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This Issue in the Journal

Management of patients admitted with an Acute Coronary Syndrome in New Zealand: results of a comprehensive nationwide audit

C Ellis, G Gamble, J French, G Devlin, P Matis, J Elliott, S Mann, M Williams, H White (For the New Zealand Acute Coronary Syndromes [NZACS] Audit Group)

In a report from the Cardiac Society of New Zealand Acute Coronary Syndrome (ACS) Audit, treatments and investigations were compared between 8 intervention (5 public, 3 private) and 28 non-intervention hospitals (all public). Patients admitted to a non-intervention hospital were less likely to have coronary angiography (17% vs 30%, p<0.01)—and for patients with a definite ACS, angioplasty (3% vs 14%, p<0.01) and coronary surgery (3% vs 5%, p=0.16) were also less frequent. Patients with an ACS receive inequitable treatments in New Zealand. A comprehensive national strategy with appropriate funding is required.

Acute Coronary Syndrome patients in New Zealand receive less invasive management when admitted to hospitals without invasive facilities

C Ellis, G Devlin, P Matis, J Elliott, M Williams, G Gamble, S Mann, J French, H White (For the New Zealand Acute Coronary Syndromes [NZACS] Audit Group)

A 2-week Cardiac Society audit of 930 patients admitted with suspected definite acute coronary syndromes to 36 hospitals across New Zealand, reports low levels of evidence-based treatments (aspirin 82%, clopidogrel 63%, beta-blockers 63%, ACE inhibitors 43%, statins 55%), investigations (angiography 21%), and revascularisation (angioplasty 7.3%, coronary surgery 3.8%). There were marked variations between hospitals, and rates are low by international standards. There is a need for a comprehensive national strategy including continuing audit.

Cardiac rehabilitation services in New Zealand: access and utilisation

F Doolan-Noble, J Broad, T Riddell, D North

Cardiac rehabilitation following a major heart event can reduce the risk of death and disability and improve the person’s quality of life. This study highlights gaps in the provision of services in New Zealand: women and older people were less likely to be referred for rehabilitation, and lack of transport was identified as an important obstacle to attending. It suggests we should consider other options for delivering the service and for improving the uptake of cardiac rehabilitation services.

Exponential increase in clinical use of plasma brain natriuretic peptide (BNP) assays

T Yandle, S Fisher, J Livesey, E Espiner, M Richards, G Nicholls
A survey was made to document utilisation of measurements for a heart hormone, BNP, by doctors in the Christchurch area between 1995 and 2002. It was found that the number of measurements requested had increased steadily from around 50 per month in 1995 to approximately 400 per month in 2002. Whereas the majority of requests were for inpatients in Christchurch hospitals, a sizeable minority were for patients in primary care (26%) or in the emergency department (14%). It is apparent that doctors in the region are relying increasingly on BNP measurements, presumably in the diagnosis of heart failure and in guiding drug therapy for this condition.
Quality and equity in cardiovascular health in New Zealand: the need for agreed achievable standards of care, cohesive planning, and action

Norman Sharpe, Gerard Wilkins

Context

The current context of heart health in New Zealand represents an unprecedented convergence of needs and opportunities. The New Zealand Health Strategy 2000 outlines 13 priority health objectives—6 relate to cardiovascular disease or related preventive objectives (including diabetes). The fundamental principles on which the Health Strategy is based include the Treaty of Waitangi relationship, a lifespan approach to health, focus on the disadvantaged, and a collaborative intersectoral approach. Further principles relate to equitable access, system performance, and involvement of consumers and communities. These principles and objectives provide guidance for District Health Boards (DHBs), Primary Health Organisations (PHOs), and other providers.

For the first time, the current health-sector changes (and particularly the emergence of the PHOs) explicitly designate priority and responsibility for population health promotion, as well as individual healthcare. In response to this, and based on the New Zealand Health Strategy objectives, DHBs and PHOs are now according cardiovascular disease and diabetes appropriate priority in their planning and development of specific strategies and actions.

Underlying such plans are a range of evidence-based cardiovascular guidelines, which have been developed principally through the New Zealand Guidelines Group process. Relevant guidelines recently launched include cardiac rehabilitation, cardiovascular risk assessment and management, type 2 diabetes management and stroke management.

This Issue of the Journal contains guidelines for pre-hospital administration of fibrinolytic therapy by New Zealand general practitioners. Several other guidelines are in development or revision—particularly those related to the management of acute coronary syndromes, atrial fibrillation, heart failure, and smoking cessation. To complete the continuum, the Ministry’s recently launched Healthy Eating Healthy Action Plan provides the necessary platform for preventive health promotion action ‘upstream’ in the community.

Finally, the National Heart Foundation Strategic Plan 2003 includes focused preventive objectives related to smoking, nutrition, physical activity, and obesity as well as clinical care objectives related to high risk patient management and rehabilitation. These objectives and underlying principles are well aligned with the New Zealand Health Strategy.

The Heart Foundation’s implementation plan is based on a continuum from primary prevention through to tertiary treatment and rehabilitation, as well as a range of
project work (increasingly in partnership or close alliance with other agencies). Direct health-sector engagement is being promoted through collaborative associations with providers and initiation of evaluable pilot projects based on guideline standards.

**Knowledge, culture, and systems**

In cardiovascular health, there is an extensive evidence base in most areas of practice on which to base recommendations for effective care to provide predictable improvement in patient outcomes. However, there are numerous issues and barriers which prevent translation of evidence into practice (with or without guidelines), and consequently there is general under-utilisation of effective care.

A pertinent example is the access to (and uptake of) statin medication for cholesterol-lowering in patients with (or at high risk of) coronary heart disease. Clear evidence of the efficacy for this treatment was provided during the 1990s. Although uptake in New Zealand has increased gradually, and particularly since the Special Authority requirement was widened in 1997 to include general practitioners (and then removed in 2002), it is estimated that more than 100,000 people eligible for this effective preventive treatment still remain to be identified and treated.

Guideline implementation requires identification of resources and a culture of collaboration and teamwork at a local or regional service level. Such implementation is often best initiated through evaluable local pilot or demonstration projects, which can show success and then be replicated. Broad representative stakeholder involvement in the guideline-development process is also crucial for credibility and to ensure that recommended standards are practical, affordable, and achievable.

Guideline development may therefore inevitably create a tension between best practice advocacy and the funding framework. Unrealistic recommendations may only tend to increase existent disparities. Indeed, there is a need for understanding that healthcare resources are constrained and that sectional advocacy needs to be reasonable and balanced against the need for broader community stewardship in a climate of rationing. On the other hand, guideline recommendations that are out of step with other published international documents will lack professional credibility.

In setting targets for healthcare performance, improved systems and processes are required for safe, effective, and equitable implementation. Such systems could include increased use of registers and checklists with some redundancy. While high performance healthcare systems and processes are evident in some areas, they are lacking in most. The comparative example of the airline industry, where flight fatality rates approach zero, is apt to some extent. In contrast, in healthcare, many recommended beneficial approaches to assessment and treatment have administrative deficiency rates that commonly exceed 50% in many settings.

**Current inequities**

Coronary heart disease death rates for men and women have fallen steadily in New Zealand since 1970. Between 1970 and 2000, there has been a 73% decline in mortality rate for men aged 45–64 years, as well as a 71% decline for women in the same age range. Similar declines have occurred in other Western countries during the same time period, although more steeply in Australia and the United States. In the United Kingdom, it has been estimated that 58% of the decline can be explained from
population risk-factor reductions, and 42% due to individual treatments. While the decline in New Zealand during this period has occurred in different socioeconomic and ethnic groups, disparities amongst these groups have increased.

Through the 1980s and 1990s, the relative risk for cardiovascular mortality comparing lowest and highest income quintiles for people aged 25–77 years (as a measure of socioeconomic position) increased progressively, to approach a two-fold higher risk for men in the lowest quintile during the years 1996–1999.

Coronary heart disease ethnic mortality rates for males aged 35–64 years show even wider disparity—being 3.5 times higher for Maori males compared with non-Maori and non-Pacific males, with rates for Pacific males being intermediate between Maori and non-Maori. In Maori males aged 45–64 years, heart failure mortality rates and hospital admissions due to heart failure are more than 8 times greater than for non-Maori; a similar trend is noticed for Maori females compared with non-Maori.

In this Issue of the Journal, the Maori Cardiovascular Advisory Group outline a Maori specific cardiovascular action plan that has been developed with a call to action to address these and related disparities. Apart from socioeconomic and ethnic disparities, there are also major disparities in access to recommended treatments highlighted by publications in the Journal in this and recent editions. For example, a retrospective comparison of management of patients with acute coronary syndromes (ACS) between Taranaki and Waikato Hospitals showed significantly higher cardiac angiography and revascularisation rates in Waikato Hospital—where management was performed by cardiologists with immediate access to invasive intervention facilities (revascularisation 16.7% vs 4% p=0.0002). A similar (larger) audit, encompassing 36 hospitals, documented patient management—and compared intervention rates between the 8 centres equipped for intervention, with the other 28 without such facilities. Cardiac investigation levels, revascularisation rates, and use of discharge medications of proven benefit were all generally low. As with the Taranaki-Waikato comparison, cardiac angiography and revascularisation rates were significantly higher in the intervention centres (percutaneous coronary intervention [PCI] 14% vs 3%, p<0.0001; coronary artery bypass graft [CABG] surgery 5% vs 3%, p=0.16). Patients had similar age and risk profiles in both types of centre but there were more Maori admitted to non intervention centres than intervention centres (8.2% vs 3.8%, p=0.0063). Comparison with contemporary practice in other Western countries shows that rates in all New Zealand centres, with or without invasive facilities, are relatively low.

There is almost a three-fold variation in the rate of coronary revascularisation generally (acute and elective CABG surgery and coronary angioplasty together) across all DHBs. After adjustment for age, ethnicity and socioeconomic deprivation structure of each DHB region, the observed and expected discharge rates have provided a standardised discharge ratio for each region for 2002–3. The rates for some few DHBs were approaching half the overall national rate and others were up to 1.5 times the national rate. Again, aside from this regional variation within New Zealand, the overall national rate is low in comparison with other countries.
An extensive national audit of cardiac rehabilitation services published in this Issue of the Journal also highlights limitations of access and utilisation in that part of the heart healthcare continuum. Limitations were shown at each stage of referral and only 12% of all originally eligible hospital patients completed a Phase 2 programme after discharge. Lack of transport was associated with reduced referral and attendance, and the 65-74 year age group were most likely to complete the programme.

**Changing paradigms for coronary heart disease and risk factors**

Different levels of causation of cardiovascular risk are now well recognised. Biological risk factors result from interaction of distal environmental, socioeconomic, and sociocultural factors with genetic and ethnic factors. Psychological and behavioural factors are intermediate determinants. Healthcare access can further influence risk progression and health outcomes.

Counterfactual modelling of the causes of all deaths by risk factor in New Zealand lists diet, tobacco, deprivation, cholesterol, blood pressure, body mass index, insufficient physical activity, and pre-diabetes as the top eight risk factors. Acknowledging the various assumptions and approximations that this modelling approach is based on, it nevertheless highlights the importance of ‘looking upstream’ to achieve ongoing improvements through various public and population health strategies. Of particular importance are those strategies directed towards child health and the obesity epidemic, to moderate the downstream burden of disease in the long-term.

**National standards for heart healthcare**

It is impressive and unacceptable that there are such large inequities in cardiovascular health outcomes and access to cardiovascular care in New Zealand. While there is agreement on the principles and priority objectives for healthcare, including cardiovascular health, there is a need for cohesive national planning and action to ensure general achievement of agreed quality standards (while acknowledging the constraints of current resources and systems).

The present planning and progression of cardiovascular health plans by a few providers with enlightened leadership and participation could potentially increase inequities between regions. To avoid this problem, such advanced organisations could assist others through national forums and provision of templates for suitable cardiovascular health action plans—this should ensure that standards of care and inequities are improved (within and between regions simultaneously).

In summary, to achieve more rapid improvement in standards and outcomes of heart healthcare, specific considerations and solutions are:

- Provider needs assessment for heart health, based on the New Zealand Health Strategy principles and objectives (requiring organisational commitment, leadership, broad participation, and intersectoral co-operation)
- Commitment by providers to the Maori specific cardiovascular action plan.
- Pilot or demonstration projects across the heart health continuum with an ‘equity lens’ applied in health promotion, risk assessment, and management; acute coronary syndromes; heart failure management and cardiac rehabilitation.
- Cardiovascular guideline implementation to an agreed high standard of care on a local basis recognising environmental constraints
- Population registration and a lifespan approach to prevention and risk management.
- Regionalisation of acute coronary syndromes management (to agreed national standards for intervention).
- Appropriate budgeting for patient transfers and cross-boundary funding.
- Revision of coding and funding of cardiovascular case weights.
- Revision of coronary scoring and surgical prioritisation methods.
- Forward planning nationally and regionally for facilities and workforce.
- Fostering of a culture shift for care providers towards quality improvement, ongoing evaluation and practice audit against guideline recommendations and indicators
- Research investment weighted to relevance and need and aimed at improving socioeconomic, ethnic, and other inequities.

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Extracranial carotid artery disease: the stroke burden of intervention

Tim Buckenham

In the 1960s, carotid endarterectomy was the most commonly performed arterial procedure in the world. Level 1 data from the European Carotid Surgery Trialists (ECST) Collaborative Group and the North American Symptomatic Carotid Endarterectomy Trial (NASCET) Collaborators have allowed evidence-based practice and better understanding of risk-benefit ratios—leading to a substantial reduction in carotid endarterectomy surgery.

Since the publication of these landmark trials, sub-group analysis of the original data has raised issues concerning the number of interventions required to prevent a single stroke in the range of patients traditionally regarded as suitable for endarterectomy. Another way of looking at this issue is to ask what is the ratio between ‘strokes caused’ and ‘strokes saved’—this is determined by the iatrogenic stroke burden of carotid repair, and the risk of stroke from the underlying disease. This ratio gains further importance when one considers that iatrogenic strokes occur at the time of intervention, whereas strokes due to the underlying disease may express themselves some years in the future (thus giving a significant stroke-free interim while the burden of iatrogenic stroke is immediate).

This equation is further confused by the rapid development of endoluminal repair (stenting of the carotid artery) enhanced by the use of cerebral protection devices, which have reduced the iatrogenic stroke burden of this alternative to carotid endarterectomy. This minimally invasive alternative to surgery has many attractions as it can be performed as a day stay—compared with a mean hospital stay in New Zealand for surgical endarterectomy of 4 days. The SAPPHIRE trial showed that endoluminal repair may have a reduced non-stroke morbidity and mortality, primarily from reduction in myocardial ischaemia and cranial nerve injury, however this trial had strict selection criteria applied to the trial interventionalists to overcome the steep learning curve.

The aim of this paper is to assess the various sub-group analyses and further develop the concept of strokes caused versus strokes saved (in order to identify the groups where this ratio is the most favourable).

What is the iatrogenic stroke burden arising from carotid repair?

The New Zealand Society of Vascular Surgery audit gives a risk of 4.8% for stroke or death from surgical carotid repair. Randomised trials such as CAVATAS (primary angioplasty versus surgery) gave a risk of stroke and/or death of 10% in both groups. In the NASCET trial, risk of stroke and death was 6%. NASCET however selected patients and surgeons, and 40% of surgeons were rejected in view of their track record. Whilst NASCET was under-way, Hsai recorded the mortality following endarterectomy amongst non NASCET centres in this group—the operative morbidity and mortality and stroke rate was 5 times higher than NASCET at 20%. In 1998,
morbidity and mortality were still double. Considering non NASCET centres perform over 90% of all endarterectomies in the USA, these figures are important. If the true stroke and death rate reaches 10% (as reported in CAVATAS), one could reasonably argue that ‘strokes caused’ and ‘strokes saved’ are an equilibrium and no endarterectomies should be performed.

Who benefits the most from carotid intervention?

Without specific knowledge of local carotid intervention morbidity and mortality figures, it is difficult for clinicians to minimise the ratio of strokes caused to strokes saved, however the selection of appropriate sub-groups with a high risk of stroke from the underlying disease will help maximise the benefit of carotid intervention. Elderly patients have increased long-term benefits despite the perception that they may have higher operative risk. This perception is unjustified and there is no reason to with-hold carotid endarterectomy from patients older than 75 years who are otherwise fit.

The ‘strokes caused’ versus ‘strokes saved’ ratio in this group is low because the iatrogenic burden is the same—but this sub group of patient has a higher stroke risk with medical treatment alone. Patients with retinal symptoms only in the presence of a severe ipsilateral carotid stenosis have a much less favourable ratio—as the stroke risk in these patients is much lower than patients presenting with hemispheric events (6% compared with 43.5% over 2 years). This considerably increases the ratio between ‘strokes caused’ and ‘strokes saved’ on the assumption the iatrogenic stroke burden rate of intervention in this group is unchanged. Surgery on this group would mean two strokes caused to prevent three at 2 years.

Sex has a significant influence on this ratio. Women have a lower risk of ipsilateral ischaemic stroke on medical treatment, and a higher operative risk. Other sub-groups that have a higher iatrogenic stroke burden are those with very tight stenosis (95–99%) with non-visualisation or collapse of the distal ICA (String sign). These patients also have a reduced risk of stroke from the disease itself. The actual risk of ipsilateral stroke in 1 year in this group is approximately 4.4%; this compares to 35% for a 90–94% stenosis. Assuming an iatrogenic stroke burden of 4.5%, there is no net gain in surgical management of these patients at 1 year.

How does timing alter the risk benefit ratio?

The risk of stroke decreases with time after the presenting neurological event. To optimise the stroke prophylaxis for carotid intervention, evidence now suggests that the benefit is greatest if the intervention is performed as close as possible to the neurological event. Furthermore, conventional interpretation of NASCET and ECST trials suggest that (6 months after the event) a symptomatic patient would have the same risk of stroke as an asymptomatic patient.

Conversely, if there is a very high risk of stroke (ie, a 90–99% stenosis, excluding String sign), these patients have a sustained increased risk of stroke—18% in the first year and 14% in the second, dropping to 3% after 2 years. In these patients, the 6-month threshold may be extended depending on the iatrogenic stroke burden of the carotid intervention. There is a reasonable argument that males with hemispheric symptoms and stenoses between 80 and 99% (excluding String sign) may still benefit from endarterectomy beyond 6 months. Indeed, many investigators now feel the benefit is greatest if the intervention is performed within 2 weeks of the event.
What is the role of endovascular repair?

