Impact of New Biomarkers of Myocardial Damage on Trends in Myocardial Infarction Hospital Admission Rates from Population-based Administrative Data

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Use of troponin testing in the diagnosis of myocardial infarction substantially increases the number of cases diagnosed as myocardial infarction among suspected cases in comparison with previous criteria. However, the impact of troponin testing on rates reported in national statistics that use routinely collected hospital morbidity data is uncertain. The authors developed Poisson regression models to estimate the effect of troponin testing on long-term trends in hospital admission rates in Perth, Western Australia, from 1980 to 2004. Troponin tests were used for 10.5% of patients with suspected myocardial infarction in 1996, rising rapidly to more than 90% of patients from 2001 onward. Fitted models that assumed a continuing linear decline estimated that 100% use of troponin testing in cases of suspected myocardial infarction would lead to an apparent increase in hospital admission rates of 42% (95% confidence interval (CI): 28, 56) in men and 21% (95% CI: 4, 41) in women as compared with rates that would be expected if previous linear trends had continued. Smaller effects of 30% (95% CI: 14, 48) in men and −2% (95% CI: −21, 20) in women were found in fitted models that assumed an underlying attenuating trend in the rates. Similarly constructed logistic regression trend models found no significant effect of troponin testing on trends in 28-day case-fatality.

coronary disease; diagnosis; medical record linkage; mortality; myocardial infarction; troponin


Mortality from coronary heart disease in Australia has been declining for nearly 40 years, but the disease remains a major cause of death and disability (1). Monitoring of trends in the incidence and prevalence of coronary heart disease is therefore essential for guiding public health initiatives and planning clinical services.

At the population level, there is no practical method for monitoring changes in the prevalence of coronary heart disease. Trends in acute coronary events (coronary heart disease death or hospitalization for myocardial infarction) are widely accepted as surrogate measures of incidence, but these measures should ideally be based on dedicated disease registers that apply standardized objective diagnostic criteria over time (2–4). However, such registers are costly, and despite the participation of collaborating centers in Newcastle and Perth in the World Health Organization MONICA Project (Monitoring of Trends and Determinants in Cardiovascular Disease; 1984–1993), there are no registers in Australia at present. In their absence, routinely collected mortality data and hospital morbidity data provide acceptable alternative sources of information for population surveillance of trends if they are supported by periodic validation studies of disease coding (3, 5–8), including previous studies using Western Australian data (5, 8).

Apart from consistency of disease coding, the accuracy of trends in hospital admissions for myocardial infarction may
be affected by many factors, including changes in admission policies, changes in clinical terminology, and changes in the sensitivity and specificity of diagnostic tests. Of particular importance in the past 10 years has been the widespread adoption in clinical practice of immunoassays for troponins, which, compared with previously available biomarkers of myocardial infarction, are highly specific and sensitive tests for myocardial necrosis. In 2000, the European Society of Cardiology and the American College of Cardiology recommended in a joint statement that any elevation of troponin in the context of symptoms of acute coronary heart disease should be considered diagnostic of myocardial infarction (9). The statement acknowledged that this would lead to an increase in the diagnosis of mild cases of myocardial infarction and have major implications for epidemiologic studies that had previously relied on traditional diagnostic biomarkers. This prediction has been confirmed in several single-hospital studies, as summarized by Roger et al. (10), and in registry studies, with estimates of increased myocardial infarction in cases of acute coronary heart disease ranging from 23 percent to 195 percent (10, 11). Changes in case mix resulting in reduced case fatality have also been reported, but with less consistency than increases in the number of cases of myocardial infarction (10, 11). However, it is unclear what impact the adoption of troponin testing has had on trends in myocardial infarction in national statistics based on administrative data.

Our objectives in this study were to 1) describe long-term trends in hospital admissions for myocardial infarction in the Perth Statistical Division of Western Australia from 1980 to 2004 and 2) evaluate the hypotheses that the widespread use of troponin testing in Perth hospitals since 1998 was associated with a relative increase in admission rates for myocardial infarction and a relative decline in 28-day case fatality in 1998–2004 as compared with previous periods.

