CONTENTS

This Issue in the Journal

A summary of the original articles featured in this issue

Editorials

Turning the tide of inequalities in heart health
*Norman Sharpe, Lance O’Sullivan*

Where to next with tobacco smokers?
*Ross McCormick, Doug Sellman, Geoff Robinson*

Original Articles

Will a web-based cardiovascular disease (CVD) risk assessment programme increase the assessment of CVD risk factors for Māori?
*Robyn Whittaker, Dale Bramley, Sue Wells, Alistair Stewart, Vanessa Selak, Sue Furness, Natasha Rafter, Paul Roseman, Rod Jackson*

Maternal smoking: risks related to maternal asthma and reduced birth weight in a Pacific Island birth cohort in New Zealand
*Sarnia Carter, Teuila Percival, Janis Paterson, Maynard Williams*

Prescribing oxygen therapy. An audit of oxygen prescribing practices on medical wards at North Shore Hospital, Auckland, New Zealand
*Matthew Boyle, Janice Wong*

Community hospital versus tertiary hospital comparison in the treatment and outcome of patients with acute coronary syndrome: a New Zealand experience
*Eng Wei Tang, Cheuk-Kit Wong, Peter Herbison*

Troponin testing for chest pain in primary healthcare: a survey of its use by general practitioners in New Zealand
*Kate Law, Raina Elley, James Tietjens, Stewart Mann*

Troponin testing for chest pain in primary healthcare: a New Zealand audit
*Stewart Mann, James Tietjens, Kate Law, Raina Elley*

Case Report

Recurrent muscle infarction (with involvement of the arm) in a diabetes patient
*Mohammed Alansari, David Hamilton, Kim Wong*
Special Series

Quality improvement in healthcare in New Zealand. Part 2: are our patients safe—and what are we doing about it?

Alan Merry, Mary Seddon, on behalf of EPIQ

100 Years Ago in the NZMJ

Case of rudimentary uterus and appendages

Medical Image

Meningococcal septic shock with adrenal apoplexy—Waterhouse-Friderichsen syndrome

Rajiv Sinha, Dipak Kanabar

Methuselah

Selected excerpts from Methuselah

Letters

More on alcohol and youth. Has the NZMA demonstrated that it is not a credible source of advice to Parliament?

John Langley, Kypros Kypri

PHARMAC and statins—getting the best population health gains

Peter Moodie, Sean Dougherty, Scott Metcalfe

Optimising Chlamydia testing within constrained funding

Arthur Morris, Michael McCarthy

Prioritisation in a stressed-out service

Roger Ridley-Smith

Quality versus value

Jim Vause

Obituary

Douglas Ian Chisholm

Notice

Index of NZMJ Obituaries: 1887–June 2006

Basil Hutchinson and NZMA
This Issue in the Journal

Will a web-based cardiovascular disease (CVD) risk assessment programme increase the assessment of CVD risk factors for Māori?
R Whittaker, D Bramley, S Wells, A Stewart, V Selak, S Furness, N Rafter, P Roseman, R Jackson

Cardiovascular disease (CVD) is a leading cause of death for Māori. National guidelines recommend assessment of CVD risk be carried out for all Māori men over 35 years of age and Māori women over 45 years of age—and 10 years later for people of non-Māori, non-Pacific, non-Indian subcontinent ethnicity. This study looked at the documentation of CVD risk assessment or risk factors by general practitioners (GPs) before and after the introduction of a computer-based tool—PREDICT-CVD. After its introduction, the documented assessment of CVD risk in electronic medical records (EMRs) increased from 3.2% to 14.7% of EMRs for Māori patients, and from 2.8% to 10.5% of EMRs for non-Māori. Therefore the implementation of PREDICT-CVD was just as likely to increase documentation of CVD risk assessment and risk factors in Māori as in non-Māori. However documentation was still low in Māori despite known high prevalence of CVD risk factors. A comprehensive quality-driven implementation programme is recommended, including targeting risk assessment for those most in need.

Maternal smoking: risks related to maternal asthma and reduced birth weight in a Pacific Island birth cohort in New Zealand
S Carter, T Percival, J Paterson, M Williams

It is widely known that cigarette smoking has been linked to harmful consequences for both maternal and child health. This study investigated associations between smoking during pregnancy and maternal asthma versus two indicators of pregnancy outcome: birth weight and preterm delivery among 1368 biological mothers of a Pacific birth cohort. Findings showed that smokers had over twice the risk of having asthma as well as a low birth weight or small for gestational age infant than non-smokers. Smoking significantly reduced mean birth weight. However, no significant association was found between smoking and preterm birth. This study provides further evidence that smoking confers increased risk to both maternal and reproductive health and reinforces the need for additional effort to reduce smoking during pregnancy among Pacific women.

Prescribing oxygen therapy. An audit of oxygen prescribing practices on medical wards at North Shore Hospital, Auckland, New Zealand
M Boyle, J Wong

Oxygen therapy may be life-saving, however it can cause dangerous effects and should therefore only be available by prescription. In order to monitor and promote
the safe administration of oxygen at North Shore Hospital, Auckland, we conducted a brief audit of inpatient oxygen prescription. Of the 100 medical inpatients receiving oxygen during the audit, only 8 had it prescribed in their medication chart. The majority (75%) of oxygen prescriptions were inadequate. This poor prescription rate carries serious potential consequences.

Community hospital versus tertiary hospital comparison in the treatment and outcome of patients with acute coronary syndrome: a New Zealand experience
E W Tang, C-K Wong, P Herbison

We compared the treatment and outcome of patients with heart attacks managed in a community hospital without, and a tertiary hospital with, interventional facility in the years 2000–2002. We found a higher use of evidence-based medications, coronary angiography (65.5% vs 20.2%, p<0.00001), and operative interventions (46.7% vs 16.4%, p<0.0005) in patients admitted into tertiary hospital. The in-hospital, 6-months, and 1-year mortality was significantly lower (absolute mortality difference of 4.3%, 9.5%, and 10.0%, p<0.05, respectively) for heart attacks managed in a tertiary hospital thus suggesting a disparity in outcome. The use of evidence-based medicine in all heart attack patients must be encouraged for all patients even if operative interventions are not needed.

Troponin testing for chest pain in primary healthcare: a survey of its use by general practitioners in New Zealand
K Law, R Elley, J Tietjens, S Mann

Troponin blood tests identify patients who have had a heart attack and are increasingly being used by general practitioners (GPs). However, the test may not show up as positive until 9 hours after chest pain. If GPs are relying on this test too soon after the chest pain, or if they are waiting for the result before sending the person with chest pain to hospital, this may delay important admission to hospital. We surveyed 216 GPs in Wellington to see how they would use the troponin test and what their knowledge of the test was. We found that almost all GPs referred people with chest pain to hospital and used the troponin test appropriately. They also had a good knowledge of the 9 hour window when the test may be negative.

Troponin testing for chest pain in primary healthcare: a New Zealand audit
S Mann, J Tietjens, K Law, R Elley

Troponin blood tests identify patients who have had a heart attack and are increasingly used by general practitioners (GPs). We audited 433 tests requested from primary care in the greater Wellington region, 10 of which were positive. The test proved very useful, particularly when chest pain occurred some days beforehand. Tests can be negative for up to 9 hours following a heart attack; some tests were done within this period and results may have been interpreted inappropriately.
Turning the tide of inequalities in heart health

Norman Sharpe, Lance O’Sullivan

A constant commentary through our national media, rightly and understandably, relates to the present economic indicators and the future economic forecast for our small country. In contrast, we are also regularly reminded of the “nuisance” value and increasing costs of modern healthcare. This “nuisance” viewpoint requires adjustment.

Quality and equity in healthcare (including heart health risk, outcomes, and access) will be a prerequisite for true economic wellbeing in the long-term. Indeed, increased investment in health promotion and preventive care is crucial, with a careful balance needed between this investment and the ever-increasing demand for clinical care.

At present there are at least three to four-fold (ethnic, socioeconomic, and geographic) differences in general health risk, outcomes, and access to care in New Zealand. These differences are particularly evident for heart health. Specifically, poor people, Māori, and those in some rural areas are particularly disadvantaged. These well-described inequalities are intolerable, and yet the situation threatens to worsen.

New Zealand appears particularly vulnerable to the current worldwide epidemic of obesity and diabetes which is leading to a new wave of heart disease in relatively younger people and particularly in Māori. But until equity and fairness are achieved for the disadvantaged groups, economic prosperity will remain a distant aspiration.

While the pathway for improvement is in view, commitment and more urgent, intensive, and balanced actions are required to initiate and accelerate progress. The continuum view and life-course approach to good health should be relevant for all providers who have the agreed responsibility of pursuing the New Zealand Health Strategy principles and priority objectives.

Leadership, coordination, and cohesion across all agencies and providers are required. Mainstream and Māori interests and providers should look to support and complement each other for maximum efficiency and effectiveness. Any “them and us” or other adversarial approaches will only distract us from our goals and waste time, energy, and resources.

Realistic timelines for improvement across the continuum need to be acknowledged. For the far horizon, a high-level strategic and sustained approach to obesity and diabetes action is required to turn the obesity “supertanker” around. This approach should be joined to implementation of the Healthy Eating Healthy Action (Oranga Kai Oranga Pumau) Plan, which is already underway but is requiring increased commitment and resources.

Regional solutions need to be found to achieve improved and equitable management of patients with acute heart disease and for elective coronary revascularisation. Such solutions are already being progressed in an exemplary way in the Midland and MidCentral regions where access has been most limited.
Chronic disease management approaches and cardiac rehabilitation have led to demonstrable improvement in some regions but they need wide extension to meet the needs of Māori and Pacific people and low-income groups in particular.

The “keystone” area of cardiovascular risk assessment and management is in the midst of these potential continuum improvements, although the benefits in this particular area are yet to be realised. The article published in this issue of the Journal shows that documentation of cardiovascular risk through primary care in one group of Primary Healthcare Organisations could be achieved as well for Māori as for non-Māori.

Documentation for both groups was still only achieved for relatively few people however, and risk assessment is only the first step towards effective management. The requirements for progress with systematic implementation of the Cardiovascular Risk Guideline (The Assessment and Management of Cardiovascular Risk NZGG 2003) are well evidenced from a number of successful projects already underway in New Zealand.

Systematic implementation in primary care (as a provider priority) requires strong leadership and a collaborative team (clinician/nurse) approach. Adequate resources must be identified for assessment, effective intervention, and monitoring plus follow-up of the high-risk people identified. A particular focus on disadvantaged and high-risk groups (with appropriate cultural fit) should be supported.

Cardiovascular risk assessment and management, if carried out comprehensively, could provide considerable heart health benefits for many people in a relatively short period of time. It is something that we can do now to improve the heart health of all New Zealanders and to reduce inequalities at the same time. Improvements in adjacent areas of the continuum will take longer to change however.

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*Te Hotu Manawa Māori assists health promotional activities and resources aimed at raising awareness of the main risk factors of heart disease among Māori. Priority areas are:

- Auahi Kore – Smokefree Māori
- Aukati Kai Paipa – Smoking Cessation & Guidelines
- Kai Totika me Whakapakari Tinana – Nutrition & Physical Activity

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References:


Where to next with tobacco smokers?

Ross McCormick, Doug Sellman, Geoff Robinson

Just under 25% of New Zealanders smoke tobacco cigarettes,\(^1\) which is virtually the same as in the United States, but significantly higher than Australia (20%).\(^2\) However the New Zealand statistic includes a 50% prevalence rate of smoking amongst Māori and 30% for Pacific people (most of whom are of Samoan, Tongan, Niuean, or Cook Islands descent).\(^1\)

It is nearly 20 years since the United States Surgeon General report made strong, unequivocal statements about the nature of nicotine addiction in the form of two major conclusions:

- “Cigarettes and other forms of tobacco are addicting”; and
- “Nicotine is the drug in tobacco that causes addiction”.\(^3\)

These conclusions have been more recently reiterated in a special 2000 Report of the Royal College of Physicians, the central conclusion of which was; “Cigarette smoking should be understood as a manifestation of nicotine addiction, and the extent to which smokers are addicted to nicotine is comparable with addiction to ‘hard’ drugs such as heroin or cocaine”.\(^4\) Their Report comments that about two-thirds of smokers say they would like to quit and about one-third try to quit in any 1 year, yet only about 2% succeed.

In New Zealand, considerable efforts have been made to reduce smoking prevalence. The approaches used have been public health focussed, using a broad range of methods including education campaigns, warning labels, legal restrictions on where people can smoke tobacco, taxation on cigarettes, and Quit Lines (with free access to nicotine substitution for 8 weeks to help overcome nicotine withdrawal symptoms).

However, despite these approaches producing admirable success over the years, how much further can a purely public health approach go? As prevalence now slowly decreases, it is probable that the remaining smokers are the more severely addicted ones and this may make further reductions in prevalence difficult. Indeed, the remaining highly dependent smokers are likely to need more intensive treatment.\(^5\)

What can be learnt from the experience with other drugs of addiction, such as opioids? In New Zealand, the strategy to reduce harm due to opioid dependence includes supply control, demand control, and problem limitation.\(^6\) For instance, supply control includes legal restrictions, and demand control includes education campaigns. Problem limitation involves a variety of objectives. Some services aim to assist an addicted person to become abstinent from opioids, others aim to reduce harmful administration of opioids by supplying clean needles, and still others aim to normalise the opioid dependent person’s life by prescribing oral substitutes such as methadone.

There are a variety of professionals involved in helping the dependent opioid user including general practitioners, pharmacists, addiction specialists, nurses, psychologists, recovering addicts, and counsellors.
Like opioids, nicotine is rewarding and addictive. Nicotine’s relaxation effect, improved mood effect, and improved cognitive performance effect are greatest when the nicotine is delivered rapidly to the brain through inhaled smoke or through other rapid high-dose delivery systems. However, there are well-known dangers of rapid opioid self-injecting delivery systems (e.g. hepatitis C infection), and similarly there are well-known dangers of rapid nicotine smoking delivery systems (e.g. severe lung disease) where the risks are mostly due to some of the 4000 or so chemicals in cigarette smoke other than nicotine.

Both opioids and nicotine are highly addictive drugs, yet their ongoing use is compatible with a relatively normal life: it is the delivery system that causes the most harm.

In New Zealand, there are no services offering problem limitation services for tobacco addiction analogous to those offered opioid addicts. The goal of most existing tobacco dependence treatment services is abstinence from nicotine, achieved through quitting cigarette smoking. Tobacco addiction treatment remains locked in a 1970s opioid addiction service model of sequential abstinence and relapse, often many times over.

A harm minimisation approach to tobacco addiction would argue that substitution drug treatment, such as Swedish snuff, should be an option for longer periods than 8 weeks; possibly indefinitely. People would remain dependent on nicotine, but their ongoing use of nicotine would have far reduced risk of harm compared with smoking tobacco cigarettes.

Products such as Swedish snuff are themselves not thought to be risk-free, but then neither are clean needles and oral methadone for opioid-addicted people. However, the risks appear to be significantly less than those of smoked tobacco, and that alone would justify trialling their use.

We consider it is time for a paradigm shift in the way the harm due to tobacco in New Zealand is approached. This paradigm shift is needed by policymakers, researchers, and health services.

Policymakers need to talk with tobacco companies to encourage them to shift to smokeless tobacco products with increased safety profile compared to smoked cigarettes. Furthermore, researchers need to be funded for projects that will evaluate the risks and benefits of harm minimisation approaches such as substitution of cigarettes by rapid-acting non-inhaled high blood level nicotine products.

Māori researchers and policy makers are best placed to ensure any proposed changes meet the needs of Māori. There needs to be public debate about the best sales system: direct sales to the public, monitored sales through pharmacists, or prescription.

Finally, the medical profession needs to debate this issue widely to help develop the best harm-minimisation approaches for the increasingly hard-core group of nicotine-addicted people.

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Will a web-based cardiovascular disease (CVD) risk assessment programme increase the assessment of CVD risk factors for Māori?

Robyn Whittaker, Dale Bramley, Sue Wells, Alistair Stewart, Vanessa Selak, Sue Furness, Natasha Rafter, Paul Roseman, Rod Jackson

Abstract

**Background** Māori suffer disproportionately from cardiovascular disease despite the national priority of reducing inequalities. National guidelines on the clinical management of CVD risk recommend a comprehensive risk assessment be completed as a prerequisite for identifying patients most likely to benefit from treatment.

**Methods** A retrospective audit of GPs using PREDICT-CVD (an electronic risk assessment and management tool) was designed with adequate explanatory power for Māori to determine if it could increase CVD risk assessment without increasing inequalities. 1680 electronic medical records (EMRs) prior to implementation and 1884 after implementation of PREDICT were audited.

**Results** Documentation of CVD risk increased from 3.2% of EMRs to 14.7% of EMRs in Māori, and from 2.8% to 10.5% in non-Māori. The documentation of individual CVD risk factors also increased post-implementation of the tool.

**Conclusions** The implementation of PREDICT-CVD was as likely to increase documentation of CVD risk assessment and risk factors in Māori as in non-Māori. However documentation was still low in Māori despite known high prevalence of CVD risk factors. A comprehensive quality-driven implementation programme is recommended, including targeting risk assessment for those most in need.

Cardiovascular disease (CVD) is the leading cause of death for Māori.1 Despite priority being placed on CVD by the health system in New Zealand (NZ), inequalities in cardiovascular health outcomes have a negative impact on Māori.2 Māori also have high prevalence of many cardiovascular risk factors, such as smoking,3 diabetes, high cholesterol, and elevated blood pressure.4

Waitemata District Health Board (WDHB), in concordance with the Ministry of Health’s national priority areas,5 has a focus on CVD prevention and on reducing inequalities.6 Current New Zealand guidelines recommend cardiovascular risk assessment and management for all Māori men over 35 years of age and Māori women over 45 years of age—and 10 years later for people of non-Māori, non-Pacific, non-Indian subcontinent ethnicity.7

A web-based tool to facilitate risk assessment and management in primary care (PREDICT-CVD) has been developed in New Zealand and implemented using the brand name “Prompt” in ProCare Health Limited (a group of three Primary Health Organisations (PHOs) under one Management Services Organisation). This PREDICT–CVD programme is integrated into the patient management system to allow systematic cardiovascular risk assessment and provide within-seconds
evidence-based patient-tailored decision support according to the national guidelines for the management of cardiovascular risk.

Prior to supporting the implementation of such a tool, Waitemata DHB sought evidence that the use of PREDICT-CVD would not adversely impact inequalities. Therefore a retrospective before-after study was designed to investigate the effect of PREDICT-CVD on documentation of CVD risk and risk factors for Māori and non-Māori.

Methods

Eligible general practitioners (GPs) were those who had used MedTech32 electronic medical records for at least 1 year, had installed PREDICT-CVD in their practice prior to May 2004, and used systems categorising patients according to their registered GP. They were invited by ProCare Health Ltd to participate in the study. Researchers conducted electronic queries to create lists of patients meeting the NZ guideline criteria for cardiovascular risk assessment (Māori/Pacific/Indian subcontinent men aged 35 years and over; Māori/Pacific/Indian subcontinent women aged 45 years and over—and 10 years later respectively for others), who had visited an eligible GP within a 4-week period that was either 1 month after the GP first used PREDICT or the same 4-week period 1 year earlier (pre-PREDICT period).

These time periods ranged from August 2001 to June 2004, with considerable overlap between pre-PREDICT and post-PREDICT installation time periods. Both time periods were audited retrospectively, and GPs did not know in either period that they would be audited.

The study was designed to provide adequate explanatory power for Māori. Therefore all Māori patients identified, and a randomly selected 15% sample of non-Māori patients identified, were included in the audit of electronic medical records (EMRs).

Patients whose ethnicity was documented as NZ Māori or equivalent (Māori, M, or New Zealand Health Information Service [NZHIS] code 21) in the EMR were included as Māori. Where no ethnicity was recorded, patients were assumed to be non-Māori.

Trained and experienced audit nurses conducted the EMR audits in the general practices, recording any cardiovascular risk notation (range, %, text description), and any documentation of smoking status (smoker, non-smoker, past smoker), cholesterol level (total cholesterol:HDL ratio or total cholesterol), blood pressure measurements, and diabetes status (type 1 or type 2 diabetes stated, diabetes documented as absent; or no documented diagnosis but evidence of impaired glucose tolerance, raised HbA1c, or prescriptions for insulin/test strips/oral hypoglycaemics).

All analyses were conducted using SAS statistical software Version 9.1. For the outcomes of interest, the proportions for the total population were derived from Māori and non-Māori sampling populations. To calculate odds ratios together with 95% confidence intervals, a multivariate analysis was conducted using a mixed logistic regression model in which GPs were regarded as random effects and all other variables regarded as fixed effects.

The model included the practice that the GP worked in and patient characteristics that may influence GP risk-assessment behaviour: age, gender, ethnicity, presence of existing cardiovascular disease, diabetes, High Use Health Card (HUHC – for those with medical conditions requiring frequent GP visits), and Community Services Card (CSC – for low income families). To assess if documentation of risk or risk factors differed by ethnicity after the implementation of PREDICT, an interaction term was used.

The PREDICT-CVD Evaluation Study gained ethics approval from the Auckland Regional Ethics Committee (AKY/04/07/185).

