Can a HEART Pathway Improve Safety and Diagnostic Efficiency for Patients With Chest Pain?

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In the dark days before Y2K, emergency physicians misdiagnosed 2% to 4% of acute myocardial infarctions, approximately 11,000 cases per year in the United States.[1-4] These misses were associated with high mortality, major adverse outcomes, and medicolegal exposure.[2,3] They were stressful times, and the thought of a patient dropping dead after emergency department (ED) discharge was terrifying. It was never realistic to admit everyone and diagnostic perfection was a pipe dream when your only tools were the ECG and an serum glutamic-oxaloacetic transaminase (SGOT) test. But times changed and biomarkers evolved. The serum glutamic-oxaloacetic transaminase (SGOT) test gave way to lactate dehydrogenase isoenzymes, creatine kinase, creatine kinase-muscle/brain (CK-MB), troponin, and high-sensitivity troponin. Things are easier now. These days, death is rare after ED discharge, and missed myocardial infarction is usually a troponin bump in a stable patient. With a bit of clinical judgment and a high-sensitivity troponin assay, I no longer fear missed myocardial infarction, but I wonder occasionally whether I should be using a prediction tool.

Two decades ago, researchers envisioned prediction tools that would objectify risk and improve diagnosis. Now there are more tools than you can shake a stick at. All share the same structure, combining clinical predictors with ECG and marker findings to identify low-risk patients. Of those that have been validated, GRACE and TIMI are the old-timers. They still have proponents but were derived in proven acute coronary syndrome (ACS) populations and perform relatively poorly for ED chest pain.[5-9] In 2006, the Vancouver Chest Pain Rule came along, boasting 99% sensitivity for 30-day ACS outcomes and identifying 26% of patients for safe early discharge without further investigation,[10] but subsequent validation studies identified fewer patients appropriate for discharge.[11,12] The Emergency Department Assessment of Chest Pain Score (ED-ACS) was combined with serial troponin assay results to create the ED-ACS accelerated diagnostic protocol,[13] which reported 99% sensitivity for 30-day major adverse cardiac events and identified 42% of patients safe for discharge.[14] The Manchester ACS model and an updated version incorporating high-sensitivity troponin testing proved 98% sensitive for 30-day major adverse cardiac events and identified 40% of patients as safe for discharge.[15]

For unclear reasons, the HEART score is now the flavor of the day. Despite a pleasing acronym that suggests ease of use, it is not simple and has subjective components that challenge reliability. Scoring requires interpretation of the history (slightly, moderately, or highly suspicious), the ECG (normal, nonspecific, or significant ST-segment deviation), age category ($\leq$45, 46 to 64, and $\geq$65 years), risk factors (hypertension, diabetes, obesity, dyslipidemia, smoking within 3 months, family history [parent or sibling with cardiovascular disease before 65 years], atherosclerotic disease [myocardial infarction, revascularization, stroke, transient ischemic attack, or peripheral vascular disease]), and troponin level (normal, 1 to 3 times normal, and >3 times normal). A low-risk score of 0 to 3 identifies 37% of patients as safe for discharge and has 95.9% to 96.7% sensitivity for risk of 30-day major adverse cardiac events.[8,16,17] This level falls below the generally accepted 98% threshold.

In this issue, Sharp et al[18] describe the effect of a HEART pathway in 13 community EDs. Participating sites introduced electronic decision support that prompted physicians to enter HEART criteria in real time. The system calculated a HEART score, incorporated serial troponin values, and recommended dispositions for high-risk (admit), moderate-risk (observe or outpatient noninvasive testing), and low-risk patients (discharge with family physician follow-up). During 1-year pre- and postphases, 30,522 and 34,871 patients were studied,

respectively. After pathway implementation, the primary outcome of 30-day hospitalization or observation or noninvasive testing decreased from 35.5% to 31.8%. Admissions decreased from 14.7% to 13.2% and stress testing from 28.6% to 25.3%. Predictive modeling suggested that in the postimplementation year, the primary outcome improved by 4.39%, with no increase in 30-day mortality or myocardial infarction. The authors concluded that HEART safely reduced inpatient care and noninvasive testing.

Interrupted time series analyses such as this account for longitudinal trends and are the strongest quasi-experimental approach to evaluating interventions over time, but they aren't perfect. If something other than the intervention changes during the study period, it could generate spurious findings. In this case, physicians weren't blinded to the intervention or its purpose. Knowing their employer wanted to reduce admissions and noninvasive testing might have influenced physicians' practice and generated the modest improvement observed. Furthermore, if physicians believed they were being monitored more closely (Hawthorne effect), this could alter their practice. For reasons outlined below, I suspect that factors other than the HEART pathway contributed to the study findings.

My main concerns with the study are poor pathway compliance and low disease prevalence. Only 35% of patients had a HEART score determined, which means the pathway was not in play for most of them. Among patients who received a HEART score, the actual disposition often disagreed with the pathway recommendation. Specifically, 25% of low-risk patients were admitted or stress tested (pathway recommendation, 0%), whereas 57.8% of high-risk patients were admitted (pathway recommendation, 100%).