**MATERIALS AND METHODS**

Population-based linked administrative health data were extracted from the Western Australian Data Linkage System (12) for the period 1980–2004 to establish a person-linked file of death records and hospital admissions for cardiovascular disease or diabetes mellitus (equivalent to the *International Classification of Diseases*, Ninth Revision, Clinical Modification (ICD-9-CM) (15) was used from 1980 to 2004. We identified 28-day episodes of myocardial infarction based on hospital admissions with a principal discharge diagnosis of myocardial infarction, including cases of myocardial infarction with onset in the hospital. That is, admissions for the same person were counted as new events only if they occurred more than 28 days from the date of a previous admission for myocardial infarction (4). Fatal cases were those in which death from any cause occurred within 28 days of admission for myocardial infarction, either before or after discharge. The data were used to calculate age-specific rates, age-standardized rates, and 28-day case fatality among persons aged 35–79 years residing in the Perth Statistical Division (population 1.46 million in 2004 (14)). The study was approved by the Human Research Ethics Committee of the University of Western Australia and the Confidentiality of Health Information Committee of the Western Australian Department of Health.

### Table 1. Proportion (%) of patients aged 35–79 years with emergency hospital admissions who received troponin testing in each year relative to all cardiac biomarker tests used in emergency admissions of the same year (troponin, creatine kinase, creatine kinase-MB), Perth Statistical Division, Western Australia, 1980–2004

<table>
<thead>
<tr>
<th>Year(s)</th>
<th>Males &lt;65</th>
<th>Males ≥65</th>
<th>Females &lt;65</th>
<th>Females ≥65</th>
<th>Total</th>
<th>No. of study hospitals*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980–1995</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1996</td>
<td>10.2</td>
<td>12.0</td>
<td>7.9</td>
<td>10.2</td>
<td>10.5</td>
<td>1</td>
</tr>
<tr>
<td>1997</td>
<td>15.3</td>
<td>17.0</td>
<td>10.2</td>
<td>12.5</td>
<td>14.4</td>
<td>5</td>
</tr>
<tr>
<td>1998</td>
<td>44.8</td>
<td>41.4</td>
<td>41.9</td>
<td>39.5</td>
<td>42.1</td>
<td>8</td>
</tr>
<tr>
<td>1999</td>
<td>65.2</td>
<td>60.9</td>
<td>61.8</td>
<td>58.6</td>
<td>61.8</td>
<td>8</td>
</tr>
<tr>
<td>2000</td>
<td>81.2</td>
<td>79.2</td>
<td>79.6</td>
<td>78.8</td>
<td>79.8</td>
<td>8</td>
</tr>
<tr>
<td>2001</td>
<td>92.9</td>
<td>91.8</td>
<td>93.1</td>
<td>92.7</td>
<td>92.6</td>
<td>8</td>
</tr>
<tr>
<td>2002</td>
<td>95.7</td>
<td>94.6</td>
<td>96.5</td>
<td>95.3</td>
<td>95.4</td>
<td>8</td>
</tr>
<tr>
<td>2003</td>
<td>94.8</td>
<td>94.5</td>
<td>97.0</td>
<td>96.0</td>
<td>95.3</td>
<td>8</td>
</tr>
<tr>
<td>2004</td>
<td>95.1</td>
<td>95.6</td>
<td>96.5</td>
<td>96.7</td>
<td>95.8</td>
<td>8</td>
</tr>
</tbody>
</table>

* Calculations were based on cardiac biomarker tests performed in the eight major hospitals (three teaching and five private) in Perth, Australia, in which cardiac admissions are investigated and treated. The numbers indicate how many of these hospitals began using troponin testing, starting from 1996 (initially there was gradual uptake, followed by more rapid uptake beginning in 1999).

Diagnoses in the hospital morbidity data are recorded by trained clinical coders in each hospital using ICD codes and rules which are revised at various intervals. During 1980–2000, there were three such revisions: the ICD-9 (13) was used from 1980 to 1987, the *International Classification of Diseases*, Ninth Revision, Clinical Modification (ICD-9-CM) (15) was used from 1988 to June 30, 1999 (the Australian Version (16) of the ICD-9-CM was used from July 1, 1995, to June 30, 1999), and the *International Classification of Diseases*, Tenth Revision, Australian Modification (ICD-10-AM) (17) has been used since July 1, 1999. The following ICD codes were used to identify hospital admissions for myocardial infarction: 410 (ICD-9 and ICD-9-CM) and I21 and I22 (ICD-10-AM).