Results

Of the 107 eligible GPs, 84 (78.5%) consented to participate (4 were absent from their practices, 1 could not be contacted, and 18 declined). Four practices were unable to supply data, thus leaving 80 contributing GPs.
A total of 3564 audits were conducted (1680 in the pre-PREDICT period and 1884 in the post-installation period). Māori participants made up 28.2% (n=474) of audited EMRs in the pre-PREDICT period, and 25.7% (n=484) in the post-PREDICT period. The audited Māori participants were significantly different from the non-Māori participants in all parameters evaluated. Māori participants were younger (due to the different sampling criteria from the screening guidelines) with very few aged over 74 years; were more likely to be male, have diabetes, and a CSC; and were less likely to have a HUHC and previous CVD, than non-Māori participants.

The characteristics of Māori participants did not differ greatly between the two time periods. The only significant difference among non-Māori participants was a higher level of previous CVD in the post-PREDICT period (25% of participants compared to 21% in the pre-PREDICT period, Chi-squared=5.65, p=0.0175).

Table 1. Characteristics of Māori and non-Māori audited populations

<table>
<thead>
<tr>
<th>Variable</th>
<th>Māori N=958</th>
<th>Non-Māori N=2606</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
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<tr>
<td>Age</td>
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<tr>
<td>35–44yrs</td>
<td>187</td>
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<td>45–54yrs</td>
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<td>55–64yrs</td>
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<td>75–84yrs</td>
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<td>Over 95yrs</td>
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<tr>
<td>Difference between Māori &amp; non-Māori participants: Chi Square 760.73 p&lt; 0.0001</td>
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<tr>
<td>Gender</td>
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</tr>
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<tr>
<td>Male</td>
<td>513</td>
<td>53.5</td>
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<td>Difference between Māori &amp; non-Māori participants: Chi Square 8.26 p= 0.0041</td>
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<tr>
<td>High Use Health Card (HUHC) status</td>
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<tr>
<td>No HUHC</td>
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<td>HUHC</td>
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<td>Difference between Māori &amp; non-Māori participants: Chi Square 4.84 p=0.028</td>
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<td>Community Services Card (CSC) status</td>
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<td>CSC</td>
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<td>Difference between Māori &amp; non-Māori participants: Chi Square 13.29 p=0.0003</td>
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<td>Diagnosed diabetes or on diabetes treatment</td>
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<td>No Diabetes</td>
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<td>Diabetes</td>
<td>218</td>
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<td>Difference between Māori &amp; non-Māori participants: Chi Square 56.86 p&lt; 0.0001</td>
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<tr>
<td>Previous cardiovascular disease (CVD) event or on nitrates</td>
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<tr>
<td>No CVD</td>
<td>801</td>
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<tr>
<td>CVD</td>
<td>157</td>
<td>16.4</td>
</tr>
<tr>
<td>Difference between Māori &amp; non-Māori participants: Chi Square 19.23 p&lt;0.0001</td>
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</tbody>
</table>

Prior to PREDICT installation, Māori had slightly higher recording of cardiovascular risk than non-Māori (in 3.2% vs 2.8% of EMRs). Where the EMR contained information on all the risk factors necessary to conduct a risk assessment but overall
risk was not documented, the GP may have used a paper-based risk assessment chart to calculate CVD risk but not documented it in the EMR. The baseline proportion of EMRs with either risk documented or all necessary risk factors documented was 12.4% in Māori and 9% in non-Māori. (Figure 1)

There was an increase in the recording of risk after the installation of PREDICT for both Māori and non-Māori. This increase appears greater in Māori participants (from 3.2% of EMRs to 14.7% of EMRs) than in non-Māori (from 2.8% to 10.5% of EMRs), although it was not statistically significant.

When the recording of CVD risk factors was included, documentation of risk or risk factors increased from 12.4% to 24.0% of EMRs for Māori and 9.0% to 17.6% for non-Māori, but again this increase was not statistically different between Māori and non-Māori.

**Figure 1. Proportion of EMRs with CVD risk, and risk factors, documented pre- and post-PREDICT by Māori and non-Māori**

![Bar chart showing the proportion of EMRs with CVD risk and risk factors pre- and post-PREDICT for Māori and non-Māori.](chart.png)

The recording of both smoking and diabetes status was higher for Māori than non-Māori in both periods. The recording of smoking status for Māori increased from 49.6% of EMRs prior to PREDICT to 59.3% in the post PREDICT period. Recording of diabetes status also increased from 21.5% to 23.3% (Table 2). The increase in the
recording of all risk factors from pre- to post-installation was not statistically different for Māori compared to non-Māori.

Table 2. Proportion of EMRs with risk factors documented

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-Prompt period</th>
<th>Post-Prompt period</th>
<th>Difference in pre-to post-increase between Māori &amp; non-Māori: p value*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Māori N=474 % (n)</td>
<td>Non-Māori N=1206 % (n)</td>
<td>Māori N=484 % (n)</td>
</tr>
<tr>
<td>CVD risk</td>
<td>3.2 (15)</td>
<td>2.8 (34)</td>
<td>14.7 (71)</td>
</tr>
<tr>
<td>CVD risk or risk factors present</td>
<td>12.5 (59)</td>
<td>9.0 (108)</td>
<td>24.0 (116)</td>
</tr>
<tr>
<td>Smoking status</td>
<td>49.5 (235)</td>
<td>38.3 (462)</td>
<td>59.3 (287)</td>
</tr>
<tr>
<td>Diabetes status</td>
<td>21.5 (102)</td>
<td>14.0 (169)</td>
<td>23.4 (113)</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>71.5 (339)</td>
<td>85.8 (1035)</td>
<td>83.7 (405)</td>
</tr>
<tr>
<td>TC/HDL or total cholesterol</td>
<td>57.6 (273)</td>
<td>64.3 (776)</td>
<td>69.2 (335)</td>
</tr>
</tbody>
</table>

*Model includes: age, gender, presence of CVD, diabetes, HUHC, CSC.

Discussion

As District Health Boards have a legislative mandate to reduce inequalities and improve Māori health, it was considered vital for Waitemata DHB to assess the potential effect of a comprehensive cardiovascular risk assessment and management programme on inequalities prior to the introduction of such a programme. Therefore an evaluation study was designed specifically to have adequate explanatory power for Māori.

The use of this methodology could be considered an innovation by the DHB as it attempts to find ways to increase health service responsiveness to the need to reduce health inequalities. This type of study methodology could also be used in the future by other DHBs in order to evaluate new or existing services and their impact on inequalities.

The results of this study are encouraging in that risk factor documentation and risk assessment increased similarly for both Māori and non-Māori after the installation of the PREDICT-CVD risk assessment tool. It appears that this type of tool will not increase inequalities and, if applied in an appropriate manner, could potentially lead to a reduction in inequalities. However, introduction of the risk assessment tool alone is not enough as evidenced by the low overall level of documented risk assessment.

In future we recommend introduction be accompanied by a comprehensive implementation programme to ensure that the entire target population is assessed. Also, the ensuing management of risk still needs to be followed up to ensure that risk assessment does lead to better treatment and health outcomes, and to avoid differences in treatment by ethnicity.

It is of interest that the recording of cardiovascular risk and risk factors for Māori was so low in this population given the demonstrated high prevalence of cardiovascular risk factors in Māori in other studies. For example, the New Zealand Health Survey...
(2002/03) found that 23.7% of Māori adults had been told they had high blood pressure (vs 17.6% European/Other) and 15.9% high cholesterol (vs 14.6% European/Other). Also CVD is known to occur at an earlier age in Māori, as reflected in the cardiovascular risk guidelines recommendation to screen this group 10 years earlier. Should Māori patients be prioritised for risk assessment and risk factor documentation, the health gains are likely to be large.

The documentation of smoking and diabetes status were higher in Māori than non-Māori. Just under one in every two Māori are smokers compared to one in every five non-Māori, and the prevalence of diabetes is 6.7% in Māori compared to 2.4% in European/Others. Therefore documentation is likely to be higher among Māori if GPs are more likely to record a positive finding than a negative one.

The audited sample contained very few older Māori. This is likely to be influenced by the fact that Māori live (on average) 8–10 years less than non-Māori. There were also low numbers of Māori participants with a HUHC, which is surprising given the high prevalence of diabetes and other chronic conditions in Māori. This could reflect inadequate targeting of this tool.

There was a lower rate of documented previous CVD in Māori than non-Māori in this group. The reasons for this are unknown but should be explored further.

Limitations of the study—Māori are known to be proportionately under-enrolled in PHOs in some areas therefore it is possible that the ProCare Health Ltd enrolled population may not be fully representative of the Māori population of the Auckland region. Differences in access to primary care for Māori, even when enrolled, may also have affected selection of participants.

Also, the definition of ethnicity may lead to classification error, as there was considerable variability across practices in the recording of ethnicity (NZHIS codes, other codes, free text). In the ProCare Health Ltd-enrolled population at the time of this study, 12.3% of patients had no ethnicity recorded [personal communication Ken Leech, ProCare Health Ltd, 2005], and an ensuing audit of ethnicity coding found that less than 2% of audited patients had more than one ethnicity recorded.

In this study 7.2% of audited EMRs had no ethnicity stated and these were coded as non-Māori, which may have resulted in undercounting of Māori participants. It is not known whether the documented ethnicity was self-identified by the patient or assigned by the practice.

Suggestions for the future—The implementation of the PREDICT-CVD tool should not occur in isolation without a comprehensive quality-driven programme. Aspects of such a programme could include: education for primary care staff concerning the importance of documenting disease risk factors, even if these are found to be absent/negative; active recalling of patients meeting the NZ guideline criteria from PHO-registered population lists; a standard approach to the documentation of ethnicity in primary care; automatic prompts for EMRs of patients who meet the criteria for risk assessment according to the New Zealand guidelines; and targeting risk assessment for those most in need (in particular Māori and Pacific). It should also include taking risk assessment into community settings to provide access for those not currently engaged with primary care.
An ongoing challenge for DHBs is the requirement to explore and implement healthcare practices that will lead to a reduction in health inequalities.

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We also thank Elaine Horn, Kate Moodabe, and all the GPs from ProCare Health Ltd for participating in the study; their practice teams for making us feel welcome; the audit nurses and staff from the Diabetes Project Trust; and Waitemata DHB Cardiovascular Technical Advisory Group for recommending we undertake the study.

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References:


Maternal smoking: risks related to maternal asthma and reduced birth weight in a Pacific Island birth cohort in New Zealand

Sarnia Carter, Teuila Percival, Janis Paterson, Maynard Williams

Abstract

Aims This study investigated associations between smoking and maternal asthma and two indicators of pregnancy outcome: birth weight and preterm delivery.

Methods Data were gathered as part of the Pacific Islands Families (PIF) Study. Mothers of a cohort of 1398 Pacific infants born in South Auckland, New Zealand during 2000 were interviewed when their infants were 6 weeks old. Mothers were questioned regarding maternal health and lifestyle behaviours such as cigarette smoking. Additional data were obtained from hospital records. Analyses focused on 1368 biological mothers.

Results Approximately 20% of mothers reported smoking during their last trimester of pregnancy. Logistic regression analyses showed that smokers had over twice the risk of having maternal asthma as well as a low birth weight (LBW) or small for gestational age (SGA) infant than non-smokers. Smoking significantly reduced mean birth weight from between 149.2 grams (1–9 cigarettes) to 204.3 grams (10+ cigarettes). No significant association was found between smoking and preterm birth.

Conclusions Smoking is preventable, yet continues to have negative consequences for mothers and their offspring. Findings can inform public health policy and smoking cessation programmes for Pacific families.

Little data exist pertaining to smoking behaviour and associated consequences among the Pacific population in New Zealand (i.e. mostly people of Samoan, Tongan, Niuean, and Cook Islands origin). As Pacific infants tend to weigh more than other ethnic groups in New Zealand, it is possible that the effects of smoking on birth weight may go largely undetected until such associations are specifically examined. Thus further understanding of the effects of smoking among this relatively socioeconomically disadvantaged group is important given the potential harm in regard to both child and adult health.

Evidence is sufficient to suggest a causal relationship exists between active smoking and acute respiratory illnesses and symptoms including wheeze. The relationship between smoking and asthma is less clear with underlying causes of asthma not well understood.

Although some studies show an association between smoking and increased likelihood of asthma or asthma symptoms, it is possible that smoking aggravates airways in susceptible people, exacerbating symptoms rather than causing development of the disease. Smoking has serious implications for asthmatics, being repeatedly linked to greater severity of symptoms, poorer control, increased use of
hospital services, impaired lung function, and asthma-related morbidity and mortality.

Smoking carries additional risk to women with greater likelihood of reproductive complications. The heightened risk of foetal and neonatal mortality among offspring of smoking mothers is thought to stem mostly from increased incidence of low birth weight infants (LBW) weighing less than 2500 grams, infants with intrauterine growth retardation (and thus abnormally small for their gestational age [SGA]), or from pregnancy complications including abruptio placentae.

Research has consistently shown mothers who smoke during pregnancy are more likely (than non-smoking mothers) to have preterm births, LBW, or SGA infants. A mean weight reduction of approximately 150–250 grams is frequently observed in infants of smoking mothers. Prevention of LBW is crucial as it continues to be the most important determinant of perinatal mortality and impaired later development. Many LBW and/or preterm infants require admission to high-cost neonatal intensive care units resulting in a significant economic burden.

**Smoking in New Zealand**

New Zealand has one of the highest prevalences of asthma in the World, and available data indicate that Māori and Pacific adults are more likely than other groups to have asthma. Asthma has been estimated to cost the country at least NZ$825 million annually. A significant number of female smokers are endangering their own health, and (in parallel) a sizeable proportion of pregnancies may be at risk for adverse outcome. In 2001, 55.9% of Māori, 26.3% of Pacific, and 20.7% of European/other New Zealand women were smokers.

Smoking rates specific to pregnancy closely mirror rates seen for female smoking in the general population. Although small fluctuations over time have been observed, pregnancy smoking has remained fairly stable at approximately 30% for 20 years. A study in the Canterbury region during 1993–94 showed the overall prevalence of smoking during pregnancy to be 33.0%; smoking prevalence was substantially higher (close to 50%) in areas associated with socioeconomic disadvantage.

In 1997, 26.8% of participants smoked during the first, 25.0% smoked during the second, and 23.0% smoked during the third trimester of pregnancy.

**Smoking among Pacific women**

Pacific people comprise approximately 6.5% of the New Zealand population, and (largely due to high fertility rates) are one of the fastest growing groups, projected to be 12% of the population by 2051.

Pacific peoples have generally fared worse than the New Zealand population as a whole on a range of health and social indicators. For instance, compared with national rates, Pacific children have high rates of hospitalisation as well as a higher incidence of respiratory infections, meningococcal disease, and common infectious diseases such as measles.

Few studies document smoking among Pacific women. Smoking prevalence for Pacific women in 2002 was 28.5% compared with 25.5% for all New Zealand women, while research conducted approximately 10 years ago revealed 23.6% of
Pacific mothers smoked during pregnancy compared with 33.2% for the whole population sampled.  

In an Australian study, 16% of Pacific mothers smoked during pregnancy, substantially lower than the 36% indicated for Caucasian mothers. However, the study contained only 80 Pacific mothers and it is possible that smoking behaviours and other characteristics differ between Pacific mothers residing in Australia and New Zealand.

Findings from our New Zealand cohort study of Pacific infants (the Pacific Islands Families [PIF] Study) supports this view, with 24.9% of mothers smoking during pregnancy. Using data from the PIF Study, this study investigated associations between smoking and maternal asthma and two indicators of pregnancy outcome: birth weight and preterm delivery.

Methods

Data collection—Data were collected as part of the PIF Study, a longitudinal investigation of a cohort of 1398 infants born at Middlemore Hospital, South Auckland, New Zealand during the year 2000. The majority (67%) of Pacific communities reside in the Auckland area. Middlemore Hospital was chosen for recruitment as it has the largest number of Pacific births and is representative of the major Pacific ethnicities (Samoan, Cook Island Māori, and Tongan). Eligibility criteria for entry to the PIF Study included having at least one parent who self-identified as being of Pacific ethnicity and being a New Zealand permanent resident. Thus, infants of non-Pacific mothers were eligible in cases where the father was of Pacific descent.

Potential participants were identified in conjunction with Middlemore’s Pacific Islands Cultural Resource Unit and the Birthing Unit. Following delivery, study personnel approached potential participants, provided brief information, and obtained permission for later contact. All procedures and interview protocols were granted ethical approval from the National Ethics Committee. Detailed information about the cohort and procedures is described elsewhere.

Approximately 6-weeks postpartum, Pacific interviewers fluent in English and a Pacific language visited the mothers at home. Of the 1376 mothers, 1368 were biological and 8 were foster or adoptive mothers. Eligibility criteria were confirmed and informed consent was gained for participation in an interview and access to Middlemore Hospital discharge records. For the present study, responses based on the 1368 biological mothers and first-born twin for twin pairs were utilised in analyses.

Mothers participated in 1-hour interviews concerning the health and development of the child and family functioning. As part of this interview, mothers approximated how many cigarettes they had smoked per day prior to pregnancy, during the three trimesters of pregnancy, and yesterday (current smoking).

Mothers were asked whether they currently had any of a range of health problems (including asthma) that had been diagnosed by a doctor or for which the mother was presently taking medications. Sociodemographic data were also collected—including maternal age, ethnicity (self-identified), education, marital status, and household income. Birth weight (grams) and preterm delivery status (<37 weeks of completed gestation) were extracted from hospital birth records. Data were coded and double-entered into the SPSS (version 12.0.1) statistical software package.

Smoking during the last trimester of pregnancy was categorised into three groups: non-smoking, light/moderate smoking (1–9 cigarettes daily), and heavy smoking (10+ cigarettes daily).

The associations between smoking and three outcome variables (maternal asthma, preterm birth, and birth weight) were examined in the following manner:

Maternal asthma—Univariate logistic regression analyses were initially performed with the measure of effect being the odds ratio (OR) with its 95% confidence interval (CI). As it is likely that other factors also contribute to a heightened risk of asthma, a multiple logistic regression analysis was then undertaken to control for potential confounding effects. Five demographic variables (maternal age, education, ethnicity, marital status, and household income) along with the smoking variable were therefore entered into a multiple regression model.
Preterm birth—Univariate and multivariate logistic regression analyses also were conducted to determine any associations between smoking during pregnancy and preterm birth. The five demographic variables (maternal age, education, ethnicity, marital status, and household income) were again entered as control variables in the multivariate model.

Birth weight—To examine the effect of third-trimester smoking on birth weight, three sets of analyses were conducted:

- First, differences between means were tested using analysis of variance. Cuzick’s non parametric test was employed to test for a significant trend of decreasing birth weight with increasing smoking dosage.\(^{32}\) Multiple regression analyses were then performed to control for potential confounding effects of maternal age, ethnicity, marital status, education, household income, twin birth, and gestational age (<37, 37–40, >40 weeks).

- Second, LBW was examined using births of 37 or more weeks of gestation (i.e. excluding preterm births). Due to the small number of LBW infants, smoking-dose categories were combined into smoking and non-smoking during the third trimester. Logistic regression was used to determine whether smoking increased risk of LBW (≤2500 grams). However, multiple logistic regression analyses controlling for other factors were not possible due to small numbers of LBW infants.

- Third, a SGA variable was created using births of 37 or more weeks of gestation categorised into five groups (37-38, 39, 40, 40+ weeks) and tested against each groups’ sex-specific 10\(^{th}\) percentile weight to determine if they were small or of appropriate weight for their gestational age grouping. Due to small frequency counts in some weeks of gestation, it was necessary to group ages. Univariate logistic regression analyses were conducted to determine any associations between smoking during pregnancy and SGA. To control for potential confounding effects a multiple logistic regression analysis was then undertaken. The same five demographic variables used previously, along with twin status and smoking, were entered into the model.

Results

Initially, 1708 mothers of Pacific infants (born between 15 March 2000 and 17 December 2000) were identified as potentially meeting eligibility criteria. After excluding cases where the infant died or non-resident status was confirmed whilst the mother was in hospital, a potential group of 1657 mothers were then invited to participate in the study.

Ninety-six percent (N=1590) of these potentially eligible mothers gave consent to be visited at 6 weeks postpartum. Ten (1\%) mothers were then determined as ineligible, 103 (6\%) mothers were of indeterminate eligibility (largely due to leaving Auckland or being untraceable), and 1477 mothers were contacted and confirmed eligible. Of these 1477 mothers, 1376 (93.2\%) agreed to participate in the PIF Study.

The study presented here is based on data obtained for the 1368 biological mothers in the PIF study. Table 1 presents the basic demographic characteristics of the biological mothers.

