Comparing risk-prediction methods using administrative or clinical data in assessing excess in-hospital mortality in patients with acute myocardial infarction

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ABSTRACT

Objectives: To compare results of statistical process-control analyses of in-hospital deaths of patients with acute myocardial infarction by using either administrative or clinical data sources and prediction models, and to assess variation in results according to selected patient characteristics.

Design: Retrospective, cross-sectional study comparing variable life-adjusted display (VLAD) curves derived by using administrative or clinical prediction models applied to a single patient sample.

Participants and setting: Data from 467 consecutive patients admitted to a tertiary hospital in Queensland, between 1 July 2003 and 31 March 2006, with a coded discharge diagnosis of acute myocardial infarction.

Main outcome measure: Statistical estimates of cumulative lives gained or lost in excess of those predicted at the end of the study period.

Results: The two prediction models, when applied to all patients, generated almost identical VLAD curves, showing a steadily increasing excess mortality over the study period, culminating in an estimated 11 excess deaths. Risk estimates for individual patients from each model were significantly correlated ($r = 0.46$, $P < 0.001$). After exclusion of misclassified cases, out-of-hospital cardiac arrests and deaths within 30 minutes of presentation, replotting the curves reversed the mortality trend and yielded, depending on the model, a net gain of three or seven lives. After further exclusion of transfers in from other hospitals and patients whose care had a palliative or conservative intent, the net gain increased to seven or 10 lives.

Conclusion: Appropriate patient selection is more important than choice of dataset or risk-prediction model when statistical process-control methods are used to flag unfavourable mortality trends suggestive of suboptimal hospital care.

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Quality and safety concerns have prompted calls for institutions to monitor condition-specific patient outcomes with methods that permit early, real-time identification of unfavourable trends.[1] Statistical process-control methods can quickly identify excess numbers of infrequent events, such as death, by comparing observed outcomes with those expected on the basis of risk-prediction models.

Simple graphical displays include the risk-adjusted cumulative sum[2,3] and the variable life-adjusted display (VLAD).[4,5] Had these tools been in routine use at the time, the Bristol cardiac surgery deaths and the Shipman murders would have been identified as statistical outliers long before proof was forthcoming from conventional comparative statistics.[6] Beginning in July 2006, Queensland Health has adopted regular internal reporting of VLAD analyses of mortality, long stays, readmission rates and complications for 17 different diagnoses and procedures involving 87 public and private hospitals in the state.[7]

Statistical process-control analyses and their inherent condition-specific risk-prediction models can be of two types: (1) based on routinely collected administrative data, such as principal diagnoses and comorbid conditions listed in coded hospital discharge data (administrative method, used by Queensland Health); or (2) based on abstracted clinical datasets of prognostically important patient and process-of-care variables recorded at or after presentation of a patient to hospital (clinical method). Previous comparisons of administrative and clinical databases have found that administrative data were frequently incomplete or miscoded, and analyses were unable to fully adjust for confounders related to administrative data.[8] However, more recent studies of elective surgical admissions suggest that administrative data compare favourably with clinical databases.[9,10] Whether this remains true for acute medical diagnoses is uncertain. Also unclear is the effect on predictive accuracy of the inappropriate inclusion of misdiagnosed cases or patients whose risk of death bears little or no relation to quality of care.

We conducted a study involving patients with acute myocardial infarction (AMI) admitted to a single tertiary hospital. We aimed to: (1) compare, for in-hospital deaths, VLAD analyses that used either administrative or clinical datasets for prediction models; and (2) determine, by sensitivity analysis, the degree to which VLAD results varied according to changes in selected patient and hospital characteristics.

METHODS

Participants, setting and data sources

Study participants were 467 consecutive patients admitted to a tertiary hospital in Queensland, between 1 July 2003 and 31 March 2006, with a primary discharge diagnosis of AMI (International classification of diseases, version 10.3, Australian modification codes I121 and I122) and who met the following criteria: age 30–89 years; hospital stay < 30 days; Queensland resident; acute admission via the emergency department (ED); and not transferred to another hospital. Coded diagnoses were ascertained by review of medical records and, in the case of deaths, of death certificates. Administrative data were collected, retrospectively, by coders on all cases within 3 months of discharge, and clinical data were abstracted by a single clinical investigator (S N) between 1 October and 22 December 2006. Coders and the investigator were blind to study objectives and collected data on standard forms. The study was approved by the chair of the district quality and safety council.

