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This Issue in the Journal

The most deprived Auckland City Hospital patients (2005–2009) are 10 years younger and have a 50% increased mortality following discharge from a cardiac or vascular admission when compared to the least deprived patients

Chris Ellis, Andie Pryce, Garth MacLeod, Greg Gamble

We looked at the 19,500 patients discharged from Auckland City Hospital from July 2005 to December 2009, who were admitted with a cardiac (heart) or vascular (artery, vein) cause. Socioeconomic deprivation (SED) was assessed for patients, and those most deprived (poorer) were 10 years younger at admission than those least deprived (richer). After discharge, the most deprived patients had a 50% increased mortality compared to the least deprived, after adjustment for age and gender. Despite our current efforts to minimise health disparities, further effort is needed to improve on health inequalities in New Zealand.

The use of troponin in general practice

Sally Aldous, Peter Gent, Graham McGeoch, Denise Nicholson

General Practitioners (GP) are able to measure a blood test called cardiac troponin (cTn) in order to help triage patients with symptoms suspicious of a heart attack. This study showed that 8.3% of patients tested by their GP had abnormal cTn levels, most were admitted to hospital and approximately half of these abnormal tests were due to a heart attack. Those with abnormal results were at higher risk of adverse events within the following 6 months than those without (death rates 8.5% versus 1.1%, heart attack rates 2.2% versus 1.2% and heart failure rates 3.1% versus 1.0%). Those with normal cTn levels were low risk and can be managed in the community although the GP may feel admission in some is still necessary. It can take up to 10 hours before the cTn becomes abnormal and therefore repeat testing in patients presenting soon after symptom onset is recommended. Only 12.1% had repeat testing in this study.

Availability of troponin testing for cardiac patients in New Zealand 2002 to 2011: implications for patient care

Mohammad Latif, Chris Ellis, Alexei Chataline, Greg Gamble, Cam Kyle, Harvey White

The modern diagnosis of a heart attack includes a blood test to detect troponin: a heart protein released when the heart is damaged by a blocked artery. The 2 types of troponins released (troponin T and I) can be assessed by various laboratory machines (analysers), produced by various companies. We reviewed troponin tests available at New Zealand hospitals which admitted heart attack patients from 2002 to 2011, and found in 2010–2011 that there were 9 different troponin analysers in 43 hospitals provided by 5 companies. Hence test thresholds and units vary, even for the same test, which can confuse the diagnosis of a heart attack, especially if a patient is transferred
across Health Boards. We consider that this situation is sub-optimal. We suggest that a coordinated national approach is needed with the development of new biochemical tests, such as troponins, which may result in better use of resources and better patient care.

**Mortality by ethnic group to 2006: is extending census-mortality linkage robust?**
Lavinia Tan, Tony A Blakely

Mortality rates continued to fall for all ethnic groups up to 2006. Gaps in death rates for all diseases for Māori compared to European/Other over this time were probably stable in relative or percentage terms. But gaps in cardiovascular disease death rates for Māori compared to European/Other probably decreased—which is good news. Linkage of mortality data to census data up to 5 years after the last census seems viable, but we suspect increasing underestimation of Pacific and Asian mortality rates with increasing time between the census and death due to migration out of New Zealand. Mortality data for 2006–11 will soon be linked to the 2006 census, allowing a more recent update.

**Improving healthcare through the use of co-design**
Hilary Boyd, Stephen McKernon, Bernie Mullin, Andrew Old

Co-design is a way of actively involving patients in the design of services by focussing on understanding and improving patient experiences. Through its Patient Co-design of Breast Service Project, Waitemata District Health Board worked with patients and staff to improve the breast journey and, on a small scale, trial a methodology that had, at the time, not been widely acknowledged or used in New Zealand in the health sector. Using patient journey mapping, experience-based surveys and co-design workshops, we identified four key issue for patients: timely/accessible information, compassionate communication, navigation and coordination, and a pleasant, easy navigable physical environment. Improvements made included a patient information folder, patient leaflets, a patient held record and patient journey guide.

**Doctors and the nurse endoscopist issue in New Zealand**
Mohammad I Khan, Robert Khan, Wanda Owen

Training and recruitment of Nurse Endoscopists (NEs) is currently actively debated in medical circles. The aim of this survey was to obtain the views of doctors regarding the role of NEs in New Zealand. Fifty percent of the 84 respondents worked in tertiary hospitals. Only 30% had a positive attitude towards the introduction of NEs in NZ. The majority (62%) believed that doctors would deliver better quality of endoscopy services than NEs. Only 37% thought that the introduction of NEs will reduce the cost of services. Forty one percent thought it was inappropriate for the NEs to be enrolled in the Bowel Cancer Screening Programme and only 6 doctors (18%) thought that NEs should be allowed to perform therapeutic endoscopic procedures. In conclusion only a minority of doctors had a positive attitude towards the role of NEs. The
majority considered doctors to deliver ‘higher’ quality of service and only a minority thought that the introduction of NEs will lower the cost of services.

Establishment of the New Zealand Drivers Study
John Langley, Dorothy Begg, Rebecca Brookland, Shanthi Ameratunga, Anna McDowell, John Broughton

Despite a significant improvement since graduated licensing was introduced, traffic-related injury remains the leading cause of death and hospitalisation among young New Zealanders. The New Zealand Drivers Study (NZDS) was established to provide information which would lead to an improvement in this situation. We successfully established a study group of 3992 newly licensed car drivers (including 825 Māori for separate analyses) including substantial differences sociodemographic, behavioural, and driving experiences. So far the response rates to interviews at the restricted and full licence stages have been very high at 87% and 93%, respectively. The NZDS is well placed to make a significant contribution to our knowledge of young driver road safety behaviour. This process has already commenced.

Christchurch earthquakes: how did former refugees cope?
Mohamud Osman, Andrew Hornblow, Sandy Macleod, Pat Coope

Seventy-two former refugees from five ethnic groups completed a questionnaire regarding the impact of the Christchurch earthquakes, and how they had coped. Despite high levels of anxiety and concern, greater among older and married participants, three-quarters of participants reported that they had coped well; spirituality and religious practice being an important support. Most (72%) reported that they had not experienced a traumatic event or natural disaster before. Less than 20% received support from mainstream agencies. More engagement from local services is needed to strengthen cooperation between refugee and local communities.

Dabigatran: rational dose individualisation and monitoring guidance is needed ((viewpoint article))
Stephen B Duffull, Daniel F B Wright, Hesham S Al-Sallami, Paul J Zufferey, James M Faed

Dabigatran is the first oral anticoagulant to be introduced in New Zealand without prescribing restrictions for over 50 years. Not surprisingly, the drug has created a great deal of interest amongst health care providers as well as the general public and media. There seems to be a general feeling that warfarin, with its requisite dose adjustments and blood monitoring, is an outdated drug and should be shelved in favour of this novel agent. The assumption is that the newer drug must be better and safer as well as easier to use. Much of the literature associated with dabigatran encourages this view, stressing that dabigatran is a ‘game changer’ with the advantage that the same dose can be used most patients and no need for blood monitoring. In this paper we question whether dabigatran can really live up to these expectations. We suggest that the safe and effective prescribing of dabigatran, like all anticoagulants
used in therapeutic doses, will most likely require dose individualisation and selective blood monitoring. This requirement should not be viewed as a failure for dabigatran. Rather, the individualisation of dabigatran dosages should indicate that the needs of patients are being met.
Beyond equity of access to equity of outcome

Andrew W Hamer, Andrew J Kerr

Equity of access to cardiac services in New Zealand is essential if we are to improve outcomes. The National Cardiac Surgery Network was established in 2009 to lead the implementation of the recommendations of the Cardiac Surgery Services Development Working Group Report.¹

A key issue of concern was the geographic inequity in access to cardiac surgery in New Zealand. District Health Boards (DHBs) with high cardiovascular mortality tended to have lower levels of cardiac surgery. The increased funding for cardiac surgery was therefore distributed across all DHBs with a target of all moving to a rate of at least 6.5/10,000 (SDR adjusted) cardiac operations. This aimed to improve the geographic equity of delivery of cardiac surgery. The Central Region required a 28% increase, Canterbury a 25% increase and the Northern Region a 15% increase. Good progress towards these goals continues.

The lack of access was most notable in the Midland Region where a 68% increase in cardiac surgery was required to achieve equity. This remarkable increase has been achieved.² As access to cardiac surgery improved low referral rates from some DHBs made it evident that equity of access to broader cardiac services varied significantly between DHBs. The developing regional cardiac clinical networks identified the need to target “the inequalities in access to cardiac services” as a key objective.³

In May of 2011 every regional cardiac surgical and cardiology clinical director attended a national meeting, along with the Chair of the Cardiac Society and the Medical Director of the Heart Foundation. The Chairman of DHB CEOs, the Directors of Nursing representative, and directors of the National Health Board and the National Health IT Board were also in attendance, along with other key ministry stakeholders. The meeting debated the best way to “network the networks”.

The New Zealand Cardiac Network was formed. At the end of the meeting the Director General and the Minister of Health joined the meeting to be presented with the proposed structure and goals of the network. The principle goal is “equity of access to high quality cardiac services for all New Zealanders.”

It was agreed that the first national initiative should be the development of registries for acute coronary syndrome, cardiac surgery and interventional cardiology to “develop a body of evidence to support evidence based approaches to improving the quality of care and the equity of access to care”. When combined with linkage of these registries to New Zealand’s national outcomes and pharmaceuticals datasets there is an exciting opportunity to support continuous quality improvement in cardiac care. In collaboration with the National Health IT Board, a business case for funding of the registries was developed. It is extremely pleasing to have the national funding for these registries now approved.
The article by Ellis and colleagues, in this issue of the Journal, clearly demonstrates the need for such outcome-linked continuous quality improvement registries. Their retrospective analysis of all patients presenting with a cardiac or vascular condition, found that the most deprived Auckland City Hospital patients are 10 years younger and have a 50% increased age-adjusted mortality at median 2.4 years after discharge from hospital.

They propose possible reasons for this difference in three main categories; differences in presentation, different treatment during inpatient admission, and differences in follow-up, lifestyle changes or adherence to medication. Whilst there is convincing data for variation in care both pre-hospital, in-hospital and post-discharge, the picture is incomplete and much further study is needed. A key aim of the cardiac registries initiative is to better understand this variation in care across the continuum of primary and secondary care and use the combination of registry and linked national data to drive improvement in evidence-based care.

In a related development, to support post-discharge care, the national datasets have been used by the Northern Regional Cardiac Network to develop key performance indicators for secondary prevention medicine adherence. On request of the leadership group of the New Zealand Cardiac Network, the Northern network has expanded this continuous reporting to the whole country, and in a collaboration between the Northern Cardiac Network and the School of Population Health at the University of Auckland and the Health, Quality and Safety Commission—this work has now been developed into a national medication adherence report as the first phase in development of a National Vascular Atlas of Healthcare Variation. This is currently in the evaluation phase. These reports will facilitate design and evaluation of quality improvement initiatives to improve cardiac medication adherence.

In the post-discharge phase medication adherence is just one of the goals of cardiac rehabilitation programmes. Lifestyle change after a cardiovascular event is harder to monitor than medication adherence but is very important, as much of the socioeconomic disparity in the prevalence of cardiovascular disease relate to lifestyle factors, including higher rates of tobacco use and obesity.

It is essential that cardiac rehabilitation programmes are made relevant and accessible to poorer New Zealanders and to those from different ethnic backgrounds. To support this we need better processes for tracking cardiac rehabilitation attendance. Lifestyle change also needs to be supported by public health programmes.

In New Zealand it is planned to increase the tax on cigarettes by 10% a year for the next 4 years to support the goal of a Smokefree New Zealand by 2025. However, should consumption not fall as predicted, a 20% yearly tax increase or a one-off 40% increase may be warranted. There is also potential to influence New Zealanders’ dietary choices in a way that goes beyond simple educational campaigns. Incentives for both the food industry and consumers is a potentially powerful tool to improve diets and subsequent cardiovascular outcomes for poorer New Zealanders.

With the establishment of the national cardiac registries and National Vascular Atlas the type of valuable data provided by Ellis et al will become routinely and regularly available. Most importantly, regular reporting of national data will facilitate continuous quality improvement by allowing rapid identification of variation in
cardiovascular care and supporting evaluation of the effectiveness of any changes made to the delivery of this care.

Pilot programmes can be performed in different regions and outcomes compared with national trends. The potential for accelerating improvements in patient care for the entire country is exciting. The integration of data from the national cardiac registries with existing national outcomes data is expected to then move us beyond equity of access to equity of outcome for all New Zealanders.

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The use of troponin testing in primary care

Stewart Mann, Lynn McBain

The triage and diagnosis of patients with acute or recent chest pain is a common worldwide challenge that is met in various healthcare settings. A majority of patients with this complaint will not have cardiac pain, some will have identifiable non-cardiac causes and a considerable number end up with a diagnosis of non-specific chest or chest wall pain. A few will have an acute coronary syndrome (ACS) and the consequences of missing such a patient can be severe. However, the pathophysiology of an acute coronary event may mean that even the best professionals equipped with the most sophisticated tests will not always be able to dismiss this diagnosis with complete accuracy.

Much relies on the usual pillars of history and examination which can take into account the background risk of coronary disease, nature of the pain, other accompanying symptoms and signs. An electrocardiogram (ECG) can help define particular ACS syndromes but many patients with ACS will have a normal ECG (or non-specific abnormalities) at initial presentation. Recent studies (ironically evaluating biomarkers) do show how identification of a low risk group can be made with confidence even at this point.

Over recent decades we have come to rely increasingly on biochemical markers in the blood released by damaged myocardium to help define the condition. Initially, such biomarkers were not particularly specific and could be released from skeletal muscle or liver but the increasing use of cardiac troponins has made a step change in both sensitivity and specificity. Their high performance here has tended to bring about a clinical dependence on them as final arbiters of appropriate triage decisions.

Two important lessons have emerged here which apply to all laboratory and other tests: (1) widespread use of even a highly specific test can mislead when used in a population with low risks of the condition in question and (2) increasing sensitivity leads to a trade-off in lower specificity and decisions made as a result can result in adverse consequences for both patient and health service.

Over the last 15 years or so, the measurement of cardiac troponin levels in the blood has become the dominant arbiter of whether there has been damage to cardiac muscle cells. These proteins are highly specific to myocardium and generally have very low circulating levels. Indeed, until recently, these ‘normal’ levels and even small abnormal elevations were below the limits of analytical sensitivity. Thus, at the higher thresholds used, a single raised troponin test would indicate a high probability of a cardiac problem and, in the right context, a likely ACS. Traps in this attractively simple interpretation do exist; a sample taken too early in the course may yield a ‘false negative’ result and there is an ever-growing list of other conditions likely to produce a ‘false positive result’.

To try to help navigate this diagnostic swamp, the European and American Cardiac bodies convened panels (on which New Zealand has been directly represented) to
produce a consensus definition of myocardial infarction with reports being promulgated in 2000² and 2007³. The definitions do depend heavily on biomarkers, especially troponin, and have set a high bar for diagnostic companies to produce accurate assays at low levels of troponin, a standard that several are now capable of achieving. A feature of the definitions that does not appear to have been widely adopted despite being specified on both occasions has been the requirement to demonstrate a ‘typical rise and/or fall’ in the biomarker.

Those assessing patients for a possible ACS face particular timing pressures. In Primary Care, provision for obtaining the first test result can be challenging, let alone awaiting a second test result. Physicians in a hospital Emergency Department are constrained by targets of patient discharge within 6 hours or less which again do not fit well with an ideal troponin testing protocol.

There are of course situations where decisions could or should be made without recourse to a biomarker level; acute ST elevation on ECG is an emergency requiring urgent revascularisation and any patient with a clinically probable ACS appropriate for hospital admission should proceed there directly by ambulance without awaiting a biochemical result. Given the false negative trap, perhaps the best use of troponin testing in General Practice is for the patient who had chest pain possibly representing an ACS some hours or even a few days ago. Practitioners should also guard against the false positive trap by minimising the use of the test in those with a low likelihood of an ACS.

Aldous et al report in this issue⁴ on an audit of troponin testing in Primary Care and conclude that management appears to have been appropriate, although without detailed clinical information about the duration of chest pain prior to testing, confirmation of this is lacking. It should also be noted that the survey was performed with a Troponin I assay which would not have been fully compliant with international guidelines⁵. Nevertheless, the paper is a useful snapshot of recent usage of this test in that environment. Some patients had serial troponin measurements although the rate was low. Clinical pre-test probabilities were not recorded. Of those with positive tests referred to hospital only around half ended with an ACS diagnosis but the prognostic significance of a raised troponin level was confirmed by the presence of other conditions, not all cardiac.

One recent development has been the introduction of troponin T and I tests with higher degrees of sensitivity⁶,⁷, so much so that levels can be registered in a reasonable proportion of the ‘normal’ population. With high-sensitivity tests, an arbitrary cut-off point has to be used, defined by the international consensus³ as the 99th percentile of the local ‘normal’ population. By definition then, 1% of ‘normal’ people will have an abnormal result and there are now many other non-ACS conditions where raised levels will be found. This gives greater weight also to the need for the use of serial testing to improve specificity.

The International consensus panel did not specify the degree of a necessary rise or fall in troponin level but various schemes have been proposed. An algorithm we have developed after local research⁸,⁹ has been implemented successfully locally and is given below, with units referring to a high sensitivity assay for troponin T. Another major current issue is that the adoption of the newer sensitive tests (and with them— for Troponin T—a 1000-fold shift in expression of units from ng/mL or mcg/L to
ng/L) is not universal across New Zealand resulting in a potential source of clinical error and confusion.

Perhaps one advantage of the adoption of tests with higher sensitivity is that clinicians will have to apply a little more qualitative thoughtfulness to referral decisions that can no longer be predicated on a single test result.

Figure 1. Algorithm to aid diagnosis of myocardial infarction using high sensitivity troponin T in patients with a clinical presentation suggestive of an acute coronary syndrome

Adapted with the author’s permission from White and see http://journal.nzma.org.nz/journal/125-1357/5245/content.pdf

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The most deprived Auckland City Hospital patients (2005–2009) are 10 years younger and have a 50% increased mortality following discharge from a cardiac or vascular admission when compared to the least deprived patients

Chris Ellis, Andie Pryce, Garth MacLeod, Greg Gamble

Abstract

Aim To examine how socioeconomic deprivation affects medium to long-term patient outcomes following hospital discharge for a cardiac or vascular admission.

Methods We performed a retrospective analysis of all patients aged ≥15 years of age discharged from Auckland City Hospital between 1/7/2005 and 31/12/2009 with a cardiac or vascular diagnostic-related group (DRG) using prospectively collected data from their Auckland District Health Board (ADHB) discharge, including their ‘deprivation index’, a small area marker of socioeconomic deprivation graded as 1 (the least deprived) to 10 (the most deprived). We then matched these data with the ADHB admissions patient data (for subsequent readmissions) and with the National death registry.

Results In these 4.5 years, 252,974 patients, resident in the ADHB region, were discharged from the ADHB, of whom 19,545 patients had presented with a cardiac or vascular DRG. Of these, 3,609 (18%) patients [mean age 66 (SD18) years] with a deprivation index of 1 or 2 were classified as ‘least deprived’, with 3812 (20%) patients [mean age 57 (SD19) years] with a deprivation index of 9 or 10 being classified as ‘most deprived’. The most deprived patients were, on average, 10 years younger (P<0.0001). 344 [1.8% (95%CI 1.6–1.9)] patients died in hospital and 2970 [15.2% (95%CI 14.8–15.6)] died within a mean follow-up of 2.5 (SD1.4) years. Compared with those least deprived (NZDep Index 2006 1-2) the age and gender adjusted risk of death in the most deprived (NZDep Index 2006 9-10) at median 2.4 years after discharge, was increased by 50% [OR 1.5 (95%CI 1.3–1.7) P<0.0001].

Conclusions Following discharge after a cardiac or vascular related admission and after adjusting for imbalance in age and gender, and also in models with adjustment for age, gender and ethnicity, socioeconomic deprivation was associated with an increased chance of death and hospitalisation following discharge. Despite programmes to minimise health disparities, comprehensive strategies to improve on this health inequality are still needed within the New Zealand health care environment.

Patients admitted to a public hospital should have the same access to investigations and treatment regardless of their socioeconomic status and thus anticipate similar outcomes. Equity is a stated aim of the New Zealand Medical Association. However, for nearly all causes of death in New Zealand large socioeconomic mortality gradients have been reported. Inequities have been observed in clinical management of acute coronary syndrome patients in hospitals with and without invasive facilities.
The long-term intervention with pravastatin (LIPID) trial showed that in both Australia and New Zealand increased mortality was associated with decreased income, a marker for socioeconomic status in patients. These findings may have been exacerbated by poor health delivery structures with sparse clinical (doctor and nursing) input into management.

Heart failure hospitalisations and death also increase with increasing deprivation in New Zealand. In New Zealand, Canada, England and the United States of America, analyses of national statistics showed worsening health care indicators in the most deprived compared with the least deprived quintile of socioeconomic status.

We examined how medium to long-term patient outcomes may be affected by social deprivation following hospital discharge. It has been previously stated that New Zealand needs to consider the totality of information to manage its health system effectively. However to date no descriptive data have been published on patient outcome following a cardiac or vascular discharge from a New Zealand hospital. Further, a descriptive audit of post discharge mortality in relation to admission deprivation level is lacking.

We aimed to describe the 30 day and 1 year mortality of any cardiac or vascular discharge in adult patients (age ≥15 years). We also planned to compare the mortality rates of these patients, by deprivation index (after adjustment for age and gender).

Methods

Study design and subjects—The study population comprised all patients resident within the ADHB region aged 15 years or more discharged from an Auckland District Health Board (ADHB) hospital between 1 July 2005 and 31 December 2009 with a cardiac or vascular diagnosis related group (DRG) (Appendix 1). Each of these patients was matched for subsequent events to 1 June 2010. The study was judged by expedited review by the North Health Ethics committee to be an audit and therefore approved without further review. The definition of an admission is that the patient had been in hospital at least three hours after being seen by a doctor or the patient had a procedure. If two or more admissions were present within the audit period the first admission was counted as the index admission.

Each patient hospital discharge is coded and categorised into a DRG which classifies admissions according to the resources consumed. DRGs are assigned on the basis of the International Classification of Diseases (ICD) diagnoses, procedures, age, gender, discharge status, and the presence of complications and/or comorbidities.

Within the discipline of cardiology there are 67 DRGs which, for the purposes of aggregating numbers, can be amalgamated further into: acute coronary syndrome (ACS)/circulatory management, congestive heart failure (CHF), electrophysiology (EP), general cardiology, cardiac surgery and peripheral vascular disease (PVD) management.

During the audit period each unique patient contributed an index (i.e. first) admission and the date of a second (and number of subsequent) readmission(s) to any ADHB hospital. Patients with an index admission were matched against the national death register and the date of death and the cause of death integrated.

The domicile code for each patient was obtained from the hospital information system and was linked to the New Zealand Deprivation 2006 (NZDep2006) Index. Socioeconomic status was then reported as decile of deprivation from 1 (least deprived) to 10 (most deprived). The NZDep2006 is a small area index of deprivation that provides a score for each mesh block in New Zealand based on nine variables (material and social domains of deprivation) from the 2006 Census. The NZ Deprivation Index is more strongly correlated with cardiovascular disease and diabetes than the NZSEI or income.

In official statistics “prioritised ethnicity” is assigned as Māori if one of the three possible self-identified ethnicity responses was Māori. This represents the total Māori ethnic group. For those not
allocated as Māori, the person is assigned as Pacific Island ethnic group if one of the self-identified ethnic groups was Pacific Island ethnic group, then Asian if an Asian ethnic group is recorded. Residual patients are then classified as European or ‘other’ ethnicities. Since the Indian ethnic group comprised a significant proportion of the ‘Asian’ ethnic groups these data have been presented separately from the ‘Asian’ category in this paper.

**Statistics**—Data were summarised as rates and 95% confidence intervals. Logistic regression was used to make comparisons after adjustment for age, gender and ethnicity and the results are presented as odds ratios and 95% confidence intervals. Cox proportional hazards models (with adjustment for age, gender and ethnicity) were used to compare survival time amongst the groups of interest. Results were presented as hazard ratios (with 95% confidence intervals) and the proportionality assumption was tested. Tests of linear trend were performed using orthogonal contrasts for normally distributed tests and the Cochran-Armitage test for ordinal data. All analyses and data manipulations were performed using SAS statistical software (SAS Institute Inc, v 9.2). Patient years were calculated for each patient as the time from index admission to the end of follow-up or death. Events per patient year were calculated using OpenEpi (www.openepi.com) All tests were two-tailed and P<0.05 was considered statistically significant.

**Results**

These data show that when compared to the least deprived patients, the most deprived patients are 10 years younger and have a 50% age and gender-adjusted increased mortality following discharge from a cardiac or vascular admission to Auckland City Hospital in 2005–2009.

**Unadjusted data**—From 1 July 2005 to 31 December 2009 there were 19,545 cardiac patient discharges from patients resident in the Auckland District Health Board (ADHB) region. Most (50%) were related to acute coronary syndrome or circulatory management, 30% were classified as ‘general cardiology’ with small proportions (<10% each) categorised as CHF, EP, cardiac surgery or PVD management (Table 1). Overall, the numbers of cardiac discharges were similarly spread across the NZDep Index ranging from 18 to 23% (P=0.87, Table 1). However, those patients discharged following cardiac surgery showed increasing proportions of discharges from more deprived patients (P=0.037). In contrast the number of patients discharged following EP management showed increasing proportions from less deprived patients (P=0.04, Table 1).

All-cause mortality following a hospital discharge over a median 2.4 years follow-up was 6.2 deaths per 100 patient years follow-up (Table 1). Unadjusted mortality was higher (6.9 deaths/100 patient years) in those attributed to the least deprived category in comparison with the most deprived category (5.7 deaths/100 patient years) (P<0.05, Table 1). However, the most deprived patients tended to be 10 years younger than the least deprived (57 years vs 66 years P<0.0001), and were more likely to be male (55% v 50%, P<0.0043, Table 2).

The average age at discharge was 61 years (SD 19) (Table 2). All-cause unadjusted mortality was highest for those following a CHF discharge (28 deaths/100 patient years) and lowest following ACS and circulatory management discharge (4.1 deaths/100 patient years) (Table 1).
### Table 1. DRGs by NZDep Index and Deaths at median 2.4 years follow-up (unadjusted data)

<table>
<thead>
<tr>
<th>NZDep Index</th>
<th>“ACS and Circulatory Management”</th>
<th>CHF</th>
<th>EP</th>
<th>General Cardiology</th>
<th>Cardiac surgery</th>
<th>PVD</th>
<th>N</th>
<th>All DRGs</th>
<th>Deaths/100 patient years</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–2 (least deprived)</td>
<td>1660 (17%)</td>
<td>225 (18%)</td>
<td>128 (25%)</td>
<td>1222 (21%)</td>
<td>96 (14%)</td>
<td>277 (18%)</td>
<td>3608 (18%)</td>
<td>6.9 (6.3–7.4)</td>
<td></td>
</tr>
<tr>
<td>3–4</td>
<td>2042 (21%)</td>
<td>220 (18%)</td>
<td>113 (22%)</td>
<td>1213 (21%)</td>
<td>113 (16%)</td>
<td>325 (21%)</td>
<td>4026 (21%)</td>
<td>6.1 (5.6–6.6)</td>
<td></td>
</tr>
<tr>
<td>5–6</td>
<td>2252 (23%)</td>
<td>292 (24%)</td>
<td>124 (24%)</td>
<td>1395 (24%)</td>
<td>151 (22%)</td>
<td>350 (23%)</td>
<td>4564 (23%)</td>
<td>6.3 (5.8–6.7)</td>
<td></td>
</tr>
<tr>
<td>7–8</td>
<td>1803 (18%)</td>
<td>241 (20%)</td>
<td>83 (16%)</td>
<td>975 (17%)</td>
<td>141 (20%)</td>
<td>293 (19%)</td>
<td>3536 (18%)</td>
<td>5.9 (5.4–6.4)</td>
<td></td>
</tr>
<tr>
<td>9–10 (most deprived)</td>
<td>2004 (21%)</td>
<td>254 (21%)</td>
<td>72 (14%)</td>
<td>1025 (18%)</td>
<td>189 (27%)</td>
<td>268 (18%)</td>
<td>3812 (20%)</td>
<td>5.7 (5.3–6.2)</td>
<td></td>
</tr>
<tr>
<td>Overall (%)</td>
<td>9761 (50%)</td>
<td>1232 (6.3%)</td>
<td>520 (2.7%)</td>
<td>5830 (30%)</td>
<td>690 (3.5%)</td>
<td>1513 (7.7%)</td>
<td>19545</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P linear trend</td>
<td>0.75</td>
<td>0.28</td>
<td>0.040 (-)</td>
<td>0.28</td>
<td>0.037 (+)</td>
<td>0.62</td>
<td>0.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths/100 patient yrs</td>
<td>4.1</td>
<td>28</td>
<td>7</td>
<td>5.7</td>
<td>6.5</td>
<td>8.6</td>
<td>6.2</td>
<td>(95%CI)</td>
<td></td>
</tr>
<tr>
<td>(95%CI)</td>
<td>(3.8–4.3)</td>
<td>(26–31)</td>
<td>(5.7–8.6)</td>
<td>(5.3–6.1)</td>
<td>(5.3–7.8)</td>
<td>(7.7–9.6)</td>
<td>(6.0–6.4)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Overall circulatory system Diagnosis Related Group (n=67) discharges categorised into six sub-groups (see Appendix 1) from 1 July 2005 to 31 December 2009 in Auckland District Health Board Residents aged 15 years and over. [(-)]indicates trend for decreasing number with increasing deprivation [(+)]indicates trend for increasing number with increasing deprivation.]

ACS: Acute coronary syndrome; CHF: Congestive heart failure; EP: Electrophysiology; PVD: Peripheral vascular disease; NZDep: New Zealand Deprivation [Index].
Table 2 Patient characteristics by New Zealand Deprivation Index (*unadjusted data*)

<table>
<thead>
<tr>
<th>Variables</th>
<th>1–2 Least deprived</th>
<th>3–4</th>
<th>5–6</th>
<th>7–8</th>
<th>9–10 Most deprived</th>
<th>Overall</th>
<th>P (trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>3609 (18%)</td>
<td>4026 (21%)</td>
<td>4564 (23%)</td>
<td>3536 (18%)</td>
<td>3812 (20%)</td>
<td>19545</td>
<td>&lt;0.0001 (-)</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>66 (18)</td>
<td>62 (19)</td>
<td>61 (19)</td>
<td>61 (18)</td>
<td>57 (19)</td>
<td>61 (19)</td>
<td>&lt;0.0001 (-)</td>
</tr>
<tr>
<td>% Male</td>
<td>1794 (50%)</td>
<td>2179 (54%)</td>
<td>2446 (54%)</td>
<td>1847 (52%)</td>
<td>2076 (55%)</td>
<td>10342 (53%)</td>
<td>0.0043 (+)</td>
</tr>
<tr>
<td>LOS, mean (SD)</td>
<td>2.9 (4.8)</td>
<td>2.7 (4.4)</td>
<td>3.1 (5.3)</td>
<td>3.2 (5.2)</td>
<td>3.2 (6.1)</td>
<td>5.0 (5.1)</td>
<td>&lt;0.0001 (+)</td>
</tr>
<tr>
<td>No. Diagnoses at discharge median (IQR)</td>
<td>3 (2-6)</td>
<td>3 (2-6)</td>
<td>3 (2-6)</td>
<td>4 (2-6)</td>
<td>4 (2-6)</td>
<td>4 (2-6)</td>
<td>0.0001 (+)</td>
</tr>
<tr>
<td>Prioritised ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>186 (5.2%)</td>
<td>286 (7.1%)</td>
<td>369 (8.1%)</td>
<td>284 (8.0%)</td>
<td>275 (7.2%)</td>
<td>1400 (7.2%)</td>
<td>0.87</td>
</tr>
<tr>
<td>European</td>
<td>3073 (85%)</td>
<td>2899 (72%)</td>
<td>2970 (65%)</td>
<td>2024 (57%)</td>
<td>1674 (44%)</td>
<td>12640 (65%)</td>
<td>0.037 (-)</td>
</tr>
<tr>
<td>Indian</td>
<td>83 (2.3%)</td>
<td>241 (6.0%)</td>
<td>386 (8.5%)</td>
<td>287 (8.1%)</td>
<td>250 (6.6%)</td>
<td>1247 (6.4%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Māori</td>
<td>58 (1.6%)</td>
<td>159 (3.9%)</td>
<td>267 (5.9%)</td>
<td>265 (7.5%)</td>
<td>500 (13.1%)</td>
<td>1249 (6.4%)</td>
<td>0.037 (+)</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>57 (1.6%)</td>
<td>252 (6.3%)</td>
<td>346 (7.6%)</td>
<td>470 (13.3%)</td>
<td>900 (23.6%)</td>
<td>2025 (10.4%)</td>
<td>&lt;0.0001 (+)</td>
</tr>
<tr>
<td>Unknown/Other</td>
<td>151 (4.2%)</td>
<td>189 (4.7%)</td>
<td>226 (5.0%)</td>
<td>206 (5.8%)</td>
<td>212 (5.6%)</td>
<td>984 (5.0%)</td>
<td>0.19</td>
</tr>
</tbody>
</table>

Note: Overall circulatory system Diagnosis Related Group (n=67) discharges from 1 July 2005 to 31 December 2009 in Auckland District Health Board Residents aged 15 years and over. 
[(-)indicates trend for decreasing number with increasing deprivation (+)indicates trend for increasing number with increasing deprivation.]

LOS: Length of stay; SD: Standard deviation; IQR: Interquartile range.
The proportion of European ethnicity decreased and the proportions of patients with prioritised Māori or Pacific Island ethnicity increased with increasing deprivation (P<0.0001) (Table 2). The most deprived patients had a significantly longer length of stay, (on average one day more P<0.0001) and they had more diagnoses reported at discharge (P<0.0001) which is a simple surrogate for patients who are more unwell with more comorbidities (Table 2).

Across categories of unadjusted deprivation there were no differences in the in-hospital death rates (overall 1.8%, P=0.23), or death at 30 days (overall 3% P=0.075) or at 1 year (overall 8.1%, P=0.40) after a cardiac discharge (Table 3). However, by a median follow-up of 2.4 years, unadjusted least deprived patients had a greater mortality (but were approximately 10 years older). Increasing deprivation was associated with increased readmission rates (Table 3).

### Table 3. Readmissions to an Auckland District Health Board hospital until 1 June 2010

<table>
<thead>
<tr>
<th>Variables</th>
<th>New Zealand Deprivation Index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1–2 (least depr.)</td>
</tr>
<tr>
<td>Died in Hospital</td>
<td>73 (2.0%)</td>
</tr>
<tr>
<td>Dead: 30 days</td>
<td>124 (3.4%)</td>
</tr>
<tr>
<td>Dead: 365 days</td>
<td>302 (8.4%)</td>
</tr>
<tr>
<td>Dead: 2.4yrs FU</td>
<td>607 (17%)</td>
</tr>
<tr>
<td>Length of FU</td>
<td>2.5 (1.4)</td>
</tr>
<tr>
<td>Readmiss./10 patient years (95% CI)</td>
<td>2.3 (2.2, 2.4)</td>
</tr>
<tr>
<td>Death or Readmis.</td>
<td>1446 (40%)</td>
</tr>
<tr>
<td>At least one Readmiss.</td>
<td>1076 (30%)</td>
</tr>
</tbody>
</table>

**Note:** All-cause death to 1 March 2009 (unadjusted data). Outcome following any cardiac discharge 1 July 2005 to 31 December 2009 in Auckland District Health Board Residents aged 15 years and over.

[(-) indicates trend for decreasing number with increasing deprivation (+) indicates trend for increasing number with increasing deprivation.]
The characteristics of patients discharged following ACS and circulatory management show a similar pattern to the entire cardiac cohort: deprived patients are younger, more likely to be male and to have a larger proportion of patients of Māori or Pacific Island origin (Table 4). They also had increased number of discharge diagnoses (a crude measure of co-morbidities) and longer length of stay (Table 4).

Table 4 “ACS and Circulatory Management” diagnosis-related group discharges 1 July 2005 to 31 December 2009 in ADHB residents aged 15 years and over (unadjusted data).

<table>
<thead>
<tr>
<th>Variables</th>
<th>1–2 (Least depr.)</th>
<th>3–4</th>
<th>5–6</th>
<th>7–8</th>
<th>9–10 (Most depr.)</th>
<th>Overall</th>
<th>P (trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>1660 (17%)</td>
<td>2042 (21%)</td>
<td>2252 (23%)</td>
<td>1803 (18%)</td>
<td>2004 (21%)</td>
<td>9761</td>
<td>&lt;0.0001 (-)</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>61 (18)</td>
<td>58 (17)</td>
<td>57 (18)</td>
<td>57 (17)</td>
<td>54 (17)</td>
<td>58 (17)</td>
<td>0.43</td>
</tr>
<tr>
<td>% Male</td>
<td>885 (53%)</td>
<td>1181 (58%)</td>
<td>1306 (58%)</td>
<td>1012 (56%)</td>
<td>1117 (56%)</td>
<td>5501 (56%)</td>
<td>0.87</td>
</tr>
<tr>
<td>LOS, mean (SD)</td>
<td>2.3 (3.9)</td>
<td>2.1 (3.3)</td>
<td>2.3 (3.6)</td>
<td>2.4 (3.7)</td>
<td>2.3 (3.7)</td>
<td>3.0 (5.2)</td>
<td>&lt;0.0001 (+)</td>
</tr>
<tr>
<td>No. diagnoses at discharge median (IQR)</td>
<td>3 (2-5)</td>
<td>3 (2-5)</td>
<td>3 (2-6)</td>
<td>3 (2-6)</td>
<td>4 (2-6)</td>
<td>3 (2-5)</td>
<td>0.0001 (+)</td>
</tr>
</tbody>
</table>

Prioritised ethnicity

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>1–2 (Least depr.)</th>
<th>3–4</th>
<th>5–6</th>
<th>7–8</th>
<th>9–10 (Most depr.)</th>
<th>Overall</th>
<th>P (trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian</td>
<td>92 (5.5%)</td>
<td>173 (8.5%)</td>
<td>200 (8.9 %)</td>
<td>162 (9.0%)</td>
<td>157 (7.8%)</td>
<td>784 (8.0%)</td>
<td>0.87</td>
</tr>
<tr>
<td>European</td>
<td>1359 (82%)</td>
<td>1371 (67%)</td>
<td>1356 (60%)</td>
<td>963 (53%)</td>
<td>784 (39%)</td>
<td>5833 (60%)</td>
<td>0.037 (-)</td>
</tr>
<tr>
<td>Indian</td>
<td>61 (3.7%)</td>
<td>174 (8.5%)</td>
<td>258 (11.5%)</td>
<td>198 (11%)</td>
<td>171 (8.5%)</td>
<td>862 (8.8%)</td>
<td>0.62</td>
</tr>
<tr>
<td>Māori</td>
<td>29 (1.8%)</td>
<td>88 (4.3%)</td>
<td>145 (6.4%)</td>
<td>131 (7.3%)</td>
<td>272 (13.6%)</td>
<td>665 (6.8%)</td>
<td>0.037 (+)</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>28 (1.7%)</td>
<td>117 (5.7%)</td>
<td>151 (6.7%)</td>
<td>222 (12.3%)</td>
<td>472 (23.6%)</td>
<td>990 (10.1%)</td>
<td>0.037 (+)</td>
</tr>
<tr>
<td>Unknown/Other</td>
<td>91 (5.59%)</td>
<td>119 (5.8%)</td>
<td>142 (6.3%)</td>
<td>127 (7.0%)</td>
<td>148 (7.4%)</td>
<td>627 (6.4%)</td>
<td>0.87</td>
</tr>
</tbody>
</table>

Note: [see Appendix for acute coronary syndrome/invasive management 20 Diagnosis Related Group codes). (-)indicates trend for decreasing number with increasing deprivation (+)indicates trend for increasing number with increasing deprivation.]