### Trends in the use of troponin testing

We determined trends in the use of troponin testing in cases of suspected myocardial infarction through linkage of laboratory test results for cardiac biomarkers (troponin, creatine kinase, and creatine kinase-MB) to corresponding hospital morbidity records of emergency hospital admissions (fatal and nonfatal) for any variant of coronary heart disease or chest pain. Laboratory data were provided by pathology laboratories servicing major public and private hospitals. The level of use of troponin testing in each year (regardless of test result) was calculated as the number of patients aged 35–79 years having troponin tests divided by the total number of patients aged 35–79 years in which any cardiac biomarker test was ordered (table 1). These proportions were used in the Poisson regression trend models.
Statistical methods

Age-specific rates were calculated as the number of hospital admissions for myocardial infarction in each year and each 5-year age group (35–79 years) divided by the Perth Statistical Division population of the same age group and year obtained from the Australian Bureau of Statistics. Age-standardized rates were calculated by the direct method using as the standard population the age distribution of the Australian resident population of June 30, 2001 (the most recent population census) (18). Case fatality was calculated as the number of patients who died of any cause within 28 days of admission for myocardial infarction divided by the number of admissions for myocardial infarction. For plotting, case fatality was age-standardized by the direct method using 5-year age groups and the age distribution of the 28-day myocardial infarction cohort.

Poisson log-linear regression models were used to estimate trends in rates with calendar year and to estimate the effect of the introduction of troponin testing on rates. Troponin tests were introduced in Perth in 1996 but were not adopted by all public and private hospitals admitting cases of myocardial infarction until 1998, which we defined as the beginning of the troponin era. To examine overall changes in the slope of the trend over time, Poisson models were initially fitted separately for the three periods 1980–1988, 1989–1997, and 1998–2004. For each period, a Poisson linear model including 5-year age group (35–39, . . . , 75–79 years) and calendar year of admission (as a continuous variable) was fitted, and the rate ratio describing the relative annual change was calculated from the beta coefficient for calendar year. Similarly, logistic regression was used to estimate the relative annual change in case fatality for these three periods.

To estimate the effect of troponin testing on rates of admission for myocardial infarction, we fitted Poisson regression models for the period 1989–2004. The earlier period was excluded because the trends in rates showed a marked increase in the rate of decline from 1989 onward. These models included variables for age and calendar year of admission and a term reflecting the use of troponin testing. Further models including a term for interaction between age (35–64 years or 65–79 years) and the troponin variable were fitted to evaluate whether the effect of troponin testing on rates differed for the two age groups. Linear and quadratic trend models were fitted. The linear trend model assumes a linear trend in the rate (on a log scale) throughout the period 1989–2004 with a troponin-test effect superimposed, whereas the quadratic trend model assumes a curved (attenuating) trend in the rate (on a log scale) with a troponin-test effect superimposed.

The variable representing the use of troponin testing took two forms. The first was a binary variable indicating whether or not troponin testing was used in a particular year. This variable was 0 for years prior to 1998 (the year of full uptake of troponin testing in Perth) and 1 for the years 1998–2004. The second was a continuous variable in which individual years were given a value from 0 to 1, calculated as the proportion of troponin tests used relative to all cardiac biomarker tests as described above. This variable was 0 for 1989–1995 and then increased from 0.10 in 1996 to 0.96 in 2004 (table 1). There was little variation in troponin test proportions by age and sex, and therefore the overall proportions were used for all age and sex groups.

In a similar manner, we used logistic regression trend models to examine the effect of troponin testing on unadjusted 28-day case fatality. All p values reported are two-sided.

RESULTS

During the 25-year period 1980–2004, there were 36,028 hospital admissions for a 28-day episode of myocardial infarction as the principal discharge diagnosis among persons aged 35–79 years in the Perth Statistical Division. In the 16-year period 1989–2004, there were 22,896 admissions for myocardial infarction (16,252 for men and 6,644 for women), and 2,095 patients died within 28 days of admission (1,246 men and 849 women). Troponin tests were used in 10.5 percent of patients with admissions for coronary heart disease variants or chest pain in 1996, rising rapidly to 42.1 percent in 1998 and 95.8 percent in 2004 (table 1).

Admission rates for myocardial infarction

Age-standardized rates of admission for myocardial infarction showed a declining trend over the period 1980–2004, with declines of 3.0 percent per year in men and 3.3 percent per year in women. Rates declined more rapidly during the period 1989–1997 than in 1980–1988, and there was an upward shift in rates for the period 1998–2004 (the troponin period) (figure 1). In men, myocardial infarction admission rates declined by 5.9 percent per year in 1989–1997 as compared with 2.5 percent per year in 1998–2004, while in women the rates declined by 5.7 percent per year in 1989–1997 as compared with 1.7 percent per year in 1998–2004. Figure 2 shows that the declines in rates of myocardial infarction during the period 1980–2004 were similar in younger and older men and women.

