EMA Emergency Medicine Australasia

Emergency Medicine Australasia (2014) **26**, 34–44 doi: 10.1111/1742-6723.12164

RESEARCH

Development and validation of the Emergency Department Assessment of Chest pain Score and 2 h accelerated diagnostic protocol

Martin Than,[1] Dylan Flaws,[2,3] Sharon Sanders,[4] Jenny Doust,[4] Paul Glasziou,[4] Jeffery Kline,[5] Sally Aldous,[1] Richard Troughton,[6] Christopher Reid,[7] William A Parsonage,[2,3] Christopher Frampton,[6] Jaimi H Greenslade,[2,3,8] Joanne M Deely,[9] Erik Hess,[10] Amr Bin Sadiq,[1] Rose Singleton,[1] Rosie Shopland,[1] Laura Vercoe,[1] Morgana Woolhouse-Williams,[1] Michael Ardagh,[6] Patrick Bossuyt,[11] Laura Bannister[1] and Louise Cullen[2,8]
[1]Christchurch Hospital, Christchurch, New Zealand; [2]Royal Brisbane and Women's Hospital, Herston, Queensland, Australia, [3]University of Queensland, Brisbane, Queensland, Australia, [4]Bond University, Gold Coast, Queensland, Australia; [5]Indiana University, Indianapolis, Indiana, USA; [6]University of Otago, Christchurch, New Zealand; [7]Monash University, Melbourne, Victoria, Australia, [8]Queensland University of Technology, Brisbane, Queensland, Australia; [9]Canterbury District Health Board, Christchurch, New Zealand; [10]Mayo Clinic, Rochester, New York, USA; and [11]Amsterdam Medical Centre, Amsterdam, The Netherlands

Abstract

Objective: Risk scores and accelerated diagnostic protocols can identify chest pain patients with low risk of major adverse cardiac event who could be discharged early from the ED, saving time and costs. We aimed to derive and validate a chest pain score and accelerated diagnostic protocol (ADP) that could safely increase the proportion of patients suitable for early discharge.

Methods: Logistic regression identified statistical predictors for major adverse cardiac events in a derivation cohort. Statistical coefficients were converted to whole numbers to create a score. Clinician feedback was used to improve the clinical plausibility and the usability of the final score (Emergency Department Assessment of Chest pain Score [EDACS]). EDACS was combined with electrocardiogram results and troponin results at 0 and 2 h to develop an ADP (EDACS-ADP). The score and EDACS-ADP were validated and tested for reproducibility in separate cohorts of patients.

Results: In the derivation (n = 1974) and validation (n = 608) cohorts, the EDACS-ADP classified 42.2% (sensitivity 99.0%, specificity 49.9%) and 51.3% (sensitivity 100.0%, specificity 59.0%) as low risk of major adverse cardiac events, respectively. The intra-class correlation coefficient for categorisation of patients as low risk was 0.87.

Conclusion: The EDACS-ADP identified approximately half of the patients presenting to the ED with possible cardiac chest pain as having low risk of short-term major adverse cardiac events, with high sensitivity. This is a significant improvement on similar, previously reported protocols. The EDACS-ADP is reproducible and has the potential to make considerable cost reductions to health systems.

Key words: *accelerated diagnostic protocol, acute coronary syndrome, emergency department, major adverse cardiac event, risk score.*

Correspondence: Dr Martin Than, Department of Emergency Medicine, Canterbury District Health Board, Private Bag 4710, Christchurch, New Zealand. Email: martinthan@xtra.co.nz

Martin Than, MBBS, Specialist; Dylan Flaws, MBBS, Resident Medical Officer; Sharon Sanders, MPH, Assistant Professor; Jenny Doust, PhD, Professor; Paul Glasziou, PhD, Director; Jeffery Kline, MD, Professor; Sally Aldous, MD, Specialist; Richard Troughton, PhD, Cardiologist; Christopher Reid, PhD, Professor, Associate Director; William A Parsonage, DM, Senior Staff Cardiologist; Christopher Frampton, PhD, Biostatistician; Jaimi H Greenslade, PhD, Research Fellow; Joanne M Deely, PhD, Medical Writer; Erik Hess, MD, Associate Professor; Amr Bin Sadiq, MBBS, House Officer; Rose Singleton, MBBS, Senior House Officer; Rosie Shopland, MBBS, Senior House Officer; Laura Vercoe, MBBS, Registrar; Morgana Woolhouse-Williams, MBBS, Senior House Officer; Michael Ardagh, PhD, Professor; Patrick Bossuyt, PhD, Professor; Laura Bannister, MBChB, Registrar; Louise Cullen, MBBS, Specialist.