LOS: Length of stay; SD: Standard deviation; IQR: Interquartile range.

There are no differences in deaths following ACS and circulatory management admissions across deprivation levels using the unadjusted data however the number of readmissions increases with worsening deprivation (Table 5).
Table 5. Readmissions to an Auckland District Health Board hospital until 1 June 2010. (All-cause death to 1 March 2009 (unadjusted data) for acute coronary syndrome/circulatory diagnoses.)

<table>
<thead>
<tr>
<th>Variables</th>
<th>New Zealand Deprivation Index</th>
<th>1–2 Least Deprived</th>
<th>3–4</th>
<th>5–6</th>
<th>7–8</th>
<th>Most Deprived</th>
<th>Overall</th>
<th>P (trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died in Hospital</td>
<td></td>
<td>23 (1.4%)</td>
<td>36 (1.8%)</td>
<td>32 (1.4%)</td>
<td>25 (1.4%)</td>
<td>28 (1.4%)</td>
<td>144 (1.5%)</td>
<td>0.64</td>
</tr>
<tr>
<td>Dead: 30 days</td>
<td></td>
<td>42 (2.5%)</td>
<td>53 (2.6%)</td>
<td>55 (2.4%)</td>
<td>35 (1.9%)</td>
<td>42 (2.1%)</td>
<td>227 (2.1%)</td>
<td>0.075</td>
</tr>
<tr>
<td>Dead: 365 days</td>
<td></td>
<td>90 (5.4%)</td>
<td>118 (5.8%)</td>
<td>139 (6.2%)</td>
<td>88 (4.9%)</td>
<td>104 (5.2%)</td>
<td>539 (5.5%)</td>
<td>0.39</td>
</tr>
<tr>
<td>Dead: 2.4yrs FU</td>
<td></td>
<td>181 (11%)</td>
<td>209 (10%)</td>
<td>246 (10%)</td>
<td>182 (10%)</td>
<td>204 (10%)</td>
<td>1022 (10%)</td>
<td>0.5</td>
</tr>
<tr>
<td>Length of FU</td>
<td></td>
<td>2.5 (1.4)</td>
<td>2.6 (1.4)</td>
<td>2.5 (1.4)</td>
<td>2.6 (1.4)</td>
<td>2.6 (1.4)</td>
<td>2.6 (1.4)</td>
<td>0.43</td>
</tr>
<tr>
<td>Readmissions/10 patient years</td>
<td></td>
<td>1.8 (1.7–1.9)</td>
<td>2.1 (2.0–2.2)</td>
<td>2.2 (2.1–2.3)</td>
<td>2.2 (2.0–2.3)</td>
<td>2.6 (2.5–2.8)</td>
<td>2.2 (2.1–2.2)</td>
<td>&lt;0.0001(+)</td>
</tr>
</tbody>
</table>

Note: Outcome following an acute coronary syndrome/circulatory management cardiac discharge 1 July 2005 to 31 December 2009 in Auckland District Health Board Residents aged 15 years and over. [(-)] indicates trend for decreasing number with increasing deprivation (+) indicates trend for increasing number with increasing deprivation.

Adjusted data—From the unadjusted data it is seen that the most deprived patients are 10 years (57 v 66 years, P<0.0001) younger and males are more frequently (54% v 50% P<0.0001) admitted in the most deprived group. Therefore to assess overall mortality over time the data needs to be age and gender adjusted to fully assess the impact of deprivation.15

Figure 1 shows the age and gender adjusted risk estimates of death, and the composite death/readmission for each NZDep Index level, compared to the least deprived group. Throughout the follow up, death at 1 year and at 2.4 years of follow-up, and the composite endpoint of death/readmission, were significantly increased (all P<0.0001) in the most deprived NZ Dep Index: category 9-10 compared with the least deprived NZ Dep Index: category 1-2. For these endpoints the trend was a linear increase across the groups.

Figure 1 also shows that short term 30-day mortality has a similar pattern, with mortality increasing with increasing levels of deprivation, however the confidence intervals were wide and the trend failed to reach conventional statistical significance. The same pattern was apparent for an analysis restricted to acute coronary syndrome/circulatory management patient admissions (Figure 2).
Figure 1. Mortality after any cardiac admission (all 67 cardiac Diagnosis Related Groups see Appendix 1) to an Auckland District Health Board hospital by deprivation index. (Age and gender adjusted data.)

Figure 2. Mortality after an acute coronary syndrome/circulatory management admission (20 Diagnosis Related Group categories see Appendix 1) to Auckland City Hospital. (Age and gender adjusted data.)
Cox proportional hazards modelling was performed for time to all-cause death at a median follow up of 2.4 years after a cardiac admission and adjusted for age and gender (Figure 3A) and with additional adjustment for ethnicity (Figure 3B).

**Figure 3A. Age/Gender adjusted** hazard of all cause mortality after a cardiac admission to Auckland City Hospital

![Figure 3A](image1)

**Figure 3B. Age/Gender/Ethnicity adjusted** hazard of all cause mortality after a cardiac admission to Auckland City Hospital

![Figure 3B](image2)
Adjusting for age, gender, ethnicity and length of stay attenuated the result slightly however increasing deprivation remained significantly associated with increased mortality (NZ DepI 1–2 HR=1, NZ DepI 2–3 HR=1.08 (1.0,1.2) P=0.18, NZDep I 4–6 HR=1.14 (1.0, 1.3) P=0.02, NZDepI 6–8 HR=1.15 (1.0,1.3) P=0.02, NZDep I 8–10 HR=1.20 (1.01, 1.08) P<0.0001).

Similar modelling was performed for an ACS and circulatory management admission and adjusting for age, gender (Figure 4A) and additionally for ethnicity (Figure 4B). Overall, and for those with ACS and circulatory management discharges the same pattern was seen, a statistically significant increase in risk of early death with increasing deprivation. Adjustment for ethnicity, in addition to age and gender did not change this observation.

Unadjusted all-cause mortality was higher at a median follow up of 2.4 years (17%) in the least deprived group (NZDep 1–2) compared with the most deprived group (NZDep 9–10) (14%) (P=0.0018, Table 6).

Table 6. All-cause mortality at a median follow-up of 2.4 years by ethnic group in the least deprived and most deprived groups

<table>
<thead>
<tr>
<th>Ethnic Group</th>
<th>NZDep 1–2 (least deprived) 18% of total cardiac admissions</th>
<th>NZDep 9–10 (most deprived) 20% of total cardiac admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>unadjusted</td>
<td>Age/Gender adjusted risk of death</td>
</tr>
<tr>
<td></td>
<td>% Total Cohort</td>
<td>% Dead</td>
</tr>
<tr>
<td>European</td>
<td>85%</td>
<td>555/3073</td>
</tr>
<tr>
<td>Asian</td>
<td>5.20%</td>
<td>20/186</td>
</tr>
<tr>
<td>Indian</td>
<td>2.30%</td>
<td>9/83</td>
</tr>
<tr>
<td>Māori</td>
<td>1.60%</td>
<td>5/58</td>
</tr>
<tr>
<td>Other</td>
<td>4.20%</td>
<td>11/151</td>
</tr>
<tr>
<td>Pacific Is.</td>
<td>1.60%</td>
<td>7/57</td>
</tr>
<tr>
<td>Total</td>
<td>100%</td>
<td>607/3608</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnic Group</th>
<th>NZDep 9–10 (most deprived) 20% of total cardiac admissions</th>
<th>Unadjusted</th>
<th>Age/Gender adjusted risk of death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% Total Cohort</td>
<td>% Dead</td>
<td>AGE (SD)</td>
</tr>
<tr>
<td>European</td>
<td>44%</td>
<td>313/1674</td>
<td>19%</td>
</tr>
<tr>
<td>Asian</td>
<td>7.20%</td>
<td>9/275</td>
<td>3.3%</td>
</tr>
<tr>
<td>Indian</td>
<td>7%</td>
<td>18/250</td>
<td>7.2%</td>
</tr>
<tr>
<td>Māori</td>
<td>13%</td>
<td>69/500</td>
<td>14%</td>
</tr>
<tr>
<td>Other</td>
<td>5.6%</td>
<td>11/213</td>
<td>5.2%</td>
</tr>
<tr>
<td>Pacific Is.</td>
<td>24%</td>
<td>121/900</td>
<td>13%</td>
</tr>
<tr>
<td>Total</td>
<td>100%</td>
<td>541/3812</td>
<td>14%</td>
</tr>
</tbody>
</table>

There were no differences in all-cause age/gender adjusted mortality across ethnic groups for those in the least deprived group however in those classed most deprived, Māori and
Pacific people were at increased risk of age/gender adjusted death compared to Europeans following cardiac discharge whilst Asians were at reduced risk (Table 6).

In comparison to least deprived (NZDep 1-2) Europeans, the most deprived (NZDep 9–10) Europeans, Māori and Pacifica peoples had significantly increased age and gender adjusted all-cause mortality (Figure 5).

Overall, men had a 20% age-adjusted increased risk of all-cause death at a median follow-up of 2.4 years compared with women (HR 1.2 (95%CI 1.14–1.32, P<0.0001) and the risk of all-cause mortality increased with increasing deprivation in both men and women (Figure 6).

When considering the age of the patient (Figure 7), the association with deprivation was most pronounced in those aged <60 years with increasing risk of gender adjusted death with increasing level of deprivation (p=0.0063 for trend across first to last Dep levels).

In patients aged 60 to 75 years the association was present, but less pronounced (p=0.0006 across Dep levels). In those aged ≥75 years there was no association between risk of all-cause death and worsening deprivation (p=0.096 across Dep levels, Figure 7). These data suggest that deprivation is associated with a marked shortening of life in younger age groups.

Figure 4A. Age/Gender adjusted hazard of all cause mortality after an acute coronary syndrome or circulatory management admission to Auckland City Hospital
Figure 4B. Age/Gender/Ethnicity adjusted hazard of all cause mortality after an acute coronary syndrome or circulatory management admission to Auckland City Hospital

Figure 5. Age/gender adjusted risk of all cause death in those most (New Zealand Deprivation Index 9-10) and least deprived (New Zealand Deprivation Index 1–2) by ethnic origin (data are hazard ratio 95% CI). P is for comparison against referent group (least deprived Europeans).
Figure 6. All-cause mortality for men and women by New Zealand Deprivation Index (overall age adjusted) for men compared to women is 1.2 (95% CI: 1.14, 1.32) P<0.0001.

Figure 7 All cause mortality for men and women by New Zealand Deprivation Index (overall gender adjusted) for those aged ≥ 75, 60-65 and < 60 at discharge.
Discussion

About 8% of all discharges from an Auckland District Heath Board hospital are cardiac or vascular, comprising roughly equal proportions of people across the categories of the NZ Deprivation Index. About half of all cardiac or vascular admissions are for “acute coronary syndrome and circulatory management”.

Overall, subsequent mortality is low (6.2 deaths/100 patient years). Patients who were most deprived were 10 years younger than those who were least deprived and more likely to be male. Hence, although fewer deaths are observed in those who are most deprived (5.7 deaths/100 patient years) than in those who are least deprived (6.9 deaths/100 patient years), an age and gender adjustment is clearly needed, and then the overall risk of all-cause mortality over a median 2.4 years follow-up is 50% greater for those most deprived in comparison with the least deprived group.

Previous studies have shown large socioeconomic mortality gradients exist for nearly all causes of deaths in the New Zealand community. The New Zealand census-mortality study examined age and ethnicity standardised mortality for 25-94 years. They compared NZ deprivation index groups 9 and 10 (most deprived) with groups 1 and 2 (least deprived).

All-cause mortality was 2.0 (1.9, 2.2) for men and 2.1 (1.9, 2.3) for women. Cardiovascular disease rate ratios were 2.4 (2.1, 2.8) for men and 2.4 (2.0, 3.0) for women. The same pattern persists in these data, in patients following a cardiac or vascular admission to Auckland City Hospital.

More Māori and Pacific Islands people are likely to be classified as most deprived. After adjustment for this imbalance, a consistent linear increase in risk of early death is still seen with increasing levels of deprivation, suggesting that deprivation results in a worse outcome independent of ethnicity.

When a patient’s ethnicity is considered, in patients identified as least deprived there is no difference in age and gender adjusted all-cause mortality across ethnic groups. However in the most deprived patients, Māori and Pacific Islands people have significantly increased mortality compared to similarly deprived European patients. Asian patients tend to have reduced all-cause mortality compared to Europeans.

This observation suggests an added risk for those of Māori or Pacific Islands ethnicity in those who are most deprived. This may reflect barriers to primary or secondary healthcare services which might prevent or delay acute hospital admissions, and/or failures of primary screening.

Marked ethnic and socioeconomic disparities in the prevalence of cardiovascular disease in New Zealand has been shown in a 2006-2007 cohort. Māori had the highest age-standardised prevalence compared to the rest. Prevalence of cardiovascular disease increased with increasing deprivation. Deprivation is also known to be associated with increased risk of heart failure death or heart failure admission which has been shown to be worse overall for Māori.

There are several possible reasons for the more deprived having a worse outcome. First, there may be differences at presentation. At presentation more deprived patients may have
more (or more severe) comorbid conditions, or may delay their presentation to hospital or may have experienced less effective primary care.

Younger patients presenting to the coronary care unit at Middlemore Hospital have a worse cardiovascular risk profile, and are more likely to be obese, to smoke, to have low HDL and high triglyceride levels. However Malcolm found that in 127,426 primary health organisation (PHO) patients that Māori actually had better access to cardiology inpatient services than non-Māori.

Further, the New Zealand Acute Coronary Syndrome Audit of 2002 showed that in-patient management was a reflection of geographical location and service provision, and not of ethnicity.

Second, there may be different treatment of patients during their inpatient admission as it is possible that treatment within hospital differs by socioeconomic status, although this seems unlikely. Supporting data for the absence of a treatment bias within the hospital might come from the survival curves from these data.

Early deaths are not split by deprivation; it is late deaths that are driving this effect. Hence the comprehensive public practice within a hospital may be unbiased, but after hospital discharge, the ongoing medical effects of socioeconomic deprivation are seen.

Third, there may also be differences with pre-hospital deaths, outpatient follow-up, or patient adherence to appropriate lifestyle or medication, or a range of other unknown differences.

The differences in access to treatment are more likely to be greater between hospitals than within a hospital and may present as a socioeconomic difference when the hospital supports a lower SES demographic. Ellis et al have shown that patients are less likely to receive interventional cardiology if they don’t present to an interventional centre. Non-interventional centres are more likely to be rural and poor.

These data also suggest that deprivation is associated with a marked shortening of life in younger age groups after a cardiac or vascular admission. It is unclear why the effect of deprivation is not seen in the older age groups. Possible reasons include: the younger more deprived members have already died; older patients may live in less deprived suburbs, and since the NZ Deprivation Index is driven by address, some older patients may be misclassified as they may have now a standard income: the pension, the same as with people from ‘more deprived’ people, and therefore may be less able to access extra assistance e.g. private health care which could result in better outcomes with earlier assessment and management of medical problems.

There are a number of potential limitations for this study including the relatively small number of Māori or Pacific Islands people in the lower deprivation categories, a focus on all-cause mortality, reliance on information extracted from the official admissions record (which has no information on the severity of casemix at presentation) and using patients self-reported (but verified) address to model socioeconomic status, as a few individuals may have been misclassified by providing incorrect address information.

Whilst the external validity (generalisability) of these conclusions is unchanged, since these represent an analysis of official statistics for a large number of consecutive admissions at a major tertiary hospital, the conclusion must be tempered by the understanding that the internal validity might be reduced by confounding factors.
Conclusion

Socioeconomic deprivation is associated with earlier age at presentation and a markedly increased chance of death and hospitalisation following discharge from a cardiac or vascular related admission to a public hospital in New Zealand. These data are of considerable concern and highlight the clear need to develop and implement comprehensive strategies to improve on this health inequality within the New Zealand health care environment.

Competing interests: None known.

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References:


Appendix 1. Cardiac and Vascular Australian Refined Diagnosis-Related groups (AR-DRG) v5 Codes. (DRGs are grouped according to system. Circulatory systems are prefixed with ‘F’)

1) **Cardiology-Acute Coronary Syndrome and Circulatory Management**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>F10Z</td>
<td>Percutaneous Coronary Intervention W AMI</td>
</tr>
<tr>
<td>F15Z</td>
<td>Percutaneous Coronary Intervention W/O AMI W Stent Implantation</td>
</tr>
<tr>
<td>F16Z</td>
<td>Percutaneous Coronary Intervention W/O AMI W/O Stent Implantation</td>
</tr>
<tr>
<td>F19Z</td>
<td>Other Trans-Vascular Percutaneous Cardiac Intervention</td>
</tr>
<tr>
<td>F21A</td>
<td>Other Circulatory System O.R. Procedures W Catastrophic CC</td>
</tr>
<tr>
<td>F21B</td>
<td>Other Circulatory System O.R. Procedures W/O Catastrophic CC</td>
</tr>
<tr>
<td>F40Z</td>
<td>Circulatory System Diagnosis W Ventilator Support</td>
</tr>
<tr>
<td>F41A</td>
<td>Circulatory Disorders W AMI W Invasive Cardiac Inves Proc W Cat or Sev CC</td>
</tr>
<tr>
<td>F41B</td>
<td>Circulatory Disorders W AMI W Invasive Cardiac Inves Proc W/O Cat or Sev CC</td>
</tr>
<tr>
<td>F42A</td>
<td>Circulatory Disorders W/O AMI W Invasive Cardiac Inves Proc W Complex DX/Pr</td>
</tr>
<tr>
<td>F42B</td>
<td>Circulatory Disorders W/O AMI W Invasive Cardiac Inves Proc W/O Complex DX/Pr</td>
</tr>
<tr>
<td>F60A</td>
<td>Circulatory Disorders W AMI W/O Invasive Cardiac Inves Proc W Cat or Sev CC</td>
</tr>
<tr>
<td>F60B</td>
<td>Circulatory Disorders W AMI W/O Invasive Cardiac Inves Proc W/O Cat or Sev CC</td>
</tr>
<tr>
<td>F60C</td>
<td>Circulatory Disorders W AMI W/O Invasive Cardiac Inves Proc; Died</td>
</tr>
<tr>
<td>F72A</td>
<td>Unstable Angina W Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F72B</td>
<td>Unstable Angina W/O Catastrophic or Severe CC</td>
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<tr>
<td>F74Z</td>
<td>Chest Pain</td>
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<tr>
<td>F75A</td>
<td>Other Circulatory System Diagnoses W Catastrophic CC</td>
</tr>
<tr>
<td>F75B</td>
<td>Other Circulatory System Diagnoses W Severe CC</td>
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<tr>
<td>F75C</td>
<td>Other Circulatory System Diagnoses W/O Catastrophic or Severe CC</td>
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2) **Cardiology-Heart Failure**

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<tr>
<th>Code</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>F62A</td>
<td>Heart Failure and Shock W Catastrophic CC</td>
</tr>
<tr>
<td>F62B</td>
<td>Heart Failure and Shock W/O Catastrophic CC</td>
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3) **Cardiology-Electrophysiology**
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<th>Description</th>
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<tbody>
<tr>
<td>F01A</td>
<td>Implantation or Replacement of AICD; Total System W Cat or Sev CC</td>
</tr>
<tr>
<td>F01B</td>
<td>Implantation or Replacement of AICD; Total System W/O Cat or Sev CC</td>
</tr>
<tr>
<td>F02Z</td>
<td>AICD Component Implantation/Replacement</td>
</tr>
<tr>
<td>F12Z</td>
<td>Cardiac Pacemaker Implantation</td>
</tr>
<tr>
<td>F17Z</td>
<td>Cardiac Pacemaker Replacement</td>
</tr>
<tr>
<td>F18Z</td>
<td>Cardiac Pacemaker Revision Except Device Replacement</td>
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4) **Cardiology-General**

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<tr>
<td>F61Z</td>
<td>Infective Endocarditis</td>
</tr>
<tr>
<td>F66A</td>
<td>Coronary Atherosclerosis W CC</td>
</tr>
<tr>
<td>F66B</td>
<td>Coronary Atherosclerosis W/O CC</td>
</tr>
<tr>
<td>F67A</td>
<td>Hypertension W CC</td>
</tr>
<tr>
<td>F67B</td>
<td>Hypertension W/O CC</td>
</tr>
<tr>
<td>F68Z</td>
<td>Congenital Heart Disease</td>
</tr>
<tr>
<td>F69A</td>
<td>Valvular Disorders W Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F69B</td>
<td>Valvular Disorders W/O Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F70A</td>
<td>Major Arrhythmia and Cardiac Arrest W Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F70B</td>
<td>Major Arrhythmia and Cardiac Arrest W/O Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F71A</td>
<td>Non-Major Arrhythmia and Conduction Disorders W Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F71B</td>
<td>Non-Major Arrhythmia and Conduction Disorders W/O Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F73A</td>
<td>Syncope and Collapse W Catastrophic or Severe CC</td>
</tr>
<tr>
<td>F73B</td>
<td>Syncope and Collapse W/O Catastrophic or Severe CC</td>
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5) **Cardiac Surgery**

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<th>Code</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>F03Z</td>
<td>Cardiac Valve Proc W CPB Pump W Invasive Cardiac Investigation</td>
</tr>
<tr>
<td>F04A</td>
<td>Cardiac Valve Proc W CPB Pump W/O Invasive Cardiac Inves W Cat CC</td>
</tr>
<tr>
<td>F04B</td>
<td>Cardiac Valve Proc W CPB Pump W/O Invasive Cardiac Inves W/O Cat CC</td>
</tr>
<tr>
<td>F05A</td>
<td>Coronary Bypass W Invasive Cardiac Inves W Catastrophic CC</td>
</tr>
<tr>
<td>F05B</td>
<td>Coronary Bypass W Invasive Cardiac Inves W/O Catastrophic CC</td>
</tr>
<tr>
<td>F06A</td>
<td>Coronary Bypass W/O Invasive Cardiac Inves W Catastrophic or Severe CC</td>
</tr>
</tbody>
</table>
### F06B
Coronary Bypass W/O Invasive Cardiac Inves W/O Catastrophic or Severe CC

### F07A
Other Cardiothoracic/Vascular Procedures W CPB Pump W Catastrophic CC

### F07B
Other Cardiothoracic/Vascular Procedures W CPB Pump W/O Catastrophic CC

### F08A
Major Reconstruct Vascular Procedures W/O CPB Pump W Catastrophic CC

### F08B
Major Reconstruct Vascular Procedures W/O CPB Pump W/O Catastrophic CC

### F09A
Other Cardiothoracic Procedures W/O CPB Pump W Catastrophic CC

### F09B
Other Cardiothoracic Procedures W/O CPB Pump W/O Catastrophic CC

6) **Peripheral Vascular Disease Management**

### F11A
Amputation for Circ System Except Upper Limb and Toe W Catastrophic CC

### F11B
Amputation for Circ System Except Upper Limb and Toe W/O Catastrophic CC

### F13Z
Upper Limb and Toe Amputation for Circulatory System Disorders

### F14A
Vascular Proc Except Major Reconstruction W/O CPB Pump W Cat CC

### F14B
Vascular Proc Except Major Reconstruction W/O CPB Pump W Sev CC

### F14C
Vascular Proc Except Major Reconstruction W/O CPB Pump W/O Cat or Sev CC

### F20Z
Vein Ligation and Stripping

### F63A
Venous Thrombosis W Catastrophic or Severe CC

### F63B
Venous Thrombosis W/O Catastrophic or Severe CC

### F64Z
Skin Ulcers for Circulatory Disorders

### F65A
Peripheral Vascular Disorders W Catastrophic or Severe CC

### F65B
Peripheral Vascular Disorders W/O Catastrophic or Severe CC

W=with; AMI= Acute myocardial infarction; W/O= Without; OR=Operating Room; CC=complications or co morbidities; DX/Pr=Diagnosis/procedure; Sev=Severe; Cat=Catastrophic
The use of troponin in general practice

Sally Aldous, Peter Gent, Graham McGeoch, Denise Nicholson

Abstract

Background General practitioners are able to measure cardiac troponin in order to help triage patients with symptoms suggestive of acute coronary syndrome. The aim of this study was to assess the utilisation of cardiac troponin testing in the community.

Methods An audit of all cardiac troponin testing in an urban community from a single laboratory in 2010 was performed. Data regarding admissions and adverse events over a 6-month period was carried out in all patients.

Results Cardiac troponin was measured during 2662 patient events during 2010. There were 223 patients episodes (8.4%) in which ≥1 troponin result was elevated, 184 (82.5%) were admitted to hospital, 101 (54.9%) were diagnosed as acute coronary syndrome. Of the 2439 with normal troponin results, 344 (14.1%) were admitted, 42 (12.2%) were diagnosed as acute coronary syndrome. Only 12.1% had serial troponin measurements. The 6-month rates of death were 8.5% versus 1.1%, myocardial infarction were 2.2% versus 1.2%, revascularisation were 1.8% versus 0.7%, heart failure were 3.1% versus 1.0% in those with elevated versus normal troponin respectively.

Conclusion The use of troponin in the community appropriately triages patients regarding the need for admission. However, many patients had elevated troponin due to non-coronary causes. The indication for testing only in cases of suspected ACS and the use of serial cTn measurement in early presenters should be emphasised.

International guidelines recommend serial cardiac troponin (cTn) measurement in patients presenting with symptoms suggestive of acute coronary syndrome (ACS). Cardiac troponin is the gold standard biochemical criterion for the diagnosis of acute myocardial infarction (AMI) and investigations and treatments guided by cTn results have been shown to influence outcomes. As such, cTn is not only a diagnostic tool but is also highly effective in risk stratification.

The primary care physician or general practitioner (GP) faces the challenge of identifying patients with ACS and therefore those at risk of adverse cardiac events such as death, AMI and heart failure. It is often the task of the GP to initiate further diagnostic procedures in a timely manner in those with suspected ACS. However, although symptoms such as chest pain are common in general practice, they are due to acute coronary artery disease in only a minority of cases. It is therefore also the role of the GP to protect patients from over-diagnosis and inappropriate treatment, and prevent overcrowding of specialist and emergency services.

Cardiac troponin tests are available in the community and GPs are making increasing use of these tests to help triage such patients.
Concerns with the use of cTn in the community are:

- The delay in referral for admission in those with ACS whilst waiting for cTn results to become available.\(^7,11\)
- Over-interpretation of positive results, especially when borderline, due to a lack of knowledge regarding the many non-coronary conditions that can lead to elevation of cTn.\(^11\)
- Failure to measure serial cTn in patients presenting early after symptom onset which may lead to false negatives.\(^7\)

**Methods**

A retrospective audit was performed of all cTn requests referred to Canterbury Health Laboratories from General Practice in Christchurch, New Zealand, in 2010. Canterbury Health Laboratories is one of three laboratory groups in the region that received cTn requests during this time period. Patient data was collected by means of a regional and then national health events search (which identifies any hospital attendance using an alpha numeric identifier unique to that patient). Admissions to hospitals outside of New Zealand were not sought. Data was collected regarding whether admission occurred after index cTn testing and the subsequent diagnosis according to the discharge notice from the admitting team.

Patients were followed for 6 months for adverse events including death, non-fatal AMI, revascularisation (percutaneous coronary intervention or coronary artery bypass surgery) and admission for heart failure with diagnoses again according to the discharge diagnosis of the admitting team. These were analysed in those above and those below 75 years of age as an arbitrary (but not ideal) marker of comorbidity.

The cTn assay utilised by Canterbury Health Laboratories is Abbott Architect Troponin I (99\(^{th}\) percentile 0.028 mcg/L, 10% coefficient of variation 0.032 mcg/L, limit of detection 0.010 µg/L, decision cut-point as per manufacturer >0.03 µg/L).

**Results**

There were 2662 patient episodes in 2575 patients during 2010 in which ≥1 sample for cTn testing were sent to Canterbury Health Laboratories. The median age was 63 (interquartile range 51 to 74) and 1186 (44.6%) were male. Other patient characteristics were not available. There were 321 (12.1%) patient episodes in which serial samples were taken.

There were 223 patients episodes (8.4%) in which ≥1 cTn results were elevated above the decision threshold, median age 73 (63–83), 131 (58.7%) male. Twenty-one (9.4%) had serial measurements, of which 11 were elevated on the second sample only. 184 (82.5%) were admitted to hospital for further evaluation, median age 71 (62–81), 111 (60.3%) male, median cTn 0.14 (0.06–0.61) µg/L. The discharge diagnoses of those who were admitted are shown in Figure 1.
Figure 1. Discharge diagnosis of patients admitted following elevated troponin in general practice


Those with a diagnosis of myocardial infarction had higher median cTn levels, 0.27 (0.10–1.65) µg/L than those with other diagnoses, 0.08 (0.05–0.22) µg/L. Those not admitted had a median cTn of 0.05 (0.03–0.09) µg/L.

Figure 2. Six month event rates according to troponin result and whether patient was admitted at index testing

(a) Troponin positive/Admitted

(b) Troponin positive/Not admitted
Of the 184 admitted, 5 (2.7%) underwent stress testing, 86 (46.7%) underwent coronary angiography and 49 (26.6%) were revascularised, 4 with coronary artery bypass surgery. Events post discharge are shown in Figure 2. The 6-month event rates of the 39 with elevated cTn who were not admitted, median age 81 (66–88), 19 (48.7%) male, are also shown in Figure 2. The median cTn of those with (any) 6-month event was 0.10 (0.05–0.58) µg/L compared with those without events, 0.12 (0.05–0.41) µg/L.
Figure 3. Discharge diagnosis of patients admitted following normal level troponin in general practice

STEMI – ST elevation myocardial infarction, NSTE ACS – non ST elevation acute coronary syndrome. Other cardiac diseases=peri/myo/endocarditis, heart failure, valvular disease, Arrhythmia=atrial fibrillation, supraventricular tachycardia, complete heart block, Respiratory disease=chronic obstructive pulmonary disease, asthma, pulmonary embolus, pneumonia, influenza, malignancy, Gastrointestinal disease=cholecystitis/cholangitis/pancreatitis, ileo/colitis, appendicitis, peptic ulcer disease.

There were 2439 patients in whom all cTn measurements were below the decision cut-point, median age 62 (50–73), 1056 (43.1%) male. Two hundred and ninety seven (12.2%) of these had serial troponin measurement. Three hundred and forty four (14.1%) were admitted to hospital for further evaluation, median age 65 (54–78), 136 (39.5%) male. The discharge diagnoses of those who were admitted are shown in Figure 3.

Of the 344 admitted, 69 (20.1%) underwent stress testing, 31 (9.0%) underwent coronary angiography and 13 (3.8%) were revascularised, 5 with coronary artery bypass surgery. Events post discharge are shown in Figure 2. The 6 month event rates of the 2095 with normal level cTn who were not admitted, median age 62 (50–73), 917 (43.8%) male, are also shown in Figure 2.

Discussion

This audit investigates the use of cTn testing in an urban community setting over a 1 year period and shows that such a test is highly utilised.
The utility of cTn in the community should be to:

- Influence the decision to refer for admission or
- In those not suitable for admission, to influence the management of such patients.

Previous studies have shown that if suspicion for ACS is low, GPs are more likely to either order cTn and wait for the result or manage medically. If suspicion is intermediate, GPs tend to either refer for admission without cTn results (especially when patients present early after symptom onset) or order cTn and wait for the result. In contrast, if the suspicion is high, patients are referred without cTn results.11

This study shows that the vast majority of patients with elevated cTn were admitted. This appears highly appropriate as those with elevated cTn were much more likely to come to harm in the short term with higher 6 month rates of death (8.5% versus 1.1%), AMI (2.2% versus 1.2%), revascularisation (1.8% versus 0.7%) and heart failure (3.1% versus 1.0%) than those with normal cTn levels. Those with and without events had similar median cTn levels.

Those with elevated cTn who were not admitted had higher event rates than those who were admitted (12.8% versus 7.6% for death, 7.7% versus 1.1% for AMI, 2.6% versus 1.6% for revascularisation and 7.7% versus 2.2% for heart failure). Although not all comorbidities were known, the median age of these patients was 81, suggesting that admission may not have been appropriate and that community treatment was possibly medical/palliative. However, 2.6% of these patients were revascularised within 6 months suggesting that this was not the case in all patients. Figure 2 shows how those ≥75 (a surrogate but not ideal marker for comorbidity) were more at risk of events, as expected.

The median cTn levels in these patients showed only borderline elevations which may also contribute to the decision not to admit, unfortunately we do not have data regarding the level of probability for AMI assigned by the General Practitioner.

It is likely that a significant number of patients would have been referred for admission irrespective of the cTn results, for example the 13 patients with STEMI and the 14.4% of those with negative cTn results who were still admitted. If admission is inevitable, it is unlikely that measurement of cTn in the community is necessary and may have economic implications.11 It can be seen that a negative result for cTn in the community indicates low risk of subsequent adverse cardiac events. As such, it seems reasonable to suggest that current management of these patients in the community is appropriate.

Only approximately 12% of patients had serial testing. Current International Guidelines recommend serial cTn measurement at presentation and again at 8–12h after symptom onset2 or 6–9h post presentation.1 Previous audit data at Christchurch Hospital has shown that the cTn used in this study is reliably negative by 10 hours post symptom onset.

Unfortunately the time from symptom onset to presentation was unknown. Although a negative cTn in the community appears to indicate low risk, serial cTn would still be recommended in those presenting less than 10 hours from symptom onset. It was
shown that of the 21 patients with elevated cTn undergoing serial measurements, only 10 had elevations on the first test.

A previous community audit performed elsewhere in New Zealand showed that 12% of negative tests were performed in patients presenting less than 10 hours after the onset of symptoms. It may be therefore, that most of the early presenters in this study did in fact have serial measurements.

The indication for cTn testing in all patients was unknown as clinical information at the time of the test was not collected. Therefore this did not allow an assessment of the clinical appropriateness of testing. However, the discharge diagnosis of the patients admitted, demonstrated a wide variety of conditions.

Previous audit data in Christchurch Hospital shows that in patients admitted for rule out of ACS, approximately 20–25% have elevated cTn and approximately 90–95% of these have a diagnosis of AMI. In contrast, this audit shows that only 8.3% have elevated cTn in the community and only just over 50% of these had a diagnosis of AMI.

This illustrates the wide variety of conditions that can lead to cTn elevation although those without AMI had lower median cTn levels than those with AMI. It should therefore be emphasised that cTn is to only be measured in patients with suspected ACS. Previous studies have shown that patients with symptoms such as chest pain presenting in the community are most likely to have musculoskeletal conditions and a significant number of patients have psychogenic disorders, respiratory infections and gastrointestinal diseases. Only a minority have ACS, 1 study had an ACS rate of 3.6% compared with our 5.4%. The American Heart Association guidelines have suggested that symptoms suggestive of ACS include acute chest, epigastric, neck, jaw or arm pain or discomfort or pressure without apparent non-cardiac source. Symptoms such as fatigue, shortness of breath, syncope or arrhythmia can in a minority of patients be secondary to AMI but are not symptoms that automatically indicate the need to measure cTn if ACS is otherwise not suspected. While it is accepted that many patients present atypically, this highlights perhaps that cTn testing in the community may not be measured in the appropriate clinical context in a significant proportion of patients.

A previous study showed that only 25–40% of GPs could correctly identify other causes of cTn elevation. Other studies suggest that community cTn testing is indicated to determine the need for acute referral in those with intermediate suspicion of ACS and not those with a high likelihood (who should be referred without cTn testing) and is also not for reassurance of the patient in whom there is low suspicion of ACS.

In conclusion, this study suggests that the use of cTn in the community appropriately triages patients regarding the need for admission. However, the indication for testing only in cases of suspected ACS and the use of serial cTn measurement in early presenters should be emphasised.

A clinical pathway for management of suspected ACS in the community is currently being developed. Each patient is categorised as to whether their likelihood of ACS is high, intermediate, low or unlikely using predefined criteria to help determine the best management. If high risk, immediate referral of patients without prior cTn testing
should occur. Low risk patients should be managed in the community with cTn testing, including serial measurements in patients presenting less than 10 hours after onset of symptoms. If unlikely to be ACS, cTn testing is not advised.

However, there is always a limit to how specific such an algorithm can be and clinical judgment will always be required, especially in cases of greater uncertainty. It is our intent to repeat this audit in 1 year following the instigation of this pathway.

Competing interests: None declared.

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References:
Availability of troponin testing for cardiac patients in New Zealand 2002 to 2011: implications for patient care

Mohammad Latif, Chris Ellis, Alexei Chataline, Greg Gamble, Cam Kyle, Harvey White

Abstract

Aims For patients presenting with an acute coronary syndrome (ACS), troponin T or I levels are crucial for the diagnosis of myocardial infarction (MI). We investigated troponin tests, analyser types and thresholds used in New Zealand (NZ) from 2002 to 2011.

Methods We reviewed troponin tests available at hospitals in NZ which admitted ACS patients and those who had troponin testing in 2002 (n=41), 2007 (n=43) and 2011 (n=43). We also contacted community laboratories and manufacturers.

Results In 2010-11 there were nine different troponin analysers in 43 hospitals provided by five companies. Troponin T assays were used in 58% of the hospitals and 42% used troponin I as their first-line method. Quoted cutpoints have become more aligned since 2002 and 2007, but are still different from laboratory cutpoints using point of care methods.

Conclusions There are differences in troponin tests available across NZ. Test thresholds and units vary, even for the same test, and available diagnostic information cannot always be used to identify a troponin rise and fall. Care is needed when comparing results from different methods and when point of care instruments are used. A coordinated national approach to the development of new biochemical tests, such as troponins, may result in better use of resources and better patient care.

The American College of Cardiology (ACC) and the European Society of Cardiology (ESC) have published a joint consensus statement redefining acute myocardial infarction (MI).¹ The cornerstone of the new definition of MI is the elevation of cardiac troponin (T or I) concentration in the appropriate clinical context and, due to the range of other causes of troponin elevation, evidence of a rise and/or fall.²,³ Troponins are not just useful for the diagnosis of MI but also have been shown to be prognostically important.⁴–⁶

Over the past 2 decades, immunoassays have been developed and refined for cardiac troponins T and I which are increasingly sensitive and specific for cardiac muscle injury. A large number (10–20) of manufacturers have produced assays for troponin I. Only Roche Diagnostics has produced and marketed a troponin T assay, although it has licensed the assay for one point of care analyser marketed by another company (Radiometer AQT90).

Different manufacturers use their own antibodies raised against different antigen epitopes on the cardiac troponin I protein, and also use different calibrator and control materials.⁷ While there has been progress in standardising troponin I assays using an international reference preparation, this has not resolved the inherent difficulties in comparing troponin results between different methods for an individual
It is very difficult, and may be dangerous, to try to diagnose a rise and/or fall in troponin when different troponin assays and platforms are used.

Use of different methods and different assay platforms could lead to diagnostic confusion and inconsistencies between centres and/or regions, affect treatment, and could confound health statistics. Hence we conducted an audit looking at the various troponin assays (including point of care), providers, analyser types and the local cutpoints used across New Zealand, in hospitals and private providers and reviewed how this has changed over time (2002, 2007 and 2011).

Methods

We reviewed the troponin tests, analysers and cutpoints available at hospital laboratories across New Zealand which admitted patients with a probable acute coronary syndrome (ACS) in 2002, 2007 and 2011. These hospitals also participated in the comprehensive Cardiac Society of New Zealand Audit in 2002 and 2007 which represented all hospitals admitting ACS patients in New Zealand.

The development of the New Zealand Acute Coronary Syndrome (NZACS) Audit Group and the methodology for the national audits has been published previously. A few smaller hospitals, not routinely admitting ACS patients, had troponin testing facilities and these were also included in this survey.

Data were collected from the hospital laboratory staff and/or clinicians by e-mail or telephone in order to establish which assays and analysers were being used, as well as the local cutpoints for a positive test. Community laboratory staff were contacted to provide similar data.

We consulted representatives from a number of major New Zealand troponin testing providers to aid in data collection, to enquire about manufacturer cutpoints, and identify other sites where point of care analysers measuring troponin were being used for patient management.

Results

Distribution of assays used in hospital laboratories—In 2011, the 43 hospitals across New Zealand which admit possible ACS patients used 10 different troponin analysers provided by five companies. In comparison there were 11 analysers in use in 2007 and 8 analysers in 2002 (Table 1). Additionally, the Siemens Centaur assay is in use by a private laboratory (Labtests) in 2011, making a total of 11 different analysers in current use.