Mortality risk-prediction models

Administrative

The model based on administrative data was derived from multivariate logistic regression analysis of data from 7491 patients admitted to 31 Queensland hospitals (four tertiary, 27 other) between 1 July 2003 and 30 June

1 Independent predictors of mortality in the administrative risk-prediction model

Predictor	Odds ratio (95% CI)
Sex (female v male)	1.10 (0.94–1.28)
Age group (years)	
85–89 v 30–69	3.61 (2.86–4.58)
80–84 v 30–69	3.22 (2.58–4.00)
75–79 v 30–69	2.21 (1.74–2.79)
70–74 v 30–69	2.44 (1.90–3.15)
Dementia	1.47 (1.07–2.02)
Hypotension	3.62 (3.01–4.37)
Renal failure	1.84 (1.54–2.20)
Heart failure	1.31 (1.11–1.54)
Dysrhythmia	2.70 (2.32–3.14)
Malignant cancer	2.12 (1.49–3.03)
Stroke	2.27 (1.66–3.11)
Hypertension	0.70 (0.60–0.82)
Diabetes	1.33 (1.06–1.65)

2006 with a coded discharge diagnosis of AMI. The model comprised 11 independent risk predictors (Box 1) and demonstrated good discrimination (C statistic[11] = 0.80) (Justin Collins, Program Director, Quality Management Statistical Unit, Queensland Health, personal communication, 2007). A very similar model, derived from data from Ontario between 1994 and 1997 (C for 30-day mortality = 0.78), had been validated in 117 070 patients (C = 0.77).[12]

Clinical

The model using clinical data was a logistic regression model based on eight clinical variables (Box 2) derived from a large international registry of 11 389 patients with clinician-verified diagnoses of acute coronary events, including AMI. Studies showed very good discrimination within two cohorts totalling 16 114 patients (C = 0.83 and C = 0.79–0.84 in derivation and validation cohorts, respectively).[14] As most patients in the registry survived to 24 hours after admission and had not presented with out-of-hospital cardiac arrest, we arbitrarily assigned the model's highest possible risk of death (52%) to patients presenting with such arrests and those who died within 30 minutes of ED presentation or lacked data on one or more variables.

The clinical diagnosis of AMI was confirmed in all cases by using internationally accepted criteria of an elevated troponin level and the presence of ischaemic chest pain or unequivocal electrocardiographic changes.[15]

Derivation of statistical process-control displays

The graphical tool used in this study was an adaptation of the VLAD, which incorporated control limits derived from the risk-adjusted cumulative sum.[16,17] Briefly, the VLAD shows a plot, for a series of consecutive patients over time, of the cumulative risk-adjusted difference between observed and expected deaths, expressed as the statiscal estimate of excess lives gained or lost. Upper and lower control limits (based mathematically on the sequential probability ratio test[2] applied to 10 000 iterations of specified datasets of 10 000 patients) were added which, when breached by the curve, corresponded, respectively, to a real 30% decrease or increase in mortality with 95% confidence. Each time the control limit was breached, the limits were reset, with the breach point taken as the new baseline. More in-depth explanations of the mathematics involved can be found elsewhere.[4,5,17]

2 Independent predictors of mortality in the clinical risk-prediction model

Predictor	Odds ratio (95% CI)
Initial serum creatinine, per 88.4 µmol/L increase	1.2 (1.15–1.35)
Heart rate, per 30 beats/min increase	1.3 (1.16–1.48)
Systolic blood pressure, per 20 mmHg decrease	1.4 (1.27–1.46)
Initial cardiac enzyme level elevation	1.6 (1.32–2.00)
Age, per 10-year increase	1.7 (1.55–1.85)
Killip class,* per increase in class	2.0 (1.81–2.29)
ST-segment deviation on admission ECG	2.4 (1.90–3.00)
Cardiac arrest on arrival at hospital	4.3 (2.80–6.72)

ECG = electrocardiogram.
* Killip classification of heart function. Class I: absence of any signs of heart failure; Class II: inspiratory crackles on chest auscultation, with or without elevated jugular venous pressure; Class III: pulmonary oedema on chest x-ray; Class IV: cardiogenic shock.[13] ◆

