A line is drawn between the two points with the morphology termed as follows: concave morphology (benign early repolarization) if area is noted below the line and above the ST segment (**A**), or non-concave morphology (acute myocardial infarction) if area is noted above the line and below the ST segment or the line falls directly on the ST segment (**B**).

Those ECGs demonstrating STE were then reviewed by three different attending EPs—who knew only the patient's age, gender, and complaint of chest pain. The second phase of the review considered the morphology of the elevated ST segment, classifying the ECGs as either concave or non-concave. The morphologic analysis was performed in the following fashion (Fig. 2). Determination of ST-segment morphology was made by noting two points on the initial, upsloping portion of the elevated ST segment: the J point (the point at which the QRS complex ends and the ST segment begins) and the apex of ST-segment/T-wave complex. A line is drawn between the two points with the morphology termed as follows: 1) concave morphology if area is noted below the line and above the ST segment (Fig. 2A) or 2) non-concave morphology if area is noted above the line and below the ST segment or the line falls directly on the ST segment (Fig. 2B). Majority opinion prevailed in terms of determining the morphology of the elevated ST segment (either concave or non-concave)

TABLE 1. Statistical Characteristics of ST-segment Elevation Morphology as an Adjunct to the Diagnosis of Acute Myocardial Infarction

Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
77%	97%	94%	88%

—i.e., at least two attending EPs must have indicated a particular morphology of STE for the ECG to be classified as such.

Acute myocardial infarction was diagnosed by abnormal serum troponin I values (>0.1 mg/dL) followed by a rise and fall of the serum marker, which occurred in the setting of a chest pain chief complaint and an abnormal ECG.[21] Unstable angina pectoris (USAP) was defined according to the clinical diagnosis rendered by the treating clinicians; confirmation of troponin I values was made in these cases to ensure that no AMI cases were included in the USAP category. Non-coronary diagnoses were recorded based on the treating clinicians' diagnoses; confirmation of troponin I values was made in these cases to ensure that no AMI cases were included in the non-coronary category.

Data Analysis. The impact of STE morphology analysis was investigated in the identification of AMI and non-AMI causes of STE. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated using standard formulas for the ability of the ST-segment waveform analysis to detect AMI. Interobserver reliability among the EP ECG interpreters concerning the presence or absence of STE and the STE morphology was determined. A kappa statistic was calculated for each pair of observers. Since three independent observers rated each variable, three kappas were calculated—one for each pair of observers. The interrater reliability reported is the mean kappa from three kappa coefficients. The rates of occurrence of the various causes of STE as well as the determinations of STE and STE morphology were also calculated.

RESULTS

Five hundred ninety-nine patients were entered in the study; all had medical records available for review. The mean age for the study population was 62.1 years, with 54% male gender. One hundred seventy-one (29%) individuals had electrocardiographic STE; this patient group, the group used for data analysis, had a mean age of 61.3 years, with 57% male gender. From the perspective of final diagnosis, of the 171 patients with STE, 56 (33%) had AMI, 50 (29%) had USAP, and 65 (38%) had non-coronary diagnoses. From the perspective of ST-segment morphology, of the 171 patients with STE, 49 (29%) had non-concave STE (Table 2) with the following final hospital diagnoses: 46 (94%) with AMI and three (6%) with USAP; no patient with a non-coronary diagnosis had a non-concave morphology. One hundred twenty-two (71%) patients had concave morphologies. Of the patients with STE AMI, 46 (82%) had non-concave morphologies and ten (18%) concave morphologies. The sensitivity and specificity of the non-concave STE morphologies for AMI diagnosis were 77% and 97%, respectively; the positive and negative predictive values for the non-concave morphologies in AMI diagnosis were 94% and 88%, respectively (Table 1).

The various electrocardiographic diagnoses responsible for the STE include AMI, 56 (32.7%); left ventricular hypertrophy (LVH), 46 (26.9%); bundle branch block, 20 (11.7%); ventricular paced rhythm, six (3.5%); benign early repolarization, 21 (12.3%); pericarditis, six (3.5%); left ventricular aneurysm, six (3.5%); and other, ten (5.8%) (Table 2).

Interobserver reliability in the determination of STE was 0.83 (kappa coefficient) and in the STE morphology determination was 0.87 (kappa coefficient).

DISCUSSION

The morphology of the elevated ST segment may hold additional useful information for an emergent interpretation of ECGs in ED chest pain patients. We have pursued the morphology of the elevated segment as an additional diagnostic tool in chest pain patients with possible AMI, while other investigators have attempted to use this information in a prognostic sense. Kosuge et al.[22] investigated the morphology of the ST segment as a marker of infarct size and left ventricular function in patients with AMI. These investigators reviewed the ECGs of 77 patients with a first anterior wall AMI immediately after presentation and prior to any reperfusion therapy; these ECGs were then divided into three categories as a function of the ST-segment morphology, or "pattern," as termed by the authors. The three pattern categories included concave, convex, and straight—similar to our delineation into concave and non-concave, corresponding to Kosuge et al.'s convex and straight.[22] Used as a prognostic marker, the pattern of the ST segment in these AMI patients was useful. Among the patients with reperfused anterior AMI, left ventricular function was excellent in patients with concave-type STE, intermediate in those with straight-type STE, and relatively poor in those with convex-type STE at hospital discharge.[22]