Troponin analysers from 2002, 2007 and 2011 are shown in Table 2 by geographical distribution. In 2011, 58% of the hospitals (25/43) use troponin T assays and 42% (18/43) use troponin I assays as their first-line method. Troponin T assays also slightly predominated in 2002 (68%) and 2007 (70%).

In 2011 Ortho and Bayer are no longer present in New Zealand, and there are two new commercial providers: Siemens and Beckman Coulter. Siemens now provide a reformulated troponin test on the Centaur (previously Bayer) platform, in addition to the Dimension and Vista analysers. Radiometer provides a bench top analyser, the AQT90, which is standardised against the Roche Troponin T assays and uses an identical antibody configuration.

Of hospital laboratories measuring troponin I, the Abbott Architect assay is the most widely used in 2011. Abbott provided 78% (14/18) of the assays measuring troponin I as the first-line test, mostly using the Architect assay. Other troponin I assays are also provided in regional North Island locations; Siemens Vista assay at North Shore and
Waitakere, and Beckman Coulter DXI and Access platforms in Bay of Plenty and Whakatane.

Table 1. Major providers, number and different types of analysis methods in use at hospitals admitting acute coronary syndrome patients across New Zealand in 2002, 2007 and 2011

<table>
<thead>
<tr>
<th>Assay</th>
<th>Company</th>
<th>Types of instruments</th>
<th>Instruments in use 2002 (41 hospitals)</th>
<th>Instruments in use 2007 (43 hospitals)</th>
<th>Instruments in use 2011 (43 hospitals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin T</td>
<td>ROCHE</td>
<td>H232/ CARDIAC READER/ RAPID T</td>
<td>10</td>
<td>13(4)*</td>
<td>3(3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MODULAR E170</td>
<td>2</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ELECSYS 1010</td>
<td>6</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ELECSYS 2010/2020</td>
<td>8</td>
<td>6(1)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>COBAS 601</td>
<td>0</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>RADIOMETER</td>
<td>AQT 90</td>
<td>0</td>
<td>0</td>
<td>2(2)</td>
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<tr>
<td>Troponin I</td>
<td>ABBOTT</td>
<td>I- STAT</td>
<td>0</td>
<td>2(2)</td>
<td>7(5)</td>
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<tr>
<td></td>
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<td>AsSYM</td>
<td>11</td>
<td>2</td>
<td>0</td>
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<tr>
<td></td>
<td></td>
<td>ARCHITECT i2000</td>
<td>0</td>
<td>4</td>
<td>7</td>
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<tr>
<td></td>
<td>BAYER</td>
<td>ADVIA CENTAUR</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>DADE BEHRING/SIEMENS**</td>
<td>DIMENSION XPAND</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>VISTA</td>
<td>0</td>
<td>0</td>
<td>2</td>
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<tr>
<td></td>
<td></td>
<td>STRATUS II</td>
<td>1</td>
<td>0</td>
<td>0</td>
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<tr>
<td></td>
<td>BECKMAN-COULTER</td>
<td>ACCESS</td>
<td>0</td>
<td>1</td>
<td>(1)</td>
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<td></td>
<td></td>
<td>DXI</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>JOHNSON &amp; JOHNSON</td>
<td>ORTHO-CLINICAL VITROS</td>
<td>1</td>
<td>0</td>
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</tr>
</tbody>
</table>

* Brackets () denote number of times instrument is also used as backup instrument

Note:
- If a hospital has two instruments of a type of analyser (e.g. two Architect i-2000 or two E170 machines) this is still only recorded once.
- Numbers of analysers in the community are considered separately.
- In some cases a laboratory may have access to a backup analyser but is it not in routine use (e.g. no available cartridges for point of care instruments), and is therefore not counted.

** One other method (Siemens Centaur assay) is being used in the Auckland community by Labtests.

Table 2. Analysers and cutpoints across New Zealand in 2002, 2007 and 2011×
<table>
<thead>
<tr>
<th>Hospital</th>
<th>Trop-</th>
<th>2002</th>
<th>Lab.</th>
<th>Tn/</th>
<th>2007</th>
<th>Lab.</th>
<th>Tn/</th>
<th>Jan 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>onin Man*</td>
<td>Analysers</td>
<td>cut-point</td>
<td>Man*</td>
<td>Analysers</td>
<td>cut-point</td>
<td>Man*</td>
<td></td>
</tr>
<tr>
<td>Intervention Centres (2011)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Waitemata (North Shore)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland City Hospital</td>
<td>I-B</td>
<td>Centaur</td>
<td>I-B</td>
<td>0.04</td>
<td>I-S</td>
<td>Vista</td>
<td>0.00</td>
<td>40ng/L</td>
</tr>
<tr>
<td>Greenlane Hospital</td>
<td>I-R</td>
<td>Elecsys 2010</td>
<td></td>
<td></td>
<td>N/A*</td>
<td>N/A*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mercy, Auckland (P)</td>
<td>I-R</td>
<td>AxSym 2.0</td>
<td>I-A</td>
<td>0.04</td>
<td>I-A</td>
<td>Architect i2000</td>
<td>40ng/L</td>
<td></td>
</tr>
<tr>
<td>Ascot, Auckland (P)</td>
<td>I-D</td>
<td>Stratus II^</td>
<td>1.0</td>
<td>0.03</td>
<td>T-R</td>
<td>Cardiac reader*</td>
<td>0.03ug/L</td>
<td></td>
</tr>
<tr>
<td>Waikato</td>
<td>T-R</td>
<td>Modular E170</td>
<td>T-R</td>
<td>0.03</td>
<td>T-R</td>
<td>Cardiac reader*</td>
<td>0.03ug/L</td>
<td></td>
</tr>
<tr>
<td>Wellington</td>
<td>T-R</td>
<td>Elecsys 2010</td>
<td>T-R</td>
<td>0.03</td>
<td>T-R</td>
<td>Cobas 601</td>
<td>14ng/L</td>
<td></td>
</tr>
<tr>
<td>Wakefield, Wellington (P)</td>
<td>T-R</td>
<td>Rapid T slide</td>
<td>+/-</td>
<td>0.03</td>
<td>T-R</td>
<td>Cobas 601</td>
<td>14ng/L</td>
<td></td>
</tr>
<tr>
<td>Nelson</td>
<td>I-A</td>
<td>AxSym 1.0</td>
<td>T-R</td>
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<td>T-R</td>
<td>Cobas 601</td>
<td>14ng/L</td>
<td></td>
</tr>
<tr>
<td>Christchurch</td>
<td>T-R</td>
<td>Elecsys 2010</td>
<td></td>
<td>0.03</td>
<td>T-R</td>
<td>Architect i2000</td>
<td>14ng/L</td>
<td></td>
</tr>
<tr>
<td>Dunedin</td>
<td>I-A</td>
<td>AxSym 2.0</td>
<td>I-A</td>
<td>0.03</td>
<td>T-R</td>
<td>Cobas 601</td>
<td>14ng/L</td>
<td></td>
</tr>
</tbody>
</table>

| Non-intervention Centres (2011) |             |             |          |        |             |          |        |          |
| Auckland/Northland, North Island|             |             |          |        |             |          |        |          |
| Kaitaia*                       | T-R         | Rapid T slide| +/-     | 0.10   | T-R        | Cobas 601  | 13ng/L |
| Dargaville*                    | T-R         | Cardiac reader| <0.05  | 0.03   | T-R        | Cobas 601  | 14ng/L |
| Rawene*                        | T-R         | Rapid T slide| +/-     | 0.03   | T-R        | Cobas 601  | 13ng/L |
| Kawakawa / BOI                 | T-R         | T-R slide    | +/-     | 0.03   | T-R        | Cobas 601  | 14ng/L |
| Whangarei                      | I-A         | AxSym 1.0   | I-A      | 0.04   | T-R        | Cobas 601  | 14ng/L |
| Waitakere*                     | I-A         | AxSym N/A*  | I-B      | 0.04   | I-S        | Vista    | 40ng/L |

| Waikato/Central, North Island  |             |             |          |        |             |          |        |          |
| Tauranga                      | I-B         | Centaur     | 0.2      | 0.04   | I-B        | DXI (Access)| 0.06ug/L |
| Whakatane                     | T-R         | Elecsys 2010| 0.03     | 0.04   | I-B        | DXI       | 0.06ug/L |
| Thames                        | T-A         | Modular E170| 0.03     | 0.03   | T-R        | Elsco 2010| <14ng/L |
| Tokoroa                       | T-R         | Cardiac reader| 0.03  | 0.03   | T-R        | Radiometer| <0.03ug/L |
| Te Kuiti                      | T-R         | Cardiac reader| 0.03  | 0.03   | T-R        | Radiometer| <0.03ug/L |
| Taumarumui                    | T-R         | Elecsys 1010| 0.03     | 0.03   | T-R        | Elsco 2010| <14ng/L |
| Taupo                         | I-A         | AxSym 0.5   | T-R      | 0.03   | T-R        | Cobas 601  | 14ng/L |
| Rotorua                       | I-A         | AxSym 0.5   | I-A      | 0.03   | T-R        | Architect i2000| 0.04ug/L |
| Hawke's Bay/Hastings          | I-A         | AxSym 0.5   | I-A      | 0.03   | T-R        | Architect i2000| 0.04ug/L |
| Wairoa Hospital *             | T-R         | Elecsys 1010| 0.03     | 0.03   | T-R        | Architect i2000| 0.04ug/L |
| Gisborne (T-lab)              | I-A         | AxSym 1.0   | I-A      | 0.15   | I-A        | Architect i2000| 0.04ug/L |

| New Plymouth                  | I-0         | Vitros Ecl  | 0.08     | 0.03   | T-R        | Elsco 2010| <14ng/L |

| Wellington/Southern, North Island/Top South Island |             |             |          |        |             |          |        |          |
| Wanganui                       | T-R         | Elecsys 2010| 0.03     | 0.04   | I-A        | Architect i2000| <0.03ug/L |
| Palmerston North               | I-A         | AxSym 1.0   | T-R      | 0.03   | T-R        | Elsco 2010| <14ng/L |
| Tairora (Maesterton/Wai-aro)   | T-R         | Elecsys 2010| 0.03     | 0.03   | T-R        | Elsco 2010| <14ng/L |
| Hutt                           | T-R         | Elecsys 1010| 0.03     | 0.03   | T-R        | Modulat E170| 14ng/L |
| Blenheim                       | T-R         | Elecsys 2010| 0.01     | 0.03   | T-R        | Cobas 601  | 14ng/L |

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While considerable effort has been taken, data may not be entirely accurate due to its historical nature and numerous means b
quoted upper reference limit
Brackets denote method used as backup.
*Hospital not in 2002 National ACS Audit.
^ Indicates first-line troponin method used, whether on-site or off-site.
* Greenlane Hospital no longer admitting ACS patients in 2007 or 2011
^ Samples sent off-site for either first line or followup testing (2011 survey only).
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Figure 1A shows the geographical distribution of troponin use across the 20 New Zealand district health boards (DHBs) in 2011. Even within the same DHB, different troponin assays and analysers are sometimes used.

**Distribution of assays used in private laboratories**—There is an even distribution of troponin T and I users among private laboratory providers across New Zealand. Almost half of them, (8/15) use the Roche troponin T assays in their main laboratories, whereas the rest use Abbott (4/15), Beckman Coulter Access (2/15) and Siemens (1/15) troponin I assays.

Table 3 and Figure 1B show the analysers and cutpoints of the private laboratory providers across New Zealand. Note that some peripheral hospitals are also run by private providers, and may have assays that differ from the main laboratory. For example, Southern Community Laboratories (SCL) runs the service for Queenstown, Oamaru and Clyde-Dunstan, all of which have Abbott i-stat instruments, whereas their main Dunedin and Christchurch labs use Roche Troponin T.

**Introduction of Roche high sensitivity (5th generation) assay**—Since the introduction of the 5th generation high sensitivity assay (hs Troponin T), from May 2010 onwards there has been a rapid uptake among existing Roche users, initially in the lower North Island, then in Auckland/Northland and later in the South Island. By February 2011 all Roche laboratory analyser platforms had switched to the hs Troponin T test.

Despite the readiness to accept this improved assay, the changeover was delayed in some places through uncertainty among clinical staff on the diagnostic and workflow implications of the new test, and there were concerns over a possible significant
increase in “abnormal” results and therefore emergency department presentations, before diagnostic and management algorithms were established.

Both 4th and 5th generation tests were typically available for a short time to enable clinicians to become more familiar with the more sensitive 5th generation test during the changeover. In some regions, such as the upper North Island, the introduction was coordinated between laboratories to occur at the same time.

Figure 1A. Distribution of troponin T, troponin I and mixed assays by District Health Board Hospital laboratories as at 2011 (Chatham Islands [troponin T] not shown)
Figure 1B. Distribution of troponin T and troponin I assays used by community laboratories as at 2011
Table 3. Main testing platforms of private community laboratories: 2007 and 2011

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Northland Path.</td>
<td>I-B</td>
<td>Centaur</td>
<td>T-R</td>
<td>Elecsys 2010</td>
<td>&lt;14ng/L</td>
</tr>
<tr>
<td>Diagnostic Medlab</td>
<td>T-R</td>
<td>Modular</td>
<td>T-R</td>
<td>Modular</td>
<td>&lt;14ng/L</td>
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<td>N/A</td>
<td>I-S</td>
<td>Centaur</td>
<td>&lt;40ng/L</td>
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<td>I-BC</td>
<td>DXI/Access</td>
<td>0.06ug/L=</td>
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<td>I-BC</td>
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<td>0.06ug/L=</td>
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<td>Architect</td>
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<td>Centaur</td>
<td>T-R</td>
<td>Cobas 601</td>
<td>&lt;14ng/L</td>
</tr>
<tr>
<td>Wellington (Aotearoa)</td>
<td>I-A</td>
<td>Architect</td>
<td>T-R</td>
<td>Modular</td>
<td>&lt;14ng/L</td>
</tr>
<tr>
<td>T Lab Ltd Gisborne</td>
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<td>I-A</td>
<td>Architect</td>
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<tr>
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</table>

Manufacturer Codes: R=Roche, D=Dade Behring, A=Abbott, B=Bayer, 0=Orthoclinical J&J, BC=Beckman Coulter, S = Siemens; I=Troponin I, T=Troponin T, = quoted upper reference limit.

Note:
- In a number of cases these laboratories will also be recorded as hospital laboratories, since the community laboratory is either responsible for the hospital service (e.g. Pathlab runs both community and hospital services in Bay of Plenty and Whakatane), or there may be a joint venture between the DHB laboratory and a private lab (e.g. Gisborne).
- Private laboratories may also be responsible for managing testing in small rural or peripheral hospitals, using a different analyser, e.g. Southern Community Labs (SCL) performs troponin I testing using i-stat at Queenstown, Oamaru and Clyde-Dunstan Hospitals, while using Roche Troponin T (Modular) in its Dunedin laboratory.

Laboratory reporting practices—With the introduction of the Roche hs Troponin T method, the reporting units have changed from micrograms per litre (ug/L) to nanograms per litre (ng/L), to reflect greater sensitivity and minimise confusion. However, troponin I tests are still reported in ug/L by most laboratories in New Zealand.

The exception is Auckland where all four laboratories measuring troponin I (Middlemore, North Shore, Waitakere, Labtests) report results in ng/L, effectively by multiplying the analyser result by a thousand using their laboratory information system.

The number of different ‘cutpoints’ used by different NZ hospitals in 2002, 2007 and 2011 was reviewed. Table 4 shows the recommended manufacturer and laboratory cutpoints at these times. During these periods there were differences in quoted cutpoints for major assays in use. For example in 2007 three cutpoints (0.03, 0.04 and 0.15ug/L) were reported for the Architect platform. There were also three cutpoints for the Roche Troponin T assay (0.03, 0.04 and 0.01ug/L).
By February 2011, all laboratories using Roche hs Troponin T had the same assay cutpoint, with all but one reporting <14ng/L with their report (Kaitaia reported 0-13ng/L as their reference interval). Importantly, however, a different cutpoint is still currently used for troponin T point of care instruments (0.03ug/L).

Table 4. Quoted cutpoint levels for New Zealand laboratories and recommended manufacturer cutpoints 2002, 2007 and 2011*

<table>
<thead>
<tr>
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<th></th>
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<td>N/A</td>
<td>N/A</td>
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<td>ABBOTT Troponin I</td>
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<td>0.04ug/L</td>
<td>0.08ug/L</td>
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<td></td>
<td></td>
<td>0.5ug/L</td>
<td>1.2ug/L</td>
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<td>0.08ug/L</td>
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<td>0.03ug/L^</td>
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<td></td>
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<td>2.0ug/L</td>
<td>0.15ug/L</td>
<td>0.03ug/L</td>
<td>0.04ug/L</td>
<td>0.03ug/L^</td>
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<td></td>
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<td>Stratus II</td>
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<td>N/A</td>
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<td>Access/DXI</td>
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<td>N/A</td>
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<td>0.04ug/L</td>
<td>0.06ug/L</td>
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<td></td>
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<td>N/A</td>
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<tr>
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<td>N/A</td>
<td>0.04ug/L</td>
<td>0.04ug/L</td>
<td>0.06ug/L</td>
<td>0.06ug/L*</td>
</tr>
</tbody>
</table>

Troponin I assays are also marketed by a number of other manufacturers, but have not been used in New Zealand

* 99th healthy population percentile
^ 10% assay coefficient of variation
# Unequivocal myocardial infarction cutpoint (based on comparisons with CKMB data)
% Correlated with Roche Cardiac reader cut off
$ 97.5th population percentile
= Quoted laboratory upper normal limit, Man=manufacturer, tn=troponin

While considerable effort has been taken, the data may not be completely accurate in view of its historical nature and numerous contacts by which it was gathered.
Troponin I assays also have different cutpoints in different laboratories. For example, for the Abbott i-stat machine four different cutpoints are quoted (0.03, <0.04, 0.08 and <0.09ug/L).

**Point of care testing**—Point of care testing (POCT) is widely used in a number of rural hospitals both for initial patient management and also as backup when laboratories cannot be viably staffed for a full 24 hours. Table 1 includes laboratories where POCT analysers are available after hours. The Abbott i-stat machine is a common choice with 7 rural labs using this instrument for routine first-line testing and 5 using it as a backup.

POCT is also used in some general practice settings where obtaining a laboratory result urgently is otherwise impractical, especially after hours, e.g. Waiheke Island (Cardiac reader), and in rural practice settings, e.g. Hawera, Waipukurau (i-stat) (data not shown).

Results reported by POCT are less precise, and therefore less sensitive, than laboratory analysers. Therefore laboratory reports using POCT analysers have a comment emphasising that the test is helpful to diagnose myocardial damage if positive, but a negative result does not exclude it, i.e. a positive test is useful to ‘rule in’ myocardial injury in the right clinical setting, but a negative result also does not rule it out.

POCT methods using both Troponin T (Cardiac reader, Radiometer AQT) and troponin I (Abbott i-stat) currently continue to be expressed in ug/L, although the Roche POCT analysers will be calibrated in ng/L in the near future.

The quoted sensitivity cutpoints for POCT analysers measuring troponin T (Roche cardiac reader/H232, Radiometer AQT) are similar at 0.03ug/L, although the Cardiac reader provides a semi-quantitative (‘low’) result in the lower abnormal range (0.03-0.1ug/L), while the AQT instrument provides a numerical result. For the i-stat machine, the manufacturer (Abbott Diagnostics) quotes two different cutpoints, either 0.08ug/L corresponding to the 99th population percentile, or 0.03ug/L, representing the 97.5th population percentile.

The individual choice of POCT instrument depends on a range of factors, including clinical preference, cost (of analyser, reagents and staff time/training) and alignment with other laboratory needs such as the ability of the POCT analyser to measure other analytes besides troponin. For example, the Abbott i-stat, Roche H232 analyser and Radiometer AQT instruments can measure other tests, making them useful options in an emergency department or rural/community setting. Thus the i-stat is used in Ashburton and Buller, while in the Waikato the Radiometer AQT is used as the first-line method in Tokoroa and Te Kuiti, and as a backup in Thames and Taumarunui.

In some centres (e.g. Rawene), the wide menu of other tests offered by the i-stat means that this instrument is used to measure troponin I, even though the referral hospitals (Whangarei, Auckland) measure hs Troponin T.
Discussion

Troponin measurement is pivotal in the diagnosis, risk stratification and management of ACS. The definition of MI requires a rise and/or fall in cardiac troponin concentration. Strictly, this means measurement by the same method, in the absence of close correlation between different assays. The magnitude of troponin elevation correlates with risk of death or non-fatal MI following ACS. Patients with elevated troponin levels also benefit more from antithrombotic therapy, glycoprotein 2b/3a receptor antagonists and revascularisation than patients with normal troponin levels.

Troponin cutpoints—In 2000 the ESC/ACC Consensus group recommended that the appropriate cutpoint for troponin levels for diagnosis of MI should be the 99th percentile of a healthy population. They also stated that the assay reproducibility (the imprecision measured as coefficient of variation) should be 10% or less at that concentration. The International Federation of Clinical Chemistry (IFCC) Committee on Standardisation of Markers of Cardiac Damage (C-SMCD) also supports these guidelines.

Since 2000 there have been major advances in assays. With improved assay precision and sensitivity, recommended cutpoints have changed progressively from CKMB-derived thresholds (using the now outdated WHO definition of MI), to those based on 10% assay precision, to the 99th population percentile. Until recently, no assay in clinical use had sufficient sensitivity to satisfy this 99th percentile cutpoint. The Roche hs Troponin T and Siemens ‘Ultra’ now have this precision.

Rather than provide a single ‘cutpoint’, it is widespread practice for manufacturers to provide a summary of their own ‘in-house’ data (usually validated by independent published studies), showing the 10% CV and 99th population percentiles for their assay. There is routinely a recommendation that each laboratory validate a reference range for its own population, which is a difficult task, particularly for small rural laboratories with staffing and cost-constraints.

These reporting difficulties are also compounded as laboratories generally report ‘rounded’ results generated by their analysers to two decimal places (e.g. 0.032 becomes 0.03ug/L, while 0.037 becomes 0.04ug/L). Some laboratories also reported a reference range with an upper normal limit, while others report a ‘less than’ number (e.g. <0.04ug/L).

Some manufacturers still quote a threshold that is “unequivocal” or “diagnostic” of MI, referring back to comparisons with previous CKMB results. For example Abbott uses an “unequivocal” threshold of 0.3ug/L and Beckman a threshold of 0.5ug/L. Both are about 10 times higher than ranges routinely quoted by NZ laboratories, although some still refer to this “unequivocal” threshold with a comment in their reports. While hard to quantify, anecdotal evidence suggests local clinician preferences played a role in some cases in influencing laboratory cutpoints.

Interpretation and standardisation of troponin results—Despite significant assay improvements, there are persisting problems with interpretation and standardisation of serum troponin measurements. This is more so for cardiac troponin I as different
manufacturers use different antibodies raised against different epitopes on the cardiac troponin I protein.\textsuperscript{21}

These different antibodies vary in their ability to bind troponin I. It is also important to note that troponin I can exist in multiple forms in the serum:

- Free,
- Bound as a two unit binary complex (cTroponin I–cTnC), and
- Bound as a three-unit ternary complex (cTroponin T–cTroponin I–cTnC).

There are potentially several additional forms that also exist for each of these three forms, representing N- and C-terminal degradation products, oxidised and reduced forms, and phosphorylated forms.\textsuperscript{22}

Different assays do not react on an equimolar basis with these different molecular species. Therefore, different assays do not produce equivalent concentration results and comparisons of absolute troponin I concentrations cannot be reliably made.\textsuperscript{23}

There have been considerable efforts towards international standardisation of troponin I assays.\textsuperscript{7} However, troponin I results can still differ by at least several-fold, sufficient to make direct comparisons of results between assays unhelpful and potentially dangerous when serial samples are taken in an ACS setting.\textsuperscript{24,25}

By contrast assay standardisation issues are minimal with troponin T because the same antibodies are used—even when the assay has been released for use on another manufacturer’s platform (e.g. Radiometer AQT).

Given the heterogeneity of troponin assays, Apple\textsuperscript{26} has proposed a cardiac troponin assay scorecard. This lists the various assays according to the total imprecision at the 99\textsuperscript{th} percentile and whether the assays are “clinically usable” or “guideline acceptable”.

This scorecard may be useful in providing some guidance to clinicians and laboratories regarding the strengths and weakness of various assays. He suggests only two assays are guideline compliant (Siemens 'Ultra' troponin I and Roche hs Troponin T) and, of the two assays available in New Zealand only one, hs Troponin T, is classified as high sensitivity. However, a recent comparison of several 'sensitive' troponin assays suggested comparable performance in the diagnosis of MI in patients presenting acutely with ACS but superiority to the 4th generation troponin T assay.\textsuperscript{27}

This study included three of the assays currently in use in New Zealand, the Abbott Architect troponin I, Roche hs Troponin T, and Siemens troponin I Ultra assays. Receiver operator curve (ROC) analysis showed high diagnostic accuracy with identical areas under the curve of 0.96 for these three assays. A separate recent comparison study also indicated that the Beckman Coulter assay had excellent diagnostic sensitivity.\textsuperscript{28}

Use of these high sensitivity troponin tests allows for earlier diagnosis, or exclusion of MI. The recent update of the Cardiac Society of Australia and New Zealand ACS Management Guidelines suggest that clinicians now take specimens 3 hours apart, with 1 at least 6 hours after symptom onset.\textsuperscript{29}

**Troponins and clinical management**—Although laboratories have provided education to users as well as interpretive comments with their results, clinicians have
taken time to become familiar with the newer assays and the implications of elevated levels, especially in the low range. Assay 99th population percentiles quoted by manufacturers are based on a ‘healthy’ (typically relatively young) population. However, many elderly patients have small degrees of troponin elevation above this ‘healthy’ cutpoint for multiple reasons. This can cause diagnostic difficulty if symptoms are non-specific and accompanied by “mild elevation” on a first test.

Diagnosis in such patients requires follow-up tests performed using the same assay platform to confirm or exclude acute myocardial injury. In New Zealand, based on biological and analytical variability considerations, a hs Troponin T initial level of 14-49 ng/L and a change of 50% or more in serial blood tests is currently required for the diagnosis of myocardial infarction in the appropriate clinical setting. If the initial hs Troponin T level is ≥50 ng/L (at some sites 53 ng/L), a change of 20% or more is required for diagnostic purposes.

In some referral networks there has been a trend towards similar methodology as instrument platforms are updated. The Northland regional centres (Kaitaia, Kawakawa, Dargaville and Whangarei) which routinely send their patients to Auckland City Hospital have since 2007 changed their assays to Roche troponin T which is also being used at LabPlus in Auckland.

Peripheral and regional hospitals in Waikato and the far North also use troponin T, similar to their major referral hospital. Conversely, in Whakatane hospital troponin testing has moved from troponin T to Beckman Coulter troponin I, also using the same test performed in the local community.

However, despite uniformity in some areas, in a number of regions different troponin assays and analysers are being used so that patients frequently have troponin measurements by more than one (even several) methods. Since results from different methods cannot be directly compared this has implications for ACS patients transferred from one centre to another.

Extra costs are incurred in repeating troponin tests and hospital stay may be prolonged while repeat tests are performed. The same applies to patients who have had troponin tests done in the community or by a private laboratory and are subsequently referred to the public hospital. Table 5 illustrates the potential transfer pathways of cardiac patients across New Zealand and the troponin assays in use among different centres.

Changes in troponin testing are related to multiple other factors besides the choice of the troponin assay itself. In the past 10 years national and regional district health board initiatives with tendering for bulk-funded laboratory services have resulted in significant consolidation of laboratories in New Zealand, but with little coordination across district health boards as each has pursued individual solutions for laboratory services.

Troponin tests in some provincial hospitals are routinely performed by their local private laboratories (e.g. Palmerston North, Whakatane), as part of a contract to run the hospital laboratory. Conversely some private laboratories in the community send their troponin requests on to their local public hospital (e.g. Taranaki). Further, there has been little attention to the impact of different troponin tests on patient management across New Zealand’s 5 major Cardiothoracic Regions which collaborate to manage ACS patients who require invasive management at the Regional Centre.
For cost and efficiency reasons the choice of assay has often been determined not just by clinician and pathologist preference, but by what is considered to be the most cost-effective overall analyser configuration, due to the need to purchase large and expensive analyser platforms that perform many different tests.

Table 5. Examples of transfer pathways for cardiac patients across New Zealand and various troponin assays used 2011

<table>
<thead>
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<th>First patient encounter ^</th>
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<td>I-S</td>
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</table>

Manufacturer Codes:
R=Roche, Ra= Radiometer, A=Abbott, BC=Beckman Coulter, S = Siemens
I=Troponin I, T=Troponin T

^ Usually a community laboratory or peripheral hospital. In these sites the same laboratory usually handles both community and hospital work. In some cases patients are referred directly from community labs or peripheral hospitals to referral hospitals. In others they may be referred to regional hospitals, and then sent on to referral hospitals for intervention or further evaluation.

* In Hawke’s Bay some GP practices get troponin T (Roche) through Southern Community Laboratories (SCL), while others get Abbott troponin I (Hawke’s Bay DHB laboratory). After hours all GPs get troponin I through the hospital laboratory.

The difficulties of providing a 24 hour service for this important test have also led to individual solutions to provide backup testing. The constraints and cost-implications of doing this have not always been well understood by funders. Point of care testing (POCT) is a common solution, depending on the staffing needs of the hospital, the range of other platforms available, and distance from other laboratories able to also measure troponin.

Clinical trials are increasingly using troponin values as part of their enrolment as well as outcome criteria. Use of different assays could lead to diagnostic inconsistencies between different centres and/or regions, potentially confounding trial results. This
also can affect the accuracy of community wide disease rates and compromise the ability to apply trial results to the general population.

**Mechanisms of troponin release**—Our understanding of the underlying mechanisms of troponin release has also increased. Very small amounts of troponin may be released by means other than gross myocyte destruction (infarction). Recently White has suggested six possible major pathobiologic mechanisms for troponin elevations. Troponin elevations could be due to myocyte necrosis, apoptosis, normal myocyte cell turnover, cellular release of proteolytic troponin degradation products, increased cellular wall permeability or passage of membranous blebs.

**Study limitations**—A potential limitation of this study is that the data regarding ‘cut points’ was obtained via personal communication, and repeated phone and email questioning of laboratory and clinical staff. Much of the data obtained were also retrospective from up to 10 years ago.

The implications of varied laboratory testing platforms, in terms of the cost of retesting troponin results on different platforms for transferred patients, would require further study. In addition, the risk to a patient where a cut point has been inappropriately applied, and the frequency with which errors of interpretation are related to different platforms, would also require further exploration.

**Conclusion**

Troponin assays have improved throughout New Zealand in the last 10 years reflecting international improvements in assays and clinical practice. However, there remain differences in the troponin tests and reporting practices across New Zealand hospitals.

Point of care troponin tests are widely used in smaller regional hospitals and in some rural and community settings. Care is required in interpretation, especially when comparing with follow-up by a more sensitive test.

The use of different assay platforms, units and sometimes different cutpoints for the same assay can result in difficulties in interpretation of changes when referring patients from the community or peripheral hospitals to tertiary referral hospitals. This is a particular problem when diagnosis requires clinical interpretation based on a change in troponin results.

Clinicians and District Health Board management need to be aware of these issues. Physicians and pathologists need to collaborate in establishing acceptable and uniform criteria for troponin assays and their use. Some standardisation of available tests, thresholds and testing protocols is likely to improve patient management and the accuracy of community wide disease rates.

A more coordinated national approach may result in better use of medical resources and improve patient care.

**Competing interests:** None declared.

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Correspondence: Dr Chris Ellis, Cardiology Department, Green Lane CVS Service, Level 3, Auckland City Hospital, Grafton, Auckland 1023, New Zealand. Email: chrise@adhb.govt.nz

References:
11. Ellis C, Devlin G, Elliott J, et al for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group. ACS patients in New Zealand experience significant delays to access cardiac investigations and revascularisation treatment especially when admitted to non-interventional

Mortality by ethnic group to 2006: is extending census-mortality linkage robust?

Lavinia Tan, Tony A Blakely

Abstract

**Objective** To update trends in mortality by ethnic group from the New Zealand Census-Mortality Study (NZCMS), by additionally linking 2004–06 mortality records to the 2001 Census. To investigate possible bias from this extended linkage, especially for Pacific and Asian people who emigrate more frequently.

**Methods** Anonymous and probabilistic record linkage of 2004–06 mortality records with the 2001 Census was undertaken. Age-standardised 1–74 year old mortality rates by sex and age group, and for all-cause and selected causes of death, were calculated using the direct method for first 30 months post 2001 Census (2001–03) and second 30 months (2003–06).

**Results** Observed all-cause mortality rates continued to fall in 2003–06 compared to previous periods, but more so for Pacific (18.3% and 21.7% for males and females for 2003–06 compared to 2001–04, respectively) and Asian (22.2%, 16.7%), than for Māori (13.2%, 14.2%) and European/Other (13.0%, 10.4%). Observed rate ratios for Māori, compared to European/Other were 2.43 (95% CI 2.31–2.57) for males and 2.72 (2.56–2.89) for females, the same (males) and slightly less (7%, females) than in 2001–03.

Declines in cardiovascular disease (CVD) and injury mortality were the main drivers of all-cause mortality rate reductions for all ethnic groups. Relative inequalities in CVD between Māori and European/Other remain high (three to four-fold relative risks), but reduced by 8% for both males and females from 2001–03 to 2003–06, which in turn means that absolute inequalities closed by as much as 20%.

**Conclusion** We suspect that analyses comparing mortality rates over time within one of the closed NZCMS cohorts (e.g. 2001–03 compared to 2003–06) is prone to bias due to our inability to censor people when they migrate out of New Zealand. This limitation means mortality rates in the NZCMS are increasingly underestimated with time since census night, particularly for Pacific and Asian people. However, previously published NZCMS trends remain valid as the duration of follow-up (3 years) is short, and cohorts were not split by time since census.

Nevertheless, it is safe to conclude that mortality rates continued to decline from 2001-03 to 2003-04 for all four ethnic groups. All-cause mortality inequalities for Māori compared to European/Other over this time were probably stable in relative terms and decreasing in absolute terms, but cardiovascular disease (CVD) inequalities probably decreased in both absolute and relative terms.
The measurement and monitoring of ethnic inequalities in health have long been of interest in New Zealand, both for research and policy. There are large differences in mortality between ethnicities in New Zealand.\textsuperscript{1-4}

Māori and Pacific have higher mortality rates than non-Māori non-Pacific non-Asian (nonMPA), while mortality rates of Asian people (people from East, South East and South Asia, but excluding those from the Middle East and Central Asia) are less than nonMPA in New Zealand.\textsuperscript{1} The main contributors to these inequalities are mortality in the older age groups (45+ years), particularly for cancer and cardiovascular disease.\textsuperscript{1}

Mortality rates change over time with changes in society, culture and economy, and perhaps differentially by ethnicity. Māori mortality decreased markedly up to the 1980’s then slowed its decline, such that the gap between Māori and non-Māori increased. Since the late 1990s, mortality rates have declined at a faster rate among Māori, and absolute differences in rates (and life expectancy) are again closing between Māori and non-Māori.\textsuperscript{2,3}

In the past, we have only linked 3 years of mortality data to each census to secure more rapid monitoring data after each census and also because linkage success decayed with time following census. However, we noted in the 2001–04 mortality linkage and in parallel linkage of 5 years of cancer registrations to the census (CancerTrends: www.uow.otago.ac.nz/CancerTrends-info.html) that linkage now deteriorates little with time for events 4 to 5 years after census night. This improvement is due to numerous sources of residential geocodes, enabling us to better select a geocode for the decedent at about census night even when their death occurred 4 to 5 years after census night. To provide more complete data on social group trends in mortality in New Zealand, we linked 2004–06 mortality records to the already existing 2001–04 census-mortality cohort.

This paper presents trends in mortality, by ethnicity, for the period 1981–2006, with a particular focus on comparisons of the first and second 30 months post-2001 Census (hereafter 2001–03 and 2003–06, respectively) with existing 1996–99 NZCMS data. We investigate the validity of the extended census-cohort, and changes in ethnic-specific all-cause and cause-specific mortality rates and inequalities.

**Method**

**Linkage and weighting**—Methods for linkage of each of the 1981, 1986, 1991, 1996 and 2001 Censuses with 3 years of subsequent mortality data have been described elsewhere.\textsuperscript{1-9} A technical note on the 2004-06 linkage can be found at the NZCMS website.\textsuperscript{10} Briefly, probabilistic record linkage was used to link census and mortality records anonymously using the variables sex, date of birth, country of birth and ethnic group. The blocking variable used for matching was address or census area unit, using multiple health dataset sources for address geocodes to attempt to bracket the date of the 2001 Census.

Mortality records for 2001–06 eligible for linkage were those where the decedent was alive at the 2001 Census and was living in New Zealand on 2001 Census night according to the duration in New Zealand variable recorded on the mortality file.

The percentage of eligible mortality records linked in 2001–04 was 79.6%, and in 2004-06 was 79.8%. Because not all mortality records were linked to a corresponding census record, it was necessary to correct for any linkage bias and consequent underestimation of mortality rates.
Weights were calculated based on variables that were predictors of linkage in logistic regression analyses: age at census, sex, prioritised ethnicity, rurality, residential mobility of area unit, Territorial Authority, NZ deprivation index, months since census night at death, and cause of death. To improve efficiency of data processing, the method for weight calculation for the full 2001–06 data differed from previous years. Cells within a stratum that met the numerical criterion of > 5 linked records, were separated and assigned an independent weight, whereas the remaining cells were collapsed.

The order of collapsing of strata variables to ensure sufficient cell sizes was based on the strength of their relationship with linkage (see\textsuperscript{10}). The initial weights were secondarily weighted (in the same manner as previous cohorts) by age, sex and ethnicity so that the sum of all the weighted linked mortality records was forced to equal the total number of linked and unlinked mortality records within age, sex and ethnicity strata. Overall, the revised weighting procedure was found to perform as satisfactorily as previous methods (see\textsuperscript{10} for a more detailed description of the weighting process).

Due to larger than expected reductions in mortality rates from 2001–03 to 2003–06 (as reported in Results section of this paper), we undertook an additional weighting to force the exact sum of weighted and eligible deaths for the first and second 30-month periods following the 2001 Census to exactly agree (see\textsuperscript{10}). That is, we were concerned that despite including ‘months since census’ in our primary weighting strategy, our above algorithm of aggregating cells did not adequately correct for any residual deterioration in linkage success – particularly for the younger Māori and Pacific age groups. However, there were no systematic differences between our preferred weights and the additional weights based on duration since census night.

We therefore concluded that residual linkage bias by time since census was not occurring. Consequently, the preferred linkage weights were used for all analyses by the 30 month split (i.e. 2001–03 and 2003–06). We also include previously published 2001–04 rates for comparison.\textsuperscript{2,11}

\textbf{Cohort analyses}—As in previous NZCMS reports and publications (e.g.\textsuperscript{11}) and consistent with ethnicity standards,\textsuperscript{12} a “Total” definition of ethnicity was used for Māori, Pacific and Asian in analyses, using (all) ethnic groups self-identified by the census question. Thus individuals could be assigned to up to three of Māori, Pacific and Asian ethnicities. A mutually exclusive group of non-Māori non-Pacific non-Asian (hereafter called European/Other) was used as the reference group for ethnicity comparisons.

Age-standardised rates were calculated for all four ethnic groups, using the WHO World population as the standard. We examined changes in mortality rates and relative (standardised rate ratios; SRR) and absolute (standardised rate differences; SRD) inequalities in rates by sex and age group. All data analyses were conducted in SAS.

\textbf{Results}

\textbf{All-cause mortality rates}—Figure 1 shows age standardised all-cause mortality rates for 1–74 year old males and females. Each observation is plotted at the midpoint of its period of follow-up according to the X-axis, meaning that the 2001–03 and 2003–06 series are closer together than the 5-year gaps in the existing 1981–84 to 2001–04 series. The 2001–03 rates commencing the new series agree reasonably closely with the previous 2001–04 rates, with any difference being due to slightly different follow-up periods (30 versus 36 months) and the modified linkage weights.