During the third trimester of pregnancy, 1089 (79.7\%) mothers reported to be non-smokers, 194 (14.2\%) smoked an average of 1–9 cigarettes daily, and 84 (6.1\%) smoked 10 or more cigarettes daily (smoking data were missing for one mother).
Table 1. Frequencies (percentages) of basic demographic variables measured at 6-weeks postpartum for biological mothers (N=1368) in 2000

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Samoan</td>
<td>647</td>
<td>(47.3)</td>
</tr>
<tr>
<td>Cook Island Māori</td>
<td>229</td>
<td>(16.7)</td>
</tr>
<tr>
<td>Tongan</td>
<td>287</td>
<td>(21.0)</td>
</tr>
<tr>
<td>Niuean</td>
<td>59</td>
<td>(4.3 )</td>
</tr>
<tr>
<td>Other Pacific*</td>
<td>47</td>
<td>(3.4 )</td>
</tr>
<tr>
<td>Non-Pacific</td>
<td>99</td>
<td>(7.2 )</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>110</td>
<td>(8.0 )</td>
</tr>
<tr>
<td>20-29</td>
<td>750</td>
<td>(52.6)</td>
</tr>
<tr>
<td>30+</td>
<td>538</td>
<td>(39.3)</td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partnered</td>
<td>1100</td>
<td>(80.4)</td>
</tr>
<tr>
<td>Non-partnered</td>
<td>268</td>
<td>(19.6)</td>
</tr>
<tr>
<td><strong>New Zealand-born</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>452</td>
<td>(33.0)</td>
</tr>
<tr>
<td>No</td>
<td>916</td>
<td>(67.0)</td>
</tr>
<tr>
<td><strong>Highest educational qualifications</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal qualifications</td>
<td>533</td>
<td>(39.0)</td>
</tr>
<tr>
<td>Secondary</td>
<td>460</td>
<td>(33.6)</td>
</tr>
<tr>
<td>Post-secondary</td>
<td>375</td>
<td>(27.4)</td>
</tr>
<tr>
<td><strong>Household income (annual)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$0–$20,000</td>
<td>454</td>
<td>(33.2)</td>
</tr>
<tr>
<td>$20,001–$40,000</td>
<td>708</td>
<td>(51.8)</td>
</tr>
<tr>
<td>&gt; $40,000</td>
<td>159</td>
<td>(11.6)</td>
</tr>
<tr>
<td>Unknown</td>
<td>47</td>
<td>(3.4 )</td>
</tr>
</tbody>
</table>

*Includes mothers identifying equally with two or more Pacific groups, equally with Pacific and Non-Pacific groups, or with Pacific groups other than Tongan, Samoan, Cook Island or Niuean

Maternal asthma

99 (7.2%) mothers reported having asthma (as diagnosed by a medical professional or for which they were taking medication). Table 2 shows the association between smoking (in the third trimester of pregnancy) and maternal asthma. The numbers and percentages of mothers who reported smoking are given along with the univariate and adjusted OR (95% CI) indicating likelihood of asthma.

Table 2. Association between daily cigarette dose and incidence of maternal asthma (N=1366)

<table>
<thead>
<tr>
<th>Daily cigarette dose</th>
<th>Maternal asthma n (%)</th>
<th>Univariate odds ratio (95% CI)</th>
<th>Adjusted odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>58 (5.3)</td>
<td>1.00</td>
<td>1.94 (1.14–3.30)*</td>
</tr>
<tr>
<td>1–9</td>
<td>23 (11.9)</td>
<td>2.41 (1.45–4.00)†</td>
<td>3.63 (1.93–6.85)‡</td>
</tr>
<tr>
<td>10+</td>
<td>18 (21.4)</td>
<td>4.85 (2.70–8.70)‡</td>
<td></td>
</tr>
</tbody>
</table>

*p<0.05; †p<0.01; ‡p<0.001.
Compared to non-smokers, univariate analyses depicted in Table 2 show that mothers who smoked 1–9 cigarettes daily had over twice the risk (and mothers who smoked 10+ cigarettes daily had almost five times the risk) of having asthma. Table 2 also shows that smoking remained significantly associated with maternal asthma for both cigarette doses levels following adjustment for demographic variables. The degree of risk was reduced, however, once these factors were controlled for.

The adjusted OR for 1–9 cigarettes was 1.9 (95%CI=1.1–3.3; p<0.05), and for the heavier smoking dose, the adjusted OR was 3.6 (95%CI=1.9–6.9; p<0.001). Ethnicity and age were also independently associated with maternal asthma, with younger mothers (<20 years) being just over two times more likely (OR=2.1; 95%CI=1.0–4.4; p<0.05) to have asthma than older mothers aged 30 or more years; ‘Other Pacific’ (OR=0.2; 95%CI=0.1–0.6) and Cook Islands (OR=0.4; OR=0.2–0.8) women less likely to exhibit asthma than Samoan women (p<0.01).

### Preterm birth

Of the 1346 births for which data were available, 106 (7.9%) were considered preterm (less than 37 weeks gestation). As indicated in Table 3, no significant association between smoking during pregnancy and preterm birth was found.

### Table 3. Association between daily cigarette dose and preterm births (<37 weeks) (N=1346)

<table>
<thead>
<tr>
<th>Daily cigarette dose</th>
<th>Preterm birth n (%)</th>
<th>Univariate odds ratio (95%CI)</th>
<th>Adjusted odds ratio (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>87 (8.1)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>1–9</td>
<td>15 (7.9)</td>
<td>0.96</td>
<td>0.97</td>
</tr>
<tr>
<td>10+</td>
<td>4 (4.8)</td>
<td>0.57</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Odds ratios adjusted for mother’s age, education, ethnicity, social marital status, and household income.

### Birth weight

**Mean birth weight**—Smoking was significantly associated with reduced birth weight (p<0.001). The mean birth weight of infants born to non-smoking mothers was 3636.2 (n=1073; SD=619.8). The corresponding mean weights of infants born to light-to-moderate smoking mothers was 3392.6 grams (n=191; SD=553.3), and 3358.4 grams (n=84; SD=551.0) for heavy smokers.

There was a significant trend: decreasing birth weight with increasing smoking dose (p<0.001). On average, infants born to light-to-moderate smokers weighed 243.5 grams less and infants born to heavy smokers weighed 277.7 grams less than infants born to non-smokers.

Adjusting for potential confounders reduced the strength of the associations between smoking during pregnancy and birth weight. Bonferroni tests confirmed that the associations remained significant (p<0.01) with mothers who smoked 1–9 cigarettes daily having infants that weighed on average 149.2 grams less and mothers who smoked 10 or more cigarettes daily delivering infants weighing 204.3 grams less than their non-smoking counterparts.
Low birth weight (<2500 grams)—Univariate analyses conducted on births of 37 or more weeks of gestation (n=1240) showed that smokers were significantly (p<0.01) more likely to deliver LBW infants compared to non-smokers (OR=6.3; 95%CI=2.1–19.5).

Small for gestational age (SGA)—Table 4 shows that (compared to non-smoking mothers) mothers who smoked 1–9 cigarettes daily had over twice the risk (and mothers who smoked 10+ cigarettes daily had over three times the risk) of delivering an infant considered SGA.

Table 4. Numbers (row percentages) as well as univariate and adjusted odds ratios of small for gestational age (SGA) and low birth weight (LBW) babies by smoking dose of mothers (N=1240)

<table>
<thead>
<tr>
<th>Variable</th>
<th>SGA n (%)</th>
<th>Univariate OR (95%CI)</th>
<th>Adjusted OR (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily cigarette dose</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>76 (7.7)</td>
<td>1.00 (1.00–1.49)‡</td>
<td>2.10 (1.30–3.41)†</td>
</tr>
<tr>
<td>1–9</td>
<td>29 (16.5)</td>
<td>2.36 (1.49–3.74)‡</td>
<td>2.72 (1.44–5.20)†</td>
</tr>
<tr>
<td>10+</td>
<td>18 (22.5)</td>
<td>3.47 (1.95–6.16)‡</td>
<td></td>
</tr>
<tr>
<td>Smoked last trimester</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of pregnancy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>5 (0.5)</td>
<td>1.00 (1.00–1.49)‡</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>8 (3.1)</td>
<td>6.32 (2.05–19.48)†</td>
<td></td>
</tr>
</tbody>
</table>

†p<0.01; ‡p<0.001

Multiple logistic regression analyses showed that smoking remained significantly associated with increased likelihood of SGA. The adjusted OR for 1–9 cigarettes was 2.1 (95%CI=1.3–3.4; p<0.01)—and for the heavier smoking dose, the adjusted OR was 2.7 (95%CI=1.4–5.1; p<0.01).

Income and twin status were also independently associated with SGA. Compared to those with household incomes <$20,000 per annum, mothers with incomes $20,001–$40,000 per annum were at a reduced risk of SGA (OR=0.4; 95%CI=0.3–0.7; p<0.01). Mothers delivering twins exhibited over 15 times the risk of SGA than singleton births (OR=15.8, 95%CI=4.2–59.3; p<0.001).

Discussion

Three health indicators were examined in relation to maternal smoking among Pacific communities, two pertinent to infant wellbeing (preterm birth and birth weight) and one to maternal wellbeing (asthma).

Prior to discussing findings, some limitations are acknowledged. Measurement of smoking was based on use during a specific timeframe, thus data regarding non-smokers may also include former smokers. Maternal reporting may have underestimated smoking and recall bias cannot be ruled out.

Studies comparing the use of self-report versus biomarkers of smoking such as cotinine tests have shown self-report to be an accurate measure of smoking status, although dose may be underreported. Thus, it is possible that cigarette consumption data could be conservative. However, bias was minimised with smoking questions...
forming a small part of the overall interview and interviewers not being health workers.

Birth data were extracted from hospital records recognising that estimation of some gestational ages may lack precision without confirmation by ultrasound scans. Inaccuracies may also have occurred with asthma data, as it was not feasible to confirm diagnoses through testing or review of records.

As many Pacific people do not have a regular doctor or use preventive medication, estimates of asthma are likely to be conservative, especially given that measurement was based on diagnosis and medication rather than the presence of symptoms. Data pertaining to onset and duration of asthma and other known risk factors such as family history or personal atopy were not available so it is not known how these and other unmeasured factors would have influenced the relationships observed between smoking and asthma. Recognising possible limitations, this study adds to the overwhelming, accumulating evidence that smoking has adverse consequences for both the smoker and their offspring.

The link between smoking and negative health consequences (including respiratory illness) is widely accepted. Furthermore, exposure to tobacco smoke is a recognised risk factor for asthma symptoms in children, however, inconsistent findings have been reported for adults.

Although research indicates that smoking negatively affects asthma, direct causation has not been confirmed due to methodological differences in measurement coupled with biases arising from alterations in smoking habits by asthmatics. In this study, analyses controlling for sociodemographic factors revealed a significant association between current smoking and maternal asthma. Dose response effects were evident, with light-to-moderate smokers being approximately twice as likely (and heavy smokers being over three and a half times as likely) to have asthma than non-smokers.

Little is known about the mechanisms of how smoking influences asthma morbidity, although genetic and environmental factors are thought to underlie the development of the disease. Smoking may heighten or suppress inflammatory responses of the airways and may modify immunological responses. Thus smoking cessation is important, particularly for those with compromised respiratory health. Along with the need to reduce individual suffering from asthma morbidity, the economic impact of the condition could be reduced.

In addition to adverse consequences of smoking for maternal respiratory health, research has consistently shown smoking to be associated with negative pregnancy outcomes, including preterm delivery and lower birth weight.

In New Zealand, Pacific women tend to have fewer preterm deliveries compared to women of other ethnic groups. When risk of preterm delivery from smoking was examined in our cohort, no association (contrary to most research) between increased smoking and increased preterm deliveries was found. Our findings are in line with others who have also failed to find an association.

It is possible that other factors not measured, such as pregnancy complications, are stronger predictors of preterm delivery than smoking. Others have suggested that methodological differences in estimation of gestational age and possible publication
biases may contribute to the occasional observed lack of association between smoking and preterm birth. 18

Pacific infants tend to weigh more than infants of other ethnic groups in New Zealand, possibly masking the effects of smoking. Analyses examining mean birth weight, LBW, and SGA were used to investigate the relationship between birth weight and smoking. Irrespective of which birth weight variable was examined, an adverse association with smoking was found. Despite controlling for potential confounders and corroborating previous research, 11,12,16–18 smoking was significantly associated with reduced birth weight with a trend towards a dose-response effect.

In line with previous research, 10,12,15,36 smokers were significantly more likely to deliver LBW infants compared to non-smokers. Although it was not possible to control for other factors, smoking was associated with a six-fold increased risk of a LBW infant. Consistent with the literature, 14,15,38 mothers who smoked 1–9 cigarettes daily had just over twice the risk (and mothers who smoked 10+ cigarettes daily had almost three times the risk) of delivering an infant considered SGA compared to non-smoking mothers.

Adverse risks associated with a lower birth weight or being SGA can be significant and include compromised immunocompetence, subnormal growth, increased morbidity and mortality in infancy, with some risks persisting over several years 39 (including increased risk of childhood obesity, type 2 diabetes, and cardiovascular disease). 40 Maternal smoking is a preventable contributor to poor foetal growth that precedes the development of these conditions.

Evidence is sufficient to regard the relationship between smoking and intrauterine growth restriction and LBW as causal, 2,14 although specific mechanisms remain unclear. Smoking during pregnancy exposes the foetus to higher concentrations of nicotine than present in the mother and carbon monoxide interferes with the release of oxygen into foetal tissues, retarding growth. 41,42

Although attention has been directed at nicotine and carbon monoxide, there are thousands of chemicals in cigarettes and little known about the interaction among these components. 43

This study of Pacific families provides further evidence that smoking confers increased risk to both maternal and reproductive health and these results reinforce the need to commit additional effort into smoking prevention and cessation initiatives.

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References:


Prescribing oxygen therapy. An audit of oxygen prescribing practices on medical wards at North Shore Hospital, Auckland, New Zealand

Matthew Boyle, Janice Wong

Abstract

Aim To assess the frequency and accuracy of inpatient oxygen prescription at North Shore Hospital, Auckland.

Method Between 14 April 2005 and 14 May 2005, 100 medical inpatients receiving oxygen therapy were randomly selected for chart review. For each patient, the clinical diagnosis, oxygen prescription (if present), and initial medical plan were analysed in conjunction with the oxygen flow rate and oxygen saturations (as documented in the observation chart).

Results Only 8% of patients receiving oxygen had it prescribed in their medication chart. The majority (75%) of oxygen prescriptions were inadequate.

Conclusion Current rates of oxygen prescription on medical wards at North Shore Hospital, Auckland, are unsatisfactory. This poor oxygen prescription rate carries serious potential consequences.

Oxygen may be a life-saving therapy and is an important aide in the treatment of various conditions (Figure 1). Oxygen can, however, cause dangerous effects (Figure 2); it should be considered a drug and therefore only be available by prescription.

Figure 1. American College of Chest Physicians and National Heart, Lung, and Blood Institute recommendations for instituting oxygen therapy.¹

- Cardiac and respiratory arrest
- Hypoxaemia (PaO₂<7.8 kPa, SaO₂<90%)
- Hypotension (systolic blood pressure <100 mmHg)
- Low cardiac output and metabolic acidosis (bicarbonate <18 mmol/L)
- Respiratory distress (respiratory rate >24/min)

It is generally accepted that in order to ensure safe and effective treatment, oxygen prescriptions should cover the flow rate, concentration, delivery system, duration, and monitoring of treatment.¹ It is, however, recognised that oxygen is poorly prescribed by doctors.¹
Figure 2. Dangers of oxygen therapy.\textsuperscript{1,2}

- Respiratory system toxicity (e.g. tracheobronchitis, absorption atelectasis, bronchopulmonary dysplasia, acute and chronic parenchymal lung injury)
- Maladaptive physiologic response and hypercapnia in patients with chronic obstructive pulmonary disease
- Nonmedical adverse effects (e.g. fire hazards)
- Paul-Bert effect (hyperbaric oxygen causing severe cerebral vasoconstriction and epileptic fits)

A previous study,\textsuperscript{3} conducted in Manchester, England, showed that only 55\% of inpatients receiving oxygen therapy had it prescribed. After introduction of a specific oxygen prescription chart, this oxygen prescription rate rose to 91\%.

Another study,\textsuperscript{4} conducted in 1992 in Christchurch, New Zealand, showed that one third of inpatients receiving oxygen did not have it prescribed. A further study,\textsuperscript{5} conducted in Sunderland, England, showed that only 16\% of inpatients receiving oxygen therapy had it prescribed. Additional studies have shown that oxygen is not prescribed and administered with the same procedural care as other medications,\textsuperscript{6} and that oxygen therapy is often administered excessively.\textsuperscript{7}

Waitemata District Health Board Clinical Practice Guidelines clearly state that the assessment of oxygen requirement and the prescription of oxygen should only be carried out by medical staff. However, in an emergency and for patient safety, the Guidelines allow oxygen to be initiated by a nurse or midwife while the patient is awaiting medical assistance.

When administered correctly, with careful evaluation of its potential benefits and side effects, oxygen may be life-saving. It is clear, however, that oxygen is often administered to patients without prescription.

In order to monitor and promote the safe administration of oxygen at North Shore Hospital, we conducted a brief audit of inpatient oxygen prescription.

**Method**

The audit was undertaken between 14 April 2005 and 14 May 2005.

Patients were randomly selected for chart review from the general medical wards at North Shore Hospital, Auckland. A random number generator was used to select dates of review and wards for review. Inpatients on reviewed wards were then assigned numbers and a random number generator was used to select patients for chart review. Those patients receiving oxygen therapy as documented in their observation chart were included in the audit.

To avoid bias, those patients under the care of the author’s medical team were excluded. All ward personnel, other than the author’s medical team, were unaware of the audit. To reduce the effect of inaccurate recharting, the precise date at which oxygen had been administered was correlated with the patient’s medication chart for that date to examine whether the administrated oxygen had been prescribed.

A total of 100 inpatients from general medical wards were included in the audit, with principle diagnostic categories of respiratory (52 patients), cardiovascular (37 patients), neurology (6 patients), and other (5 patients).
For each patient, the clinical diagnosis, oxygen prescription (if present), and initial medical plan were analysed in conjunction with the oxygen flow rate and oxygen saturations (as documented in the observation chart). Laboratory records were reviewed to assess arterial blood gas analyses.

**Results**

Only 8 of the 100 patients (8%) receiving oxygen had oxygen prescribed in their medication chart (Figure 3), and the majority (75%) of the oxygen prescriptions were inadequate with respect to recommended guidelines for safe oxygen prescription. As a result, only 2 of the 100 patients (2%) receiving oxygen had an adequate oxygen prescription. Fourteen of the 100 patients (14%) receiving oxygen had oxygen therapy listed in their initial medical plan.

![Figure 3. Rate of oxygen therapy prescription during audit](image)

The level of oxygen that was administered during the study varied between 1–5 L/min.

Of the 92 patients receiving oxygen without prescription, 14 had a previous diagnosis of chronic obstructive pulmonary disease, with 5 of these patients having previously documented carbon dioxide retention on arterial blood gas analysis.

Of the 100 patients receiving oxygen, 19 had arterial blood gas analysis performed during the audit.

**Discussion**

Oxygen therapy can be life-saving; treatment of hypoxaemia is essential. Oxygen is a drug, however, and should be prescribed as it can have detrimental effects. Current rates of oxygen prescription in medical wards at North Shore Hospital are unsatisfactory; only 8% of patients who were receiving oxygen during the study had oxygen prescribed, with the majority of oxygen prescriptions being inadequate.
It should be noted that 19% of patients receiving oxygen had arterial blood gas analysis performed during the study; this indicates a moderately higher level of medical monitoring than is suggested by the low prescription rate. In any case, the poor oxygen prescription rate seen during our audit carries serious potential consequences.

The possible adverse effects of oxygen therapy include respiratory system toxicity (e.g. tracheobronchitis, absorption atelectasis, bronchopulmonary dysplasia, and acute and chronic parenchymal lung injury), maladaptive physiologic responses (e.g. hypercapnia in patients with chronic obstructive pulmonary disease), nonmedical adverse effects (e.g. fire hazards) and the Paul-Bert effect (an adverse effect which is only seen in patients exposed to hyperbaric oxygen).1,2

Although all very serious potential adverse effects, it is uncommon to see these effects in non-sedated medical inpatients receiving low flow oxygen such as those in our audit.

Tracheobronchitis, absorption atelectasis, bronchopulmonary dysplasia, and acute parenchymal lung injury have been observed in patients breathing high concentrations of oxygen, however have not been seen in patients receiving low-flow oxygen therapy.8–12 In contrast, histologic changes consistent with chronic parenchymal lung injury have been seen in patients receiving low-flow oxygen therapy (1–6 L/min).13,14 These changes are only seen after at least 7 months of oxygen therapy, however, and do not appear to contribute toward mortality.13

Elevation of arterial carbon dioxide tension in patients with chronic obstructive pulmonary disease treated with oxygen has been noted for years.15 Fourteen patients who were receiving oxygen without prescription during our audit had a previous diagnosis of chronic obstructive pulmonary disease; five of these patients had previously documented carbon dioxide retention on arterial blood gas analysis. Although our audit did not extend to recording unfavourable events, this raises the very serious concern of oxygen-associated respiratory depression and detrimental hypercapnia. Due to this concern, it is generally recommended that (until arterial blood gas analysis is available) only low-flow oxygen is used in patients with chronic obstructive pulmonary disease.16

Nonmedical hazards of oxygen therapy include fire hazards and the hazards associated with high-pressure oxygen cylinders, oxygen concentrators, and oxygen delivery systems. These hazards are more commonly associated with long-term oxygen therapy than inpatient oxygen therapy.17

Despite the dangers of unregulated oxygen therapy, hypoxaemia is a much graver situation. Hypoxaemia accounts for more deaths and permanent disability than can be justified by the relatively small possible risks associated with oxygen therapy.1 Descriptions in the literature of inpatient low flow oxygen therapy resulting in clinically significant adverse effects are scarce. While striving to improve oxygen prescription rates, care must be taken not to overstate the potential dangers of oxygen and inadvertently promote inadequate management of hypoxaemia.

Our audit clearly illustrates the need for improved oxygen prescription at North Shore Hospital. Such improvements may be seen with educational initiatives such as lectures and in-service instruction for junior doctors, or with possible revisions of the
current prescription chart to provide a more specific prescription chart for oxygen. Further more in-depth audits of oxygen prescription at North Shore Hospital are warranted to examine any adverse effects stemming from the current poor prescription rate, and to evaluate any future initiatives.