Patient and hospital characteristics selected for sensitivity analyses

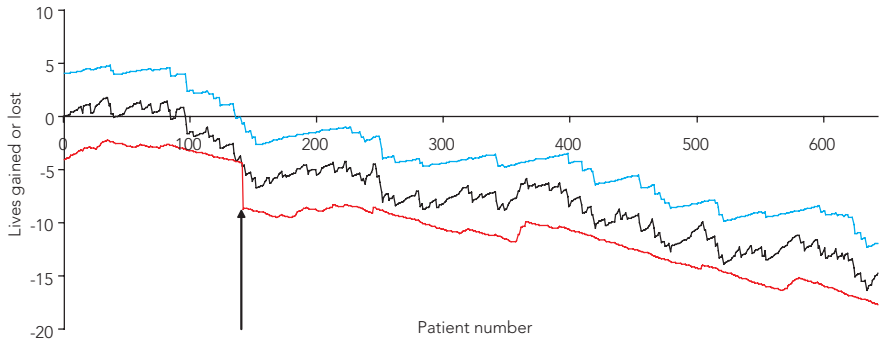
When preliminary results of the VLAD based on administrative data were presented to

3 Patient characteristics and statistically significant mortality associations (all $P < 0.001$) of 467 patients with coded discharge diagnosis of acute myocardial infarction

Factor*	Total ($n = 467$)	Died ($n = 67$)	Survived ($n = 400$)	Odds ratio† (95% CI)
Age at admission: mean (± SD)‡	65.4 (13.7)	73.1 (11.9)	64.1 (13.6)	—
Male sex§	314 (67.2%)	33 (49.3%)	281 (70.3%)	0.41 (0.24–0.69)
Misclassification (misdiagnosis or unsubstantiated diagnosis)¶	20 (4.3%)	9 (13.4%)	11 (2.8%)	5.49 (2.18–13.8)
Out-of-hospital cardiac arrest¶	14 (3.0%)	14 (20.9%)	0 (0%)††	106 (14–820)
Death in ED within 30 min of presentation¶**	5 (1.1%)	5 (7.5%)	0 (0%)	NA
Patients deemed to have had palliative or conservative care§	75 (16.1%)	23 (34.3%)	52 (13.0%)	3.50 (1.85–6.47)
Patients cared for by non-cardiology teams§	111 (23.8%)	39 (58.2%)	72 (18.0%)	6.35 (3.53–11.4)
Estimated probability of death, median (IQR): administrative model‡	0.06 (0.03–0.15)	0.22 (0.10–0.40)	0.05 (0.03–0.10)	—
Estimated probability of death, median (IQR): clinical model‡	0.05 (0.03–0.16)	0.40 (0.18–0.52)	0.05 (0.02–0.10)	—

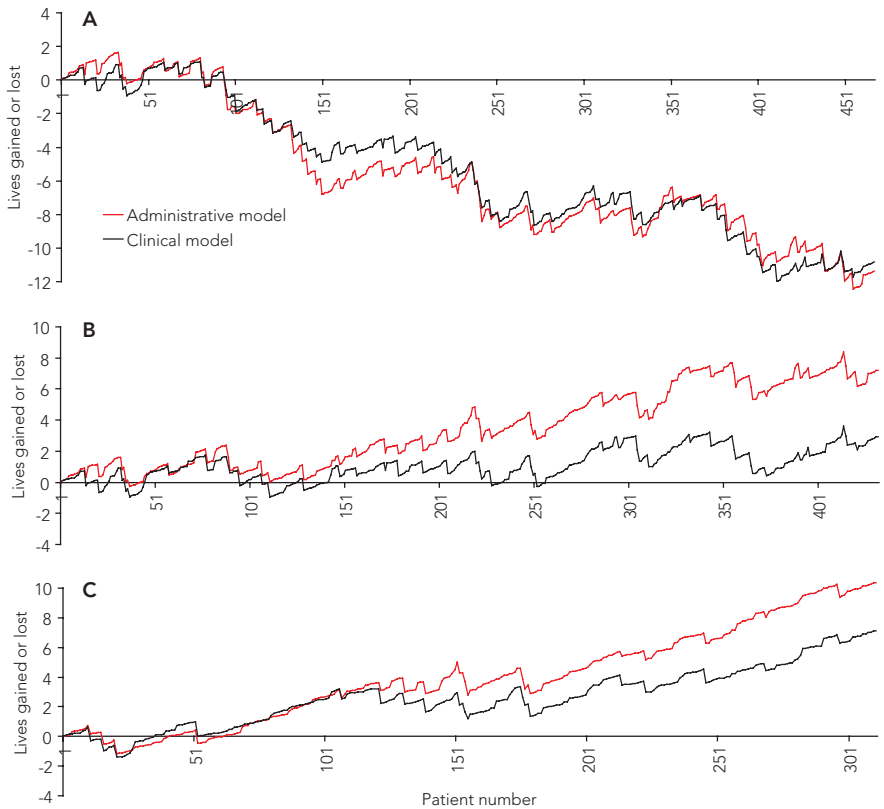
ED = emergency department. NA = not applicable, as this is a mortality outcome not a risk factor. IQR = interquartile range. Numbers are *n* except where specified.
* Except for age and sex, categories are not mutually exclusive. † Odds ratio (OR) indicates the extent to which the stated binary factor increases (OR > 1.0) or decreases (OR < 1.0) the risk of death on the basis of univariate (unadjusted) analysis. ORs do not apply to age and estimated death probabilities, as these are continuous variables. ‡ Mann–Whitney test (2-sided). § Pearson χ^2 (2-sided). ¶ Fisher's exact test (2-sided). ** Missing data = 1. †† OR calculated by imputing 1 in the zero cell. ◆