Introduction

Approximately 5–10% of annual presentations to EDs and 25% of hospital admissions are from patients with symptoms suggestive of acute coronary syndromes (ACS).[1] Assessment of these patients is a major challenge for clinicians because a missed ACS can lead to death or other adverse outcomes.[2]

Although cardiac troponin and the ECG are the principal tests used to assess patients with chest pain, estimating the pretest probability of a cardiac event is necessary to interpret these tests. Traditionally, pretest probability has been determined using clinical acumen, primarily involving historical variables and risk factors learnt during early training and reinforced in clinical practice. When used in the ED, evidence suggests that neither symptomatic history nor the presence of chronic risk factors for coronary artery disease are as predictive of ACS as previously thought.[3–10]

There is a clear need for a well-developed and effective ED-based chest pain prediction rule. There have been multiple attempts to provide clinicians with tools to assist with the risk stratification of possible cardiac chest pain.[11] Recent systematic reviews of prediction rules for assisting with the diagnosis of ACS in the ED setting found that many such rules had been developed retrospectively and have methodological flaws.[12,13] Further, existing prediction rules using solely cardiac troponin to aid early discharge (within the first few hours of ED arrival) show a relatively low potential early discharge rate. The ADAPT study[14] identified 20% of patients as suitable for early discharge, with a sensitivity of 99.7%; the HEART score[15,16] has been reported to identify 20.4–28.2% of such patients with sensitivity of 96.3–99.0%; and the updated Vancouver Chest Pain Rule[17] identifies 14.5–20.4% of patients with a sensitivity of 99.2–100%.

Methods for developing prediction rules have been described in detail.[18–22] Their development involves identifying important predictors of the disease or outcome of interest from a selection of candidate clinical variables. This might be performed using expert opinion or, more commonly, by using multivariable statistical modelling techniques. Statistical modelling results in the assignment of a relative 'weighting' of each predictor according to its diagnostic value. An important consideration during development is the clinical sensibility of the resulting prediction rule (Box 1a). Evaluation of sensibility requires judgment rather than statistical methods.[23] A sensible rule is easy to use, and has content and face validities. This means that it must contain predictors appropriate for the purpose of the rule that are combined in a sensible way, with no obvious items missing (Box 1). Ease of use might be facilitated by presenting a rule developed from logistic regression as a score, where the original predictor weights have been converted to integers that are easy to add together[19] or as a nomogram, or a risk stratification chart. Though less precise than the original regression formula, such presentations are less complex, easier to apply by memory and usable without electronic assistance. Prediction rules are unlikely to be applied in practice if they are not considered sensible by the end-user, even if they are accurate.[24]

The present study had two goals. First, we wanted to develop a clinically sensible score to predict the short-term risk of major adverse cardiac events (MACE) in adults presenting to the ED with possible cardiac chest pain. This will be referred to as the Emergency Department Assessment of Chest pain Score or EDACS (Box 1b). Second, we combined EDACS with normal troponin results at 0 and 2 h and an ECG finding of no new ischaemia, to develop an EDACS-accelerated diagnostic protocol (EDACS-ADP). The proposed purpose of the ADP was to identify a subgroup of adults presenting to the ED with chest pain that is at very low short-term risk of MACE. Such patients could then be safe for early discharge and further investigations performed as an outpatient. We externally validated the EDACS-ADP on a temporally separate cohort of patients.

Methods

Setting

The EDACS and EDACS-ADP were derived and studied in urban EDs of the Royal Women's and Brisbane Hospital (Australia, ~72 000 ED patients, annually) and Christchurch Hospital (New Zealand, ~75 000 ED patients, annually).

Participants

Patient recruitment criteria were the same as previously published in the prospective observational ADAPT study.[14] We enrolled consecutive patients aged 18 years or over, with at least 5 min of symptoms consistent with an acute coronary syndrome, such that the attending physician planned to perform further investigations for this potential diagnosis. The American Heart Association case definitions[25] for possible cardiac symptoms

Box 1. *(a) Terminology, definitions associated with 'Sensibility' of clinical prediction rules; (b) terminology and abbreviations of derived score and diagnostic protocol*

Terminology	Meaning
(a)	
Sensibility	This refers to whether a prediction rule is both clinically reasonable and easy to use. This is more based on opinion than statistical methodology.
1. Content validity	For a rule to have content validity, the items included must be sensible, with no obvious omissions and the way that these variables are organised appears suitable for the objectives of the rule.
2. Face validity	This is a subjective interpretation of the validity of the rule by the user. The face validity of a rule will depend on the expectations and beliefs of the user, and may not be associated with statistical validity, but is essential for end-user uptake. To maximise face validity and ensure end-user trust, it may be necessary to include variables found to be statistically suboptimal in the final prediction model.
3. User friendliness	This refers to how easy the rule is to use. This depends on the demands the rule will place on memory, complexity of calculations in the absence of electronic devices, format and layout of the rule.
(b)	
EDACS	Emergency Department Assessment of Chest pain Score A clinical score to predict the short-term risk of major adverse cardiac event for adults presenting to the ED with possible cardiac chest pain.
EDACS-ADP	Emergency Department Assessment of Chest pain Score-Accelerated Diagnostic Protocol A chest pain clinical investigation pathway that combines the EDACS below a specified score cut-off with negative ECG and troponin results to identify a low-risk subgroup of patients. These patients are at low short-term risk of MACE and would be safe for rapid discharge to early outpatient follow-up investigation (or could proceed more quickly to further inpatient investigations).

were used (i.e. acute chest, epigastric, neck, jaw or arm pain; or discomfort or pressure without an apparent non-cardiac source).[25] Patients judged to be at high risk of MACE were also included. All patients were managed as per local guidelines.