Carotid stenting has recently been the subject of a randomised controlled trial (the SAPPHIRE trial) in which the major adverse event rate of endoluminal carotid repair was 3.8%, and 4.6% for surgery. Non-stroke mortality (eg, myocardial infarction) favoured endoluminal repair over surgery. Endoluminal repair with distal protection is as safe (if not safer) than surgery, however its role in stroke prophylaxis has yet to be fully determined. There are currently sub groups in which stenting is favoured; these are based on contra indications to surgery rather than comparison of risk. Examples of these indications include hostile neck due to a previous radiotherapy or a high carotid bifurcation.

Conclusion

It must be remembered that carotid intervention is a prophylactic procedure, but in order to prevent stroke, some strokes will be caused. The significance of these strokes is greater as they occur at the time of intervention. To maximise the stroke prevention of carotid intervention, sub-groups can be selected where the risk of medical treatment is high but surgical risk unchanged. Timing of carotid intervention is important—the closer to the presenting neurological symptom, the greater the benefit as the risk of stroke declines quickly with time.

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References:
1. The European Carotid Surgery Trialists Collaborative Group. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. Lancet. 1991;337:1235–43.
Management of patients admitted with an Acute Coronary Syndrome in New Zealand: results of a comprehensive nationwide audit

Chris Ellis, Greg Gamble, John French, Gerald Devlin, Philip Matsis, John Elliott, Stewart Mann, Michael Williams, Harvey White. (For the New Zealand Acute Coronary Syndromes [NZACS] Audit Group.)

Abstract

Aims To audit all patients presenting to a New Zealand hospital with a myocardial infarction or unstable angina (an acute coronary syndrome [ACS]) over a 14-day period, to assess their number, presentation type and patient management during the hospital admission.

Methods We formed a group of clinicians to lead the local audit process with one representative for each hospital (n=36) that admitted ACS patients. A comprehensive data form was used to record individual patient information for patients admitted between 0000 hours on 13 May 2002 to 2400 hours on 26 May 2002.

Results 930 patients were admitted with a suspected or definite ACS: 11% with a ST-segment-elevation myocardial infarction (STEMI), 31% with a non-STEMI, 36% with unstable angina pectoris (UAP), and 22% with another cardiac or medical diagnosis. Cardiac investigations were limited: echocardiogram (20%), exercise treadmill test (20%), cardiac angiogram (21%).

In-hospital revascularisation rates were low for those patients with a definite presentation with an ACS (STEMI, non-STEMI, UAP, n=721). Percutaneous coronary intervention (PCI) rates were 13%, 8%, and 4%—with coronary artery bypass grafting (CABG) rates being 4%, 3%, and 4% respectively. The use of discharge medications of proven benefit was also generally low (n=695): aspirin (82%), clopidogrel (8%), beta-adrenergic blockers (63%), angiotensin converting enzyme (ACE) inhibitors (43%), and statins (55%).

Conclusions A collaborative group of clinicians has performed a nationwide audit of acute coronary syndrome patients, which has demonstrated low levels of investigations, evidence-based treatments, and revascularisation. There is a need for a comprehensive national strategy—particularly for continuing audit of the treatment of patients presenting with a suspected or definite acute coronary syndrome to a New Zealand hospital.

There is unequivocal evidence that certain treatments improve the outcome of patients presenting with an ACS. International and local guidelines support intensive medical treatment, and for many patients early revascularisation, which is of proven benefit and shown to be cost-effective in high-risk patient groups.

Comprehensive national surveys of ACS patients have previously been attempted in several countries, including Argentina and Italy. Other countries such as Britain and the United States of America have performed less complete surveys. In addition,
some international trials and registries, such as the Global Registry of Acute Coronary Events (GRACE)\(^1\) and the European Heart Survey\(^2\) have attempted to compare the treatment of ACS patients between countries and regions.

In New Zealand, both the number and management of patients presenting with an ACS, is unknown, although an earlier Auckland-based study provided useful demographic and outcomes data from 1993.\(^3\) Many clinicians are unable to optimally manage the ACS patients under their care due a limited provision of service and a relatively low level of funding.

In May 2001, the Cardiac Society of New Zealand supported a meeting, which invited representatives from all major New Zealand hospitals to discuss the appropriate management of patients with ACS. At this meeting, the need for a national audit was further developed and endorsed as an important aspect of the strategy to improve patient care. New Zealand has favourable characteristics to undertake a comprehensive national survey. There is a history of good collaboration in cardiovascular research, which is strengthened by the small specialist community across the country, and the personal contact between clinicians.

We therefore created a network of practicing clinicians representing every New Zealand hospital that admits ACS patients and performed a comprehensive National audit of the in-hospital management of all ACS patients across New Zealand. We chose to undertake the audit in a 2-week period in the autumn of 2002, to minimise the known influence of seasonal change on the numbers of ACS patients.\(^4\)

**Methods**

**Data collection**—A network was created—consisting of one physician for every hospital in New Zealand that admitted ACS patients (n=36). Most centres also co-opted one or more research nurses or registrars to assist with data collection for the study.

The data collection form recorded patient demographics, initial and discharge diagnosis, medication use in hospital and at discharge, as well as investigations undertaken and invasive treatments received by patients. The inclusion criterion for the audit was ‘a patient admitted overnight with a suspected or definite acute coronary syndrome’.

A 2-week audit period was accepted as a compromise between the need to collect sufficient patient numbers to obtain an accurate representative cohort versus the ability of unfunded clinicians and nurses to collect the consecutive patient data. We collected data from 0000 hours on Monday 13\(^{th}\) May to 2400 hours on Sunday 26\(^{th}\) May 2002.

Following input from the 14 other local ethics committees across New Zealand, ethical approval was obtained from the North Health Ethics Committee. As an audit of current practice, individual patient consent was not required. The ethics committees encouraged the collection of patient names and National Health Index (NHI) numbers to assist with accurate data collection.

Data (including revascularisation procedures) from patients subsequently transferred to another institution are ‘attributed’ to their original admitting hospital. Patients readmitted within the 2 weeks have all admissions included in the data; they only represented a small % of the overall patient number. Ethnicity was self-reported at hospital admission.

All 36 hospitals had the facilities for assessing troponin levels. Five methods were used: troponin T [Roche] (16 hospitals), troponin I [Abbott] (13 hospitals), troponin I [Bayer] (3 hospitals), troponin I [Ortho] (1 hospital), ‘Rapid’ troponin T [Roche] (3 hospitals). In order to divide non-STEMI and unstable angina patients by means of a ‘positive’ troponin,\(^5\) we defined ‘normal’ or ‘abnormal’ troponin levels using the ‘cut-off’ for ‘positive’ troponins as Troponin T [Roche] >0.03ug/L, Troponin I [Abbott] >0.4ug/L, Troponin I [Bayer] >0.2ug/L, Troponin I [Ortho] >0.08ug/L, ‘Rapid’ troponin T ‘positive’ [Roche].
Hypertension and dyslipidaemia were defined as patients on treatment, or with a previous clinical diagnosis. Patients with diabetes mellitus were those on diet control, oral hypoglycaemic, or insulin treatment. Cardiogenic shock was defined as: a systolic blood pressure of <90mmHg for at least 30 minutes, or the need for supportive measures to maintain a systolic blood pressure of ≥90mmHg with end organ hypoperfusion. Sustained ventricular tachycardia was defined as >30 seconds of ventricular tachycardia, or requiring electrical cardioversion.

Statistics—Continuous data are summarised as median and interquartile range. Differences in frequencies were tested using chi-squared procedures. All tests were two-tailed and a 5% significance level was used.

Results

930 patients with a suspected or definite ACS were admitted to 36 New Zealand hospitals and enrolled in the ACS audit over the 14-day period (Figure 1). Thirty-six patients were readmitted—within the 2 weeks, 35 patients were admitted once and 1 patient was admitted twice (29 readmissions to the same hospital, and 7 to another hospital). Fifty-seven patients were transferred from their admitting hospital to another institution for further management (53 [93%] to an intervention centre). Over the 2 weeks, one hospital had no admissions, 9 hospitals admitted 40 or more patients, and one hospital admitted 131 patients.

Figure 1. New Zealand ACS hospitals (n=36) and patient numbers (n=930)

Patient demographics—The median age was 69.6 (IQR 58-78, range 21-102) years. Forty-two percent of patients were female, 81% Caucasian, 7% Maori, 2% Indian, 1% Pacific Islander, 1% Asian, and 5% were another ethnic group—and in 5% the ethnicity was unspecified. Baseline demographics are shown in Table 1.

Patient diagnoses—Using both the admission clinical diagnosis and the measurement of a positive troponin level, we found that 101 (11%) patients presented with a ST-segment-elevation myocardial infarction (STEMI), 287 (31%) with a non-STEMI,
333 (36%) with unstable angina pectoris (UAP), and 209 (22%) patients with another cardiac or medical diagnosis.

Table 1. Baseline demographics (n=930)

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<th>Baseline demographic</th>
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<th>Percentage</th>
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<tr>
<td>Median age (IQR): 69.6 yrs (58–78 yrs)</td>
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<tr>
<td>Gender (male)</td>
<td>535 (58%)</td>
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<td>Ethnicity</td>
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<tr>
<td>- Caucasian</td>
<td>753 (81.0%)</td>
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<tr>
<td>- Maori</td>
<td>62 (6.7%)</td>
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<td>- Other groups</td>
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<td>Hypertension</td>
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<tr>
<td>Dyslipidaemia</td>
<td>326 (35%)</td>
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<td>Prior MI</td>
<td>325 (35%)</td>
<td></td>
</tr>
<tr>
<td>Prior angiogram</td>
<td>257 (28%)</td>
<td></td>
</tr>
<tr>
<td>Prior PCI</td>
<td>105 (11%)</td>
<td></td>
</tr>
<tr>
<td>Prior CABG surgery</td>
<td>91 (9.8%)</td>
<td></td>
</tr>
<tr>
<td>Prior peripheral arterial disease</td>
<td>93 (10%)</td>
<td></td>
</tr>
<tr>
<td>Prior TIA/stroke</td>
<td>112 (12%)</td>
<td></td>
</tr>
<tr>
<td>Prior atrial fibrillation</td>
<td>126 (13%)</td>
<td></td>
</tr>
</tbody>
</table>

IQR: Interquartile range; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; TIA: Transient ischaemic attack.

Medical management—Medical treatments are shown in Table 2. Overall, 55% of STEMI patients received thrombolytic therapy. Of 77 STEMI patients who were admitted within 12 hours of symptom onset, 53 (69%) received thrombolytic therapy and 3 (3.9%) received primary PCI. Sixty-four percent of non-STEMI patients were treated with low molecular weight heparin (54% enoxaparin, 12% daltaparin) and 8.8% unfractionated heparin. A few patients received more than one type of heparin, 92 (32%) patients were not treated with any heparin. Three percent of non-STEMI patients received a glycoprotein 2b/3a inhibitor, and 13% clopidogrel therapy.

Cardiac Investigations—Investigations are listed in Tables 2 and 3. Of the 930 patient admissions, 184 (20%) underwent an echocardiogram, 190 (20%) received an exercise treadmill test, and 199 received (21%) a cardiac angiogram. 583 (63%) patients received neither an exercise treadmill test nor a cardiac angiogram.

Revascularisation—Of 721 ‘definite’ ACS patients (STEMI, non-STEMI, UAP), 159 (22%) patients underwent a cardiac angiogram, 50 (6.9%) patients received a PCI, and 25 (3.5%) patients received CABG (Tables 2 and 3).

Hospital outcomes—Thirty-eight (4%) patients died during their hospital admission: 14 (14%) of STEMI patients, 12 (2%) of non-STEMI/UAP patients, and 11 (5%) of ‘other cardiac or medical diagnosis’ patients. Twenty (2%) patients had a recurrent or subsequent myocardial infarction, and 144 (16%) had recurrent angina. Cardiogenic shock developed in 41 (4%) patients. Twenty (2%) patients received an intra-aortic...
balloon pump, 12 (1%) received a temporary pacemaker, and 2 patients received a permanent pacemaker. Six patients developed a stroke (5 non-haemorrhagic), and 13 (1%) sustained ventricular tachycardia. Only 1% of patients were enrolled in a research project whilst in hospital.

Table 2. Treatments and investigations of STEMI, Non-STEMI, and UAP patients

<table>
<thead>
<tr>
<th></th>
<th>STEMI</th>
<th>Non-STEMI</th>
<th>UAP</th>
<th>P**</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>101 (11%)</td>
<td>287 (31%)</td>
<td>333 (36%)</td>
<td></td>
</tr>
<tr>
<td><strong>Treatments in hospital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>56 (55%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary PCI</td>
<td>3 (3.0%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>33 (34%)</td>
<td>156 (54%)</td>
<td>126 (39%)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Daltaparin</td>
<td>6 (5.9%)</td>
<td>33 (12%)</td>
<td>39 (12%)</td>
<td>0.23</td>
</tr>
<tr>
<td>UF heparin</td>
<td>28 (28%)</td>
<td>25 (8.8%)</td>
<td>22 (6.6%)</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>No heparin***</td>
<td>40 (40%)</td>
<td>92 (32%)</td>
<td>159 (48%)</td>
<td>0.0011a</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>5 (5.0%)</td>
<td>6 (2.1%)</td>
<td>1 (0.3%)</td>
<td>0.0046c</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>2 (2.0%)</td>
<td>2 (0.7%)</td>
<td>1 (0.3%)</td>
<td>0.20</td>
</tr>
<tr>
<td>Abciximab</td>
<td>1 (1%)</td>
<td>0</td>
<td>0</td>
<td>0.05</td>
</tr>
<tr>
<td>Aspirin</td>
<td>87 (88%)</td>
<td>228 (79%)</td>
<td>268 (81%)</td>
<td>0.22</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>14 (14%)</td>
<td>35 (13%)</td>
<td>21 (6.2%)</td>
<td>0.015d</td>
</tr>
<tr>
<td><strong>Investigations in hospital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>89 (89%)</td>
<td>265 (92%)</td>
<td>269 (81%)</td>
<td>0.0001e</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>35 (35%)</td>
<td>61 (22%)</td>
<td>54 (16%)</td>
<td>0.0003b</td>
</tr>
<tr>
<td>Exercise test</td>
<td>18 (18%)</td>
<td>52 (18%)</td>
<td>86 (26%)</td>
<td>0.04c</td>
</tr>
<tr>
<td>Angiogram</td>
<td>31 (31%)</td>
<td>71 (35%)</td>
<td>57 (16%)</td>
<td>0.006d</td>
</tr>
<tr>
<td>No ETT/Angio</td>
<td>57 (57%)</td>
<td>180 (63%)</td>
<td>203 (63%)</td>
<td>0.51</td>
</tr>
<tr>
<td>No Echo/Angio</td>
<td>50 (50%)</td>
<td>178 (62%)</td>
<td>241 (72%)</td>
<td>&lt;0.0001e</td>
</tr>
<tr>
<td>PCI</td>
<td>13 (13%)</td>
<td>24 (8.4%)</td>
<td>13 (3.9%)</td>
<td>0.004d</td>
</tr>
<tr>
<td>CABG surgery</td>
<td>4 (4.0%)</td>
<td>8 (2.8%)</td>
<td>13 (3.9%)</td>
<td>0.72</td>
</tr>
<tr>
<td><strong>In-hospital deaths</strong></td>
<td>14 (14%)</td>
<td>10 (3.5%)</td>
<td>2 (0.9%)</td>
<td>&lt;0.0001f</td>
</tr>
<tr>
<td><strong>Discharge medications (n=721-26 deaths: n=695)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>77 (89%)</td>
<td>228 (83%)</td>
<td>266 (80%)</td>
<td>0.78</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>14 (14%)</td>
<td>26 (9.5%)</td>
<td>17 (5.1%)</td>
<td>0.01f</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>68 (76%)</td>
<td>177 (63%)</td>
<td>193 (59%)</td>
<td>0.22</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td>43 (51%)</td>
<td>127 (45%)</td>
<td>128 (39%)</td>
<td>0.33</td>
</tr>
<tr>
<td>Statins</td>
<td>58 (67%)</td>
<td>153 (55%)</td>
<td>172 (52%)</td>
<td>0.59</td>
</tr>
<tr>
<td>Fibrates</td>
<td>0</td>
<td>7 (2.5%)</td>
<td>9 (2.4%)</td>
<td>0.26</td>
</tr>
</tbody>
</table>

**Compares STEMI/Non-STEMI/UAP Post hoc tests.

aUAP different from Non-STEMI, Non-STEMI different from STEMI.

bSTEMI different from Non-STEMI and UAP.

cSTEMI different from UAP, Non-STEMI different from UAP.

dUAP different from Non-STEMI and STEMI.

eUAP and Non-STEMI different.

fAll different.

gUAP and STEMI different.

***Neither enoxaparin, daltaparin, nor UF heparin.

PCI: Percutaneous coronary intervention; UF: Unfractionated; CABG: Coronary artery bypass grafting; Angio: Angiogram; ETT: Exercise treadmill test; UAP: Unstable angina pectoris; ACE: Angiotensin converting enzyme.
Clinicians from 13 hospitals, which admitted ACS patients exclusively to their coronary care unit (CCU), were confident of collecting all ACS patient admissions (n=202). 10 hospitals that admitted patients with an ACS to either a CCU or to a medical ward were able to fully enrol these patients into the audit (n=320). 4 hospitals, which admitted patients to either the CCU or to a medical ward, were able to enrol all CCU patients and most of the medical ward patients (n=172), estimating that 2–5% of medical ward patients (6 patients) were missed. 9 hospitals, which admitted ACS patients to both the CCU and the medical ward, were able to enrol all CCU patients (n=266) but none of the medical ward patients, and missed an estimated 5 to 30% of patients (37 patients). Hence, an estimated total of 43 ACS patients (4%) were admitted to a medical ward over the 2 weeks, and not included nor further considered in this audit.

