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ORIGINAL RESEARCH

Effect of change of high-sensitivity troponin I assay on emergency department diagnosis and disposition of patients with possible acute coronary syndrome

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Abstract

Objective: Changing cardiac troponin (cTn) assays may affect a hospital's admission and myocardial infarction rates. The effect of changing from a contemporary to high-sensitivity (hs) cTnI assay has been well described, but the real-life impact on disposition and diagnosis of changing from one hs-cTnI assay to another has not.

Methods: All patients who received a troponin measurement in the ED during 16 weeks were adjudicated to identify those the attending physician investigated for possible acute coronary syndrome (ACS) and for clinical outcomes. The Abbott ARCHITECT hs-cTnI assay was in use for the first 8 weeks, followed by the Beckman Coulter hs-cTnI assay for the second 8 weeks.

Results: Fewer patients were investigated with the Abbott assay (2213), than the Beckman assay (2683). A larger proportion were investigated for ACS in the Abbott (64.8%) than the Beckman (60.3%) arm. Among those investigated for ACS the rate of myocardial infarction decreased on changing from Abbott (12.8%) to Beckman (8.8%). Adjusted odds of a myocardial infarction were lower for the Beckman arm, Odds Ratio 0.69 (95% CI 0.55 to 0.88). A lower proportion of Abbott than Beckman had myocardial injury (at least one ED cTnI ≥ upper reference limit) 26.4% compared with 29.8%. The proportion admitted to hospital decreased from the Abbott arm (42.5%) to the Beckman arm (36.8%).

Conclusions: A change in cTnI assay resulted in a decreased rate of myocardial infarction and admission despite an increase in rate of myocardial injury among patients investigated for ACS.

Key words: *cardiovascular diagnostic technique, emergency departments, myocardial infarction, troponin.*

Key findings

- Changing from one high-sensitivity troponin I assay (Abbott) to another (Beckman) decreased the rate of diagnosis of myocardial infarction.
- Changing Abbott to Beckman reduced the overall proportion of patients admitted to a hospital ward.
- Changing from the Abbott to Beckman increased the proportion of patients with troponin results in the ED above the upper reference limit.

Introduction

All cardiac troponin I (cTnI) assays measuring the circulating concentration target the cardiac isoform of troponin I. However, they vary by methodology and target epitopes on the cTnI molecule. This alone is likely to result in different result concentrations. They also vary in calibration and analytical characteristics (upper reference limits [URLs], limit of quantitation [LoQ], limit of detection [LoD] and coefficients of variation [analytical precision] at decision threshold).[1–3] Additionally, decision thresholds, URLs and low-risk thresholds, are likely to have been developed in different cohorts with different numbers of subjects. At the URLs, for example, some

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discordant results between assays are likely.[4–6] These thresholds are used in accelerated diagnostic pathways (ADPs) to risk-stratify patients in the ED. Therefore, it is expected, that changing assays within hospital may change both the risk stratification for acute coronary syndrome (ACS) and the rates of diagnosis of ACS.[7,8] To date studies have compared the effect of changing from a contemporary to a high-sensitivity troponin assay on MI diagnosis,[9–11] but not the real-life impact on disposition and diagnosis of changing between two high-sensitivity I assays. The primary aim of the present study was to assess the effect of a change in high-sensitivity troponin assay on the disposition and diagnosis of patients being assessed for possible ACS.

Methods

This before-after study examines a change of troponin assay in the ED of a tertiary hospital, Christchurch (New Zealand), which received about 130 000 presentations per-annum. Christchurch has been using ADPs as routine care for assessing possible ACS since the end of the first randomised controlled trial to assess the effectiveness and safety of such pathways in 2012.[12] Since then the ADP has been adapted to include the Emergency Department Assessment of Chest Pain score (EDACS),[13] low-risk stratification with just one troponin measurement with an hs-cTnI assay,[14] and a next-day troponin arm to the ADP for other low-risk patients.[15] Data collection was from 0000 h 11 June 2021 to 2359 h 12 August 2021 (before period: 63 days) and 0000 h 01 September 2021 to 2359 h 02 November 2021 (after period: 63 days). A 2-week gap between periods where no data was collected was included to allow imbedding of the pathway with the second assay. The Abbott ARCHITECT hs-cTnI assay was used in the before period (Abbott arm), and the Beckman Coulter hs-cTnI assay in the after period (Beckman arm). Analytical characteristics of the assays are given in the Supporting Information.