Model derivation

The derivation cohort consisted of patients recruited between June 2007 and February 2010 for the ADAPT study.[14]

Model validation

The validation cohort was recruited between October 2010 and December 2011 at the same two hospitals, as part of their ongoing cardiac studies. The same participant symptom criteria, outcomes and data definitions were used as in the ADAPT study.[14,26]

Predictor variables

Data were prospectively collected for 37 candidate variables commonly used in clinical care, or reported as having value in predicting acute myocardial infarction among ED patients with chest pain (Table 1). Research nurses collected the data using explicit definitions for each candidate variable from a published data dictionary.[26] Clinical symptoms and medical history were recorded as reported by the patient, without incorporating information from the patient's medical records. If a patient was unsure of an answer to a question (e.g. history of hypertension), a response of 'No' was recorded. We used reported rather than previously recorded variables, so that the reproducibility of the final prediction rule would not be reliant upon access to the patient's records.

Outcome assessment

Patients were followed up to determine the occurrence of MACE within 30 days of presentation using the following methods: telephone contact, review of patients' hospital notes and an electronic health events search (which identifies any deaths). An adjudicated discharge diagnosis was made by two independent cardiologists using information obtained during each patient's

Table 1. Demographics of derivation cohort (n = 1974)

Variable	Mean (range or 95% CI)	Estimate	P value
Age	60.5 (19.8–98.0) 95% CI ± 0.7	0.045	<0.0001
	Prevalence (%)	Phi coefficient	
Male sex	1184 (60.0), 95% CI ± 2.2	0.076	0.0008
Symptoms			
Pain on inspiration	427 (21.6), 95% CI ± 1.8	–0.10	<0.0001
Pain on palpation	166 (8.4), 95% CI ± 1.2	–0.084	0.0002
Pain radiates to arm or shoulder	674 (34.1), 95% CI ± 2.1	0.096	<0.0001
Pain radiates to throat	425 (21.5), 95% CI ± 1.8	0.024	0.28
Pain radiates to back	330 (16.7), 95% CI ± 1.6	–0.046	0.043
Diaphoresis	998 (50.6), 95% CI ± 2.2	0.046	0.043
Past history			
Previous MI	473 (24.0), 95% CI ± 1.9	0.061	0.0065
Previous angina	712 (36.1), 95% CI ± 2.1	0.049	0.031
Previous ventricular tachycardia	21 (1.0), 95% CI ± 0.5	0.010	0.65
Previous congestive heart failure	153 (7.8), 95% CI ± 1.1	0.033	0.14
Previous transient ischaemic attack	218 (11.0), 95% CI ± 1.4	0.023	0.30
Previous peripheral arterial disease	69 (3.5), 95% CI ± 0.1	0.071	0.0016
Previous CABG	170 (8.6), 95% CI ± 1.2	0.088	<0.0001
Previous PCI	358(18.1), 95% CI ± 1.7	–0.0054	0.81
Hypertension	1019 (51.6), 95% CI ± 2.2	0.093	<0.0001
Diabetes mellitus	130 (6.5), 95% CI ± 1.1	–0.035	0.12
Dyslipidaemia	1119 (56.7), 95% CI ± 2.2	0.067	0.0031
Family history of coronary artery disease	1123 (56.9), 95% CI ± 2.2	0.042	0.061
Smoking	373 (18.9), 95% CI ± 1.7	–0.0065	0.77
Medications			
Nitrates	266 (13.5), 95% CI ± 1.5	0.076	0.0035
Aspirin	807 (40.9), 95% CI ± 2.2	0.072	0.0057
Clopidogrel	649 (32.9), 95% CI ± 2.1	0.099	<0.0001
Warfarin	116 (5.9), 95% CI ± 1.1	0.019	0.71
Oral beta blockers	601 (30.4), 95% CI ± 2.1	0.052	0.067
Calcium channel blockers	316 (16.0), 95% CI ± 1.6	0.10	<0.0001
ACE inhibitors	479 (24.3), 95% CI ± 1.9	0.084	0.001
Diuretics	325 (16.5), 95% CI ± 1.7	0.11	<0.0001
Statins	713 (36.1), 95% CI ± 2.1	0.052	0.071
Angiotensin II antagonists	64 (3.2), 95% CI ± 0.8	0.051	0.081
Heparin		0.087	0.0001
Low-molecular-weight heparin		0.42	<0.0001
GBIII2A		0.50	<0.0001
3+ cardiac risk factors	864 (43.8), 95% CI ± 2.1	0.79	0.0004
Observations	Mean		
Systolic blood pressure	143, 95% CI ± 1.1		
Mean heart rate	76.9, 95% CI ± 0.9		
Mean respiratory rate	18.1, 95% CI ± 0.2		
Outcome	Prevalence (%)		
MACE	305 (15.5), 95% CI ± 1.5		

ACE, angiotensin-converting enzyme; CABG, coronary artery bypass graft; MACE, major adverse cardiac event; PCI, percutaneous intervention.

standard management. While making these diagnoses, the adjudicators were blinded to information that was collected by the research nurses on the study case record forms. Successful follow up was achieved in all patients. MACE was deemed to have occurred in any patient who had suffered an ST-elevation or non-ST-elevation MI, had required an emergency revascularisation procedure, had died of cardiovascular causes,

and had suffered a ventricular arrhythmia, cardiac arrest, cardiogenic shock or a high atrio-ventricular block.