Table 3. Investigations and invasive treatments

<table>
<thead>
<tr>
<th>Procedure</th>
<th>All patients (n=930)</th>
<th>‘Definite’ ACS patients* (n=721)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td>794 (85%)</td>
<td>623 (86%)</td>
</tr>
<tr>
<td>- Pulmonary oedema **</td>
<td>96 (10%)</td>
<td>76 (11%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>184 (20%)</td>
<td>150 (21%)</td>
</tr>
<tr>
<td>Exercise treadmill test</td>
<td>190 (20%)</td>
<td>156 (22%)</td>
</tr>
<tr>
<td>Cardiac angiogram</td>
<td>199 (21%)</td>
<td>159 (22%)</td>
</tr>
<tr>
<td>Exercise test and cardiac angiogram</td>
<td>42 (4.5%)</td>
<td>38 (5.3%)</td>
</tr>
<tr>
<td>Exercise test or cardiac angiogram</td>
<td>347 (37%)</td>
<td>277 (38%)</td>
</tr>
<tr>
<td>Neither exercise test or cardiac angiogram</td>
<td>583 (63%)</td>
<td>444 (62%)</td>
</tr>
<tr>
<td>PCI</td>
<td>69 (7.3%)</td>
<td>50 (6.9%)</td>
</tr>
<tr>
<td>CABG</td>
<td>35 (3.8%)</td>
<td>25 (3.5%)</td>
</tr>
</tbody>
</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; *Patients with STEMI (n=101), non-STEMI (n=287), and unstable angina pectoris (n=333); **Physician/Radiologist assessment.

Discussion

The major strength of this study has been the demonstration that a collaborative group of clinicians can perform a prospective, comprehensive audit of patients admitted to 36 hospitals in New Zealand with an ACS. Our approach has allowed clinicians to collect the data, which we believe has resulted in an accurate collection. It also allows clinicians to collectively have responsibility for the data. By benchmarking current practice, the ACS audit will enable physicians to examine the provision of equitable and good practice throughout the country. Moreover, the infrastructure created will facilitate future audit with the goal of assessing temporal trends and improving patient outcomes.

We identified 930 patients admitted with presentations of STEMI (11%), non-STEMI (31%), UAP (36%), and other cardiac or medical diagnosis (22%). The hospital management and outcomes of these patients throughout New Zealand had not previously been known.

In-hospital investigations: For the entire cohort, the use of a chest X-ray following presentation with a suspected or definite ACS was 85%. The use of an
echocardiogram (20%), exercise treadmill test (20%), or cardiac angiogram (21%) was low.

For the STEMI and non-STEMI patients (n=388), with myocardial damage and at the highest risk, the use of echocardiography (25%) or angiography (26%) was low, with 59% receiving neither as a method of assessing left ventricular systolic function, which is important in risk stratification.17

Furthermore, for the same group, the use of an exercise treadmill test (18%) or a cardiac angiogram (26%) as methods of risk assessment was also low—with 61% of patients not receiving either test. These levels of investigation contrast with the recommendations of local and international guidelines,2–6 which recommend that all STEMI and non-STEMI patients be considered for assessment in these ways.

International comparisons—Previous international ACS patient cohorts7–12 have selected different patient populations, which limits the ability to compare these studies with the New Zealand ACS Audit. The GRACE registry11,18 is probably the most appropriate comparator for our audit, although the methods used to enrol patients were similar, but not identical. It was not designed as a comprehensive national survey, rather as a collection of 95 hospitals in 14 countries in North and South America, Europe, Australia, and New Zealand (2 sites) which has allowed comparisons to be made between countries.

Patients entered in the GRACE registry had to be admitted with an ACS as the presumptive diagnosis and to have ≥1 of the following: electrocardiographic changes consistent with ACS, serial increases in serum biochemical markers of cardiac necrosis, and/or documentation of coronary artery disease.18

Reperfusion therapy, heparin, and platelet inhibitor use—In the current audit, 77 STEMI patients were admitted within 12 hours of symptom onset and had the most to gain from reperfusion: 53 (69%) received thrombolytic therapy and 3 (3.9%) were treated with PCI. In the widely spread population of New Zealand, this finding was expected as 24-hour cover for primary PCI was routinely available at only 1 centre in New Zealand, with the 4 other public interventional centres offering it to a variable extent. The overall level of thrombolytic therapy for STEMI patients (55%) is consistent with data from GRACE where thrombolytic therapy use was 47%—although primary PCI use was higher in GRACE at 18%,19 compared to 3% in the New Zealand ACS Audit.

Some non-reperfused patients are likely to have contraindications to treatment (although we did not specifically record this)—but some patients will probably have missed an opportunity for reperfusion, which has also been reported from GRACE.20 Further study of this issue would be helpful.

Overall, we found that there was a low level (68%) of heparin use (unfractionated or low molecular weight) for non-STEMI patients. Patients not treated would be unable to benefit from an estimated 47% reduction in death or myocardial infarction.21 Although there will certainly be a small group of non-STEMI patients with a clear contraindication to the use of heparin, these treatment levels are lower than expected and may be an area where clinicians can improve their medical treatment. Again, further study of this issue would be helpful. All 36 hospitals had access to heparins—although not all hospitals had access to enoxaparin, which is specified as preferable to...
unfractionated heparin by the Australia and New Zealand, and the United States Guidelines.\textsuperscript{2,6}

There was a very low use of intravenous glycoprotein 2b/3a inhibitors (3\%) for the management of non-STEMI patients, despite the recommendations of international and local guidelines, and despite the expected 9\% reduction in death or myocardial infarction.\textsuperscript{22}

Many hospitals do not have glycoprotein 2b/3a inhibitor drugs available for clinicians to use, as a result of local pharmaceutical policies. In addition, some clinicians try to ‘target’ glycoprotein 2b/3a inhibitor drugs to particularly ‘high-risk’ patients, such as those with elevated troponins and diabetes mellitus.\textsuperscript{22}

Medically treated patients were also largely unable to benefit from the use of clopidogrel (used in 13\%)—despite a 20\% reduction in cardiovascular death, myocardial infarction or stroke demonstrated in similarly treated patients in the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial.\textsuperscript{23} Patients receiving a PCI were the only group to routinely access clopidogrel.

**In-hospital revascularisation**—Overall, revascularisation was undertaken in 17\% of STEMI, 11\% of non-STEMI and 8\% of UAP patients. We found that PCI during the hospital admission was performed in 13\%, 8\%, and 4\% of patients and CABG in 4\%, 3\% and 4\% of patients, from each group. This intervention level is low compared to figures from the GRACE Registry,\textsuperscript{11} which reported PCI rates as being 40\%, 28\%, 18\%, and CABG surgery rates being 4\%, 10\% and 5\% (Table 4 and Figure 2).

**Table 4. Cardiac interventions by baseline condition: comparison with the GRACE Registry\textsuperscript{11}**

<table>
<thead>
<tr>
<th>Patients</th>
<th>STEMI</th>
<th>Non-STEMI</th>
<th>UAP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GRACE n=3419</td>
<td>NZACS n=101</td>
<td>GRACE n=2893</td>
</tr>
<tr>
<td>Angiogram</td>
<td>55%</td>
<td>31%</td>
<td>53%</td>
</tr>
<tr>
<td>PCI</td>
<td>40%</td>
<td>13%</td>
<td>28%</td>
</tr>
<tr>
<td>CABG surgery</td>
<td>4%</td>
<td>4%</td>
<td>10%</td>
</tr>
</tbody>
</table>

STEMI: ST-segment-elevation myocardial infarction; UAP: Unstable angina pectoris; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

Indeed, many STEMI, non-STEMI and UAP patients will potentially benefit if treated with revascularisation,\textsuperscript{2–6,24–27}—and the low levels found in the current audit are a cause of concern.
Discharge medications—Patients (without clear contraindications), and discharged following a presentation with an ACS, should be routinely prescribed aspirin—resulting in a reduction in vascular events of about 25% and clopidogrel with a 20% reduction for those with a non-STEMI presentation.
In addition, a beta-blocker, with a reduction in risk of death of around 20%, and a statin, with a reduction in risk of death of approximately 30% in 5 years, should be given to these patients. Furthermore, an angiotensin converting enzyme (ACE) inhibitor should be given acutely for all patients with anterior MIs, second or subsequent MIs, and for those patients with heart failure and an ejection fraction of <40%—resulting in a reduction of the risk of death of 25%.

ACE-inhibitors should also be prescribed for all patients with coronary or other vascular disease, due to their role in preventing vascular events. In addition, other medications may also be appropriate for some sub-groups of these patients.

For STEMI/non-STEMI/UAP patients, the use of various therapies at discharge (May 2002) was generally lower than the rates reported in patients enrolled in GRACE from April 1999 to December 2000: aspirin (80–89% vs 90–95%), beta-blockers (59–76% vs 75–81%), ACE inhibitors (39–51% vs 50–57%), and statins (52–67% vs 37–51%), respectively.

Unfortunately we did not record the presence of contraindications for the use of secondary prevention therapies. Nevertheless, only half of New Zealand ACS Audit patients received a statin—a sub-optimal level, which in New Zealand has been partly due to PHARMACs previous funding restrictions, resulting in a low level of statin use across the community.

The results of the Heart Protection Study showed a benefit for vascular patients (individuals with prior coronary, cerebral or peripheral vascular events, or with diabetes mellitus or hypertension, at high-risk of developing events) with a cholesterol level of 3.5mmol/L and above, and implied that all vascular patients should be considered for statin therapy after presentation with an ACS.

Although there are Australia and New Zealand guidelines for the management of patients with ACS (endorsed by the Cardiac Society of Australia and New Zealand), it is recognised that it would be of value to have local guidelines addressing some of the unique aspects of the New Zealand health scene where there are restrictions to funding and the availability of various therapies. These local, guidelines are being developed with the help of the Cardiac Society and the New Zealand Guidelines Group.

Modern medical and revascularisation treatments enhance patient outcomes and many have been shown to be ‘cost-effective’. These include the use of statin therapy following a MI, and the use of an invasive revascularisation strategy for non-STEMI patients. It should be emphasised how cost-ineffective it is to not have adequate facilities available for use, or for them not to be used. This point must be emphasised to politicians, health administrators, PHARMAC, physicians and the public of New Zealand.

Study limitations include the fact that a short audit may produce some chance findings and bias in patient selection. Furthermore, we were reliant on local investigators to check the accuracy of individual patient data, without there being a central system of review. Nevertheless, this audit has revealed deficiencies with the delivery of optimal management for ACS patients, with a low level of service provision for this high-risk group. These data show that few hospitals in New Zealand are practising evidence-based medicine according to local and international guidelines. It is probable that
this situation results, at least in part, from both a limited central coordination of clinical service as well as a lack of local support services.

**Conclusions**—There appear to be deficiencies in the availability and use of modern medicines, as well as inadequate facilities for appropriate investigations and revascularisation of patients with acute coronary syndromes. Continuing audit and feedback to local clinicians will probably help to improve the clinical service. Furthermore, open, informed public debate needs to be encouraged as to the level of service provision available in all regions of New Zealand. We believe that there is an urgent need to develop a comprehensive nation-wide strategy for patients presenting to a New Zealand hospital with an ACS.

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We thank these audit leaders and assistants in the following hospitals—from north to south by region. (Patient numbers in the study are given inside brackets.) #Chairman. *Steering Committee member.

**Auckland/Northland (North Island)**

Kawakawa Hospital, Dr P Burgoyne, Ms S August (4), Whangarei Hospital, Dr B Wong, Ms K O'Keefe (30), North Shore Hospital, Auckland, Dr H Hart, Ms J Wickham (66), Auckland Hospital, Dr C Ellis*, Mr G Gamble*, Ms W Benjamin (48), Mercy Private Hospital, Auckland, Dr T Clarke, (4), Green Lane Hospital, Auckland, Assoc Prof J French*, Prof H White*, Ms B Williams (26), Ascot Private Hospital, Auckland, Dr A Maslowski, (0), Middlemore Hospital, Auckland, Dr A Ko, Dr M Lund, Dr H Oettli (40).

**Waikato/Central North Island**

Thames Hospital, Dr J Lennane, Dr Aftabuzzaman (23), Tauranga Hospital, Dr J Tisch, Dr G Porter, Ms V Watts, Ms J Braid (48), Waikato Hospital, Hamilton, Dr G Devlin*, Ms D Penney (63), Whakatane Hospital, Dr E Edwards, Ms D Garner (13), Rotorua Hospital, Dr K Logan, Ms A Morley (26), Tokoroa Hospital, Dr P Reeve, Dr F Kanan (1), Te Kuiti Hospital, Dr P Reeve, Dr J Pusupati (2), Taupo Hospital, Dr A Ludbrook (11), Gisborne Hospital, Dr F Aitcheson, Ms K Weytmans (7), Tauramunui Hospital, Dr P Reeve, Dr R Shepherd (1), New Plymouth Hospital, Dr I Ternouth (28).
Wellington/Southern North Island

Hastings Hospital, Dr R Luke, Ms J Mackenzie (66), Wanganui Hospital, Dr T Thompson, Ms K Olsen (30), Palmerston North Hospital, Dr R Shameem (27), Masterton Hospital, Dr T Matthews, Ms K Lee (12), Hutt Hospital, Dr S Mann*, Ms A Cuthbert (19), Wellington Hospital, Dr P Matis*, Ms D Middlemitch, Ms B Scott (50), Wakefield Private Hospital, Wellington, Dr M Abernethy (2), Nelson Hospital, Dr A Hamer, Ms R Price (21), Blenheim Hospital, Dr M Heynike, Ms M Udy (16).

Christchurch, Canterbury (South Island)

Greymouth Hospital, Dr Y Al Khairulla, Ms L Skeats (9), Christchurch Hospital, Dr J Elliott*, Prof M Richards, Ms L Campbell, Ms A Alspach (131), Ashburton Hospital, Dr N Abdul-Ghaffar, Ms A Smart (11), Timaru Hospital, Dr M Hills, Ms Maria Hammond, Ms C Barker (16).

Dunedin, Otago (South Island)

Oamaru Hospital, Dr P Curzon (4), Dunstan Hospital Clyde, Dr G Nixon, Ms S Meaden (9), Dunedin Hospital, Dr MJ Williams*, Ms M McLelland (42), Invercargill Hospital, Dr C Renner, Dr A Maloney (23).

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References:


43. White HD, Willerson JT. We must use the knowledge that we have to treat patients with acute coronary syndromes. Circulation. 2004;109;745–9.
Acute Coronary Syndrome patients in New Zealand receive less invasive management when admitted to hospitals without invasive facilities

Chris Ellis, Gerald Devlin, Philip Matsis, John Elliott, Michael Williams, Greg Gamble, Stewart Mann, John French, Harvey White. (For the New Zealand Acute Coronary Syndromes [NZACS] Audit Group.)

Abstract

Aim To compare differences in the presentation and management of acute coronary syndrome (ACS) patients presenting to interventional versus non-interventional New Zealand hospitals.

Methods We assessed the data collected by the New Zealand Cardiac Society ACS Audit Group over 14 days from each hospital in New Zealand (n=36) that admits ACS patients. Patient management at intervention centres (5 public, 3 private) was compared with non-intervention centres (28 public). Investigations and revascularisation procedures performed on transferred patients were ‘attributed’ to the referring centre.

Results From 0000 hours on 13 May 2002 to 2400 hours on 26 May 2002, 930 patients were admitted to a New Zealand hospital with a suspected or definite ACS: ST-segment-elevation myocardial infarction [STEMI] (11%), non-STEMI (31%), unstable angina pectoris [UAP] (36%), or another cardiac or medical diagnosis (22%).

Patients admitted to a non-intervention centre (n=612) were the same age (median 70 years) with similar risk factors, but were more likely to be Maori (8.2% vs 3.8%, p=0.0063) and were less likely to have a history of prior cardiac angiography (26% vs 28%, p=0.02) or percutaneous coronary intervention [PCI] (9.6% vs 14%, p=0.03) than patients admitted to an intervention centre (n=318).

Patients admitted to a non-intervention centre were more likely to have a chest X-ray (88% vs 81%, p<0.0024), as likely to have an exercise treadmill test (20% vs 22%, p=0.39), but less likely to receive an echocardiogram (17% vs 26%, p<0.0005), a cardiac angiogram (17% vs 30%, p<0.0001), or neither a treadmill nor a cardiac angiogram (68% vs 53%, p<0.0001) for cardiac risk assessment.

For patients with a definite ACS presentation (STEMI, Non-STEMI, UAP, n=721), PCI was performed less often for patients admitted to non-intervention centres: 3% vs 14% (p <0.0001), although the rate of coronary artery bypass grafting was similar: 3% vs 5% (p=0.16).

Conclusion Patients admitted to a hospital without cardiac interventional facilities receive fewer investigations and less revascularisation than patients admitted to Intervention Centres. Hence patients admitted with an acute coronary syndrome in New Zealand receive inequitable management. A comprehensive National strategy is needed to improve access to optimal cardiac care.
Recent clinical trials have demonstrated that ‘high-risk’ acute coronary syndrome (ACS) patients benefit from vigorous medical management and an invasive revascularisation strategy. Local and international guidelines have recommended the benefits of this strategy.

Patients presenting with an ST-segment-elevation myocardial infarction (STEMI) should undergo urgent reperfusion of the culprit vessel, using thrombolytic therapy or primary percutaneous coronary intervention (PCI), in addition to adjunctive antithrombotic therapy with combinations of aspirin, heparin, and clopidogrel.

Patients presenting with unstable angina pectoris (UAP)/Non-STEMI should receive ‘passivation’ of the culprit lesion also using combinations of aspirin, heparin, a glycoprotein 2b/3a inhibitor, and clopidogrel. They should receive early invasive management if they are troponin positive, have dynamic ST segment changes, have an intermediate or high TIMI-risk score, ongoing ischaemia or haemodynamic instability, diabetes, or other features of a worse outcome.

These optimal management strategies have wide-ranging implications for a national programme for the treatment of ACS patients.

We aimed to determine whether patients admitted to a hospital not equipped with surgical and percutaneous revascularisation capabilities were able to access interventional management to the same extent as patients admitted directly to such centres, and we compared these rates with international registries. We used data from a comprehensive national audit from each New Zealand hospital (n=36) that admitted such patients during a 14-day period in May 2002.

Methods

Data collection—The development of the New Zealand Acute Coronary Syndrome (NZACS) Audit Group and the methodology for the national audit, which was supported by the Cardiac Society of New Zealand, has been published elsewhere. The inclusion criterion for the audit was ‘a patient admitted overnight with a suspected or definite acute coronary syndrome’. An extensive four-page case report form was used to obtain patient demographics, initial and discharge diagnosis, medication use in hospital and at discharge, as well as investigations undertaken and invasive treatments received by patients. Ethnicity was self-reported at hospital admission.

Data from the NZACS Audit was used to compare patients’ presentation and management at intervention centres (5 public hospitals and 3 private hospitals), with non-intervention centres (28 public hospitals) [Table 1]. An additional 5 public hospitals in the non-intervention centres had the ability to perform a cardiac angiogram, without percutaneous intervention or coronary artery bypass grafting (CABG) surgery. Cardiac angiography, PCI, and CABG surgery was also performed at a private hospital in Christchurch; however, this hospital does not plan to admit ACS patients and hence is not further considered with this audit. Investigations and revascularisation of transferred patients was attributed to the referring centre. The data was collected from 0000 hours on Monday 13th May to 2400 hours on Sunday 26th May 2002.