4 Original variable life-adjusted display derived from the administrative model of risk prediction



The black line shows the cumulative risk-adjusted difference between observed and expected deaths in the sample of patients with acute myocardial infarction (n = 467) from the administrative risk-prediction model. Blue and red lines represent upper and lower control limits, respectively. Lives gained are shown by positive values on the x axis and lives lost by negative values. The arrow indicates that 30% excess mortality was signalled with 95% confidence at Patient 134. ◆

5 Variable life-adjusted display curves derived from administrative and clinical risk-prediction models, with and without patient deselection



A: The lines show the cumulative risk-adjusted difference between observed and expected deaths in the sample of patients with acute myocardial infarction (n = 467) for the administrative and clinical models. Lives gained are shown by positive values on the x axis and lives lost by negative values.

B: Misclassified cases, out-of-hospital cardiac arrests and deaths in the emergency department within 30 minutes of presentation have been excluded (n = 430, after exclusion of 37 cases).

C: Misclassified cases, out-of-hospital cardiac arrests, deaths in the emergency department within 30 minutes of presentation, transfers in, and patients having care with palliative or conservative intent or residing in nursing homes have been excluded (n = 311, after exclusion of 156 cases). ◆

senior clinicians in the hospital for interim feedback, they requested sensitivity analyses of the results to exclude: (1) misclassified cases (a clinically evident alternative diagnosis or unsubstantiated diagnosis of AMI); (2) out-of-hospital, out-of-ambulance cardiac arrests or deaths in the ED within 30 minutes of presentation, both scenarios representing patients whose very high mortality risk was independent of quality of hospital care, and in whom AMI as the cause of death listed on many death certificates had not been confirmed by autopsy or test results; (3) complicated patients transferred in from community hospitals whose high mortality risk might be underestimated by risk-prediction models; (4) patients with end-stage or terminal comorbidity who warranted a more conservative or palliative approach (dialysis-dependent, non-transplantable renal failure, severe oxygen-dependent chronic obstructive pulmonary disease, metastatic cancer, severe frailty or dementia, severe chronic liver failure, or advanced age [>85 years] associated with poor quality of life); and (5) patients residing in high-level care nursing homes whose care had not already been classified as being of palliative intent.

Clinicians also questioned the use of a risk-prediction model whose reference population was all patients admitted to all Queensland hospitals, as opposed to patients at high risk admitted to tertiary hospitals only.

Statistical analysis

Associations between patient variables and mortality were identified by using χ^2 methods and were expressed as odds ratios (ORs) with 95% confidence intervals. Independent predictors were determined by multivariate logistic regression models. Two VLAD curves plotted from the administrative and clinical models were compared, and the models were assessed for their discriminatory power (C statistic[11]) and goodness of fit (Hosmer–Lemeshow χ^2 test, a measure of calibration),[18] as applied to the study sample. The curve was also replotted by using an altered administrative risk-prediction model whose reference population included only patients admitted to tertiary hospitals.