Statistical performance goals

We aimed to achieve a sensitivity of at least 99% as it has been reported that this is the level of sensitivity many ED physicians are comfortable with.[27] All modifications aimed to maximise specificity and achieve a proportion of patients classified as low risk >40% (which would be higher than in previously reported studies) while maintaining a sensitivity of ≥99%.

Development of the EDACS and the EDACS-ADP

We used a two-stage development process (Box 2). In the first stage, a statistically predictive model of MACE was derived without accounting for clinical sensibility. In the second stage, the risk score (EDACS) was derived by improving the clinical sensibility and usability of the statistical model. In addition, EDACS was combined with diagnostics tests (negative troponins and ECGs) to arrive at an accelerated diagnostic protocol (EDACS-ADP).

Stage 1: Statistical predictors and derivation of a statistical risk model

We performed univariate analysis on 37 candidate variables and identified variables with statistical significance of $P < 0.05$ for multivariate analysis (Table 1). Next, useful independent predictors were identified using logistic regression, with backwards elimination at $P < 0.05$, to arrive at a parsimonious model. Overall model fit was assessed with the Hosmer–Lemeshow test.[28] These independent predictors formed the foundation for onward creation of the risk score. Statistical analysis was performed using SAS (SAS Institute, Cary, NC, USA).

Diagnostic tests such as ECG and cardiac biomarkers (i.e. troponin) were not used in model building in order to make the score more versatile. The score would thus be independent of the type of ECG and troponin testing available in EDs. This will minimise the changes needed if new forms of ECG or blood tests such as high-sensitivity troponin assays and multi-vector ECG analysis are introduced in the future.

Stage 2a: Presentation of the model as a preliminary score

The statistical prediction model was then converted into a simpler, more user-friendly scoring system that could be easily calculated by clinicians without electronic assistance.[18] The coefficients from the statistical prediction model were converted into integers by multiplying with whole numbers and rounding to nearest integers. The aim was to find a low-multiplier that allowed all the variables to be rounded to the smallest integers. This ensured that the resulting score was easily calculable while still preserving the predictive value of the statistical model.

Stage 2b: Interim determination of optimal cut-off

The objective of the next stage was to create a preliminary ADP combining negative ECG and troponin results with the preliminary risk-prediction score (below a specified cut-off). The ADP would accurately identify patients at very low risk of MACE. The preliminary risk prediction score's performance was studied in conjunction with the results of ECGs and troponin tests taken on arrival at hospital and 2 h later. A cut-off was sought that maximised specificity while keeping sensitivity ≥99%. The test results were dichotomised so that new cardiac ischaemia on the ECG and positive troponin

Box 2. *Development of the EDACS and the EDACS Accelerated Diagnostic Protocol (EDACS-ADP)*

Stage 1: Statistical derivation	A statistically predictive model for possible MACE was derived.
Stage 2: Clinical sensibility	The clinical sensibility was improved and an ADP developed.
2a	Statistical coefficients were converted to whole numbers to create a preliminary risk score.
2b	An initial score threshold was determined and combined with ECG and troponin results to give a preliminary ADP.
2c	Modifications were made to improve the clinical sensibility and create the EDACS and the EDACS-ADP.

EDACS, Emergency Department Assessment of Chest pain Score; MACE, major adverse cardiac event.

results (above the 99th percentile) were classified as positive results (see Supporting Information Table S1 for test definitions). Although it was not intended for this strategy to be the final ADP, these interim calculations were made so that it was possible to measure the impact of subsequent modifications designed to improve clinical sensibility.

Stage 2c: Final EDACS and EDACS-ADP

Feedback was sought from a convenience sample of clinicians involved in day-to-day assessment of chest pain in the ED. It was used to make modifications to improve clinical sensibility. The statistical impact of the modifications was examined to ensure that the target sensitivity and optimum specificity were maintained.

Interrater agreement

The interrater agreement of the EDACS and the EDACS-ADP was tested. Convenience groups of three clinical staff per patient independently calculated the score and categorised risk according to the ADP in a separate cohort of 60 patients that presented to the ED with chest pain. Results were analysed using inter-class correlation coefficients.

Results

Derivation phase

The derivation cohort contained 1974/1975 participants from the ADAPT cohort, which included 975 recruited in Brisbane, and 999 in Christchurch. Participants were predominantly Caucasian, older men with risk factors for coronary artery disease and a significant rate of known coronary artery disease (Table 1).

Stage 1: Statistical predictors and derivation of a statistical risk model

We identified six statistical predictors after backwards elimination. These were age, sex, pain on inspiration, pain on palpation, pain radiating to arm or shoulder, and diaphoresis (Table 2).

Stage 2a: Presentation of the model as a preliminary score

The beta coefficients were multiplied by eight, which was the smallest common multiplication factor possible to obtain a sensible score that used whole numbers and facilitated clinical ease of use. Age was the only continuous variable to be included in the final score. It was converted to a categorical variable, using 5-year age bands with increasing increments of +2 points.

