**Points & Pearls**

- The EDACS accelerated diagnostic protocol (EDACS-ADP) study included any symptoms lasting longer than 5 minutes that the attending physician thought were worth working up for possible acute coronary syndromes (ACS). This is a broader definition than other studies, such as the Vancouver Chest Pain Score, which only included chest pain patients.
- Like other chest pain evaluation studies, the primary outcome for the EDACS-ADP study was a major adverse cardiac event (MACE), as defined by any of the following:
  - ST-elevation or non-ST-elevation myocardial infarction
  -requiring an emergency revascularization procedure
  - Death from cardiovascular causes
  - Ventricular arrhythmia
  - Cardiac arrest
  - Cardiogenic shock
  - High atrioventricular block
- The goal of these rules is to identify a low-risk population of patients who need less testing than other, higher-risk patients. As a rule-out calculator, the EDACS is good at identifying who is relatively safe to go home (highly sensi-

**Why to Use**

Patients requiring serial blood testing (serial troponin markers, typically at 0 and 6 hours, to rule out myocardial infarction) and further risk stratification require an extended ED evaluation or hospital admission, leading to crowding, bed allocation problems, and exposure of patients to side effects of increased testing. The study authors were able to find a group of low-risk patients (~45%) who could be safely discharged from the ED after 2 biomarkers just 2 hours apart, along with ECG, history, and physical examination.

**When to Use**

Use in patients with chest pain or other anginal symptoms requiring evaluation for possible ACS, who may potentially be at low risk and appropriate for early discharge from the ED.

**Next Steps**

- For low-risk patients, consider other causes of chest pain due to aortic, esophageal, pulmonary, cardiac, abdominal, or musculoskeletal sources prior to discharge.
- For non–low-risk patients, physicians should use best judgment to work up and treat as per usual chest pain protocols, including but not limited to consideration of aspirin, nitroglycerin, and serial ECGs and biomarkers.

**Abbreviations:** ED, emergency department; ECG, electrocardiogram.
tive), but not good at identifying who does have the disease (not terribly specific). The fairly extreme goal of ≥ 99% sensitivity was achieved in the study (see the Evidence Appraisal section below).

- The score was initially created without electrocardiogram (ECG) or biomarkers. These were added into the EDACS-ADP, which includes an ECG and troponin at 0 hours and at 2 hours.
- The score was internally validated in the original paper, but has not yet been externally validated.

Advice

Barring other concerning features for ACS or other life-threatening causes of chest pain (pneumothorax, pulmonary embolism, cardiac tamponade, aortic dissection, esophageal rupture, etc), patients who meet the low-risk criteria can be considered for discharge after negative 0-hour and 2-hour troponin testing, with close follow-up by a primary care physician.

Patients who do not meet the low-risk criteria cannot be ruled out using the EDACS or EDACS-ADP. As a rule-out calculator, the EDACS does not provide definitive guidance for treatment of patients who fail the rule, so physicians should use best judgment and follow other evidence-based chest pain guidelines.

Critical Actions

Patients deemed to be at low risk are safe for discharge to early outpatient follow-up investigation, or to proceed to earlier inpatient testing. For patients who are not at low risk, physicians should use best judgment, as this rule-out calculator was not designed to “rule in” patients with ACS. Physicians cannot use the EDACS to rule out ACS.

Evidence Appraisal

The EDACS-ADP was prospectively validated in the original paper (Than et al, 2014), but would be strengthened by an external validation as well. The EDACS-ADP was 99% to 100% sensitive for correctly identifying patients as low-risk and identified 45% of its cohort as low-risk. This is much higher than other emergency department-based risk scores such as the HEART Score (history, ECG, age, risk factors, troponin), the Vancouver Chest Pain Score, ADAPT (Two-hour Accelerated Diagnostic Protocol to Assess Patients with Chest Pain Symptoms Using Contemporary Troponins as the Only Biomarker), the Marberg Score, and the GRACE (Global Registry of Acute Coronary Events ) ACS Risk Score.