Mortality rates were consistently highest for Māori, followed by Pacific, then European/Other ethnicities, and lowest for Asians, and inequalities were large at all points in time. The long-run trend of steadily decreasing mortality rates over time for European/Other and Asian continued and recent accelerations in Māori mortality decline seemed to be maintained.

Pacific mortality rates were more variable pre-1996, but also exhibited the same decrease from 1996 onwards. More specifically, observed all-cause mortality rates fell for all ethnic groups from 2001–03 to 2003–06, but more so for Pacific (18.3\% and
21.7% for males and females, respectively) and Asian (22.2%, 16.7%), than for Māori (13.2%, 14.2%) and European/Other (13.0%, 10.4%) (Table 1 and Table 2).

**Figure 1. All cause age-standardised mortality rate (per 100,000) for males and females by ethnicity**

The first line (starting from the left) for each ethnic group is for the previously published 1981–84, 1986–89, 1991–94, 1996–99 and 2001–04 series (i.e. three years of mortality data each linked to a census). The second line is that for the new series, 2001-03 and 2003-06. Error bars are 95% confidence intervals.
Table 1. Person years and numbers of weighted deaths for all ages (1-74 years) combined for 2001–03 and 2003–06

<table>
<thead>
<tr>
<th>Variables</th>
<th>Sex</th>
<th>Total</th>
<th>Māori (%)</th>
<th>Pacific (%)</th>
<th>Asian (%)</th>
<th>European/Other (%)</th>
<th>Missing (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Person-years</td>
<td>2001–03</td>
<td>Females 4,092,544</td>
<td>609,966</td>
<td>269,221</td>
<td>2,887,506</td>
<td>288,506</td>
<td>37,345</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Males 3,926,711</td>
<td>572,319</td>
<td>258,051</td>
<td>2,794,626</td>
<td>258,404</td>
<td>43,311</td>
</tr>
<tr>
<td>2003–06</td>
<td>Females</td>
<td>4,014,015</td>
<td>605,543</td>
<td>268,236</td>
<td>2,816,768</td>
<td>287,307</td>
<td>36,161</td>
</tr>
<tr>
<td></td>
<td>Males</td>
<td>3,845,348</td>
<td>566,905</td>
<td>256,782</td>
<td>2,722,324</td>
<td>257,098</td>
<td>42,239</td>
</tr>
<tr>
<td>Deaths</td>
<td>2001–03</td>
<td>Females 10,443</td>
<td>2022</td>
<td>564</td>
<td>7,470</td>
<td>258</td>
<td>129</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Males 15,714</td>
<td>2667</td>
<td>837</td>
<td>11,601</td>
<td>411</td>
<td>198</td>
</tr>
<tr>
<td>2003–06</td>
<td>Females</td>
<td>9,624</td>
<td>1905</td>
<td>480</td>
<td>6906</td>
<td>231</td>
<td>102</td>
</tr>
<tr>
<td></td>
<td>Males</td>
<td>14,091</td>
<td>2520</td>
<td>750</td>
<td>10,299</td>
<td>357</td>
<td>165</td>
</tr>
</tbody>
</table>

Table 2. Standardised mortality rates and percentage change from preceding cohort for all cause mortality by ethnicity, period and sex. (Full tables of rates for all age groups and all causes of death are available as web annex tables at [http://www.uow.otago.ac.nz/nzcms-info.html](http://www.uow.otago.ac.nz/nzcms-info.html).)

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Cohort</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Std Rate (95% CI)</td>
<td>% change†</td>
</tr>
<tr>
<td>Māori</td>
<td>1996–99</td>
<td>823 (788–858)</td>
<td>541 (514–568)</td>
</tr>
<tr>
<td></td>
<td>2001–04</td>
<td>697 (668–726)</td>
<td>488 (465–512)</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>735 (701–768)</td>
<td>517 (490–543)</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>638 (607–668)</td>
<td>443 (420–467)</td>
</tr>
<tr>
<td>Pacific</td>
<td>1996–99</td>
<td>625 (573–677)</td>
<td>361 (325–397)</td>
</tr>
<tr>
<td></td>
<td>2001–04</td>
<td>526 (486–567)</td>
<td>323 (294–353)</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>554 (508–600)</td>
<td>338 (305–372)</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>453 (412–493)</td>
<td>265 (237–293)</td>
</tr>
<tr>
<td>European/Other</td>
<td>1996–99</td>
<td>339 (332–345)</td>
<td>194 (190–199)</td>
</tr>
<tr>
<td></td>
<td>2001–04</td>
<td>294 (288–300)</td>
<td>178 (173–182)</td>
</tr>
</tbody>
</table>
We suspect that there is a systematic bias in the NZCMS due to the inability to censor respondents who migrate out from New Zealand after census night, and we also suspect this bias might be greater for Pacific and Asian people. We consider this further in the Discussion. Thus, the ‘observed’ rates, SRRs and SRDs for Asian and Pacific people in 2003–06 need to be treated with considerable caution and accordingly, the remainder of the Results section is mostly focussed on rates calculated for Māori and European/Other.

Observed rate ratios for Māori, compared to European/Other, were 2.43 (95%CI 2.31–2.57) for males and 2.72 (2.56–2.89) for females in 2003–06, the same as in 2001–03 for males (2.44) and 7% less for females (2.84) (Table 3 and Figure 2). The observed SRDs decreased by 13% for males (from 434 to 376 per 100,000) and by 16% for females (from 335 to 281 per 100,000).

### Table 3. Age-standardised rate ratios (SRRs) and rate differences (SRD) for selected age groups and causes of death, by sex by ethnic group, for 1996-99 onwards. (Full tables are available as web annex tables at [http://www.uow.otago.ac.nz/nzcms-info.html](http://www.uow.otago.ac.nz/nzcms-info.html))

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Cohort</th>
<th>Males Std Rate (95% CI)</th>
<th>% change†</th>
<th>Females Std Rate (95% CI)</th>
<th>% change†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2001–04</td>
<td>187 (166–208)</td>
<td>26.4</td>
<td>106 (91–121)</td>
<td>21.8</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>198 (174–222)</td>
<td>22.2</td>
<td>113 (96–130)</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>160 (139–181)</td>
<td>19.0</td>
<td>88 (74–101)</td>
<td>22.4</td>
</tr>
</tbody>
</table>

† 2001–04 compared to 1996–99; 2001–03 compared to 1996–99; and 2003–06 compared to 2001–03.
Mortality rates by age group—Mortality rates were greatest for Māori, followed by Pacific, then European/Other ethnicities, and lowest for Asians for adults aged 15+ yrs (Figure 3; actual rates and percentage changes in Web Annex Table 1 at [www.uow.otago.ac.nz/nzcms-info.html](http://www.uow.otago.ac.nz/nzcms-info.html)). Across all age groups and for both sexes, there is a long-term, steadily decreasing trend in mortality rates.

Rates for Pacific tend to show larger than plausible decreases from 2001–03 to 2003–06 for 45–64 year olds (22.2% and 21.8%, males and females respectively) and for 65–74 year olds (17.1% and 18.6%). By way of comparison, the corresponding percentage declines for European/Other were 12.4% and 7.9% for 45–64 year olds, and 13.8% and 10.4% for 65–74 year olds. Percentage declines from 2001–03 to 2003–06, were also particularly notable among these older age groups for Asian and, to a lesser extent, Māori.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Year</th>
<th>SRR (95% CI)</th>
<th>SRD (95% CI)</th>
<th>SRR (95% CI)</th>
<th>SRD (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular Māori</td>
<td>1996–99</td>
<td>2.81 (2.60–3.03)</td>
<td>203 (181–225)</td>
<td>3.98 (3.60–4.40)</td>
<td>133 (117–149)</td>
</tr>
<tr>
<td>Disease</td>
<td>2001–03</td>
<td>3.10 (2.85–3.37)</td>
<td>182 (161–202)</td>
<td>4.18 (3.75–4.65)</td>
<td>118 (104–133)</td>
</tr>
<tr>
<td>Pacific</td>
<td>2003–06</td>
<td>2.93 (2.67–3.21)</td>
<td>140 (122–158)</td>
<td>3.94 (3.49–4.44)</td>
<td>89 (77–102)</td>
</tr>
<tr>
<td>Pacific</td>
<td>1996–99</td>
<td>2.15 (1.87–2.46)</td>
<td>129 (96–161)</td>
<td>2.64 (2.20–3.16)</td>
<td>73 (53–94)</td>
</tr>
<tr>
<td>Asian</td>
<td>2001–03</td>
<td>2.40 (2.09–2.77)</td>
<td>121 (93–150)</td>
<td>2.74 (2.27–3.31)</td>
<td>65 (46–83)</td>
</tr>
<tr>
<td>Asian</td>
<td>2003–06</td>
<td>2.20 (1.88–2.57)</td>
<td>87 (63–111)</td>
<td>2.41 (1.95–2.99)</td>
<td>43 (28–58)</td>
</tr>
<tr>
<td>Unintentional Injury Māori</td>
<td>1996–99</td>
<td>2.36 (2.01–2.76)</td>
<td>41 (31–51)</td>
<td>2.14 (1.66–2.75)</td>
<td>11 (7–16)</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>1.88 (1.57–2.25)</td>
<td>26 (17–36)</td>
<td>2.50 (1.92–3.26)</td>
<td>15 (9–20)</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>2.06 (1.70–2.48)</td>
<td>27 (18–36)</td>
<td>2.57 (1.93–3.42)</td>
<td>12 (7–16)</td>
</tr>
<tr>
<td>Pacific</td>
<td>1996–99</td>
<td>1.31 (0.98–1.76)</td>
<td>9 (2–21)</td>
<td>1.02 (0.60–1.75)</td>
<td>0 (-5–6)</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>1.43 (1.08–1.91)</td>
<td>13 (1–25)</td>
<td>0.77 (0.41–1.45)</td>
<td>-2 (-7–3)</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>0.99 (0.69–1.43)</td>
<td>0 (-9–9)</td>
<td>0.84 (0.44–1.61)</td>
<td>-1 (-5–3)</td>
</tr>
<tr>
<td>Asian</td>
<td>1996–99</td>
<td>0.91 (0.61–1.36)</td>
<td>4 (-14–8)</td>
<td>0.68 (0.34–1.36)</td>
<td>-3 (-8–2)</td>
</tr>
<tr>
<td></td>
<td>2001–03</td>
<td>0.57 (0.38–0.84)</td>
<td>12 (-20–6)</td>
<td>0.72 (0.39–1.31)</td>
<td>-3 (-7–2)</td>
</tr>
<tr>
<td></td>
<td>2003–06</td>
<td>0.34 (0.17–0.68)</td>
<td>17 (-23–10)</td>
<td>0.86 (0.48–1.53)</td>
<td>-1 (-5–3)</td>
</tr>
</tbody>
</table>
Figure 2. Trends in relative (SRR; left) and absolute (SRD; right) inequalities in all cause mortality for Māori, Pacific and Asian each compared to European/Other, by sex, for all ages (1–74 years)

The first line (starting from the left) for each ethnic group is for the previously published 1981-84, 1986-89, 1991-94, 1996-99 and 2001-04 series (i.e. three years of mortality data each linked to a census). The second line is that for the new series, 2001-03 and 2003-06. Error bars are 95% confidence intervals.

Trends in relative inequalities (SRRs) between Māori and European/Other by age group from 1996–99 to 2001–03 to 2003–06 were unclear across age groups, and therefore arguably best assumed as stable over time (Web Annex Table 3).

Given the background trends for mortality reduction in all ethnic by age groups, there was necessarily a pattern of reducing absolute inequalities between Māori and European/Other over time across age groups. For example, the SRDs among 45–64 year olds reduced by 29% for males and 21% for females from 1996–99 to 2003–06 (Web Annex Table 3), although these may be somewhat overestimated due to the suspected migration bias within NZCMS cohorts (see Discussion).

Mortality rates by specific cause of death— Figure 4 shows cause-specific mortality rates. Declines in cardiovascular disease and injury mortality were the main drivers of all-cause mortality rate reductions for all ethnic groups (Figure 4). Relative inequalities in CVD between Māori and European/Other remain very high (three to four-fold relative risks), but reduced by 8% for both males and females from 2001–03 to 2003–06, which in turn means that absolute inequalities closed by as much as 20%.
Figure 3. All-cause age-standardised mortality rates (per 100,000) by age group, by ethnicity

Each line plots the previously published 1981-84, 1986-89, 1991-94, and 1996-99 series (but not 2001-04), and then continues with the new 2001-03 and 2003-06 series. Error bars are 95% confidence intervals.
Figure 4. Cause-specific age-standardised mortality rates (per 100,000) by sex, by ethnicity

Each line plots the previously published 1981-84, 1986-89, 1991-94, and 1996-99 series (but not 2001-04), and then continues with the new 2001-03 and 2003-06 series. Error bars are 95% confidence intervals.
Māori unintentional injury mortality rates are higher than other ethnicities, for both males and females. Inequalities were unstable over time, and not measured with sufficient precision to make confident conclusions about trends in Māori European/Other inequalities. Injury rates were comparable between Pacific and European/Other ethnicities since 1996–99, and lower among Asian.

Rates for other causes of death can be found at the NZCMS website: www.uow.otago.ac.nz/nzcms-info.html

Discussion

Observed all-cause mortality rates continued to fall in 2003–06 compared to previous periods for all ethnic groups, but especially so for Pacific and Asian people (and probably affected by bias—see below). The falling mortality rates are largely driven by falls in CVD and unintentional injury.

Relative inequalities in mortality between Māori and European/Other have stabilised since the late 1990s—and possibly decreased for females. Given this stability or slight reduction in relative inequalities, and the overall decreasing trend in mortality for all ethnic groups, the absolute inequalities between Māori and European/Other have necessarily decreased.

Methodologically, we believe we have encountered a bias in the NZCMS when results are reported by time since census. As shown in Figure 1, Pacific and Asian rates seem to fall by an implausibly large amount from 2001–03 to 2003–06. We thoroughly checked our linkage bias weights (see Methods and elsewhere10), and do not believe that inadequate adjustment for (any) decline in linkage success is the problem. Rather, we think that the number of deaths among the people alive on census night is progressively underestimated with time since census night, due to our inability to censor New Zealanders as they emigrate.

Put more simply, we do not identify deaths among those New Zealand residents who completed the 2001 Census and subsequently emigrated, and possibly died overseas. As there is relatively frequent migration to and from the Pacific13 14, and this likely applies to a more recent immigration population such as Asian people, we suspect this bias is greater among Pacific and Asian people.

A direct test of this hypothesis would require data on emigration by ethnicity to allow censoring, or identification of deaths among those emigrating. We do not have such data. However, we do have data on ‘permanent and long-term departures’ from New Zealand by country/region of destination (personal communication, Robert Didham, Statistics New Zealand, July 2010).

If our hypothesis about differential emigration by ethnicity is correct, we might also expect emigration of older people (at great risk of death) to be more common among Pacific and Asian populations. Those people emigrating to the Pacific or Asia are skewed towards older age groups, both using simple counts and also a ‘crude’ proportion measure using the 2006 New Zealand Pacific, Asian and all population count data as denominators (Web Annex Figure 1 and 2).
If our suspicions are correct and our observed mortality rates are a result of emigrant bias, then mortality rates for Asian and Pacific in 2003–06 are too low, and SRRs in 2003–06 comparing either Pacific or Asian with European/Other are also too low (due to misclassification bias of the mortality outcome that is differential by ethnicity). We do not have enough evidence to suggest any difference in this bias (in relative terms) for Māori and European/Other; consequently, the SRRs comparing Māori with European/Other are probably valid.

A possibly greater underestimation of Pacific and Asian mortality rates is consistent with a study that examined “unhealthy return migrant” (URM) bias mortality rates for Pacific. Tobias showed that it is possible to estimate the extent of this bias using lung cancer as a “tracer condition” whereby spuriously high survival could only be explained by emigration, and in turn allowed an estimate of approximately 20% undercount for Pacific deaths in older people. However, its accuracy is unknown and immeasurable. For example, the URM adjustor might account for both return migration and missed NHI links between the National Cancer Registry and mortality files.

There are potential methods to correct this bias in the NZCMS in the future, if we are to present mortality rates by year since census night. First, we currently only attempt to link deaths when the mortality record states they have been in New Zealand long enough to have answered the previous census. Thus, if we were to assume that immigration and emigration roughly cancel each other out (not currently true for Asian people), then we could make these excluded mortality records ‘eligible’, and when they fail to be linked to a census record they will then contribute to a large linkage bias weight, thus also (potentially) correcting for this emigrant bias. Second, we could attempt to link NZCMS data to migration data, and ‘properly’ censor the cohorts. However, this would be a large undertaking, and possibly not justifiable from either a SNZ or a funders perspective.

Third, we could attempt to create a database of New Zealand residents (as of census night) dying overseas in the following 5 years. This would also be a very large undertaking, and probably unreliable. Fourth, we could use external data on emigration rates by sex, age and ethnicity to undertake quantitative bias analyses (or sensitivity analyses) of the results presented here.

Unfortunately, migration data is not collected by ethnic group (the closest approximation is country of birth), and we would have to estimate the mortality rate among the emigrants (which would not be the same as non-emigrants). Finally, and currently our preferred option, once 5 years of mortality data have been linked to the 2006 census, we will have a continuous series of 10 years of linked data, with the fifth and sixth years bracketing the beginning of the new cohort. By using regression modelling, we would be able to estimate the magnitude of unexpected ‘jumps’ in mortality rates across this boundary by sex, age and ethnic group, and thereby estimate sex by age by ethnic group specific adjustment factors.

**Conclusions**

We suspect that analyses comparing mortality rates over time within one of the closed NZCMS cohorts (e.g. 2001–03 compared to 2003–06) is prone to bias due to our inability to censor people when they migrate out of New Zealand. This limitation to
the NZCMS means mortality rates are increasingly underestimated with time since census night, particularly for Pacific and Asian people. However, previously published NZCMS trends remain valid as the duration of follow-up (3 years) is short, and cohorts were not split by time since census.

Nevertheless, it is safe to conclude that mortality rates continued to decline from 2001–03 to 2003–04 for all four ethnic groups. All-cause mortality inequalities for Māori compared to European/Other over this time were probably stable in relative terms and decreasing in absolute terms, but CVD inequalities probably decreased in both absolute and relative terms.

Statistics NZ Security Statement: Access to the data used in this study was provided by Statistics New Zealand under conditions designed to give effect to the security and confidentiality provisions of the Statistics Act 1975. The results presented in this study are the work of the author, not Statistics New Zealand’s.

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References:


Improving healthcare through the use of co-design

Hilary Boyd, Stephen McKernon, Bernie Mullin, Andrew Old

Abstract

**Aim** This paper describes how co-design methods can be used to improve patient experiences and services within healthcare organisations. Using the Patient Co-design of Breast Service Project as an example, we describe how patient experiences were captured and understood, the improvements made and implications for future work.

**Method** We used a six-step process: engage, plan, explore, develop, decide and change. Tools and techniques employed were based on service design approaches. These included patient journey mapping, experience-based surveys and co-design workshops.

**Results** Information, communication, navigation and co-ordination, and environment emerged as key themes for the Breast Service. And as a result, a suite of improvements were made. Key methodological learnings included using co-design alongside traditional quality improvement methodologies, engaging with patients early, the importance of staff buy-in and the necessity of trying things outside one’s comfort zone.

**Conclusion** Use of co-design within the Breast Service has resulted in tangible improvements and has demonstrated the value of engaging patients and focusing on their experiences. It is recommended that: evaluation phases are factored into future co-design work, further research is conducted on sustainability and funding and support is given to allow co-design to become more widespread throughout New Zealand.

Co-design challenges the existing quality improvement paradigms commonly used in New Zealand hospitals in three major ways. Firstly, it encourages patients to take an equal role in the review and development of services. Secondly, it focuses strongly on designing services around patient experiences. Thirdly, it uses techniques and tools derived from service design—e.g. prototyping and storyboards, rather than manufacturing environments as well as process maps and statistical process control.

Within a health context, co-design (also known as experience based design or co-production) is “… a method of designing better experiences for patients, carers and staff”. It involves patients and staff exploring the care pathway and the emotional journey patients experience along it, capturing experiences, then working together to understand these experiences and improve them.

Co-design’s innovative way of actively involving patients in healthcare design has been gaining traction overseas for a number of years. Originally piloted in the Head and Neck Cancer Service in Luton and Dunstable, UK, (in 2006) it has successfully spread to other parts of the UK and Australia and more recently New Zealand. The range of health services where co-design has been applied now includes head and
The changing role of patients and their families/whanau in quality

Traditionally patients and their families were seen as passive recipients of health services but in recent years the importance of more meaningful consumer input into the review and design of services has gained currency.

District Health Boards (DHBs) and their predecessor organisations have historically endeavoured to listen to and incorporate patients’ perspectives through mechanisms such as the complaints (and compliments) process, surveys, feedback boxes, representation on reference groups, health literacy groups, consultation meetings and hui and so on. Service improvement and quality projects, too, have recognised the value of listening to, and understanding, patient perspectives.

The way in which patients have input into service improvement in healthcare in New Zealand is gradually evolving from what Bate & Robert describe as a passive (or low involvement) patient mode towards a partnership approach on the ‘continuum of patient influence’ scale. This change has been influenced by:

- The proactive approach of various industries to improving customer experiences.
- The growth of service design.
- A more organised and active consumer voice.
- The prevalence of instant public feedback via the internet and social networking technologies.

Co-design approach

In our New Zealand work, co-design projects incorporate six main elements or phases. The first three elements are primarily about capturing and understanding the patient experience. While the latter three focus on improving the patient experience.

- **Engage:** proactively establishing and maintaining meaningful relationships with patients (and staff) to understand and improve health services.
- **Plan:** working with patients and staff to come up with ideas about the goals of the improvement work and how to go about doing it.
- **Explore:** learning about and understanding patient and staff experiences of services, and identifying things that can be improved.
- **Develop:** turning the ideas into specific improvements.
- **Decide:** choosing what improvements to make and how to make them.
- **Change:** turning improvement ideas into action.

While described as a series of steps, in reality each element may overlap and the order, and even the omission, of some elements is not necessarily important. The common element is the active engagement of patients and their families in each activity undertaken.
The core principles underpinning our co-design work are equity, understanding experiences and improving services.\(^{10}\)

This paper discusses how Waitemata DHB, through its Patient Co-design of Breast Service Project, has worked with patients and staff to improve the breast journey and, on a small scale, trial a methodology not yet widely acknowledged or used in New Zealand.

**Method**

The Breast Service at Waitemata DHB provides services at both North Shore and Waitakere Hospitals. At the time of the project, the Service comprised two breast nurse specialists, four surgeons and four oncologists. Weekly surgical and oncology clinics were held at North Shore Hospital. Breast surgery was performed at both North Shore and Waitakere Hospitals. The Breast Service averaged more than 2,500 referrals per year and approximately 10% of these resulted in a diagnosis of breast cancer.

In 2007, the Patient Co-design of Breast Service Project was set up to work alongside a sister project focussed on improving the referral process and developing clinical guidelines for patients with breast disease. Its aims were to use an innovative co-design approach to understand patient experiences, make small, focussed changes with patients, make further recommendations for changes in the service and develop a model for working with patients that could be used in other services. Further, it strived to involve patients in a deeper, more participatory way, than previously had been done.

In order to capture and understand patient experiences, a number of tools were used. Each tool used produced results which then influenced the type of tool which would be used next and the overall direction of the project. The tools were: patient journey mapping, experience-based surveys and co-design workshops.

**Patient journey mapping**—A patient journey map is a summary of the service experiences patients have over time. It includes patient journey phases, the people they have contact with, the emotions they experience during their journey, touch points (significant points of contact—tangible and intangible— that patients have with the Service) and suggested improvement ideas.

Twenty-one people attended a journey mapping workshop including patients and their supporters (14), staff (5) and workshop organisers (2). Participants were guided through the development of patient journey maps in groups. These were subsequently developed into a summary map (Appendix 1).

Participants discussed ideas for change and improvements at the conclusion of the workshop and came up with a summary list of improvements.

**Experience-based surveys**—Experience-based surveys are one-page surveys to find out how patients experience a specific part of the hospital journey. They allow patients to come up with specific suggestions for improving their experiences.

To gain a deeper understanding of patients’ experiences, and to ascertain benchmark data for the Service, we developed an experience-based survey (adapted from the NHS) (Figure 1).

Over a 6-week period, all patients who attended a Breast Clinic appointment or a mammogram were given a survey. The survey asked patients and their family/whanau to rate their experience of elements of their journey. 182 surveys were completed (97 from those attending a breast clinic appointment and 85 from mammography/ultrasound) representing approximately 31% and 14% response rates respectively. It is important to note that the surveys were not intended to be scientific and as such no demographic information was collected. Further, responses were sought passively and were not followed up which is likely to have affected the response rate.

**Co-design workshops**—Co-design workshops involve a wide variety of people who have an interest in the project getting together in one place to discuss issues, learn together and make decisions. These workshops may be based around starting up a project (start-up or planning workshop), understanding patient or staff experiences or delving in depth into an issue (journey mapping or ideas groups) or coming up with tangible solutions (using tools such as prototyping).
Figure 1. The breast clinic experience survey

The co-design workshop aimed to find out:
- What information given to patients was most useful?
- What other information would patients find useful to improve their experiences?
- When is the best time to get this information?
- What format would people like information provided in?

Twenty-nine people attended including patients and their supporters (12), staff (11), community group representatives (3) and workshop organisers (3). We asked participants to talk about their response when they had the ‘right’ information and what difference the ‘right information’ could make to them (Table 1).

Table 1. Information dynamics

<table>
<thead>
<tr>
<th>Without right information</th>
<th>With right information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely upset</td>
<td>Confidence</td>
</tr>
<tr>
<td>Angry</td>
<td>Powerful, empowered</td>
</tr>
<tr>
<td>Frustrated</td>
<td>Competent</td>
</tr>
<tr>
<td>Scared</td>
<td>Trust</td>
</tr>
<tr>
<td>Confused</td>
<td>Relief</td>
</tr>
<tr>
<td>Anxious</td>
<td>Empathy</td>
</tr>
<tr>
<td>Bewildered</td>
<td>Partnership</td>
</tr>
<tr>
<td>Pressured</td>
<td>Understood</td>
</tr>
<tr>
<td>Let down</td>
<td>A whole person, not just a number</td>
</tr>
<tr>
<td>In conflict</td>
<td>Supported</td>
</tr>
</tbody>
</table>
In small groups, participants then identified what they needed to know at each step in the journey, why they needed to know it, how they could best find out about it and the best media or format for the information.

**Results**

**Patient journey mapping**—Participants identified a range of improvement ideas (see Table 2). There was an agreement that many improvements were oriented towards the beginning of the journey because they have the capacity to influence everything else that follows.

**Table 2. Key identified improvements summarised by phase**

<table>
<thead>
<tr>
<th>Journey phase</th>
<th>What patients wanted</th>
<th>Tools and actions suggested</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Encourage women to go to their GP earlier for check-ups.</td>
<td>Develop diagnosis and referral guide for GPs.</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>A supporter with them when they had their results appointment (and for any other such meetings where they might be told bad news).</td>
<td>Staff to recommend and emphasise the benefits of this when the appointment is made.</td>
</tr>
<tr>
<td></td>
<td>Kind and empathetic staff.</td>
<td>Have patients give talks about their experiences and problems to staff and patients on training courses.</td>
</tr>
<tr>
<td></td>
<td>A ‘host’ for women on their entry to the Service to act as their ‘navigator’ through their journey.</td>
<td>Provide one constant point of contact throughout the whole journey.</td>
</tr>
<tr>
<td>Treatment</td>
<td>The option of a longer stay in hospital after surgery to prepare psychologically and practically for the return home.</td>
<td>Help people prepare for recovery at home, building their confidence and skills (method to be decided).</td>
</tr>
<tr>
<td></td>
<td>Minimal delays in waiting for surgery and other treatments.</td>
<td>Stop treating cancer patients as ‘elective’ cases.</td>
</tr>
<tr>
<td></td>
<td>Earlier/ quicker appointments with oncology after surgery.</td>
<td>Provide more staff and communicate about delays and help people deal with the stresses of waiting.</td>
</tr>
</tbody>
</table>

**Experience-based surveys**—Most respondents had a very positive experience while attending the hospital and greatly appreciated the efforts of staff to make their experience as positive as possible (Figure 2). Comments such as “excellent service” and “staff were great” were common. However, about one in 10 patients had a ‘bad’
or ‘very bad’ experience, and these made a big difference to the average rating of the service.

Most negative experiences arose through:

- Increasing anxiety while waiting at any time, especially if staff were uninformative (the bigger problem) or impolite.
- Anxiety and pain during mammography, biopsy and clinic appointments, especially if staff were uninformative, rough or impolite, or if patient expectations of pain were not actively managed.

Further suggestions made as to ways of improving the service included:

- Better-written information about what the appointments are for and what patients can expect when they attend.
- Improved facilities ranging from layout the waiting rooms through to design of mammography gowns.
- Better communication about likely pain levels and how to minimise them.
- A clear explanation at the end of appointments about what will happen next and when.

**Figure 2. Patient ratings of journey experiences**

Co-design workshops—At the start of their journey, patients wanted answers to ‘big picture’ questions to orient themselves to the news of their cancer and to gain a picture of how the Breast Service would be helping them. They wanted reliable, relevant facts about cancer and the Breast Service. Many were too shocked to take in
detailed information and needed time to adjust to the news of cancer. At the start of treatment patients typically wanted information to help them understand what their possible future outcomes and their best treatment options were.

Recommendations focussed on ways of developing processes to improve communication, and included:

- Developing a staff communication guide for use by all clinical staff who have contact with patients
- Designing a patient held record
- Rationalising information given out within the DHB
- Developing a sectionalised/care diary.

Further workshops were held to develop these ideas.

Table 3. Key questions patients wanted answered during their breast cancer journey

<table>
<thead>
<tr>
<th>Question</th>
</tr>
</thead>
<tbody>
<tr>
<td>When will I know the radiology results? Who will tell me?</td>
</tr>
<tr>
<td>What sort of breast cancer do I have?</td>
</tr>
<tr>
<td>What is my prognosis?</td>
</tr>
<tr>
<td>How bad is my cancer and what are the treatment options?</td>
</tr>
<tr>
<td>Do I have to have treatment?</td>
</tr>
<tr>
<td>What is my best treatment option? Why?</td>
</tr>
<tr>
<td>How do I know that the treatment will be successful?</td>
</tr>
<tr>
<td>Will I get sick with my cancer treatment?</td>
</tr>
<tr>
<td>When will my appointment be?</td>
</tr>
<tr>
<td>Is there a chance of the cancer coming back? How will I know?</td>
</tr>
<tr>
<td>What can I do to lessen the chance?</td>
</tr>
<tr>
<td>How long do I have to live? What should my priorities be?</td>
</tr>
</tbody>
</table>

Emerging themes for the Breast Service—Each of the three tools yielded different information that was then analysed to identify key themes. Specific improvements were prioritised during a co-design workshop. There were four emerging themes.

- The provision of timely, accessible information was a key issue. Equally important was a way of managing the vast array of information that breast patients received. Patients were keen that the information they received was streamlined and that tools, e.g., folders and hand held records, were developed to help them manage the information.
The role of compassionate communication. It was important for patients that staff were able to communicate clearly and with compassion. Simple things such as smiling, introducing oneself and one’s role, explaining concepts accurately and being clear about what would happen next were vital for patients. The ability of staff to understand the patient experience was seen as fundamental.

The need for navigation and co-ordination. Patients wanted a person who could meet and greet them on arrival and help them navigate their way through the journey, both literally and metaphorically. There were various opinions as to who would be best suited to provide this role – patient buddies or a dedicated staff member – and the scope, i.e., whether it would extend to being a service co-ordination role.

A desire for a pleasant, easily navigable physical environment. This encompassed a wide range of issues ranging from getting a car park and finding the clinic through to the layout of the clinic and the design of the mammography gown.

Changes made—As stated earlier, amongst other things the project aimed to implement small, focussed changes. The improvements we made are listed in Table 4.

<table>
<thead>
<tr>
<th>A map of the patient journey</th>
<th>A strategic tool for staff allowing them to see the experiences over time. Provides a framework for evolving current and future improvements.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient information folder</td>
<td>A folder of information to help patients navigate their way through the Service.</td>
</tr>
<tr>
<td>Patient leaflets</td>
<td>A suite of seven new patient information leaflets. Enabled local information to be included and a constancy of supply of leaflets.</td>
</tr>
<tr>
<td>Patient held record</td>
<td>A double-sided card for patients to track their appointments. (Useful for staff too.)</td>
</tr>
<tr>
<td>Patient journey guide</td>
<td>A high level visual map of the journey. Staff can use this with patients to explain their journey and where they are in the process.</td>
</tr>
<tr>
<td>Communication guide</td>
<td>A poster in cartoon format with tips for patients and staff on how to communicate better.</td>
</tr>
<tr>
<td>Mammography gown</td>
<td>A gown, specifically designed to address usability problems for patients and staff was developed.</td>
</tr>
<tr>
<td>Co-design toolkit</td>
<td>Development of a co-design toolkit and website for healthcare services. The toolkit has 18 tools matching six key project phases.</td>
</tr>
</tbody>
</table>

Table 4. List of improvements

Discussion

Implementing co-design in healthcare can be a challenging endeavour, especially when clinical workloads are high and the organisational environment is fiscally constrained. However, the benefits of co-design, both in terms of increased staff understanding of patients’ experiences and improved experiences for patients, are potentially enormous.

Key learnings about the process:

- Use of co-design does not mean the abandonment of more standard, well-recognised quality improvement methodologies. Co-design can potentially
work well alongside Lean and Six Sigma methods (which focus on more measurable areas of service improvement such as prioritisation, queuing and adherence to guidelines) as long as one method does not subsume the other.

- Engage with patients early. Engagement is absolutely critical to true and successful co-design. Having patients involved early means that their experiences and requirements can be taken into account at the start of the process rather than people presuming to know what is required. In our project patients were invited to a workshop before decisions were made about its final scope and structure. Relationships with patients were developed and continued throughout the project to varying degrees. An important consideration was the acknowledgement that many patients were still receiving treatment during the project so energy levels and availability varied accordingly.

- Work hard to ensure a representative spread of patients. It is acknowledged that self-selecting patients may not be representative of the patient population more generally. Specific methods should be considered to target involvement across the patient spectrum.

- Staff buy-in is fundamental. Clinical, management and administrative staff are busy people, yet their involvement in co-design work is vital. Staff attendance at workshops with patients gives them a unique opportunity to understand patients’ experiences in a different way. In a supportive, workshop environment where staff and patients are equal, patients will often open up and share their perspectives in a way they would never do in the clinic room. The success of co-design work then, is greatly enhanced through communication with staff and their active participation.

- Be prepared to try things outside your comfort zone. Many elements of co-design involved trying new things and that required faith in the process. Early on a workshop was held with a wide variety of stakeholders including medical staff, patients, people interested in innovation and improvement specialists from external industries. Having extensive input early on in the project provided a richness of ideas from which to build a strong foundation. Use of service design tools, such as emotional journey mapping, involved learning new ways of thinking and challenging existing ways in which things have been done.

**Spreading the word**—As a result of the success of the project, co-design has been used within Melanoma Services at Waitemata DHB and more recently looking at advance care planning at Auckland DHB.

While co-design work has not yet become widespread in New Zealand, there is certainly a real interest in the method. The Ministry of Health funded the development of a health co-design toolkit and website www.healthcodesign.org.nz. Training sessions organised at Waitemata DHB proved popular, with a willingness and desire amongst staff to learn about and use co-design. In 2011, the Central Cancer Network facilitated a series of health service co-design workshops within their region, primarily aimed at a cancer control audience. The need for using a co-design approach
to develop supportive care strategies for adults with cancer in New Zealand has also been recognised.12

With ongoing funding and support, co-design could spread to other DHBs and health services throughout New Zealand. The use of co-design could initially be targeted to high need areas. For example, it could have particular benefit for services where there has been a lot of staff dissatisfaction or patient complaints, where there are high DNA (Did Not Attend) rates or where a new service or facility is being developed.

Recommendations:

- A limitation of this work that planned evaluation was not able to be undertaken on the effectiveness of the improvements, i.e., what (measurable) positive difference did the improvement have on patients’ experience of the service? We recommend that co-design projects factor in time and resources to allow an evaluation to take place after the improvements have been made and bedded down. Note that a co-design project identifies process and outcome criteria as part of designing improvements. These can then be used in evaluation work.

- Many organisations simply implement improvement initiatives without consideration of ongoing sustainability of their work. Organisations that are successful are the ones that can both implement and sustain improvement over time leading to increased quality and patient experience at lower cost. Co-design's emphasis on working with all stakeholders on an ongoing basis, and service users in particular, suggests it is implicitly more sustainable than conventional approaches. Further research in this area is recommended.

- The Patient Co-design of Breast Service Project not only made tangible improvements but it has demonstrated the potential value of engaging patients and focusing on their experiences. Although not formally evaluated, a limitation noted above, our work supports the findings of people who have adopted the method overseas: using co-design within the healthcare context is valuable and worthwhile. We recommend that DHBs and other health services in New Zealand recognise the value and benefits of co-design and consider adopting it as a key approach to service improvement.

Competing interests: None declared.

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(Note: Hilary Boyd, Bernie Mullin and Andrew Old all worked in the Healthcare Improvement Team at Waitemata District Health Board at the time this piece of work was undertaken.)

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References:


Appendix 1. A map of the patient journey
Doctors and the nurse endoscopist issue in New Zealand
Mohammad I Khan, Robert Khan, Wanda Owen

Abstract

Aim Training and recruitment of Nurse Endoscopists (NEs) is currently actively debated in medical circles. The aim of this survey was to obtain the views of doctors regarding the role of NEs in New Zealand (NZ).

Methods A web-based, self-administered questionnaire was sent to 84 endoscopists currently working in 25 public hospitals across all the 20 District Health Boards. The survey period was July 2011. Data was analysed using descriptive statistics.

Results The response rate was 47.5%. Fifty percent of the respondents worked in tertiary hospitals. Only 30% had a positive attitude towards the introduction of NEs in NZ. The majority (62%) believed that doctors would deliver better quality of endoscopy services than NEs. Only 37% thought that the introduction of NEs will reduce the cost of services. Forty-one percent thought it was inappropriate for the NEs to be enrolled in the Bowel Cancer Screening Programme and only 6 doctors (18%) thought that NEs should be allowed to perform therapeutic endoscopic procedures.

Conclusion Only a minority of doctors had a positive attitude towards the role of NEs. The majority considered doctors to deliver ‘higher’ quality of service and only a minority thought that the introduction of NEs will lower the cost of services.

The recent international trend of training and recruiting non-medical personnel (mainly nurses) in different medical fields has two main drivers. Firstly, there is a chronic shortage of doctors in certain specialities and secondly, health economics has recently been playing an increasingly important role in the healthcare industry. The possibility of cheaper healthcare provision in times of harsh budgetary constraints has attracted many supporters.

In New Zealand (NZ), within the field of Gastroenterology, nurses have taken up the role of Nurse Specialists and are running ‘Dyspepsia clinics’, Inflammatory Bowel diseases clinics’ and ‘Hepatitis Clinics’. However, unlike some of the other developed countries like the United Kingdom and the United States of America, NZ, so far, has no Nurse Endoscopist (NE).

NZ has an established shortage in the provision of colonoscopy services in public hospitals. Although, free endoscopy unit sessions are available they are not utilized because of a nationwide shortage of both trained endoscopy nursing staff and endoscopists. This shortage is likely to increase with the launch of the National Bowel Cancer Screening Programme. Yeoman and Parry have briefly mentioned in their survey that 25% of the NZ public hospitals (including only two of the main centres) will be willing to employ non-medical endoscopists but did not elaborate further on this topic.

The introduction of NEs in NZ can partly cover the capacity shortage of endoscopy services. However, there is no evidence in literature that the introduction of NEs can also save health dollars. The stepped up role of nurses as NEs has created a lot of
controversy, especially in medical circles. We carried out a survey of doctors to obtain their views on the role of NEs in NZ.