Rate ratios from the Poisson trend models incorporating a troponin variable showed the estimated effect of the introduction of troponin testing on admission rates (table 2). In men, the introduction of troponin testing produced estimated increases of 19 percent (linear model with binary troponin variable) and 14 percent (quadratic model with binary troponin variable) in the rate of admission for myocardial infarction. That is, rates in the troponin period were 19 percent (or 14 percent) higher than would have been expected if troponin testing had not been introduced and the linear (or quadratic) trend had continued during the troponin period. Estimates from trend models with the continuous troponin variable reflect the gradual uptake of troponin testing, and the reported rate ratio shows the estimated increase in the rate that is associated with 100 percent use of troponin testing. The estimated increase was 42 percent from the linear trend model and 30 percent from the quadratic trend model. When examined within age groups, the estimated effect of troponin testing in men was slightly (but not significantly) larger in the age group 35–64 years than in the age group 65–79 years.
For women, a similar pattern of results was seen, with a significantly greater effect of troponin testing in the age group 35–64 years than in the age group 65–79 years; however, the effect sizes were smaller and not statistically significant, except for the age group 35–64 years. Overall, for women the introduction of troponin testing produced estimated increases of 9 percent (binary linear model) and 3 percent (binary quadratic model) in the rate of admission for myocardial infarction. The corresponding effect sizes for women aged 35–64 years were 22 percent and 16 percent, respectively. Estimates of the effect size associated with 100 percent use of troponin testing (from respective continuous...
During the period 1980–2004, age-standardized 28-day case fatality decreased from 18.1 percent to 7.1 percent in women and from 13.5 percent to 4.7 percent in men (figure 3). Table 3 shows the estimated effects of troponin use on 28-day case fatality in men and women based on trend models for the period 1989–2004. The overall estimated effects of troponin testing in emergency cardiac admissions in each of the years 1996–2004. The binary troponin variable estimated the average effect over the period 1998–2004, using a continuous variable reflecting the actual level of use of troponin testing (possibly overestimating the effect of troponin testing), while the quadratic models captured the effect of troponin testing additional to real attenuation of the downward trends (possibly underestimating the effect of troponin testing). Both models were fitted using “troponin testing” as a binary variable or alternatively using a continuous variable reflecting the actual level of use of troponin testing in emergency cardiac admissions in each of the years 1996–2004. The binary troponin variable estimated the average effect over the period 1998–2004, with a modest decline in the first 9 years (1.4 percent decline per year in men, 2.6 percent in women) being followed by an accelerated decline in 1989–1997 (approximately 6 percent per year for both men and women) before slowing to 2.5 percent per year in men and 1.7 percent in women in 1998–2004. The departure in the last period from the linear decline of the previous period of 1989–1997 is consistent with our hypothesis that the uptake of troponin testing would be associated with a relative increase in admission rates for myocardial infarction. This does not exclude the possibility of real attenuation of the rate of decline, although there is no evidence of slowing of the long-term decline in mortality from ischemic heart disease in Australia to support this (19).

To estimate the effect of troponin testing on rates of myocardial infarction, we fitted both linear and quadratic Poisson regression trend models. The former assumed that recent linear trends would have continued were it not for troponin testing (possibly overestimating the effect of troponin testing), while the quadratic models captured the effect of troponin testing additional to real attenuation of the downward trends (possibly underestimating the effect of troponin testing). Both models were fitted using “troponin period (1998–2004)” as a binary variable or alternatively using a continuous variable reflecting the actual level of use of troponin testing in emergency cardiac admissions in each of the years 1996–2004. The binary troponin variable estimated the average effect over the period 1998–2004, with a modest decline in the first 9 years (1.4 percent decline per year in men, 2.6 percent in women) being followed by an accelerated decline in 1989–1997 (approximately 6 percent per year for both men and women) before slowing to 2.5 percent per year in men and 1.7 percent in women in 1998–2004. The departure in the last period from the linear decline of the previous period of 1989–1997 is consistent with our hypothesis that the uptake of troponin testing would be associated with a relative increase in admission rates for myocardial infarction. This does not exclude the possibility of real attenuation of the rate of decline, although there is no evidence of slowing of the long-term decline in mortality from ischemic heart disease in Australia to support this (19).