In the EDACS-ADP cohorts, the prevalence of MACE in the study overall was 13% to 15%.

EDACS may help rule out ACS in patients with chest pain, but still requires strong external validation before widespread use.

Selected Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ACS</td>
<td>Acute coronary syndromes</td>
</tr>
<tr>
<td>ADAPT</td>
<td>Two-hour Accelerated Diagnostic Protocol to Assess Patients with Chest Pain Symptoms Using Contemporary Troponins as the Only Biomarker</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
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<tr>
<td>EDACS</td>
<td>Emergency Department Assessment of Chest Pain Score</td>
</tr>
<tr>
<td>EDACS-ADP</td>
<td>Emergency Department Assessment of Chest Pain Score accelerated diagnostic protocol</td>
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<td>GRACE</td>
<td>Global Registry of Acute Coronary Events [Risk Score]</td>
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<tr>
<td>HEART</td>
<td>History, ECG, Age, Risk Factors, Troponin [Score]</td>
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<td>MACE</td>
<td>Major adverse cardiac event</td>
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Calculator Creator

Martin Than, MD
Click here to read more about Dr. Than.

References

Original/Primary Reference


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Related Calculators

- Click here to access the ADAPT Protocol for Cardiac Event Risk.
- Click here to access the GRACE ACS Risk and Mortality Calculator.
HEART Pathway for Early Discharge in Acute Chest Pain

Introduction: The HEART Pathway was designed to aid in efficiently evaluating patients with acute chest pain, using the previously validated HEART Score.

Why to Use
Chest pain is one of the most common and potentially life-threatening chief complaints in emergency medicine. Many patients presenting with chest pain undergo unnecessarily extensive and costly evaluations to rule out ACS. The HEART Pathway can reduce the number of prolonged and invasive evaluations, while maintaining high sensitivity and negative predictive value for ACS.

Unlike other scoring systems such as the TIMI Risk Index or the GRACE Risk Score, the HEART Pathway is designed to predict the likelihood of ACS in the patient presenting to the ED with acute chest pain. TIMI and GRACE scores are used to risk stratify patients who have been diagnosed with ACS.

When to Use
• Use in patients aged ≥ 21 years presenting with symptoms suggestive of ACS.
• Do not use in patients with new ST-segment elevation ≥ 1 mm or other new ECG changes, hypotension, life expectancy < 1 year, or noncardiac medical/surgical/psychiatric illness determined by the provider to require admission.

Next Steps
• Low-risk patients with a follow-up troponin (at 3 hours) can be considered for safe discharge home with appropriate follow-up.
• High-risk patients require admission, serial cardiac biomarkers and ECG, and cardiology consult.

Abbreviations: GRACE, Global Registry of Acute Coronary Events [Risk Score]; TIMI, Thrombolysis in Myocardial Infarction [Risk Index].

Any patient presenting with chest pain and subsequently discharged should be informed that even with a negative workup, there is still a small risk of ACS. Patients should have close follow-up arranged and be given extensive return precautions prior to discharge.
Critical Actions
Clinician judgment should prevail, even if patients are deemed to be at low risk by the HEART Pathway. If there is some other cause for concern for an acute cardiac event, workup should be individualized to the patient.

All patients presenting to the ED with chest pain concerning for ACS should receive aspirin unless there is an absolute contraindication, such as known allergy, active bleeding, or if the patient has received a therapeutic aspirin dose prior to arrival.

Evidence Appraisal
The HEART Pathway was developed by Mahler et al in 2015 in a randomized controlled single-center trial. The control arm was managed at the discretion of care providers encouraged to follow American College of Cardiology/American Heart Association guidelines for acute chest pain.

The use of the HEART Pathway in this study was designed to mimic the real world in that it was used as an accelerated diagnostic pathway. Patient care was at the discretion of the healthcare provider and not mandated by the outcome of the HEART Pathway.