Statistics: Continuous data were summarised as median and interquartile range (IQR). Differences in frequencies were tested using standard chi-squared procedures. All tests were two-tailed and a 5% significance level was maintained throughout.

Results

Admissions and transfers—Over the 14-day period, 318 suspected or definite ACS patients were admitted to an intervention centre and 612 were admitted to a non-intervention centre. Of these, 36 patients were readmitted within the 2 weeks (35 patients readmitted once, and 1 patient readmitted twice) and 57 patients were
transferred to another institution for further management (93% to an intervention centre). Data from patients transferred were attributed only to the hospital to which they were initially admitted (Table 1).

Table 1. Admissions and transfers to Intervention and Non-Intervention Centres.

<table>
<thead>
<tr>
<th>FACILITIES</th>
<th>No of Patients</th>
<th>Transferred Out</th>
<th>Transferred In</th>
<th>Angiogram</th>
<th>PCI</th>
<th>CABG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intervention Centres (n=8)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Green Lane, Auckland</td>
<td>26</td>
<td>6</td>
<td>v</td>
<td>v</td>
<td>v</td>
<td>v</td>
</tr>
<tr>
<td>Mercy, Auckland (Private)</td>
<td>4</td>
<td>2</td>
<td>v</td>
<td>v</td>
<td>v</td>
<td>v</td>
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<tr>
<td>Ascot, Auckland (Private)</td>
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<td>0</td>
<td>v</td>
<td>v</td>
<td>v</td>
<td>v</td>
</tr>
<tr>
<td>Waikato</td>
<td>63</td>
<td>21</td>
<td>v</td>
<td>v</td>
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</tr>
<tr>
<td>Wellington</td>
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<td>16</td>
<td>v</td>
<td>v</td>
<td>v</td>
<td>v</td>
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<td>Wakefield Wellington (Private)</td>
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<td>v</td>
<td>v</td>
<td>v</td>
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<tr>
<td>Christchurch</td>
<td>131</td>
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<td>v</td>
<td>v</td>
<td>v</td>
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<tr>
<td>Dunedin</td>
<td>42</td>
<td>7</td>
<td>v</td>
<td>v</td>
<td>v</td>
<td>v</td>
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<td><strong>Non-Intervention Centres (n=28)</strong></td>
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<td>100%</td>
<td>100%</td>
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<tr>
<td>Kawakawa</td>
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<td>x</td>
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<tr>
<td>Whangarei</td>
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<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>North Shore</td>
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<td>Middlemore</td>
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<td>x</td>
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<td>x</td>
</tr>
<tr>
<td><strong>Total</strong></td>
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<td>10</td>
<td>53</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
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<td></td>
<td></td>
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<tr>
<td>Thames</td>
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<td>x</td>
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<td>x</td>
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<tr>
<td>Tauranga</td>
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<td></td>
<td>v</td>
<td>x</td>
<td>x</td>
<td>x</td>
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<td>x</td>
<td>x</td>
<td>x</td>
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<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Tokoroa</td>
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<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Te Kuiti</td>
<td>2</td>
<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Taupo</td>
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<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Gisborne</td>
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<td>x</td>
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<tr>
<td>Taumarunui</td>
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<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>New Plymouth</td>
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<td></td>
<td>v</td>
<td>x</td>
<td></td>
<td>x</td>
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<tr>
<td><strong>Total</strong></td>
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<td>21</td>
<td>1</td>
<td>20%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Wellington/Southern, North Island</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hastings</td>
<td>66</td>
<td></td>
<td>v</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Wanganui</td>
<td>30</td>
<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Palmerston North</td>
<td>27</td>
<td></td>
<td>v</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
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<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Hutt</td>
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<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Nelson</td>
<td>21</td>
<td></td>
<td>v</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Blenheim</td>
<td>16</td>
<td></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>191</td>
<td>17</td>
<td>0</td>
<td>43%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Christchurch/Canterbury, South Island</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Patients admitted to each type of hospital were the same age (median 70 years, p=0.88) with similar risk factors, but at a non-intervention centre they were more likely to be Maori (8.2% vs 3.8%, p=0.0063) [Table 2]. Patients admitted to a non-intervention hospital were more likely to have had a prior MI, (38% vs 28%, p=0.003) but less likely to have previously undergone a cardiac angiogram (26% vs 28%, p=0.02) or PCI (9.6% vs 14%, p=0.03).

### Table 2. Baseline demographics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hospital type</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n=318)</td>
<td>Non-intervention (n=612)</td>
</tr>
<tr>
<td>Age median (range)</td>
<td>69.7 (21.2-102.3)</td>
<td>69.6 (24.7-96.5)</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>170 (53%)</td>
<td>365 (60%)</td>
</tr>
<tr>
<td>Ethnicity:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Caucasian</td>
<td>259 (81%)</td>
<td>494 (81%)</td>
</tr>
<tr>
<td>- Maori</td>
<td>12 (3.8%)</td>
<td>50 (8.2%)</td>
</tr>
<tr>
<td>- Others</td>
<td>47 (15%)</td>
<td>68 (11%)</td>
</tr>
<tr>
<td>Tobacco smoker:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Current</td>
<td>59 (19%)</td>
<td>112 (18%)</td>
</tr>
<tr>
<td>- Previous</td>
<td>115 (36%)</td>
<td>264 (43%)</td>
</tr>
<tr>
<td>- Never</td>
<td>138 (43%)</td>
<td>209 (34%)</td>
</tr>
<tr>
<td>- Not reported</td>
<td>6 (1.9%)</td>
<td>27 (4.4%)</td>
</tr>
<tr>
<td>Hypertension (drug treatment)</td>
<td>149 (47%)</td>
<td>293 (48%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>58 (18%)</td>
<td>103 (17%)</td>
</tr>
<tr>
<td>- Type 1</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>- Type 2</td>
<td>49</td>
<td>91</td>
</tr>
<tr>
<td>- Not defined</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Dyslipidaemia (drug treatment)</td>
<td>96 (30%)</td>
<td>230 (38%)</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Within 1 month</td>
<td>92 (28%)[12 (13%)]</td>
<td>233 (38%)</td>
</tr>
<tr>
<td>Prior angiogram</td>
<td>99 (28%)</td>
<td>158 (26%)</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>46 (14%)</td>
<td>59 (9.6%)</td>
</tr>
<tr>
<td>- Within 6 months</td>
<td>9 (20%)</td>
<td>21 (36%)</td>
</tr>
<tr>
<td>Prior CABG</td>
<td>33 (10%)</td>
<td>58 (9.5%)</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>30 (8.1%)</td>
<td>64 (11%)</td>
</tr>
<tr>
<td>Prior TIA/Stroke</td>
<td>37 (12%)</td>
<td>75 (12%)</td>
</tr>
<tr>
<td>Length of Stay: Days (Median + IQR)</td>
<td>3 (2-7)</td>
<td>4 (2-6)</td>
</tr>
</tbody>
</table>
**In-hospital investigations**—Overall, patients admitted to a non-intervention centre were more likely to have had a chest X-ray (88% vs 81%, *p*<0.0024), but had the same rates of exercise treadmill tests (20% vs 22%, *p*=0.39) as patients admitted to an intervention centre (Table 3). Patients admitted to non-intervention centres were less likely to receive an echocardiogram (17% vs 26%, *p*<0.0005), or a cardiac angiogram (17% vs 30%, *p*<0.0001), or either test (68% vs 53%, *p*<0.0001) for cardiac risk assessment. For patients with a definite ACS (STEMI, Non-STEMI, UAP, *n*=721), those admitted to a non-intervention centre received less angiography (17% vs 32%, *p*<0.0001) (Table 4).

**Table 3. Investigations and revascularisations (all patients: *n*=930)**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Hospital type</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n=318)</td>
<td>Non-intervention (n=612)</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>256 (81%)</td>
<td>538 (88%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>83 (26%)</td>
<td>101 (17%)</td>
</tr>
<tr>
<td>Exercise test</td>
<td>70 (22%)</td>
<td>120 (20%)</td>
</tr>
<tr>
<td>Angiogram</td>
<td>95 (30%)</td>
<td>(17%)</td>
</tr>
<tr>
<td>Exercise test or angiogram</td>
<td>143 (45%)</td>
<td>176 (29%)</td>
</tr>
<tr>
<td>Exercise test and angiogram</td>
<td>15 (4.7%)</td>
<td>27 (4.4%)</td>
</tr>
<tr>
<td><strong>Neither exercise test nor angiogram</strong></td>
<td>168 (53%)</td>
<td>415 (68%)</td>
</tr>
<tr>
<td>PCI</td>
<td>40 (13%)</td>
<td>29 (4.7%)</td>
</tr>
<tr>
<td>CABG</td>
<td>19 (6.0%)</td>
<td>16 (2.6%)</td>
</tr>
</tbody>
</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

**Table 4. Investigations and Revascularisations (Patients with a ‘definite’ ACS: STEMI, non-STEMI, UAP: *n*=721)**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Hospital type</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n=251)</td>
<td>Non-intervention (n=470)</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>199 (79%)</td>
<td>424 (90%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>67 (45%)</td>
<td>83 (55%)</td>
</tr>
<tr>
<td>Exercise test</td>
<td>61 (24%)</td>
<td>95 (20%)</td>
</tr>
<tr>
<td>Angiogram</td>
<td>81 (32%)</td>
<td>78 (17%)</td>
</tr>
<tr>
<td>Exercise test or angiogram</td>
<td>118 (47%)</td>
<td>134 (29%)</td>
</tr>
<tr>
<td>Exercise test and angiogram</td>
<td>30 (12%)</td>
<td>27 (6%)</td>
</tr>
<tr>
<td><strong>Neither exercise test nor angiogram</strong></td>
<td>133 (53%)</td>
<td>336 (72%)</td>
</tr>
<tr>
<td>PCI</td>
<td>35 (14%)</td>
<td>15 (3%)</td>
</tr>
<tr>
<td>CABG</td>
<td>12 (5%)</td>
<td>13 (3%)</td>
</tr>
</tbody>
</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

In-hospital treatments—**Only two patients admitted to an intervention centre, and one patient rapidly transferred from a non-intervention centre, received a primary PCI. Intervention centre STEMI patients received the same rate of thrombolytic therapy as non-intervention centre STEMI patients (50% vs 57%),**
p=0.65)—including those patients admitted within 12 hours of symptom onset (67% vs 69%, p=0.99). (Table 5).

Table 5. Investigations and treatments of ST-segment-elevation myocardial infarction (STEMI) (n=101) and Non-STEMI (n=287) patients according to hospital type

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>26</td>
<td>75</td>
<td>96</td>
<td>191</td>
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</tr>
</tbody>
</table>

**Treatments**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombolysis</td>
<td>13 (50%)</td>
<td>43 (57%)</td>
<td>-</td>
<td>-</td>
<td>0.15</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>2 (7.7%)</td>
<td>1 (1.3%)</td>
<td>-</td>
<td>-</td>
<td>0.16</td>
</tr>
<tr>
<td>Aspirin</td>
<td>24 (92%)</td>
<td>64 (85%)</td>
<td>80 (83%)</td>
<td>148 (77%)</td>
<td>0.19</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>10 (38%)</td>
<td>4 (5.3%)</td>
<td>30 (31%)</td>
<td>5 (2.6%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>12 (46%)</td>
<td>21 (28%)</td>
<td>64 (67%)</td>
<td>92 (48%)*</td>
<td>0.0003</td>
</tr>
<tr>
<td>Daltaparin</td>
<td>1 (3.9%)</td>
<td>5 (7%)</td>
<td>3 (3.1%)</td>
<td>30 (16%)*</td>
<td>0.0027</td>
</tr>
<tr>
<td>UF heparin</td>
<td>7 (27%)</td>
<td>21 (28%)</td>
<td>6 (8.3%)</td>
<td>16 (10%)</td>
<td>0.24</td>
</tr>
<tr>
<td>No heparin*</td>
<td>8 (31%)</td>
<td>32 (43%)</td>
<td>29 (30%)</td>
<td>63 (33%)</td>
<td>0.30</td>
</tr>
<tr>
<td>Ticagrel</td>
<td>2 (8.0%)</td>
<td>3 (4.0%)</td>
<td>1 (1.0%)</td>
<td>5 (2.6%)</td>
<td>0.76</td>
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<tr>
<td>Eptifibatide</td>
<td>1 (3.9%)</td>
<td>1 (1.3%)</td>
<td>2 (2.0%)</td>
<td>0</td>
<td>0.06</td>
</tr>
<tr>
<td>Ticlopidimab</td>
<td>1 (3.9%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.14</td>
</tr>
</tbody>
</table>

**Investigations in hospital**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td>23 (88%)</td>
<td>66 (88%)</td>
<td>83 (86%)</td>
<td>182 (95%)*</td>
<td>0.04</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>16 (62%)</td>
<td>19 (25%)*</td>
<td>26 (27%)</td>
<td>35 (18%)</td>
<td>0.0003</td>
</tr>
<tr>
<td>Exercise test</td>
<td>3 (12%)</td>
<td>15 (20%)</td>
<td>18 (19%)</td>
<td>34 (18%)</td>
<td>0.77</td>
</tr>
<tr>
<td>Angiogram</td>
<td>12 (46%)</td>
<td>19 (25%)*</td>
<td>40 (42%)</td>
<td>31 (16%)*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No ETT or Angio</td>
<td>12 (46%)</td>
<td>45 (60%)</td>
<td>44 (46%)</td>
<td>136 (71%)*</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

**Revascularisation in hospital**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCI</td>
<td>7 (27%)</td>
<td>6 (8%)*</td>
<td>19 (20%)</td>
<td>5 (2.6%)*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>CABG</td>
<td>2 (7.7%)</td>
<td>2 (2.7%)</td>
<td>4 (4.2%)</td>
<td>4 (2.1%)</td>
<td>0.16</td>
</tr>
</tbody>
</table>

**In hospital deaths**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 (12%)</td>
<td>11 (15%)</td>
<td>2 (2.1%)</td>
<td>8 (4.2%)</td>
<td>0.25</td>
</tr>
</tbody>
</table>

**Discharge medications**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>23 (88%)</td>
<td>54 (72%)</td>
<td>71 (74%)</td>
<td>157 (82%)</td>
<td>0.61</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>7 (27%)</td>
<td>7 (9.3%)*</td>
<td>18 (19%)</td>
<td>8 (4.2%)*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>18 (69%)</td>
<td>50 (67%)</td>
<td>60 (62%)</td>
<td>117 (61%)</td>
<td>0.83</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td>13 (50%)</td>
<td>30 (40%)</td>
<td>33 (35%)</td>
<td>94 (49%)*</td>
<td>0.10</td>
</tr>
<tr>
<td>Statins</td>
<td>18 (69%)</td>
<td>40 (58%)</td>
<td>50 (52%)</td>
<td>103 (54%)</td>
<td>0.72</td>
</tr>
<tr>
<td>Fibrates</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7 (3.6%)</td>
<td>0.07</td>
</tr>
<tr>
<td>Warfarin</td>
<td>1 (3.9%)</td>
<td>2 (2.7%)</td>
<td>2 (2.1%)</td>
<td>8 (4.2%)</td>
<td>0.51</td>
</tr>
</tbody>
</table>

**Length of stay**

<table>
<thead>
<tr>
<th></th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>Non-STEMI Intervention</th>
<th>Non-STEMI Non-Intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5 (3-8)</td>
<td>5 (3-7)</td>
<td>4 (2-7)</td>
<td>4 (3-8)*</td>
<td>0.67</td>
</tr>
</tbody>
</table>

*P<0.05: Comparisons between same patient groups.
‡Comparing intervention with non-intervention groups.
*Neither enoxaparin, daltaparin or UF heparin.
**In days (median and Interquartile range).

PCI: Percutaneous coronary intervention; UF: Unfractionated; ETT: Exercise treadmill test; CABG: Coronary artery bypass grafting; Angio: Cardiac angiogram; ACE: Angiotensin converting enzyme.
Patients with non-STEMI admitted to an intervention centre were as likely to receive some type of heparin treatment (70% vs 67%, p=NS), but more likely to receive enoxaparin (67% vs 48%, p<0.05) [Table 5]. The use of glycoprotein 2b/3a inhibitors was low in patients admitted to either group of hospitals (3% vs 2.6%, p=NS).

UAP patients had similar medical management when admitted to either an intervention or a non-intervention centre (Table 6), although they were more likely to receive enoxaparin (47% vs 32%, p<0.05) at an intervention centre. The use of clopidogrel was low for patients at the non-intervention centres, probably largely reflecting different levels of PCI treatment.

Table 6. Investigations and treatments of unstable angina pectoris [UAP] (n=333) patients according to hospital type

<table>
<thead>
<tr>
<th></th>
<th>UAP Intervention</th>
<th>UAP Non-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>129</td>
<td>204</td>
</tr>
</tbody>
</table>

**Treatments in hospital**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Intervention (%)</th>
<th>Non-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>108 (89%)</td>
<td>160 (78%)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>12 (9.3%)</td>
<td>9 (4.4%)</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>61 (47%)</td>
<td>65 (32%)*</td>
</tr>
<tr>
<td>Daltafarin</td>
<td>4 (3.1%)</td>
<td>35 (17%)*</td>
</tr>
<tr>
<td>UF heparin</td>
<td>7 (5.4%)</td>
<td>15 (7.4%)</td>
</tr>
<tr>
<td>No heparin+</td>
<td>63 (49%)</td>
<td>96 (47%)</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>0</td>
<td>1 (0.5%)</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>1 (0.8%)</td>
<td>0</td>
</tr>
<tr>
<td>Abciximab</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Investigations in hospital**

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Intervention (%)</th>
<th>Non-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td>93 (72%)</td>
<td>176 (86%)*</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>25 (19%)</td>
<td>29 (14%)</td>
</tr>
<tr>
<td>Exercise test</td>
<td>40 (31%)</td>
<td>46 (23%)</td>
</tr>
<tr>
<td>Cardiac angiogram</td>
<td>29 (22%)</td>
<td>28 (13%)*</td>
</tr>
<tr>
<td>No ETT or Angio</td>
<td>67 (52%)</td>
<td>140 (69%)*</td>
</tr>
</tbody>
</table>

**Revascularisation in hospital**

<table>
<thead>
<tr>
<th>Revascularisation</th>
<th>Intervention (%)</th>
<th>Non-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCI</td>
<td>9 (7.0%)</td>
<td>4 (2.0%)*</td>
</tr>
<tr>
<td>CABG</td>
<td>6 (4.7%)</td>
<td>7 (3.4%)</td>
</tr>
</tbody>
</table>

**In hospital deaths**

<table>
<thead>
<tr>
<th>In hospital deaths</th>
<th>Intervention (%)</th>
<th>Non-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (0.8%)</td>
<td>1 (0.5%)</td>
<td></td>
</tr>
</tbody>
</table>

**Discharge medications**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Intervention (%)</th>
<th>Non-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>97 (75%)</td>
<td>169 (83%)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>9 (7.0%)</td>
<td>8 (3.9%)*</td>
</tr>
</tbody>
</table>
Beta-blockers 66 (51%) 127 (62%)*  
ACE-inhibitors 49 (38%) 79 (39%)  
Statins 59 (46%) 113 (55%)  
Fibrates 2 (1.6%) 7 (3.4%)  
Warfarin 9 (7.0%) 7 (3.4%)  

Length of stay** 2 (1-5) 2 (1-4)  

* P<0.05: Comparisons between same patient groups.  
* Neither enoxaparin, daltaparin or UF heparin.  
** Length of stay (In days and Interquartile range).