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whereas the continuous variable estimated the expected effect if troponin testing were used in all cases. Estimates from the continuous linear model suggest that in men admission rates for myocardial infarction would be approximately 40 percent higher with complete use of troponin testing, whereas the continuous quadratic model was associated with a 30 percent increase in rates. The effects were not modified by age.

FIGURE 3. Age-standardized 28-day case fatality (all causes) following myocardial infarction during 1980–2004 among patients aged 35–79 years residing in the Perth Statistical Division, Western Australia. OR(slope) is the odds ratio for annual change in case fatality from a logistic regression model with 5-year age group (35–39, 40–44, ... 75–79 years) and calendar year (as a continuous variable from 1980 to 2004), and the 95% confidence interval (CI) is shown in parentheses. Case fatality was calculated for 28-day events from linked administrative health data.

TABLE 3. Estimated effects of use of troponin testing (logistic regression trend models) on 28-day case fatality (any cause) following hospital admission for myocardial infarction during the period 1989–2004 among patients aged 35–79 years residing in the Perth Statistical Division, Western Australia*

<table>
<thead>
<tr>
<th>Sex</th>
<th>No. of deaths</th>
<th>Troponin testing model</th>
<th>Age group (years)</th>
<th>Linear models</th>
<th>Quadratic models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>OR ± 95% CI</td>
<td>OR ± 95% CI</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>p value §</td>
<td>p value §</td>
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<tr>
<td>Male</td>
<td>1,246</td>
<td>Binary</td>
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<td>1.11 0.87, 1.42</td>
<td>1.09 0.83, 1.42</td>
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<tr>
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<td></td>
<td></td>
<td>65–79</td>
<td>1.17 0.90, 1.51</td>
<td>1.14 0.87, 1.51</td>
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<tr>
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<td></td>
<td></td>
<td>35–64</td>
<td>0.96 0.69, 1.34</td>
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<tr>
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<td></td>
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<td>All</td>
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<td></td>
<td>65–79</td>
<td>1.21 0.82, 1.79</td>
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<tr>
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<td>35–64</td>
<td>0.99 0.62, 1.59</td>
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<td>849</td>
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<tr>
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<td>65–79</td>
<td>1.04 0.77, 1.42</td>
<td>1.07 0.76, 1.49</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>1.16 0.70, 1.92</td>
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<td>All</td>
<td>0.93 0.58, 1.48</td>
<td>0.90 0.44, 1.84</td>
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<td></td>
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<td></td>
<td>65–79</td>
<td>0.92 0.57, 1.48</td>
<td>0.89 0.44, 1.83</td>
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<td></td>
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<td>35–64</td>
<td>0.99 0.51, 1.93</td>
<td>0.96 0.41, 2.27</td>
</tr>
</tbody>
</table>

* Results were based on 28-day events from linked administrative health data.
† The binary model indicates whether troponin testing was used (1 for 1998–2004) or not used (0 for 1989–1997). The continuous model used the proportion of patients who received troponin testing in a given year out of all patients who had biomarker tests (troponin, creatine kinase, and creatine kinase-MB tests for men and women combined) in the same year. Values used were those shown in table 1, expressed as fractions from 0 to 1. Period was a continuous variable ranging from 1 (1989) to 16 (2004).
‡ OR, odds ratio; CI, confidence interval.
§ The p value for all ages was obtained from a two-sided Wald chi-squared test for OR = 1; the p value for age group (two-sided Wald chi-squared test) tested whether the ORs were equal for the two age groups.
In women, the estimated effects of troponin testing were smaller, and there was a significant interaction with age. In the continuous linear model, rates were estimated to be nearly 40 percent higher (95 percent confidence interval: 15, 64) in women under age 65 years as compared with 14 percent higher (95 percent confidence interval: −3, 33) in women aged 65–79 years. In the quadratic models, there was no significant effect of troponin testing except for the age group 35–64 years, which showed a 16 percent increase (binary model). These findings, which contrast with those of previous studies, are unexpected, especially in older women, since troponin testing should identify more cases of myocardial infarction among patients in whom the diagnosis of myocardial infarction is often difficult (11, 20). Our study nevertheless suggests that troponin testing is associated with a substantial apparent increase in admission rates for myocardial infarction, at least among men.