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**References:**

Community hospital versus tertiary hospital comparison in the treatment and outcome of patients with acute coronary syndrome: a New Zealand experience

Eng Wei Tang, Cheuk-Kit Wong, Peter Herbison

Abstract

**Aims** To compare the baseline characteristics, use of evidence-based medications, rate of revascularisation, and mortality of acute coronary syndrome (ACS) patients managed in a community hospital (Invercargill Hospital) without, and a tertiary teaching hospital (Dunedin Hospital) with, catheterisation and an interventional facility.

**Methods** All patients with ACS admitted into Dunedin and Invercargill coronary care units (CCUs) between 2000–2002 inclusive were included in the study.

**Results** Major baseline characteristics including age, history of diabetes, heart rate and systolic blood pressure at presentation were not different between the two centres. However, the proportions of patients with ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI) were higher in Invercargill CCU. More Invercargill patients experienced a cardiac arrest or clinical heart failure on hospital arrival.

The use of evidence-based medications, coronary angiography (65.5% vs 20.2%, p<0.00001), and revascularisation (46.7% vs 16.4%, p<0.0005) were significantly higher in patients admitted into Dunedin CCU.

The in-hospital, 6-months, and 1-year mortality was significantly lower (absolute mortality difference of 4.3%, 9.5%, and 10.0%, p<0.05, respectively) for ACS patients admitted into Dunedin CCU. Using multivariable logistic regression incorporating baseline characteristics, use of evidence-based medicine on arrival and transfer for angiography, the 1-year adjusted hazard ratio 3.02 (95%CI 1.60–5.71) remains significantly higher for patients in Invercargill Hospital.

**Conclusion** There was a disparity in ACS outcome between community and tertiary hospitals in New Zealand. The use of evidence-based medicine in all ACS patients should be encouraged even if revascularisation was not offered.

Information derived from randomised clinical trials are used to formulate recommended treatment guidelines for the management of acute coronary syndrome (ACS). Registry studies published locally and overseas have demonstrated the underuse of evidence-based therapies (both medications and revascularisation) in ‘real-life’ high-risk ACS patients. The lower rate of revascularisation in community hospitals could be due to the lack of facilities, necessitating transfer of patients to tertiary centres.

Although revascularisation is generally beneficial for high-risk ACS patients, the GRACE registry showed similar short-term (6 months) outcome between patients...
admitted into community hospitals for whom revascularisation required transferral and patients admitted directly into tertiary centres.

There is currently no local data comparing the treatment and outcome of ACS patients admitted into a community hospital (staffed by general physician without catheterisation facilities) versus a tertiary teaching hospital (with cardiologists and catheterisation laboratory). However this information is crucial to both the public and health professionals/policymakers when we strive to offer an equitable healthcare service to all New Zealanders.

Hence the aim of this study was to compare the management (use of evidence-based medications and revascularisation) and mortality of patients with ACS managed in a community hospital versus those managed in a tertiary hospital.

Methods

This is a retrospective registry study including all consecutive patients with ACS admitted into the coronary care units (CCUs) of two related centres in New Zealand, including the tertiary hospital in Dunedin, Otago Province (Dunedin Hospital) and the regional hospital in Invercargill, Southland Province (Invercargill Hospital) from the years 2000–2002 inclusive. Dunedin Hospital served as the referral centre for Invercargill Hospital during the study. Stable patients were transferred from Invercargill to Dunedin via ambulance, a 3-hour journey. Unstable patients were retrieved via helicopter (Dunedin Hospital has a helipad).

All patients had ACS as their discharge diagnosis and were above 18 years of age. Patients having ACS precipitated by significant non-cardiac comorbidity, trauma, or surgery were excluded. The first admission with ACS was used for analysis if readmissions with ACS were present. Patients initially admitted to Invercargill Hospital and subsequently transferred to Dunedin Hospital were categorised as Invercargill patients. This study protocol was in accordance with local hospital research guidelines.

All clinical data were collected by a research physician, which includes:

- Baseline demographic characteristics: age, sex, cardiac risk factors, history of ischaemic heart disease, history of stroke, peripheral vascular disease, smoking, time to presentation, and door-to-needle time for thrombolysis.
- Presenting clinical features: heart rate, blood pressure, Killip class, episodes of cardiac arrest on arrival, and cardiogenic shock.
- ECG characteristics: degree of ST-deviation and T-waves changes in the initial ECG.
- Laboratory findings: initial and maximum troponin rise, creatinine level at presentation.
- Left ventricular function on echocardiography or left ventriculography during cardiac catheterisation.
- Treatment: in-hospital medications in the first 24-hour, reperfusion and revascularisation therapy, and the use of intra-aortic balloon pump.

Death was defined as all-cause mortality during hospitalisation and over a 1-year follow-up period. Information on the deaths was obtained from medical records and the national death registry.

Definition of ACS—Patients with ACS were classified into:

- **ST-segment elevation myocardial infarction (STEMI):** defined as having ST segment elevation ≥1 mm in two contiguous leads (or ≥2 mm in V1 to V3 leads), or new left bundle branch block (LBBB) together with chest pain for > 30 minutes and/or evidence of myonecrosis with elevated troponin I (Abbott AxSYM assay) ≥2.0 mcg/L.
- **Non-ST-segment elevation myocardial infarction (NSTEMI):** defined as no ST-segment elevation on ECG despite elevated troponin I (Abbott AxSYM assay) ≥ 2.0 mcg/L and chest pain for more than 30 minutes.
- **Unstable angina:** defined as ischaemic chest pain lasting more than 30 minutes with no evidence of myonecrosis or ST elevation.
Statistical analysis—Statistical analysis was carried out in STATA v8 software. Data are presented as mean with 95% confidence intervals, or median with interquartile ranges or actual numbers with percentages as appropriate. Chi-squared tests were used to compare proportions, and t-tests or Mann-Whitney U tests for continuous data. The test was double-sided and considered to be statistically significant at $\alpha<0.05$.

Multivariable analysis was performed using Poisson regression with robust standard errors for patients who died in hospital and Cox’s proportional hazards regression for hospital survival up to 1-year to examine the association between admission to Invercargill Hospital vs Dunedin Hospital and mortality during in-hospital stay, 6-months, and 1-year follow-up.

These models were adjusted for age, sex, history of hypertension, diabetes mellitus, history of ischaemic heart disease, history of coronary artery bypass graft, history of stroke, heart rate, systolic blood pressure, Killip class and cardiac arrest on presentation, maximum troponin elevation, renal impairment, and the subset of acute coronary syndrome.

Further models were run with adjustments for the use of evidence-based medications in the first 24 hours of admission and the use of coronary angiography, as well as those already in the model.

Results

Patients—From 1 January 2000 to 31 December 2002, 843 patients and 299 patients were admitted into the tertiary (Dunedin Hospital) and community (Invercargill Hospital) CCUs respectively (Table 1).

There was no difference in age, sex—and history of hypertension, diabetes, smoking, dyslipidaemia, cerebral vascular disease, or peripheral vascular disease—between patients admitted to Dunedin Hospital versus patients admitted to Invercargill Hospital.

More patients admitted to Dunedin Hospital had a history of ischaemic heart disease (49.2% vs 40.5%, $p=0.010$), previous coronary artery bypass graft (10.1% vs 2.0%, $p<0.0005$) and renal impairment (23.5% vs 15.4%, $p=0.003$).

There were a higher proportion of patients with STEMI (56.5% vs 33.3%) or NSTEMI (34.5% vs 26.1%, $p<0.0005$ for both) amongst patients admitted to Invercargill Hospital. Furthermore, there was a higher incidence of cardiac arrest (12.7 vs 7.3%, $p<0.004$) and clinical heart failure (24.1% vs 17.8%, $p=0.039$).

Amongst patients with non-ST-elevation acute coronary syndrome (NSTEACS), those admitted into Invercargill more frequently had presenting ECG showing ischaemic ST depression ($p=0.006$).

Management—In the first 24-hours, patients with STEMI were as likely to receive aspirin, beta-blockers, statins, and thrombolysis in both centres. However, the use of statins in both hospitals was low (21.1% vs 17.2%, $p=0.312$).

In patients admitted into Dunedin Hospital, percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) during the index admission was more frequently performed (46.4% vs 16.6%, $p<0.0005$; Table 2). In addition, patients in Dunedin Hospital were more likely to be discharged on statins (66.4% vs 42.3%, $p<0.0005$) or an ACE-inhibitor (60.2% vs 49.7%, $p=0.040$; Table 2) than patients admitted to Invercargill Hospital, despite having a similar incidence of dyslipidaemia (60.4% vs 60.7%, $p=0.940$), clinical heart failure (Killip class $>1$) on admission (20.7% vs 18.9%, $p=0.737$) and echocardiographic measurements of left ventricular systolic dysfunction ($p=0.772$; Table 2).
Table 1. Baseline characteristics of ACS patients

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Tertiary interventional hospital</th>
<th>Community hospital</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of patients</td>
<td>n=842</td>
<td>n=299</td>
<td></td>
</tr>
<tr>
<td>% Of Mm</td>
<td>511 (60.6%)</td>
<td>165 (65.2%)</td>
<td>0.159</td>
</tr>
<tr>
<td>Age</td>
<td>65.9 (65.1-66.8)</td>
<td>64.7 (63.2-66.2)</td>
<td>0.166</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>430 (51.0%)</td>
<td>134 (44.8%)</td>
<td>0.068</td>
</tr>
<tr>
<td>Diabetes</td>
<td>141 (16.7%)</td>
<td>51 (17.1%)</td>
<td>0.889</td>
</tr>
<tr>
<td>Smoking history</td>
<td>414 (53.8%)</td>
<td>156 (52.2%)</td>
<td>0.630</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>323 (39.0%)</td>
<td>191 (63.8%)</td>
<td>0.600</td>
</tr>
<tr>
<td>History of IHD</td>
<td>41 (49.0%)</td>
<td>121 (40.1%)</td>
<td>0.010</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>8 (10.1%)</td>
<td>6 (2.0%)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>CVA/TIA</td>
<td>110 (13.5%)</td>
<td>20 (0.6%)</td>
<td>0.071</td>
</tr>
<tr>
<td>PVD</td>
<td>50 (6.0%)</td>
<td>19 (6.4%)</td>
<td>0.744</td>
</tr>
</tbody>
</table>

### Presenting characteristics

| Time to presentation (hr)              | 1.5 (1.6-1.9)                    | 10.9 (7.4-14.5)    | 0.714   |
| Symptom to thrombolysis for STEMI      | 4.4 (3.4-5.5)                    | 5.1 (3.7-6.5)      | 0.453   |
| Heart rate, median (bpm)              | 77 (71-79)                       | 83 (77-82)         | 0.087   |
| SBP, median (IQR), mmHg               | 138 (136-140)                    | 135 (133-139)      | 0.026   |
| DEP, median (IQR), mmHg               | 77 (75-78)                       | 81 (79-82)         | 0.0001  |
| Killip class                           |                                  |                    |         |
| I                                       | 69 (32.2%)                       | 127 (75.9%)        |         |
| II                                      | 112 (13.7%)                      | 60 (20.1%)         | 0.039   |
| III+IV                                  | 32 (4.1%)                       | 12 (4.0%)          |         |

### ACS subgroup

| STEMI                                   | 28 (33.3%)                       | 165 (66.5%)        |         |
| NSTEMI                                  | 220 (26.1%)                      | 103 (34.5%)        | <0.0005 |
| Unstable angina                         |                                  |                    |         |
|                                         | 34 (4.0%)                        | 27 (9.0%)          |         |

### S-T depression for non-ST-elevation acute coronary syndrome (NSTEMI) on arrival

| No S-T change                          | 559 (66.3%)                      | 171 (57.3%)        |         |
| 1 mm                                   | 156 (18.3%)                      | 43 (14.5%)         |         |
| Between 1-2 mm                         | 56 (6.7%)                        | 20 (6.3%)          | 0.006   |
| 2 to <3 mm                             | 49 (5.8%)                        | 31 (10.3%)         |         |
| 23 mm                                  | 23 (2.7%)                        | 24 (8.1%)          |         |

### T-wave changes for NSTEMI on arrival

| No T-wave change                       | 348 (47.2%)                      | 106 (35.4%)        |         |
| Flat/Low T wave                        | 241 (32.8%)                      | 113 (37.9%)        | 0.064   |
| T-wave inversion                       | 174 (23.6%)                      | 72 (24.4%)         |         |
| Deep T inversion                       | 30 (4.2%)                        | 7 (2.4%)           |         |

| Positive troponin                      | 501 (50.4%)                      | 372 (91.3%)        | <0.0005 |
| Cardiac arrest on admission            | 62 (7.8%)                        | 38 (12.7%)         | 0.004   |
| Renal impairment (Cr>0.12 mmol/L)      | 198 (23.3%)                      | 66 (15.4%)         | 0.003   |
| Initial creatinine level, mmol/L       | 109 (135-112)                    | 102 (98-108)       | 0.052   |

### LV dysfunction

| Good function                          | 350 (41.5%)                      | 103 (34.5%)        |         |
| Mild                                    | 215 (25.3%)                      | 50 (19.2%)         | 0.288   |
| Moderate                                | 179 (21.5%)                      | 62 (20.7%)         |         |
| Severe                                  | 99 (11.8%)                       | 42 (4.5%)          |         |

*Age and creatinine levels were reported as mean and 95% confidence intervals*
Table 2. Evidence-based medicine and revascularisation procedures

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Tertiary interventional hospital</th>
<th>Community hospital</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>For STEMI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st 24-hour</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>278/99.3% (95% CI)</td>
<td>166/98.2% (95% CI)</td>
<td>0.300</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>226/80.7% (95% CI)</td>
<td>128/75.7% (95% CI)</td>
<td>0.211</td>
</tr>
<tr>
<td>Beta-blocker if Killip class = I</td>
<td>176/61.1% (95% CI)</td>
<td>113/62.5% (95% CI)</td>
<td>0.454</td>
</tr>
<tr>
<td>Statins</td>
<td>59/21.1% (95% CI)</td>
<td>29/17.2% (95% CI)</td>
<td>0.312</td>
</tr>
<tr>
<td>Lytics in &lt;12hr</td>
<td>213/76.1% (95% CI)</td>
<td>139/82.3% (95% CI)</td>
<td>0.123</td>
</tr>
<tr>
<td>Clinical characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Killip class I</td>
<td>159/60.4% (95% CI)</td>
<td>102/60.7% (95% CI)</td>
<td>0.940</td>
</tr>
<tr>
<td>Killip class II, III, or IV</td>
<td>218/79.3% (95% CI)</td>
<td>137/81.1% (95% CI)</td>
<td></td>
</tr>
<tr>
<td>LV function</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good function</td>
<td>72(30.3%) (95% CI)</td>
<td>33(33.6%) (95% CI)</td>
<td>0.737</td>
</tr>
<tr>
<td>Mild</td>
<td>565(33.3%) (95% CI)</td>
<td>318(31.9%) (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>50(23.7%) (95% CI)</td>
<td>28(24.3%) (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>27(11.4%) (95% CI)</td>
<td>13(9.7%) (95% CI)</td>
<td></td>
</tr>
<tr>
<td>On discharge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>253/93.8% (95% CI)</td>
<td>147/93.7% (95% CI)</td>
<td>0.881</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>214(83.6%) (95% CI)</td>
<td>115/77.2% (95% CI)</td>
<td>0.111</td>
</tr>
<tr>
<td>ACE-I</td>
<td>154(60.2%) (95% CI)</td>
<td>74(49.7%) (95% CI)</td>
<td>0.040</td>
</tr>
<tr>
<td>Statins</td>
<td>150(64.4%) (95% CI)</td>
<td>83(42.3%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Received angiography</td>
<td>162(57.5%) (95% CI)</td>
<td>36(21.4%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>PCI/CABG</td>
<td>118(46.4%) (95% CI)</td>
<td>23(15.6%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td><strong>For NSTEMI—Non-ST STEMI + Unstable Angina</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st 24-hour</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>552/98.1% (95% CI)</td>
<td>124/95.4% (95% CI)</td>
<td>0.077</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>449(79.9%) (95% CI)</td>
<td>90(69.2%) (95% CI)</td>
<td>0.028</td>
</tr>
<tr>
<td>Beta-blocker if Killip class = I</td>
<td>387(84.3%) (95% CI)</td>
<td>73(81.1%) (95% CI)</td>
<td>0.421</td>
</tr>
<tr>
<td>Statins</td>
<td>238(73.0%) (95% CI)</td>
<td>30(23.1%) (95% CI)</td>
<td>0.003</td>
</tr>
<tr>
<td>Heparin</td>
<td>522(92.6%) (95% CI)</td>
<td>120(92.3%) (95% CI)</td>
<td>0.924</td>
</tr>
<tr>
<td>Clinical characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>On discharge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>523(96.7%) (95% CI)</td>
<td>113(95.8%) (95% CI)</td>
<td>0.625</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>416(76.6%) (95% CI)</td>
<td>76(64.4%) (95% CI)</td>
<td>0.009</td>
</tr>
<tr>
<td>ACE-I</td>
<td>274(50.1%) (95% CI)</td>
<td>57(48.3%) (95% CI)</td>
<td>0.638</td>
</tr>
<tr>
<td>Statins</td>
<td>350(64.6%) (95% CI)</td>
<td>48(40.7%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Received PCI/CABG</td>
<td>276(48.9%) (95% CI)</td>
<td>25(18.5%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Medical management</td>
<td>256(51.1%) (95% CI)</td>
<td>106(81.5%) (95% CI)</td>
<td>&lt;0.0005</td>
</tr>
</tbody>
</table>

NZMJ 21 July 2006, Vol 119 No 1238
URL: http://www.nzma.org.nz/journal/119-1238/2078/
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Table 3. Baseline characteristics of NSTEACS patients from the community hospital (Invercargill Hospital) transferred and not transferred to the tertiary centre (Dunedin Hospital)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Transferred (n=33)</th>
<th>Not transferred (n=97)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of Men</td>
<td>20(60.6%)</td>
<td>59(60.3%)</td>
<td>0.982</td>
</tr>
<tr>
<td>Age</td>
<td>60(55-65)</td>
<td>68(66-71)</td>
<td>0.003</td>
</tr>
<tr>
<td>Hypertension</td>
<td>17(11.5%)</td>
<td>52(33.6%)</td>
<td>0.833</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5(15.2%)</td>
<td>27(37.8%)</td>
<td>0.144</td>
</tr>
<tr>
<td>History of IHD</td>
<td>19(57.6%)</td>
<td>51(52.6%)</td>
<td>0.619</td>
</tr>
<tr>
<td>Median heart rate/min</td>
<td>71(65-77)</td>
<td>82(76-87)</td>
<td>0.030</td>
</tr>
<tr>
<td>SBP, median (IQR), mmHg</td>
<td>137(129-144)</td>
<td>138(133-145)</td>
<td>0.771</td>
</tr>
<tr>
<td>DBP, median (IQR), mmHg</td>
<td>80(75-85)</td>
<td>81(78-84)</td>
<td>0.548</td>
</tr>
<tr>
<td>Creatinine level, μmol/L</td>
<td>87(80-94)</td>
<td>110(108-121)</td>
<td>0.012</td>
</tr>
<tr>
<td>Renal impairment (Cr&gt;0.12mmol/L)</td>
<td>3(9.1%)</td>
<td>20(20.6%)</td>
<td>0.134</td>
</tr>
<tr>
<td>Killip class</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>29(37.9%)</td>
<td>61(52.9%)</td>
<td>0.024</td>
</tr>
<tr>
<td>II</td>
<td>41(12.1%)</td>
<td>32(33.0%)</td>
<td></td>
</tr>
<tr>
<td>III-IV</td>
<td>0(0.0%)</td>
<td>4(4.1%)</td>
<td></td>
</tr>
<tr>
<td>S-T depression for non-ST-elevation acute coronary syndrome (NSTEACS) on arrival</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No S-T change</td>
<td>18(54.6%)</td>
<td>53(58.2%)</td>
<td>0.690</td>
</tr>
<tr>
<td>&lt;= 1 mm</td>
<td>6(18.2%)</td>
<td>12(12.3%)</td>
<td></td>
</tr>
<tr>
<td>Between 1-2 mm</td>
<td>3(9.1%)</td>
<td>6(6.9%)</td>
<td></td>
</tr>
<tr>
<td>2 to &lt;3mm</td>
<td>2(6.1%)</td>
<td>11(11.2%)</td>
<td></td>
</tr>
<tr>
<td>23mm</td>
<td>4(12.1%)</td>
<td>6(6.6%)</td>
<td></td>
</tr>
<tr>
<td>T-wave changes for NSTEACS on arrival</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No T-wave change</td>
<td>10(30.3%)</td>
<td>34(37.0%)</td>
<td>0.903</td>
</tr>
<tr>
<td>Flat/biphasic T</td>
<td>14(42.4%)</td>
<td>34(37.0%)</td>
<td></td>
</tr>
<tr>
<td>T-wave inversion</td>
<td>9(26.9%)</td>
<td>22(23.9%)</td>
<td></td>
</tr>
<tr>
<td>Deep T inversion</td>
<td>1(3.0%)</td>
<td>2(2.2%)</td>
<td></td>
</tr>
<tr>
<td>LV dysfunction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good Function</td>
<td>10(32.6%)</td>
<td>14(14.9%)</td>
<td>0.143</td>
</tr>
<tr>
<td>Impaired Function</td>
<td>9(27.4%)</td>
<td>33(30.2%)</td>
<td></td>
</tr>
<tr>
<td>On-discharge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>32(100.0%)</td>
<td>21(98.3%)</td>
<td>0.163</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>26(81.9%)</td>
<td>30(58.1%)</td>
<td>0.020</td>
</tr>
<tr>
<td>ACEI</td>
<td>15(46.9%)</td>
<td>42(48.3%)</td>
<td>0.850</td>
</tr>
<tr>
<td>Stattes</td>
<td>24(73.5%)</td>
<td>24(73.5%)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>PCI/CABG</td>
<td>24(72.7%)</td>
<td>0(0.0%)</td>
<td>&lt;0.0005</td>
</tr>
</tbody>
</table>

*Age and creatinine levels were reported as mean and 95% confidence intervals
For patients with non-ST-elevation acute coronary syndrome (NSTEACS), there were no difference in the use of aspirin, beta-blockers, and heparin during the first 24 hours amongst the two centres. For patients admitted to Dunedin Hospital, the use of statins was more frequent both in the first 24 hours of admission (37.0% vs 23.1%, p<0.003) and on discharge (64.4% vs 40.7%, p<0.0005), despite similar rates of dyslipidaemia (62.4% vs 67.7%, p=0.303). More patients with NSTEACS received revascularisation (PCI/CABG) during index admission in Dunedin Hospital (48.9% vs 18.5%, p<0.0005).