Kosuge et al.[22] reported that a significant minority of their patients had a concave ST-segment morphology on the initial ECG in the setting of AMI. They found that most anterior AMI ST-segment patterns were either convex or straight (what we have termed non-concave)—totaling 69% in their population. In our study population, we found a much higher rate of non-concavity—82% of AMI patients had a non-concave ST-segment morphology. The difference in the rates of occurrence of the ST-segment patterns may be explained, at least in part, by the anatomic distribution of the infarcts under study. In our work, we looked at all STE AMI presentations (anterior, inferior, lateral, and combinations), while Kosuge et al.[22] considered only first AMI of the anterior wall.

The application of ST-segment morphologic analysis has been suggested as a useful tool in distinguishing between AMI and non-AMI causes of STE,[18–20,23] although it has never been tested in any scientific trial. We found that morphologic analysis, namely, the presence of a non-concave ST segment, has a rather poor sensitivity (77%) for the diagnosis of STE AMI but an impressive specificity (97%) for the diagnosis of acute infarction. While the majority of AMI patients had a non-concave ST-segment morphology, a significant minority had a concave STE pattern—hence the poor sensitivity. This electrocardiographic interpretative tool is therefore a poor choice to "rule-out" acute infarction in a patient with chest pain and STE. Conversely, its quite high specificity for the diagnosis of AMI makes it an ideal tool for "ruling in" AMI in the patient with chest pain and STE. In general, specific tests are very useful to confirm the diagnosis of an illness when its presence has been suggested by other data. In this particular instance, the patient with chest pain whose electrocardiographic analysis shows STE (the other data) has AMI confirmed by the presence of a non-concave STE pattern.

The predictive values of this electrocardiographic tool were also rather high, in particular, the positive predictive value—94% in this study. Positive predictive value is the probability of disease presence in a patient with a positive or abnormal test finding; in this case, the finding of a non-concave ST-segment morphology suggests AMI with a very high probability. It is important to note that the four patients who had non-concave STE and a non-AMI etiology responsible for the STE had unstable angina as their final hospital diagnosis. Thus, the positive predictive value of non-concave STE for acute coronary syndromes is 100%. Conversely, a negative predictive value is the chance of not having the disease when the test is negative or normal; though less robust, the negative predictive value in this study population confirms previous assumptions[18–20,23] stating that the absence of a non-concave morphology (i.e., the presence of a concave pattern) suggests a non-AMI cause of the STE.

TABLE 2. Causes of Electrocardiographic ST-segment Elevation among 171 Patients

Electrocardiographic Syndrome	Number of Patients
Acute myocardial infarction	56
Left ventricular hypertrophy	46
Bundle branch block	20
Benign early repolarization	21
Pericarditis	6
Left ventricular aneurysm	6
Paced rhythm	6
Other	10

ST-segment elevation is a common finding on the ECG of the chest pain patient; its cause less often involves AMI.[8] The occurrence of numerous other noninfarctional STE syndromes only reinforces the point that STE is an insensitive marker of AMI. One out-of-hospital study of adult chest pain patients demonstrated that the majority of patients who had STE on their ECGs did not have AMI as a final hospital diagnosis; rather, LVH and left bundle branch block accounted for the majority of the cases.[6] Further, in a review of adult ED chest pain patients with STE on the ECG, STE resulted from AMI in only 15% of this population; LVH, seen in 30% of adult chest pain patients, was the most frequent cause of this STE.[8] In the coronary care unit population, Miller and colleagues[7] demonstrated that STE was diagnostic for acute infarct in only half of patients with a past history of ischemic heart disease with such ST-segment changes.

In our study population, we found similar rates of occurrence of most syndromes, with the exception of AMI and bundle branch block, when compared with our previous work investigating the cause of STE in the chest pain patient.[8] ST-segment elevation due to AMI was noted more frequently in this study population, 31% of cases compared with the previous report of 15% of instances.[8] This discrepancy can be explained by sampling error as well as the specific patient population under scrutiny. In this most recent population, we included only patients undergoing the R/O MI evaluation, while the previous work included all adult ED chest pain patients with STE. As expected, the prevalence of AMI was higher in the selected population of patients undergoing R/O MI when compared with the general ED population with chest pain and STE. Bundle branch block occurred less often in this study population for unknown reasons other than may be explained by sampling error.

LIMITATIONS AND FUTURE QUESTIONS

This study is limited by several issues, primarily involving study design. The structure of the study itself—prospective identification of the patient population with retrospective ECG review—is a partially hypothetical, contrived situation, unlike the actual ED encounter. In a real-time interpretation of the ECG, the EP has numerous other diagnostic tools that may assist in arriving at the correct etiology of the STE—such as an expanded history, past medical history, the physical examination, both prior and serial ECGs, various other diagnostic studies, and consultants. Essentially, the ECG is a test that must be interpreted in the context of a particular patient event. The study design clearly removed this option from the participants.