**Methods**

*Ethical approval* for the survey was taken from the Multi-region Ethics Committee. Twenty five public hospitals across all of the 20 District Health Boards in NZ were selected for the study. Small peripheral hospitals with only basic endoscopy facilities were excluded from the study. The survey was carried out in July, 2011. *The study population* included all of the Gastroenterologists currently working part time or full time in the selected public hospitals. In hospitals without gastroenterologists, the local surgeons providing the endoscopy services were included in the study. *Contact data* on the participants were obtained from each endoscopy unit. A postal letter with a web-link to the web-based survey (ss Appendix) was sent to each study participant. The letter included an introductory note and an explanation of the anonymous and confidential nature of the survey. *Descriptive statistics* were used to analyse the data.

**Results**

Eighty four study participants were identified. Two have since left their public jobs and were removed from the study. Forty doctors completed the survey (response rate of 47.5%). Eighty seven percent of the doctors were male, 50% were working in tertiary centres and 59% percent were practicing endoscopy both in public and private sector.

Sixty-two percent of the doctors thought that they will offer better quality of endoscopy services compared to trained NEs. Reasons included that endoscopy procedures are more than just a technical skill and findings need to be co-related to the clinical scenarios and that NEs will have a lower standard at trouble shooting. Thirty-two percent thought that there will be no difference in the quality of services if the NEs are properly trained (Figure 1).

**Figure 1. Clinical quality of endoscopy services**

<table>
<thead>
<tr>
<th>Your expectation of the clinical quality of services provided by the Nurse endoscopists compared to medically qualified endoscopists (doctors).</th>
<th>Response Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Better performance by the Nurse endoscopists</td>
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</tr>
<tr>
<td>No difference</td>
<td>12</td>
</tr>
<tr>
<td>Better performance by the doctors</td>
<td>23</td>
</tr>
</tbody>
</table>

Sixty-one percent of the doctors said that they expect no difference in patient experiences between the services offered by the doctors or NEs while the rest were almost evenly split between moderately better performance by the NEs and doctors (Figure 2).
Forty-seven percent of the doctors thought that there will be no impact on the running cost of endoscopy services while 37% thought that the overall cost will reduce with the introduction of NEs. Thirty-six percent thought the endoscopy practice for nurses should be limited to diagnostic upper endoscopy procedures only while 47% thought that it should be restricted to both upper and lower diagnostic endoscopies. The rest were in favour of full provision of both diagnostic and therapeutic upper and lower endoscopy procedures by the NEs (Figure 3).

Forty-three percent of the doctors thought that it was appropriate to enrol NEs in providing screening colonoscopies as part of the National Bowel Cancer Screening Programme. Forty-three percent thought it was not appropriate while the rest were not sure (Figure 4).
Sixty-seven percent of the doctors were willing to provide voluntary supervision if NEs were recruited in their department. Forty-five percent of the doctors said that they have a neutral attitude towards the introduction of NEs, 25% had a negative attitude and only 30% had a positive attitude (Figure 5).

Discussion

Worldwide, the role of NEs in the delivery of endoscopy services has probably caused more controversy than the role of any other Nurse Specialist in the field of Gastroenterology.

In some countries, like the UK and the US, NEs have expanded their role to both academic and district level hospitals. The span of procedures they are allowed to perform has also increased from simple diagnostic procedures to a wider array of both diagnostic and therapeutic procedures.

However, in other countries, like Australia and New Zealand, the role of NEs has not established at all. The reasons for these variations in the individual health care systems are unclear. Our survey shows that at present there is little enthusiasm among doctor for the role of NEs in NZ.
There have been many studies on the quality, safety and efficacy of endoscopies delivered by NEs. There is more data in literature for support of the role of NEs in upper gastrointestinal endoscopies and flexible sigmoidoscopies.\textsuperscript{5,6} Data from such studies have shown that NEs are comparable to doctors in terms of the quality, safety and efficacy of endoscopic procedures.

Our survey shows that majority of our doctors want the role of NE to be limited to the diagnostic upper and lower endoscopy. This may be because almost 62\% of the participants thought that, in terms of the quality of endoscopy services, the doctors will perform better than NEs and hence their reluctance to allow NEs the full scope of endoscopic practice. There is no robust literature to support this viewpoint.

A recent feasibility study by Koonstra et al showed that the learning curve of a nurse for training in colonoscopy is similar to that of a doctor trainee and involves 150 supervised colonoscopies.\textsuperscript{7} The colonoscopy procedure generally requires the patient to be sedated and again the evidence so far is that nurses are as good as doctors in supervising sedation.\textsuperscript{8} Dellon, Lippmann, Sandler, & Shaheen, have reported that procedures staffed by less-experienced gastrointestinal endoscopy nurses have increased odds of missing polyps.\textsuperscript{9} However, a different study of well trained NEs has reported a higher adenoma detection rate by the NEs.\textsuperscript{10}

Our survey shows that only 43\% of the doctors thought that it is appropriate to enrol NEs in the National Bowel Cancer Screening programme if they meet accredited standards. Forty-one percent were against it and the rest were not sure. This is less than the 2009 survey of US Gastroenterologist, where the majority were supportive of the role of NE in screening endoscopies.\textsuperscript{11}

In our survey majority of the doctors did not believe that endoscopy costs will decrease with the introduction of NEs. In their open comments they have pointed to two specific issues relevant to the health economics of introducing NEs. Firstly, there will be at least initially, a spike in cost as the training programme for NE is established. Secondly, NEs will still require supervision from doctors (and hence their time) even if they are fully accredited as is happening in other developed countries. In public hospitals, the reimbursement rate for the endoscopist is a small proportion of the overall cost of endoscopy. Therefore, the potentially lower reimbursement rate for NEs is unlikely to influence the overall cost of delivery of endoscopy services.

Richardson et al, in their MIINuEt study, conclude that endoscopy delivered by nurses are unlikely to be more cost effective than doctors.\textsuperscript{12} In their analysis, although endoscopies by doctors were more costly, patients in the doctors group also gained more Quality Adjusted Life Years (QALY) than those in the nurses group.

Potential areas of cost cutting in endoscopy services in certain countries include registered nurse-administered propofol sedation for endoscopy instead of anaesthesiologists.\textsuperscript{13} This, however, does not apply to NZ as conscious sedation is administered in NZ by the doctors performing the procedure and not by the anaesthesiologists.

Patient satisfaction with NEs has been studied in literature with patient satisfaction rates comparable or even better in some surveys, than those of doctors.\textsuperscript{14} Majority of the doctors (62\%) in our survey also thought that patient satisfaction rates will be the
same with NEs as for doctors. However, the same number of doctors also thought that the clinical quality of services will be better delivered by the doctors.

Only 28% of the doctors had a positive attitude towards the introduction of NE in the provision of endoscopy services. One of the objections was the lack of teaching slots for such trainees as they will have to compete with medical/surgical trainees for such positions. Another potential reason may be their perception that NEs will deliver inferior quality of endoscopy services compared to doctors, although, there is clear support for that in literature.

At present, few of the doctors in NZ have exposure to NEs. It will be interesting to observe that, if NEs are introduced in the NZ setting, whether a subsequent survey of doctors will show any change in their opinion. Health Work Force New Zealand (HWNZ) recommends facilitation of nurse specialization including training NEs to free up doctors to do high level procedures but acknowledge that significant barriers have to be overcome.15 HWNZ recommends close collaboration between the involved stakeholders to develop the role of NEs in NZ.

It is important to end the discussion on the potential limitations of the study. It was not possible to obtain an in-depth and wide ranging qualitative data from each respondent. The quantitative nature of the study and the use of survey strategy to obtain data precluded that. The questionnaire was deliberately kept short for a better response rate.

The survey was restricted to the public sector and mainly to the Gastroenterologists, except for the smaller public hospitals without the services of Gastroenterologists, where the surgeons providing the endoscopy services were included in the study. Also, because of the basic version of the survey tool used (Survey monkey) it was not possible to compare the opinions of the respondent Gastroenterologists with the respondent surgeons.

Competing interests: This survey was carried out as a part of a ‘Masters’ degree with the Massey University.

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References:

APPENDIX

1. Gender
   - [ ] Male
   - [ ] Female

2. Type of practice
   - [ ] Public
   - [ ] Private
   - [ ] Both

3. Place of practice
   - [ ] Tertiary centre
   - [ ] Non Tertiary centre
   - [ ] Other (please specify)

4. Your expectation of the clinical quality of services provided by the Nurse endoscopists compared to medically qualified endoscopists (doctors).
   - [ ] Better performance by the Nurse endoscopists
   - [ ] No difference
   - [ ] Better performance by the doctors

Any comment
5. Your expectation of the patient experiences of endoscopy services provided by the Nurse Endoscopist compared to medical endoscopists

- Substantially better performance by the Nurse endoscopist
- Moderately better performance by the Nurse endoscopist.
- No difference
- Moderately better performance by the Medical endoscopist
- Substantially better performance by the Medical endoscopist.

Any comment

6. The expected impact on cost upon introduction of Nurse Endoscopists to the endoscopy service

- Decrease in cost
- No effect
- Increase in cost

Any comment

7. In your opinion the appropriate procedures that could be delegated to Nurse endoscopist are

- Diagnostic upper endoscopy only
- Diagnostic and therapeutic upper endoscopy only
- Diagnostic upper and lower endoscopy only
- Diagnostic and therapeutic upper and lower endoscopy

Other (please specify)

Any comment
8. In your opinion, is it appropriate to enrol Nurse endoscopist in the National Bowel Cancer Screening programme.

☐ Yes
☐ No
☐ Not sure

Any comment

9. Your attitude towards the introduction of Nurse endoscopist

☐ Positive
☐ Neutral
☐ Negative

Any comment

10. Will you be willing to provide voluntary supervision for Nurse endoscopist if they are recruited in your department.

☐ Yes
☐ No

Any comment
Establishment of the New Zealand Drivers Study

John Langley, Dorothy Begg, Rebecca Brookland, Shanthi Ameratunga, Anna McDowell, John Broughton

Abstract

Aim Despite a significant improvement since graduated licensing was introduced, traffic related injury remains the leading cause of death and hospitalisation among young New Zealanders. The New Zealand Drivers Study (NZDS) was established with a view to providing information that would lead to an improvement in this situation. The NZDS is a prospective cohort study designed to explore the relationship between a comprehensive range of driving and traffic safety related factors and subsequent traffic crashes and convictions among newly licensed drivers. We describe key process objectives in establishing the cohort, and our success in meeting them and the implications arising thereof.

Methods We compare what occurred with what was proposed in the research protocol.

Results We successfully established a cohort of 3992 newly licensed car drivers with substantial heterogeneity in sociodemographic, behavioural, and driving experiences. We have 825 Māori that will allow us to undertake a separate Māori analyses. Response rates to interviews at the restricted and full licence stages have been very high at 87% and 93%, respectively. We have been successful via linkage in following them through the stages of licensure and via linkage obtaining national data on the outcomes of interest.

Conclusions The NZDS is well placed to make a significant contribution to our knowledge of young driver road safety behaviour. This process has already commenced.

Motor vehicle traffic crashes are the leading cause of mortality and morbidity among adolescents and young adults (15–25 years of age) in New Zealand and many other developed countries. In response to the high motor vehicle crash rate among young drivers in New Zealand a Graduated Driver Licensing System (GDLS) was introduced in 1987. The key elements of the GDLS are: a 6 months learner licence stage of supervised driving; a restricted licence stage of 18 months that allows unsupervised driving except at night-time (10pm–5am) or with young passengers in the car; a full licence stage with no restrictions. Further details of the New Zealand GDLS have been described elsewhere.

Despite a significant improvement since graduated licensing was introduced, traffic related injury remains the leading cause of death and hospitalisation among young New Zealanders, especially young Māori. Most New Zealand research on young drivers to date has used routinely collected crash data, such as the police traffic crash reports and the national hospital inpatient records, but has been limited in that these
databases do not, and realistically cannot, include the level of detail required to ensure that learner driver policy and programmes are based on sound scientific evidence applicable to young drivers in the current New Zealand context.\(^6\)

There have been a small number of studies examining the role of parents/caregivers in the learning to drive process. These studies have shown that high parental monitoring of driving by teens can reduce risky driving and crashes, especially during the high risk first few months of driving, and interventions designed to encourage parental monitoring have shown promise.\(^6\) None of these studies were undertaken in New Zealand. The lack of relevant information relevant to young drivers in New Zealand lead to the establishment of the New Zealand Driver’s Study (NZDS).

The NZDS is a prospective cohort study designed to explore the relationship between a comprehensive range of driving and traffic safety related factors and subsequent traffic crashes and convictions among newly licensed drivers. The study is designed to capitalise on the unique characteristics of New Zealand’s multistage graduated driver licensing system with the interview stages for collecting exposure data coinciding with the three testing stages of graduated driver licensing (Figure 1).

The protocol for this study has been described in detail elsewhere.\(^6\) Briefly, study participants are recruited at the learner licence stage and complete a self-administered baseline questionnaire. They are then followed-up at the restricted and full licence stage, by telephone interview. Official traffic crash and offence outcome data are obtained from the authorities that maintain these databases, and self-reported crashes are obtained at the restricted and full licence interview.

The study was established following a comprehensive pilot study which included: consultation with Māori; consultation with agencies involved in driver licensing; trialling procedures for recruitment; and developing, pre-testing and piloting questionnaires.\(^6,7\)

In this paper we describe:

- The goals and objectives of the study.
- Recruitment methods,
- Explanatory, exposure and outcome variables,
- Our success in following up the cohort, and
- Sociodemographic and behavioural characteristics of the cohort.

**Goals and objectives**—The goal of the New Zealand Drivers Study is to provide an evidence base that would facilitate efforts aimed at reducing traffic-related injury in New Zealand, and especially among the high-risk adolescent/young adult population. The primary objective of the study is to explore the relationship between a comprehensive range of driving and traffic safety related factors (e.g. driving experience, motivation for driving/licensing, driver training, alcohol use, risk-taking) and subsequent traffic crashes and convictions among newly licensed drivers, and from this identify specific areas that can be targeted to reduce traffic-related injury among this high-risk group.
The secondary objectives are to:

- Examine this relationship specifically for newly licensed Māori drivers,
- Evaluate the impact of current novice driver training programmes on driving-related outcomes, and
- Examine the role of parents/caregivers as supervisors of newly licensed drivers.

An overview of the study design is provided in Figure 1.

**Figure 1. Overview of study design**
Study population size—We aimed to recruit 5,000 newly licensed drivers of whom 1500 (minimum 1000) would be Māori, over a 12-month period. In reality we recruited 4282 over a 2-year period but 290 were ineligible (e.g. overseas licence conversions, truck licences, unsigned consent form) leaving a final cohort of 3992, of whom 825 self-identified as Māori.

Although the total cohort, and the number of Māori, were less than our original targets, the power calculations as given in the study protocol show that these numbers provide adequate statistical power to show effect sizes of around 1.5 to 2.0 for policy issues (e.g. full cohort) and 2.0 to 2.5 for programme issues (e.g. Māori cohort), which we considered satisfactory.

Recruitment methods—Our aim was to recruit participants from a diverse range of locations throughout NZ. During the pilot study recruitment was undertaken by the University research team. Given the differing needs of the local populations, we anticipated that different methods would be required in different regions.

In the larger urban areas, (Auckland, Christchurch, Dunedin), research assistants (RAs) (mostly postgraduate students) were employed part-time to undertake face-to-face recruitment at the local driver licensing centres. For example, in areas with a relatively high proportion of Māori, such as South Auckland (Manukau), and on the East Coast of the North Island (Hastings/Napier, Wairoa, Gisborne and Ruatoria) it was preferred that the recruitment be done by local Māori community groups. Six such groups, with whom we had consulted and established a collaborative partnership during the pilot, were subcontracted to undertake the recruitment among their local people.

All of these community groups had many years of experience delivering learner licence courses, and the source of participants for the NZDS. Five of these community/iwi groups were also subcontracted to undertake recruitment at their local driver licensing centre.

To boost the number of rural participants, postcards were distributed to all NZ Automobile Association (NZAA) licensing centres, except those in the large urban centres. On this postcard was an invitation for all newly licensed learner drivers to take part in the study, a mention of the $20 petrol voucher all participants would receive for each completed interview, and our contact details (email address, a free telephone number, and cell phone number) which they needed to contact if they wished to take part. This recruitment method was reasonably successful, and in particular helped boost the number of rural participants.

The licensing centres chosen for the recruitment were those that had issued the highest number of learner licences in the previous year. However, as there was no booking system for learner licence testing, there was a degree of uncertainty as to how many potential study participants would turn up at any centre, at any given time. Most of the recruitment took place at NZAA licensing centres, and although the recruitment took two years instead of one, the NZAA staff were very accommodating and
supportive of the research staff. This highlighted the importance of having established a good rapport with the NZAA head office personnel during the pilot study.

Recruitment commenced on 1 February 2006 and finished on 31 January 2008. Of the 3992 study participants, 2685 (67%) were recruited at a licensing centre, 916 (23%) at learner licence courses, and 391 (10%) by postcards. At the licensing centres, the recruitment rate across all research assistants was approximately 75% and for the learner licence courses it was around 90%. We were unable calculate a recruitment rate for postcards as we did not know the denominator.

Explanatory/exposure measures—Figure 1 provides an overview of the explanatory and the exposure measures, and the stage of the study when this information is collected.

Outcome measures—We intended that official data be sourced for traffic offences (any recorded breach of the Road Code), and injury crashes. We got participants consent to access crash and offence data at learner, restricted and full licence stage for up to 10 years after full licence. Our pilot work indicated a significant portion may not give consent for access to official data, especially for police records, and the period for which we sought it. In reality, this was not a problem. Of the 3992 study participants 98% gave consent to access all the listed traffic and injury records, 1% refused access to all records, and 1% denied access to some records.

For the 98% who gave consent, we have successfully accessed the traffic infringement and offence and convictions notices from the Driver Licence Registry (with assistance from the Department of Justice who hold these records, and not the NZ police as stated in the protocol) using the driver licence number. As at 31 December 2010, 1589 participants had at least one traffic offence, and there were 5751 offences in total.

We have also been successful in accessing crash information from the NZ Crash Analysis System (CAS), which is maintained by the Ministry of Transport. Crashes in which the study participant was a driver are identified using their driver licence number, and verified by personal data such as name and date of birth.

To identify crash records with no licence number name, date of birth, and address variables were used in the linkage. These variables are recorded on the bulk of the crash records. As at 31 December 2010 the participants had been involved in 143 police reported crashes.

Since official crash reports are known to have significant biases associated with them we also sought information from the participants about injury crashes. The intention is to use official and self-report information to derive a single outcome measure of crash experience.

Follow-up of the cohort—Central to the study design was the expectation that participants who had obtained their learner licence would be followed up when they got their restricted and full licences. Critical to achieving this was the establishment of a study specific database by the New Zealand Driver Licence Registry (DLR) that would allow us to track the participants’ progress as they moved through the licensing system. With the cooperation of the DLR we achieved this objective. Each week the DLR provide us with an updated electronic file of any changed records in the
database. A changed record indicates progression to the next licence stage, and the eligibility of the participant for the next licence stage interview.

Figure 2 is a snapshot in time of participants’ progress through the licensing stages and demonstrates that our tracking has been very successful. As at 30 April 2011, 74% had passed their restricted licence test and 87% of these completed the restricted licence interview, and 39% had passed the full licence test and 93% of them had completed the full licence interview.

Figure 2. Cohort progression as at 30 April 2011
After a minimum of 2.5 years had elapsed since obtaining a learner licence we conducted telephone interviews to ascertain reasons for non-progression to the restricted licence. Up to six attempts were made to contact non-progressors. As at 30 April 2011 we had attempted to contact all non-progressors. Of these 548 (46%) have been interviewed, 101 (8%) refused, and 540 (45%) could not be contacted.

**Sociodemographic and behavioural characteristics of the cohort**—One of the objectives of the recruitment process was to recruit a cohort that reflected the geographic and cultural diversity of the newly licensed driving population of New Zealand, and to recruit sufficient Māori to be able to conduct a separate analysis for Māori. As noted above, to achieve this, recruitment was undertaken in regions that represented both rural and urban areas, North and South Islands, in ethnically diverse communities, and included several regions with a relatively high proportion of Māori in the population.

Table 1 shows the sociodemographic characteristics of the study population at the learner licence stage. Gender was evenly distributed and, the majority were young with 77% aged 17 years or younger. The results show we successfully recruited an ethnically diverse cohort, with 825 (21%) and 516 (13%) identifying as Māori and Pacific origins, respectively. It should be noted that participants could identify with as many ethnic groups as they wished.

Table 1. Sociodemographic characteristics of the NZDS participants at learner licensing stage

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
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<tbody>
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<tr>
<td>Males</td>
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<td>49</td>
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<tr>
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</tr>
<tr>
<td>15 yrs</td>
<td>1975</td>
<td>49</td>
</tr>
<tr>
<td>16 yrs</td>
<td>712</td>
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<tr>
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<td><strong>Ethnicity</strong></td>
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<td>NZ European</td>
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<tr>
<td>Māori</td>
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<tr>
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<tr>
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</tr>
<tr>
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</tr>
<tr>
<td><strong>Place of residence</strong></td>
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<tr>
<td>Main urban areas</td>
<td>3557</td>
<td>89</td>
</tr>
<tr>
<td>Independent &amp; satellite urban areas</td>
<td>122</td>
<td>3</td>
</tr>
</tbody>
</table>
Rural areas with high or moderate urban influence 152 4
Rural areas with low urban influence 91 2
Highly rural/remote areas 70 2

**Main activity**
Secondary school student 2747 69
University or other student 405 10
Full-time, part-time employed 460 12
Homemaker 84 2
Unemployed 134 3
Other (includes missing) 162 4

**Deprivation**
Least deprived 1 488 12
2 414 10
3 383 10
4 374 9
5 323 8
6 340 9
7 318 8
8 317 8
9 400 10
Most deprived 10 635 16

*% Some totals many not equal 100 due to rounding
**More than one ethnicity could be recorded therefore total exceeds 3992

The residential address of each study participant was classified according to Statistics New Zealand “Urban/rural profile”. The majority (89%) lived in an urban location. The NZDep2006 score is a measure of socioeconomic deprivation created by combining nine variables, which reflect eight dimensions of deprivation, from the 2006 census. The scores in Table 1 show a reasonably even distribution (8%–10%) across all levels, except for the level of highest (16%) and lowest (12%) deprivation.

Table 2 shows a similar degree of diversity within behavioural factors. Scores for impulsivity/sensation seeking and aggression/hostility were derived using Zuckerman’s personality measure. Of note, there are substantial numbers at the extremes on measures of impulsivity/sensation seeking and aggression/hostility. The cohort also includes significant numbers of hazardous drinkers (measured by the AUDIT-C) and occasional and regular drug users. Also of significance is that half the cohort had driven (illegally) on a public road prior to obtaining their learner licence and approximately a quarter have either been in a crash themselves or know someone who had.
Table 2. Behavioural characteristics of the NZDS participants at learner licensing stage

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Impulsivity/Sensation Seeking</strong></td>
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<tr>
<td>low 0–4</td>
<td>625</td>
<td>17</td>
</tr>
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<td>5–9</td>
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<td>38</td>
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<tr>
<td>10–14</td>
<td>1352</td>
<td>36</td>
</tr>
<tr>
<td>high 15–19 (missing 221)</td>
<td>361</td>
<td>10</td>
</tr>
<tr>
<td><strong>Aggression/Hostility</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>low 0–2</td>
<td>487</td>
<td>13</td>
</tr>
<tr>
<td>3–5</td>
<td>907</td>
<td>24</td>
</tr>
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<td>6–8</td>
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<td>12–14</td>
<td>402</td>
<td>11</td>
</tr>
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<td>high 15–17 (missing 222)</td>
<td>78</td>
<td>2</td>
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<td><strong>Hazardous alcohol use</strong></td>
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<td></td>
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<tr>
<td>No</td>
<td>2452</td>
<td>66</td>
</tr>
<tr>
<td>Yes</td>
<td>1354</td>
<td>34</td>
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<tr>
<td>(missing 186)</td>
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</tr>
<tr>
<td><strong>Cannabis use</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>3437</td>
<td>89</td>
</tr>
<tr>
<td>Monthly or less</td>
<td>208</td>
<td>5</td>
</tr>
<tr>
<td>2–4 times a month</td>
<td>68</td>
<td>2</td>
</tr>
<tr>
<td>2–3 times a week</td>
<td>51</td>
<td>1</td>
</tr>
<tr>
<td>4 or more times a week</td>
<td>97</td>
<td>3</td>
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<tr>
<td><strong>Recreational drug use</strong></td>
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<td></td>
</tr>
<tr>
<td>Never</td>
<td>3796</td>
<td>99</td>
</tr>
<tr>
<td>Monthly or less</td>
<td>37</td>
<td>1</td>
</tr>
<tr>
<td>2–4 times a month</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>2–3 times a week</td>
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<tr>
<td>4 or more times a week</td>
<td>8</td>
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<td></td>
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<tr>
<td><strong>Herbal highs/party pills</strong></td>
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<td></td>
</tr>
<tr>
<td>Never</td>
<td>3615</td>
<td>94</td>
</tr>
<tr>
<td>Monthly or less</td>
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</tr>
<tr>
<td>2–4 times a month</td>
<td>38</td>
<td>1</td>
</tr>
<tr>
<td>2–3 times a week</td>
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<tr>
<td>4 or more times a week</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>(missing 141)</td>
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<td></td>
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<tr>
<td><strong>Trouble staying awake</strong></td>
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</tr>
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Figure 3. Parent recruitment

<table>
<thead>
<tr>
<th>Frequency of Drink and Drive</th>
<th>Count</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>not during the last month</td>
<td>2954</td>
<td>81</td>
</tr>
<tr>
<td>less than once a week</td>
<td>446</td>
<td>12</td>
</tr>
<tr>
<td>once or twice a week</td>
<td>184</td>
<td>5</td>
</tr>
<tr>
<td>three or more times a week</td>
<td>75</td>
<td>2</td>
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</table>

<table>
<thead>
<tr>
<th>Unlicensed driving-public road</th>
<th>Count</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>no</td>
<td>1935</td>
<td>51</td>
</tr>
<tr>
<td>yes</td>
<td>1870</td>
<td>49</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Crash experience</th>
<th>Count</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>no</td>
<td>2775</td>
<td>74</td>
</tr>
<tr>
<td>yes</td>
<td>1000</td>
<td>26</td>
</tr>
</tbody>
</table>

Stage 1

3992 NZDS Cohort

Stage 2

3099 Aged 15 - 17 years at learner licence stage

893 Aged 18 years or older at learner licence stage

1594 Passed restricted test by August 2008

1435 Restricted licence stage interview completed

159 Refused Unable to contact

1405 Parent contact details

30 Excluded (e.g., no parent contact details)

1200 (85%) Parent interviews complete

205 Parent refusal or Unable to contact

Parent Interviews
Parents’ study—One objective of this study was to examine the role of parents/caregivers as supervisors of newly licensed drivers. As planned a parent or caregiver was eligible for the parents’ study if their child:

- Was aged 15–17 years when they passed their learner licence, and
- Passed their restricted licence test, and
- Completed their first follow up interview (i.e. restricted licence interview)

In the protocol we proposed to recruit the first 1000 eligible parents. This, however, ran the risk of recruitment bias as 15–17 year olds recruited in the latter part of the recruitment phase would have had insufficient time to progress to the restricted licence stage, and therefore their parents would not be eligible for the study. Accordingly recruitment for the parent study continued until August 2008, seven months after the NZDS cohort recruitment phase finished. This meant that every 15–17 year old held their learner licence for the minimum length of time (6 months), and thus could progress to the restricted licence stage, should they have chosen to.

Figure 3 shows the numbers that met the successive recruitment criteria. In all, 85% of the parents invited to take part in the study agreed to do so, resulting in 1200 parent participants (773 mothers, 427 fathers).

Discussion

Cohort studies provides excellent means of providing insight into the factors associated with adverse outcomes as young drivers commence their driving careers. There are major challenges to mounting such a study, nevertheless we have been successful in mounting such study, albeit with some limitations.

While the study population size was less than we had planned for, as indicated above, it has not seriously compromised the statistical power of the study. It was particularly pleasing that we were able to achieve sufficient numbers of Māori to undertake a separate Māori analyses. This is important for the credibility of the study in the New Zealand context. Internationally, given the dearth of injury epidemiology which is focused on indigenous populations we are well placed to add significantly to knowledge in this area. This process has already commenced with an examination of unlicensed driving among Māori and another on attitudes and opinions of newly licensed drivers.

The greatest challenge to the study to date has been the recruitment. By extending the recruitment period to two years instead of one, we were able to achieve an adequate number of study participants. This was especially important for Māori, as an analysis solely for Māori, was a specific objective of this study. The Māori community/iwi groups that undertook the recruitment must be given credit, for making this possible. Our experiences confirm those of others who have shown community engagement and local assistance is critical to successful recruitment of indigenous people.

Critical to the success of this study is our ability to track outcomes for all participants, via official data sources. Our very high levels of consent to access outcome data (traffic crashes, infringements and offences) during the licensing years, and for ten
years after obtaining a full licence, means we can access outcomes for 98% of the cohort, on an ongoing basis. Also, as we are able to link the official outcome data to our study data using driver’s licence numbers, because the majority of crashes do have the driver licence number recorded on the file, we are confident we can access complete data. The risk of bias in terms of those we do not have outcomes information for is minimal.

A major threat to validity of the findings of a cohort study such as this is attrition bias due to failure to monitor some participants through the licensing stages. Figure 2 shows that we have been very successful in tracing participants and subsequently obtaining follow-up information from them. It should also be noted that the response rates to interviews at the restricted and full licence stages have been very high at 87% and 93%, respectively. Failure to follow-up at the restricted licence stage should not be interpreted as precluding follow-up at the full licence stage.

Currently, we have successfully followed up at the full licence stage two thirds of those we failed to follow-up at the restricted stage. In this context it is important to note that we have followed-up approximately half of those who have failed to progress from the learner to the restricted licence stage, to ascertain their reasons for failing to progress through the graduated licensing system and to obtain information on driving behaviour comparable to that obtained from those who have progressed to the restricted licence stage.

The characteristics of the study population showed substantial heterogeneity in demographic characteristics at the learner licensing stage. For example, the range of ages potentially allows us to determine the independent contributions of age (maturity) and driving experience to crash risk. The relatively small number of participants from rural settings will restrict our ability to determine rurality as a factor for various outcomes. Aside from the sociodemographic factors, the results show we have heterogeneity in terms of impulsivity/sensation seeking, aggression, alcohol and other drug use, and road safety experience. Of particular note is that we have a significant number of the cohort who had been driving (illegally) on public roads prior to obtaining their learner licence.

Our results also show high response rates, and thus low risk of bias, for all the items in Table 2. Even the more sensitive issues, such as drug use, had less than 4% who did not complete the item.

The recruitment for the parent study was successful with 85% of parents agreeing to participate. This is a unique feature of this cohort study of young drivers and will enable us to determine how central parents are in how young drivers progress, or do not, through the graduated licensing system.

A limitation of this study is that we have not recruited a representative cohort. This means that the prevalences we report cannot be considered to represent young drivers in New Zealand. This decision was deliberate since recruitment of representative cohort would have required us to have a far larger cohort to ensure, for example, we had sufficient Māori to allow separate analyses. This would have been very expensive and not an efficient means of addressing our primary objectives. Irrespective we considered the recruitment of a representative cohort would have been extremely difficult, if not impossible, given high refusal rates for certain sub-populations of
young drivers. As has been demonstrated by Dunedin Multidisciplinary Health and Development Study non-representative cohorts have the potential to make a substantial contribution to our knowledge.

Although analyses addressing the primary objectives of the study have only recently commenced other results from the NZDS are achieving our main aim, that is to influence young driver policy in New Zealand. Results from the NZDS have been used by the Ministry of Transport in the development of New Zealand’s young driver policy\textsuperscript{f}, and learner driver policy currently under development.

Results from the parents’ study have been requested by the New Zealand Transport Agency to assist in the development of a young driver programme for parents. We are also currently preparing a number of papers of direct relevant to policy. For example, the government recently indicated that as one of its supporting actions to increase the safety of young drivers it intended to investigate the introduction of maximum licence time limits for learner and restricted licences.\textsuperscript{19}

Despite the fact that staged progression is central to graduated licensing schemes there is limited information on the sociodemographic and behavioural characteristics which are associated with non-progression, whether non-progression results in negative traffic outcomes, and why non-progressor choose not to progress. We have recently addressed this issue and the findings have been forwarded to the Ministry of Transport.\textsuperscript{20}

**Conclusion**

A significant investment was made in pretesting and piloting for the NZDS. This has paid off as there have only been minor variations to the protocol for the main study. Moreover the study population displays significant variation in range of factors at the learner licensing stage. The NZDS study has already made a contribution to the development of young driver policy and, as has been demonstrated here with the example of non-progressors, it is well placed to significantly increase that contribution.

**Competing interests:** None declared.

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References:


Christchurch earthquakes: how did former refugees cope?
Mohamud Osman, Andrew Hornblow, Sandy Macleod, Pat Coope

Abstract

Aim This study investigated how former refugees now living in Christchurch (Canterbury Province, New Zealand) communities coped after the 4 September 2010 and subsequent earthquakes.

Method A systematic sample of one in three former refugees from five ethnic groupings (Afghanistan, Kurdistan, Ethiopia, Somalia and Bhutan) was selected from a list of 317 refugees provided by the Canterbury Refugee Council and invited to participate in the study. Seventy-two out of 105 potential participants completed a 26 item questionnaire regarding the impact of the quakes, their concerns and anxieties, coping strategies and social supports. The methodology was complicated by ongoing aftershocks, particularly that of 22 February 2011.

Results Three-quarters of participants reported that they had coped well, spirituality and religious practice being an important support for many, despite less then 20% receiving support from mainstream agencies. Most participants (72%) had not experienced a traumatic event or natural disaster before. Older participants and married couples with children were more likely to worry about the earthquakes and their impact than single individuals. There was a significant difference in the level of anxiety between males and females. Those who completed the questionnaire after the 22 February 2011 quake were more worried overall than those interviewed before this.

Conclusion Overall, the former refugees reported they had coped well despite most of them not experiencing an earthquake before and few receiving support from statutory relief agencies. More engagement from local services is needed in order to build trust and cooperation between the refugee and local communities.

On 4 September 2010, at 4:35am local New Zealand time, the city of Christchurch experienced an earthquake of magnitude 7.1 on the Richter scale. The epicentre of the quake was 40 km west of Christchurch at a depth of 11 km.1 Many residents suffered serious damage to their property, with thousands of homes temporarily or permanently uninhabitable, but miraculously no fatalities were reported. After the September 4 earthquake, there were regular ongoing aftershocks which ranged between magnitudes 2 and 5 on the Richter scale.

On 22 February at 12:51pm the city was devastated by a second major quake, measured at 6.3 on the Richter scale; the epicentre was 10 km south-east of Christchurch at a depth of just 5 km.1 This resulted in widespread further destruction to property, including the destruction of much of the central business district, and a final death-toll of 181, making it the second-deadliest natural disaster recorded in New Zealand.2
Earthquakes may cause profound emotional and psychological trauma to thousands of people. Livanou et al. investigated the level of post-traumatic stress disorder (PTSD) in 157 Greek survivors of the 1999 Parnitha earthquake in a 4-year follow-up study. They concluded that there is an association between exposure to a traumatic event such as an earthquake and the development of PTSD.

Maldonado et al. conducted a longitudinal survey in Guadalajara, Mexico, to examine the factors associated with acute stress reaction. Their results showed that exposure to traumatic events such as an earthquake increases the risk of developing anxiety-spectrum disorder.

Refugee communities are part of the spectrum of Christchurch residents affected by the quakes. The definition of “refugee” according to the United Nations Refugee Convention, 1951 is:

“any person who, owing to well-founded fear of being persecuted for reasons of race, religion, nationality, membership of a particular social group or political opinion, is outside the country of his nationality and is unable or, owing to such fear, is unwilling to avail himself of the protection of that country; or who, not having a nationality and being outside the country of his former habitual residence as a result of such events, is unable or, owing to such fear, is unwilling to return to it.”

New Zealand has had a long-established custom of welcoming refugees from around the world. Refugees can come to New Zealand as asylum-seekers, through the United Nations High Commissioner for Refugees (UNHCR) mandated quota programme, humanitarian migrant intake or the family reunification programme. There is an existing body of research which has consistently documented that refugees are more likely to experience PTSD due to past traumatic events or political violence. There is also evidence of the resilience of former refugees.

The 4 September 2010 and 22 February 2011 Christchurch earthquakes and subsequent continuing aftershocks have had a significant impact on the Christchurch population as a whole, but, arguably, particularly on refugee communities whose location, circumstances or past history have made them more vulnerable.

In this study we investigated how the refugee communities responded to and coped with the 4 September and subsequent earthquakes.

Method
A total of 105 former refugees aged over 18 years, who were living in Christchurch at the time of the 4 September 2010 earthquake, were systematically selected every 1 in 3 from a list of 317 refugees provided by the Canterbury Refugee Council. The participants were drawn from five ethnic and geographic groups of former refugees, representing major and different communities: Afghanistan, Kurdistan, Ethiopia, Somalia and Bhutan. It may be noted that earthquakes are very rare in the Horn of Africa, though not infrequent in Afghanistan, Kurdistan and Bhutan.

Former refugees belonging to any of these groups were eligible for inclusion in the study provided they were resident in Christchurch at the time of and following the 4 September 2010 quake. Potential participants who had been selected for the study were contacted individually and invited to take part by completing a questionnaire regarding their experience of the September 4 earthquake and aftershocks, and how they had coped. All those contacted agreed to participate and so, after obtaining verbal consent, information sheets and consent forms were posted. Each interview lasted not more than one hour. All interviews were conducted by the first author, who was himself from the Somali refugee community, and, for the subjects comfort, conducted in their home.
Interviews were structured, with a 26-item questionnaire which included questions on the participant’s experience of and response to the quakes, their coping processes and level of support, past experience, and demographic information. The questionnaire used a 5-point Likert scale (1 = ‘not at all’ and 5 = ‘extremely’).

After the 22 February 2011 quake, and part way through the study, just under one-third (N=33) of the sample evacuated the city and were lost to the study before being interviewed.

All statistical analyses were done using statistical (SPSS) software; significance being determined from use of the Chi-squared ($\chi^2$) and Mann Whitney U tests at a 5% significance level.

Ethical approval for the project was given by the University of Canterbury Human Ethics Committee.

Results

After the devastating 22 February 2011 earthquake, and part way through the study, just under one-third (N=33) of the sample evacuated the city and were lost to the study. Participants lost to the study were mostly from Kurdistan (N=16) and Afghanistan (N=13); most were male and employed, and all were in the age range 25–39 years.

This loss of participants may have biased our results in that it is plausible that those leaving Christchurch may have been more severely affected than those remaining. However it was reported in the media that about a third of the residents of the city departed at this time. Most have since returned. As the circumstances in which the participants fled could not be controlled the best option we had was to continue interviewing those who wished to stay and take part in the study.

Seventy-two participants (69%) out of 105 completed interviews; 40% from Somalia, 19% from Bhutan, 14% from Ethiopia and Kurdistan, and 13% from Afghanistan. Table 1 shows some demographic characteristics of the participants broken down by their country of origin.