Patients with NSTEMI admitted into Dunedin Hospital were more likely to receive clopidogrel (27.4% vs 0%, p<0.0005) and glycoprotein IIbIIIa (20.1% vs 8.7%, P=0.008), which parallel the higher revascularisation rate: PCI (37.8% vs. 14.1%, P<0.0005) and CABG (19.8% vs. 8.3%, p=0.008).

Demographics of NSTEACS patients transferred to a tertiary centre—Compared to patients not transferred to Dunedin Hospital, transferred patients were younger (60 vs 68, p=0.003); with a lower incidence of diabetes mellitus (15.2% vs 27.8%, p=0.144); more clinically stable with a lower heart rate on hospital admission (71 vs 82 beats/minute, p=0.030); and a lower incidence of clinical heart failure (12.1% vs. 37.1%, p=0.024, Table 3). Their mean creatinine level was also significantly lower (87 vs 110 µmol/L, p=0.012).

Clinical outcome—The mortality rate for ACS is significantly higher for patients admitted into Invercargill Hospital during index admission, at 6 months and at 1 year (Table 4). At 1-year, there was a 10.0% absolutely mortality difference between the two hospitals (12.1% vs 22.1%, p<0.0005). The adjusted and unadjusted hazard ratios for death are shown in Table 4.

Amongst the subgroup of ACS, patients with NSTEMI (but not STEMI or unstable angina) in Invercargill Hospital have a significantly higher in-hospital, 6-months, and 1-year mortality and risk of dying compared to patients admitted into Dunedin Hospital (Table 5 and Table 6).

Adjusting for patient’s baseline characteristics (including cardiac arrest on arrival) and use of evidence-based medications during the first 24 hours, the higher risk of death remains for patients admitted to Invercargill Hospital without catheterisation facilities (Table 4).

After further adjusting for angiography and/or revascularisation, the higher risk of death for Invercargill patients remains significant. Patients first admitted there were at a 202% increased risk of death at 1-year (adjusted hazard ratio 3.02, 1.60–5.71).
Table 4. Mortality rates of patients with acute coronary syndrome followed up to 1 year comparing patients admitted into a tertiary vs a community hospital

<table>
<thead>
<tr>
<th></th>
<th>Hospital with catheterisation facility and on-site cardiologists</th>
<th>Hospital without catheterisation facility</th>
<th>Unadjusted hazard ratio (95% C.I.)</th>
<th>Adjusted hazard ratio(^1) (95% C.I.)</th>
<th>Adjusted hazard ratio(^2) (95% C.I.)</th>
<th>Adjusted hazard ratio(^3) (95% C.I.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died in hospital</td>
<td>6.4%</td>
<td>10.7%(^*)</td>
<td>1.67 (1.10-2.54)</td>
<td>1.45 (0.97-2.17)</td>
<td>1.46 (0.91-2.30)</td>
<td>1.36 (0.81-2.31)</td>
</tr>
<tr>
<td>Died in 6-months</td>
<td>9.6%</td>
<td>19.1%(^**)</td>
<td>2.06 (1.10-3.83)</td>
<td>2.60 (1.29-5.27)</td>
<td>2.91 (1.32-6.44)</td>
<td>3.15 (1.21-7.55)</td>
</tr>
<tr>
<td>Died in 1-year</td>
<td>12.1%</td>
<td>22.1%(^**)</td>
<td>1.90 (1.19-3.04)</td>
<td>2.33 (1.39-3.91)</td>
<td>2.61 (1.45-4.68)</td>
<td>3.02 (1.60-5.71)</td>
</tr>
</tbody>
</table>

\(^*\) P < 0.05  
\(^**\) P < 0.005

\(^1\) Adjusted for patient’s baseline characteristics including age, sex, history of hypertension, diabetes mellitus, history of ischaemic heart disease, history of coronary artery bypass graft, history of stroke, heart rate on presentation, systolic blood pressure on admission, Killip class and cardiac arrest on arrival, maximum troponin elevation, renal impairment and subset of acute coronary syndrome.

\(^2\) Adjusted further for use of evidence-based medicine during 1st 24-hours (aspirin, anticoagulant, beta-blockers, statins).

\(^3\) Adjusted further for angiography & revascularisation.
Table 5. Mortality figures (percentages) for ACS patients at different follow-up periods after being admitted into a tertiary (catheterisation laboratory/cardiologists) vs a community (general physicians) hospital

<table>
<thead>
<tr>
<th></th>
<th>STEMI</th>
<th></th>
<th>NSTEMI</th>
<th></th>
<th>Unstable Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients admitted</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tertiary</td>
<td>281</td>
<td>community</td>
<td>165</td>
<td></td>
<td></td>
</tr>
<tr>
<td>died in hospital</td>
<td>31(11.0%)</td>
<td>20(11.3%)</td>
<td>18(5.3%)</td>
<td>12(11.7%)</td>
<td>5(2.3%)</td>
</tr>
<tr>
<td>died by 6-months</td>
<td>33(13.2%)</td>
<td>33(19.5%)</td>
<td>33(9.9%)</td>
<td>22(21.4%)</td>
<td>10(4.6%)</td>
</tr>
<tr>
<td>died by 1-year</td>
<td>43(15.3%)</td>
<td>37(21.9%)</td>
<td>42(12.6%)</td>
<td>27(25.2%)</td>
<td>18(7.2%)</td>
</tr>
</tbody>
</table>

*P<0.05
**P<0.005
* Base numbers were 281, 169, 333, 103, 218 and 27 respectively
* Base numbers were 281, 169, 334, 103, 218 and 27 respectively

Table 6. Adjusted hazard ratio (95% confidence interval) for patients first admitted into a community hospital vs a tertiary hospital, according to final ACS diagnosis

<table>
<thead>
<tr>
<th></th>
<th>STEMI</th>
<th></th>
<th>NSTEMI</th>
<th></th>
<th>Unstable Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>died in hospital</td>
<td>1.07(0.63- 1.82)</td>
<td></td>
<td>2.22(1.11-4.46)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>died by 6-months</td>
<td>3.06(0.90-10.46)</td>
<td></td>
<td>2.31(0.96-5.57)</td>
<td>1.18(0.14-9.57)</td>
<td></td>
</tr>
<tr>
<td>died by 1-year</td>
<td>1.95(0.83-4.60)</td>
<td></td>
<td>2.38(1.23-4.53)</td>
<td>1.37(0.31-6.14)</td>
<td></td>
</tr>
</tbody>
</table>

† Figure not able to be calculated

Discussion

In this study, patients with ACS admitted into a tertiary hospital (Dunedin Hospital) have significantly lower in-hospital, 6-months, and 1-year mortality (absolute mortality difference of 4.3%, 9.5%, and 10.0% respectively) compared to patients admitted into a community hospital (Invercargill Hospital).

This difference in survival outcome cannot be accounted for by the baseline patient characteristics because our multivariable regression model adjusted for multiple important prognostic factors. These included age and sex; history of hypertension, diabetes mellitus, ischaemic heart disease, coronary artery bypass graft, and stroke; heart rate and systolic blood pressure on admission; Killip class and cardiac arrest on arrival; maximum troponin elevation; renal impairment; and the subset of acute coronary syndrome.
We further adjusted our model for the use of evidence-based medicine in the first 24 hours and found an over two-fold increase in 1-year mortality in patients admitted to a community hospital.

Invercargill Hospital is a community hospital serving the Southland districts without any onsite cardiologist or catheterisation facilities. Referrals for angiography are made to the attending cardiologist in Dunedin Hospital before patient transfer (a 3-hour ambulance journey) can be organised.

After adjusting for the use of angiography and revascularisation, the adjusted hazard ratio for 6-month and 1-year mortality remains significantly higher in patients initially admitted into Invercargill Hospital than patients admitted into Dunedin Hospital. This difference in mortality is particularly marked in the NSTEMI subgroup.

Published New Zealand guidelines on the management of NSTEMI in 2005 recommends the use of an early invasive strategy for patients with NSTEMI (positive troponins) showing high risk features including dynamic ST changes, patients with diabetes, patient with continuous or recurrent ischaemic symptoms at rest or on exertion despite medical therapy and patients with clinical evidence of left ventricular failure.

Selecting high-risk patients who would benefit most from revascularisation is important to maximise the cost-benefit gains from invasive intervention. A meta-analysis of seven major randomised trials on routine versus selective invasive approach for patients with NSTEMACS found a better outcome with patients managed with a routine invasive approach, in those with positive cardiac markers such as troponins and those managed with a more contemporary treatment strategy (studies published after 1999). There was, however, an early hazard with a routine invasive strategy leading to higher in-hospital mortality and non-fatal myocardial infarction.

The benefit of routine revascularisation is seen after discharge, resulting in fewer subsequent deaths or myocardial infarction. Overall, there was a benefit after a mean follow-up period of 17 months from hospital admission (the time of randomisation to trials), including a non-statistically significant 8% relative reduction in death, a significant 18% reduction in combined death and non-fatal myocardial infarction, and a one-third reduction in severe angina and rehospitalisation.

Interestingly, the current study also demonstrated a bigger survival difference at 6 months and 1 year between the two centres at hospital discharge. This may also reflect the delayed benefit from the more frequent use of revascularisation in Dunedin Hospital.

Recently the ICTUS study found routine invasive revascularisation strategy (98% catheterisation rate, 76% rate of in-hospital PCI or CABG) to confer no additional benefit over a selective invasive (53% catheterisation rate, 40% PCI or CABG) strategy in NSTEMI patients. Of note, the Dunedin rate of revascularisation for patients with NSTEMI (71% catheterisation, 58% PCI or CABG) represented a midway between routine and selective revascularisation. In centres with catheterisation facilities in the GRACE registry, the average revascularisation rate (PCI or CABG) is 48%.

The significantly higher mortality in Invercargill patients admitted with NSTEMI (Table 5) is concerning. The in-hospital revascularisation rate was low (26%...
catheterisation, 22.4% PCI or CABG) but similar to the findings from the New Zealand Acute Coronary Syndrome audit performed in 2002 (25% angiography, 11.2% PCI or CABG). However, even after adjusting for transfer and angiography, the hazard ratio (risk of dying) remains increased.

We found that transferred patients from Invercargill Hospital were generally lower-risk ACS patients (younger, clinically stable, and with fewer premorbidities), than those not transferred. These lower-risk patients would be expected to benefit less from revascularisation, at least within the one-year follow-up. However, numerous practical issues requires consideration when transferring high-risk ACS patients, including the need for sufficient medical escorts, the possible reluctance of medical staff in Dunedin Hospital to accept patients with significant comorbidities (renal insufficiency as a contraindication for angiography, severe non-reversible airways disease) and non-medical logistic reasons (bed availability).

The delay in angiography because of the time required for transfer may also have impacted negatively on survival, although the optimal timing for coronary intervention post-NSTEACS is yet to be determined.\textsuperscript{12,13}\textsuperscript{13}

The fact that higher-risk ACS patients (who potentially would derive a greater absolute benefit from angiography) were less likely to receive these procedures is not unique to New Zealand. This had been shown in a large survey of 158,831 elderly Medicare patients with acute myocardial infarction in United States in the mid 1990s.\textsuperscript{14} In the contemporary CRUSADE Registry\textsuperscript{15,16}—17,926 patients from 248 United States hospitals with angiographic facilities—interventions again were more likely to be offered to younger patients, those under cardiology care and those without heart failure, renal dysfunction, or ischaemic ECG changes.

Further analysis of CRUSADE registry found high-risk ACS patients managed conservatively solely with medical therapy had higher in-hospital mortality.\textsuperscript{17} It may also be argued that interventions, even when performed in lower risk patients, will confer prognostic benefit over a longer time period after the first 1 or 2 years.

Interestingly, patients with interventions were more often prescribed evidence-based medicine (Table 3), findings similar to those in the United States.\textsuperscript{14} The use of evidence-based medicine in ACS patients should be encouraged even if intervention/revascularisation were not offered.

**Study limitations**—This is a retrospective registry study. Although a vigorous multivariable logistic regression was used incorporating multiple known prognostic predictors, there were possibly other relevant factors (which we did not record or adjust for) which may account for the discrepancy in mortality observed between the two centres.

As this study included only patients admitted into the CCUs of the two hospitals, issues like bed availability and CCU admission policies might have influenced our results.

The only documented endpoint was all-cause mortality—secondary endpoints (such as myocardial infarction, stroke, major bleeding, recurrent angina, or rehospitalisation) were not studied. Furthermore, we did not study management (use of medications and revascularisation) after hospital discharge which could have affected 6-month and 1-year mortality.
Conclusion

There was a disparity in ACS outcome between community and tertiary hospitals in New Zealand, even after adjustment has been made for baseline characteristics, use of evidence-based medicine on arrival, and transfer for angiography. The use of evidence-based medicine in all ACS patients should be encouraged even if revascularisation was not offered.

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Acknowledgements: Dr EW Tang received support from The Cardiac Society of Australia and New Zealand / MSD Fellowship as well as partial support from the University of Otago Frances G Cotter Scholarship.

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References:


Troponin testing for chest pain in primary healthcare: a survey of its use by general practitioners in New Zealand

Kate Law, Raina Elley, James Tietjens, Stewart Mann

Abstract

Aim Serum troponin is now the preferred biochemical marker for myocardial infarction. The aim of this study was to investigate general practitioner (GP) knowledge and use of serum troponin testing in primary healthcare.

Methods We sent a postal survey about troponin testing to all GPs in the Wellington region (n=299) of New Zealand.

Results Of the 299 surveys sent, 216 replies were received (72%). 54% (n=115) of participants were male and 58% (n=113) in full time practice. 92% were using troponin tests (58% monthly). ECG (79%) and serum troponin (78%) were the tests most commonly used to triage patients with chest pain. GPs had excellent knowledge of false negative scenarios (84% correctly identified false negative if test undertaken within 6 hours) and less knowledge of false positive scenarios (39% answered ‘Don’t know’).

Conclusions The majority of GPs use serum troponin tests, and have sufficient knowledge of the test for use in a primary care setting. Most GPs use the tests appropriately, although a small proportion of doctors may defer rapid admission to hospital while waiting for the test result (7%) or manage the patient within general practice (5%) in those patients who have chest pain considered ‘possibly’ due to myocardial infarction.

Serum troponins are now the preferred biomarker for the routine diagnosis of myocardial infarction. Troponins are a group of molecules that control the myofibril elements of the muscle cells. There are three distinct sub-groups (troponin I, T, and C), and both the I and T subunits are specific to the cardiac myocyte. This study focuses on the use of serum troponin T as this is the isoform commonly used in local laboratories.

Serum troponin tests have been made accessible to general practitioners (GPs) in New Zealand via community laboratories. A recent audit of acute coronary syndrome patients in hospital identified the concern that the availability of the tests could potentially have an adverse effect on the early triage of patients with acute chest pain. It is possible that some GPs might come to rely on troponin test results as a key triage factor. Given the delay in a result becoming positive after initial chest pain (up to 10 hours), some may defer referral of patients who previously might have referred to hospital care immediately.

There are concerns that dependence on such a test result—especially if a delay is involved—may lead to some results being missed in the crucial time period because of changing duty rosters. There are also issues around over-interpretation of borderline results and the occasional false positive (some patients, for example, have a
long-term slight elevation of troponin levels). It is also possible that patients presenting with very atypical symptoms (a few of whom will have an acute coronary syndrome) will now be identified when this would never have happened in the past.

Thresholds for rapid referral of patients to hospital care may vary, especially with geographical factors. For example, rapid transfer of a patient to a hospital facility greater than 50 km away creates different pressures from those in the urban environment.

There are many potential benefits, as well as potential hazards, with using troponin testing in general practice. However, there are currently no guidelines regarding the use of troponin testing in general practice in New Zealand. Therefore, it is important that we know the way in which it is used to determine whether guidelines are needed and to inform the content of the guidelines, if necessary.

The aim of this survey was to investigate general practitioner views, knowledge, and use of troponin testing in primary healthcare. In conjunction with the survey, a linked study, Troponin testing for chest pain in primary healthcare: a New Zealand audit, was also undertaken, which provided data on the actual clinical circumstances and outcomes of troponin tests that were ordered in the greater Wellington region over a 5-week period.5

Methods

Participants—The target population for this study was all general practitioners in the greater Wellington region (Wellington city, Porirua, Kapiti, and Hutt Valley) in New Zealand. A list of GPs was compiled based on a database available within the Department of General Practice at the Wellington School of Medicine. GPs practicing north of Paekakariki were considered ‘rural’. There were initially 341 GPs identified, including 32 ‘rural’ GPs. However after phoning several practices, and receiving letters ‘returned to sender’, 42 GPs were found to be no longer at the practice, or had retired from general practice. This left a total of 299 GPs, 30 of whom were from rural practices.

Survey—A survey to examine individual general practitioner’s knowledge of troponin tests and their policies for use of the test was developed in consultation with several GPs (Appendix 1). The survey was piloted amongst a variety of GPs and adapted accordingly.

The survey attempted to address the following areas:

- How often do GPs use troponin tests?
- Is there any evidence of the inappropriate use of troponin tests which might defer otherwise advisable rapid admission of a patient to coronary care facilities?
- How do GPs usually triage patients with chest pain?
- Is there evidence of sufficient knowledge among GPs to use the test safely and appropriately? (e.g. knowledge of common false negative and false positive scenarios)

The survey was conducted during November and December 2004, with non-responders receiving a telephone call then repeat survey. A wrapped chocolate was attached to each survey to encourage participation. The study was approved by the Wellington Ethics Committee.

Analysis—The data was entered into a spreadsheet using Microsoft Excel software. Response rates for each individual question were calculated, and for each question percentages were calculated for each possible response.

Results

The response rate of the survey was 72% (216/299). Of those who responded, 54% (n=115) of participants were male and 58% (n=113) were working full time as GPs (8/10ths of a full time equivalent [FTE] or more).
Figure 1 shows the frequency with which GPs order troponin tests. This question had a 98% response rate. The greatest proportion of GPs order the test monthly (58%). Almost all GPs use the test in their practice (92% answered yearly or more often) but the frequency of use varies greatly.

**Figure 1. Frequency of troponin tests ordered by GPs**

Table 1 and Figures 2–4 show proportions of GPs selecting each management option for nine different clinical scenarios. Table 1 is based on the table presented in the survey. Several participants also made comments regarding this question. Where the option “order troponin test and wait for the result before referring” was ticked, and the pain was less than 2 hours ago, several respondents commented that they would do bedside troponin dipstick tests or would do serial / repeat test / test in 12 hours time.

Several participants had difficulty with some of the scenarios commenting that management would depend on ECG findings, patient age, clinical history, and time of day. In particular, participants commented that they found the ‘possible’ myocardial infarction scenario difficult to answer, especially because the percentage range was broad (5–50%).

Table 2 lists the tests commonly used by GPs to triage patients with recent chest pain suggestive of myocardial infarction, with the percent of GPs that indicated each test. The most commonly used tests were ECG and troponin T.

Ten percent (21/210) of participants responded that they would not do any tests, and 5% (11/210) qualified this by adding that they would refer the patient immediately. Several participants commented that the test they would order would depend on how recent the ‘recent’ chest pain was.
Table 1. Proportions of GPs selecting each management option for different clinical scenarios

<table>
<thead>
<tr>
<th>SCENARIO: Likelihood that chest pain is due to myocardial infarction on clinical grounds</th>
<th>Order troponin test but refer immediately to hospital</th>
<th>Refer immediately to hospital without ordering troponin test</th>
<th>Order troponin test and wait for result before referring</th>
<th>Manage within general practice without troponin test or referral to hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unlikely (&lt;5% likelihood of myocardial infarction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms ≥ 12 hours ago RR 99%</td>
<td>0%</td>
<td>0%</td>
<td>53%</td>
<td>46%</td>
</tr>
<tr>
<td>≥ &lt;2 hours ago RR 99%</td>
<td>1%</td>
<td>1%</td>
<td>64%</td>
<td>33%</td>
</tr>
<tr>
<td>Possible (5–50% likelihood of myocardial infarction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms ≥ 12 hours ago RR 99%</td>
<td>4%</td>
<td>15%</td>
<td>78%</td>
<td>2%</td>
</tr>
<tr>
<td>≥ &lt;2 hours ago RR 99%</td>
<td>10%</td>
<td>36%</td>
<td>53%</td>
<td>1%</td>
</tr>
<tr>
<td>Probable (50–100% likelihood of myocardial infarction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms ≥ 12 hours ago RR 99%</td>
<td>16%</td>
<td>51%</td>
<td>32%</td>
<td>1%</td>
</tr>
<tr>
<td>≥ &lt;2 hours ago RR 100%</td>
<td>15%</td>
<td>76%</td>
<td>2%</td>
<td>0%</td>
</tr>
</tbody>
</table>

*The highest percentage for each scenario is indicated in bold type, and response rates are given beside each scenario. RR=response rate of each scenario.