There were 282 patients studied, with 141 patients in each treatment group. The primary outcome was the rate of objective cardiac testing (stress test, coronary computed tomography angiogram, or invasive coronary angiography) within 30 days of presentation. Secondary outcomes were early discharge rate, index length of stay, cardiac-related recurrent ED visits, and nonindex hospitalization at 30 days.

The rate of objective cardiac testing in the HEART Pathway group was 12% less than the usual care group. The rate of early discharge in the HEART Pathway was 21% higher than the usual care group. The index length of stay was 12 hours shorter using the HEART Pathway. There was no significant difference between the 2 groups for cardiac-related recurrent ED visits or nonindex hospitalization at 30 days.

The use of the HEART Pathway compared to usual care, using the same dataset as the original HEART Pathway trial. There were 270 patients studied. Billing data were missing for 12 patients from the original study. Cost metrics considered in each group were index visit cost, total cost at 30 days, cardiac-related healthcare cost at 30 days, cardiac and noncardiac diagnostic testing cost, ED cost, inpatient cost for index visit, and outpatient cost. HEART Pathway patients had a significantly lower mean and median cost for both index visit and 30-day follow-up. There was no significant difference between the median and mean costs of the other metrics. Average savings per patient was $216, using the HEART Pathway. On a larger scale, this would mean approximately $2 billion in savings per year for undifferentiated chest pain.

Mahler et al in 2017 also published a secondary analysis looking at high-sensitivity cardiac troponin I and high-sensitivity cardiac troponin T. The study compared risk stratification using cardiac troponin I versus high-sensitivity cardiac troponin I and high-sensitivity cardiac troponin T in calculating the HEART Pathway score. Blood samples were sent for troponin I, high-sensitivity troponin I, and high-sensitivity troponin T for 133 patients.

All of the troponin assays had poor sensitivity for predicting MACE when used separately from the HEART score. There was no difference in the predicted risk of MACE between the use of serial troponin I and 3-hour high-sensitivity troponin I in the HEART Pathway. Using high-sensitivity troponin T in the HEART Pathway led to 1 patient with a non-ST-segment elevation myocardial infarction being misclassified as low-risk. The study found the HEART Pathway using serial troponin I or 3-hour high-sensitivity troponin I to have sensitivity and negative predictive value of 100% for 30-day MACE. Although hs-cTnT use in the HEART Pathway caused a non-ST-segment elevation myocardial infarction to be misclassified as low-risk, the reduction in sensitivity was not statistically significant, given the small study population. The authors recommend further appropriately powered studies to determine small differences in the accuracy of the high-sensitivity troponin assays.

Calculator Creator
Simon A. Mahler, MD
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References
Original/Primary References

Other References

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HEART Score for Major Cardiac Events

Introduction: The HEART Score predicts 6-week risk of major adverse cardiac events, including all-cause mortality, myocardial infarction, or coronary revascularization, and can be used to identify patients who are safe for discharge.

Points & Pearls

- The HEART Score helps emergency department (ED) providers risk stratify patients with chest pain into low-, moderate-, and high-risk groups.
- HEART is an acronym of its components: history, ECG, age, risk factors, and troponin. Each of these components is scored with 0, 1, or 2 points.
- It was designed to risk stratify patients with undifferentiated chest pain, not those already diagnosed with acute coronary syndromes (ACS).
- The HEART Score helps identify patients with higher risk of having a major adverse cardiac event (MACE) within the following 6 weeks. MACE is defined as all-cause mortality, myocardial infarction, or coronary revascularization.
- The user needs some experience taking a detailed chest pain history and reading electrocardiograms (ECGs) to adequately apply these 2 components of the score.
- The HEART Score is sometimes compared to the TIMI (Thrombolysis in Myocardial Infarction) Risk Score for UA (unstable angina) / NSTEMI (non-ST-segment elevation myocardial infarction) and the GRACE (Global Registry of Acute Coronary Events) ACS Risk Score, which are older ACS scores; however, the latter 2 scores are different from the HEART Score in that they measure risk of death for patients with ACS.
- The HEART Score outperforms the TIMI Risk Score for UA/NSTEMI, safely identifying more low-risk patients.