Table 1. Demographic characteristics of 72 Canterbury former refugees exposed to the 4 September 2010 and 22 February 2011 earthquakes

<table>
<thead>
<tr>
<th>Variables</th>
<th>Afghanistan (%) (n=9)</th>
<th>Kurdistan (%) (n=10)</th>
<th>Ethiopia (%) (n=10)</th>
<th>Somalia (%) (n=29)</th>
<th>Bhutan (%) (n=14)</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>13 (n=9)</td>
<td>14 (n=10)</td>
<td>14 (n=10)</td>
<td>40 (n=29)</td>
<td>19 (n=14)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>89</td>
<td>80</td>
<td>50</td>
<td>41</td>
<td>43</td>
<td>55</td>
</tr>
<tr>
<td>Female</td>
<td>11</td>
<td>20</td>
<td>50</td>
<td>59</td>
<td>57</td>
<td>45</td>
</tr>
<tr>
<td>Status</td>
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<tr>
<td>Married</td>
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<td>50</td>
<td>40</td>
<td>90</td>
<td>64</td>
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<td>Single</td>
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<td>50</td>
<td>60</td>
<td>10</td>
<td>36</td>
<td>33</td>
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<td></td>
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<td>10</td>
<td>40</td>
<td>45</td>
<td>7</td>
<td>26</td>
</tr>
<tr>
<td>Employed</td>
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<td>50</td>
<td>10</td>
<td>31</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>*Student</td>
<td>67</td>
<td>40</td>
<td>50</td>
<td>24</td>
<td>93</td>
<td>44</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18–24</td>
<td>67</td>
<td>40</td>
<td>10</td>
<td>14</td>
<td>29</td>
<td>26</td>
</tr>
<tr>
<td>25–39</td>
<td>0</td>
<td>20</td>
<td>80</td>
<td>38</td>
<td>21</td>
<td>33</td>
</tr>
<tr>
<td>40+</td>
<td>33</td>
<td>40</td>
<td>10</td>
<td>48</td>
<td>50</td>
<td>40</td>
</tr>
</tbody>
</table>

* Students are classified as being those in schools, tertiary education or language centres.
Table 2 compares how the former refugees responded to questions on to their level of worry, fear, coping, damage experienced, access to information and help received following the Canterbury earthquakes. More than 85% of Kurdish, Bhutanese, Ethiopian and Somali refugees were very worried after experiencing constant aftershocks, whereas Afghans were less worried (33%).

Across the ethnic groups, the only significant differences related to worry ($\chi^2=16.734$, df=4, $p=0.02$), feelings of helplessness ($\chi^2=28.859$, df=4, $p=0.025$), and disturbing thoughts or images about the earthquakes ($\chi^2=32.973$, df=4, $p=0.007$). Afghan participants, predominantly young single males, were less worried and had fewer feelings of helplessness than other ethnic groups. Participants from Bhutan, Ethiopia and Somalia had more disturbing thoughts than those from Afghanistan and Kurdistan.

Analyses by marital status and gender, using a cut-off score of 3, indicated that married participants with children were more likely to suffer from high levels of worry and anxiety than participants who were single (U=392.5, $p=0.012$), and more women were highly anxious than men (73% and 39% respectively, U=396, $p=0.002$).

Worry levels varied with age as the younger participants aged 18-24 were relatively less worried compared to older participants 40+ (58% vs 96%, $\chi^2=20.9$, $p=0.007$). Additionally, in term of occupation and experiencing fear, students reported having a higher level of fear compared to employed and unemployed participants (88% vs 56% and 54% respectively) though the differences were not statistically significant ($\chi^2=9.2$, $p=0.34$).

When we assessed the impact of the earthquakes on the remembering of past traumatic experiences, also how well participants were prepared, we found that 72% of participants had never been exposed to traumatic events or natural disasters before, nor had they any emergency supplies for natural disasters. In addition, the majority of Somali (83%) and Afghani (67%) participants used spirituality and religious practices as a form of coping mechanism post earthquake experience, these coping mechanisms also being important, tho’ to a lesser degree, for Ethiopian (47%), Kurdish (43%) and Bhutanese (21%) participants.

Twenty-nine participants were interviewed after the second major earthquake on 22 February 2011 and so we were able to compare their responses with those of the 43 participants interviewed prior to that earthquake (Table 3). Differences between the two groups were not statistically significant, except on the fear question. When participants were asked what has been their biggest fear 83% feared death and had concerns for their family safety after experiencing the 4 September 2010 earthquake, 100% after the 22 February earthquake (U=414, $p=0.016$).

Despite the worry about aftershocks, when participants were asked to score their level of coping from 1–5 (1=not at all and 5=extremely), over three-quarters of all participants scored 3 or more. Over 80% of all participants did not receive help or support from the City Council or Earthquake Commission, and over two-thirds reported difficulty in accessing help and information.
Table 2. Responses of 72 former refugees exposed to the Canterbury earthquakes

<table>
<thead>
<tr>
<th>Variables</th>
<th>Afghanistan (%)</th>
<th>Kurdistan (%)</th>
<th>Ethiopia (%)</th>
<th>Somalia (%)</th>
<th>Bhutan (%)</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continually worry about aftershocks</td>
<td>33</td>
<td>90</td>
<td>90</td>
<td>86</td>
<td>93</td>
<td>82</td>
</tr>
<tr>
<td>Support from external agencies</td>
<td>11</td>
<td>20</td>
<td>10</td>
<td>21</td>
<td>14</td>
<td>17</td>
</tr>
<tr>
<td>Fear for family safety or death</td>
<td>75</td>
<td>80</td>
<td>90</td>
<td>93</td>
<td>93</td>
<td>90</td>
</tr>
<tr>
<td>How well family coped</td>
<td>100</td>
<td>60</td>
<td>80</td>
<td>76</td>
<td>71</td>
<td>80</td>
</tr>
<tr>
<td>*Damage to house/ properties</td>
<td>22</td>
<td>30</td>
<td>30</td>
<td>55</td>
<td>79</td>
<td>49</td>
</tr>
<tr>
<td>Feeling hyper-vigilant</td>
<td>89</td>
<td>80</td>
<td>80</td>
<td>79</td>
<td>93</td>
<td>84</td>
</tr>
<tr>
<td>Feeling helpless</td>
<td>67</td>
<td>80</td>
<td>80</td>
<td>90</td>
<td>100</td>
<td>86</td>
</tr>
<tr>
<td>*Avoid people/places</td>
<td>11</td>
<td>0</td>
<td>10</td>
<td>28</td>
<td>43</td>
<td>22</td>
</tr>
<tr>
<td>*Disturbing images about earthquakes and aftershocks</td>
<td>56</td>
<td>30</td>
<td>70</td>
<td>75</td>
<td>71</td>
<td>72</td>
</tr>
<tr>
<td>Difficulty accessing help/information</td>
<td>100</td>
<td>60</td>
<td>70</td>
<td>76</td>
<td>57</td>
<td>72</td>
</tr>
</tbody>
</table>

**Note:** All percentages are for responses scored 3 or more on the 1-5 scale, except those marked by an asterisk which indicates yes/no questions.
Table 3. Percentage of responses comparing former refugees interviewed before and after the 22 February earthquake

<table>
<thead>
<tr>
<th>Variables</th>
<th>Interviewed before 22 February (n=43)</th>
<th>Interviewed after 22 February (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continually worry about aftershocks</td>
<td>79</td>
<td>86</td>
</tr>
<tr>
<td>Support from external agencies</td>
<td>16</td>
<td>17</td>
</tr>
<tr>
<td>Fear for family safety or death</td>
<td>83</td>
<td>100</td>
</tr>
<tr>
<td>How well family coped</td>
<td>77</td>
<td>76</td>
</tr>
<tr>
<td>*Damage to house/properties</td>
<td>49</td>
<td>48</td>
</tr>
<tr>
<td>Feeling hyper-vigilant</td>
<td>79</td>
<td>90</td>
</tr>
<tr>
<td>Feeling helpless</td>
<td>84</td>
<td>90</td>
</tr>
<tr>
<td>*Avoid people/places</td>
<td>23</td>
<td>21</td>
</tr>
<tr>
<td>*Disturbing images about earthquakes and aftershocks</td>
<td>74</td>
<td>66</td>
</tr>
<tr>
<td>Difficulty accessing help/information</td>
<td>68</td>
<td>79</td>
</tr>
</tbody>
</table>

Note: All percentages are for responses scored 3 or more on the 1–5 scale, except those marked by an asterisk which indicates yes/no questions.

Discussion

The 4 September 2010 and 22 February 2011 earthquakes and subsequent aftershocks have had a significant impact on the Christchurch population as a whole, including on refugee communities whose location, circumstances and past history has arguably made them more vulnerable.

The dead and injured in the 22 February earthquake included members of the close-knit refugee communities, adding to the overwhelming feeling of the earthquakes and aftershocks as a devastating and ongoing experience generating high levels of worry and anxiety, challenging personal resilience and coping resources.

The survey was designed for five former refugee groups in Christchurch, chosen because they represented the majority of the refugee population in the region. The aim initially was to investigate how they coped after the 4 September earthquake, assessing the level of anxiety across the groups, whether their experience of the earthquake and subsequent aftershocks reminded them of past traumatic experience and how supportive the local services were.

The Somali, Afghani, Bhutanese, Kurdish and Ethiopian participants were systematically selected from the refugee contact list provided by the Canterbury Refugee Council to reduce the effect of selection bias. Following the 22 February 2011 earthquake 30% of the participants excluded themselves from the study as they left Christchurch and could not be traced. However the initial sample size of 105 was adequate, though the impact on the results of the loss of 33 potential participants is unknown.
The circumstances in which the participants fled the city could not be controlled and the best option we had was to continue interviewing those who wished to stay and take part in the study.

Distressing and ongoing worry and anxiety, hyper-vigilance in expectation of further aftershocks, feelings of helplessness, disturbing earthquake-related thoughts and images, and fear of further earthquake trauma were the norm across all ethnic groups in the study.

The Afghani participants were the least anxious compared to the other ethnic groups, perhaps because over two-thirds were young and single and our results have shown that younger participants without family responsibilities were less worried than older married participants. Also, earthquakes occur occasionally in Afghanistan, and thus this population has prior earthquake knowledge and experience.

Married participants with children were more anxious than single participants, and females were significantly more anxious than males. Other studies indicate that females are more likely to experience anxiety following earthquakes than males\(^8,10\) and a May 2011 Christchurch media report also indicated males to be less worried than females, 55% compared to 71\(^\%\)\(^{11}\). Other research reports parents as being more psychologically affected by earthquakes\(^12\).

In terms of occupation and experiencing fear, while our study suggested that students were more likely to experience fear compared to employed and unemployed participants, these differences were not statistically significant. Whether or not students participating in our study were personally affected by the deaths of a group of students in the 22 February earthquake is unknown.

The possibility that the Christchurch earthquakes might remind participants of past trauma or distressing experiences was considered in the development of our survey. It is noteworthy that 72\(^\%\) of participants in our study reported having no prior experience of a traumatic event or natural disaster, and responses to open ended questions in our study indicated that the Christchurch earthquakes did not reactivated memories of earlier experiences.

Three quarters of participants reported coping either satisfactorily or well after both the 4 September 2010 and 22 February earthquakes, this being attributed by many of the participants to their strong cultural beliefs and spiritual practices. Religious and spiritual beliefs have been identified as an active form of coping which decreases the level of stress and improves the acceptance of challenging situations\(^13\). Whatever the mechanisms of psychological and social support, the high level of coping reported is a tribute to the resilience of the refugee communities.

A limitation of this study was the lack of a control group which could compare support of refugee and non refugee communities. Nevertheless, access to appropriate support was a major issue for participants. The majority of the participants (80\%) did not receive support from local government or the Earthquake Commission, and it took some time for them to access help, two-thirds having difficulty doing so.

The low support from mainstream agencies could be an added factor influencing the level of anxiety among the refugee communities. The language barrier could also be
an issue, as some refugees are not confident enough to call for help, relying on family and friends for support when difficulties or crises arise.

The issue of barriers to access to care has been raised in previous New Zealand research,\textsuperscript{14,15} and the resourcing of health sector responsiveness to the needs of refugees resettled in New Zealand has been highlighted in a recent policy review.\textsuperscript{16} The apparent difficulty in accessing information and help which the participants in our study experienced is cause for concern.

In New Zealand’s increasingly diverse society, and particularly in circumstances such as the recent Christchurch earthquakes, more engagement by both national and local services is needed to build trust and cooperation between the communities of former refugees, also other ethnic minority groups, which are an increasingly significant part of our wider community.

Competing interests: None declared.

Author information: Mohamud Osman, Graduate Student, Andrew Hornblow, Adjunct Professor; Sandy Macleod, Adjunct Associate Professor; Pat Coope, Statistical Advisor; Health Sciences Centre, University of Canterbury, Christchurch

Acknowledgements: We are particularly grateful to those former refugees who participated in this study and thank them sincerely for their patience and responsiveness; we are also grateful for the support of their communities. Special appreciation is extended to Mr Ahmed Tani, Chairman of Canterbury Refugee Council, for his initiation of and contribution to this project. We are grateful also to Partnership Health Canterbury (PHO) for encouragement and support throughout the study and funding of a summer studentship for the first author.

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References:


New Zealand 2012 guidelines for the management of non ST-elevation acute coronary syndromes

Non ST-Elevation Acute Coronary Syndrome Guidelines Group and the New Zealand Branch of the Cardiac Society of Australia and New Zealand

(see Appendix 1 for author names)

Glossary

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACC</td>
<td>American College of Cardiology</td>
</tr>
<tr>
<td>ACE</td>
<td>Angiotensin converting enzyme</td>
</tr>
<tr>
<td>ACS</td>
<td>Acute coronary syndromes</td>
</tr>
<tr>
<td>ACUITY</td>
<td>Acute Catheterisation and Urgent Intervention Triage strategY</td>
</tr>
<tr>
<td>AHA</td>
<td>American Heart Association</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin-receptor blocker</td>
</tr>
<tr>
<td>BNP</td>
<td>Brain natriuretic peptide</td>
</tr>
<tr>
<td>CAD</td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>CABG</td>
<td>Coronary artery bypass grafting</td>
</tr>
<tr>
<td>CAPRIE</td>
<td>Clopidogrel versus Aspirin in Patients at Risk of Ischaemic Events</td>
</tr>
<tr>
<td>CREDO</td>
<td>Clopidogrel for the Reduction of Events During Observation</td>
</tr>
<tr>
<td>CURE</td>
<td>Clopidogrel in Unstable Angina to Prevent Recurrent Events</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>ESC</td>
<td>European Society of Cardiology</td>
</tr>
<tr>
<td>FRISC-II</td>
<td>Fragmin and fast Revascularisation during In Stability in Coronary artery disease</td>
</tr>
<tr>
<td>hsCRP</td>
<td>High sensitivity C-reactive protein</td>
</tr>
<tr>
<td>hsTNT</td>
<td>High sensitivity Troponin T</td>
</tr>
<tr>
<td>HPS</td>
<td>Heart Protection Study</td>
</tr>
<tr>
<td>ICTUS</td>
<td>Invasive versus conservative treatment in unstable coronary syndromes</td>
</tr>
<tr>
<td>IHD</td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td>ISAR</td>
<td>Intracoronary Stenting and Antiithrombotic Regimen trials</td>
</tr>
<tr>
<td>LDLc</td>
<td>Low Density Lipoprotein Cholesterol</td>
</tr>
<tr>
<td>LMWH</td>
<td>Low-molecular weight-heparin</td>
</tr>
<tr>
<td>LV</td>
<td>Left ventricular</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>NNT</td>
<td>Number needed to treat</td>
</tr>
<tr>
<td>NSTEACS</td>
<td>Non ST-elevation acute coronary syndromes</td>
</tr>
<tr>
<td>NT-proBNP</td>
<td>N-terminal pro-B-type natriuretic peptide</td>
</tr>
<tr>
<td>PCI</td>
<td>Percutaneous coronary intervention</td>
</tr>
<tr>
<td>OASIS-5</td>
<td>Fifth Organisation to Assess Strategies in Acute Ischemic Syndromes</td>
</tr>
<tr>
<td>RITA</td>
<td>Randomised Intervention Trial of Unstable Angina (RITA-3)</td>
</tr>
<tr>
<td>SYNERGY</td>
<td>Superior Yield of the New Strategy of Enoxaparin, Revascularisation and Glycoprotein IIb/IIIa Inhibitors (SYNERGY) trial</td>
</tr>
<tr>
<td>TACTICS</td>
<td>Treat Angina With Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy-Thrombolysis in Myocardial Infarction</td>
</tr>
<tr>
<td>TNI</td>
<td>Troponin I</td>
</tr>
<tr>
<td>TNT</td>
<td>Troponin T</td>
</tr>
<tr>
<td>UFH</td>
<td>Unfractionated heparin</td>
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</table>
Purpose

These guidelines apply to the management of patients with non-ST elevation acute coronary syndromes (NSTEMI). The purpose is to provide a summary of the most up to date New Zealand and overseas evidence and to make recommendations based on the evidence that will lead to the best practice for patients with NSTEMI in New Zealand. The guideline is aimed at all health providers who care for patients with NSTEMI.

These guidelines are based on the New Zealand branch of the Cardiac Society of Australia and New Zealand (2005) Guidelines on the Non ST-elevation acute coronary syndromes: New Zealand management guidelines, the 2011 addendum to the National Heart Foundation of Australia/Cardiac Society of Australia and New Zealand guidelines for the management of ACS, and consensus of doctors, recommended by the Head of Department from every major New Zealand hospital.

For a detailed description of the levels of evidence cited in this guideline please see Appendix 2. These guidelines are intended for best clinical practice and include some drugs which are not approved yet for funding by PHARMAC. Where physicians or hospitals are not able to meet the guidelines it is recommended that there is documentation that there have been communications between clinicians and managers clearly defining the clinical implications of any resource shortages.

Early risk assessment

Introduction—Risk assessment of patients with NSTEMI for both ischaemia and bleeding, plays an important role in predicting patient prognosis and determining treatments. This also enhances the cost-effectiveness of patient care by enabling evidence-based treatments including antiplatelet, antithrombotic, and revascularisation therapies to be targeted at the patients who are most likely to benefit and not to be harmed from their use.

Ischaemic risk assessment—The clinical history, examination findings, electrocardiographic changes, and blood levels of cardiac marker and troponins are all critical factors in determining risk.

Risk assessment should be considered as a dynamic process and patients should be assessed when first seen, after several hours, 6–8 hours, 24 hours and prior to discharge. The presence of continuing symptoms and response to therapy are important in risk assessment. Refractory ischaemia or evidence of ongoing (including silent) ischaemia (ST elevation see STEMI guidelines, ST depression ≥0.5 mm) on the electrocardiogram (ECG) or monitoring, haemodynamic instability or life-threatening ventricular arrhythmias should mandate early angiography. Risk assessment may be enhanced by determining the number and severity of flow-limiting coronary artery stenoses and the presence or absence of left ventricular impairment. Risk assessment in patients with NSTEMI allows prediction of low, intermediate or high risk of death or nonfatal myocardial infarction (MI) and particularly the risk of events occurring in the short term.

The important features contributing to ischaemic risk assessment are shown in Table 1. Various risk scores can be used—e.g. the Global Registry of Acute Coronary
Events (GRACE) score [Table 1] or the Thrombolysis In Myocardial Infarction TIMI risk score. The Global Registry of Acute Coronary Events (GRACE) score is recommended as it has been shown to correlate the best with risk related to the inclusion of heart rate, blood pressure and renal function which are not included in the TIMI risk score. It is available on IPODs (www.outcomes.org/GRACE).

Table 1a. GRACE risk score

<table>
<thead>
<tr>
<th>Variables</th>
<th>Points</th>
<th>Total points</th>
<th>Probability of in-hospital death (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>0</td>
<td>≤60</td>
<td>≤0.2</td>
</tr>
<tr>
<td>40–49</td>
<td>18</td>
<td>70</td>
<td>0.3</td>
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<tr>
<td>50–59</td>
<td>36</td>
<td>80</td>
<td>0.4</td>
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<tr>
<td>60–69</td>
<td>55</td>
<td>90</td>
<td>0.6</td>
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<tr>
<td>70–79</td>
<td>73</td>
<td>100</td>
<td>0.8</td>
</tr>
<tr>
<td>≥80</td>
<td>91</td>
<td>110</td>
<td>1.1</td>
</tr>
<tr>
<td>Heart rate (beats per min)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>&lt;70</td>
<td>0</td>
<td>130</td>
<td>2.1</td>
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<tr>
<td>70–89</td>
<td>7</td>
<td>140</td>
<td>2.9</td>
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<td>90–109</td>
<td>13</td>
<td>150</td>
<td>3.9</td>
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<tr>
<td>110–149</td>
<td>23</td>
<td>160</td>
<td>5.4</td>
</tr>
<tr>
<td>150–199</td>
<td>36</td>
<td>170</td>
<td>7.3</td>
</tr>
<tr>
<td>&gt;200</td>
<td>46</td>
<td>180</td>
<td>9.8</td>
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<tr>
<td>Systolic blood pressure (mmHg)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>&lt;80</td>
<td>63</td>
<td>190</td>
<td>13</td>
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<td>80–99</td>
<td>58</td>
<td>200</td>
<td>18</td>
</tr>
<tr>
<td>100–119</td>
<td>47</td>
<td>210</td>
<td>23</td>
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<tr>
<td>120–139</td>
<td>37</td>
<td>220</td>
<td>29</td>
</tr>
<tr>
<td>140–159</td>
<td>26</td>
<td>230</td>
<td>36</td>
</tr>
<tr>
<td>160–199</td>
<td>11</td>
<td>240</td>
<td>44</td>
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<tr>
<td>&gt;200</td>
<td>0</td>
<td>≥250</td>
<td>≥52</td>
</tr>
<tr>
<td>Creatinine (μmol/L)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>0–34</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35–70</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>71–105</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>106–140</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>141–176</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>177–353</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥354</td>
<td>31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Killip class</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>0</td>
<td></td>
<td></td>
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<tr>
<td>Class II</td>
<td>21</td>
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<td></td>
</tr>
<tr>
<td>Class III</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class IV</td>
<td>64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This score should be recorded in all ACS patients to aid medical management to determine whether an invasive strategy is appropriate and its timing taking into account co-morbidities, including frailty and renal failure, risk of an invasive procedure, likelihood to benefit and patient preferences. A score >140 is high risk.
Other risk factors
Cardiac arrest at admission 43
Elevated cardiac markers 15
ST segment deviation 30

Table 1b. GRACE risk score and mortality 28(White & Chew, Table 1, Adapted with permission)

<table>
<thead>
<tr>
<th>Mortality</th>
<th>&lt;96</th>
<th>96–112</th>
<th>113–133</th>
<th>&gt;133</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-day death</td>
<td>3.1%</td>
<td>5.3%</td>
<td>5.9%</td>
<td>11.2%</td>
</tr>
<tr>
<td>12-month death</td>
<td>4.2%</td>
<td>9.6%</td>
<td>11.9%</td>
<td>27.2%</td>
</tr>
</tbody>
</table>

Bleeding risk assessment—Major bleeding occurs in approximately 4.7% of patients with non-STEMI and 2.3% with unstable angina. 14 Major bleeding is associated with increased in-hospital mortality; 5.3–15.3% in non-STEMI and 3.0–16.1% in unstable angina. Major bleeding15,16 and transfusions17 are strong predictors of mortality in non-STEACS and the increased risk is comparable to that of a recurrent MI.15 Reducing bleeding improves outcomes and reduces costs. A consensus definition of bleeding has recently been defined [Table 2].18

A patients’ risk of bleeding should be assessed with risk scores [Table 3].1B19 The CRUSADE risk score20 includes creatinine clearance, anaemia, female sex, tachycardia, hypotension, severe hypertension, heart failure, diabetes and peripheral vascular disease. Other risk factors associated with bleeding are; age >75 years; history of bleeding; history of stroke or TIA; creatinine clearance rate <60 mL/min; blood pressure <120 mmHg or ≥180 mmHg; concomitant use of a GP IIb/IIIa inhibitor; administration of enoxaparin 48 hours prior to intervention; switching between UF heparin and enoxaparin; procedural factors associated with increased risk (femoral artery versus radial artery access, prolonged procedure, intra-aortic balloon pulsation, right heart catheterisation).

Not all of these factors are also risks for ischaemic events. Bleeding may be reduced by using the radial approach21 for angiography and PCI, bivalirudin instead of UFH and IIb/IIIa antagonists,22 avoiding upstream IIb/IIIa antagonists23,24 and avoiding switching between UFH and enoxaparin.IIa A25 Patients can be switched from UFH or enoxaparin to bivalirudin.IIa B26

Table 2. Bleeding Academic Research Consortium definition for bleeding18

**Type 0:** no bleeding

**Type 1:** bleeding that is not actionable and does not cause the patient to seek unscheduled performance of studies, hospitalisation, or treatment by a healthcare professional; may include episodes leading to self-discontinuation of medical therapy by the patient without consulting a healthcare professional

**Type 2:** any overt, actionable sign of haemorrhage (e.g. more bleeding than would be expected for a clinical circumstance, including bleeding found by imaging alone) that does not fit the criteria for type 3, 4, or 5 but does meet at least one of the following criteria:

1. requiring nonsurgical, medical intervention by a healthcare professional,
2. leading to hospitalisation or increased level of care, or
3. prompting evaluation
Type 3:
Type 3a
Overt bleeding plus haemoglobin drop of 3 to <5 g/dL* (provided haemoglobin drop is related to bleed)
Any transfusion with overt bleeding
Type 3b
Overt bleeding plus haemoglobin drop ≥5 g/dL* (provided haemoglobin drop is related to bleed)
Cardiac tamponade
Bleeding requiring surgical intervention for control (excluding dental/nasal/skin/haemorrhoid)
Bleeding requiring intravenous vasoactive agents
Type 3c
Intracranial haemorrhage (does not include microbleeds or hemorrhagic transformation, does include intraspinal)
Subcategories confirmed by autopsy or imaging or lumbar puncture
Intraocular bleed compromising vision
Type 4: CABG-related bleeding
Perioperative intracranial bleeding within 48 h
Reoperation after closure of sternotomy for the purpose of controlling bleeding
Transfusion of ≥5 U whole blood or packed red blood cells within a 48-h period†
Chest tube output ≥2 L within a 24-h period
Type 5: fatal bleeding
Type 5a
Probable fatal bleeding; no autopsy or imaging confirmation but clinically suspicious
Type 5b
Definite fatal bleeding; overt bleeding or autopsy or imaging confirmation

CABG indicates coronary artery bypass graft. Platelet transfusions should be recorded and reported but are not included in these definitions until further information is obtained about the relationship to outcomes. If a CABG-related bleed is not adjudicated as at least a type 3 severity event, it will be classified as not a bleeding event. If a bleeding event occurs with a clear temporal relationship to CABG (i.e., within a 48-h time frame) but does not meet type 4 severity criteria, it will be classified as not a bleeding event.

*Corrected for transfusion (1 U packed red blood cells or 1 U whole blood=1 g/dL haemoglobin).
†Cell saver products are not counted.

Adapted with permission: Mehran et al. Circulation. 2011;123(23):2736 – Table 3.18

### Table 3a. Assessment of bleeding risk19

<table>
<thead>
<tr>
<th>Variables</th>
<th>Add to score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>0</td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;50</td>
<td>0</td>
</tr>
<tr>
<td>50–59</td>
<td>3</td>
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<tr>
<td>60–69</td>
<td>6</td>
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<td>70–79</td>
<td>9</td>
</tr>
<tr>
<td>≥80</td>
<td>12</td>
</tr>
<tr>
<td><strong>Serum creatinine (µmol/L)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;88</td>
<td>0</td>
</tr>
<tr>
<td>88</td>
<td>2</td>
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<tr>
<td>106</td>
<td>3</td>
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<td>124</td>
<td>5</td>
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<tr>
<td>141</td>
<td>6</td>
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<td>159</td>
<td>8</td>
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<tr>
<td>177</td>
<td>10</td>
</tr>
<tr>
<td>&lt;10</td>
<td>0</td>
</tr>
<tr>
<td>10–</td>
<td>2</td>
</tr>
<tr>
<td>Total Score</td>
<td>Non-CABG major bleeding within 30 days (%)</td>
</tr>
<tr>
<td>-------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>0</td>
<td>0.9</td>
</tr>
<tr>
<td>5</td>
<td>1.6</td>
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<tr>
<td>10</td>
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<td>15</td>
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<td>20</td>
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<td>30</td>
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<tr>
<td>35</td>
<td>30.7</td>
</tr>
<tr>
<td>40</td>
<td>43.5</td>
</tr>
</tbody>
</table>

Choice of antiplatelet regimens with lower bleeding risk (clopidogrel in preference to prasugrel or ticagrelor) and optimal dosing of antithrombotic therapy in relation to age; sex; weight and renal function may also reduce bleeding risk.

**Measurement of troponins**

In patients presenting with symptoms within the last 24 hours suggestive of acute myocardial ischaemia cardiac troponins T or I have the best sensitivity and specificity for the diagnosis of MI and these are the markers of choice. In both short- and long-term follow-up studies, the magnitude of troponin elevations has correlated consistently with the risk of death and the composite risk of death or nonfatal MI and troponin levels have been shown to be more powerful prognostic indicators than CKMB levels. It is recommended that CKMB no longer be measured.

Troponin point of care testing is recommended when hospital logistics cannot consistently deliver laboratory-assayed results within 1 hour.

Troponins are very sensitive markers of myocyte necrosis, and elevated levels can occur in settings other than with myocardial ischaemia. Apart from acute coronary syndromes (ACS), the most frequent causes of elevated troponin levels are:

- STEMI – raised biomarkers
- NSTEMI – normal biomarkers
- Heparin plus a GPI
- Bivalirudin monotherapy

*If patient is on bivalirudin alone rather than heparin plus glycoprotein IIb/IIIa inhibitor (GPI), the total score should be reduced by 5.

myocarditis, atrial or ventricular tachycardia (often with hypotension and an increased myocardial oxygen demand), pulmonary emboli with right ventricular infarction, and cardiac failure where troponins may be elevated due to myocardial stretch. Other causes of elevated troponin levels include cardiac surgery, Takotsubo cardiomyopathy, and renal failure. There are 6 mechanisms causing troponin elevations. Table 4. Decreased renal excretion is not considered a cause of troponin elevation.

Table 4. Pathobiology of troponin elevations

<table>
<thead>
<tr>
<th>Type</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Myocyte necrosis</td>
</tr>
<tr>
<td>Type 2</td>
<td>Apoptosis</td>
</tr>
<tr>
<td>Type 3</td>
<td>Normal myocyte turnover</td>
</tr>
<tr>
<td>Type 4</td>
<td>Cellular release of proteolytic troponin degradation products</td>
</tr>
<tr>
<td>Type 5</td>
<td>Increased cellular wall permeability</td>
</tr>
<tr>
<td>Type 6</td>
<td>Formation and release of membranous blebs</td>
</tr>
</tbody>
</table>

Adapted with permission: White HD. J Am Coll Cardiol. 2011;57(24):2406 – Table 1.

The diagnostic criteria for MI for high sensitivity troponin T is a discrimination level of $\geq 15$ ng/L, with a rise and or fall of $\geq 50\%$ over 3–6 hours (Figure 1). There are different cutpoints for troponin I.

Figure 1. Use of hsTnT to diagnose MI in a clinical setting consistent with myocardial ischemia.
MI can be ruled out with high sensitivity troponins\(^3\) if there is a level below the 99\(^{th}\) percentile 6 hrs after the onset of ischaemic symptoms\(^3\) in the absence of ongoing ischaemic symptoms.

The levels of troponins predict the benefits of therapy with low molecular weight heparins (LMWH)\(^4\), glycoprotein IIb/IIIa antagonists,\(^4\) and of an early invasive/revascularisation strategy.\(^{IIa\ B}\) Troponins are also recommended to diagnose reinfarction.\(^{IIa\ B}\)

**Initial medical management**

A 12-lead ECG should be obtained within 10 minutes of patient presentation.\(^1B\) If there is persistent (≥20 minutes) ST elevation patients should be considered for reperfusion therapy (See STEMI guidelines). Abnormalities may involve ST depression (≥0.5 mm)\(^8\) transient ST elevation and or T wave changes.

If the initial ECG is normal or non-diagnostic additional recordings should be made if there are further symptoms and repeated at 3 and 6 hours after presentation.\(^1B\)

A completely normal ECG does not exclude non-STEACS and recordings should be performed for detecting ischaemia in the circumflex territory (V\(_7\)–V\(_9\)) and the right ventricle (V\(_3\)R and V\(_4\)R).\(^{1C}\)

Blood samples for troponins, full blood count, glucose and lipids should be obtained within 10 minutes of presentation.\(^1C\) If a chest pain unit pathway is used patients should be observed and have repeat measurements of troponins at 3-6 hours after symptom onset.\(^1A\)

A second high sensitivity troponin sample within 3 hours of presentation increases the sensitivity for the diagnosis of MI to nearly 100%.\(^{1B}\)\(^44,45\)

Early discharge decisions can then be made based on clinical features, including the presence or absence of recurrence of ischaemia, troponin levels, electrocardiographic changes, and testing for inducible ischaemia as appropriate, usually with exercise testing. CT angiography has the potential to exclude significant fixed coronary artery stenoses.\(^{1B}\)\(^46,47\) An echocardiogram is recommended in all patients with elevated troponins and those with ECG abnormalities to assess global and regional left ventricular function, assess the valves for defining differential diagnoses.\(^1C\)

Where to manage patients is an important consideration. It is recommended that all high risk patients should be managed in a CCU or CCU step-down until further risk stratification shows them to be at lower risk or revascularisation is performed.\(^1C\)

The very important role of nurses in the management of these patients is acknowledged and highly valued.

**Analgesia**

Sub-lingual nitroglycerine is recommended for symptoms of ischaemia.\(^1C\) Morphine together with an antiemetic should be used to relieve severe pain.\(^1C\) Intravenous nitroglycerine can also achieve symptomatic relief and be used for blood pressure lowering.\(^1C\)
Oxygen therapy

A recent Cochrane meta-analysis identified three trials with a total of 387 patients evaluating the value of oxygen therapy in whom 14 deaths occurred. The relative risk of death for those receiving oxygen therapy was 2.88 (95%CI 0.88–9.39) by intention-to-treat analysis and 3.03 (95%CI 0.93–9.83) amongst patients with confirmed acute MI. Although these analyses lacked adequate power the findings suggest increased hazard and the routine use of supplemental oxygen is not recommended. **IIa A** Oxygen therapy is indicated for patients with hypoxia (oxygen saturation <93%) and those with evidence of shock, to correct tissue hypoxia. In the absence of hypoxia, the benefit of oxygen therapy is uncertain, and in some cases oxygen therapy may be harmful. **IIa C**

Antiplatelet agents

Table 5 summarises the recommended dosage regimens for various antiplatelet therapies.

**Aspirin**—Aspirin reduces progression to MI and cardiac mortality by about 50% and all patients without contraindication should immediately receive aspirin 150–300 mg, which should be chewed if enteric coated. Long-term, lower doses of 75-100 mg in enteric coated formulations to maintain efficacy and to minimise bleeding risk should be given indefinitely.

**Clopidogrel**—The CURE trial and the separately reported PCI-CURE results provide important evidence for the use of clopidogrel in patients with NSTEACS regardless of whether they are managed conservatively or invasively. In the CURE trial which randomised 12,562 patients (77% managed conservatively), clopidogrel reduced the incidence of death, non-fatal MI and stroke by 20% over an average 9-month follow-up period (9.3% with clopidogrel vs 11.5% with placebo, P<0.001). There were also reductions in the rates of revascularisation, as well as need for thrombolytic therapy and intravenous glycoprotein IIb/IIIa inhibitors in the clopidogrel group.

There was an excess of major bleeding with clopidogrel (3.7% vs 2.7%, P=0.003) but life-threatening bleeding was not increased. In patients undergoing CABG within 5 days of receiving clopidogrel, there was an increase in major bleeding from 6.3% to 9.6%, p=0.05. This compares with 7 major events per 1 000 patients (cardiovascular death, MI or stroke) prevented within the first 24 hours with clopidogrel. Clopidogrel should be stopped 5 days prior to surgery.

In the PCI-CURE trial with 2658 patients, pre-treatment with clopidogrel for 10 days prior to PCI reduced 30-day composite of death, non-fatal MI and urgent target vessel revascularisation by 30% after PCI (4.5% vs 6.4%, P=0.03). Long-term administration of clopidogrel after PCI for 12 months was associated with a lower rate of cardiovascular death, MI, or any revascularisation (p=0.03), and of cardiovascular death or MI (p=0.047).

Overall (including events before and after PCI) there was a 31% reduction in cardiovascular death or MI (p=0.002). Long-term benefit of clopidogrel plus aspirin after PCI in patients with chronic stable angina was also shown in the CREDO trial. At 1 year, the composite endpoint of death, myocardial infarction or stroke was
reduced by 27% in the clopidogrel group. Greater benefit was achieved in patients receiving clopidogrel >6 hours prior to PCI.

In the CAPRIE trial\textsuperscript{54} in patients with previous MI, stroke or peripheral vascular disease clopidogrel had an 8.7% greater benefit than aspirin on reducing vascular death, MI and ischaemic stroke. Clopidogrel is therefore a useful alternative to aspirin when there is intolerance to aspirin.\textsuperscript{1A}

The CURRENT trial compared, in patients with ischaemic ECGs or elevated biomarkers, clopidogrel with 600 mg loading followed by 150 mg daily for 7 days and then 75 mg/day compared with 300 mg followed by 75 mg/day. There was no difference between the groups for the primary endpoint of CV death, MI or stroke at 30 days.\textsuperscript{55} In a prespecified post randomisation subgroup analysis of patients undergoing PCI (63.1% with non-STEACS) the primary endpoint was reduced with the higher dose clopidogrel regimen; 3.9% vs 4.5%, HR 0.86; 95%CI 0.74–0.99, p=0.039. Stent thrombosis (ARC definition for definite or probable)\textsuperscript{56} was also reduced; HR 0.69; 95%CI 0.56–0.87, p=0.001. CURRENT defined major bleeding was increased but TIMI major bleeding was not; 1.0% high dose vs 0.7% standard dose clopidogrel, p=0.07.

The efficacy of clopidogrel is affected by a number of factors including age; diabetes; and genetic polymorphisms.\textsuperscript{57,58} High levels of platelet reactivity after clopidogrel are associated with increased risks of ischaemic events and stent thrombosis.\textsuperscript{36} However in a trial targeting higher doses of clopidogrel (150 mg vs 75 mg) in patients with high platelet reactivity there was no advantage of the higher dose regimen.\textsuperscript{60}

There are two approaches, one is to give clopidogrel only at the time of PCI after the coronary anatomy is known and the other is to give it to all patients prior to angiography, except those in whom urgent CABG is likely as there is increased bleeding if clopidogrel has been given within 5 days of surgery.\textsuperscript{51} These patients include those with ECG changes suggestive of ≥50% left main stenosis (i.e. ST deviation in ≥2 coronary artery territories), known coronary anatomy from a previous angiogram which is inappropriate for PCI, the presence of multiple regional wall motion abnormalities on echocardiography, haemodynamic instability or heart failure. All of these patients should be considered for expeditious angiography.

Clopidogrel (600 mg loading dose, 150 mg for 7 days and then 75 mg daily in patients undergoing an invasive strategy; 75 mg daily after the loading dose in patients managed with a conservative strategy) is recommended in addition to aspirin or as an alternative to aspirin \textsuperscript{IIa B} and continued for 12 months \textsuperscript{1A} if ticagrelor and prasugrel are not available.

\textbf{Prasugrel}\textmd{—}\textmd{Prasugrel produces more rapid and consistent platelet inhibition than clopidogrel}\textsuperscript{61} and is not affected by polymorphisms that affect clopidogrel. In the TRITON trial, prasugrel (60 mg loading and 10 mg daily) was compared with clopidogrel 300g loading and then 75 mg/day.\textsuperscript{62}

The composite primary endpoint (cardiovascular death, non-fatal MI, or stroke) occurred in 11.2% of clopidogrel-treated patients and in 9.3% of prasugrel-treated patients (HR 0.82; 95%CI 0.73–0.93; P = 0.002), mostly driven by a significant risk reduction for MI (from 9.2% to 7.1%; RRR 23.9%; 95%CI 12.7–33.7; P < 0.001). Definite or probable stent thrombosis was reduced from 2.4% to 1.1%; HR 0.48,
95%CI 0.36–0.64. There was a significant increase in the rate of non-CABG-related TIMI major bleeding (2.4% vs. 1.8%; HR 1.32; 95%CI 1.03–1.68; P = 0.03). Life-threatening bleeding was significantly increased with prasugrel; 1.4% vs. 0.9% (HR 1.52; 95%CI 1.08–2.13; P = 0.01), as well as fatal bleeding, with 0.4% vs. 0.1% (HR 4.19; 95%CI 1.58–11.11; P = 0.002). There was net harm with prasugrel in patients with a history of TIA or stroke. There was no apparent net clinical benefit in patients >75 years of age and in patients with low body weight (<60 kg). Greater benefit without increased risk of bleeding was observed in diabetic patients.