Stage 2b: Interim determination of optimal cut-off

When combined with ECG and troponin results, the optimum interim risk-prediction score cut-off was 30. Using this cut-off, the preliminary ADP had a sensitivity of 99.0% in the derivation cohort, with a specificity of 52.8% (Table 3). This would have allowed 44.6% of presentations to be classified as low risk, if the interim score was <30 and the cardiac biomarkers and ECG were normal (Supporting Information Box S1).

Stage 2c: Final EDACS and EDACS-ADP

The following issues were found when the clinicians provided feedback on the preliminary score and ADP. First, the interim score would classify all young patients (aged <41 years) as lower risk; this was considered clinically implausible. Second, 17 age bands (with up to +40 points) were considered too many and would make arithmetic difficult. Last, the absence of traditional cardiac risk factors or a history of known coronary artery disease in younger patients (<50 years) was considered to decrease the score's credibility.

Based on the clinician feedback, the interim risk-prediction score was modified as follows to form the final EDACS (Box 3). First, the youngest age groupings were merged into a single group from 18 to 45 years, which made it possible for patients from all age groups to be classified as higher risk. Second, age was categorised into 10 groups by forming one group for patients ≥86 years because the very elderly formed only 3.6% of the derivation cohort. This resulted in 10 age bands increasing by increments of two points, starting with +2

Table 2. Predictors after backwards elimination

Parameter	Beta coefficient	*P* value	Odds ratio
Age	0.051	<0.0001	(per 10 years) 1.500
Male	0.750	<0.0001	2.110
Diaphoresis	0.380	0.0038	1.460
Pain radiates to arm (or shoulder)	0.620	<0.0001	1.850
Pain on inspiration	–0.460	0.0190	0.630
Pain on palpation	–0.760	0.0379	0.470

Estimated with multivariable logistic regression – The intercept –5.73.

Table 3. Accuracy of the preliminary clinical prediction rule and EDACS-ADP

	Preliminary ADP		EDACS-ADP	
	Derivation	Validation	Derivation	Validation
Participants (n)	1974	608	1974	608
MACE				
Not low risk	302	78	302	79
Low risk	3	1	3	0
Sensitivity	99.0 (96.9–99.7)	98.7 (97.9–99.6)	99.0 (96.9–99.7)	100.0 (94.2–100.0)
No MACE				
Not low risk	788	198	836	217
Low risk	881	331	833	312
Specificity	52.8 (50.6–55.0)	62.6 (58.7–66.4)	49.9 (47.4–52.3)	59.0 (54.6–63.2)
Per cent low risk†	44.6 (42.4–46.8)	54.4 (50.5–58.4)	42.2 (40.1–44.5)	51.3 (47.7–55.4)

†*Low risk of MACE within 30 days. ADP, accelerated diagnostic protocol; EDACS, Emergency Department Assessment of Chest pain Score; MACE, major adverse cardiac events.*

points (18–45 years age group) to +20 points (≥86 year age group). Third, traditional risk factors and history of coronary artery disease were incorporated in the score. These were initially univariate predictors of MACE, but were not statistically significant predictors in the final multivariable model, after backwards elimination. Clinicians are already familiar with the Thrombolysis In Myocardial Infarction risk score,[29] which utilises the presence of ≥3 risk factors as an indicator of high risk of an adverse event. Risk factors are reported as more predictive in younger patients,[7] so we assigned ≤50 years as the cut-off below which +4 points were allocated for ≥3 risk factors or proven coronary artery disease. This way, a patient aged 18 years would be allocated the same risk score as a patient aged 51 years who had similar presentation characteristics. The score correlated well with the prevalence of MACE (Fig. 1).

The EDACS was combined with ECG and troponin results to create the EDACS-ADP. A cut-off of 16 was the optimal score to maintain sensitivity near 99% and maximise specificity. There was minimal difference in the diagnostic accuracy between the preliminary and final ADP. In particular, safety (proportion of patients categorised as low risk who had a MACE) was the same in the preliminary and final ADPs (Supporting Information Fig. S1). The EDACS-ADP had the same sensitivity as the preliminary ADP (99.0%; Table 3), similar specificity (49.9% *vs* 52.8%) and a similar proportion of patients eligible for early discharge (42.2% *vs* 44.6%).

Interrater agreement

The intra-class correlation coefficient (ICC) from the calculation of the numerical value of EDACS was 0.80. For the EDACS-ADP categorisation of patients as low risk or not low risk, the ICC was 0.87.

Validation phase results

The EDACS and EDACS-ADP were tested on a separate prospectively recruited validation cohort of 608 patients (431 from Christchurch and 177 from Brisbane). Both the preliminary score and final EDACS and EDACS-ADP performed better in the validation cohort than in the derivation cohort, with similar sensitivity while specificity improved (Table 3). Combining the risk score with troponin and ECG results, 51.3% of the participants recruited were classified as low risk and eligible for discharge at 2 h.