Why to Use

As one of the most commonly encountered ED complaints, chest pain often leads to extensive workup, with long ED or inpatient stays. In addition, it often leads to high resource utilization to try to determine which patients have life-threatening pathology. The HEART Score facilitates communication between providers and, more objectively, risk stratifies patients into low-, moderate-, and high-risk categories. This helps guide management, leading to better resource utilization, shorter hospital and ED stays for low-risk patients, and earlier interventions for moderate- and high-risk patients.

When to Use

The HEART Score can be applied to any ED patient presenting with chest pain whom the physician deems appropriate to evaluate for possible ACS.

Next Steps

- **Scores 0-3:** 0.9% to 1.7% risk of adverse cardiac event. In the HEART Score study, these patients were discharged (0.99% in the retrospective study, 1.7% in the prospective study).
- **Scores 4-6:** 12% to 16.6% risk of adverse cardiac event. In the HEART Score study, these patients were admitted to the hospital (11.6% in the retrospective study, 16.6% in the prospective study).
- **Scores ≥ 7:** 50% to 65% risk of adverse cardiac event. In the HEART Score study, these patients were candidates for early invasive measures (65.2% in the retrospective study, 50.1% in the prospective study).

Abbreviation: HEART, History, ECG, Age, Risk Factors, Troponin (Score).
Critical Actions
Do not use if the ECG shows new ST-segment elevation requiring immediate intervention or with clinically unstable patients.

Evidence Appraisal
The HEART Score was originally developed by Backus et al (2008) in a cohort of 122 patients with chest pain in an ED setting. The study included any patients admitted to the ED due to chest pain, irrespective of age, prehospital assumptions, and previous medical treatments. It excluded patients with chest pain and significant ST segment elevations. End points in this study were acute myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft, and death. HEART scores of 0-3 points confer a risk of 2.5% for any end point, and therefore were used to support discharge from the ED. Conversely, HEART scores of 4-6 points confer a risk of 20.3% for any end point, implying admission for clinical observation is necessary. HEART scores of ≥ 7 points have a risk of 72.7% for any end point, and support early invasive strategies.

In a retrospective multicenter validation study also by Backus et al (2010), 880 patients presenting with chest pain were evaluated. The primary end points studied were the same as in the original study. In this study, 158 patients (17.95%) reached a primary end point. Of the 303 patients with HEART scores of 0-3 points, 3 (0.99%) had a MACE. Among 413 patients with HEART scores of 4-6 points, 48 cases (11.6%) resulted in a MACE, and among patients with HEART scores of 7-10 points, a MACE was identified in 107 of 164 cases (65.2%).

Backus et al (2013) externally validated the HEART Score with a prospective multicenter study. The study evaluated 2440 patients presenting with chest pain to 10 EDs in the Netherlands. The primary end point was the occurrence of any MACE within 30 days in patients who were identified for early discharge. The study found that the HEART Score identified more patients as low-risk than the TIMI Score did, which would have led to potential cost savings of €64,107 (~USD $76,000) by using the HEART Score cutoffs versus cost savings of €14,670 (~USD $17,000) using the TIMI Score cutoffs.

The HEART Pathway developed by Mahler et al (2015) combined the HEART Score with 0- and 3-hour cardiac troponin tests in a decision aid designed to identify ED patients who are safe for early discharge. The study found that the HEART Pathway decreased length of stay by 12 hours, decreased objective cardiac testing by 12%, and increased early discharges by 21%. No MACE was seen within 30 days in patients who were identified for early discharge. Of note, this study (and the original HEART Score studies) used regular-sensitivity cardiac troponin testing.