The detailed ADPs in use in both the before and after periods are presented in the Supporting Information. Briefly, only patients with no new ischaemia on ECG (negative ECG), a low, <16, Emergency Department Assessment of Chest Pain Score (EDACS), and a first cTn result less than the low-risk threshold (as previously determined for each assay) were to be considered for early discharge. Patients with EDACS <16 and non-ischemic ECG, but with the first cTn slightly above the low-risk threshold and below an intermediate threshold, which is less than the URL, may be discharged to receive cTn measurement in a community laboratory the following day. If that cTn was elevated, they were called back to the ED. The safety of this process has been established.[14] Patients with EDACS ≥16, possible new ischaemia on ECG, or first cTn above the intermediate threshold had a second cTn measurement from a blood sample taken 2 h after the first.

Patients were included if they were ≥18 years old and had at least one troponin measurement in the ED within 4 h of presentation. Patients did not fit the criteria for use of the ADP if they had a STEMI diagnosed in the ED, crescendo angina, unstable vital signs or signs of haemodynamic instability, or had other conditions that required in-hospital investigation or treatment. Patients were also excluded from the analysis if the clinician commented in the patient's note that they did not investigate for ACS, that the pain was clearly musculoskeletal, or that there was no indication for cTnI, or that the patient had been initially assessed at another hospital with an ED and subsequently transferred to Christchurch Hospital.

Adjudicators applied exclusion criteria after review of the patient's electronic health record comprising laboratory test results, the ED assessment and triage notes, cardiology or other specialty consult notes, General Practitioner (GP) referral notes if the patient was referred to the ED or cardiology by a GP, and patient admission and discharge summaries if the patient was admitted to hospital. Where there were inconsistencies or discrepancies between notes, ED assessment notes were given priority, then cardiology or other specialty consult notes, then admission notes, then discharge notes, then GP referral notes, and finally ED triage notes.

Three regular adjudicators were used. A standard operating procedure (SOP) was developed at the beginning of the data collection process to ensure consistency between adjudicators (Supporting Information). The SOP included detailed definitions for different categories that the adjudicators were collecting, to reduce variability in what information was being recorded. The primary categories patients were put in are in Table 1.

To ensure consistency between adjudicators a sample data set from 2 days prior to the study was used by all adjudicators at the beginning of their data collection period. Any disagreements between adjudicators about specific patients were discussed until consensus was achieved. Where an adjudicator was unsure of any aspect of a specific patient's data, they could flag the patient for review by one of the other adjudicators. This second adjudicator would review the data, identify if any changes needed to be made, and record these with their initials with the patient's data. Further escalating to an ED consultant or cardiologist for review happened when there was disagreement between the two adjudicators.

Outcomes were classified by ICD10 codes (Supporting Information). We purposefully chose to use ICD10 coding for this real-life study as they are based on the attending physician's notes and diagnoses.

We report differences in the rate of troponin use among all ED presentations, the rates of troponin results above the URL, and the rate of ACS investigation among those in the adjudicated cohort.

The primary category of interest was those undergoing ACS investigation (Category 1: ACS). Within this category the primary outcome of interest was myocardial infarction. We also report the admission to a

TABLE 1. *Patient categories*

Category	Definition (short form)
1: ACS	The ED assessment notes show evidence of gestalt judgement from the attending physician in ED to investigate the patient for ACS
2: Re-present	The patient had re-presented to the ED after a follow-up cTn test organised during previous investigations for ACS.
3: Other condition	The ED assessment notes showed evidence that the physician did not suspect ACS and the cTn has been ordered to investigate a different suspected condition (e.g. myocarditis, aortic dissection, SCAD, Takotsubo, myocardial contusion)
4: STEMI	Patient presented to the ED with a STEMI (on arrival) [excluded].
5: CA	Patient arrived in ED post cardiac arrest or arrested very soon after arrival.
6: N	None of the above

All categories require at least one troponin measurement had been made in the ED.

hospital ward rate, the rates of troponin results above and below the low-risk threshold and URL for each assay, and the ED length of stay. A cox-regression model was constructed for the outcome of subsequent MI or death within 1 year adjusted for age and sex. Demographic differences between study arms were also investigated. Subgroups were by sex and ethnicity.