Discussion

In the present study, we developed a clinically sensible score (EDACS) to predict the short-term risk of MACE in adults presenting to the ED with possible cardiac chest pain. We combined EDACS with normal troponin results at 0 and 2 h, and an ECG finding of no new ischaemia to develop the EDACS-ADP. The EDACS-ADP more than doubles the proportion of patients classified as having low short-term risk of MACE compared with the previously reported ADP from the ADAPT study.[14] When validated, the preliminary and final EDACS-ADP classified more than 50% of patients as having low risk of MACE within 30 days of discharge. This is a significant increase in the proportion of patients identified as low risk by studies of similar protocols. In both the derivation and validation cohorts, the sensitivity remained at ≥99%.

Box 3. *EDACS and EDACS-ADP*

EMERGENCY DEPARTMENT ASSESSMENT OF CHEST PAIN SCORE (EDACS)

Clinical Characteristics	Score
a) Age (Please Circle SINGLE Best Answer)	
18–45	+2
46–50	+4
51–55	+6
56–60	+8
61–65	+10
66–70	+12
71–75	+14
76–80	+16
81–85	+18
86 +	+20
b) Male sex (Please circle if true)	+6
c) Aged 18–50 years and either: (i) known coronary artery disease or (ii) ≥3 risk factors	+4
d) Symptoms and signs (Circle each if present)	
Diaphoresis	+3
Radiates to arm or shoulder	+5
Pain† occurred or worsened with inspiration	−4
Pain† is reproduced by palpation	−6
EDACS Total (Please Add all circled figures and enter to right)	_____

EDACS-ACCELERATED DIAGNOSTIC PROTOCOL (EDACS-ADP)

Low risk*	(i) EDACS <16 (ii) No new ischaemia on ECG (iii) 0 and 2 h troponin both negative
Recommendation	Patient safe for discharge to early outpatient follow-up investigation (or proceed to earlier inpatient testing)
Not low risk	(i) EDACS ≥16 (ii) New ischaemia on ECG Either 0 or 2 h‡ troponin positive (see footnote)
Recommendation	Proceed with usual care with further observation and delayed troponin

*Coronary artery disease (CAD) = previous acute myocardial infarction, coronary artery bypass graft or percutaneous intervention. Risk factors = family history of premature CAD, dislipidaemia, diabetes, hypertension, current smoker. †Pain that caused presentation to hospital. ‡A 2 h troponin is only required if other parameters are low risk. ***Safety point**: patients with an unstable presentation (abnormal vital signs or pain that is ongoing or in a crescendo pattern) should not be considered for the low-risk protocol.*

Using the new ADP, more patients could be safely discharged early to outpatient follow-up investigation (or proceed more quickly to further inpatient testing). The ADP has potential to shorten hospital length of stay in a larger proportion of patients than reported previously.[14] It could also make a significant impact in reducing ED overcrowding at minimal cost to the health systems where it is deployed.

The new risk score (EDACS) is flexible in any clinical setting. Clinicians can modify the score cut-off and combine it with any biomarker tests and ECG technology available now or in the future. The good correlation between EDACS and prevalence of MACE in both cohorts suggest that the score might be useful in identifying patients who are at higher risk, need additional investigations or require telemetry.