Selected Abbreviations

| ACS | Acute coronary syndromes |
| ECG | Electrocardiogram |
| GRACE | Global Registry of Acute Coronary Events [Risk Score] |
| HEART | History, ECG, Age, Risk Factors, Troponin [Score] |
| MACE | Major adverse cardiac event |
| NSTEMI | Non-ST-segment elevation myocardial infarction |
| TIMI | Thrombolysis in Myocardial Infarction [Risk Score] |
| UA | Unstable angina |
TIMI Risk Score for UA/NSTEMI

Introduction: The TIMI risk score for UA/NSTEMI estimates mortality in patients with unstable angina and non-ST-segment elevation myocardial infarction.

Points & Pearls

• The TIMI (Thrombolysis in Myocardial Infarction) Risk Score for UA (unstable angina) / NSTEMI (non-ST-segment elevation myocardial infarction) has historical significance as one of the earliest chest pain decision rules that was widely implemented.
• A TIMI Risk Index and TIMI Risk Score for STEMI (ST-segment elevation myocardial infarction) also exist; however, in this review, “TIMI Risk Score” refers only to the TIMI Risk Score for UA/NSTEMI.
• The TIMI Risk Score was originally derived with patients with known UA or NSTEMI.
• A TIMI Risk Score of 0 or 1 point does not equal zero risk of adverse outcome. The original study showed 4.7% of patients with a score of 0 or 1 point had adverse outcomes within 14 days.
• Validation studies showed 1.7% to 2.1% of patients with a score of 0 still had adverse outcomes within 30 days.
• It is unclear whether this risk score can be used in patients with chest pain in the setting of cocaine use.
• The TIMI Risk Score was further studied as part of an accelerated diagnostic protocol in the ADAPT (Two-hour Accelerated Diagnostic Protocol to Assess Patients with Chest Pain Symptoms Using Contemporary Troponins as the Only Biomarker) trial, which included estimation of pretest probability using TIMI, plus abnormal electrocardiogram (ECG) and troponin (high-sensitivity cardiac troponin I).

Advice

Patients in the 0-1 point group should be further risk stratified using another risk score or institutional practices, as risk is not low enough to safely discharge these patients from the hospital. Many
guidelines recommend aggressive medical intervention and/or early invasive management for higher-risk patients.

**Critical Actions**

A TIMI Risk Score of 0 does not equate to zero risk of adverse outcome.

**Evidence Appraisal**

Antman et al (2000) used a merged database of 7081 UA/NSTEMI patients in the TIMI 11B and ESSENCE (Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q-wave Coronary Events) trials for the original derivation and validation of this TIMI Risk Score. The risk score was originally derived from 1957 UA/NSTEMI patients receiving unfractionated heparin in the TIMI 11B trial, and was internally validated in 3 cohorts of patients from the rest of the merged data: 1953 patients receiving enoxaparin in the TIMI 11B trial; 1564 patient receiving unfractionated heparin in the ESSENCE Trial; and 1607 patients receiving enoxaparin in the ESSENCE Trial. The study included UA/NSTEMI patients with chest pain at rest who presented within 24 hours of symptoms, and who had ST-segment deviation on their presenting ECG, history of coronary artery disease, and a measured cardiac enzyme that was elevated. Patients were excluded if revascularization was within 24 hours or if the patient had a contraindication to anticoagulation. The primary end points were composite all-cause mortality, myocardial infarction, or urgent revascularization within 14 days.

By the end of the 14 days, 16.7% of the derivation group had died, had a myocardial infarction, needed urgent revascularization. An increase of the TIMI Risk Score correlated with an increase in all-cause mortality, myocardial infarction, or urgent revascularization. The same pattern was seen in the internally validated groups. There have been many external validation studies since the original derivation.