Data are presented as *n* (%), mean (standard deviation) or median (interquartile range). Differences between the Abbott and Beckman arms are expressed with 95% confidence intervals calculated using bootstrapping or a test of proportions. All statistical analyses were performed in R.[16]

The study was deemed out of scope by the National Health and Disability Ethics Committee as it was considered an audit, and approved by the University of Otago Human Ethics Committee under their Minimal Risks Health Research procedure after peer review.

Results

During the 126-day study duration, 4896 patients received at least one troponin measurement. More patients received troponin measurements in the Beckman arm (2683; 42.6 per day) than the Abbott arm (2213; 35.1 per day), Table 2, representing an increased rate of troponin testing of 7.5 (21.2%) per day (95% CI 5.2 to 9.7). The mean age was 2 years younger in the Beckman arm than the Abbott arm. The sex distribution also changed with a higher proportion of females in the Beckman arm (net difference 3.7%, 95% CI: 0.9% to 6.5%).

More patients had at least one troponin measurement greater than or equal to the assay's URL for the Beckman (31.2%) than the Abbott (26.1%) assay (difference: 5.1%, 95% CI 2.6% to 7.7%).

A larger proportion of patients were investigated for possible ACS with the Abbott assay (64.8%) than the Beckman assay (60.3%), difference 4.5% (95% CI: 1.7% to 7.2%) (Table 3).

Disposition and diagnosis in category 1: ACS

Among those being investigated for possible ACS, those investigated using the Beckman assay were younger on average, more likely to be female and more likely to have a cTnI ≥ URL (sex-specific) (Table 4).

The rate of MI was considerably lower, with 8.8% ($n = 143$) of those investigated with the Beckman assay, and 12.8% for the Abbott assay ($n = 184$). This corresponds to a 34% lower likelihood of being diagnosed with an MI (OR: 0.66, 95% CI 0.52 to 0.83). After adjustment for age and sex the decrease was 31% (OR: 0.69, 95% CI 0.55 to 0.88).

Within category 1, a smaller proportion were admitted to hospital with the Beckman (36.8%) than the Abbott assay (42.5%).

The proportion of patients with their first blood draw cTnI below the low-risk threshold for each assay was similar, 48.6% for Abbott, 46.5% for Beckman; difference −2.1% (95% CI: −5.7% to 1.5%). The patients were younger than the overall cohort, though the Abbott were still older than the Beckman (median 55 years compared with 53 years) and less likely to be female (527% compared with 56.6%). Of these, 18.5% Abbott and 12.2% Beckman were admitted to a ward, a 34% decrease; absolute difference −6.3% (95% CI: −10.0% to −2.6%). The differences in ages in those admitted was greater, median 66 years for Abbott and 61 years for Beckman, as was the differences in females, 48.8% for Abbott and 58.7% for Beckman.

The lengths of stay in the EDs were similar between arms, $P = 0.51$ (median [IQR] for Abbott 4.3 h [3.1 h–5.9 h] and Beckman 4.2 h [3.2 h–5.7 h]).

The adjusted hazard ratio for subsequent MI or death for Beckman relative to Abbott was 0.91 (95% CI: 0.70 to 1.16, $P = 0.44$). Among those not admitted it was 0.81 (0.50 to 1.3, $P = 0.37$).