In sensitivity analyses, curves for both models were replotted, after sequential exclusion of the previously defined patient groups, and compared. The incidence of non-fatal adverse events related to health care occurring during admission in patients who survived more than 24 hours after presentation was determined.

RESULTS

Patient characteristics and risk associations

Of the 467 patients included in the original administrative dataset (Box 3), most were male, and the mean age was 65 years. Of these patients, 416 (89%) were acute admissions to the ED from the community, and 447 (96%) had a confirmed diagnosis of AMI. Of 67 patients who died in hospital, nine represented either misdiagnosis (two with alternative diagnoses of septic shock and pulmonary thromboembolism) or unsubstantiated diagnoses (seven with AMI listed as an unconfirmed cause of death on the death certificate), 14 had out-of-hospital, out-of-ambulance cardiac arrests in which circulation was lost for at least 15 minutes, and 23 were deemed as receiving care with palliative or conservative intent. Of 400 survivors, 11 cases were misdiagnosed cases of unstable angina.

Variables associated with a significantly increased risk of death on univariate analysis were out-of-hospital arrest, misclassification, palliative or conservative care intent and care received from non-cardiology teams, while being male was associated with less risk (Box 3). After multivariate regression analysis, misclassification (OR = 4.52; 95% CI, 1.49–13.68; $P = 0.008$), transfers from other hospitals (OR = 3.20; 95% CI, 1.30–0.92; $P = 0.01$) and care from a non-cardiology team (OR = 3.56; 95% CI, 1.50–8.44; $P = 0.004$) remained significant as independent associations.

When applied to the total patient sample, the clinical model showed slightly better discrimination (C = 0.89; 95% CI, 0.85–0.94) than the administrative model (C = 0.84; 95% CI, 0.81–0.88). Goodness of fit of both models was low (Hosmer–Lemeshow $\chi^2 = 22.69$ and $\chi^2 = 25.57$, respectively; $P \leq 0.004$ for both), although the clinical model generated significantly higher risk estimates for patients who died than did the administrative model: median (interquartile range) values were, respectively, 40.0% (34.0%) and 21.5% (29.8%), $P < 0.001$.

Comparison of VLADs derived from administrative and clinical risk models

The original VLAD for the study hospital based on the administrative risk-prediction model (Box 4) showed a steadily increasing excess mortality over the 33-month study period, culminating in an estimated 11 excess deaths, with 30% excess mortality signalled with 95% confidence at Patient 134. A replotted administrative curve using tertiary hospital patients only as the reference population (not shown) showed a nearly identical pattern, with signalling of excess mortality delayed marginally to Patient 139.

Applying the clinical prediction model to the original VLAD also produced a nearly identical curve (Box 5 [A]), with significant correlation between models in risk estimates for individual patients ($r = 0.46$, $P < 0.001$) and similar ranges for risk estimates: administrative 1.6% to 63.9%, clinical 0.2% to 52.0%.

Sensitivity analyses

Replotting of data from both models, after exclusion of misclassified cases, out-of-hospital, out-of-ambulance cardiac arrests and deaths in ED within 30 minutes of presentation ($n = 37$), yielded two upwardly directed curves (Box 5 [B]) which, depending on the model, represented a net gain of three or seven lives at the end of the study period, with no breaching of control limits (not shown). This upward pattern persisted as previously defined patient groups were sequentially excluded (data not shown), with the final plot after deselection of 156 patients showing a net gain of seven or 10 lives (Box 5 [C]).

In-hospital adverse events

There were three health care-related serious events (two episodes of major bleeding requiring transfusion and one of reversible renal failure requiring temporary dialysis) among 400 survivors (0.6%) and none among the 67 who died.

DISCUSSION

The results of this study indicate that appropriate patient selection is more important than the choice of dataset or risk-prediction model when employing statistical process-control methods to detect excess (and potentially avoidable) in-hospital deaths in patients admitted with AMI. In particular, patients with a misdiagnosis or those in whom diagnosis of AMI cannot be substantiated, those presenting with prolonged out-of-hospital cardiac arrest, and those dying very soon after admission need to be removed from the cohort of evaluable cases, as their outcomes bear no relation to the quality of in-hospital care. The inclusion of patients deemed to be receiving care with palliative or conservative intent or requiring high-level nursing care should also be viewed cautiously,[19] although we are by no means implying that a therapeutically nihilistic approach be adopted in all such cases.