PCI was performed significantly less for all patients (n=721) with a definite ACS admitted to a non-intervention centre vs an intervention centre: (3% vs 14%, p<0.0001) [Table 4]. This finding was consistent across the 3 patient groups: STEMI (8% vs 27%, p<0.05), non-STEMI (2.6% vs 20%, p<0.05), UAP patients (2% vs 7%, p<0.05) [Tables 5 and 6]. CABG rates during hospital admission were not different between different centres: STEMI patients (2.7% vs 7.7%, p=NS), non-STEMI (2.1% vs 4.2%, p=NS), UAP patients (3.4% vs 4.7%, p=NS) for non-intervention vs intervention centres.

Discharge medications—The use of aspirin, beta-blockers, ACE-inhibitors, and statins was broadly similar for patients admitted to either a non-intervention or intervention centre. (Tables 5 and 6). However, non-STEMI patients had a higher rate of ACE-inhibitor medication at discharge from a non-intervention centre, and UAP patients had a higher rate of beta-blocker medication at discharge from a non-intervention centre. Patients presenting with a STEMI, non-STEMI, or UAP had a higher rate of use of clopidogrel at discharge from an intervention centre.

Discussion

We have shown that New Zealand ACS patients admitted to a non-intervention centre receive lower levels of investigational procedures and revascularisation than patients admitted to an intervention centre. However, medical management was generally similar at both types of centre. Of the 36 hospitals in New Zealand that admit such patients, the NZ ACS Audit Group identified 318 ACS patients admitted to an intervention centre (n=8 hospitals) and 612 ACS patients admitted to a non-intervention centre (n=28 hospitals) over 14 days. These findings have significant implications for the equitable management of ACS patients.

ST-segment-elevation myocardial infarction patients: Primary PCI is only routinely available at one intervention centre, over 24 hours and 7 days, and it is of no surprise that little primary PCI is performed on patients initially admitted to a non-intervention centre. In particular, benefit over thrombolytic therapy has been shown for STEMI patients with contraindications for thrombolysis (approximately 5–10%), and for those patients presenting more than 3 hours after symptom onset. For patients presenting less than 3 hours after symptom onset, outcomes are more similar. Across New Zealand, thrombolytic therapy is likely to remain the standard
method of reperfusion for most eligible STEMI patients. However facilitated PCI (thrombolysis followed by PCI) is being actively investigated and may be shown to be a better strategy than either therapy alone.

Approximately one quarter of patients following thrombolysis\textsuperscript{21} may need to urgently access ‘rescue angioplasty’. Advanced planning for this eventuality should be in place for the efficient transfer of these patients. At non-intervention centres, early land or air transport could allow patients to access ‘rescue angioplasty’.\textsuperscript{7,22} at the regional intervention centre. This would be a significant benefit for a small number of severely unwell STEMI patients. A cohesive referral structure should be readily achievable in New Zealand.

**Non-ST-segment-elevation myocardial infarction patients**—Non-STEMI patients admitted to either type of centre received similar rates of some type of heparin therapy. However, patients admitted to intervention centres were more likely to receive enoxaparin therapy than those admitted to a non-intervention centre.

This finding may reflect policies in smaller hospitals where the subcutaneous heparin chosen for use across the Hospital has had to satisfy the wishes of a variety of clinicians—some favouring daltaparin for surgical prophylaxis and others favouring enoxaparin for the treatment of ACS patients.\textsuperscript{53}

The European guidelines\textsuperscript{14} favour a strategy of low molecular weight heparins over unfractionated heparin, with enoxaparin being specified as preferable in the American\textsuperscript{15} and Australia and New Zealand guidelines.\textsuperscript{16} Some flexibility in pharmaceutical provision at each hospital would allow different groups of clinicians to access optimal medications for their patient group.

For non-STEMI patients, the overall use of glycoprotein 2b/3a inhibitors is low across both intervention and non-intervention centres. An improvement in this rate of treatment is important particularly for high-risk patients including those with elevated troponins and diabetes mellitus.\textsuperscript{4,24} Clinicians’ access to these medicines is often denied by local hospital pharmaceutical policies. Of the 36 hospitals admitting ACS patients, only 14 hospitals had tirofiban on their formulary, and another 2 hospitals had some non-formulary stock. A less restrictive policy appears to be needed across New Zealand. The Health Ministry may need to direct local District Health Boards to allow appropriate formulary listing.

Discharge medications are generally similar for patients leaving either type of centre but rates are low compared to internationally. Use of clopidogrel in all patient groups was more common from the intervention centres, and the use of ACE-inhibitors for non-STEMI patients, and the use of beta-blockers for UAP patients was more common from non-intervention centres.

**In-hospital investigations**—There is a disparity between patients admitted to intervention and non-intervention centres. Non-intervention-centre patients were more likely to have the basic examination of a chest X-ray, and the same (low) rates of exercise treadmill tests as intervention centre patients.

Patients admitted to a non-intervention centre had fewer echocardiograms (17% vs 26%), fewer cardiac angiograms (17% vs 30%), and more patients received neither a treadmill nor an angiogram for cardiac risk assessment (68% vs 53%). The low level of investigations undertaken on patients at New Zealand intervention centres, when
compared to international figures,\textsuperscript{19} is compounded by the even lower level of investigations for patients at the non-intervention centres (Tables 3 and 4).

**In-hospital revascularisation**—There is a major discrepancy in the rate of PCI between patients admitted to interventional and non-interventional hospitals in each category of ACS: STEMI, non-STEMI, and UAP. Patients admitted to a non-intervention centre were 3 to 8 times less likely to receive an in-hospital PCI, compared to those patients admitted to an intervention centre. Few patients underwent CABG at intervention or non-intervention centres.

Over the past 7 years, several clinical trials have demonstrated a benefit for ACS patients who were randomised to receive an ‘invasive’ rather than a ‘conservative’ management strategy.\textsuperscript{6–10} For STEMI patients, post-thrombolytic therapy, the DANAM-1 study\textsuperscript{6} revealed a significant benefit for an invasive strategy in patients who had spontaneous or inducible post-infarct ischaemia.

For non-STEMI/UAP patients, three randomised trials of invasive versus conservative management of patients\textsuperscript{8–10} have shown significant benefits of an invasive over a conservative strategy when accompanied by antithrombotic therapy: daltaparin for 4–7 days,\textsuperscript{8} unfractionated heparin, and tirofiban for 4–48 hours,\textsuperscript{9} or enoxaparin for 2–8 days.\textsuperscript{10}

In the FRISC 2 trial, with one year follow-up, for every 100 patients randomised to an invasive strategy on the background of daltaparin therapy saved 1.7 lives, prevented 2 myocardial infarctions, and prevented 20 re-admissions to hospital—as well as providing earlier and better symptom relief for patients.\textsuperscript{25} To achieve this result, 21 more PCI procedures and 15 more CABG surgery operations were required per 100 patients.

Because the hospital re-admission rate with the additional costs was so much lower, the calculated overall cost of this strategy after 1 year was only an additional 12\%\textsuperscript{,26} prompting the accompanying editorial to the health economics paper to be entitled ‘What are we waiting for?’\textsuperscript{,27} Indeed, the cost-analysis paper for TACTICS\textsuperscript{9} (which showed a 22\% reduction in the composite of death, nonfatal MI, and rehospitalisation with an ACS within 6 months) showed a cost neutral effect of an invasive strategy in combination with unfractionated heparin and tirofiban.\textsuperscript{28}

Comparison of the NZACS non-STEMI/UAP patient data with both the ‘invasive’ and ‘conservative’ rates of investigations and revascularisation of these three studies\textsuperscript{8–10} is revealing (Table 7 and Figure 1). In many cases, even the ‘conservative’ strategy in the three trials led to higher rates of investigations and revascularisation than those found in the NZACS Audit patients. However, data collection in these three trials was somewhat different to the NZACS data collection methods, where a review of current hospital practice was being audited. Hence, the discrepancies identified could be partly explained by methodology.

However, the large differences in practice do suggest that New Zealand non-STEMI/UAP ACS patients are currently not benefiting from these proven advances in treatment. New treatments including the addition of ADP receptor antagonists and drug eluting stents are likely to lead to even better patient outcomes of an invasive strategy.
Figure 1. Comparison of NZACS Audit non-STEMI/UAP patients, and their centre—with FRISC 2, TACTICS, and RITA 3 data

Data collection—The traditional method of data collection has been to code patients’ admissions at each hospital according to the current International Classification of Diseases (ICD) standard. These data are collected by the Health Information Service (NZHIS) group within the Ministry of Health, and are used to plan hospital services.
There are two major limitations to this system. Firstly, the ICD coding structure is progressively developed to reflect current diagnostic thinking, which limits the accuracy of data comparisons over time. Secondly, the quality of this data is limited by the complexities of patient presentation, and both the junior doctor and coding clerk’s interpretation of this. The current NZACS audit uses modern terminology and accurately outlines the type and number of ACS patients, who are currently requiring treatment in New Zealand. These data should prove a valuable addition to health information already collected.

Over 14 days, the audit enrolled 930 patients, which extrapolates to approximately 24,180 (930 x 26) patients over 1 year if a similar admissions policy existed throughout this time. This is likely to be a reasonable annual estimate as data was collected in the autumn, hence avoiding the seasonal difference of 38% which exists between winter and summer admissions of ischaemic heart disease patients in New Zealand. Furthermore, it is close to the figure of 27,573 given by the New Zealand Health Information Service for 1999/2000. By extrapolation, 2626 STEMI, 7462 Non-STEMI, and 8658 UAP patients would be admitted to a New Zealand hospital in 1 year.

Cost analysis—Over 14 days, 199 definite or suspected ACS patients received a cardiac angiogram, 69 patients received a PCI, and 35 patients received a CABG operation. Extrapolated over 1 year, these numbers approximate to 5174 angiograms, 1794 PCIs, and 910 CABG operations, respectively.

The approximate costs at a public hospital are: cardiac angiography $2,000, PCI $10,000, and CABG $20,000. A commitment to increase these procedures for ACS patients by 50%, (2587 more angiograms, 897 more PCI procedures, 455 more CABG operations) would equate to an undiscounted financial cost of approximately $23 million per year. However as major cost savings can be made (mainly from a reduction in hospital re-admissions of UAP/non-STEMI patients) the actual figure may be cost neutral, or there might be a saving on costs due to the current in-hospital waiting lists for angiograms and revascularisation. These make it not uncommon for patients to spend 7 or more days waiting for their treatment. Costs of transfers from non-intervention centres to intervention centres should also be considered.

Hence the additional hospitalisation costs of this current ‘rationed’ revascularisation policy could easily approach the cost of the subsequent investigation and revascularisation, and gives no advantage to the patient or the New Zealand taxpayer. Furthermore, the costs associated with the loss of earnings, and the requirement for State support of patients who can no longer actively contribute to the community, are likely to be very large. Patients who unnecessarily develop heart failure and chronic angina will only add to subsequent health costs, which will be considerable.

Study limitations—There are a number of limitations of our audit, including the fact that we did not collect data for investigations and treatment following hospitalisation. However the current data suggest that there is a lower level of investigation and revascularisation performed on ACS patients admitted to a non-intervention centre when compared to ACS patients admitted to an intervention centre and indicates the need for further study of this finding. New Zealand intervention
centres themselves perform fewer investigations and revascularisations when compared to international comparisons. \(^{19}\)

Several areas need to be considered if there is to be an improvement in the treatment of New Zealand patients with an ACS. Each regional intervention centre and referral hospital should encourage optimal in-hospital medical management and facilitate the transfer of appropriate ACS patients for invasive investigations and revascularisation.

Each regional intervention centre and non-interventional centre should review the local provision for the emergency transfer of acutely unwell ACS patients. More centres with facilities for angiography, and the expansion of Intervention Centres will be required.

**Conclusion**—We have demonstrated that a collaborative group of clinicians can perform a nation-wide audit of ACS patients. This has revealed an inequitable service provision in New Zealand. In particular, patients admitted to a non-intervention centre have lower levels of modern cardiac investigations and are markedly less likely to receive optimal revascularisation treatment. Having collected this data, the opportunity now exists for clinicians to work with the health department to develop a comprehensive nation-wide strategy for patients presenting with an ACS.

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We thank these audit leaders and assistants in the following hospitals—from north to south by region. (Patient numbers in the study are given inside brackets.) #Chairman. *Steering Committee member.

**Auckland/Northland (North Island)**

Kawakawa Hospital, Dr P Burgoyne, Ms S August (4), Whangarei Hospital, Dr B Wong, Ms K O’Keefe (30), North Shore Hospital, Auckland, Dr H Hart, Ms J Wickham (66), Auckland Hospital, Dr C Ellis*, Mr G Gamble*, Ms W Benjamin (48), Mercy Private Hospital, Auckland, Dr T Clarke, (4), Green Lane Hospital, Auckland, Assoc Prof J French*, Prof H White*, Ms B Williams (26), Ascot Private Hospital, Auckland, Dr A Maslowski, (0), Middlemore Hospital, Auckland, Dr A Ko, Dr M Lund, Dr H Oettli (40).

**Waikato/Central North Island**
Thames Hospital, Dr J Lennane, Dr Aftabuzzaman (23), Tauranga Hospital, Dr J Tisch, Dr G Porter, Ms V Watts, Ms J Braid (48), Waikato Hospital, Hamilton, Dr G Devlin*, Ms D Penney (63), Whakatane Hospital, Dr E Edwards, Ms D Garner (13), Rotorua Hospital, Dr K Logan, Ms A Morley (26), Tokoroa Hospital, Dr P Reeve, Dr F Kanan (1), Te Kuiti Hospital, Dr P Reeve, Dr J Pusupati (2), Taupo Hospital, Dr A Ludbrook (11), Gisborne Hospital, Dr F Aitcheson, Ms K Weytmans (7), Tauramunui Hospital, Dr P Reeve, Dr R Shepherd (1), New Plymouth Hospital, Dr I Ternouth (28).

Wellington/Southern North Island

Hastings Hospital, Dr R Luke, Ms J Mackenzie (66), Wanganui Hospital, Dr T Thompson, Ms K Olsen (30), Palmerston North Hospital, Dr R Shameem (27), Masterton Hospital, Dr T Matthews, Ms K Lee (12), Hutt Hospital, Dr S Mann*, Ms A Cuthbert (19), Wellington Hospital, Dr P Matisis*, Ms D Middlemitch, Ms B Scott (50), Wakefield Private Hospital, Wellington, Dr M Abernethy (2), Nelson Hospital, Dr A Hamer, Ms R Price (21), Blenheim Hospital, Dr M Heynike, Ms M Udy (16).

Christchurch, Canterbury (South Island)

Greymouth Hospital, Dr Y Al Khairulla, Ms L Skeats (9), Christchurch Hospital, Dr J Elliott*, Prof M Richards, Ms L Campbell, Ms A Alspach (131), Ashburton Hospital, Dr N Abdul-Ghaffar, Ms A Smart (11), Timaru Hospital, Dr M Hills, Ms Maria Hammond, Ms C Barker (16).

Dunedin, Otago (South Island)

Oamaru Hospital, Dr P Curzon (4), Dunstan Hospital Clyde, Dr G Nixon, Ms S Meaden (9), Dunedin Hospital, Dr MJ Williams*, Ms M McLelland (42), Invercargill Hospital, Dr C Renner, Dr A Maloney (23).

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References:


Cardiac rehabilitation services in New Zealand: access and utilisation
Fiona Doolan-Noble, Joanna Broad, Tania Riddell, Diana North

Abstract

Aim To identify factors associated with patient referral to, uptake of, and completion of cardiac rehabilitation programmes in New Zealand.

Methods Information was collected on referrals to cardiac rehabilitation during February 2002. Routinely collected hospitalisation data were obtained for men and women aged over 35 years with specified coronary episodes. The data were merged, and four predictive logistic regression models developed.

Results There were 2001 people either hospitalised or referred to cardiac rehabilitation. Of the 1696 hospitalised, 36% were referred for rehabilitation. After adjusting for ethnicity, women were less likely to be referred: odds ratio (OR)=0.72 [95% confidence interval (CI) 0.57–0.91]. With each 10-year age increase, there was a lower likelihood of referral (OR=0.74; 95%CI 0.67–0.82).

Of those people who were referred to inpatient rehabilitation, 83% were referred to an outpatient programme. Lack of access to transport was associated with reduced likelihood of referral (OR=0.44 95%; CI 0.28–0.70) and with attendance (OR=0.54; 95%CI 0.33–0.88). Those who had previously attended a cardiac rehabilitation programme were significantly more likely to attend, and compared to those aged 65 to 74 years, those older or younger were less likely to complete the programme. Some associations with deprivation were found, but none with ethnicity.

Conclusion This study demonstrated considerable scope for improvement in referral to, uptake of and completion of cardiac rehabilitation programmes in New Zealand. It highlighted the need to improve referral processes, promotion, provision, delivery and monitoring of cardiac rehabilitation services.