The relative increase in rates of myocardial infarction during the troponin period in our study was substantially smaller than the increases observed in two major population-based registry studies in Finland (11) and the United States (10), in which the introduction of troponin testing increased the numbers of cases diagnosed as myocardial infarction by 83 percent and 74 percent, respectively. The major difference between these studies and ours was that the present study, based on administrative data, related relative changes in trends in myocardial infarction rates to levels of overall use of troponin testing, whereas the other studies compared troponin testing in diagnostic algorithms in place of traditional cardiac biomarkers. However, the US study (10) also found that the increase in cases with dismissal diagnoses of myocardial infarction (ICD-9 code 410) when meeting troponin-based criteria was substantially smaller (42 percent vs. 74 percent). The authors attributed this discrepancy to the failure of clinicians to fully adopt the recommended criteria for the diagnosis of myocardial infarction incorporating troponin results, perhaps because they were reluctant to label as myocardial infarction all additional cases diagnosed from troponin testing (10). Anecdotal evidence suggests that this was also the case in Perth, particularly in later years, in which there was progressive lowering of diagnostic thresholds for troponin testing. If this is the case generally, a lag of several years may occur before the new troponin-based criteria for myocardial infarction are fully expressed in administrative data nationally.

Our second hypothesis, that the decline in 28-day case fatality would accelerate due to an increase in the number of less severe cases of myocardial infarction diagnosed because of troponin testing, was not confirmed. In women, case fatality was further reduced by an apparent 7–10 percent, but it increased by 11–16 percent in men. However, in neither instance were the changes statistically significant. It is possible that our initial assumption that troponin testing would lead to the diagnosis of less severe cases of myocardial infarction was incorrect, as several studies have shown that cases of non-ST-elevation myocardial infarction with low troponin levels, which account for the majority of cases reclassified as myocardial infarction, are often associated with poor outcomes (10, 11).

In interpreting these inconclusive results, readers should recognize that by 1998, 28-day case fatality for myocardial infarction in Perth had already fallen by more than 50 percent in men and women since 1980. Hence, the numbers of deaths in each year were relatively low, as reflected by the wider confidence intervals in logistic regression models. This improvement coincided with major and rapid changes in proven medical treatment of myocardial infarction, particularly during the period 1984–1993, in Perth and in the majority of the populations included in the MONICA Project (21–23). The benefits of these advances in treatment may have been largely realized by the time troponin testing was introduced.

**Limitations of the study**

During the period of introduction of troponin testing, there was also progressive lowering of diagnostic troponin thresholds for myocardial infarction as the precision of measurement improved. This may have led to the diagnosis of more cases of myocardial infarction in later years, which could have affected our results, but this could not be confirmed statistically.

The accuracy of long-term trends in rates of hospital admission for myocardial infarction based on administrative data may be affected by factors other than changes in diagnostic tests. During the study period, there were three major changes and one minor revision in the version of the ICD used in Western Australian hospitals, as noted above in Materials and Methods. These transitions did not produce any obvious discontinuities in the coding of myocardial infarction, but they did affect the coding of unstable angina pectoris from 1996 onward, with reciprocal declines in cases coded as “other angina.” It is unlikely that this has affected the coding of myocardial infarction.

The clinical terminology for acute coronary heart disease has changed during the last decade. New insights into the pathophysiology of myocardial infarction and unstable angina pectoris, together with common approaches to treatment, have led to the use of the broad clinical term “acute coronary syndromes” (24). The extent to which these changes in clinical nomenclature have influenced the coding of myocardial infarction in hospitals is uncertain. Finally, publicity advising persons with sudden chest pain to seek immediate hospital care has undoubtedly increased the numbers of people visiting emergency departments for chest pain, possibly increasing admissions of persons with less severe cases of myocardial infarction in many jurisdictions (25). Nevertheless, previous studies in which the coding of myocardial infarction in administrative data was validated against myocardial infarction registers suggested that administrative data reflect the trends in myocardial infarction reasonably well (5, 7, 8, 26).

The present study and the US study (10) suggest that the validity of trends based on administrative data is now in doubt and will remain so until the new diagnostic criteria for myocardial infarction are fully accepted by clinicians and there is stability in diagnostic thresholds for troponin testing. Given the importance of monitoring rates of myocardial infarction for both public health and health-service
purposes, we suggest that there is a need for further studies to validate the coding of myocardial infarction in administrative data in different jurisdictions and to develop correction factors for adjusting rates of myocardial infarction and other variants of coronary heart disease. Ideally, such studies should be based on a standardized international protocol incorporating the new diagnostic criteria (27, 28).

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REFERENCES