Scirica et al (2002) externally validated the TIMI Risk Score in patients from 9 sites in the TIMI III Registry. This study included UA/NSTEMI patients with ischemic chest pain lasting more than 5 minutes, presenting within 96 hours of symptom onset. Patients were excluded if they had a STEMI, chest pain of other origin, planned revascularization, or if the patient was in a prior TIMI trial. Primary end points were death, myocardial infarction, and recurrent ischemia within 6 weeks and at 1 year. As in the original derivation study and internal validation studies, there was an increase in mortality, myocardial infarction, and recurrent ischemia with each increase in the TIMI Risk Score. However, this study modified the TIMI Risk Score definitions to some degree by substituting a patient’s history of myocardial infarction or revascularization history for “known coronary artery stenosis > 50%,” and assigning 1 point for aspirin use in the past 24 hours, not the past 7 days as in the original trial by Antman et al. Still, this population was in a registry for patients with known UA/NSTEMI. Thus, this validation is less useful for patients with undifferentiated chest pain seen in the acute care setting of the emergency department (ED).

Pollack et al (2006) externally validated the TIMI Risk Score in a prospective observational cohort study of 3929 adult patients with chest pain in the ED. The study included adult chest pain patients aged > 24 years who were evaluated with ECG. Patients with a score of 0 or 1 point are at lower risk of adverse outcome (death, myocardial infarction, urgent revascularization) compared to patients with a higher risk score. However, the risk is not zero.

**Why to Use**

Chest pain is one of the most common complaints bringing patients to the emergency department for evaluation. The identification and acute management of ST-elevation myocardial infarction is rarely a conundrum. However, UA/NSTEMI can go missed. Traditionally, the TIMI Risk Score for UA/NSTEMI can correlate the risk of adverse outcome in chest pain patients.

**When to Use**

The TIMI Risk Score for UA/NSTEMI can be used to help risk stratify patients with presumed ischemic chest pain. However, it was originally derived in patients with confirmed unstable angina or non-ST elevation myocardial infarction.

**Next Steps**

- Patients with a score of 0 or 1 point are at lower risk of adverse outcome (death, myocardial infarction, urgent revascularization) compared to patients with a higher risk score. However, the risk is not zero.
- Patients with a higher risk score may require more aggressive medical or procedural intervention.
- Newer chest pain risk scores such as the HEART Score have been shown to be better at risk stratification than the TIMI Risk Score for UA/NSTEMI, particularly in the undifferentiated chest pain patient.

Abbreviations: HEART, History, ECG, Age, Risk Factors, Troponin [Score]; NSTEMI, non-ST-segment elevation myocardial infarction; TIMI, Thrombolysis In Myocardial Infarction [Risk Score]; UA, unstable angina.
with patients for up to 30 days from presentation for adverse outcomes of death, myocardial infarction, or revascularization. As in prior studies, the higher the TIMI Risk Score, the higher the likelihood of adverse outcome within the measured time period, which was 30 days in this study. However, the patient population was different in that there were more black patients and more female patients. Also, if no cardiac markers were ordered, a score of 0 was assumed and assigned to the category of cardiac enzymes.

Chase et al (2006) externally validated the TIMI Risk Score in a prospective observational study of 1458 patient visits in the ED. The study included patients aged > 30 years with nontraumatic chest pain who had an ECG performed in the ED. Whereas Pollack et al included patients with cocaine use, Chase et al excluded patients if cocaine was used in the 7 days prior to presentation. Like Pollack et al, Chase et al assigned a score of 0 to cardiac enzymes if they were not drawn. Chase et al also followed patients for up to 30 days. Within 30 days, 12.8% of patients had an adverse outcome of death, myocardial infarction, or revascularization. In patients with a TIMI Risk Score of 0, 1.7% had an adverse outcome. Although there was a general correlation of an increase in adverse outcome with higher TIMI Risk Score, this study did not show a similar stepwise increase. This is likely secondary to having a study population that was dissimilar to the original derivation group or other validation studies, as this study had patients with mostly low TIMI scores and included STEMI patients in the study population.

### Selected Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tr>
<td>ADAPT</td>
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<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
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<td>ESSENCE</td>
<td>Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q-wave Coronary Events [Trial]</td>
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<td>HEART</td>
<td>History, ECG, Age, Risk Factors, Troponin [Score]</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>Non-ST-segment elevation myocardial infarction</td>
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<td>STEMI</td>
<td>ST-segment elevation myocardial infarction</td>
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<td>TIMI</td>
<td>Thrombolysis in Myocardial Infarction [Risk Score]</td>
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<td>UA</td>
<td>Unstable angina</td>
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### Calculator Creator

Elliot M. Antman, MD
Click here to read more about Dr. Antman.