Comprehensive multifactorial cardiac rehabilitation following a myocardial infarction (MI) has been shown to reduce mortality and morbidity, and to improve quality of life.1–5 Benefits have also been shown for people following percutaneous transluminal coronary angioplasty (PTCA), coronary artery bypass grafting (CABG), and those with stable angina and chronic heart failure.6,7 However international research has demonstrated generally low rates of referral and participation to cardiac rehabilitation.8–10 Referral and participation have been found to be an issue for the elderly, women, ethnic minorities and those of lower socioeconomic status.11–16

In New Zealand, an audit of a cardiac rehabilitation centre showed that over 25% of patients admitted to the coronary care unit or high dependency ward were not referred to cardiac rehabilitation, and 56% of eligible patients did not attend the programme.17

This study investigates factors associated with referral and utilisation of cardiac rehabilitation programmes in New Zealand. It is the second of a two part national
audit designed to benchmark service provision prior to the launch of the new guidelines for cardiac rehabilitation in New Zealand.18

Methods

Data collection—The audit aimed to include all cardiac patients who were hospitalised during February 2002 and met the following inclusion criteria: men and women aged 35 and over admitted to a public hospital in New Zealand with MI, acute coronary syndrome, PTCA, CABG, or heart failure.

Thirty of the 38 cardiac rehabilitation centres agreed to take part in the audit: four were in recess, two were serviced by larger nearby centres (part of the audit), one centre agreed too late to participate and one was a private hospital.

Data was collected by cardiac rehabilitation nurses using a form which had been pre-tested by cardiac rehabilitation nurses to ensure clarity and acceptability, and its content and format modified following responses received. Audit forms were posted to all cardiac rehabilitation centres and one of the lead investigators acted as a point of contact to resolve queries.

Centre staff were asked to initiate an audit form on receipt of each referral to their programme, and to retain it until the patient had either completed the phase II programme or a period of 16 weeks had elapsed. The completed forms were then returned to the National Heart Foundation for data entry and analysis. No patient names were included, but to enable matching against the national hospital discharge records, patients were identified by their unique NHI number, sex, birthdate, and residential address.

The cardiac rehabilitation programme audit form sought basic information on: patient demographics and ethnicity, admitting condition, source of referral, referral to inpatient cardiac rehabilitation (phase I), referral and utilisation of the outpatient cardiac rehabilitation programme (phase II), potential barriers to attending phase II, and factors which influenced completion of phase II.

Potential barriers included employment status (in full-time or part-time paid work, retired, or other), physical or mental disability, no private telephone, living alone, lack of access to transport, not receiving a written invitation, whether the person had previously attended a phase II programme, and whether English was their first language. Completion of the phase II programme was deemed to be attendance at four or more sessions during a period of 12 weeks from the date of hospital discharge.

A data set was provided by the New Zealand Health Information Service (NZHIS) that included all patients with a New Zealand hospital stay during February 2002—with any one or more of the following International Classification of Diseases (ICD 9) codes as a discharge diagnosis: 402, 410.1–410.9, 413, 422, 428, 429.1, or 429.3—or one of the procedures: 36.01, 36.02, 36.06, 36.07, 36.11–36.14.

Approval for the study was obtained from the Auckland Ethics Committee on behalf of the other 12 regional ethics committees in New Zealand.

Data management of audit forms—Audit forms were coded as described below, entered into a database, and then merged with hospitalisation data.

Ethnicity provided for multiple responses to nine categories including ‘other’. These were converted to a single ethnicity variable using the following rule: if any mention of Maori, then code as Maori; if no mention of Maori but any mention of Pacific Island ethnicity, then code as Pacific; if no mention of Maori or Pacific but any mention of Asian ethnicity, then code as Asian; otherwise, code as ‘NZ European or other’.19

The main medical condition provided for classification into one (only) of the following five categories: myocardial infarction, acute coronary syndrome/unstable angina, post-CABG, post-PTCA (with or without stent), and heart failure. Although terminology around diagnosis is changing, at the time of the study, some hospitals were using myocardial infarction and some were using acute coronary syndrome; hence the need to provide for both. Source of referral to cardiac rehabilitation provided for one only of the following eight categories: medical team, ward nurse, cardiac rehabilitation nurse, ward nurse, other hospital, general practitioner, practice nurse, patient, and other health professional (for example physiotherapist). These were coded as categorical variables.

Utilisation and outcome data included whether the patient was referred to Phase II; and if so, whether that included a written invitation, whether they attended, and how many visits they attended.
In addition to the unique identifying number of the patient (NHI), the NZHIS data included sex, date of birth, discharge diagnoses and procedures, and latest residential area coded using the NZ Census area unit. By matching against 1996 census area unit, the NZ Deprivation Index (NZDep96) was obtained. This is a score developed from New Zealand census data to indicate economic and social deprivation of neighbourhoods, based on several indicators of socioeconomic deprivation.

Validity of NHI numbers recorded on the audit forms was checked and corrected (where possible) through the centres. The two sources of data were merged based on NHI number. For those individuals where there was an audit form and no corresponding hospital record, the identifiers were sent to NHI to establish any recent discharge with a cardiac diagnosis, even if not coded as its primary diagnosis. When this process could not match a hospital discharge record, NZHIS provided the NZDep96 code for the latest address known for the patient.

Data management of NZHIS records—NZHIS discharge records, which were not associated with an audit form, were coded according to the discharge codes into the same medical categories as the audit form (MI, PTCA, CABG, etc). Where more than one condition was coded, a priority system was established—so MI was coded as a priority before acute coronary syndrome/unstable angina, ahead of post coronary artery bypass grafting, ahead of post angioplasty, and finally ahead of heart failure. Likewise, ethnicity was classified for NZHIS data as for the audit form.

NZDep96 codes were grouped into quintiles (coded Q1–Q5) and a separate category (Q6) made for those for whom no NZDep96 quintile had been allocated (for example, there were too few people in the mesh block, indistinguishable address on the census record, or audit form not matched with NHI record).

Patients with a residential address or other details which indicated they lived overseas, or where the hospital discharge record indicated they had died during hospital admission or were day patients, were excluded from analyses.

Statistical analysis—Because of the known association between variables (particularly age, sex, and ethnicity), no univariate analysis are presented. Instead, logistic regression models were used to adjust for the many competing potential predictors.

Four predictive models were developed to identify:

1. Characteristics associated with referral to cardiac rehabilitation.
2. Factors associated with referral to Phase II in those patients referred to cardiac rehabilitation.
3. Factors associated with attending Phase II if patients were referred.
4. Factors associated with completing a cardiac rehabilitation programme if referred to Phase II.

In each predictive model, sex, age, ethnicity, and deprivation were included. Other variables (available at eligibility) were made available and were progressively eliminated according to size of estimate (closest to unity) and p-value (distance from zero) until only significant variables remained in the model. Referent groups were generally the ‘No’ category—but to distinguish associations for either high or low deprivation areas for NZDep96, the referent group was the central group (quintile 3).

In all models, age was initially entered as continuous and regression co-efficients calculated for each 10 year increase. In the fourth model, age as a continuous variable was not significant, and, instead, age was entered as a single categorical variable (under 65 years, 65–74, and over 75 years) with the central group as the referent group.

Once the most parsimonious model was produced, each variable was systematically reinserted to check whether it would improve the model fit in combination with the variables already present.

To assess the correlation between Maori ethnicity and NZDep decile, a Cochran–Armitage Trend test was conducted with all available records. Throughout, the SAS 8.02 package was used for analysis, and p values of 0.05 or less were taken as statistically significant.

Results

2266 people were found for the specified admission period—comprising 1956 with hospital records and 948 with a cardiac rehabilitation audit form. 2001 patients remained after excluding patients who died during admission (91), day patients (154), and overseas residents (21). Of these 2001 patients, 1085 had a hospital record but no
audit form, 611 had both an audit form and a hospital record, and 305 had an audit form only. The data matching process and study flowchart are shown in Figure 1.

**Figure 1. Flowchart of cardiac rehabilitation audit**

Of the 1085 eligible patients with a hospital record and no audit form, 52.1% were men and 47.9% were female (Table 1). The majority (74.4%) were between the ages of 55–84 years, and 13.9% were aged over 85 years. The ethnic composition of this group of patients was ‘New Zealand Europeans and others’ (82.2%), Maori (11.6%), Asian (4.1%), and Pacific Islanders (2.1%). Heart failure and myocardial infarction were the main admitting conditions—35.2% and 34.5% respectively.

Of the 916 eligible patients with cardiac rehabilitation audit forms; 64.6% were men and 34.9% were female. Most (74.8%) were between the ages of 55–84 years, with 3.3% aged over 85 years. ‘New Zealand Europeans and others’ were the largest ethnic
group (84.1%), followed by Maori (9.9%), Asians (2.5%), and Pacific Islanders (3.3%). Myocardial infarction (45.3%) and unstable angina (35.4%) were the main cardiac conditions (Table 1).

Table 1. Characteristics of cardiac hospitalisations and rehabilitation patients

<table>
<thead>
<tr>
<th></th>
<th>Unmatched hospitalised patients</th>
<th>Matched CRP patients</th>
<th>Unmatched CRP patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=1085 (54.2%)</td>
<td>N=611 (30.5%)</td>
<td>N=305 (15.2%)</td>
</tr>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>565</td>
<td>400</td>
<td>192</td>
</tr>
<tr>
<td>Women</td>
<td>520</td>
<td>211</td>
<td>109</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15–44</td>
<td>36</td>
<td>28</td>
<td>22</td>
</tr>
<tr>
<td>45–54</td>
<td>89</td>
<td>89</td>
<td>48</td>
</tr>
<tr>
<td>55–64</td>
<td>177</td>
<td>151</td>
<td>79</td>
</tr>
<tr>
<td>65–74</td>
<td>302</td>
<td>203</td>
<td>89</td>
</tr>
<tr>
<td>75–84</td>
<td>329</td>
<td>120</td>
<td>44</td>
</tr>
<tr>
<td>85+</td>
<td>151</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European and other</td>
<td>822</td>
<td>508</td>
<td>246</td>
</tr>
<tr>
<td>Maori</td>
<td>126</td>
<td>59</td>
<td>32</td>
</tr>
<tr>
<td>Asian</td>
<td>45</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>23</td>
<td>25</td>
<td>6</td>
</tr>
<tr>
<td>Not specified</td>
<td>69</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Main cardiac condition*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>372</td>
<td>361</td>
<td>54</td>
</tr>
<tr>
<td>Unstable angina &amp; ACS</td>
<td>277</td>
<td>202</td>
<td>123</td>
</tr>
<tr>
<td>Post CABG</td>
<td>30</td>
<td>19</td>
<td>14</td>
</tr>
<tr>
<td>Post-PTCA</td>
<td>24</td>
<td>11</td>
<td>59</td>
</tr>
<tr>
<td>Heart failure</td>
<td>382</td>
<td>18</td>
<td>27</td>
</tr>
<tr>
<td>NZDep96</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quintile 1 (least deprived)</td>
<td>197</td>
<td>139</td>
<td>60</td>
</tr>
<tr>
<td>Quintile 2</td>
<td>199</td>
<td>125</td>
<td>52</td>
</tr>
<tr>
<td>Quintile 3</td>
<td>186</td>
<td>114</td>
<td>42</td>
</tr>
<tr>
<td>Quintile 4</td>
<td>188</td>
<td>109</td>
<td>41</td>
</tr>
<tr>
<td>Quintile 5 (most deprived)</td>
<td>214</td>
<td>108</td>
<td>48</td>
</tr>
<tr>
<td>Not available</td>
<td>101</td>
<td>16</td>
<td>62</td>
</tr>
</tbody>
</table>

*Only one choice permitted. CRP=cardiac rehabilitation programme; ACS: acute coronary syndrome; CABG: coronary artery bypass grafting; PTCA: percutaneous transluminal coronary angioplasty; Note: Number may not always add to the total due to missing data.

Characteristics if patients in relation to referral to and attendance at cardiac rehabilitation are shown in Table 2.

The factors most associated with being referred to cardiac rehabilitation following a hospital admission with a cardiac condition are shown in Table 3. Being female (OR=0.72; 95%CI 0.57–0.91), increasing age (OR=0.74; 95%CI 0.67–0.82), or having a diagnosis of heart failure (OR=0.11; 95%CI 0.06–0.19) were all independently associated negatively with referral to the cardiac rehabilitation team.
(Phase 1) after adjusting for ethnicity and deprivation quintile. Compared to those with unstable angina or acute coronary syndrome, revascularisation and MI patients were much more likely to be referred for cardiac rehabilitation. No association with ethnicity was evident.

Table 2. Characteristics of patients at different stages of cardiac rehabilitation

<table>
<thead>
<tr>
<th></th>
<th>Referred to</th>
<th>Attended</th>
<th>Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase I</td>
<td>Phase II</td>
<td>Phase II</td>
</tr>
<tr>
<td>n=916</td>
<td>n=767</td>
<td>n=319</td>
<td>n=198</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Employed:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In paid work</td>
<td>31</td>
<td>34</td>
<td>30</td>
</tr>
<tr>
<td>Retired</td>
<td>56</td>
<td>53</td>
<td>57</td>
</tr>
<tr>
<td>Not retired or in paid work</td>
<td>12</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>First language is English</td>
<td>87</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>Has physical or mental disability</td>
<td>13</td>
<td>13</td>
<td>12</td>
</tr>
<tr>
<td>Lives alone</td>
<td>21</td>
<td>21</td>
<td>18</td>
</tr>
<tr>
<td>No private phone</td>
<td>18</td>
<td>18</td>
<td>17</td>
</tr>
<tr>
<td>Transport:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Has access to private transport</td>
<td>83</td>
<td>89</td>
<td>89</td>
</tr>
<tr>
<td>Programme provides transport</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>No access to private or programme transport</td>
<td>15</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Written invitation to Phase II</td>
<td>71</td>
<td>86</td>
<td>88</td>
</tr>
<tr>
<td>Attended Phase II previously</td>
<td>35</td>
<td>16</td>
<td>14</td>
</tr>
</tbody>
</table>

Table 4a shows that referral to phase II was negatively associated with increasing age (OR=0.76; 95%CI 0.63–0.90) and no access to transport (either private or programme) (OR=0.44; 95%CI 0.28–0.70). Those post-revascularization (OR=4.30; 95%CI 2.51–7.35) and post-myocardial infarction (OR 2.72; 95%CI 0.63–0.90) were more likely to receive referral to phase II cardiac rehabilitation.

Attendance at phase II was significantly associated with a person being post myocardial infarction (OR=1.56; 95%CI 1.03–2.35) or having previously attended the programme (OR=2.38; 95%CI 1.46–3.91). Lack of access to either private or programme transport was negatively associated with attendance (OR=0.54; 95%CI 0.33–0.88) (Table 4b).

Patients were less likely to complete the phase II cardiac rehabilitation programme (four or more sessions) if they were aged under 65 years (OR=0.60; 95%CI 0.41–0.87) or over 75 years (OR=0.55; 95%CI 0.33–0.92) or were living in middle quintile...
Maori ethnicity and high NZDep decile were highly associated in the trend test ($z$-score=5.97, $p<0.0001$), showing evidence of increasing proportions of Maori in deciles that are more deprived.

### Table 3. Factors associated with referral of patients hospitalised with heart condition(s) to phase 1 cardiac rehabilitation

<table>
<thead>
<tr>
<th>Medical condition(s)</th>
<th>Adjusted OR* (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable angina or ACS</td>
<td>1.00 -</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Heart failure only</td>
<td>0.11 (0.06, 0.19)</td>
<td></td>
</tr>
<tr>
<td>Post revascularisation</td>
<td>2.66 (1.91, 3.70)</td>
<td></td>
</tr>
<tr>
<td>(PTCA or CABG)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1.68 (1.25, 2.24)</td>
<td></td>
</tr>
<tr>
<td>Age (10 year increase)</td>
<td>0.74 (0.67, 0.82)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Women</td>
<td>0.72 (0.57, 0.91)</td>
<td>0.006</td>
</tr>
</tbody>
</table>

**Notes**

Only people with a relevant hospital admission and known ethnicity were eligible for this analysis.

*Adjusted for ethnicity and decile of NZ Deprivation index.

### Discussion

This prospective audit aimed to identify factors significant in the referral of patients to cardiac rehabilitation, their uptake of the intervention and their completion of the programme in New Zealand. Having no access to transport, being a woman, being older, and a diagnosis of heart failure were all significantly related to a reduced likelihood of referral to either phase I or phase II. Previous attendance at a programme and having a diagnosis of myocardial infarction were both predictive of attendance at phase II. No structural factors were found which influenced completion of the programme but age and coming from the middle deprivation quintiles were determinants of non-completion.

Limitations in the study design may have had some impact on accuracy of the results—the short duration over which the audit was conducted, and consequent small sample sizes of particular groups, may have resulted in measurement error. This audit could not consider the potential effects coexisting co-morbidities may have had on referral to and uptake of cardiac rehabilitation. The absence of hospitalisation records for a substantial portion of those with referrals to cardiac rehabilitation during the period of the audit suggests that these patients may have been referred from other areas; for example, outpatient clinics or primary care. Incomplete data on the audit forms will account for some of the non-matching between the audit forms and the
NZHIS data. Using busy clinical staff to collect data may have led to incomplete audit data, although possible errors or omissions in completion of forms were avoided by contacting staff for clarification.

Table 4. Models showing factors associated with various outcomes following referral to phase I cardiac rehabilitation

a) Referral to Phase II, given referral to Phase I

<table>
<thead>
<tr>
<th>Medical condition(s)</th>
<th>Adjusted OR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable angina or ACS</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>Post revascularisation (PTCA or CABG)</td>
<td>4.30 (2.51, 7.35)</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2.72 (0.63, 0.90)</td>
<td></td>
</tr>
<tr>
<td>Age (10 year increase)</td>
<td>0.76 (0.63, 0.90)</td>
<td>0.002</td>
</tr>
<tr>
<td>Transport:</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Has access to private transport</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>No access to private or programme transport</td>
<td>0.44 (0.28, 0.70)</td>
<td></td>
</tr>
</tbody>
</table>

b) Attendance at Phase II, given referral to Phase I

<table>
<thead>
<tr>
<th>Medical condition(s)</th>
<th>Adjusted OR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable angina or ACS</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1.56 (1.03, 2.35)</td>
<td></td>
</tr>
<tr>
<td>Previously attended Phase II</td>
<td>2.38 (1.46, 3.91)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Transport:</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Has access to private transport</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>No access to private or programme transport</td>
<td>0.54 (0.33, 0.88)</td>
<td></td>
</tr>
</tbody>
</table>

(c) Completion of Phase II, given referral to Phase II

<table>
<thead>
<tr>
<th>Age:</th>
<th>Adjusted OR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>65-74 years</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>Over 75 years</td>
<td>0.55 (0.33, 0.92)</td>
<td></td>
</tr>
<tr>
<td>Under 65 years</td>
<td>0.60 (0.41, 0.87)</td>
<td></td>
</tr>
<tr>
<td>Deprivation:</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>Quintile 1 (least deprived)</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>Quintile 3</td>
<td>0.42 (0.23, 0.76)</td>
<td></td>
</tr>
<tr>
<td>Quintile 4</td>
<td>0.49 (0.28, 0.87)</td>
<td></td>
</tr>
</tbody>
</table>

Notes:
All people with a CRP audit form and known ethnicity were eligible for all these analyses. Adjusted for age, gender, ethnicity, and decile of NZ Deprivation index if not shown.
International research has demonstrated lower referral to cardiac rehabilitation programmes for women,\textsuperscript{14,22–24} with women being 20\% less likely to be referred.\textsuperscript{22} This audit demonstrated a 28\% lower referral rate for women after adjustment for age, medical condition, ethnicity and deprivation score. Cardiac rehabilitation programmes have proven benefits to women. Women who experience a cardiac event tend to have poorer psychological adjustment, are older, more likely to have other co-morbidities, and be more likely to be retired or living alone.\textsuperscript{22} Further efforts are required to promote cardiac rehabilitation programmes for women.