Prasugrel (60 mg loading dose, 10 mg daily) is an alternative (not funded at present) when the coronary anatomy is known and the bleeding risk is low. IB Prasugrel should be stopped 7 days prior to surgery. IC

**Ticagrelor**—Ticagrelor is a rapid acting reversible (triazolopyrimidine) P2Y12 inhibitor which achieves greater platelet inhibition at 2 hours (as assessed with light transmittance aggregometry) than after clopidogrel with a 600 mg loading dose (88% vs 38%, p<0.001).63

In the PLATO trial which randomized 18,624 patients with an ACS with or without ST elevation received ticagrelor or clopidogrel (300 mg loading dose was recommended unless patients were pre-treated; ≥600 mg was given in 19.6% of patients in the clopidogrel arm) for a mean duration of 277 days. The composite of CV death, MI or stroke was reduced with ticagrelor from 11.7% to 9.8%; HR 0.84, 95%CI 0.77–0.92, p<.001. Definite stent thrombosis was reduced from 1.9% to 1.3%, p<0.01 and total mortality from 5.9% to 4.5%, p<0.001. Overall bleeding was not increased but major bleeding unrelated to CABG was increased; 4.5% ticagrelor, 3.8% clopidogrel, HR 1.19; 95%CI 1.02–1.38, p=0.03.

Ticagrelor (180 mg loading dose, 90 mg bid) is recommended (not currently funded) as the preferred P2Y12 inhibitor. IB Ticagrelor should be stopped 5 days prior to surgery. IC

Ticagrelor, prasugrel and clopidogrel should be continued for 12 months after ACS including recommencement after CABG.

**Glycoprotein IIb/IIIa antagonists**—In the EARLY ACS trial in patients with high risk non-ST elevation ACS the routine use of eptifibatide did not lower ischaemic risk on background therapy of aspirin and clopidogrel but was associated with increased risk of bleeding.23 Similar results were seen in the ACUITY trial.24

Routine upstream administration of IIb/IIIa antagonists (tirofiban or eptifibatide) is not recommended in the absence of continuing ischaemia prior to angiography. III A They may be administered at the time of PCI (eptifibatide or abxicimab IV or intracoronary) if there is thrombus present or poor coronary flow. III C
Table 5. Clinical use of antithrombotic therapies

<table>
<thead>
<tr>
<th>Oral antiplatelet therapies</th>
<th>Heparins</th>
<th>Glycoprotein IIb/IIIa antagonists</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Heparin (UFH)</td>
<td>Tirofiban (Aggrastat)</td>
</tr>
<tr>
<td>Clopidogrel (Plavix)</td>
<td>Enoxaparin (Lovenox)</td>
<td>Eptifibatide (Integrilin)</td>
</tr>
<tr>
<td>Prasugrel (efficient)</td>
<td>Prasugrel (Effient)</td>
<td>Abxicimab (ReoPro)</td>
</tr>
<tr>
<td>Ticagrelor (Brilinta)</td>
<td>Ticagrelor (Brilinta)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Heparin (UFH)</td>
<td>Tirofiban (Aggrastat)</td>
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<tr>
<td></td>
<td>Ticagrelor (Brilinta)</td>
<td></td>
</tr>
</tbody>
</table>

**Oral antiplatelet therapies**

- **Aspirin**: Initial dose of 150-300 mg followed by 75-150 mg/day of an enteric formulation.
- **Clopidogrel (Plavix)**: A loading dose of 600 mg followed by 150 mg/day for 7 days and then 75 mg daily for 12 months.
- **Prasugrel (efficient)**: A loading dose of 60 mg followed by 10 mg bid for 12 months.
- **Ticagrelor (Brilinta)**: 180 mg followed by 90 mg bid for 12 months.

**Heparins**

- **Heparin (UFH)**: Bolus 60U/kg (maximum 4000 U) IV followed by infusion of 12U/kg/h (modified to achieve an aPTT of 50-75s) with laboratory measurements and 60-85 seconds with bedside measurements.
- **Enoxaparin (Lovenox)**: 1 mg/kg subcutaneously 12 hourly; preceded by a 30 mg IV bolus. ‡ In patients aged ≥75 years no bolus and 0.75 mg/kg subcutaneous 12 hourly. If creatinine clearance <30 mL/min give 1 mg/kg daily.

**Glycoprotein IIb/IIIa antagonists**

- **Tirofiban (Aggrastat)**: 0.4 µg/kg/min for 30 minutes followed by infusion of 0.1 mcg/kg/h for 48 to 96 h and for 12–24 hours post PCI.
- **Eptifibatide (Integrilin)**: Double bolus 180 mcg/kg separated by 10 minutes followed by infusion of 2.0 µg/kg/min for 72 to 96 h and for 12-24 hours post PCI. (If creatinine clearance <50 mL/min give 1 mg/kg/min).
- **Abxicimab (ReoPro)**: 0.25 mg/kg bolus followed by infusion of 0.125 mcg/kg/min (maximum 10 mcg/min) for 12 to 24 hours post PCI. Abxicimab should not be used as upstream treatment unless coronary anatomy is known and the patient is scheduled for PCI.

‡ Adjustment required for age ≥75 years and renal dysfunction – see pharmacy guidelines.

**Antithrombotic agents**

Table 5 summarises the recommended dosage regimens for various antithrombotic therapies.

**Enoxaparin**—Low molecular weight heparins have several advantages over UFH including less platelet activation, a more predictable dose-effect relationship and a low rate of heparin induced thrombocytopenia (HIT). A meta-analysis of all enoxaparin trials shows a 16% reduction in death and MI at 30 days compared to therapy with UFH. 91

The SYNERGY trial in 10,027 high risk patients, showed similar outcomes with UFH compared with enoxaparin on a background of high usage of clopidogrel and glycoprotein IIb/IIIa antagonists and an invasive strategy with a modest increase in bleeding. 25 There was no significant increase in transfusions but there was an increase in TIMI major bleeding (See Appendix 3) (non CABG related) in all patients 1.7% UFH, 2.4% enoxaparin; p=0.025. In patients undergoing PCI there were similar TIMI major bleeding rates of 2.8% in patients receiving UFH vs 2.7% in patients receiving enoxaparin on a background of aspirin, clopidogrel, and GP IIb/IIIa inhibitors. Either enoxaparin or UFH should be continued until catheterisation or for 48 hours with the preferred therapy being enoxaparin. 1B
If patients have been pre-treated with enoxaparin no additional enoxaparin is necessary if PCI is performed within 8 hours of the previous dose. If the previous dose of enoxaparin was >8 hours an additional 0.3 mg/kg IV is required. In view of increased bleeding and events if patients are switched from one antithrombotic agent to another, patients should continue on the initial antithrombotic agent. III B

Fondaparinux—Fondaparinux was shown in the OASIS-5 study to be non-inferior to enoxaparin and to be associated with a reduction in major bleeding and 6 months mortality. It is particularly useful in patients not planned to have early invasive management. It is not is not approved for ACS in New Zealand.

Bivalirudin—Bivalirudin is a direct thrombin inhibitor which inactivates fibrin-bound as well as fluid-phase thrombin. In the ACUITY trial 13,819 moderate and high-risk patients with non-STEACS planned for an invasive strategy were randomized to bivalirudin alone, bivalirudin plus a GP IIb/IIIa antagonist, or UFH or enoxaparin with a GP IIb/IIIa antagonist. There was no difference between the first two groups for a composite ischaemic endpoint of death, MI or unplanned revascularisation for ischaemia. Bivalirudin alone was non-inferior (upper 95%CI did not exceed a relative margin of 25%) to the UFH/enoxaparin plus GP IIb/IIIa group; 7.8% vs 7.3% RR 1.08, 95%CI 0.93–1.24, p=0.32. And there was less major bleeding; 3.0% vs 5.7%, RR 0.53, 95%CI 0.43–0.65, p<0.0001. Crossing over from UFH or enoxaparin to bivalirudin maintained the benefit of reduced bleeding with bivalirudin. 26

Bivalirudin is recommended instead of UFH or enoxaparin with a IIb/IIIa antagonist and use should be considered when the time to angiography is short (<12 hours) or there is a high risk of bleeding and switching is appropriate. IB

β-blockers

Oral β-blockers are recommended if there are no contraindications (asthma, systolic BP <110 mmHg, heart rate <50 min or AV block > Mobitz Type I or Killip class ≥3). 1B

Oral B Blockade should be continued for at least 3 years and can be continued indefinitely in the absence of side effects. Class I 1C

Calcium channel blockers

If β-blockers are contraindicated, diltiazem should be given. 1B Calcium channel blockers are recommended in patients with coronary artery spasm. 1C Calcium channel blockers that increase heart rate should not be used without concomitant β-blockers therapy. III C

Lipid modifying therapy

Use of a fixed dose of simvastatin (40 mg) has been shown to reduce events by over 20% in HPS in non ACS patients. Achievement of an LDL level of 1.6 mmol/L with atorvastatin (80 mg) has been shown to reduce by 16% a composite endpoint of death, MI, readmission with unstable angina, revascularisation and stroke compared to an LDL level of 2.5 mmol/L achieved with pravastatin therapy (40 mg).
Initiation of high dose statin therapy should be commenced in hospital in all ACS patients in order to enhance adherence and to reduce events. 1B Administration of a high dose statin is reasonable before PCI to reduce the risk of periprocedural MI. IIa

ACE inhibitors

All patients with evidence of heart failure, should receive oral ACE inhibitors (or ARB if intolerant of ACE inhibitors) beginning 1 - 2 hours after admission if the systolic BP is >100 mmHg using (e.g. Inhibace 0.5 mg bid, 6.25 mg tds, or equivalent medication) and then increasing over several days to maximally tolerated doses. 1A 69,70 ACE inhibitors or ARBs are recommended in all other patients to prevent recurrent ischemia events. Drugs used in trials showing benefit and in doses of proven efficacy are recommended. 1B ACE inhibitors should be continued indefinitely. 69 1C

Aldosterone antagonists

Aldosterone antagonists are recommended in patients who have an ejection fraction ≤35%. IIb

Aldosterone antagonists should also be considered in all patients with a history of heart failure and impaired LV systolic function treated with a loop diuretic. IIb

Caution is needed in patients with impaired renal function because of an increased risk of hyperkalaemia. 1C

Early angiography and revascularisation

Early angiography and revascularisation improves symptoms, improves prognosis, and shortens hospital stay. 71-75

The FRISC-II trial demonstrated superiority in higher risk patients of an invasive approach with PCI or CABG after initial medical treatment with the low molecular weight heparin dalteparin and aspirin for 4-7 days with a reduction in mortality at 1 year from 3.9% to 2.2% P=0.01612. 92 The TACTICS trial 42 randomised 2220 high risk patients with aspirin, UFH and tirofiban to an early invasive strategy with angiography within 4–48 hours followed by revascularisation if the anatomy was suitable, or to a more conservative strategy with catheterisation only for recurrent ischaemia or a positive stress test. Death, non-fatal MI and rehospitalisation for ACS at 6 months occurred in 15.9% of patients in the invasive arm and 19.4% in the conservative arm (P=0.025). The benefit of an invasive approach was confined to medium and high-risk patients who had elevated troponins, ST segment changes or diabetes.

RITA 3 71 also showed benefit of an invasive strategy in high risk patients treated with enoxaparin for 3 days prior to intervention. The ISAR Cool study 72 showed that an immediate invasive approach in 410 patients with either ST depression or elevated troponins (time to angiography of 2.4 hours) together with aspirin, clopidogrel, UFH and tirofiban resulted in lower rates of MI (5.9% vs 10.1%) compared with delaying PCI while on the same therapy for 72 hours.

In the ICTUS study in 12000 patients all patients had elevated troponins and a strategy of early invasive therapy was compared with a selective invasive approach. 73
All patients were recommended to receive aspirin, clopidogrel 300 mg as a loading dose, enoxaparin and atorvastatin 80 mg. The invasive group was also given abxicimab. In the routine invasive group 76% had revascularisation in hospital compared to 40% in the selective invasive group. In this latter group a further 14% crossed over to the invasive arm by 12 months. At 1 year the composite of death, MI or rehospitalisation for anginal symptoms was similar in both groups; 22.7% invasive, 21.2% selective, RR 1.09, 95%CI 0.87–1.33, p=0.33.

A meta-analysis of seven trials comparing a routine invasive vs a conservative or selective approach with contemporary adjunctive therapy showed a reduction with an early routine invasive strategy at 2 years in mortality 4.9% vs 6.3% RR 0.75, 95%CI 0.63–0.90, p=0.001 and non-fatal MI 7.6% vs 9.1% RR 0.83, 95%CI 0.72–0.96, p=0.012.74

A recent meta-analysis of 8 trials showed a significant reduction in death, MI or rehospitalisation at 1 year with comparable benefit in men and high-risk women.75 A more recent meta-analysis of the FRISC-2, ICTUS and RITA 3 studies with 5-year follow-up showed a significant reduction in death and MI with the invasive strategy.76,77 There was an 11.1% absolute benefit (NNT nine) in the highest risk patients and 2–3.8% absolute benefit (NNT 26–50) in the low and immediate risk patients.

**Timing of intervention**

The optimal timing for angiography and PCI with an invasive strategy has been evaluated in a number of trials. In a meta-analysis of 4 trials intervention on the first hospital day was shown to be safe, associated with 41% lower risk of recent ischaemia and a shorter hospital stay.

In patients at higher risk [Table 6] there is strong evidence to suggest a benefit of an invasive strategy. In the TIMACS trial at 6 months there was a 38% lower risk of death MI or stroke in patients with a GRACE risk score >140 with no increase in safety concerns.79 Also in the ACUITY trial delay to PCI >24 hours was an independent predictor of 30-day and 1-year mortality.81

Patients at very high risk should go to the cath lab emergently ≤2 hours if they have refractory angina, with associated heart failure, life threatening ventricular arrhythmias, hemodynamic instability or recurrent marked (≥1 mm) dynamic ECG changes or ≥1 mm ST depression V2–V4 indicative of circumflex occlusion. 1B 5,80

- Immediate arrangement must be made for immediate transfer from a non-PCI hospital to a PCI capable Hospital. 1C
- Advanced age, frailty, co-morbidities, procedural risk, ability to benefit, and patient preferences must be taken into account. 1C
Table 6. Criteria for high risk with indication for invasive management

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Relevant rise or fall in troponin*</td>
<td>• Diabetes mellitus</td>
</tr>
<tr>
<td>• Dynamic ST- or T-wave changes (symptomatic or silent)</td>
<td>• Renal insufficiency (eGFR &lt;60 mL/min/1.73 m²)</td>
</tr>
<tr>
<td></td>
<td>• Reduced LV function (ejection fraction &lt;40%)</td>
</tr>
<tr>
<td></td>
<td>• Early post infarction angina</td>
</tr>
<tr>
<td></td>
<td>• Recent PCI</td>
</tr>
<tr>
<td></td>
<td>• Prior CABG</td>
</tr>
<tr>
<td></td>
<td>• Intermediate to high GRACE risk score (Table 2)</td>
</tr>
</tbody>
</table>

*Rise/fall of troponin relevant according to precision of assay
CABG = coronary artery bypass graft
eGFR = estimated glomerular filtration rate
GRACE = Global Registry of Acute Coronary Events
LV = left ventricular
PCI = percutaneous coronary intervention.

Adapted with permission: Hamm et al. EHJ. 2011;32:2999 – Table 9.

In patients at **high risk** with both raised troponins, and ischaemic ECG changes (elevation or depression ≥1 mm or T wave inversion ≥ 2 mm V2–V3), and especially if the patient has a GRACE score >140, angiography should optimally be performed in ≤24 hours in a PCI capable hospital. **1B**
- Immediate arrangement must be made for transfer within 24 hours from a non-PCI hospital. **1C**

In other patients angiography should be performed within 72 hours. **1A**
- Advanced age, frailty, co-morbidities, procedural risk, ability to benefit, and patient preferences must be taken into account. **Class 1C**
- It is recognised that this is the optimal goal and may not be possible over weekends and public holidays and where resources are limited.

For **low risk** patients in whom a conservative strategy is selected and recurrent ischaemia has not occurred, a non-invasive test for inducible ischaemia should be performed in hospital with management based on the results of the test. **1A**

Renal failure is a relative contraindication for angiography and revascularisation because of the hazard of contrast induced nephropathy. **1C** Randomised data on the advantage of an invasive strategy are not available.

Advanced age is not an absolute contraindication for angiography and PCI, and because of data showing reduced readmissions and reduced costs in the elderly, **84** PCI should be considered in all patients without frailty or significant co-morbidity with appropriate consideration to patient preferences. **1B**

**Patients on warfarin or dabigatran**

Decisions as to whether patients should undergo an invasive strategy when the INR with warfarin is therapeutic or the patient is on dabigatran should be the same as when
patients are not on these therapies. Treatment should be continued until angiography and adjunctive anticoagulant therapy withheld (unless the INR is subtherapeutic).

Treatment with aspirin and P2Y12 inhibitors and their duration needs to be individualised according to whether a stent is inserted (bare metal preferred) and the individualised risk of stent thrombosis and bleeding.

Triple therapy (aspirin, a P2Y12 inhibitor (prasugrel should not be used), and warfarin (INR 2.0–2.5) or dabigatran) should be used for as short a period as appropriate e.g.: with bare metal stent 1 month, drug eluting stent 6 months. There is currently no evidence base for the use of the combination of the dabigatran (lower bleeding with 110 mg bid as compared with warfarin) and ticagrelor.

**Smoking cessation**

Smokers should be advised to quit and be given nicotine patches and lozenges as appropriate on day 1. 1C

**Secondary prevention**

All patients should be referred to rehabilitation services. All patients without contraindication should be on aspirin, a β-blocker, a statin with optimisation of LDL cholesterol below 1.6mmol/L, and an ACE inhibitor or ARB indefinitely and a P2Y12 inhibitor for 12 months. Patients should stop smoking, have a cardioprotective diet to achieve ideal weight, and exercise 30 minutes on most days. 1A

**Measurement of performance indicators**

Reduction of the delay between onset of symptoms and presentation to hospital and time to an invasive strategy is recognized as an important clinical goal. Clinical networks with predefined protocols for transport from hospitals without capacity for early catheterisation to hospitals with the capacity must be further developed. 1C

Appropriate evidence-based treatments should be given to all eligible patients without contraindications. Routine audit should be integrated into all clinical services that provide care to patients with ACS. This should include prescribing and adherence with aspirin, P2Y12 inhibitor, B-blockers, ACE inhibitors or ARBS, aldosterone antagonists, statins, cardiac rehabilitation and smoking cessation. Metrics including percentages of patients undergoing angiography, PCI and CABG, and time to angiography should also be monitored with feedback. 1C

**Resource availability**

It is recognised that in New Zealand that providing expensive pharmaceuticals and equitable provision of an invasive strategy for Maori and rural populations is challenging. However, it is recognised that an invasive approach has been shown to be cost effective and it is expensive to keep patients in hospital for long periods awaiting diagnostic testing. If these patients are discharged without angiography there is a high risk of reinfarction or readmission to hospital.

In New Zealand, cost-effective and readily available therapies such as aspirin, beta-blockers and ACE inhibitors are still under-prescribed. It is important that these
treatments are used in as many patients without contraindications as possible and that PCI is equitably available to all New Zealanders.

**Competing interests:** None known.

**Author information:** See Appendix 1.

**Acknowledgement:** We are extremely grateful to Charlene Nell for secretarial assistance.

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**References:**


Appendix 1. Non ST-Elevation Acute Coronary Syndromes Guidelines Group

<table>
<thead>
<tr>
<th>Name</th>
<th>Hospital/Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrew Hamer</td>
<td>Nelson Marlborough District Health Board</td>
</tr>
<tr>
<td>Andrew Kerr</td>
<td>Middlemore Hospital, Auckland</td>
</tr>
<tr>
<td>Brandon Wong</td>
<td>Whangarei Hospital, Whangarei</td>
</tr>
<tr>
<td>Charles Renner</td>
<td>Kew Hospital, Invercargill</td>
</tr>
<tr>
<td>Cheuk-Kit Wong</td>
<td>Dunedin School of Medicine, Dunedin</td>
</tr>
<tr>
<td>Chris Ellis</td>
<td>Green Lane Cardiovascular Service, Auckland City Hospital</td>
</tr>
<tr>
<td>Chris Nunn</td>
<td>Waikato Hospital, Hamilton</td>
</tr>
<tr>
<td>David Smyth</td>
<td>Christchurch Hospital, Christchurch</td>
</tr>
<tr>
<td>Gerry Devlin</td>
<td>Waikato Hospital, Hamilton</td>
</tr>
<tr>
<td>Gerry Wilkins</td>
<td>Dunedin Hospital, Dunedin</td>
</tr>
<tr>
<td>Guy Armstrong</td>
<td>North Shore Hospital, Auckland</td>
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<tr>
<td>Hamish Hart</td>
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<tr>
<td>Harvey White</td>
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<tr>
<td>Hitesh Patel</td>
<td>North Shore Hospital, Auckland</td>
</tr>
<tr>
<td>Ian Crozier</td>
<td>Christchurch Hospital, Christchurch</td>
</tr>
<tr>
<td>Ian Ternouth</td>
<td>Taranaki Base Hospital</td>
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<tr>
<td>John Elliott</td>
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<tr>
<td>Lynne Belz</td>
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</tr>
<tr>
<td>Malcolm Abernathy</td>
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</tr>
<tr>
<td>Mark Simmonds</td>
<td>Wellington Hospital, Wellington</td>
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<td>Mark Webster</td>
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<td>Nigel Harrison</td>
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<tr>
<td>Paul Tanser</td>
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<tr>
<td>Phil Matsis</td>
<td>Wellington Hospital, Wellington</td>
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<tr>
<td>Ralph Stewart</td>
<td>Green Lane Cardiovascular Service, Auckland City Hospital (Cardiac Society Representative)</td>
</tr>
<tr>
<td>Richard Luke</td>
<td>Royston Hospital, Hastings</td>
</tr>
<tr>
<td>Scott Harding</td>
<td>Wellington Hospital, Wellington</td>
</tr>
<tr>
<td>Seif El-Jack</td>
<td>North Shore/Waitakere Hospital, Auckland</td>
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<tr>
<td>Stewart Mann</td>
<td>Wellington Hospital, Wellington (Heart Foundation Representative)</td>
</tr>
</tbody>
</table>
## Appendix 2. Classes of recommendation and grading levels of evidence

<table>
<thead>
<tr>
<th>Classes of recommendation</th>
<th>Definition</th>
</tr>
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<tbody>
<tr>
<td>Class I</td>
<td>Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.</td>
</tr>
<tr>
<td>Class II</td>
<td>Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.</td>
</tr>
<tr>
<td>Class IIa</td>
<td>Weight of evidence/opinion is in favour of usefulness/efficacy.</td>
</tr>
<tr>
<td>Class IIb</td>
<td>Usefulness/efficacy is less well established by evidence/opinion.</td>
</tr>
<tr>
<td>Class III</td>
<td>Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.</td>
</tr>
</tbody>
</table>

### Levels of evidence

<table>
<thead>
<tr>
<th>Level of Evidence</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of Evidence A</td>
<td>Data derived from multiple randomised clinical trials or meta-analyses.</td>
</tr>
<tr>
<td>Level of Evidence B</td>
<td>Data derived from a single randomised clinical trial or large non-randomised studies.</td>
</tr>
<tr>
<td>Level of Evidence C</td>
<td>Consensus of opinion of the experts and/or small studies, retrospective studies, registries.</td>
</tr>
</tbody>
</table>

Adapted with permission: Hamm CW. EHJ. 2011; 32(23): 2999- Table 1 and 2).
Appendix 3. TIMI Major Bleeding Criteria

Bleeding is associated with ≥5 g/dL decrease in hemoglobin (each unit of packed red blood cells or whole blood transfused counting as 1g of hemoglobin) or a ≥15% absolute decrease in hematocrit (each unit of packed red blood cells or whole blood transfused will count as 3% points) or it is intracranial (confirmed by magnetic resonance imaging or computer tomography).
Dabigatran: rational dose individualisation and monitoring guidance is needed

Stephen B Duffull, Daniel F B Wright, Hesham S Al-Sallami, Paul J Zufferey, James M Faed

Abstract

Dabigatran is the first oral anticoagulant to be introduced in New Zealand without prescribing restrictions for over 50 years. Not surprisingly, the drug has created a great deal of interest amongst health care providers as well as the general public and media. There seems to be a general feeling that warfarin, with its requisite dose adjustments and INR monitoring, is an outdated drug and should be shelved in favour of this novel agent. The assumption is that the newer drug must be better and safer as well as easier to use. Much of the literature associated with dabigatran encourages this view, stressing that dabigatran is a ‘game changer’ with the advantage of fixed dosing for most patients and no anticoagulation monitoring. In this paper we question whether dabigatran can really live up to these expectations. We suggest that the safe and effective prescribing of dabigatran, like all anticoagulants used in therapeutic doses, will most likely require dose individualisation and selective monitoring. This requirement should not be viewed as a failure for dabigatran but rather as a success for rational therapeutics.

Dabigatran is an orally-active direct thrombin inhibitor currently licensed for use in non-valvular atrial fibrillation and for the prevention of venous thromboembolism following joint replacement surgery. It is marketed in New Zealand under the brand name Pradaxa® and was listed in the Pharmaceutical Schedule without Special Authority in July 2011. Whilst we believe that the availability of this drug will provide more flexibility for the prescriber, and may lead to improved outcomes for some patients, we have major concerns about the lack of rational advice on dosing and monitoring, particularly for patients at high risk of bleeding.

Current dosing guidance for dabigatran

The current dosing advice for prescribers is puzzling. The New Zealand datasheet states that all patients get a fixed dose unless (1) they have atrial fibrillation and are over 80 years old, or, (2) if they have had orthopaedic surgery and have impaired renal function.1 No comment is made as to why the suggested dose reductions for age and renal impairment do not apply equally to both indications. This guidance seems to fly in the face of reason.

Drugs that are cleared renally, such as dabigatran, are reliant on glomerular filtration rate (GFR) for elimination. A normal GFR may be in the order of about 100 mL/min. If the patient has a GFR of half or one-third normal (e.g. 30–50 mL/min) the clearance of the drug will also be reduced and plasma concentrations will increase proportionately. A dose reduction is therefore required to normalise drug exposure and reduce the risk of adverse effects.
There is a paucity of independent prescribing guidance for dabigatran. The recent bulletin from the Best Practice Advocacy Centre (BPAC) is a welcome and thorough overview of dabigatran for primary care practitioners but falls short when it comes to specific advice about dosing. Rather, BPAC advocates that prescribers take a ‘cautious approach’, particularly when prescribing for patients at high risk of bleeds such as those with renal impairment. This leaves the practitioner with little practical guidance with which to meet the needs of individual patients.

Consider, for example, the hypothetical case of a 79-year-old female with atrial fibrillation who has a lean mass of 50 kg and a normal serum creatinine of 110 μmol/L. Her estimated creatinine clearance calculated by the Cockcroft and Gault formula would be 33 mL/min. This patient would clearly be at risk of increased drug exposure and bleeding if a fixed dose were given, a situation that would no doubt be recognised by a cautious prescriber. However, the fact remains that the dosing guidance in this situation is lacking and contradictory.

In the absence of a validated means of monitoring the effectiveness and safety of therapy the physician is left guessing as to the best course of action. If the patient had been post-orthopaedic surgery, then the dose would be reduced according to the recommendations in the datasheet without question. However since the patient has atrial fibrillation the manufacturer’s guidance suggests that it is perfectly reasonable to continue the dose at 150 mg twice daily without adjustment.

The need for dose individualisation

As with any anticoagulant, the use of dabigatran carries a risk of bleeding. Major bleeding is an independent predictor of death and is associated with increased cost and longer duration of hospital stay. Arguably, there is a fine line between the magnitude of anticoagulation required to prevent clots in susceptible patients and that which is sufficient to cause bleeding, especially in a patient population where a variety of vascular disorders are common. This is independent of the drug itself but relates to the innate complexity and sensitivity of the coagulation network, which is the target for anticoagulant action.

In short, we suggest that all anticoagulants used in therapeutic doses will have a narrow therapeutic range and will require selective monitoring to ensure optimal effectiveness and the prevention of side effects. In addition, analysis of the coagulation system (based on Wajima et al) suggests that there may be less natural dampening of the coagulation system with anticoagulants that target the later stages of coagulation. Hence, the anticoagulant effects of drugs like dabigatran that act close to the final stage of clot formation will, in theory, be more sensitive to the prescribed dosing regimen and to inherent variability in the dose-concentration relationship (pharmacokinetics).

When dabigatran was introduced into the New Zealand market the media release from Pharmac claimed “… [dabigatran] is literally a game-changer and demonstrates PHARMAC’s desire to move relatively swiftly to fund genuinely innovative medicines”. Although the spirit of the statement is appropriate, the suggestion that introducing a new drug is all that it takes to change the game is misleading.
Individualisation of existing medicines has often been shown to be quantitatively more important than the introduction of a new medicine. For instance, an individualised regimen of enoxaparin treatment, based on renal function and body composition, resulted in a number needed to treat (NNT) of 8 patients to reduce bleeding events when compared to conventional weight-adjusted dosing. This is a very favourable number when compared to many new interventions. For example, simvastatin was found to have an NNT for one year of 167 for preventing all cardiovascular events in the Scandinavian Simvastatin Survival Study (4S). We therefore contend that the act of introducing a new anticoagulant will not necessarily change the game without knowledge of how best to individualise its use.

So why would dabigatran be considered a games changer? Perhaps this is because it is seen as a replacement for warfarin, with its requisite INR monitoring and dose adjustments? If so, we query whether dabigatran will ultimately prove to be all that different from warfarin and whether dose individualisation and selected anticoagulation monitoring will eventually prove important for safe prescribing. If so, the monitoring required will need to be targeted to clinical situations that are more likely to be associated with bleeding or clotting.

**Is dabigatran a better choice than warfarin?**

Warfarin acts by inhibiting the formation of vitamin K dependant clotting factors (II, VII, IX and X). Daily dose requirements are highly variable between patients, ranging from < 1 mg/day to > 10 mg/day. This large variability is often interpreted as a leading problem with warfarin therapy. However it also indicates that doses are being successfully adjusted to meet the needs of individual patients. Herein lies the dichotomy between the desire for simplicity—one dose fits all, and the needs of our patients, where doses should be optimised to meet their specific requirements. Indeed from a standpoint of optimising care the warfarin dosing model is an excellent example of success. It is apparent that services that are set up for this purpose (individualising warfarin dosing) achieve better health outcomes for patients.

A related problem with warfarin is that its metabolic clearance has been found to vary between individuals due to genetic polymorphisms in cytochrome P450 enzymes (largely CYP2C9). It also interacts with vitamin K in the diet and vitamin K stores in the body resulting in variability in response. However we can measure a patient’s response to warfarin with the INR, an inexpensive test that can now be performed in the clinic or at home using a portable device.

INR results capture information about the patient’s individual response to warfarin and allow for rational dose adjustments. We could also measure a patient’s genotype to help predict a starting dose for warfarin, although this is not routinely performed in New Zealand and will only identify variability that arises from genetic differences between individuals.

Another issue is that warfarin may cause excessive bleeding after dosing that is higher than required and occasionally at the intended dose. This can be reversed by either withholding the drug or, if required promptly, administering vitamin K, and the addition of Prothrombinex-VF if urgent correction is required.
Unlike warfarin, it is claimed that dabigatran has a predictable pharmacokinetic and pharmacodynamic profile which allows for the use of fixed doses.\textsuperscript{15} Yet, the variability in dabigatran clearance, the pharmacokinetic parameter most important for determining the maintenance dose, has been reported to be in the order of 50% (coefficient of variation across the population)\textsuperscript{16} which is similar to the variability observed for warfarin clearance of 30–50%\textsuperscript{17,18}.

On this basis alone dabigatran exposure is not more predictable than warfarin. Indeed it might therefore be argued that if one dose does not fit all for warfarin then why would we expect this to be the case with dabigatran? As there is a correlation between dabigatran blood concentrations and efficacy and safety outcomes,\textsuperscript{19} unpredicted high or low blood concentrations could increase the risk of adverse events.

There is published evidence to indicate that dabigatran exposure differs predictably between individuals. The manufacturer reports that drug exposure was 1.5–6.3-fold higher in those with renal impairment compared to healthy subjects.\textsuperscript{20} Exposure to dabigatran may also be altered in patients with low body weight. The Pradaxa\textsuperscript{®} datasheet states that drug exposure was about 40–50% higher in female patients in primary VTE prevention studies.\textsuperscript{1} In atrial fibrillation patients, females had an average 30% higher trough post-dose concentration than male patients. We believe that these sex differences may relate to differences in body composition (e.g. lean body weight) between males and females. Therefore, dabigatran exposure in individuals at the extremes of body weight needs to be defined and evaluated.

Dagibatran is poorly absorbed orally because it is a substrate for P-glycoprotein,\textsuperscript{21} an efflux transporter responsible for limiting systemic xenobiotic exposure by pumping drug back into the gut. There are currently over 100 known polymorphic variants of the gene that codes for P-glycoprotein (ABCB1)\textsuperscript{22} and it is not clear what impact different genotypes will have on efflux function in many cases.

By contrast, the impact of altered hepatic enzyme activity on warfarin exposure is well understood and can be measured by determining the patients genotype.\textsuperscript{23} In addition, several drugs and drug classes have been found to inhibit P-glycoprotein\textsuperscript{22,24} such as amiodarone, atorvastatin, felodipine, verapamil, macrolides, some antifungals as well as foods such as with grapefruit and other citrus juices.\textsuperscript{25} Ingestion of these drugs and foods may result in elevated plasma dabigatran concentrations and an increased risk of bleeding.

**Monitoring dabigatran therapy**

Dabigatran shows varied effects on individual coagulation screening tests. There is limited sensitivity of the PT/INR, better sensitivity, though non-linearity at lower concentrations, of the aPTT, and marked sensitivity of the thrombin clotting time (TT).\textsuperscript{15} The Ecarin clotting time appears to be a slightly more sensitive test but availability is currently limited in New Zealand. Data suggests that three factors affect the test results: drug concentration, the patient’s intrinsic coagulation kinetics and the variability of the screening test(s).\textsuperscript{15} This situation is similar to variation in the INR testing of warfarin effect, despite strenuous international efforts to standardise the test results. Further research to validate a clotting time test for monitoring dabigatran effect is required. Research is also needed to establish guidelines for monitoring.
In the absence of a validated anticoagulation measure for dabigatran, the best widely available clotting time test for monitoring and dose individualisation is probably the aPTT. Published data indicate that prolongation of the aPTT at peak drug concentration (i.e. 2–4 hours post dose) will be around 1.9x, ranging from 1.6x–2.2x or 46–65s where the reference range is 24–34s (note that values will differ where the reference range differs). This may be useful in assessing peak dose effects to confirm the dose selected, particularly in low weight patients and in those whom P-glycoprotein function is known or suspected to be abnormal. Trough aPTT values (i.e. just before the next dose) may be useful for detecting significant drug accumulation in a renally impaired patient and where low body weight or metabolic factors lead to higher values. Trough aPTT values appear to be in the range 34–45s (reference range 24–34s) but this needs confirmation. The sensitivity of the aPTT for trough measurements will be poor at low dabigatran concentrations but can be detected by the thrombin clotting time (TT).

We suggest that monitoring may be appropriate in the following situations: initial dose individualisation in patients at risk of increased drug exposure and bleeding (aPTT), patients with deteriorating renal function (aPTT), and where a patient requires urgent/emergency surgery to detect the absence of residual drug (TT). Note that trough and peak monitoring will be time-dependent and when used will require patients to attend for sample collection at the required time pre- or post-dose.

If a patient receives sustained excessive dosing of dabigatran and experiences a bleed there is currently no antidote. Efforts to produce an inhibitor of dabigatran have been reported using monoclonal antibody technology but are still at an early stage of development. Other approaches to managing bleeding rely on non-specific and local measures as noted in the Pharmac guidance document for managing bleeds. Haematologist advice should be sought for reversing the effect of dabigatran for acute surgery or in the event of acute bleeding.

The fact that the monitoring of dabigatran therapy is currently not recommended should not be misinterpreted to mean that monitoring is not required. Although the published trial data for dabigatran indicates similar or lower bleeding rates in the selected trial participants compared with warfarin, this is not enough to claim that monitoring is not required. It is well established in medical ethics that where it is possible to reduce the risk of bleeding or thrombosis as a result of inappropriate dosing, by taking reasonable actions, these actions should be taken.

**Conclusions**

We believe there is a clear need for rational dose individualisation and monitoring guidance with dabigatran, particularly for patients at higher risk of bleeding. This would include the elderly, those at the extremes of body weight or with renal impairment and/or on drugs with potential interactions. Patients with moderate renal impairment should receive a smaller daily dose and, based on our current knowledge, the drug should be avoided in those with severe renal impairment (calculated Cockcroft-Gault GFR <30 ml/min).

There are some important questions that arise from the enthusiastic introduction of dabigatran. Is it reasonable to expect that any new anticoagulant could be safely prescribed at a fixed dose with no anticoagulation monitoring? In our opinion, no.
Indeed, we would not expect any anticoagulant used in therapeutic doses to achieve a high level of safety.

As noted, this is because of the thin line that exists between therapeutic anticoagulation and the risk of bleeding. Therefore, we propose that all new anticoagulants should be backed up by independent and relevant drug information at their time of launch and the need to individualise dosage should be the expectation. Is dabigatran a breakthrough in anticoagulation? In our opinion, yes. Dabigatran is a novel anticoagulant and we believe if used appropriately will add to our ability to meet the needs of our patients. Dabigatran is not, however, a game changer. We should not discard warfarin simply on the grounds that it may be more difficult to use in favour of a drug where: reversibility in the presence of acute bleeding has not been established, the drug has wide variability in its exposure from any given dose, and is a candidate for many drug interactions.

We contend that a wide range of prescribed dabigatran doses across the population would be a good indicator of our success in selecting doses to meet our individual patients’ needs. If we do not monitor anticoagulation effects how do we really know we are meeting our patients’ needs? A blindfold is not what the best dressed practitioner should be wearing.

Competing interests: James Faed is a paid member of the Committee of Haematologists convened by PHARMAC to advise on the release of dabigatran in New Zealand.

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References:


NZMJ 29 June 2012, Vol 125 No 1357; ISSN 1175 8716
Spontaneous gastroduodenal artery aneurysm rupture in acute surgery

Jessica Savage, Li Hsee

Abstract

Among the rarest of the visceral aneurysms, gastroduodenal artery (GDA) aneurysms often present with spontaneous rupture and are associated with a high mortality rate. Their aetiology is poorly understood. This report describes a case of haemorrhagic shock due to sudden GDA aneurysm rupture in a patient with a significant autoimmune history.

Gastroduodenal artery (GDA) aneurysms are one of the least common visceral artery aneurysms. Mortality is in the region of 30%. The aetiology is poorly understood. Many are the result of pancreatitis but true aneurysms are thought to be caused by arteriosclerosis, peptic ulcer disease, polyarteritis nodosa or Takayasu arteritis. This report describes a case of haemorrhagic shock due to GDA aneurysm rupture. The patient had a notable history of autoimmune disease.

Case report

This 67-year-old woman collapsed at home, with a 3-hour history of sudden onset, severe, epigastric pain radiating to her back. On arrival at the Emergency Department (ED) resuscitation room, she was tachycardic (110 bpm) and hypotensive (60/40 mmHg), requiring aggressive resuscitation. There was no history or sign of melaena, haematochezia or haematemesis.

Whilst in the ED, a bedside ultrasound scan showed free fluid in the subhepatic recess and pelvis, with a normal calibre abdominal aorta. Arterial blood gas demonstrated metabolic acidosis with an elevated lactate level of 5.4 mmol/L.