Study limitations

The first limitation of our study relates to incomplete ascertainment of all cases of AMI in the original administrative dataset, because of misdiagnosis by clinicians or error by coders. During the study period, the hospital pathology laboratory reported 2970 instances of elevated troponin I, a sensitive biomarker for AMI, but only 447 correctly coded cases of AMI. While a detailed audit has not been done, we suspect that at least half of these "positive" troponin assays represent non-AMI elevations (≤ 0.1 ng/mL),[20] and another third comprise serial assays performed on the same patient. This reduces the number of cases with probable AMI to 990, corresponding to a sampling fraction in our study of 45%, which we contend constitutes a representative sample.

Second, unlike some investigators,[21] we excluded patients transferred in from other hospitals, because performance within a single tertiary hospital was being assessed, not the performance of a regional health care system in which timely interhospital transfer of patients requiring tertiary care is a quality indicator. Moreover, initial management of transferred patients at the referring hospital, which may increase mortality risk, is not within the control of the study hospital.

Third, while some of the deaths we labelled as being misclassified may in fact have constituted cases of AMI, we argue that diagnostically uncertain cases lacking confirmatory autopsy or investigation results should be excluded from analysis.

Fourth, the mortality risk of patients presenting with out-of-hospital cardiac arrest or dying shortly after presentation may have been underestimated, despite assignment of the highest possible mortality risk in both models.

Fifth, we suspect the association of increased death risk with non-cardiologist care reflects selection bias, as virtually all patients presenting with AMI to this hospital are referred in the first instance to cardiologists, who may decline care on the basis of age and comorbidity burden.

Finally, while goodness of fit for both risk-prediction models was low for the total patient sample, this significantly improved after patient deselection (Hosmer–Lemeshow test $P > 0.10$ for both), with no decrease in discrimination (C = 0.92 and C = 0.85).

Implications for clinical practice

VLAD analyses have the ability to detect runs of favourable or adverse outcomes and provide timely, regular updates as "real-time" reporting. Such analyses are feasible on a large scale and at low cost when they use readily available administrative data routinely collected from all hospitals.[22] However, the accuracy of such data has been questioned because of variations in the results of same-condition mortality analyses that use different administrative datasets[23] and between those using administrative versus clinical data.[24,25]

In response, Queensland Health has adopted a multitiered flagging mechanism which begins with clinicians first examining the validity of the administrative data and risk-adjustment models used for specific indicators.[7] The accuracy of risk adjustment of administrative data may also be improved by adding a small number of readily available laboratory results[26,27] or clinical variables[28] to administrative datasets.

This study suggests that a-priori determination of appropriate patient selection criteria may be an even more important consideration than risk adjustment in preventing VLAD analyses unfairly labelling individual institutions as being poor performers. Coding practices in regard to admission status, diagnosis or complication, and verifiable cause of death need to be reviewed and standardised with input from clinicians. Audit-based coding error rates of 17% for principal discharge diagnoses and missing-diagnosis rates of 27% across Queensland hospitals (KPMG Consulting. Casemix coding audit and process review, 2002) need to be corrected if VLAD analyses are to adequately account for differences in risk between hospitals[29] and be acceptable to clinicians.[30] Mandated use of clinician-verified primary data contained within regularly audited structured discharge summaries may be one possible solution.

The early experience of Queensland Health with VLADs has shown that many flags, indicating that review is required, relate to data coding problems and inadequate risk adjustment; correction of these has resulted in flag cancellation. Data collected between 1 July 2004 and 30 September 2007 at the study hospital have not given rise to any further flags (Kirstine Sketcher-Baker, Senior Analyst, Quality Management Statistical Unit, personal communication, 2007), which may reflect improved coding.

Finally, VLADs and related tools do not, in themselves, provide definitive proof of, or explanations for, lower quality care. Their results should not be used in interhospital comparisons for purposes of ranking, but to monitor outcomes within single institutions over time. If excess mortality is found, then in-depth, clinician-led investigations should be initiated to identify and remedy system-of-care problems (including inadequate resourcing) or impaired professional performance.

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COMPETING INTERESTS

None identified.

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