TABLE 2. *Demographics*

	Abbott ($N = 2213$)	Beckman ($N = 2683$)	Total ($N = 4896$)
Age, year, mean (SD)	64.0 (17.9)	61.9 (18.9)	62.8 (18.5)
Sex, female	1015 (45.9%)	1330 (49.6%)	2345 (47.9%)
Ethnicity			
European	1766 (79.8%)	2095 (78.1%)	3861 (78.9%)
Māori	189 (8.5%)	252 (9.4%)	441 (9.0%)
Asian	148 (6.7%)	172 (6.4%)	320 (6.5%)
Pacific Peoples	64 (2.9%)	89 (3.3%)	153 (3.1%)
Middle Eastern/Latin American/African	17 (0.8%)	37 (1.4%)	54 (1.1%)
Other ethnicity	29 (1.3%)	38 (1.4%)	67 (1.4%)
Arrival method			
Ambulance	1328 (60.0%)	1587 (59.2%)	2915 (59.5%)
Walk-in	868 (39.2%)	1079 (40.2%)	1947 (39.8%)
Helicopter	15 (0.7%)	7 (0.3%)	22 (0.4%)
Police	2 (0.1%)	2 (0.1%)	4 (0.1%)
Unknown	0 (0.0%)	8 (0.3%)	8 (0.2%)
Chief complaint			
Chest pain	1161 (52.5%)	1434 (53.4%)	2595 (53.0%)
Shortness of breath	257 (11.6%)	261 (9.7%)	518 (10.6%)
Palpitations	125 (5.6%)	180 (6.7%)	305 (6.2%)
Collapse and/or syncope	136 (6.1%)	139 (5.2%)	275 (5.6%)
Abdominal pain	95 (4.3%)	130 (4.8%)	225 (4.6%)
General weakness/fatigue/unwell	49 (2.2%)	79 (2.9%)	128 (2.6%)
Dizziness/vertigo	51 (2.3%)	47 (1.8%)	98 (2.0%)
Fall(s)	35 (1.6%)	46 (1.7%)	81 (1.7%)
Fever symptoms	21 (0.9%)	42 (1.6%)	63 (1.3%)
Altered mental state	29 (1.3%)	27 (1.0%)	56 (1.1%)
Other	254 (11.5%)	298 (11.1%)	552 (11.3%)
Triage category			
1	44 (2.0%)	33 (1.2%)	77 (1.6%)
2	1130 (51.1%)	1285 (47.9%)	2415 (49.3%)
3	956 (43.2%)	1242 (46.3%)	2198 (44.9%)
4	81 (3.7%)	119 (4.4%)	200 (4.1%)
5	2 (0.1%)	4 (0.1%)	6 (0.1%)

Post-hoc investigation into the difference in MI rate in category 1: ACS

A higher proportion of those investigated for ACS had troponin results ≥ URL in the Beckman than the Abbott arms (Fig. 1). This was true for both males and females. In the Beckman arm, the proportion was slightly greater for females than males. Of those with a troponin result ≥ URL, 46.7% in the Abbott arm and 58.3% in the Beckman arm were admitted (difference net 11.6%, 95% CI 6.0% to 17.2%). However, the overall proportion of patients admitted was lower in the Beckman arm (Fig. 1). The proportion of all patients with an MI was lower for both females and males in the Beckman arm. Of those admitted, the proportion with MI was 30.2% in the Abbott arm and 24.0% in the Beckman arm (difference net 6.1%, 95% CI: 1.1% to 11.1%).

TABLE 3. *Adjudicated categories*

Adjudication category	Abbott ($N = 2213$)	Beckman ($N = 2683$)
1: ACS	1434 (64.8%)	1619 (60.3%)
2: Re-present	1 (0.0%)	2 (0.1%)
3: Other condition	54 (2.4%)	137 (5.1%)
4: STEMI	15 (0.7%)	8 (0.3%)
5: CA	14 (0.6%)	9 (0.3%)
6: N	695 (31.4%)	908 (33.8%)

Discussion

Among those investigated for possible ACS there was a lower rate of MI in the Beckman arm than the Abbott arm. We are sceptical that the 'true' rate of myocardial infarction changed as much as the results of the present study suggest over the short period. There was also a lower proportion admitted with Beckman than Abbott, but of those with troponin ≥ URL a higher proportion admitted with Beckman than with Abbott. This may reflect the process of adjusting to a new assay. However, the differences in rates of MI are difficult to attribute just to physician behaviour, in this case inpatient physicians (Cardiologists and Internists), associated with lack of familiarity with a new assay. We may speculate that it is because of a difference between the numeric values reported (Beckman are lower) or the rate of change of one assay compared to another post a myocardial injury may be different, as it is between the Abbott assay and the Roche hs-cTnT assay,[17] but we have no evidence that this is so. The differences in disposition and diagnosis between assays appears not to have affected 1-year MI or death rates. We recommend careful investigation of this phenomena in other hospitals undertaking a change of assay.

Following a change of hs-cTnI assay from the Abbott ARCHITECT to the Beckman Coulter there were more patients per day having troponin measured and including determined to be under investigation for possible ACS and the patients were on average younger and more likely to be female. This was unexpected and it is not immediately apparent why there should have been a small, but noticeable, demographic shift. We may speculate that this is an effect of clinicians acting a little more cautiously with an unfamiliar assay. This may also explain why a lower proportion of those who received a troponin measurement were determined to be under investigation for possible ACS. This may also, in part, explain some of the difference in MI rates. Of note, we are unaware of any educational initiatives or other publicity which would encourage clinicians to increase investigation for ACS in females during the period of the study.