The age distribution in Table 1 demonstrates a cardiac rehabilitation population predominantly over 65 years. It is projected the number of people aged 65 years and over will double by 2051.\textsuperscript{25} This is a population susceptible to heart failure. Table 1 also shows that 35.2\% of those patients with hospital records only had a diagnosis of heart failure compared to those patients with matched and unmatched audit forms—2.9\% and 8.9\% respectively.

Despite the documented benefits of cardiac rehabilitation for those with heart failure; improvement in disease related symptoms, quality of life and clinical outcomes\textsuperscript{26} it remains an under utilised intervention for this patient group with pharmacology providing the mainstay of treatment. The audit that formed the first part of this study\textsuperscript{27} showed that 82\% (N=33) of responding cardiac rehabilitation programmes stated they provided a service for heart failure patients. With the heart failure nurse specialist a rare commodity in New Zealand multidisciplinary cardiac rehabilitation teams are well suited to accept the challenge of providing individualised follow up for people with heart failure and their families.

It appears that programmes catered comparatively well for those aged 65–74 years: those younger than 65 years and over 74 years are less likely to complete. Although employment status did not reach statistical significance, it is possible that part of the observed lower use among those younger than 65 years is related to their employment. There is a need for programmes to consider outcomes of importance to the elderly such as disability, independence and health-related quality of life.\textsuperscript{28}

The ‘U’-shaped relationship of deprivation quintile with completion of phase II was unexpected—those in quintiles 3 and 4 were less likely to complete compared to those in the least deprived quintile. Socioeconomic inequalities in cardiovascular disease in New Zealand have become wider and as a result cardiovascular disease is increasingly associated with disadvantage.\textsuperscript{29} Indeed, cardiac rehabilitation staff need to be mindful of the impact a person’s level of deprivation may have on their ability to attend the programme. Information on the socioeconomic status of all those referred to CRPs should be routinely collected and monitored to establish how responsive the programme is to those from lower deprivation levels.

There was no evidence in any of the models for an association with ethnicity that was independent of age and deprivation. For the models shown in Table 4, this may possibly be due to the low numbers of Maori and Pacific Island people included. In this study, there was a high correlation between deprivation and Maori ethnicity as demonstrated elsewhere.\textsuperscript{50} Studies in the United States have consistently found that while both ethnicity and socioeconomic position predict access to care, ethnicity is the major determinant.\textsuperscript{50} NZDep96 is an area based measure of deprivation and may not be a good proxy measure of either an individual’s level of deprivation or their...
socioeconomic status, yet in this study it accounts for more variation in all models than ethnicity.\textsuperscript{20}

The significant relationship found between access to transport and use of cardiac rehabilitation services, both in rate of referral and in attendance confirms findings in several other studies.\textsuperscript{31–33} One potential strategy to overcome this barrier is to take rehabilitation to the person through home- or community-based cardiac rehabilitation services.\textsuperscript{34}

The study did have limitations however its strength lies in the large amount of data available for analysis and the national perspective taken. For a low cost it provided benchmarking data for the service and highlighted the need to undertake a longitudinal study of this area to examine in more detail the issues related to referral to, uptake of and utilisation of cardiac rehabilitation services from a quantitative and qualitative perspective.

This study demonstrates there is considerable scope for improvement in referral to, uptake of and completion of cardiac rehabilitation programmes in New Zealand. It highlights the need to improve referral processes, to promote the benefits of cardiac rehabilitation for certain groups, namely women, those at each end of the age spectrum, those with heart failure and those living in areas of greater deprivation. It is important to design and implement innovative and more effective ways of delivering the service and to improve monitoring of cardiac rehabilitation services.

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**Acknowledgements:** This work was supported by funding from the National Heart Foundation of New Zealand. We would like to thank all the cardiac rehabilitation nurses who completed the audit forms; Dr Dale Bramley, Dr Sue Wells, and Professor Norman Sharpe for their comments on earlier drafts; and Elizabeth Robinson for her statistical advice.

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**References:**


Exponential increase in clinical use of plasma brain natriuretic peptide (BNP) assays

Timothy Yandle, Steve Fisher, John Livesey, Eric Espiner, Mark Richards, Gary Nicholls

Abstract

Aims To document the number of requests by clinicians for plasma brain natriuretic peptide (BNP) measurements and to define which groups of practitioners have made use of the assay over the 7 years since it became available for clinical use.

Methods We reviewed the number and source of requests by clinicians for measurements of plasma BNP in the Christchurch area (from 1995—when the assay became available, until July 2002).

Results There was an exponential increase in requests for BNP measurements over the 7-year period (1995–2002). Of the 11,308 samples analysed, 47% came from hospital inpatients, 25.9% from patients in general practice, and 14% from hospital emergency departments.

Conclusions There has been a rapidly increasing uptake of the assay for plasma BNP by both hospital and primary care clinicians in the Christchurch area.

The peptide hormone brain natriuretic peptide (BNP) is released from the heart into the circulation where it acts to counterbalance contrary systems, especially the renin-angiotensin system, thereby protecting against sodium and volume overload.

Research reports suggest that plasma BNP levels might be potentially useful to the clinician under a number of circumstances—for example: in the differential diagnosis of dyspnoea; in providing a prognostic index after myocardial infarction or in established heart failure; as a guide to pharmacotherapy in patients with heart failure; and in the detection of left ventricular dysfunction in the general population.1

Whether this experience from research studies will be translated into routine clinical practice, however, is not known. Accordingly, we reviewed the number and source of requests by clinicians for measurements of plasma BNP in Christchurch city and surrounds since the assay became available for clinical use in 1995.

Methods

After our report on its application for the differential diagnosis of dyspnoea,2 a radioimmunoassay for measuring BNP in plasma, validated in our research laboratory in the early 1990s,3 became available in 1995 to clinicians in the city of Christchurch and surrounds (total drainage population 500,000).

We searched the database of the Canterbury District Health Board Laboratory for requests from clinicians to measure plasma BNP between February 1995 and July 2002. Ours was the only laboratory measuring the peptide in the country over this period of time. Assay samples for research purposes could be identified and were excluded. The data were further dissected according to whether samples came from general practice (primary care) or from clinicians working in the city public (Christchurch or Princess Margaret) hospitals, or from the public hospital in the town of Ashburton (87km south of Christchurch).
Results

Monthly and annual assay numbers are shown in Figure 1, and the source of samples is given in Table 1. There was an exponential increase in the number of clinical samples assayed over the 7-year period (Figure 1).

Of the 11,308 samples analysed, 47% were from inpatients in the public hospitals—comprising 29.4% from the general medicine service (which includes geriatric wards), 11.8% from cardiology or respiratory services, and 4.2% from surgical wards. General practitioners requested 25.9% of tests and hospital emergency departments requested 14% (Table 1).

Table 1. Source of requests for measurement of Plasma BNP

<table>
<thead>
<tr>
<th>Source</th>
<th>Number of samples</th>
<th>Percentage of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital General Medicine inpatient service</td>
<td>3,327</td>
<td>29.4%</td>
</tr>
<tr>
<td>General Practice</td>
<td>2,934</td>
<td>25.9%</td>
</tr>
<tr>
<td>Hospital Emergency Department</td>
<td>1,585</td>
<td>14%</td>
</tr>
<tr>
<td>Cardiology/Respiratory hospital inpatient service</td>
<td>1,336</td>
<td>11.8%</td>
</tr>
<tr>
<td>Hospital outpatient service (general)</td>
<td>857</td>
<td>7.6%</td>
</tr>
<tr>
<td>Hospital Surgical inpatient services</td>
<td>472</td>
<td>4.2%</td>
</tr>
<tr>
<td>Cardiology/Respiratory hospital outpatient service</td>
<td>386</td>
<td>3.4%</td>
</tr>
<tr>
<td>Miscellaneous specialty hospital inpatient services</td>
<td>174</td>
<td>1.5%</td>
</tr>
<tr>
<td>Out of province</td>
<td>187</td>
<td>1.7%</td>
</tr>
<tr>
<td>Unknown</td>
<td>50</td>
<td>0.5%</td>
</tr>
<tr>
<td>Total</td>
<td>11,308</td>
<td>100%</td>
</tr>
</tbody>
</table>

Figure 1. Number of BNP samples measured for clinical purposes per month (columns) and per year (circles) in Christchurch 1995–2002

Discussion

BNP, a peptide hormone produced largely by the cardiac ventricles, is released into the bloodstream where it circulates normally in lower picomolar concentrations.
Secretion of BNP is increased primarily by stretch of ventricular myocardium, although many hormonal and biochemical factors also play a modulatory role. Along with atrial natriuretic peptide (ANP), it plays a protective role against sodium and fluid overload and as a counterpoise to the renin-angiotensin system which protects against sodium and fluid depletion.

As noted already, considerable enthusiasm has been expressed in the research literature regarding the potential clinical usefulness of plasma levels of BNP, or its amino-terminal peptide, in, for example, diagnosing heart failure in dyspnoeic patients, in guiding drug treatment for cardiac failure, and as a prognostic index in acute coronary syndromes or after myocardial infarction.

Our survey indicates that, in the case of Christchurch and surrounds, the uptake of BNP assays by clinicians has increased exponentially since the service became available in 1995. Christchurch has been at the forefront of research into some aspects of BNP and, inevitably, clinicians were informed early of its potential use in routine clinical practice through local scientific publications, lectures, and seminars.

The incremental uptake of the assay by clinicians in the Christchurch region occurred despite the fact that test results were forthcoming only once per week in 1995 (but increasing to twice weekly in July 1996 and thrice weekly in January 1998). Assays are now run twice daily. With a point-of-care technique (Biosite Triage BNP Test), results can be available within 15 minutes.

Our study does not provide information on how the BNP assay was used in clinical practice. We suspect that the majority of requests were to assist in the differentiation of cardiac, from pulmonary disease as a cause of dyspnoea or peripheral oedema. More recently, guidance regarding the intensity of drug treatment for established heart failure is likely to have contributed to the increasing demand.

As with any new test, questions of cost arise. The price for a single clinical measurement of plasma BNP in Christchurch has, over the last few years, varied between NZ$50–60.70. Reagent prices for the Biosite Triage BNP assay vary, depending on the number of samples being assayed, from NZ$20–40 per sample, and the Biosite Triage meter itself retails at approximately NZ$8,000.

Although these costs are considerable, they need to be set against the overall costs in the diagnosis and management of heart failure. We are not aware of a cost-benefit analysis of BNP-guided treatment of heart failure, but a Swiss study documented that use of the rapid Biosite assay in the differential diagnosis of acutely dyspnoeic patients in the emergency department was associated with a sizeable cost saving (mean US$1,854 per patient) compared with usual evaluation and management in the absence of the BNP assay.

We suggest that BNP assays will show increasing uptake in routine clinical practice elsewhere—both from general practice and in the hospital setting. This is likely since the prevalence of heart failure is increasing yet the diagnosis often remains difficult. Furthermore, plasma BNP (or its amino-terminal peptide) provides objective guidance for drug therapy. BNP levels might prove clinically useful under a variety of circumstances not mentioned already in this paper—such as in intensive care or coronary care units to assess the haemodynamic response to therapy, and in the
evaluation of cardiac function for patients with valvular heart disease.\textsuperscript{10,11} If so, clinical use of BNP assays may exceed current expectations.

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**References:**

Guidelines for pre-hospital administration of fibrinolytic therapy by New Zealand general practitioners

The Pre-Hospital Fibrinolysis Guidelines Working Party

Introduction

These guidelines have been developed in consultation with the Ministry of Health to ensure equity of access to fibrinolytic therapy (formerly known as thrombolytic therapy) throughout New Zealand. There are many rural areas in New Zealand where patients with acute myocardial infarction currently do not receive timely fibrinolytic therapy because of the time and distance involved in transporting them to hospital.

The guidelines have been written after wide consultation with various groups and a series of discussions between the members of the Pre-Hospital Fibrinolysis Guidelines Working Party (see ‘Author information’ at the end of this article). The search strategy for evidence included a Medline search, and the recommendations listed in the guidelines are based on the evidence and on the consensus of the Working Party.

The levels of evidence are graded according to the Scottish Intercollegiate Guidelines Network (SIGN) method¹ (Table 1) to differentiate between those based on strong evidence and those based on weak evidence. The grading does not signify the importance of the recommendation, but rather the strength of the supporting evidence.

Table 1. Scottish Intercollegiate Guidelines Network (SIGN) revised grading system¹

<table>
<thead>
<tr>
<th>Levels of evidence</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1++</td>
<td>High-quality meta-analyses, systematic reviews of randomised controlled trials, or randomised controlled trials with a very low risk of bias.</td>
</tr>
<tr>
<td>1+</td>
<td>Well conducted meta-analyses, systematic reviews of randomised controlled trials, or randomised controlled trials with a low risk of bias.</td>
</tr>
<tr>
<td>1-</td>
<td>Meta-analyses, systematic reviews of randomised controlled trials, or randomised controlled trials with a high risk of bias.</td>
</tr>
<tr>
<td>2++</td>
<td>High-quality systematic reviews of case-control or cohort studies, high-quality case-control or cohort studies with a very low risk of confounding, bias or chance, and a high probability that the relationship is causal</td>
</tr>
<tr>
<td>2+</td>
<td>Well-conducted case control or cohort studies with a low risk of confounding, bias or chance, and a moderate probability that the relationship is causal.</td>
</tr>
<tr>
<td>2-</td>
<td>Case control or cohort studies with a high risk of confounding, bias or chance, and a significant risk that the relationship is not causal.</td>
</tr>
<tr>
<td>3</td>
<td>Non-analytical studies, eg. case reports or case series</td>
</tr>
<tr>
<td>4</td>
<td>Expert opinion.</td>
</tr>
</tbody>
</table>

Grades of recommendation

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At least one meta-analysis, systematic review or randomised controlled trial rated as 1++, and directly applicable to the target population; or a systematic review of randomised controlled trials or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results.</td>
</tr>
<tr>
<td>B</td>
<td>A body of evidence including studies rated as 2++, directly applicable to the target population.</td>
</tr>
</tbody>
</table>
and demonstrating overall consistency of results; or extrapolated evidence from studies rated as 1++ or 1+.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C</strong></td>
<td>A body of evidence including studies rated as 2+, directly applicable to the target population, and demonstrating overall consistency of results; or extrapolated evidence from studies rated as 2++.</td>
</tr>
<tr>
<td><strong>D</strong></td>
<td>Evidence level 3 or 4; or extrapolated evidence from studies rated as 2+.</td>
</tr>
</tbody>
</table>

Over the past decade, it has been shown that fibrinolysis reduces mortality in patients suffering acute myocardial infarction (Level of evidence: 1++).\(^2\text{-}^6\) However, the mortality reduction attenuates markedly the longer that treatment is delayed after the onset of infarction (1++).\(^3\text{-}^5,^7\) Several studies have demonstrated the feasibility and safety of pre-hospital assessment and initiation of fibrinolysis (1++).\(^2,^7\text{-}^17\) The greater the distance from a hospital with fibrinolytic facilities, the greater the potential for myocardial salvage by pre-hospital fibrinolysis. This is because myocyte necrosis progresses rapidly over time,\(^18\) and many more lives are saved when patients are treated with fibrinolysis very early in the course of infarction than when they are treated later.

A retrospective analysis of the Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI-1) Study revealed that patients who received treatment within 1 hour had a 51% reduction in mortality at 21 days (2+).\(^4\) This was a hypothesis-generating analysis and must be interpreted cautiously, but it does demonstrate the potential benefit of very early treatment.

In the Fibrinolytic Therapy Trialists’ (FTT) overview of 58,600 randomised patients, the calculated mortality reduction for every hour of delay avoided was 1.6 lives saved per 1,000 patients treated with fibrinolysis, with a 30% mortality reduction at 1 hour, 25% at 2 to 3 hours, and 18% at 4 to 6 hours (1++).\(^5\) Another analysis, which excluded some of the studies in the FTT overview because they included patients with unstable angina, reported even greater mortality reductions with earlier treatment (48% at 1 hour, 44% at 2 hours, and 20% after 3 hours) (1+).\(^7\)
Figure 1. Results of randomised trials comparing pre-hospital and in-hospital fibrinolysis, showing the time saved by pre-hospital fibrinolysis and the relative risks of early mortality


<table>
<thead>
<tr>
<th>Trial</th>
<th>N</th>
<th>Fibrinolytic regimen</th>
<th>Time difference (minutes)</th>
<th>Mortality</th>
<th>Odds ratio and 95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIT⁸</td>
<td>360</td>
<td>Alteplase (100 mg over 3 hours)</td>
<td>33</td>
<td>5.7%</td>
<td>8.1% 0.69 (0.30-1.57)</td>
</tr>
<tr>
<td>EMIN⁹</td>
<td>5469</td>
<td>Anistreplase (30 IU intravenous bolus)</td>
<td>55</td>
<td>9.1%</td>
<td>10.4% 0.86 (0.72-1.03)</td>
</tr>
<tr>
<td>GREAT⁹⁰</td>
<td>311</td>
<td>Anistreplase (30 IU intravenous bolus)</td>
<td>130</td>
<td>6.7%</td>
<td>11.5% 0.56 (0.25-1.23)</td>
</tr>
<tr>
<td>Roth et al₁¹</td>
<td>113</td>
<td>Alteplase (120 mg over 6 hours)</td>
<td>43</td>
<td>5.8%</td>
<td>6.8% 0.80 (0.17-3.77)</td>
</tr>
<tr>
<td>Schofer et al₁²</td>
<td>78</td>
<td>Urokinase (2 million IU intravenous bolus)</td>
<td>43</td>
<td>2.5%</td>
<td>5.3% 0.46 (0.04-5.31)</td>
</tr>
<tr>
<td>Castaigne et al₁³</td>
<td>100</td>
<td>Anistreplase (30 IU intravenous bolus)</td>
<td>60</td>
<td>5.3%</td>
<td>7.0% 0.74 (0.14-3.86)</td>
</tr>
<tr>
<td>Overall</td>
<td>6434</td>
<td></td>
<td></td>
<td>8.5%</td>
<td>10.2% 0.83 (0.70-0.96)</td>
</tr>
</tbody>
</table>
The implied benefit of earlier treatment in the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO-I) Trial was five lives saved for every hour of delay avoided per 1,000 patients treated (2+). It is acknowledged that all of these analyses were observational, as none of the studies deliberately randomised patients to receive fibrinolytic therapy at different timepoints, and it should be remembered that patients who present later may have different baseline characteristics from those who present earlier, viz. those presenting late are more likely to be elderly, female or diabetic.