Figure 2. Management options for chest pain thought unlikely to be myocardial infarction (MI) (<5% likelihood of MI)

Figure 3. Management options for chest pain thought possibly due to myocardial infarction (5–50% likelihood of MI)
Figure 4. Management options for chest pain thought probably due to myocardial infarction (50–100% likelihood of MI)
Table 2. Tests performed in general practice on patients with recent chest pain

<table>
<thead>
<tr>
<th>Type of Test</th>
<th>%</th>
<th>(n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG</td>
<td>79%</td>
<td>165/210</td>
</tr>
<tr>
<td>ProBNP</td>
<td>2%</td>
<td>5/210</td>
</tr>
<tr>
<td>CKMB</td>
<td>24%</td>
<td>50/210</td>
</tr>
<tr>
<td>Troponin T</td>
<td>78%</td>
<td>163/210</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>0%</td>
<td>1/210</td>
</tr>
<tr>
<td>Lipids</td>
<td>3%</td>
<td>6/210</td>
</tr>
<tr>
<td>ESR</td>
<td>0%</td>
<td>1/210</td>
</tr>
<tr>
<td>FBC</td>
<td>3%</td>
<td>6/210</td>
</tr>
<tr>
<td>CRP</td>
<td>1%</td>
<td>2/210</td>
</tr>
<tr>
<td>Renal Function</td>
<td>2%</td>
<td>4/210</td>
</tr>
<tr>
<td>TSH</td>
<td>1%</td>
<td>2/210</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>1%</td>
<td>3/210</td>
</tr>
<tr>
<td>Glucose</td>
<td>2%</td>
<td>4/210</td>
</tr>
<tr>
<td>O2 Saturations</td>
<td>9%</td>
<td>1/210</td>
</tr>
</tbody>
</table>

Figure 5 shows the proportion of GPs who chose different causes of false negative troponin test results in the presence of a recent myocardial infarction. Most GPs identified the correct options.

Figure 5. Percentage of doctors identifying scenarios as common causes of false negative troponin results
Figure 6 shows the proportion of GPs who selected different causes of false positive troponin test results. Thirty-nine percent of GPs answered “Don’t know” to this question. Very few GPs chose the incorrect options: “Asthma”, “Peptic Ulcer”, or “Cellulitis”. Correct options have light shaded bars and incorrect options have darkly shaded bars.

The question: “As a GP in a more remote setting, without rapid access to hospital care, do you have any further comments to make about the use of troponin testing?” was sent to GPs in ‘rural areas’, of whom 73% (22/30) replied to the questionnaire. Of those that replied 68% (15/22) wrote comments in response to the question. The comments listed below are a sample of those collected, and represent the main themes expressed:

- *Has been a useful adjunct particularly in late presenting chest pain or atypical chest pain*
- *We use on site [instant] trop T testing with follow-up testing by lab*
- *It [troponin dipstick test] is not very reliable—had false negatives and then the lab test comes through a few hours later positive*
- *The ambulance takes anything from 5–30 minutes to arrive*
- *Troponin testing is a useful aid in the diagnosis of myocardial injury. It does not however, serve as the sole determinant of our decision-making in acute coronary syndromes. The history, ECG, examination and other comorbidities are equally important*

**Discussion**

This study has shown that GPs have a good knowledge of serum troponin testing, and most use the test appropriately in their practice. It appears that most GPs order a serum troponin T test about once per month (58%). There was a large amount of variation in the frequency with which GPs ordered the test. The variation in frequency
could be due to differing populations, but could also be an indication of different understanding about appropriate usage of the test. Overuse of the test may have economic implications, and underuse may compromise detection of acute coronary syndromes.

The survey aimed to find out whether there was any evidence of inappropriate use of serum troponin tests which might defer otherwise advisable rapid admission of a patient to coronary care facilities. This was achieved using the table format provided in Table 1. Several participants had difficulty answering the question, as an estimation of the level of risk is a multi-variable problem based on medical history, physical examination, and ECG. The layout of the questionnaire aimed to keep the scenarios as simple as possible and thus not include specific information about ECG and other findings.

The simplicity of the scenarios resulted in a few participants answering, “it depends on other factors”, rather than indicating a specific management option. It is also worth noting that not all GPs have an ECG available in their practice. The wide range of 5–50% likelihood also limited interpretation of appropriateness of actions taken in this group of patients.

Given the scenario of chest pain unlikely to be due to myocardial infarction (less than 5% likelihood) the majority of GPs replied they would order the test and wait for the result or manage within general practice without using a troponin test. Those that said they would order the test and wait when symptoms were less than 2 hours ago, often indicated that they would do a repeat test at 12 hours. Therefore, these seem to be appropriate management options.

In the “possible myocardial infarction” scenario (5–50% likelihood), few GPs said they would manage the patient without the use of troponin in their practice. The majority would order the test and wait if the pain was more than 12 hours ago, or refer immediately to hospital if the pain was less than two hours ago; for example 86%, [95%CI: 80.9%–90.4%, n=179] would refer immediately with or without ordering a troponin test first. Again, these would seem to be appropriate management options.

It is hard to judge the appropriateness of the 10% (n=20) of participants who stated they would order the test and wait for the result for pain less than 2 hours ago instead of admitting the person, because the range of 5–50% likelihood included relatively low and medium likelihood of MI.

Of those who responded that they would ‘order the test and wait’ in this situation, five came from rural practices where instant dipstick troponin testing may have been used. In this case, the test result may well be available before arrival of an ambulance so the management would be appropriate. Even excluding these respondents, a significant number of GPs selected possibly inappropriate options, 7% [95%CI: 3.7%–10.9%; n=15] replied “order and wait”, and 5% [95%CI: 1.9%–7.7%; n=10] replied “manage within general practice”. However, the range of 5–50% was again too wide to conclude that this was definitely inappropriate.

Finally, in the “probable myocardial infarction” scenario (50–100% likelihood), very few said they would manage the patient within their practice. The majority would refer the patient to hospital immediately (with or without ordering a troponin test first).
When tests are used to evaluate chest pain in primary healthcare, the most common tests are ECG and troponin T (79% and 78% respectively). It is worth noting that 21% of participants would not do ECG, possibly because they do not have one available to them within their practice. These doctors must rely more heavily on biochemical markers such as troponin T.

Creatinine Kinase MB Isoenzyme (CKMB), the previous preferred biochemical marker for myocardial infarction, was only used by 24% of participants, thus suggesting most GPs are using the markers appropriately (CKMB is useful for very recent chest pain as it rises before troponin). The fact that troponin tests are more sensitive and specific for MI than ECGs, and often easier to take, may mean that GPs sometimes feel that they do not need to do both.

Interpretation of troponin tests is not, however, without problems. Despite high levels of sensitivity and specificity, there are still false negative and false positive results. For example, acute pericarditis, acute pulmonary embolism, acute or severe heart failure, myocarditis, sepsis and/or shock, and renal failure are commonly associated with serum troponin elevation in the absence of acute myocardial infarction.\(^6\)

Timing of the test in the course of an evolving acute coronary syndrome is crucial to its usefulness. Troponins may take 6–9 hours to rise following an acute coronary event. The Joint ESC/ACC Committee (2000) consensus statement gives the following recommendation “For most patients blood should be obtained for testing on hospital admission, at 6 to 9 hours, and again at 12 to 24 hours if the clinical index of suspicion is high” (p962).\(^2\)

There was some evidence that tests were sometimes undertaken prematurely in primary care, although this would be appropriate if a follow-up test was done.

The current survey included two knowledge-based questions regarding false positives and false negatives. Knowledge regarding false negatives was excellent; with most GPs correctly answering that false negative troponin results can occur if the test is performed within 6 hours of the acute event (84% correctly answered) or 2 weeks after the event (72% correct).

Troponin usually rises 6–10 hours after the acute event, and stays elevated for about 10 days. However, GPs had much less knowledge about false negatives. The correct answers, according to Roongsritong et al (2004), were acute heart failure (25% correct), acute pulmonary embolism (25%), and renal failure (39%).\(^7\)

A large proportion (39%) responded “Don’t know”. As one respondent commented, \([I\] don’t know but [I’m] not too concerned as doesn’t change immediate management in general practice.\) This is a valid point, as most GPs use troponin to ‘rule out’ myocardial infarction rather that ‘rule in’ other diseases. Therefore, it would seem that knowledge of false negatives is sufficient for practice. That is, all positive results, whether false positives or not, are likely to need immediate referral to a hospital setting.

Finally, the survey aimed to gain some information about the views of GPs practicing in a more rural setting. It seemed that rural GPs found troponin a very useful adjunct to diagnosis. The major difference seemed to be the use of ‘instant’ troponin dipstick test, which eliminates the need to wait for the result before referral to hospital care.
For many rural GPs, the dipstick troponin result can be available before an ambulance arrives at the practice, hence the “order the test and wait for result” scenario does not apply to this population. However, several comments were made that the results were not very reliable, and a sample must be sent to the lab for analysis for every dipstick test performed.

A strength of this study is the good response rate of 72%. This was achieved by keeping the questionnaire short and simple to encourage participation, and enlisting the help of practice managers to encourage responses. The attached chocolate proved very successful with many expressions of thanks returned with completed surveys.

Possible reasons for non-response include close proximity to Christmas, high workload of GPs, and overload of mail. The high response rate and large sample size means the study has a high level of generalisability. Another strength of this study is the fact that this is the first survey conducted in the area of serum troponin testing in primary care internationally, and hence provides new information.

A limitation of the study is the fact that the rural sample was small (30 identified, 22 respondents), and the distance from hospital was not as great as other more typically ‘rural’ areas of New Zealand. A similar survey and audit conducted in a rural area would be useful in exploring the ways in which troponin tests are used in more remote settings.

The other limitation is the fact that some recipients found the questions problematic, especially question number 3, where “Possible MI” had a likelihood range from 5–50%. The likelihood ranges were the subject of much discussion in the development of the questionnaire but those chosen were felt by GP advisors to be the most likely to match different potential management strategies. Despite the comments, response rates were sufficient for all questions.

Results from this study concur with findings in the linked audit of all troponin tests ordered over a 5-week period in the same region. This audit found that the perceived frequency of use of troponin tests was accurately reflected in practice. However, the audit showed a much lower rate of ECG recording than the survey suggested.

This survey has shown that GPs are using serum troponin testing in their practice, and in most cases, are using it appropriately despite the absence of guidelines. A study by Centor et al (2003) found increasing and appropriate use of troponin testing before guideline release in a population of secondary care doctors; they suggested that guidelines may in fact codify current practice, rather than always disseminate new knowledge.7 Even so, these results provide the first information about use of troponin testing in primary care, which may be useful in the development of future guidelines.

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References:


Troponin testing for chest pain in primary healthcare: a New Zealand audit

Stewart Mann, James Tietjens, Kate Law, Raina Elley

Abstract

**Aim** Serum troponin is a widely used biomarker for the diagnosis of myocardial infarction (MI). The aim of this audit was to document the actual clinical circumstances when serum troponin tests are used to assess chest pain in primary healthcare.

**Methods** We undertook an audit of general practitioner (GP) serum troponin requests made to community laboratories in the Wellington region over a 5-week period in 2004.

**Results** 433 tests were ordered by 201 GPs and 10 tests were positive. We faxed 396 questionnaires to identifiable GPs requesting the tests and received 292 replies (74%). The time between initial onset of symptoms and troponin testing was biphasically distributed with peaks at 7–12 hours and 3.5 days. An ECG was performed in less than 50% of the cases. The GP’s estimate of the likelihood of their patient’s symptoms being due to MI was strongly influenced by both positive and negative test results. Patients were referred acutely to hospital on less than 5% of occasions.

**Conclusions** GPs used troponin testing mostly for late presentations some days after chest pain, to ‘rule-out’ MI. When used acutely, referral for admission occasionally awaited the troponin test result.

Troponin I and troponin T are proteins specific to myocardium that are released into the bloodstream with even minimal myocardial damage. These proteins are now detectable with a high degree of precision by various assays that are widely used as markers of myocardial infarction (MI) and indeed are key components of its current definition. The tests have been available from community medical laboratories in New Zealand for several years and general practitioners (GPs) are making increasing use of them to help triage patients presenting with chest pain, the majority of whom have not had MI.

While the sensitivity and specificity of the tests are both very high, there are a few causes of false negative and false positive tests. The most clinically relevant false negative situation is the period of up to 10 hours from the onset of chest pain when, even with later proven MI, the test may be negative. Ideal management of MI usually includes expedited transfer of the patient to a monitored environment such as a Coronary Care Unit where serious complications such as ventricular fibrillation can be dealt with, the peak incidence of this decaying rapidly and exponentially from the moment of chest pain.

While troponin tests allow a much greater precision in the diagnosis of myocardial damage, the delays in obtaining a result and the test becoming positive can create practical problems in management of patients in the community; those who might
otherwise have been referred to hospital immediately may be deferred pending a valid troponin result.

This has created problems for laboratory staff, for example, when a test proves positive and the referrer is not contactable (Drs Michael Crook and John McCafferty, personal communications, 2004). In a recent study of patients eventually admitted to hospital with MI, awaiting a troponin result was cited several times as a reason for delay in referral.4

We wished to explore the use made of troponin tests by GPs and accordingly conducted a postal survey,5 and an audit of tests requested through community laboratories over a month in late 2004. Specifically, the aim of this audit was to document the actual clinical circumstances and medical outcomes when serum troponin tests are used to assess chest pain in primary healthcare.

Methods

We enlisted the help of the two community laboratories in the greater Wellington region that perform the vast majority of biochemical tests for GPs, including troponin T estimations. One laboratory had registered 2570 troponin T tests over a recent 12-month period, 178 (6.9%) of which were positive. A prospective audit was planned of all tests requested of both laboratories over a 5-week period (15/11/2004 through 19/12/2004) with a single page explanation and questionnaire faxed to the requesting GP within 3 days of the test requesting a reply also by facsimile. If no response was received within 2 weeks, telephone contact was made with the practice and, if necessary, a further copy of the questionnaire sent.

In the questionnaire (see Appendix 1), enquiry was made of the duration of patient’s chest pain prior to the test, the likelihood in the GP’s mind of MI both before and after learning the results of the test, whether or not an ECG was done, and what the outcome for the patient was. Consultation was undertaken with several GPs in the development of the questionnaire and the Wellington Ethics Committee approved the study. All individual information identifying the patient and GP was anonymised after compilation although, where relevant, additional information was sought from admitting hospital records.

Results

Test numbers, results, and response rate—Of the 433 troponin T tests performed in the study period, 37 could not be tracked to a contactable source. Thus, 396 questionnaires were sent—but, in 14 cases, we found that the GP had not requested the troponin test specifically (it had been added on by the laboratory to a request made for creatine kinase for non-cardiac reasons). Of the remaining 382, a completed reply was received to 278 questionnaires (73%) on which the subsequent analysis is based.

Of the 201 GPs identified as requesting troponin T tests, 51% requested only one test in the audit period, whereas 7% requested five or more tests. Twelve tests were repeated within 24 hours and 3 patients had second tests more than one week after the first. Eight tests were ordered from Wellington or Hutt Valley After-Hours Centres.

Ten of the 433 (2.3%) tests were positive. Audit returns were received on eight of these patients, four of whom were referred acutely to hospital (as were the two other patients with positive tests with no audit returns).

Time from onset of chest pain to troponin testing—The distribution of times between the onset of pain and taking of the blood test for each patient was given in 245 (88%) of the responses and is shown in Figure 1. There is clearly a bimodal distribution with an early peak at 7–12 hours and a larger later one at 2–7 days.
Twenty-nine (12%) of negative tests were performed less than 10 hours after the onset of chest pain when false negative results might have been obtained and in only 3 of these cases was a repeat test performed in the community and one other after referral to hospital. 122 tests (50%) followed the chest pain by more than 24 hours when immediacy of action was unlikely to have been an issue. Two of the five positive tests recorded were in the 7–12 hour category, one in the 13–18 hour group and two in the 2–7 day window.

**Figure 1. Time delays between onset of symptoms and performance of the troponin test**

**Clinicians’ estimates of likelihood of myocardial infarction (MI)—** An answer was given to this question in 98% of responses. For those with an eventual negative test result (Figure 2A), pre-test probability of MI was indeed thought to be low (less than 25% likelihood in 86% of cases) and was reduced markedly by the negative result (less than 1% likelihood in 84% of cases post-test). However, in 20 cases, some residual likelihood of an infarct was held after a negative test result despite this being done in an appropriate timeframe, 12–168 hours after pain (14 reported 1–10% likelihood, 4 reported 10–25%, 1 reported 25-50%, and 1 reported 50–75%).

In the seven responses where the test had proved positive, pre-test likelihood (Figure 2B) was quite variable but the positive result did not convince GPs of a definite acute MI in 2 cases, one of these patients being known to have a chronically elevated level.

**Other tests—** We asked whether an ECG was performed at the same visit when troponin T was requested; 99% responded indicating that this had been done on 42%
of occasions, with a similar proportion (three of eight) for those with a positive test result.

**Outcomes**—According to audit returns, 7 patients were referred immediately to hospital before the test result was known (one of whom turned out to have a positive result). Three other patients with positive results were referred to hospital when the result was known; one of these having been tested 9 hours following symptoms and consequently incurred a further 5-hour delay before admission, but the other 2 being late tests, 3 days following symptoms. Three others were referred for admission following the result even though the troponin was negative.

Of the 10 patients with *positive* tests (including 2 patients for whom there were no audit returns), 1 was untraced but 5 were admitted to hospital, although 1 with a borderline result was discharged after overnight observation. Two were deemed unsuitable for acute hospital admission, residing in sheltered accommodation with severe comorbidities and 2 were referred for urgent outpatient assessment.

Of the 9 patients with *negative* tests sent to hospital, no information was traceable on 4, 2 were thought to have had non-cardiac chest pain, 1 pneumonia, 1 syncope following nitrate use and 1 arrhythmia.

197 (76%) of the remainder were thought by their general practitioner to have suffered non-cardiac symptoms and 22 (8%) known stable angina. Twenty-three (9%) were referred for further cardiac investigation as outpatients, the remainder having miscellaneous diagnoses and management.
Figure 2A. GPs’ estimates of the likelihood of each patient having suffered a myocardial infarction before and after negative troponin test results known.
Discussion

This audit was conducted in conjunction with a questionnaire-based survey of local general practitioners’ knowledge and use of troponin testing. Both studies had a sufficiently high response rate to indicate likely valid results. We are not aware of any similar survey or audit on the use of troponin tests in primary care having been reported previously.
The survey indicated that general practitioners had generally sufficient knowledge and an appropriate approach to the use of tests and interpretation of results. The audit revealed that use of the test can be dichotomised into acute and delayed testing according to the time lapse since the onset of symptoms suggestive of a possible myocardial infarction.

The test was most commonly used as a “rule-out” option performed in patients with a low probability of infarction. This was probably why ECGs were only performed in 42% of cases. Given the persistence of a positive troponin test result for some days after myocardial damage, testing in primary care at 3–7 days following symptoms appears useful, more easily accomplished than an ECG, highly sensitive, and therefore appropriate. In the absence of any strong clinical indications, acute hospital management is not indicated and a delay in obtaining the results is not critical to management of the patient.

The use of troponin testing in the initial 12 hours following the onset of symptoms raises some potential issues. It has been cited by patients in another survey4 as a reason behind delay in presentation to hospital with an infarction. However, use in patients with a low probability of acute infarction might reveal occasional positive results that would not otherwise have been recognised.

Indeed, in this audit, 3 patients with less than 10% clinical likelihood moved to ‘confirmed infarction’ with a positive result. When a test done less than 10 hours from symptom onset is negative, this does not provide confident reassurance about the absence of infarction and it is concerning that very few patients with negative tests within this timeframe underwent further testing. It is possible that some may have been falsely reassured.

Since only one patient in this group appeared to have their acute admission potentially delayed by waiting for a troponin test result, it might appear that this is a minor problem although the previous survey (of 100 later confirmed infarctions) found this was a more common occurrence.

This audit did not indicate an immediate need for comprehensive guidelines on the use of troponin tests in the primary care setting. However, there were too few positive test results, or other patients with a high likelihood of acute infarction, to conclude that the management of ‘true’ MI was never adversely affected by ordering the test and waiting for results.

It may be advantageous to remind clinicians that the test should not be used to determine the need for acute referral in ‘high likelihood’ cases, or for reassurance of the patient in ‘low likelihood’ cases without repeat samples taken, when the onset of symptoms was less than 10 hours before testing.

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Medical Laboratory Wellington; and John McCafferty and staff of Valley Diagnostic Laboratory.