The other major limitation is the testing of this technique by a small number of academic EPs—and its generalization to the larger EP pool. We did demonstrate a very high rate of interobserver reliability, in terms of both determining the presence or absence or STE and assessing the morphology of the ST segment.

We did not review previous ECGs in this study. A patient might have a baseline electrocardiographic pattern with STE of either morphology. Among patients with non-STE AMI, the study presentation with electrocardiographic STE might then reflect an ST-segment abnormality that is unrelated to AMI; the patient would actually be experiencing a non-STE AMI, with the STE's resulting from a non-infarction pattern. Such a presentation would obviously alter the test characteristics of this morphologic analysis. We also did not review the subsequent, or serial, ECGs performed in the typical care of the study patients. The additional ECGs are frequently of value in arriving at the diagnosis. The impact of this technique when applied to serial ECGs is unknown and represents an area of future endeavor.

The most significant future issue must focus on the applicability of this electrocardiographic technique in a real-time scenario by EPs in general practice.

CONCLUSIONS

A non-concave STE morphology is frequently encountered in AMI patients, representing the most frequent morphology of the elevated ST segment. The EP must realize, however, that a significant minority of patients with AMI will present with a concave morphology of the ST segment. While the sensitivity of the non-concave STE morphology for AMI diagnosis is not particularly helpful, the presence of this finding in adult ED chest pain patients with ST-segment elevation strongly suggests acute infarction with a very high positive predictive value. This technique produces consistent results among these EPs.

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Errata

Some abstracts for the May 2000 and May 2001 SAEM annual meeting issues of *Academic Emergency Medicine* contained errors as they were received at the publisher.

In abstract 334 published in the May 2000 issue (Mycyk MB, Perera TB, Ulrich AS, Mitchell P, Case B. Identification of patient preferences during death notification in the emergency department [abstract]. Acad Emerg Med. 2000; 7:538), the last-listed author's name should be spelled Benjamin Kase (not "Case").

In abstract 475 published in the May 2001 issue (Milzman D, Smith R, Calloway D, Thistle T, Greenberg D, Glasser E. Implementation of SAEM medical student curriculum to first-year students: results of an EM crash course [abstract]. Acad Emerg Med. 2001; 8:588), the third-listed author's name should be David W. Callaway (not "Dave Calloway").

Abstract 244 published in the May 2001 issue was the wrong one. The correct abstract appears below.

Feasibility and Predictive Value of Combining Two Chest Pain Algorithms Using Bayesian Theory *Rebecca R Roberts, Jeffrey J Schaider, Brendan Reilley, Arthur Evans, Krishna Das, Dobroslawa T Reschke, Kyle Prioleau, Joey Sebollena, Scott Kono, Linda M Kampe; Cook County Hospital/Rush University, Chicago, IL*

Background: Goldman's algorithm (GM) predicts cardiac complication risk (c-risk) in hospitalized patients (NEJM, 1996). The 4 categories and c-risk rates are: GM1: 0.6%, GM2: 4%, GM3: 8%, GM4: 16%. They advised observation for GMI, telemetry for GM2–3, and CCU for GM4. The Diamond & Forrester algorithm (D&F) predicts coronary artery disease (CAD) risk (NEJM, 1979). We hypothesized that c-risk predictions within GM groups can be adjusted by the underlying risk of CAD, as complications occur only in those with CAD. Because the two algorithms were developed independently, a potential limitation would be if GM c-risk closely correlated with risk of CAD, no new data would be added by D&F. **Objectives:** 1. To determine whether c-risk by GM correlates with CAD risk; 2. To calculate c-risks groups within each GM group based on risk of CAD; and 3. To compare our sample with the original study to test feasibility. **Methods:** 1,549 ED cases were prospectively enrolled using a decision aid combining GM c-risk and D&F CAD risk. Each GM group was further stratified into 4 D&F groups with the following CAD risks: low (l): 4%, mod (m): 27%, high (h): 77%, and known (k): 100% for a total of 16 risk groups. Complications in each GM group were applied only to patients with CAD in each D&F group. **Results:** GM1 patients had the following numbers (95% CI) in each D&F risk group: l: 12% (10–14), m: 35% (31–39), h: 45% (41–49), k: 8% (6–10). GM2 had: l: 5% (3–7), m: 20% (16–24), h: 52% (47–57), k: 23% (19–27). GM3: l: 9% (6–12), m: 50% (45–55), h: 27% (23–31), k: 14% (10–18). GM4: l: 26% (19–33), m: 15% (9–21), m: 22% (15–29), k: 37% (29–45). The ability to differentiate c-risk increased from 0.6–16% with GM alone to 0.07–36% using both. Proportions in each CAD group (sample vs. original) were: low: 11% vs 9%; mod: 36% vs 33%; high: 55% vs 58%. For GM groups: GMI: 45% vs 55%; GM2: 21% vs 19%, GM3: 24% vs 18%, GM4: 10% vs 7%. **Conclusions:** The combination of 2 risk assessment algorithms is feasible and can potentially improve use of hospital resources.