The rate of patients under investigation for ACS admitted to hospital was lower in Beckman than Abbott, including among those with first troponin measure below each assay's low-risk threshold. The difference was greatest for males. In part, this may be explained by the fact that not all URLs are created equal. The URL is the 99th percentile value from a healthy population. These are not measured precisely; the Abbott 90% CIs for the URLs are for females is 14 to 18 ng/L and for males 29 to 39 ng/L, whereas the respective Beckman 95% CI for the URLs are 8 to 18 ng/L and 14 to 43 ng/L.[18–20] Therefore, it is not surprising that the proportions with troponin concentration ≥ URL was different, greater in the Beckman than Abbott arms. This has been seen previously in paired measurements from stored samples from blood donors between the Abbott and an earlier generation Beckman assay (AccuTnI+3).[4] We have also observed this between the high-sensitivity Abbott and Beckman assay where the prevalence and bias adjusted kappa statistics at the URL were 0.90 (95% CI: 0.83 to 0.95).[6] We note that other assays also have imprecise URLs, and the concordance at the URLs in paired samples is similar, or worse.[6] The differences in the URL, though, do not explain the greater admission rate in the Abbott arm among those with first troponin measure below the low-risk threshold. It appears there were more older males admitted for whatever reasons in the Abbott than the Beckman cohort.

TABLE 4. *Category 1: ACS (investigated for possible ACS) demographics and outcomes*

	Abbott ($N = 1434$)	Beckman ($N = 1619$)	Difference (95% CI)
Age, year	63.1 (17.0)	61.6 (17.3)	1.5 (0.3 to 2.7)
Female sex	641 (44.7%)	784 (48.4%)	3.7% (0.2% to 7.3%)
cTn ≥ URL	378 (26.4%)	483 (29.8%)	3.5% (0.3% to 6.7%)
ED length of stay (h)	4.3 (3.1–5.9)	4.2 (3.2–5.7)	−0.1 h (−0.2 h to 0.0 h)
Admitted	610 (42.5%)	595 (36.8%)	−5.8% (−9.3% to −2.3%)
Myocardial infarction	184 (12.8%)	143 (8.8%)	−4.0% (−6.2% to −1.8%)
Unstable angina	33 (2.3%)	22 (1.4%)	−0.9% (−1.9% to 0.0%)

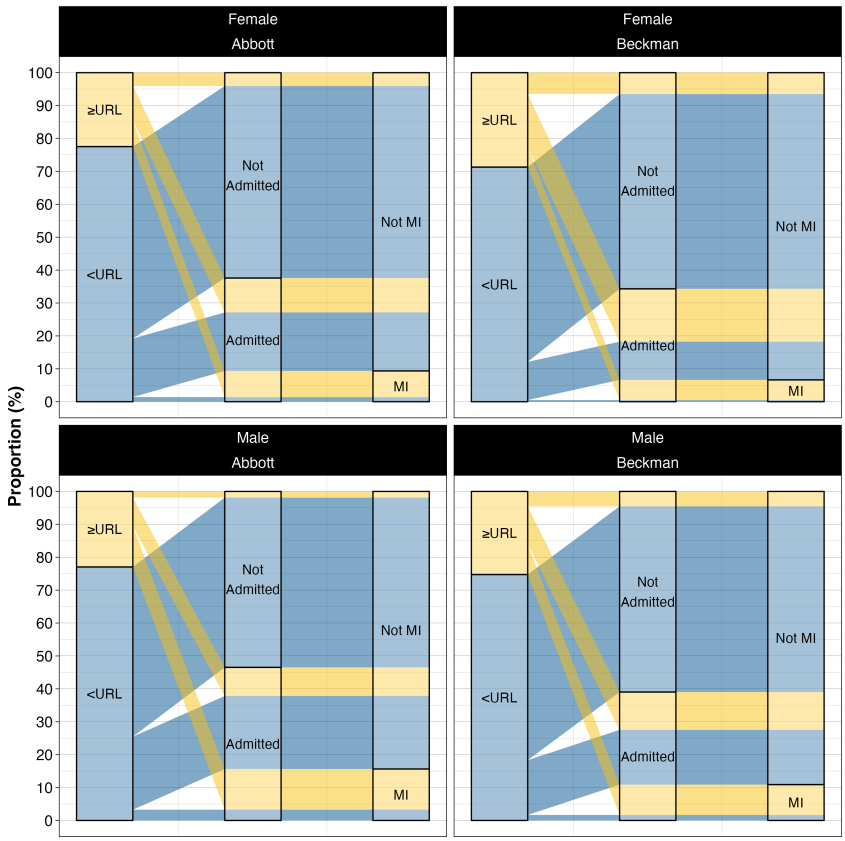

Figure 1. *Flow of patients by assay and sex split by whether they had a troponin result in the ED ≥ the (sex specific) URL of the assay.*