The patient had recently been diagnosed with multiple sclerosis, for which she had been commenced on low dose prednisone. She also has a history of ankylosing spondylitis and ulcerative colitis. There was no history of aspirin or non-steroidal anti-inflammatory use.

From the ED, the patient was transferred directly to the operating room. An urgent laparotomy was performed. The findings included a significant haemoperitoneum and a large retro-duodenal haematoma. Kocherisation of the duodenum allowed direct visualisation of an actively bleeding gastroduodenal artery. The bleeding vessel was oversewn and clipped. The patient was left with a temporary laparostomy.

Due to the acute nature of this case, there was no opportunity to take any intraoperative photos for the reader, nor was the GDA aneurysm large enough for resection and submission of histology. This surgery was performed by two consultant emergency surgeons and the diagnosis was made intraoperatively by direct visual identification.
Overnight in the Critical Care unit, the patient’s circulation normalised. The next day, she returned to the operating room for a re-look laparotomy, removal of packs and abdominal closure.

CT angiogram prior to discharge showed no evidence of other visceral aneurysms.

**Discussion**

Visceral aneurysms have been reported to have an incidence between 0.01% and 10% based on autopsy studies.²³ Splenic, hepatic and superior mesenteric aneurysms collectively comprise over 85%, whereas aneurysms of the gastroduodenal artery are generally considered one of the least common, representing just 1.5% of the total.²

Despite the infrequency of the occurrence of GDA aneurysm, rupture occurs in more than half and brings with it a 20–30% mortality risk.¹²,⁴ Often patients present in shock and thus the differential diagnosis of visceral artery aneurysm needs to be considered when assessing the shocked patient with abdominal pain.¹

The aetiology of visceral aneurysms is poorly understood. GDA aneurysms specifically have been attributed to pancreatitis and pancreatic surgery.¹² However, it has been estimated that as many as 8% of visceral aneurysms may be attributed to underlying disease, namely Ehlos-Danlos syndrome, fibromuscular dysplasia and polyarteritis nodosum.⁵

Other conditions to which visceral aneurysms may be linked include polymyalgia rheumatica, systemic lupus erythematosus, Takayasu arteritis, neurofibromatosis and Marfans syndrome.²³ Ankylosing spondylitis has been linked to aortic and coronary aneurysms,⁶ but no reports seem to previously link this to visceral aneurysms.

The proximity of the gastroduodenal artery to the first part of the duodenum, means it is more common for patients to present with upper gastro-intestinal bleeding due to erosion of this vessel by a duoduenal ulcer. However, as our patient showed no symptoms or signs of this, we suspect that autoimmune disease may be accountable for the ruptured gastroduodenal aneurysm seen at the time of laparotomy.

**Conclusion**

In summary, we present a recent case of gastroduodenal artery aneurysm rupture to promote discussion and highlight the importance of high clinical suspicion when faced with hypovolaemic shock and an acute-abdomen. We also believe this to be the first report to link GDA rupture to ankylosing spondylitis, supporting previous associations to autoimmune disease.

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Microfilaria in a facial mass—a coincidental finding in fine needle aspiration cytology

Dilip C Barman, Tapan D Bairagya

Clinical—A 42-year-old non-smoker male patient presented with a large swelling over the right side of the face around the orbital region for 7 months duration; the swelling was gradually increasing in size and painful.

On examination, it was 8 cm × 5 cm, fixed to deeper structure, had an irregular surface, and displaced his nose to the left side. The right eye was completely disfigured. The overlying skin shows ulceration and crusting. There was serosanguinous discharge from the mass.

Opposite eye was absolutely normal (Figure 1). There was no cervical lymphadenopathy. Other systems were also normal. Peripheral blood examination revealed eosinophilia with low haemoglobin level (Hb: 9 gm/dl).

Fine needle aspiration cytology (FNAC) of the swelling revealed microfilaria with a clear space at the cephalic and caudal ends and areas of undifferentiated tumour cells in dyscohesive clusters in a haemorrhagic background (Figure 2). It was purely a coincidental finding as there was no suggestive clinical history of filariasis.

Figure 1. The mass over the right side of the face
Figure 2. FNAC of the mass revealed microfilaria (left image) and undifferentiated tumour cells (right image) [Leishman’s stain, ×40]

Discussion—Filariasis is a major public health problem in a tropical country like India. It is transmitted by the Culex mosquito and caused by two closely related nematodes: Wuchereria bancrofti and Brugia malayi. Infective larvae penetrate the feeding wound in the skin, enter the lymphatics and travel to the regional lymph nodes.

Once fertilised, the female discharges several thousand microfilariae (150–300 μm), which dwell in the peripheral blood for 5–10 years. Despite the high incidence of filariasis, microfilaria in FNAC is not a common finding. There are reports of single or small number of cases of microfilariasis at various sites such as lymph node, breast lump, bone marrow, bronchial aspirate, nipple secretions, pleural and pericardial fluid, ovarian cyst fluid, and cervicovaginal smears. One proposed mechanism in this finding is rupture of lymphatic vessels and liberation of microfilaria within the mass.

In the medical literature of microfilaria with malignant neoplasm we have found some case reports describing coexistence of microfilaria with primary malignant tumour.

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Assessment of health and potential for milk based intervention to improve the nutrient intake of toddlers in New Zealand

Nutrition remains a key determinant of child health globally. Childhood malnutrition, both macronutrient over-nutrition and micronutrient under-nutrition is prevalent in New Zealand (NZ). NZ has a large burden from communicable diseases for which, in the developing world, malnutrition is known to play a causal role. Deficiencies in iron and iodine, micronutrients that play central roles in brain development, cognition and learning, are prevalent in NZ during infancy.

Community based nutrition interventions have been used successfully in developing and developed countries to prevent and treat micronutrient deficiencies. Such interventions have also been shown to prevent the adverse health effects that result from micronutrient deficiency. We recently examined the health of young children living in a socio-economically deprived urban region and completed the piloting necessary for a subsequent trial of a milk-based nutritional intervention.

Methods—Thirty-eight children aged 9 to 18 months (mean age 15 months) who were enrolled with the Tamaki Primary Health Care Organisation in Auckland, NZ were recruited between July and October 2008. Data were collected at face-to-face study enrolment and completion interviews with each child’s caregiver, at weekly telephone caregiver interviews, by abstraction of data from health care records and by assessment of the child’s growth, development and middle ear function.

Data collected included demographics, dietary intake (food frequency questionnaire), middle ear function (tympanometry), development (Bayley Scales of Infant Development III), communicable disease episodes and health care utilisation (parental report and primary care record review). Ethical approval was obtained from the Northern Y Regional Health & Disability Ethics Committee.

The children were randomised to receive either a micronutrient-fortified fresh milk (N=17) or powdered formula (N=19) based intervention. The fresh milk product was Meadowfresh Junior (Goodman Fielder). The formula product was Karicare toddler (Nutricia, New Zealand). Each 500 ml of the fresh milk product provided approximately 50% of the recommended daily intake of iron, zinc, iodine and vitamin D. Each 500 ml of the powdered milk formula contained 25 to 50% of the recommended daily intake of 16 vitamins and minerals. Both milk interventions were provided for three months.

Data analyses were undertaken using the JMP v5.1 software (SAS Inc. NC, USA). Differences in proportions of categorical variables were investigated by the Fisher’s exact test.
Results—Sixteen (44%) of children were male. The boys had a mean height of 81 cm (+1 SD height-for-age relative to WHO reference population) and a mean weight of 11.9 kg (+1.4 SD weight-for-age). The girls had a mean height of 82 cm (+1.7 SD height-for-age) and a mean weight of 11 kg (+1 SD weight-for-age). The enrolled sample was ethnically diverse (28% Māori, 28% Pacific, 22% Asian, 17% NZ European and 6% of other ethnicities). Children were followed for a median of 78 (48–97) days.

Three-quarters (72%, 26/34) of the children had abnormal tympanograms for one or both ears with abnormalities persisting up to 20 weeks. Fifteen children (44%) with persistently abnormal tympanometry were referred to the paediatric otorhinolaryngology clinic. All children had developmental testing completed. Bayley Scale III mean cognitive and language scores were one standard deviation below the reference population mean. The mean motor and socio-emotional scores approximated the reference population mean.

At weekly interviews, coughing was reported in 23 (64%) of the children, wheezing in 15 (42%), cold or flu symptoms in 25 (69%), sneezing or rhinorrhoea in 32 (89%), snoring in 20 (56%), ear infection in 9 (25%) and gastrointestinal symptoms in 16 (44%). The children experienced a mean of 3.4 days/month of coughing, 1.2 days/month of wheezing and 1.2 days/month of gastrointestinal symptoms. The children made between 0 and 42 primary health care visits. Forty-seven percent had made 11 or more visits since birth. Almost two thirds (65%) of visits were for respiratory illnesses including otitis media.

Twenty-two (61%) of the children consumed an average of 500 to 600 mls/day and parents reported that both milk interventions were acceptable. The milk volume consumed did not differ between the 2 groups. Intake of other nutritious foods from the major food groups as recorded in food frequency questionnaires did not decrease over the interval that the children received the milk intervention.

Discussion—This pilot study highlights the poor health status of young NZ children living in a socio-economically deprived urban region. These data suggest concerning rates of middle ear disease, respiratory symptoms, high primary health care usage and poor developmental assessment results. There is a paucity of contemporary data on the health of children in NZ from a primary care perspective. Our study shows the largest symptom and disease burden in our children was from respiratory illnesses.

Both fortified fresh and powdered formula milk were acceptable to the mothers and consumed in sufficient quantity to provide 25 to 50% of recommended micronutrient intakes. The provision of this milk did not reduce the consumption of other nutritious foods.

In order to improve child health, New Zealand needs to consider developing policy that helps to secure a nutritious diet for young children. The findings from this pilot study indicate that micronutrient fortified milk is a potentially important component of such a diet.

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References:


New Zealand’s smokefree prison policy appears to be working well: one year on

A comprehensive smokefree prisons policy was introduced in New Zealand a year ago (1 July 2011). The impression is that this policy appears to have been very successful so far. Initial concerns about the feasibility of establishing smokefree prisons seem to have been overridden by the reported smooth transition, from 67% of the prison population previously being smokers to a situation of a fully smokefree environment.\(^1\)

The policy was reported to have been met with cooperation and even enthusiasm from many prisoners across the country.\(^2\) In this letter, we review evidence from the media, Government departments, the scientific literature, and other sources, to describe how the policy was introduced, it’s likely effect, and explore its implications for public health and tobacco control.

We believe New Zealand is the first country in the world to implement a comprehensive country-level smokefree policy for all staff, prisoners and visitors within prison premises, indoors and outdoors (with 8690 prisoners at March 2012).\(^3\) Sweden had previously initiated a partial policy, affecting only the indoor prison environment; however, this policy was not sustained after a legal challenge.\(^4\) Other smokefree prison policies have been introduced overseas (such as various US states and Canadian jurisdictions), but these have not been country-level policies.\(^5,7\)

Overseas experience has tended to show poor results for achieving smokefree prisons. Evaluation of an indoor smokefree policy in a Canadian prison reported that 93% of inmates continued to use tobacco products inside.\(^7\) A smokefree prison policy in Taiwan was resisted by inmates, with mixed compliance, as staff were permitted to continue to smoke at work.\(^8\)

The introduction of smokefree prisons Overseas have also regularly been met with the emergence of a black market for tobacco.\(^9-11\) There was an initial rise in tobacco contraband in the first 2 months following the introduction of the New Zealand policy and the black market price of tobacco doubled;\(^12\) however prisons enhanced their methods for checking and stopping contraband entering, and no further tobacco related problems have been reported since.

Another initial reported problem was allegation of some prisoners attempting to smoke their nicotine patches mixed with tea leaves.\(^13\) However, there have been no further reports of this nature, and it is unclear if this is an ongoing issue.

Three factors were likely to have contributed to the widespread acceptance of smokefree prisons in New Zealand. First, the comprehensive preparation provided by both the Department of Corrections and individual correction facilities; second, the availability, range and standard of smoking cessation support services; and third, the opportunity to learn from overseas experience and enact a comprehensive policy (covering both indoors and outdoors) as opposed to a partial policy.
Preparation for the smokefree policy consisted of a year-long lead-in period. During this time, prisoners were provided with educational materials which outlined the health risks of smoking along with advice on how best to quit. After the proposed smokefree policy was announced and prior to its implementation, 2000 prisoners started nicotine replacement therapy (NRT).

Six voluntary smokefree units were established across the country up to 9 months before the policy was enforced, receiving unexpected support from inmates. Tobacco sales were outlawed in prisons a month before the full smokefree prison policy came into effect. Police stations also promoted the smokefree prisons policy in advance.

Smoking cessation services available to inmates have consisted of both pharmacological and behavioural support. These have included NRT, access to a national free-phone service (Quitline), access to cessation guidance books and assistance from health care staff trained in smoking cessation support. While there were initial concerns over the level of cessation support available for prisoners prior to the policy implementation, extra activities were provided as part of the smoking cessation programme including: sporting events, exercise initiatives, cultural activities and art classes. In one correctional facility, prisoners were provided with healthy snacks (carrot sticks) to assist with withdrawal symptoms.

An important observation, noted from other studies, is that policies that have enforced a 100 percent smokefree environment tended to face fewer problems than “indoor only” policies. The smokefree prison policy introduced in New Zealand prohibits smoking within the entire prison premises (both indoors and outside), thus making the policy easier to enforce.

Evidence from the US suggests that poor compliance with a smokefree prison policy is associated with a lack of strict enforcement from staff who object to the rules. Fortunately, in New Zealand, Corrections staff have been co-operative with the policy. This may partly be explained by the Department of Corrections having sponsored the development of “Workplace Champions”, a voluntary designated staff member, who was provided with smoking cessation training, with the intention of supporting colleagues and prisoners to quit, both before and after the policy was introduced.

The aims of the New Zealand smokefree prison policy are to make prisons both healthier and safer, primarily to reduce secondhand smoke exposure and risk of fires. Staff working in prisons without smokefree policies are exposed to high levels of secondhand smoke exposure. An Irish study showed that 44% of non-smoking prison workers had carbon monoxide levels in respired breath equivalent to those of a light to heavy smoker.

Studies of indoor air quality in prisons before and after smokefree policies have shown a significant decrease in nicotine concentrations in ambient air. Indeed, recent evaluation work in an Auckland prison showed indoor air pollution levels (of fine particulates associated with second-hand smoke) to have halved as a result of the new policy.

The smokefree prisons policy appears to have reduced the risk of fires. Within a month of the introduction of the policy, the number of arson-related incidents in
prisons dropped. The month before the policy was introduced 18 fires and arson-related incidents occurred compared to only four in the month after the policy was introduced, and only one the following month.\textsuperscript{29} This was likely to result from the prohibition placed on cigarette lighters, which accompanied the tobacco ban.

Smokefree prisons policies have occasionally been associated with riots\textsuperscript{30,31} and an increase in inmate violence.\textsuperscript{32} One New Zealand prison was reported to have an increase in violence between prisoners in the month following the introduction of the policy.\textsuperscript{33} The number of serious assaults in prisons since the implementation of the policy are yet to be reported.

International evidence suggests that the re-uptake of smoking once leaving a smokefree prison is high.\textsuperscript{34} Fortunately in New Zealand, relapsed smokers can get quitting support from the Quitline and many other health service providers. Relapse risk in the community will also be lowered as the price of tobacco continues to rise (with multiple tobacco tax rises planned by the current Government).

There are also other supportive environmental measures being planned on the country’s path towards achieving the “Smokefree Nation 2025” goal, such as the requirement for plain (or standard) packaging, being introduced in Australia. However, we believe the smoking relapse rate for released prisoners should be quantified and their needs for services to support them remaining smokefree assessed. Such evidence would help evaluate whether current quit support for prisoners on leaving prison is adequate, and help identify areas where further assistance may be needed.

The World Health Organization Framework Convention on Tobacco Control introduced Article 8 in 2007 for countries to protect their citizens from secondhand smoke in indoor public places and workplaces.\textsuperscript{23} Five years on, it appears that New Zealand is the first country to have successfully addressed these issues in all their prisons. The available evidence suggests that the policy has been successfully introduced, with no evidence of the problems reported in other jurisdictions.

Objective indicators have shown both improved indoor air quality\textsuperscript{17} and reduced incidence of fires\textsuperscript{29} after the policy was introduced.

Nevertheless, we believe that a more in-depth evaluation of this policy is now desirable to inform other smokefree developments in New Zealand, but also to assist the introduction of national smokefree prison policies in other countries. A systematic evaluation of this tobacco control intervention could include a more comprehensive assessment of air quality expanding the previous assessment beyond a single prison, surveying staff and prisoners, measuring health indicators amongst staff and inmates, further determining trends in fires, and assessing whether smokefree prisoners remain so after their release back to their communities.

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References:


Majority support among the public, youth and smokers for retail-level controls to help end tobacco use in New Zealand

The New Zealand Government has recently committed to the goal of making New Zealand (NZ) smokefree by 2025. There are growing discussions about the type and mix of measures needed to achieve the 2025 goal. Specific interventions have been suggested by: the Māori Affairs Select Committee (MASC) Report, a commentary on the report, a ‘Next Steps’ document from the National Tobacco Control Working Group, and a Ministry of Health internal briefing document released through an Official Information Act request.

Many of these suggestions reflect the need to target the retail availability and promotion of tobacco products, part of the increasing interest in the supply side of tobacco control. For example, the MASC Report recommended banning all retail displays of tobacco products, mandating the sale of nicotine replacement therapies (NRT) wherever tobacco is sold, increasing penalties and enforcement of bans on sales to minors, requiring all retail staff selling tobacco to be over 18 years, banning tobacco vending machines, and investigating giving Local Authorities powers to restrict the number and location of tobacco retailers in order to reduce children’s exposure to tobacco products. It also discussed making retail premises where tobacco is sold accessible only to adults aged over 18 years, but did not make a specific recommendation to this effect. The ‘Next Steps’ document further recommended mandatory registration of all tobacco retailers as an initial step towards controls on retailers, and the consideration of banning duty free sales of tobacco.

While the Government’s response to the MASC Report accepted the ‘aspirational’ goal of “reducing smoking prevalence and tobacco availability to minimal levels, thereby making New Zealand essentially a smoke-free nation by 2025”, it accepted only one retail environment recommendation in full. The Smoke-free Environments (Controls and Enforcement) Amendment Act 2011 will result in the removal of point-of-sale (POS) tobacco displays from 23 July 2012.

Other recommendations were accepted partially, deferred for further investigation or rejected. The Government will consider requiring tobacco retailers to be aged over 18 years if additional evidence of likely impact emerges. It will investigate reducing duty free sales allowances for travel with major tourism partners and giving Local Authorities powers to restrict the numbers and location of retailers, but rejected banning vending machines and mandating sales of NRT where tobacco is sold. Overall, the Government’s response has so far resulted in only a minor increase in enforcement monitoring illegal sales to minors.

We have reviewed and synthesised support for the long-term goal of reducing tobacco availability as well as specific tobacco control interventions in the retail sector (Table 1). We excluded the removal of point-of-sale displays as this intervention comes into effect in July 2012, though we note the strong support for this measure from both the public and some retailers.
Table 1. Summary of the results of New Zealand studies that consider attitudes to retail-level tobacco control interventions in New Zealand

<table>
<thead>
<tr>
<th>Survey results/ date published</th>
<th>Participants</th>
<th>Reducing the number of places that can sell tobacco</th>
<th>Other retail tobacco control interventions</th>
<th>Banning sales in 10 years’ time</th>
</tr>
</thead>
<tbody>
<tr>
<td>International Tobacco Control Survey, NZ arm (2009/2010)[11-13]</td>
<td>Smokers: A national sample of adult smokers with boosted sampling of Māori, Pacific and Asian New Zealanders. N=1376 (Wave1) N=923 (Wave 2)</td>
<td>Most (55.2%) agreed (while 38.8% disagreed), that the number of places allowed to sell tobacco products should be reduced gradually to make them less easy to buy (Wave 2). Support was highest among Māori (61.6%) and Pacific (75.1%) smokers.</td>
<td>Most (61.9%) agreed (34.1% disagreed), with restricting tobacco sales to dedicated outlets where children are not allowed to go (Wave 2). Support was highest among Māori (66.6%) and Pacific (83.4%) smokers.</td>
<td>Similar proportions agreed (46%) and disagreed (47%), that if effective nicotine substitutes became available, the Government should ban cigarette sales in 10 years’ time (Wave 1). The highest support was among Pacific smokers (62.3%).</td>
</tr>
<tr>
<td>Health Sponsorship Council – 2008 Year 10 In-depth survey (2009)[15]</td>
<td>14 and 15 year old smokers and non-smokers: N=3036 (420 current smokers)</td>
<td>Not applicable.</td>
<td>Around half (53.1%) of students agreed with the statement that tobacco companies should not be allowed to sell their products in the dairy at the checkout. Agreement was higher for non-Māori than for Māori (39.3% vs 29.7%) and for never smokers compared to current smokers (63.3% vs 22.0%).</td>
<td>Not applicable.</td>
</tr>
<tr>
<td>Survey results/ date published</td>
<td>Participants</td>
<td>Reducing the number of places that can sell tobacco</td>
<td>Other retail tobacco control interventions</td>
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<tr>
<td>Health Sponsorship Council – National 2008 Health and Lifestyle Survey (2010) ¹⁶,¹⁷</td>
<td>Adult smokers and non-smokers: N=1608 (n=422 smokers)</td>
<td>Around two-thirds (65.6%) agreed that the number of places selling cigarettes and tobacco should be reduced to make these products less easily available.</td>
<td>Not applicable</td>
<td>Around half 49.8% agreed that cigarettes and tobacco should not be sold in NZ in 10 years’ time, while 30.3% disagreed. 47.9% Māori agreed, 34.9% disagreed. 59.7% Pacific peoples agreed, 19.7% disagreed. 26.2% of smokers agreed, 55.3% disagreed.</td>
</tr>
<tr>
<td>Health Sponsorship Council- National 2010 Health and Lifestyle Survey (2011) ¹⁷,¹⁸</td>
<td>Adult smokers and non-smokers: N=1740 866 European/Other 460 Māori 301 Pacific peoples 113 Asian</td>
<td>Around two-hirds (67%)* agreed that the number of places selling cigarettes and tobacco should be reduced to make them less easily available (29% strongly agreed, 38% agreed)*. Never-smokers, people living in low deprivation status neighbourhoods, women and people with a university qualification were more likely to agree.</td>
<td>Not applicable</td>
<td>A minority (43%)* agreed that cigarettes and tobacco should not be sold in NZ in 10 years’ time Never-smokers, Pacific peoples and women were more likely to agree with this statement. A minority (42% overall)* either agreed (27%) or strongly agreed (16%), that cigarettes and tobacco are too dangerous to be sold at all.</td>
</tr>
<tr>
<td>Action on Smoking and Health (ASH) Annual Year 10</td>
<td>14 and 15 year old smokers and non-smokers:</td>
<td>Two-thirds (66.6%) agreed that the number of places selling cigarettes and tobacco should be reduced to</td>
<td>Not applicable</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>
| Snapshot Survey 2009 (2012)³⁹ | N=24,495 (2599 current smokers) | make them less easily available. (17.6% disagreed, 15.7% didn’t know)  
Students who did not have a parent who smoked were more likely to agree. |

*Results only reported to 0 decimal places.*
The results shown in the table indicate that there is strong majority support among smokers, the general adult population, and 14 to 15-year-olds for reducing the number of tobacco retailers. There was also majority support among smokers for restricting places that sell tobacco to premises that exclude children and that make cessation products available. We did not find any data on the level of support for other measures such as licensing tobacco retailers, restricting duty-free sales, and requiring that tobacco retailers are over 18 years of age. There were no quantitative data on the level of support for tobacco control interventions in the retail sector among retailers.

There was substantial levels of support for ending sales of tobacco products altogether within 10 years, though among smokers this was when the caveat was added to the question that effective nicotine substitutes became available. Support for tobacco control measures in the retail setting and for ending the sales of tobacco products were higher among Pacific peoples, with mixed findings for Māori compared to non-Māori.

Public support for proposed policy options often influences politicians’ willingness to introduce new tobacco control measures, and so it can be useful for researchers to study these issues. This may be particularly true for health issues like tobacco control policies, where policy introduction and implementation is often highly contested by the tobacco industry and their commercial allies; and where some politicians can be ideologically trapped by their opposition to the regulation of markets. The evidence presented above from recent New Zealand surveys has shown that smokers, non-smokers and youth support many of the MASC recommendations to reduce tobacco supply and retail availability.

These levels of support are high given that the surveys occurred before there had been substantial public debate about retail-focused tobacco control measures other than removal of point-of-sale displays. The usual pattern observed is that support for tobacco control measures increases greatly following such debate and policy implementation. For example, this was observed with smokefree bars and restaurants with the introduction of the 2003 Smoke-free Environments Amendment Act.20

Achieving the goal of a smokefree New Zealand by 2025 will require radical action.5 Despite the logic of supply-focused interventions, reducing the availability of tobacco products is an underdeveloped facet of tobacco control worldwide.6 7 This is also the case in New Zealand in the Government’s current approach to achieving the “Smokefree Nation 2025” goal. The findings of this study suggest that public opinion is running far ahead of the policy process, and that urgent consideration should be given to implementing the MASC report’s and National Tobacco Control Working Group’s recommendations on tobacco control interventions in the retail setting.

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Use of a reminder sticker improves rates of documentation of resuscitation status and the appropriate prescription of venous thromboembolism prophylaxis

We aimed to investigate the effect of a reminder sticker, placed in the patient chart at the time of the post-acute ward round, on the documentation of resuscitation status and appropriate prescription of venous thromboembolism (VTE) prophylaxis in adult general medical patients at Auckland City Hospital.

The adult general medical service at Auckland City Hospital consists of four ward-based teams. The sticker was trialled on the Red team with the White team acting as the control group.

The sticker contained contact details of the medical team and reminders about documenting resuscitation status, prescribing VTE prophylaxis and retaining or removing intravenous cannulae.

Before the introduction of the sticker the charts of 100 consecutive patients admitted Monday to Friday under both teams were reviewed in the afternoon following the post acute ward round. Both teams were blinded to this review.

The charts were audited for documentation of resuscitation status and the appropriate prescription of VTE prophylaxis (the VTE prophylaxis guideline for medical patients in the Auckland City Hospital RMO Handbook was used to adjudicate this).

We did not audit whether intravenous cannulae were necessary or unnecessary as we had previously shown that the use of a reminder sticker could improve the removal of unnecessary intravenous cannulae.\(^1\)

Both teams then received a teaching session highlighting the importance of documenting resuscitation status and the appropriate prescription of VTE prophylaxis. The Red team also received education about placement and completion of the sticker. Both teams were aware of the sticker and that rates of documentation of resuscitation status and appropriate prescription of VTE prophylaxis would be audited.

The nurses responsible for the Red team patients were asked to remove a patient’s intravenous cannula if the sticker requested this.

The sticker was introduced in October 2009. One week later, the charts of 100 consecutive patients admitted under both teams were again audited as above and the same information was collected.

The Red team patients' charts were also audited for presence and completeness of the sticker. The Red team patients whose sticker stated “please remove intravenous cannula” were reviewed for the presence or absence of an intravenous cannula.

The two-tailed Fisher’s exact test was used to calculate univariate p values. Ethical approval was granted by the Northern X Regional Ethics Committee.
Documentation of resuscitation status for the Red team patients improved from 79% in the pre-intervention period to 99% in the intervention period (p<0.0001) whereas for the White team patients was unchanged at 92% in both periods (p=1).

Prescription of appropriate VTE prophylaxis for the Red team patients improved from 39% in the pre-intervention period to 73% in the intervention period (p<0.0001) whereas for the White team patients fell from 35% in the pre-intervention period to 9% in the intervention period (p<0.0001).

During the intervention period the sticker was present in 76 Red team patient charts and was complete on 63 (83%) occasions.

The sticker asked for the removal of an intravenous cannula in 21 patients. When reviewed, a median of five hours after the sticker had been placed, this cannula remained in situ in 9 (43%) patients.

The use of this reminder sticker was associated with a statistically significant improvement in rates of documentation of resuscitation status and appropriate prescription of VTE prophylaxis. The sticker may have resulted in the removal of a number of unnecessary intravenous cannulae and has the potential to result in the removal of further unnecessary intravenous cannulae if nursing staff respond to the sticker request more often.

Reminder stickers have been shown to be beneficial in a wide variety of areas of medical care including prescription of VTE prophylaxis, appropriate perioperative antibiotic prescribing, cancer screening in primary care and smoking cessation.2-5

There was an unexpected significant decrease in the rate of appropriate prescription of VTE prophylaxis in the White team during the intervention period. It is possible that the White team physicians views of VTE prophylaxis may have been influenced by an article addressing the benefit-hazard ratio of VTE prophylaxis in medical patients that was published between the pre-intervention and intervention periods.6

With our study design, we felt that we were able to assess the true impact of the sticker. This audit has a number of limitations. We have only shown that this sticker is beneficial over a short period of time. It is uncertain as to whether this benefit will be maintained over a more prolonged period.

The removal of unnecessary intravenous cannulae is reliant on nursing staff reading and following the sticker request. There were unavoidable changes in the medical personnel of both the Red and White teams between the pre-intervention and intervention periods due to the regular rotation of registrars and house officers.

We plan to introduce this reminder sticker across the adult general medical service at Auckland City Hospital and to reaudit rates of documentation of resuscitation status and appropriate prescription of VTE prophylaxis after a more prolonged period of use.

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References:

Improving hand hygiene compliance in New Zealand hospitals to increase patient safety and reduce costs: results from the first national hand hygiene compliance audit for 2012

The data presented by Roberts et al in the 11 May 2012 issue of the *NZ Medical Journal* show that even modest improvements in hand hygiene compliance by hospital healthcare workers can lead to significant reductions in healthcare-associated *Staphylococcus aureus* bacteraemia rates.¹ This conclusion is supported by recently published data from the first 2 years of the Hand Hygiene Australia programme. Their data showed that an increase in hand hygiene compliance from 46% to 63% nationally (excluding the State of Victoria) was associated with a significant reduction in the national incidence of MRSA bloodstream infections.²

These reports contribute to the growing body of evidence suggesting that successful hand hygiene programmes improve patient outcomes and reduce healthcare costs.³⁻⁶ In New Zealand in 2005 the financial cost of each healthcare-associated bloodstream infection was approximately $20,000 and in 2003, the overall cost of healthcare associated infections to the New Zealand healthcare system was estimated to be NZD $140 million.⁷⁻⁸

Given these figures, it is not surprising that in one study it was concluded that every time an individual healthcare worker *fails* to perform hand hygiene at an appropriate “moment” during patient care, the cost to the healthcare system is somewhere between US$2 and US$50.⁹

With these facts in mind, we report national hand hygiene compliance data for the first quarter of 2012 and compare the results to those obtained by Roberts et al.

For the first auditing period of 2012 (ending 31 March) 10 DHBs submitted data: The total number of moments audited was 11,298 and correct hand hygiene was performed on 7356/11298 occasions, giving an overall compliance rate of 65%. When examined by healthcare worker category, medical practitioners had the lowest rate (54.8%; 95%CI: 52.5⁻57.0) and phlebotomists had the highest rate (71%; 95%CI: 66.1⁻75.6). When examined by each of the WHO 5 moments; higher rates were observed for “after” moments than “before” moments (“before patient contact” 60% versus “after patient contact” 74%; and “before a procedure” 55% versus “after a procedure or body fluid exposure risk” 71%).

Whilst a compliance rate of 65% indicates that hand hygiene in New Zealand hospitals has considerable room to improve, it should be remembered that this is the first audit since the HHNZ programme was reinvigorated in late 2011 under the auspices of the Health, Quality & Safety Commission (the Commission). For this reason, the 10 DHBs that submitted data should be commended for their efforts to improve their hand hygiene practice and for their ongoing commitment to the national programme.
As pointed out by Roberts et al, changing institutional culture with respect to hand hygiene practice is a gradual process and one that requires commitment from the highest levels of hospital management. Moreover, such commitment must consist of more than endorsement of the programme; DHB CEOs and managers who are serious about improving hand hygiene compliance need to provide the modest investment of resources necessary to allow meaningful participation in the national programme.

For example, resources need to be provided at DHB level to ensure that sufficient numbers of auditors have been trained and secondly to ensure that once trained, auditors are provided with sufficient time outside of their normal workload to perform their auditing duties. Similarly, resource needs to be provided to ensure hand hygiene coordinators have sufficient time to feed back audit results, organise educational programmes, and to organise strategic promotional activities.

Nonetheless, although investment by senior management is necessary to improve hand hygiene compliance, it is not by itself sufficient to ensure success. It is also essential to gain the support and commitment of senior opinion leaders in the hospital (such as senior doctors). Senior opinion leaders play a huge role in determining institutional culture, largely by influencing their more junior medical colleagues who in turn behave as role models to other healthcare workers. However, the power of senior opinion leaders to influence others by their example is a double-edged sword.

Just as positive role modeling can have a very positive impact on the hand hygiene practice of others negative role modeling can undo a lot of hard work. Unfortunately, the fact that among all healthcare worker groups, the lowest compliance rates were observed among medical staff (52%) indicates that when it comes to hand hygiene practice, many doctors are not currently functioning as the role models they ought to be.

Finally, the national auditing data indicate higher compliance with the “after” moments than the “before” moments. This is consistent with data reported by Roberts et al and reflects the common misperception among healthcare workers that performing hand hygiene is primarily for self-protection rather than for the safety and protection of patients.

Hand hygiene education programmes, therefore, need to strongly emphasize that hand hygiene is about patient safety. In a recent study, framing hand hygiene education programmes in this way was reported to lead directly to substantial improvements in compliance.10

In summary, participation in the HHNZ programme offers an opportunity for CEOs and management to improve patient safety while reducing financial costs. Encouragingly, many DHBs have already made the necessary investment, although recent auditing data indicate there is still scope for improvement. Of all healthcare worker groups it is senior doctors and opinion leaders in particular who need to improve their role modeling, doing so will help provide much needed leadership in this area.

Finally the primary purpose for performing hand hygiene is to improve the safety of our patients. This is a simple but key message and one that deserves repeated emphasis.
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References:

Editorial: genius and insanity

Excerpt from Editorial published in 1912 March issue of the NZMJ.

"THE question of race culture, or, as it is now called, eugenics, is one of great perplexity. No doubt, it is an admirable aim to attempt to eliminate the production of the unfit, and no one but a reactionary and obscurantist will deny that much can be done to this end, mainly by education, but also by slight degrees of compulsion.

There is, however, a great danger that harm, as well as good, may come if some of the main principles of race culture are carried into effect, and it may well be the better course "to bear those ills we have than fly to others that we know not of."

The enforcement of eugenic principles would have robbed the world of intellectual giants. The greatest men, with rare exceptions, have sprung from neurotic stock, and, in very many instances, genius has been allied to insanity. Aristotle was one of the first to point out that often great men displayed morbid mental symptoms, and late writers, for example, Moreau, have contended that genius is essentially a neurosis.

It is near the truth to say that genius cannot be explained, that it certainly is not essentially a mental aberration akin to insanity, is the product of no class and no system, and is very rarely transmitted The average man's lamp of reason burns steadily if not brightly—there is neither fitful gleam nor dazzling flash.

Darwin could not explain the cause of spontaneous variations in the lower animal kingdom where the law of survival of the physically fittest generally prevails, and in the human race, the variations of mental development pass far beyond the range of our understanding. There are more things in Heaven and earth than are dreamt of in the evolution theory, and Wallace has admitted that the "noblest and most characteristic of human faculties" do not appear to come under the Darwinian law.

The greatest men in Art, Philosophy, and Literature (and Science, also, in a less degree), are nearly all the subjects of nerve disorder, but, for our present purpose, we may confine our attention to authors and poets.

Swift, once observing a wayside tree blasted by lighting, said to a friend: "I shall be like that tree I shall die at the top." His vicious courses, his irresponsibility, his uncontrollable fits of temper, facial twitchings and giddiness, and the hereditary taint in his family were sure signs to him that his reason reeled, and indeed he finally came under the charge of a keeper.

That great and good man, Dr. Johnson, inherited "a vile melancholy," and was obsessed by two fears all his life, the fear of madness, and the fear of death and the bondage of the grave.

Goldsmith's reason might have tottered upon her throne had it not been for the solace of Samuel Johnson's approval.

Cowper varied between suicidal impulses and religious melancholia.
There was insanity in Southey's family, and although the poet himself escaped, he was the "excitablest man" Carlyle had ever met.

Shelley suffered from hallucinations, and Byron wrote "some curse hangs over me and mine." His life was blighted by heredity, and the fear of madness possessed him all his life.
Warfarin or aspirin in patients with heart failure and sinus rhythm

It is generally acknowledged that warfarin is much more efficacious than aspirin in the prevention of ischaemic strokes in patients with atrial fibrillation. This report concerns a randomised trial comparing warfarin and aspirin treatment in patients in sinus rhythm who have a reduced left ventricular ejection fraction. They followed 2305 patients for up to 6 years. The primary outcome was the time to the first event in a composite end point of ischemic stroke, intracerebral haemorrhage, or death from any cause.

Their conclusions were that among patients with reduced LVEF who were in sinus rhythm, there was no significant overall difference in the primary outcome between treatment with warfarin and treatment with aspirin. A reduced risk of ischemic stroke with warfarin was offset by an increased risk of major haemorrhage. The choice between warfarin and aspirin should be individualised. The daily dose of aspirin was 325mg and we could speculate whether a lower daily dosage might have been better or worse.


Angiotensin receptor blockers and risk of cancer

The authors of this paper note that data from randomised clinical trials have suggested an increased risk of cancer with angiotensin receptor blockers, but the most recent and comprehensive meta-analysis found no association. Their report concerns a cohort study involving 377,649 individuals, comparing the effects of exposure to angiotensin receptor blockers or ACE inhibitors between 1995 and 2010. The information was derived from UK primary practices. They report that “we found no overall association with cancer; we detected small absolute risk increases for breast and prostate cancer, but the results did not support a causal effect”.


Haemodynamic effect of nebulised frusemide in stable, advanced heart failure

The authors of this paper have previously reported on the favourable effects of nebulised frusemide in a patient in whom intravenous access was not attainable. They reported that after administering 80mg of nebulised frusemide, there was an immediate improvement in oxygen saturation, the chest was clearer on auscultation and there was increased diuresis. In order to assess the haemodynamic effects of such treatment they have performed a trial in which 32 patients with stable advanced heart failure were randomised to receive either 40mg (4ml) of nebulised frusemide or 4ml of normal saline. Ten haemodynamic functions were studied and no differences were
found between the frusemide and saline cohorts. As expected urine output was significantly increased in the frusemide treated cohort.


**Masked hypertension in hypertensive patients treated in a primary care setting**

This paper from Canada notes that it is now increasingly recognised that uncontrolled hypertension is overlooked in patients with normal office BP (OBP) but high home BP, a phenomenon termed masked hypertension (MH). Their study involved OBP measurement at baseline and after 3 months of valsartan-based therapy in 5636 hypertensive patients who had recorded their home blood pressure monitoring (HBPM) for 7 consecutive days at month 3 using an Omron HEM-711 apparatus. Their findings were that one of five hypertensive patients and more than one of three with controlled OBP will have MH. MH is associated with other cardiovascular risk factors, such as diabetes, and in non diabetics, with male sex, older age and obesity.


**Self-monitoring of oral anticoagulation with vitamin K antagonists?**

Introduction of reliable and analytically accurate point-of-care devices allows self-testing by the patient in the home setting. Patients can have their test results managed by their health-care provider (self-testing) or they can interpret their INR results, and adjust their own dose of anticoagulant accordingly (self management). This report is a meta-analysis of 11 trials that compared self-monitoring of therapy with conventional testing and medical supervision.

The authors conclude that self-monitoring and self-management of oral coagulation is a safe option for suitable patients of all ages. The very best outcomes were in patients younger than 55 yrs and those with mechanical heart valves. An editorial agrees that self-management should be offered to those with mechanical heart valves but is not enthusiastic about its widespread use. They point out that the advent of several new oral anticoagulants which do not require monitoring may displace warfarin as the anticoagulant of choice.

University of Otago Faculty of Medicine
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