### References

**Original/Primary Reference**

**Validation References**

**Additional Reference**
Points & Pearls

• The goal of the original derivation study was to have a prediction rule that allowed for the safe discharge of emergency department (ED) chest pain patients within 2 hours and without the need for further provocative testing.
• The original derivation study tested with troponin T, but an external validation study showed no difference in the performance of the rule with high-sensitivity troponin.
• The incidence of disease may be higher in the countries where the rule was developed and tested (Canada, Australia, New Zealand) than in the United States.
• The primary outcome was the diagnosis of acute coronary syndromes (ACS), defined as an acute myocardial infarction or unstable angina, within 30 days. Acute myocardial infarction was defined as positive troponin, electrocardiogram (ECG) consistent with acute myocardial infarction, or death without any other cause or event; unstable angina was defined as coronary angiogram showing 70% lesion or revascularization, with either percutaneous coronary intervention or coronary artery bypass grafting.
• Adverse events were broadly defined and included the following: tachycardia or bradycardia requiring medical intervention; respiratory failure requiring assisted ventilation; pulmonary embolism; aortic dissection or aneurysm; new congestive heart failure requiring intravenous medications; hypotension requiring vasoactive agents or an intra-aortic balloon pump; chest compressions; percutaneous coronary intervention; or coronary artery bypass grafting.

Advice

Patients who meet the low-risk criteria can be considered for discharge from the ED without further provocative testing. Otherwise, patients should be ruled out for ACS per normal chest pain protocols utilizing serial ECGs, cardiac biomarkers, and risk stratification. These patients may require additional provocative testing.

Critical Actions

Patients who meet low-risk criteria can be considered for discharge from the ED without further provocative testing for ACS. Other etiologies of chest pain should be considered, including aortic, esophageal, pulmonary, cardiac, abdominal, and musculoskeletal sources. On the other hand, patients who do not meet the low-risk criteria should be managed as per usual chest pain protocols, including but not limited to consideration of aspirin, nitroglycerin, and serial ECGs and biomarkers.

Evidence Appraisal

Scheuermeyer et al (2014) derived the Vancouver Chest Pain Rule in a prospective cohort study of 763 ED patients at St. Paul’s Hospital, an urban tertiary care center, and internally validated the rule in a prospective cohort of 906 ED patients at the same hospital. The study included ED patients complaining of anterior or lateral chest pain who were suspected of having possible ischemic chest pain. Patients were excluded for any of the following: aged < 25 years, traumatic etiology, radiologically evident alternate cause of chest pain, previous study enrollment in the past 30 days, terminal illness, or communication barriers. The primary outcome was the diagnosis of ACS, defined as an acute myocardial infarction or unstable angina within 30 days.

ECGs were labeled as having ischemic factors if there were any of the following: ST elevation > 2 mm in 2 consecutive precordial leads or > 1 mm in 2 consecutive limb leads; ST depression > 2 mm in V1/V2; ST depression > 0.5 mm or Q waves or T-wave inversions in 2 contiguous leads; left bundle branch block; left ventricular hypertrophy; or a paced rhythm. Of note, “ischemic ECG changes” in this study are not the same as those in the American Heart Association (AHA) guidelines. The AHA uses...
1 mm as the measurement cutoff for significant ST elevation, except in the anterior leads, which are age- and sex-dependent. Also, the AHA does not consider a paced rhythm or the existence of a left bundle branch block or left ventricular hypertrophy as signs of acute ischemia.