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Recurrent muscle infarction (with involvement of the arm) in a diabetes patient

Mohammed Alansari, David Hamilton, Kim Wong

Spontaneous muscle infarction is an uncommon complication in diabetic patients. It occurs usually in advanced, poorly controlled, type 1 and 2 diabetes mellitus, usually with a high prevalence of micro- and macrovascular complications.

The first case report was in 1965,¹ and up to 1999, 53 cases have been reported in the English medical literature.²⁻⁴ Thereafter, with increasing awareness of the condition, there have been numerous reports, the largest of which included 21 cases.⁵ Because of the rarity of the condition, the aetiology and management has been difficult to determine.

The following case report describes a patient (with type 2 diabetes and on chronic intermittent haemodialysis for the management of diabetic nephropathy) who suffered two separate episodes of muscle infarction.

Case report

A 47-year-old Māori gentleman was admitted with a history of progressive painful swelling on the lateral side of his left thigh for several days in the absence of any trauma.

He had insulin-dependent type 2 diabetes (diagnosed at the age of 27 years), complicated by neuropathy, autonomic neuropathy, gastroenteropathy, retinopathy, and nephropathy. He commenced continuous ambulatory peritoneal dialysis in 1999, which was converted to chronic intermittent haemodialysis 3 years later. He also suffered from hypertension, secondary hyperparathyroidism (parathyroid hormone [PTH] 200 pmol/L), and a benign paraproteinaemia.

On examination, he had marked swelling of the lateral aspect of his left thigh with localised tenderness. Blood cultures were negative; his haemoglobin level was 116g/L and white cell count 4.7 × 10⁹ with 72% neutrophils. His C-reactive protein (CRP) was 34 mg/L, erythrocyte sedimentation rate (ESR) 33 mm/1 hr, and creatine kinase 88 u/L.

Doppler ultrasound excluded deep vein thrombosis (DVT), but showed abnormal heterogeneity of the muscles of the thigh and two pockets of fluid. A magnetic resonance imaging (MRI) scan revealed localised muscle oedema, inflammation, and haemorrhage involving vastus lateralis. This was relatively central to the muscles; there was no muscular tear but extensive oedema throughout the lower thigh.

Following contrast-enhanced studies, there was reduced muscle enhancement with a possible area of vascular necrosis in an area 1.4 cm wide. The changes were compatible with muscle necrosis (Figure 1).
Figure 1. MRI of the left thigh

A: Sagittal T2 weighted MRI showing necrosis in vastus lateralis—B: Axial, T2-weighted, showing enlargement of vastus lateralis and subcutaneous oedema—C: Axial, T1-weighted MRI, showed mixture of hypoattenuation; infarcted muscle indicated by black arrow and hyperintense areas (haemorrhage) indicated by white arrow

Figure 2. MRI of the left arm

A: Sagittal T2-weighted MRI showing necrosis and oedema of the lateral head of triceps muscle—B+C: Axial T2-weighted MRI showing enlargement of triceps’s lateral head with some vascular compromise and extensive soft tissue oedema—D: T1-weighted MRI showing mixture of hypo and hyperintense signals indicating mixture of necrosis and haemorrhage—E: Post-contrast MRI showing the extensive soft tissue oedema.
Three years previously he had presented with a 4-day history of left arm pain with the posterior distal upper arm being swollen and tender, with no overlying erythema. He was afebrile with a normal white cell count.

A MRI scan showed a large area of abnormal signals of the distal triceps muscle, associated with extensive soft tissue oedema—and following contrast, there was a small amount of vascular compromise within the muscle but no evidence of abscess cavity or any localised fluid collection (Figure 2).

Doppler ultrasound showed no evidence of venous thrombosis or arterial insufficiency. He was treated conservatively with analgesics and the pain and swelling settled in a few weeks.

Discussion

The present case presents multiple characteristics of diabetic muscle infarction. The mean age of this condition from the literature is 41–46 years. Most of the reported cases have been diabetic for more than 15 years. Type 1 diabetes patients with this condition outnumber those with type 2 diabetes. In addition, multiple diabetes complications are common in this group with nephropathy affected 70–90% and 20% on dialysis. The average duration of renal replacement therapy was 25 months before the first event.

The patient with diabetic muscle infarction characteristically presents with acute or subacute onset of pain, tenderness, and swelling of muscle groups in the extremities, more commonly proximal rather than distal, with a predilection for thigh and buttock rather than calf muscles.

Rarely it can affect the muscles of the forearm, psoas, and there are recent reports of involvement of the muscles of the abdominal and thoracic wall, small muscles like peroneus brevis. This is the first report of the triceps muscle being involved. There is no racial predilection: reports include Caucasian, Hispanic, Māori, and Asian.

There are no specific markers for diabetic muscle infarction. Fever is not common. Creatine kinase and white blood cell count are not usually raised. ESR and CRP are raised in 60% of reports.

The average time for seeking medical advice was 4 weeks in one report. The diagnosis is best made by MRI scan (although ultrasound and plain X-ray images may exclude alternative diagnoses). The affected muscle shows increased intensity compared with unaffected muscles and fat associated with intense oedema of the surrounding tissue. MRI scans in the recovery period show no hyperintense signals in T2-weighted scans but some atrophic muscle changes in the affected area with fatty infiltration.

The differential diagnosis includes deep vein thrombosis, soft tissues abscess, necrotising fasciitis, pyomyositis; and other causes of myositis, muscle rupture, haematoma, muscle lymphoma, and osteomyelitis. The combination of MRI findings and the clinical findings in a diabetic patient should give the diagnosis without the necessity to undertake muscle biopsy. The usual pathological features are those of haemorrhagic necrosis of the muscles fibres,
oedema, neutrophilic infiltrate, and surrounding granulation tissue. In chronic lesions, there will be fibrosis and mononuclear infiltrate.

In many reports there were microvascular changes (including hyaline thickening of the walls of the capillaries, arterioles, and small arteries in association with luminal narrowing as well as occlusion of the small vessels with fibrin thrombi.\textsuperscript{1,12}

The pathogenesis of diabetic muscle infarction remains uncertain—three theories exist:

- Firstly, that the aetiology is secondary to macrovascular disease in the light of arteriographic changes of medium and large vessels supplying the involved muscles as well as the post-mortem findings of extensive arteriosclerosis obliterans without an embolic source.
- Secondly, a microvascular cause has been postulated. Muscle biopsies have shown conversion of normal rich collateral circulation of muscle to end-vessel circulatory pattern, rendering it vulnerable to injury. In this abnormal setting, tissue oedema and swelling may lead to intracompartmental ischaemia and myonecrosis.\textsuperscript{12} Furthermore, some reports suggest that this hypoxic environment followed by re-perfusion injury with free radicle production leads to direct myotoxicity.\textsuperscript{13}
- A third theory states that the acquired hypercoagulability in association with endothelial damage leads to muscle infarction.\textsuperscript{2,4,14} An acquired antiphospholipid syndrome may be a contributory factor in the progression of diabetic complications acting as a link between immunological and haemostatic system in the pathogenesis of diabetic microangiopathy.

Because of the nature of the problem and lack of clinical trials, it has been difficult to reach a conclusion as to the optimum treatment. Conservative treatment has been proposed management in most reports. A worse prognosis has been reported following muscle biopsy.\textsuperscript{12} Analgesics remain the mainstay. Anticoagulation has been advocated, with one report observing non recurrence,\textsuperscript{14} while another one did.\textsuperscript{15}

The short-term prognosis is usually good with pain and swelling resolving in a few weeks with regain of muscle function. Recurrence has been reported in about 50% of cases, involving the original or other muscle. Generally, the overall prognosis is poor because the patient usually has multiple diabetic complications at the time of presentation.

Our patient was treated conservatively and achieved full functional recovery after 6 weeks. To our knowledge, he is the first reported diabetes case whose upper arm was involved in muscle infarction.

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Quality improvement in healthcare in New Zealand. Part 2: are our patients safe—and what are we doing about it?

Alan Merry, Mary Seddon, on behalf of EPIQ*

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Abstract

The evidence is incontrovertible—we are inadvertently harming an unacceptable number of our patients by the very healthcare intended to help them. Most developed countries have responded to this evidence with substantial funding for dedicated patient-safety campaigns. New Zealand has a reasonable legislative foundation in relation to this problem but to date has not galvanised action at either the national or the organisational level.

The reasons for this inaction are explored in this article and include a lack of understanding of the causes of medical error and of the difference between error and violation. Insistence on randomised controlled trial evidence and a business model is to misunderstand the constructs at stake and may inhibit the implementation of urgently needed safety strategies that are clearly sensible and worthwhile.

In the first of this series of articles, the question was asked, What would a high quality healthcare system look like? In answering it, patient safety was identified as one of the most important dimensions of quality. Why? Because approximately 10% of admissions to acute-care hospitals in the developed world are associated with an adverse event, and around 1.5% are associated with permanent disability or death (Table).

Table. Adverse event rates in different countries

<table>
<thead>
<tr>
<th>Country in which the study was done</th>
<th>No of records studied</th>
<th>Adverse events % of admissions</th>
<th>Permanent harm and death % of admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>14,179</td>
<td>15.6</td>
<td>2.3</td>
</tr>
<tr>
<td>America</td>
<td>30,195</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>America</td>
<td>14,565</td>
<td>10.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Canada</td>
<td>3,745</td>
<td>7.5</td>
<td>1.6</td>
</tr>
<tr>
<td>Denmark</td>
<td>1,097</td>
<td>9.0</td>
<td>0.4</td>
</tr>
<tr>
<td>England</td>
<td>1,014</td>
<td>11.7</td>
<td>1.5</td>
</tr>
<tr>
<td>New Zealand</td>
<td>6,579</td>
<td>12.9</td>
<td>1.9</td>
</tr>
</tbody>
</table>

This startling fact comes from several studies conducted in different countries over the last 15 years. The most recent of these studies was conducted in New Zealand, and
showed, not surprisingly, that the extent of the problem here is much the same as it is everywhere else in the developed world.¹

The number of deaths attributable to this so-called “iatrogenic” harm in the USA exceeds the road toll² and equates to three jumbo jets full of passengers crashing every 2 days.³

Despite the fact that the vast majority of these iatrogenic deaths are in elderly and/or very sick patients, and it could be argued that even in the absence of an adverse event few would have been alive and well 3 months later,⁴ there is general agreement that preventable harm to patients caused by the very healthcare that was intended to help them is a substantial public health problem.

What has been the response to evidence of iatrogenic harm?

The international community was galvanised by the U.S. Institute of Medicine’s 2000 publication To Err is Human: Building a Safer Healthcare System² which made the extent of harm obvious to the public and politicians. Internationally this has been met with initial scepticism from doctors. However, Governments in Australia (Australian Patient Safety Foundation)¹¹ and the United States (Joint Commission International Center for Patient Safety)¹² and the United Kingdom (National Patient Safety Agency)¹³ have taken these data seriously, and have responded by funding organisational entities for Patient Safety.

In New Zealand, there is now a reasonable level of awareness about the issue of iatrogenic harm at a National or Governmental level. The Health and Disability Services (Safety) Act 2001¹⁴ makes explicit the responsibility to “promote the safe provision of health and disability services to the public.”

The establishment of the office of the Health and Disability Commissioner (HDC) was a response to harm done to New Zealand women by healthcare, identified in the Cartwright Report.¹⁵ The Commissioner has unusually wide ranging powers that allow him to enquire as to the contribution to an adverse event by anyone responsible for the provision of healthcare, including administrators. This has facilitated a world-leading focus on addressing aspects of the system, which contribute to patient harm rather than only seeking to identify individual scapegoats when things go wrong.

Recent changes to Accident Compensation Corporation (ACC) legislation, have also moved in this direction by removing the requirement for patients to demonstrate so-called “Medical Error” (in effect, negligence) in order to obtain compensation for harm from avoidable adverse events. This was accompanied by the establishment of a dedicated patient safety function within ACC.

Unfortunately we still have a long way to go in translating these high-level initiatives into practical gains at the organisational or facility level of the system.

What are the challenges to improving patient safety?

The first challenge is to promote understanding of the causes of error, and to be clear about the distinction between violations and error,¹⁶ because it is only with this knowledge that one can start finding effective solutions to the problem of iatrogenic harm. Making errors is part of the human condition. The propensity to err is integral to such attributes as creativity, distractibility and the facility to undertake multiple
tasks simultaneously which have contributed to the evolutionary success of our species. These attributes also underlie our ability to undertake tasks (often in teams) whose complexity and difficulty is at times astonishing. This is often the case in healthcare, and as the complexity of an activity increases so does the propensity for error.

Errors involve people trying to do the right thing but actually doing the wrong thing. Errors cannot be decreased by exhorting staff to try harder (most are trying hard already) or by punishing those at the ‘sharp end’ of the error chain. Violations on the other hand have an element of choice, they are deliberate (e.g. choosing not to wear a seatbelt or not to wash one’s hands between patients) and they can be decreased by appropriate deterrence.

To improve patient safety there needs to be an increased awareness of the difference between these two. On one hand, responding to error as though it were deliberate violation does not lead to lasting change, and indeed it is likely to lead to a culture of fear where errors and near-misses go unreported. Blaming and removing the person who made the error does not make the situation any safer for the next patient. On the other hand, all involved with healthcare, from the minister, through senior consultants and chief executive officers, to the newest trainee nurse, need to understand fully their responsibility for deliberate choices that have the potential to impact on safety.

To reduce harm to patients from error we will need to accept human fallibility, and concentrate on improving the design of the healthcare system. At present, the system is highly complex, poorly coordinated and very prone to error. What is needed are changes that reduce the chance of error, and the propensity for any errors which do occur to cause harm. Prompt and open disclosure is key to the second objective—if an error is identified immediately, its consequences can often be limited. These are the changes that high-reliability organisations (e.g. nuclear power stations) have built into their culture.

In response to the repeated error of injecting vincristine into the intrathecal space (where it is neurotoxic and usually fatal), James Reason said:

*When a similar set of conditions repeatedly provokes the same kind of error in different people, it is clear that we are dealing with an error prone situation rather than with error prone, careless, or incompetent individuals.*

Once we accept this point, the focus of the patient safety improvement programme switches from the individual to the process of care and it is only then that it is likely to be effective.

The second major challenge in the pursuit of safety lies in having to justify the cost of safety initiatives in a sector which struggles daily with substantial deficits in funding. It is often impossible to quantify precisely the reduction in risk likely to be achieved by any given intervention. Furthermore, in New Zealand, unlike many other countries, the fear of litigation is not a major financial incentive for an organisation to invest in safety.

The almost universal adoption of pulse oximetry by anaesthetists in the developed world illustrates these points. The use of this technology has been associated with a dramatic reduction in the number of patients killed or brain damaged by circuit disconnections or other causes of hypoxia in anaesthesia. However evidence for this is
only emerging now, years after the adoption of this technology. It would not have been available when business cases were needed.

Although the downstream costs of hypoxic brain damage can run to millions of dollars, these costs seldom fall on the hospital which has to fund the technology. Instead they are picked up by the community in general and by rehabilitation agencies in particular. To add to the irony of perverse financial incentives, it may often be cheaper to kill than to seriously harm a patient. Nevertheless it is highly likely that the overall financial impact to society of the introduction of this technology has been a net saving and few anaesthetists would doubt that there has been a substantial reduction in the human cost of harm attributable to anaesthesia.

A third barrier to investing in patient safety has been the misuse of the concept of grading the strength of evidence. As Sackett has made clear, evidence-based medicine is “the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients.” Which evidence is best depends substantially on the question to be answered.

For some questions, the randomised controlled trial (RCT) is the best source of evidence, but there are many questions for which other research tools are more appropriate. In 2001, in response to the To Err is Human report, the Evidence-based Practice Centre was commissioned to evaluate the evidence for safety improvement practices. The practices were ranked on the basis of the “strength of evidence” with the RCT at the top.

The resulting list was criticised for having only 11 practices, of which only 3 could be characterised as true safety issues (anticoagulation for the prevention of deep venous thrombosis, antibiotic prophylaxis to prevent surgical infections, and use of pressure-relieving materials to prevent pressure sores); many well-accepted safety measures were omitted altogether.

There are several reasons that the report failed to identify more patient safety initiatives. Error prevention is a young field which (in New Zealand as elsewhere) has received little funding. This of course means that formalised evidence is not as abundant as it is in relation to the efficacy of drugs, for example. More importantly, there is a lack of appreciation of the limitations of traditional methods of research in this area. Randomised controlled trials are not always feasible or warranted in patient safety. They may not be feasible because randomisation is impossible or because the outcomes of interest (adverse events), although sometimes catastrophic, are rare.

The inadvertent administration of intrathecal vincristine alluded to above has occurred 15 times in the UK over last decade—a totally unacceptable frequency but one far too low to lend itself to a RCT. The overall mortality rate of anaesthesia is probably about 1 in 50,000 in Australia and New Zealand. To show a halving of this rate would require an RCT of over 4 million anaesthetics (with a power of 80% and p=0.05). A study this large is simply not possible. It is therefore not surprising that attempts to demonstrate objectively and definitively the safety benefits of oximetry through RCTs have been unsuccessful. This does not mean that there is no evidence of its value, just that we have to look elsewhere for that evidence.
Leape, Berwick, and Bates have put the matter this way:

…the anesthesia community has measured its progress over time, accumulating a “time series” track record whose signal is virtually incontrovertible. To say that convincing evidence of progress and effect is lacking because randomised trials of all safe anesthesia practices have not been conducted would be Luddite.  

Randomised controlled trials are best suited to evaluating the efficacy of individual interventions or therapies, whereas patient safety is primarily a function of how well the system of care is performing—evaluating safety is more like evaluating the effectiveness of a treatment in actual practice.

For example, RCTs can prove the efficacy of prophylactic anticoagulation under protocol to prevent venous thromboembolism, but the real patient safety gains come from ensuring that all eligible patients receive anticoagulation (appropriate care) and that they receive it on time, at the right dose, every time without fail (i.e. safe care).  

The latter two elements are systems issues that will be improved with reference to human factors theory, process engineering, and systems theory by the institution of a number of small changes guided by repeated evaluations through a cycle of continuous quality improvement. These changes—like those that resulted in the dramatic improvements in aviation safety and in anaesthesia—are not amenable to RCTs.

Human factors principles such as standardisation, simplification, and the use of checklists are commonly applied to improve patient safety. These are supported by common sense and evidence from industry. There is no need for an RCT to justify their application, anymore than there is need for an RCT to justify the use of a parachute when jumping from an aeroplane at altitude.  

For example, the standardisation of prescribing to require the use of leading zeros (0.5 mg not .5 mg—which can easily be misread as 5 mg), and to eliminate trailing zeros (1 mg not 1.0 mg—which can easily be misread as 10mg), eliminates a potentially dangerous prescription—but to find RCT evidence that this reduced adverse drug events would be difficult, expensive, and actually quite pointless.

As Leape, Berwick, and Bates say:

For policymakers to wait for incontrovertible proof of effectiveness before recommending a practice would be a prescription for inaction and an abdication of responsibility. The prudent alternative is to make reasonable judgments based on the best available evidence combined with successful experiences in health care. While some errors in these judgments are inevitable, we believe they will be far outweighed by the improvement in patient safety that will result.

If we are to deal with the current ‘epidemic of iatrogenic harm’ and promote a safer healthcare system in New Zealand, we need to persuade those who are responsible for healthcare expenditure that safety really does deserve the highest priority. This is not for trite reasons; we acknowledge that some risk is necessary if we are to balance achieving acceptable levels of safety with the other elements of quality (to be discussed in the remaining articles in this series). It is because the data are
overwhelming—our hospitals are not acceptably safe at present, and until they are, the
gains on expenditure from addressing this problem will arguably be greater than
expenditure on most other initiatives in healthcare today.

Huge investment into biomedical research has improved the effectiveness of drugs
and technology and transformed healthcare: we now possess and have already largely
deployed the means to cure many important illnesses. The challenge today is to use
these therapies to help patients, rather than harm them in the attempt. The public are
now well aware of this risk, not least because of several highly visible enquiries into
failures of exactly this type: addressing concerns about patient safety will go some
considerable distance towards re-establishing trust in our healthcare system.

Conflict of interest: Dr Mary Seddon—no conflict; Professor Alan Merry—financial interests in Safer
Sleep LLC.

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Case of rudimentary uterus and appendages

This case report was written by Emily H. Siedeberg, M.B., B.Sc., L.R.C.P., and published in the New Zealand Medical Journal 1906, Volume 5 (20), p12–13.

Miss L., aet. 20 years, consulted me for complete Amenorrhoea. She is a teacher in a Kindergarten, and very fond of children. Her general health is remarkably good; indeed, she says she has always considered herself as enjoying much better health than the majority of girls. She is rosy, robust and happy looking; height 5ft. 5½in., of slight build, but well rounded.

At the age of 16 years her mother consulted a doctor about the non-appearance of menstruation and was told that it was probably slow development, and as long as she kept well there was no need to trouble about it.

As no sign had appeared at age of 20, her mother asked me to examine her.

Questioned as to presence of pain or feeling of congestion, she said she had no indication of any sort. On one or two occasions she had had bleeding from the nose, but at quite irregular and long intervals.

Examination.—There was an entire absence of pubic and vulval hair, with the exception of about a dozen scattered ones. Axillary hair was also absent. Breasts were quite undeveloped, and nipples as flat as a child’s.

Labia Majora and Minora were naturally developed. Vaginal orifice admitted finger, but hymen was tough and fibrous.