The pathway identified 7,204 patients (59%) as low-risk suitable for discharge (Table 1), but pathway sensitivity was only 76.5%, meaning it missed 23% of major outcomes. A negative predictive value of 99.8% made the pathway appear safe, but this value was based on the fact that only 0.4% of patients with documented HEART scores (51 of 12,267) experienced an outcome event. In this exceedingly low-prevalence sample, positive predictive value (the event rate in moderate- or high-risk patients designated for observation or admission) was only 0.8%. Poor pathway performance probably explains the dissonance between physician decisions and pathway recommendations. Had physicians followed HEART guidance, they would have admitted or investigated 5,063 patients, with a miss rate of 0.2% (12/5,063). Had they simply discharged *all* patients with no investigation, their miss rate would have been a very acceptable 0.4% (51/12,267). So although efficiency improved during the study, it seems unlikely the pathway was responsible.

Table 1. HEART pathway performance in a low-prevalence (0.4%) sample.

	Event (N)	No Event (N)	Total (N)	HEART Pathway Performance Characteristics
HEART (+)	39	5,024	5,063	Sensitivity 76.5% (39/51) Specificity 58.9%(7,192/12,216)
HEART (–)	12	7,192	7,204	Positive predictive value 0.8% (39/5,063)
Total	51	12,216	12,267	Negative predictive value 99.8% (7,192/7,204)

HEART (+), Moderate- and high-risk patients with scores of 4 to 9; *HEART (–)*, patients with low-risk scores (0 to 3 points) and negative serial troponin assay results. "Event" refers to the predefined safety outcome of death or myocardial infarction. "Prevalence" refers to the proportion of subjects who had an outcome event.

Pathway performance is highly dependent on disease prevalence in the population tested.[19] Data from a recent meta-analysis[17] show that HEART performs better in a higher-prevalence population, with 96.7% sensitivity and 24.9% positive predictive value (Table 2). But high sensitivity and reasonable predictive value do not add up to utility. To be useful, a pathway must improve on physician judgment,[19] which is a tall order. In a cohort of patients with normal ECG results and negative troponin values, Body et al[20] found that physician judgment identified 23% of patients as safe for discharge and achieved 100% sensitivity for adverse outcomes. Scheuermeyer et al[21] studied an algorithm that depended on physician judgment along with 2- and 6-hour troponin levels to differentiate high-risk (admit), moderate-risk (early outpatient provocative testing), and low-risk patients (discharge with family physician follow-up). In a large consecutive sample with 10.8% acute coronary syndrome prevalence, these

Table 2. HEART pathway performance in a moderate-prevalence (15.4%) sample.

	Event (N)	No Event (N)	Total (N)	HEART Performance Characteristics
HEART (+)	1,670	5,030	6,700	Sensitivity 96.7% (1,670/1,727) Specificity 47% (4,460/9,490)
HEART (–)	57	4,460	4,517	Positive predictive value 24.9% (1,670/6,700)
Total	1,727	9,490	11,217	Negative predictive value 98.7 % (4,460/4,517)

"Event" refers to major adverse cardiac events. "Prevalence" refers to the proportion of subjects who had a major adverse cardiac event.

physicians discharged 72% of patients and referred 22.8% for early outpatient provocative testing, again achieving 100% sensitivity for 30-day ACS outcomes.

A *useful* pathway must not only improve on physician judgment but also change management in a way that improves outcomes.[19] One recent study reported that a new HEART pathway, combined with serial high-sensitivity troponin results, reduced admission rates from 59% to 33%.[22] This study was limited by convenience sampling, a low recruitment rate, a before-after analysis that didn't account for temporal trends or other confounders, and a sicker cohort (eg, twice the rate of myocardial infarction) in the preimplementation period. Another recent multicenter study with stronger randomized stepped-wedge methodology found that a HEART pathway did not significantly change admission rates or safety outcomes.[23]

Whether a pathway will improve physician performance also depends on baseline performance. When deriving the North American Chest Pain Rule, Hess et al[24] demonstrated extreme practice variability across 3 participating sites. Two of the sites admitted 18% and 21% of patients, whereas the third hospitalized 48% and admitted 48% to a clinical decision unit, for a combined admission rate of 96%. The authors noted that the derived decision tool would have reduced resource use at one site but substantially increased it at the others.

The study by Sharp et al[18] provides fascinating data showing that, although prediction tools may be helpful in moderate- or even low-risk populations, they should not be applied in "no-risk" populations, where they are unlikely to improve safety and very likely to increase use. With this limitation in mind, I believe chest pain prediction tools are more good than bad. They force structured thought about difficult diagnoses, they objectify risk, they offer guidance in marginal cases, they provide evidence-based justification for our decisions should adverse outcomes occur, and they are less variable than physician judgment. In some circumstances, they may even be useful.

Sadly, their role is about to change. High-sensitivity troponin testing will transform the diagnostic landscape in the United States just as it has elsewhere in the world, and the tag team of physician judgment and high-sensitivity troponin testing is formidable. Prediction tools making headlines today may be in breadlines tomorrow.

For me, the future looks like clinical gestalt and high-sensitivity troponin testing, but I could be wrong.

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