The effect on concentrations of macrotroponin complexes between the Abbott and the Beckman assays is known to differ. Kavsak *et al.* demonstrated that there were macrocomplexes in 7 of 9 patients with much elevated Abbott concentrations compared to Beckman[21] and Lam *et al.* demonstrated that above patients with macrocomplexes were more likely to be above the URL with the Abbott than the Beckman assay.[22] It is possible that this explains some of the differences observed in the disposition and diagnosis in this present study, however the proportion of patients with macrocomplexes affecting concentration results would need to be high and is unknown. It is an area for future research.

We have been unable to find prior research investigating the impact on disposition and diagnosis of a real-life change from one high-sensitivity troponin I assay to another. That said, Canadian hospitals after the change from an Abbott assay to an Ortho-clinical diagnostics hscTnI assay noted imprecision with the Ortho assay after implementation and instigated a series of studies including simultaneously measuring some samples with both assays, a robust methodology that we suggest be adopted for a period of time after implementation of a new assay.[23,24] Discordance at the URL between Abbott and Beckman has been noted previously,[4] and some discordance with 0/2 h pathways between the Abbott and another hs-cTnI and the Roche hs-cTnT assays.[5] Indeed, the T assay is quite different from all I assays in terms of concordance at low-risk and URL thresholds which hospitals and physicians need to be aware of if they are switching between an I and T assay.[6] Our work with two I assays raises the possibility that there may also be patient flow and diagnostic differences when a switch is made between two high-sensitivity I assays. This suggests hospitals should ensure they monitor any changes.

Limitations

A limitation of our study is that it compares a change only between two assays, and it is unknown if similar differences could be encountered if the change had been made to a different I assay. While the overall time period of 18 weeks for the study is short, we cannot eliminate a possible seasonal influence or influence from other unmeasured changes. We note, that there was a rotation of about 50% of the doctors in the study period, meaning these doctors in the Abbott arm were likely a few months more experienced than those in the Beckman arm. This may affect the quality of notes and possibly the rate of troponin ordering. During the Abbott phase Christchurch as at COVID-19 alert level 1 – Prepare (face covering, managed isolation of cases).[25] On 31 August for 1 week Christchurch was at COVID-19 alert level 3 (level 2 + minimal gatherings, stay at home other than essential purposes or work, preference for virtual/non-contact health services) and from 7 September at alert level 2-Reduce (level 1 + gatherings >100 restricted, physical distancing recommended, health services as normal). There were no COVID-19 cases reported in Christchurch during the study period.[26] During level 3 alert in 2020, the MI rate dropped 20% compared to the same period in 2019. With only 1 week of alert 3 in the present study, this difference would not meaningfully impact the results here. Nevertheless, it is possible that with the changes in alert levels physicians changed behaviour. There were 5 cases in the Abbott arm where myocarditis was recorded as a possible concern and 12 in the Beckman arm, but only 3 of those were investigated for possible ACS. These numbers are too small to change the statistics. The number of adult presentations to the ED differed by on 2% between arms (15 872 in the Abbott arm and 16 188 in the Beckman arm; Fig. S1). In this first report, we have been unable to determine the reasons

for all the differences observed. Further research needs to be cognisant of these possible differences and specifically look to identify possible reasons for them.

Conclusion

A change in high-sensitivity cTnI assay impacted the rate of admission to hospital from the ED and the rate of myocardial infarction diagnosed. The reasons for this remain uncertain, nevertheless the different rates highlight that hospitals must monitor these rates when assays are changed to ensure safety is maintained.

Competing interests

JWP has undertaken statistical consultancy for Abbott Diagnostics and other troponin assay manufacturers, but not in relation to the present study. MPT has received research support, consultancy fees, and honoraria from Abbott Diagnostics, Beckman Coulter and other troponin assay manufacturers, but not in relation to the present study. No other disclosures.

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Data availability statement

The data that support the findings of this study are available from NZ Health. Restrictions apply to the availability of these data, which were used under license for this study. Data are available from the author(s) with the permission of NZ Health.

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Supporting information

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

Data S1: Supporting Information.