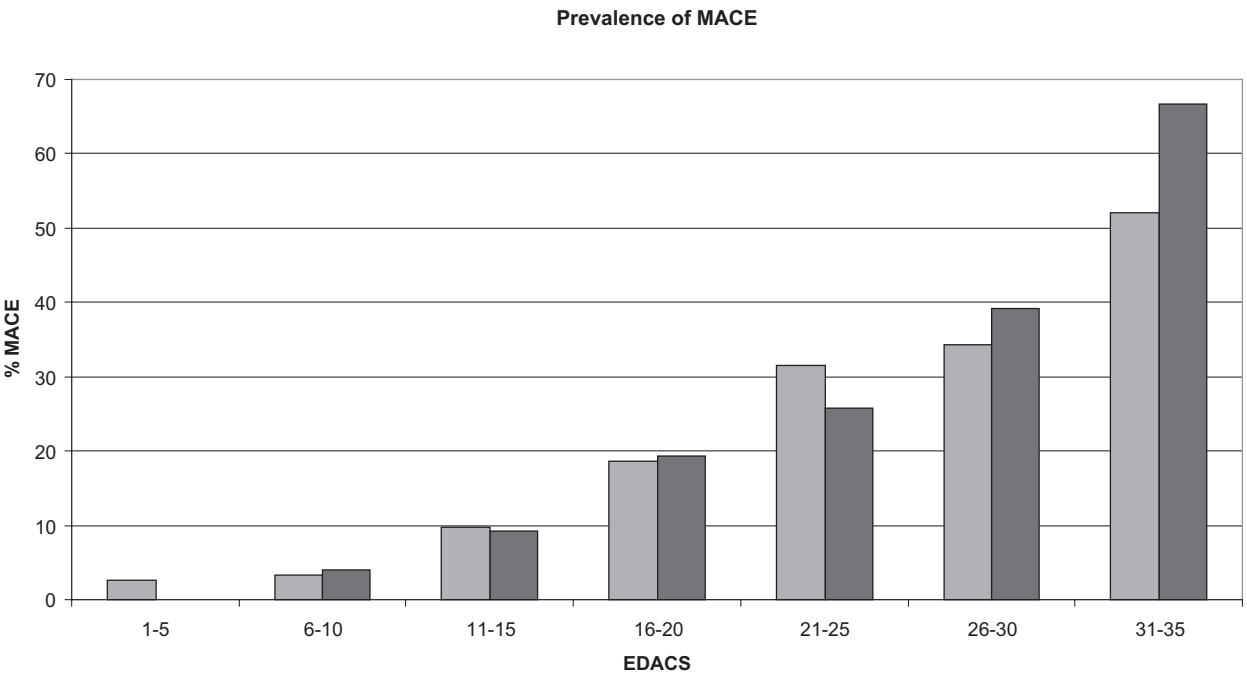

Figure 1. Prevalence of MACE in the derivation and validation cohorts. EDACS, Emergency Department Assessment of Chest pain Score. MACE, major adverse cardiac event; (□), Derivation; (■), validation.

Limitations

Although the EDACS derivation was initially based upon statistical analysis of prospectively collected data, it was modified in the present study. Several adaptations (involving age and risk factors) were made to improve clinical sensibility and the likelihood of clinician uptake, and these modifications might appear to make the score less statistically robust. However, although it might be argued that statistical models are exact, they can be more difficult to apply clinically than prediction models that are presented as a scoring model or rule.[24] Acceptance by clinicians is vital for implementation of the score,[19,24] which is why end-user feedback was sought and integrated into the score. Moreover, these adaptations were achieved with minimal change to the diagnostic performance of the prediction rule, and this was confirmed in the validation cohort. Maintenance of diagnostic performance following simplification has been reported after several well-known clinical scores such as the revised Geneva score and Canadian (Well's) rule for pulmonary embolism.[30–32] We used a statistical significance level of $P < 0.05$ as a criteria for inclusion in multivariate analysis. More lenient P values are now also common, but our priority was to avoid having large numbers of variables in the score in order to maintain simplicity (and hence clinical sensibility as stated above).

It was not logistically possible to determine the interrater reliability of individual clinical variables in the derivation dataset. Therefore, we tested and found excellent inter-class correlation for the EDACS and EDACS-ADP showing that they can be reliably reproduced in clinical practice. We validated the prediction rule in a temporal external cohort of patients. However, we acknowledge that these patients were recruited from the same Australasian centres as the derivation cohort, therefore limiting generalisability.

Conclusion

The EDACS-ADP could safely identify 40–50% of patients presenting to the ED with possible cardiac chest pain as having low risk of short-term MACE. These patients would be suitable for early discharge to outpatient investigation or expedited inpatient tests. This is a large increase in the proportion of patients identified as low risk by studies of similar protocols. The EDACS-ADP has the potential to make considerable cost

reductions to health systems. A pragmatic randomised controlled trial is now desirable to validate the new score and ADP for application in different clinical settings.

Competing interests

None declared.