The pubic rami met at the symphysis at an acute angle, as in the male pelvis. The vagina was of normal length and capacity, but no cervix was present, and no orifice could be felt in the vault. No uterus or appendages could be made out by vaginal examination. On rectal examination a slight thickening, about the thickness of a slate pencil, could be made out, stretching transversely above the vault of the vagina, but there was no indication of ovaries or tubes.

Remarks.—In such a marked rudimentary condition of uterus and appendages with non-development of breasts and pubic hair, it is usual to find the vagina also small and ill-developed, but in this case it was of normal length and capacity.

There had never been any sign of hysteria or other nervous disturbance, so frequently found associated with mal-development of these organs—the girl being particularly bright and happy in her work among children. There was also nothing masculine in her appearance, conversation or manner, indeed she was distinctly timid and retiring.

Emily Hancock Siedeberg, 1873–1968, grew up in Dunedin. Encouraged by her father, and grudgingly accepted by staff, Siedeberg became New Zealand’s first woman medical graduate in 1896. A streetside plaque commemorating this fact is near the University of Otago in Dunedin.
Meningococcal septic shock with adrenal apoplexy—Waterhouse-Friderichsen syndrome

Rajiv Sinha, Dipak Kanabar

A 1-year-old boy was transferred from a provincial hospital with profound shock, coagulopathy, and widespread purpura (Figure 1). His shock persisted despite vigorous fluid resuscitation, inotropes, and ventilatory support of perfusion. Presence of purpura and persistent hypotension, despite inotropes, led us to suspect Waterhouse-Friderichsen syndrome.

**Figure 1. Widespread purpura**

Bilateral adrenal haemorrhage was seen on ultrasound (Figure 2). A synacthen test revealed poor adrenal function, and meningococcus Group B was isolated from blood cultures.

The child made a slow recovery over a couple of weeks without any neurological sequelae.
Discussion

Waterhouse-Friderichsen syndrome, first reported in 1911 by Waterhouse, is characterised by a petechial rash, coagulopathy, cardiovascular collapse, and adrenal apoplexy.\(^1\) The presence of bilateral adrenal haemorrhage does not in itself signify adrenal insufficiency, and a formal assessment of cortisol response is required.\(^2,3\)

In inotrope-resistant hypotension due to septic shock, Waterhouse-Friderichsen syndrome should be considered.

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Mobile phone use and risk of brain tumours

An occasional report in the literature has implied a positive association between high grade astrocytoma (glioma) and phone use ipsilateral to the side of the tumour, brain tumours and phone use in rural areas, and use of analogue mobile phones.

An English case-control study involving 966 adults with glioma and 1716 random control subjects has found no such association in the short or medium term. That is good news as there are a lot of cellphone users out there.

An accompanying editorial comment makes two salient points. The first relates to the relatively short term follow up, bearing in mind that in adult humans, all known carcinogens, including radiation, require a latency period of usually more than 20 and often more than 30 years. And the second—“the biggest risk to health from mobile phones is using them while driving.”

Aspirin—the wonder drug

Aspirin is widely used to prevent cardiovascular and cerebrovascular atherothrombotic complications. This interesting paper discusses why it is not always effective. The rationale for its beneficial effect is that it reduces the activation of platelets by irreversibly acetylating cyclooxygenase-1 (COX-1), and thereby reduces thromboxane A_2 produced by platelets. Hence less platelet-derived thrombosis—and it is effective in low dosage. Laboratory studies indicate that low-dose aspirin (as low as 30 mg daily) uniformly suppresses platelet COX-1 in healthy controls and in patients.

Daily dosage in the range of 75–150 mg is recommended, as higher dosages have an increased risk of adverse effects (e.g. upper gastrointestinal symptoms and bleeding). Apparently some subjects do have genetically determined aspirin resistance but the usual cause of failure is more mundane—non-compliance—up to 40%.

Another problem is that some other non-steroidal anti-inflammatory drugs (NSAIDs), prevent access of aspirin to the COX-1 substrate binding site, and can thereby reduce the antiplatelet action of aspirin. So the aspirin needs to be taken hours before the NSAID.

BMJ 2006;332:883–6 & 864–5

Lancet 2006;367:606–17
ACE inhibitors and congenital anomalies

Angiotensin-converting-enzyme (ACE) inhibitors are among the most widely prescribed antihypertensive agents in the western world. However, they are avoided in the second half of pregnancy, as they can cause oligohydramnios, fetal growth retardation, pulmonary hypoplasia, joint contractures, hypocalvaria and neonatal renal failure, hypotension, and death. On the other hand, they have been considered safe in the first-trimester of pregnancy—until now.

A recently published study involving nearly 30,000 infants in Tennessee has shown that infants with only first-trimester exposure to ACE inhibitors had an increased risk of major congenital malformations (risk ratio, 2.71) as compared with infants who had no exposure to antihypertensive medications. Fetal exposure to other antihypertensive medications during only the first trimester did not confer an increased risk (risk ratio, 0.66). The malformations principally involved the cardiovascular and central nervous systems. The authors and an editorial commentator draw the obvious conclusion—avoid ACE inhibitors in pregnancy.


Bowel cancer screening program in Australia

Colorectal neoplasia (CRN) screening in Australia is imminent, and is likely to be based on faecal occult blood testing (FOBT). Those eligible, men who turn 55 and women who turn 65 years of age, will be invited to complete an immunochemical faecal occult blood test (FOBT) in the privacy of their own home and mail it in for analysis. Valid alternatives include endoscopic screening by flexible sigmoidoscopy (FS) or colonoscopy and possibly computed tomography colonography (CTC) (often referred to as virtual colonoscopy). Consumer choice of these various strategies has been evaluated in Australia. And the result? A choice of screening did not improve participation. Participation by FOBT was higher than by other tests. Not too surprising.


Save the banana

The world’s most popular fruit and the fourth most important food crop of any sort is in deep trouble. Why? Apparently nearly all bananas traded internationally are of a single variety, the Cavendish, the genetic roots of which lie in India. And the Cavendish is under threat from pandemics of diseases such as that caused by the black sigatoka fungus. The main hope for survival of the Cavendish lies in developing new hybrids resistant to the fungus, but this is a difficult and time-consuming task because the seedless modern fruit does not reproduce sexually and has to be bred from cuttings. The UN Food and Agriculture Organization (FAO) has warned that wild banana species are rapidly going extinct as Indian forests are destroyed, while many traditional farmers’ varieties are also disappearing. Hard to imagine a world without bananas.

New Scientist 2006;2561:5
More on alcohol and youth. Has the NZMA demonstrated that it is not a credible source of advice to Parliament?

NZMA’s responses to the queries in our letter of 19 May were evasive and ill-informed (http://www.nzma.org.nz/journal/119-1234/1994/), demonstrating that in preparing its advice to the Select Committee, it did not take a balanced view of the empirical evidence.

Below we provide examples of the nature of the assertions which underpin NZMA’s position.

Evasive response concerning the evidence

In their response, NZMA say: “Evidence that lowering the purchase age was associated with increase in harm is not evidence that raising it again will decrease harm”. We agree. They then go on to say “published evidence that we could find is that where the legal purchase age has been raised from 18 to 20, reduction in harm was minimal”. This is an evasive response to a major criticism of NZMA’s position, namely, that they were selective in their assessment of the relevant research literature.

To reiterate, in our letter of 19 May 2006 we drew to NZMA’s attention a study1 which we discuss in our paper;2 a systematic review of research in this area, which reports 23 studies where the age was raised and in 21 of which there was a positive benefit. In their reply, they made no acknowledgement of this important body of research evidence, instead selectively citing three studies which, at best, partially support their claim.

It should be noted that, in the meta-analysis by Shults et al,1 there were median reductions of 12–16% in the incidence of various traffic crash outcomes, which are large effects, relative to those achieved with other policy interventions, e.g. 8% for graduated driver licensing.3 Seldom do legislators and their advisers have such strong international and national evidence on which to base policy decisions.

It should be noted that the Shults et al review, which focuses on traffic crash outcomes, is complemented by that of Wagenaar and Toomey, who reached similar conclusions:

“The preponderance of evidence indicates there is an inverse relationship between the MLDA [minimum legal drinking age] and two outcome measures: alcohol consumption and traffic crashes” (p.206).4

Both of these reviews are published in readily available journals, indexed by Medline.

Ill-informed comments concerning the arbitrariness of the age of purchase

In their response, the NZMA argue that whether the minimum purchase age is 18 or 20 is “entirely arbitrary”, on the grounds that greater benefits might be expected by raising the age to 30, 40, or 50. This response is not only frivolous, but completely ignores the powerful interaction of maturation and alcohol consumption.
In New Zealand, alcohol consumption peaks at ages 18–19 years: they are our heaviest consumers.\(^5\)

There is good research evidence based on neuropsychological assessment and functional magnetic resonance imagining that:

1. Neural development is not complete at age 18, and that important structures (e.g., the hippocampus) continue to develop into the early 20s;\(^6\)

2. People in their teenage years have a normal propensity for sensation seeking (including drinking to intoxication), which declines with age thereafter;\(^7\)\(^8\)\(^9\), and

3. Heavy consumption of alcohol can produce anatomical and physiological damage to a developing brain,\(^10\)\(^11\) with the extent of that damage being greater than for older persons\(^6\) (see a series of reports published online by the New York Academy of Sciences for further information on brain development).

**Ill-considered reference to what others do**

NZMA says that in preparing their submission they took into account that: “Countries with which we identify such as Australia and the UK also have 18 as the legal purchase age”. What are readers meant to infer from this? We should do what others do even if the health consequences are negative?

**Other measures**

Like others in this debate, NZMA have argued that we need to strengthen our efforts on other measures to reduce alcohol-related harm, including, better enforcement and restrictions on advertising. We agree, but it should be noted that the evidence base for each of these strategies, while supportive of the actions proposed, is far less compelling than that for increasing the purchase age, in terms of volume and breadth of research, and likely public health impact. This view of the evidence is shared by a large team of international experts working under the auspices of the World Health Organization (WHO), who recently reviewed many hundreds of studies of interventions to reduce hazardous drinking.\(^12\)

In conclusion, the NZMA has demonstrated (in its response to our letter) that its submission to Parliament was not based on a balanced consideration of all of the relevant evidence. It cannot, therefore, be considered a credible source of advice to Parliament.

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References:

PHARMAC and statins—getting the best population health gains

We note the commentary by Drs Ellis and White (http://www.nzma.org.nz/journal/119-1236/2033/) on the history of statin funding in New Zealand. Many of these specific points have been raised previously in the Journal,1,2 and we have responded in detail (http://www.nzma.org.nz/journal/115-1163/203/ and http://www.nzma.org.nz/journal/116-1170/361/).3,4

In summary, access to statins was initially more restrictive than now—perhaps 45,000 patients were eligible prior to 1997, whereas around 300,000 are now eligible. The reasons for that were both clinical and financial. At that time the major studies related to secondary prevention and the price of statins was significantly higher than now—for example, 20 mg simvastatin cost over $1,000 per year (total expenditure was $16 million for perhaps 15,000 patients).

Widening access at that time in line with current NHF guidelines, at the above price could have resulted in expenditure of perhaps $200 million on one class of drugs—40% of total community pharmaceutical expenditure. Widening access to statins to allow the (now) 290,000 patients treatment was only achievable by using commercial opportunities to reduce the price of statins significantly. Wider access to statins then would have been at a significant cost to other patient groups.

PHARMAC is required to balance potential health gains for both high-risk individuals and the New Zealand population as a whole, amongst other criteria including costs, when making its decisions.5 Historically, patients at highest overall cardiovascular risk have tended not to receive statin treatment, particularly Māori and Pacific men. That is why PHARMAC is working with DHBs and communities with its One Heart Many Lives programme attempting to redress this.

Access to simvastatin is now unrestricted, and atorvastatin remains available as a second-line agent for those who genuinely need a more potent statin or cannot tolerate simvastatin. Statin usage rates in New Zealand are now the same as in Australia (see graph below),6 and a recent BMJ editorial has suggested that the United Kingdom should insist that simvastatin be the first-line statin therapy.7 However, the critical issue is not which statins are available—but rather, whether they are prescribed for, and used by, those at highest risk.
New Zealand and Australian use of statins since 2002

Peter Moodie
Medical Director
PHARMAC

Sean Dougherty
Therapeutic Group Manager
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References:


Optimising *Chlamydia* testing within constrained funding

The schedule for community pathology tests is not uniform within New Zealand. Some areas have a schedule item for molecular based testing (nucleic acid amplification tests, NAAT) for *Chlamydia trachomatis* while others, like Auckland, do not. National and local data show that the highest incidence of infection is found in those aged <25 years.¹² Ideally the best test should be provided for testing. This is particularly important for the highest risk group of women who are entering reproductive life and the implication infection has for tubal disease and neonatal infection.

NAAT have greater sensitivity over antigen-detection methods in diagnosing *C. trachomatis* infection.³⁴ Our schedule fee, however, was introduced many years ago and was based on antigen detection methods. NAAT have significantly higher reagent and labour costs. Using NAAT for all specimens, when only funded for antigen detection, would have a significant fiscal impact.

We have therefore assessed a sequential testing algorithm so we can provide NAAT to high-risk groups while controlling the cost of testing. From October–December 2005 we sequentially tested urogenital swabs submitted from females, aged 15–25, for *C. trachomatis* testing. Specimens were initially tested by enzyme immunoassay (EIA) antigen testing. Non-reactive specimens were then pooled (5 specimens/pool) and tested by NAAT. For positive pools the constituent pool samples were tested individually to confirm the individual positive specimen(s).

Over this period, 19,970 genital swabs were received from women aged 15–25 years; 833 (13.2%) were confirmed positive for *C. trachomatis*. Most, 591/833 (71%), were positive by EIA and confirmed by direct fluorescent assay (DFA) microscopy. The additional 242 (29%) positive samples were identified by NAAT testing. This represents a significant increase in infection detection but at a far lower cost than individual NAAT testing for all patients in this high-risk age group. On the basis of this evaluation we have extended our EIA/NAAT testing algorithm to this gender age group.

This testing algorithm may be of interest for other providers who do not have a specific NAAT fee for *Chlamydia* on their community pathology schedule.

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References:


Prioritisation in a stressed-out service

Since the inauguration of the British National Health Service, or NHS, in 1946, the state has come to dominate the provision of health in many countries, and with the socialism comes the shorthand. As the grip tightened in this country, we saw HBs become CHEs and RHAs, all now happily deceased, then the HFA, which disappeared as we got into DHBs.

General practice, not to be outdone, spawned IPAs and joined with the hospitals in PHOs. The skills and merits of the doctors can now be crudely measured, not by the two letters that precede their surname, but by the six to ten letters of the several abbreviated titles that come after it.

The doctor-patient relationship is breaking down, as the administration forms an unholy alliance with the law. GP obstetrics, for example, is regulated, not by the competence of the doctor meeting the needs of the patient, but by the relevant clauses of the appropriate Act. Another legal entity, the Health and Disability Commissioner, or HDC, lurks in the background, dealing with an endless stream of complaints.

Now we have a new terror, the First Specialist Assessment, or FSA. This socialist construct is about getting patients seen in the outpatients departments of our public hospitals. It, too, has collapsed here and there, as it tried, at huge expense, to take the place of a middle-aged receptionist, working for a self-employed private specialist hunched over his desk in his modest suite of rooms, where any anxious GP could get hold of him promptly.

In a seminal editorial, Frank Frizelle has drawn attention to “the significance of Case 04HDC13909” (http://www.nzma.org.nz/journal/119-1237/2072). It concerns the management of patients waiting for the FSA that, for some, never comes, as hospitals admit that they are no longer able to provide it for everyone that wants one.

In this case, the HDC probed a situation that arose not so long ago in the SDHB, or Southland District Health Board, when the Urology Department of Invercargill Hospital attempted to deal with a man aged 61 who had cancer of the prostate. I have carefully studied a number of the HDC’s opinions. They are often lengthy, and detailed to the point of ponderosity, but I am most grateful to Professor Frizelle for drawing my attention to this one. It’s on the web. It reads like a thriller. Don’t miss it (http://www.hdc.org.nz/files/pageopinions/04hdc13909urologist,dhb.pdf).

Case 04HDC13909 is about the collapse of a surgical service in a provincial hospital, a service that should never have been set up in the first place, since there was only one specialist to run it. All the patients should have been sent to Dunedin. The surgeon finally quit, and the mess was resolved when, in the course of one weekend, seven urologists from all over the country descended on Invercargill and cleaned it up.

For a long time, both the doctors and the administration appear to have adopted a see-nothing, do-nothing attitude, as things got worse and worse. It was a bad, bad, case. When it was all over, the HDC ordered some apologies, and the SDHB now says it...
has got its act together, as we usher in the infamous FSA, by its very existence an admission of delay and defeat.

The HDC endorses the Medical Council’s view that “prioritisation systems should be fair, systematic, consistent, evidence-based, and transparent.” When adjectives pile up like that, the futility and the desperation are plain. What do they want? A new bureaucracy to devise pain scales, order more investigations, and count the gallstones?

Telling consultants to accept responsibility for patients they have never seen is the stupidest thing I ever heard of. No wonder they are all being shovelled back into the trembling arms of the disgruntled GPs.

What happens next? How much more damage can the system withstand?

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Quality versus value

In the first paper of the series, *Quality improvement in healthcare in New Zealand*, Mary Seddon from EPIQ highlights the problem of understanding and implementing quality.

Prior to dissecting quality into its key components, it is important to understand that this problem arises from a misapplication of quality, particularly when applied to decisions on resource provision and distribution. Such decisions are made on “value” rather than quality, and thus we need to clarify this term prior to exploring quality.

“Value” is the relationship between quality and cost. In modern health, this is central to most decisions, from the coalface of general practice to the whole nation, from the patient to the Minister of Health. Patients, when they choose to see a GP, make a value judgement…will they receive the quality of service and outcomes for the money they spend?

At the national level, “value” is central to decisions made by the key funders such as PHARMAC, ACC, MOH, DHBs, etc. Each of these organisations hopefully judges “value” according to quality criteria (sometimes utilising appropriate quality frameworks such as exemplified by *Improving Quality (IQ): a systems approach for the New Zealand health and disability sector*) balanced against their budgets.

Value judgements are widespread but largely descriptive. Those organisations in health that try to deal with value in an explicit manner are few, and their efforts may raise more questions than answers. These explorations of value should receive wider debate.

Thus quality sits within “value”. EPIQ has taken a brave step in looking at the understanding of quality. I look forward to the rest of the series but plead for readers to appreciate that quality cannot be regarded in isolation.

Jim Vause
General Practitioner
Redwoodtown Doctors
Blenheim

References:


Douglas Ian Chisholm

25 April 1921—21 March 2006

Douglas was born in Oamaru in 1921. His father was a master at Waitaki Boy’s High School. There were four boys in the family but two died in childhood. Douglas’ younger brother, William, qualified in medicine and was a GP in Oamaru until his untimely death in his 50s.

Douglas attended Otago Boy’s High School where he was dux, a prefect, and a member of the 1st XV. He gained a University scholarship coming second in New Zealand.

His Medical School years spanned World War II. Medical students were told to stay put and qualify as the country would need doctors.

He was a good student and won the Stanley Batchelor Prize for surgery. After House jobs at Christchurch and Kew, Douglas and his bride Eileen moved to Hamilton where he set up in general practice with an interest in Anaesthetics.

The family next moved to Christchurch where Douglas was in general practice until he left for the UK and took his Fellowship. He worked in Leicester and Oxford before returning to New Zealand and to Dunedin where he was a specialist anaesthetist, lecturer in Anaesthesia, and later Assistant Director of Anaesthesia.

In 1964 came the final shift to Christchurch where he was appointed Director of Anaesthesia. He remained in this post until he retired in 1986. He gave no further anaesthetics but did work for ACC for several years. Anaesthesia was just emerging as an independent specialty. There were still many part-time GP anaesthetists and a local, national, and international shortage of anaesthetists trained to the standards required. There had to be training programmes set up, posts accredited, and examinations organised.

Douglas Chisholm was the right man in the right post. Many trainees prospered under his supervision and the number of staff members multiplied and there was specialisation within the specialty as intensive care evolved. Douglas put proposals for a day surgical unit in the late 1970s. This did not find favour in Wellington.

Inevitably he became involved with the wider hospital and was elected Chairman of the Hospital’s Medical Staff Association. He became Secretary and then President of the New Zealand Post Graduate Medical Federation.

Douglas Chisholm was recognised as a man of absolute integrity and reliability. He was knowledgeable, competent, and meticulously careful. Furthermore, he had sound judgment, common sense, and was a team worker.
Douglas was a keen golfer, and on Thursday afternoon he was to be found in a medical foursome at Shirley. He developed an interest in woodwork and was a meticulous gardener. A great reader, Douglas absorbed knowledge like a sponge and was a great lover of words well used.

Douglas and Eileen had a long and devoted marriage. Together they made a great team.

This obituary was collated from information supplied by Richard Chisholm, Vaughan Laurenson, and John Gibbs.
Index of NZMJ Obituaries: 1887–June 2006

This index of obituaries in the New Zealand Medical Journal was compiled by retired anaesthetist Basil Hutchinson.

Obituaries dating from 7 June 2002 onwards are in the online NZMJ and are accessible by looking at the archived issues or by searching with the name.

To obtain obituaries dating from before this, please contact NZMA National Office (email Lucy Wesley, lucy@nzma.org.nz), or phone 04 472 4741, or write to NZMA National Office, PO Box 156, Wellington, with your request. There is no charge for one-off requests. This index will be updated regularly.

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