In the derivation cohort, 10.1% had an acute myocardial infarction and 11.5% had unstable angina. Based on the Vancouver Chest Pain Rule, no cases of ACS were missed, and 18.6% of patients without ACS would have been able to be discharged within 2 hours without the need for additional provocative testing, resulting in 100% sensitivity, 18.6% specificity, 100% negative predictive value (NPV), and 25.3% positive predictive value (PPV).

In the internal validation cohort, 4.3% had an acute myocardial infarction and 8.8% had unstable angina. The Vancouver Chest Pain Rule missed 1 case of unstable angina: a 48-year-old male who screened as low-risk but was taken to the catheterization lab because of documented prehospital hypotension, and was found to have a 50% lesion to the left anterior descending coronary artery. Based on the validation cohort, the Vancouver Chest Pain Rule had 99.2% sensitivity, 23.4% specificity, 99.5% NPV, and 16.4% PPV.

Cullen et al (2014) externally validated the Vancouver Chest Pain Rule in a prospective cohort of 1635 ED patients in Brisbane, Australia, and Christchurch, New Zealand, comparing sensitive and highly sensitive troponin assays. The study included ED patients with ≥ 5 minutes of suspected ischemic chest pain. Patients were excluded if there was a clear non-ACS etiology of chest pain, previous study enrollment in the past 45 days, terminal illness, inability or unwillingness to consent, or pregnancy. All patients had troponins drawn on presentation and at ≥ 6 hours. Blood was also drawn at 2 hours for the highly sensitive troponin assay. Cullen et al looked at the same primary outcome of ACS, defined as acute myocardial infarction or unstable angina within 30 days. With the usual-sensitivity troponin assay, 20.4% of patients had ACS, resulting in 98.8% sensitivity, 15.8% specificity, 98.1% NPV, and 23.2% PPV. The Vancouver Chest Pain Rule, using the usual-sensitivity troponin assay, missed 4 cases of unstable angina. When the highly sensitive troponin assay was used, 20.2% of patients were identified with ACS, resulting in 99.1% sensitivity, 16.1% specificity, 98.6% NPV, and 23.3% PPV. The Vancouver Chest Pain Rule using the highly sensitive troponin assay missed 3 cases of unstable angina. There was no statistically significant difference in the function of the Vancouver Chest Pain Rule using the usual-sensitivity troponin versus the highly sensitive troponin assay.

**Why to Use**
In the absence of an obvious ST-elevation myocardial infarction, diagnosing the chest pain patient in the ED can be challenging. Typically, patients with possible ischemic chest pain undergo serial troponin testing to rule out myocardial infarction, which extends a patient’s ED length of stay and can lead to further strains on the ED in time and space. The Vancouver Chest Pain Rule identifies low-risk chest pain patients who can be safely discharged from the ED after the standard initial evaluation of history and physical examination, ECG, and 1 cardiac biomarker (usual-sensitivity troponin).

**When to Use**
- The Vancouver Chest Pain Rule can be applied to adult patients aged > 25 years who present to the ED with chest pain.
- It should not be used in patients with trauma or other radiographically identified cause for chest pain such as pneumothorax, pleural effusion, and/or pneumonia.
- It should only be used in patients without any of the following findings on ECG: ST elevation, ST depression > 0.5 mm, Q waves, left ventricular hypertrophy, paced rhythm, or left bundle branch block.

**Next Steps**
- Patients with an abnormal ECG, positive troponin at 2 hours, or history of prior ACS or nitrate use do not qualify for early discharge.
- Patients with a normal ECG, negative 2-hour troponin, no prior history of ACS or nitrate use, and reproducible pain to palpation can be discharged from the ED without further provocative testing.
- Patients with a normal ECG, negative 2-hour troponin, no prior history of ACS or nitrate use, with nonreproducible chest pain, who are aged < 50 years and have chest pain that does not radiate to the neck, jaw, or arm, can be discharged from the ED without further provocative testing.
Selected Abbreviations

ACS  Acute coronary syndromes
ECG  Electrocardiogram
NPV  Negative predictive value
PPV  Positive predictive value

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References

Original/Primary Reference

Validation Reference

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