Accepted 10 November 2013

References

1. Goodacre S, Cross E, Arnold J, Angelini K, Capewell S, Nicholl J. The health care burden of acute chest pain. *Heart* 2005; **91**: 229–30.
2. Pope JH, Aufderheide TP, Ruthazer R *et al.* Missed diagnoses of acute cardiac ischemia in the emergency department. *N. Engl. J. Med.* 2000; **342**: 1163–70.
3. Goodacre S, Locker T, Morris F, Campbell S. How useful are clinical features in the diagnosis of acute, undifferentiated chest pain? *Acad. Emerg. Med.* 2002; **9**: 203–8.
4. Goodacre SW, Angelini K, Arnold J, Revill S, Morris F. Clinical predictors of acute coronary syndromes in patients with undifferentiated chest pain. *QJM* 2003; **96**: 893–8.
5. Body R, Carley S, Wibberley C, McDowell G, Ferguson J, Mackway-Jones K. The value of symptoms and signs in the emergent diagnosis of acute coronary syndromes. *Resuscitation* 2010; **81**: 281–6.
6. Body R, McDowell G, Carley S, Mackway-Jones K. Do risk factors for chronic coronary heart disease help diagnose acute myocardial infarction in the emergency department? *Resuscitation* 2008; **79**: 41–5.
7. Han JH, Lindsell CJ, Storrow AB *et al.* The role of cardiac risk factor burden in diagnosing acute coronary syndromes in the emergency department setting. *Ann. Emerg. Med.* 2007; **49**: 145–52. 52 e1.
8. Christenson J, Innes G, McKnight D *et al.* A clinical prediction rule for early discharge of patients with chest pain. *Ann. Emerg. Med.* 2006; **47**: 1–10.
9. Hess EP, Brison RJ, Perry JJ *et al.* Development of a clinical prediction rule for 30-day cardiac events in emergency department patients with chest pain and possible acute coronary syndrome. *Ann. Emerg. Med.* 2012; **59**: 115–25 e1.
10. Swap CJ, Nagurney JT. Value and limitations of chest pain history in the evaluation of patients with suspected acute coronary syndromes. *JAMA* 2005; **294**: 2623–9.
11. Than MP, Flaws DF, Cullen L, Deely JM. Cardiac risk stratification scoring systems for suspected acute coronary syndromes in the emergency department emergency and hospital medicine reports 2013. *Curr. Emerg. Hosp. Med. Rep.* 2013; **1**: 53–63.
12. Hess EP, Thiruganasambandamoorthy V, Wells GA *et al.* Diagnostic accuracy of clinical prediction rules to exclude acute coronary syndrome in the emergency department setting: a systematic review. *CJEM* 2008; **10**: 373–82.
13. Steurer J, Held U, Schmid D, Ruckstuhl J, Bachmann LM. Clinical value of diagnostic instruments for ruling out acute coronary syndrome in patients with chest pain: a systematic review. *Emerg. Med. J.* 2010; **27**: 896–902.
14. Than M, Cullen L, Aldous S *et al.* 2-hour accelerated diagnostic protocol to assess patients with chest pain symptoms using contemporary troponins as the only biomarker: the ADAPT trial. *J. Am. Coll. Cardiol.* 2012; **59**: 2091–8.
15. Six AJ, Cullen L, Backus BE *et al.* The HEART score for the assessment of patients with chest pain in the emergency department: a multinational validation study. *Crit. Pathw. Cardiol.* 2013; **12**: 121–6.
16. Mahler SA, Miller CD, Hollander JE *et al.* Identifying patients for early discharge: performance of decision rules among patients with acute chest pain. *Int. J. Cardiol.* 2013; **168**: 795–802.
17. Xavier Scheuermeyer F, Wong H, Yu E *et al.* Development and validation of a prediction rule for early discharge of low-risk emergency department patients with potential ischemic chest pain. *CJEM* 2013; **15**: 1–14.
18. Steyerberg EW, ed. *Clinical Prediction Models: A Practical Approach to Development, Validation, and Updating*, 1st edn. New York: Springer, 2009.
19. Moons KG, Kengne AP, Woodward M *et al.* Risk prediction models: I. Development, internal validation, and assessing the incremental value of a new (bio)marker. *Heart* 2012; **98**: 683–90.
20. Royston P, Moons KG, Altman DG, Vergouwe Y. Prognosis and prognostic research: developing a prognostic model. *BMJ* 2009; **338**: b604.
21. Stiell IG, Wells GA. Methodologic standards for the development of clinical decision rules in emergency medicine. *Ann. Emerg. Med.* 1999; **33**: 437–47.
22. Laupacis A, Sekar N, Stiell IG. Clinical prediction rules. A review and suggested modifications of methodological standards. *JAMA* 1997; **277**: 488–94.
23. Reilly BM, Evans AT. Translating clinical research into clinical practice: impact of using prediction rules to make decisions. *Ann. Intern. Med.* 2006; **144**: 201–9.
24. Toll DB, Janssen KJ, Vergouwe Y, Moons KG. Validation, updating and impact of clinical prediction rules: a review. *J. Clin. Epidemiol.* 2008; **61**: 1085–94.
25. Luepker RV, Apple FS, Christenson RH *et al.* Case definitions for acute coronary heart disease in epidemiology and clinical research studies: a statement from the AHA Council on Epidemiology and Prevention; AHA Statistics Committee; World Heart Federation Council on Epidemiology and Prevention; the European Society of Cardiology Working Group on Epidemiology and Prevention; Centers for Disease Control and Prevention; and the National Heart, Lung, and Blood Institute. *Circulation* 2003; **108**: 2543–9.
26. Cullen L, Than M, Brown AF *et al.* Comprehensive standardized data definitions for acute coronary syndrome research in emergency departments in Australasia. *Emerg. Med. Australas.* 2010; **22**: 35–55.
27. Than M, Herbert M, Flaws D *et al.* What is an acceptable risk of major adverse cardiac event in chest pain patients soon after discharge from the Emergency Department?: a clinical survey. *Int. J. Cardiol.* 2013; **166**: 752–4.

28. Hosmer DW, Hosmer T, Le Cessie S, Lemeshow S. A comparison of goodness-of-fit tests for the logistic regression model. *Stat. Med.* 1997; **16**: 965–80.
29. Antman EM, Cohen M, Bernink PJ *et al.* The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision making. *JAMA* 2000; **284**: 835–42.
30. Douma RA, Gibson NS, Gerdes VE *et al.* Validity and clinical utility of the simplified Wells rule for assessing clinical probability for the exclusion of pulmonary embolism. *Thromb. Haemost.* 2009; **101**: 197–200.
31. Douma RA, Mos IC, Erkens PM *et al.* Performance of 4 clinical decision rules in the diagnostic management of acute pulmonary embolism: a prospective cohort study. *Ann. Intern. Med.* 2011; **154**: 709–18.
32. Klok FA, Mos IC, Nijkeuter M *et al.* Simplification of the revised Geneva score for assessing clinical probability of pulmonary embolism. *Arch. Intern. Med.* 2008; **168**: 2131–6.

Supporting Information

Additional Supporting information may be found in the online version of this article at the publisher's web-site:

Figure S1. Impact of clinical sensibility changes on statistical performance in derivation cohort.
Table S1. Technical characteristics and definitions for cardiac troponins and ECG.
Table S2. Data Dictionary of Clinical Variables in EDACS score.
Box S1. Preliminary Risk Prediction Score and Interim ADP.