In a meta-analysis\textsuperscript{19} of randomised trials comparing pre-hospital fibrinolysis with in-hospital fibrinolysis (Figure 1),\textsuperscript{8–13} there was an overall 17% reduction in mortality—with 16 lives saved per 1,000 patients treated with pre-hospital fibrinolysis (P=0.02) (1++). Of note, a 44% mortality reduction was observed in the Grampian Region Early Anistreplase (GREAT) Trial (1-),\textsuperscript{10} which reported a 2-hour treatment delay, whereas in the European Myocardial Infarction Project (EMIP) there was only a 14% mortality reduction despite a shorter treatment delay of only 55 minutes (2+).

In another recent trial, pre-hospital administration of tissue plasminogen activator (TPA) was found to be equivalent to primary angioplasty in reducing the combined risk of death, reinfarction and stroke.\textsuperscript{20} Follow-up at 2½ years showed that the mortality rates were 6.7% in patients treated with pre-hospital fibrinolysis versus 8.8% in those treated with primary angioplasty (P=0.05 for patients treated within 2 hours of symptom onset) (1+).

Fibrinolysis carries a small risk of intracranial haemorrhage, but this is offset by a reduction in the risk of ischaemic stroke, and as some of the strokes are fatal, they are already counted in the mortality benefit. The overall benefit/risk ratio of fibrinolysis is 16 lives saved at the cost of one nonfatal disabling stroke per 1,000 patients treated with fibrinolysis.

The likelihood of ventricular fibrillation is greater when fibrinolytic therapy is administered very early after the onset of symptoms (2+),\textsuperscript{21} but other complication rates are similar to those seen with in-hospital fibrinolysis. It is therefore recommended that communities more than 1 hour away from the nearest hospital with fibrinolytic facilities should initiate programmes to administer pre-hospital fibrinolysis.

\section*{Recommendations}

\subsection*{Programme implementation}

Before pre-hospital fibrinolysis programmes can be commenced, it is desirable that all medical practitioners involved should receive adequate training in collaboration with the central supporting service. Practice nurses, community hospital nurses and ambulance officers would also benefit from training.

\textit{The training should include information on:}

\begin{itemize}
  \item Interpretation of electrocardiograms (ECGs).
  \item The particular fibrinolytic agent chosen.\textsuperscript{4}
  \item Indications for, and contraindications against, fibrinolytic therapy (4, D).
  \item Management of the potential side-effects of fibrinolytic therapy.
\end{itemize}
- Use of defibrillators (4, D).

An ongoing audit should be maintained—with regular reviews to assess the accuracy of infarct diagnosis, the timing of fibrinolytic administration, and patient outcome.

Figure 2. Algorithm for pre-hospital administration of fibrinolytic therapy.

START

Patient with chest pain 1 to 2 hours away from nearest hospital who makes an emergency call to the ambulance service and/or GP.

Assess patient and take a clinical history at the rural site.

- Administer 150 to 300 mg of aspirin.
- Administer pain relief and/or anti-nausea drugs if needed.

Figure 2 Continues on the next page
*Some general practitioners will be very experienced and the supporting hospital may prospectively approve independent practice.

**Principles for pre-hospital administration of fibrinolytic therapy:**

- All general practitioners administering fibrinolytic therapy must have a well maintained defibrillator available at the time of fibrinolysis (4, D).
- Fibrinolysis should be considered in all patients who:
  - Meet the diagnostic criteria for acute myocardial infarction, and
  - Present within 12 hours of symptom onset, and
  - Are =1 hour away from the nearest hospital with fibrinolytic facilities (4, D).
- The treatment algorithm is outlined in Figure 2, and discussed in detail below.

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**Figure 2 above (continued from previous page)**

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- The diagnosis of acute myocardial infarction must be confirmed by a 12-lead ECG (1++, A), and approval for pre-hospital fibrinolysis must be obtained from the supervising hospital (4, D). Practitioners who are experienced in the administration of pre-hospital fibrinolysis may be granted prior approval by the supervising hospital to administer pre-hospital fibrinolysis when indicated.

- The ECG should be faxed or transmitted via modem to the supervising hospital. While it is recognised that digitally transmitted ECGs are clearer and require less processing, budgetary constraints may mean that faxing is the preferred option. This decision will need to be made on a local basis (4, D).

- Every effort should be made to minimise the delay between the diagnosis of infarction and the administration of fibrinolytic therapy (1+, A). A bolus fibrinolytic agent such as reteplase (RPA) or tenecteplase (TNK-TPA) should be administered as soon as possible after the diagnosis of myocardial infarction is confirmed. Currently, reteplase is the bolus fibrinolytic agent most commonly used for pre-hospital fibrinolysis in New Zealand. Tenecteplase has been shown to be associated with less systemic bleeding than tissue plasminogen activator (1+). Streptokinase is not recommended for pre-hospital fibrinolysis because it requires an infusion pump, is difficult to administer, and frequently causes hypotension. Tissue plasminogen activator is not recommended either because it too requires an infusion pump, and has a complex dosage regimen (4, D).

- The patient may be transported to hospital by road or air depending on the clinical stability of the patient, the distance involved, and the road and weather conditions. The transport policy will need to be assessed on a regional basis (4, D).

Indications for fibrinolytic therapy

- Absence of contraindications against fibrinolytic therapy.

- A clinical history of =30 minutes of chest discomfort beginning ≤12 hours previously and consistent with ischaemic aetiology (1++, A), plus:

  Either: An ECG showing ST-segment elevation measuring ≤1 mm in two or more inferior leads, or =2 mm in two or more contiguous anterior leads V1 to V3, or =1 mm in leads V4, V5, V6 or AVL (1++, A).

  Or: An ECG showing a left bundle branch block pattern that is not known to be pre-existing (1+, A).

Contraindications against fibrinolytic therapy

- Suspected aortic dissection (4, D).

- Any previous history of haemorrhagic stroke, or a suspected previous stroke in which haemorrhage was not excluded by scanning (1+, A).

- History of non-haemorrhagic stroke or central nervous system damage within 1 year (1+, A).

- Head trauma (1+) or brain surgery (4, D) within 6 months, recent lumbar puncture, known cerebral tumour or aneurysm (1+, A).
Internal bleeding within 6 weeks (4, D).

Active bleeding or known bleeding disorder (4, D).

Major surgery, trauma or bleeding within 6 weeks (1+, A).

Traumatic cardiopulmonary resuscitation within 3 weeks (4, C).

Oral anticoagulant therapy (3, C).

Persistent hypertension (systolic blood pressure of > 180 mmHg or diastolic blood pressure of >110 mmHg) (1+, A).

Puncture of a non-compressible blood vessel within 2 weeks (4, D).

Peptic ulcer disease documented by endoscopy with symptoms occurring within the previous 3 months (4, D).

Pregnancy (4, D).

Note: The indications for fibrinolysis and the absence of contraindications should be discussed fully with the hospital before an expeditious decision is made as to whether fibrinolytic therapy should be administered.

Dosage regimens

Reteplase should be administered intravenously as a 10 IU bolus over 2 minutes, and repeated 30 minutes later.

Tenecteplase should be administered intravenously over 10 seconds in a weight-adjusted regimen (30 mg for patients weighing <60 kg, 35 mg for patients weighing 60 to 69 kg, 40 mg for patients weighing 70 to 79 kg, 45 mg for patients weighing 80 to 89 kg, or 50 mg for patients weighing =90 kg) (1++, A).

Streptokinase is not recommended for pre-hospital administration because of the incidence of hypotension (10%) (4, B)

Adjunctive therapy

All patients should receive 150 to 300 mg of soluble or sublingual aspirin to chew as soon as the possibility of acute myocardial infarction is considered (1++, A).

*If reteplase is chosen as the fibrinolytic agent, it should be followed immediately by unfractionated heparin administered intravenously in a dose of 4,000 IU for patients weighing >70 kg or 3,500 IU for those weighing <70 kg. Ideally, a heparin infusion should then be commenced at a rate of 12 IU/kg/hour (maximum 1,000 IU/hour) (1–, B). However, administration of an infusion may be impractical for logistical reasons, and so an alternative option is to administer a second heparin bolus after 90 minutes*.

If tenecteplase is chosen as the fibrinolytic agent, it is recommended that enoxaparin be administered immediately as a 0.3 mL (30 mg) intravenous bolus followed by a 1 mg/kg subcutaneous injection (1+, A). In patients aged =75 years, the intravenous bolus should be omitted, and the subcutaneous dose should be increased to 0.75 mg/kg up to a maximum total dose of 75 mg.
Note: Alternatively, intravenous unfractionated heparin may be administered as detailed above.

- Medications such as adrenalin and atropine should be available in case of cardiac arrest.
- Adequate medication for pain relief and nausea should be available and administered as appropriate.

Equipment

Advisory defibrillators should be available for immediate use during transportation.

Indicators

*It is important that pre-hospital fibrinolysis services are audited, and so the following outcomes are to be documented along with patient demographics:

- Time from symptom onset to administration of fibrinolytic therapy.
- Time from first contact with emergency services (111 call), ambulance or primary healthcare services to administration of fibrinolytic therapy.
- Time from when the patient is first seen by a clinician to administration of fibrinolytic therapy.
- Time from administration of fibrinolytic therapy to start of transport.
- Time from administration of fibrinolytic therapy to arrival at hospital.
- Complications following acute myocardial infarction: bleeding, arrhythmia, cardiogenic shock, stroke, death.
- Death within 30 days after acute myocardial infarction.
- Baseline ECGs to determine the accuracy of diagnosis.

Summary

New Zealand is a rural country with many isolated regions that are distant from hospitals with fibrinolytic facilities. Pre-hospital fibrinolysis is thus the only way that rural patients can be efficiently managed with modern reperfusion therapy. These guidelines provide a framework for safe and appropriate administration of fibrinolytic agents in the New Zealand rural community. The guidelines will be updated 2 years after publication.

Author information (Pre-Hospital Fibronolysis Guidelines Working Party):

These guidelines were initiated and endorsed by the New Zealand Regional Committee of the Cardiac Society of Australia and New Zealand. Roche Products (New Zealand) Limited provided financial support for two meetings of the Working Party, but the guidelines were developed independently of pharmaceutical industry funding.

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A call to action on Maori cardiovascular health

Dale Bramley, Tania Riddell, Sue Crengle, Elana Curtis, Matire Harwood, Deidre Nehua, Papaarangi Reid

Introduction

Maori have the poorest cardiovascular health outcomes in Aotearoa (New Zealand).\textsuperscript{1–5} Although disparities in these health outcomes have been documented for many years, progress toward reducing them has been alarmingly slow.\textsuperscript{1}

Disparities in health are seen as being unjust and inequitable, avoidable, and potentially detrimental to all members of society. Furthermore, efforts to reduce disparities may be cost effective.\textsuperscript{6}

Full recognition of Maori rights as tangata whenua, as reflected in the Treaty of Waitangi, is an important driver towards the goal of Maori having at least the same standard of health as non-Maori. Disparities also reflect the fact that Maori health status has not been afforded the ‘protection’ the crown intends under the Treaty of Waitangi, using the government’s health framework of the guiding principles of the Treaty.\textsuperscript{7}

In 2001, the Ministry of Health in association with the New Zealand Guidelines Group convened a National Cardiovascular Advisory Committee. The aim of this group was to:

‘advise on the development of an integrated managed approach to cardiovascular disease, from primary prevention through to tertiary treatment in Aotearoa, New Zealand. The work of the committee was to draw upon the best available evidence and was to be conducted in accordance with the principles of the Treaty of Waitangi’.

A key task of this group was to facilitate the production of a Maori cardiovascular action plan. To produce this plan, a separate Maori cardiovascular group was formed.

The aims of this paper are to:

- Provide a brief overview of the current status of Maori cardiovascular health,
- Outline the key themes of the Maori cardiovascular action plan, and
- Stimulate co-ordinated action by the health sector to reduce Maori cardiovascular disparities.

Although many of the determinants of health lie outside of the realm of the health sector, the sector has a key role in ensuring that access to procedures is equitable and that healthcare responsiveness is based on demonstrable need.\textsuperscript{8}

An overview of Maori cardiovascular health status

Mortality rates for cardiovascular disease in Aotearoa have been declining. However the decline in Maori cardiovascular mortality has occurred more slowly. This has led
to an increase in disparities. \(^1\) For the period 1996–1999 the Maori male cardiovascular mortality was 3.0 times higher than that for non-Maori, non-Pacific males—and the Maori female mortality rates was 4.2 times higher than that for non-Maori, non-Pacific females. \(^1\)

In 2002, 30-day age standardised case fatality rates following acute coronary syndrome were 158 per 1,000 patients for Maori compared to 112 per 1,000 patients for Europeans/Others. \(^9\)

Maori also have the highest prevalence of many cardiovascular risk factors. The AC Neilsen Tobacco Survey (2002), found that 44% of Maori males (aged 15+ years) compared to 24% of European males and 51% of Maori females compared to 24% of European females were smokers. \(^10\) Smoking is the leading modifiable risk factor causing disease. \(^11\)

Provisional release data from the 2002/2003 New Zealand Health Survey (which included over 3,900 Maori) has provided a wealth of information regarding the prevalence of cardiovascular risk factors in Maori and non-Maori. \(^12\) From this survey, the prevalence of self reported diabetes in adults over 45 years was 21.4% in Maori males and 13% in Maori females—compared to 8.6% in non-Maori males and 7.5% in non-Maori females. \(^12\)

The prevalence of self-reported high blood pressure was 23.2% in Maori males and 22.1% in Maori females—compared to 17.5% and 18.7% respectively in European New Zealanders. \(^12\) Furthermore, the prevalence of obesity (measured by the interviewer and defined as a BMI \(=30\) kg/m\(^2\) in non-Maori and \(=32\) kg/m\(^2\) in Maori) was 31.5% in Maori males compared to 16.5% in European males, and 26.7% in Maori females compared to 19.1% in European females. \(^12\)

Given this context of high need, it would be expected that cardiovascular intervention rates would be substantially higher for Maori. However, the converse is true. Interventions for coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA) have been consistently lower for Maori over many years.

Tukuitonga and Bindman reported that over the period 1990–1999, Maori men had a mean age standardised CABG and PTCA intervention rate ratio of 0.40 and 0.29 respectively when compared to European men. \(^13\) Although these intervention rates have been increasing in recent years, they are still far below that which would be expected given the higher prevalence of risk factors and the higher incidence of disease in Maori.

Similarly (though data is sparse and is derived from administrative data-sets) it would appear that Maori have lower utilisation of diabetes screening. Only 35% of Maori people estimated to have diagnosed diabetes had a free check in 2002 compared to 51% of all people estimated to have diagnosed diabetes. \(^14\) Compared with Europeans, Maori diabetic patients were also less likely to be on cholesterol-lowering medications and ACE inhibitors, and were less likely to have good glycaemic control (HBA1c <8%). \(^14\)
The Maori cardiovascular action plan

The overall aim of the Maori Cardiovascular Action Plan is to improve Maori cardiovascular health and to remove inequalities in cardiovascular disease outcomes between Maori and non-Maori. The action plan has six categories. These categories reflect the need for a multi-level, multi-sector approach to improving cardiovascular outcomes. The categories for action include the following areas: policy development, improved information systems, needs assessment, quality standards, Maori workforce development and a proposed research agenda.

The Treaty of Waitangi and policy development

The explicit recognition of the Treaty of Waitangi is central to the Maori Cardiovascular Action Plan. Indeed, the Treaty of Waitangi and Whakatataka (the Government’s Maori health action plan 2002–2005) are to be included in all policy development. The Maori Cardiovascular Group endorses Jackson’s (2001) comment that the very reason Maori have high need is because Maori rights under the Treaty of Waitangi have not been respected.15

Flowing from the recognition of the Treaty of Waitangi is the need to prioritise Maori health gain in all health policy directives. Therefore, consultation, engagement, leadership, and representation of Maori in all areas of policy development and implementation are needed.

It is worth noting here that the Waitangi Tribunal has recently confirmed that the Treaty of Waitangi assures to Maori:

- Equal standards of healthcare,
- Equality of access to healthcare, and
- A general equality of health outcomes.16

Information systems

To monitor disparities and health status, complete and consistent collection of ethnicity data resulting from health services encounters is essential. To facilitate this, there is a need to implement a standardised ethnicity question across the health sector.

Specifically, to ensure consistency between numerators and denominators within health data-sets, the 2001 census question should be used. The way in which ethnicity data is coded and stored should also be standardised. Furthermore, resources and educational material for the training of key health personnel in the area of ethnicity data collection are also needed. The Maori Cardiovascular Group endorses the use of regular audits to monitor the accuracy and completeness of the ethnicity data collected by health providers.

Regarding health provider funding, the amount and level of health expenditure on Maori cardiovascular health should be monitored. In particular, expenditure should be consistent with Maori cardiovascular need and efforts to reduce disparities. In the short term, this will require an additional investment in Maori cardiovascular health until disparities are eliminated.
Needs assessment

Cardiovascular health needs assessments for Maori are required in order to identify
the level of met and unmet need in the community. Access barriers to preventive,
primary, secondary, and tertiary services should be identified in partnership with
Maori stakeholders. Creation and implementation of strategies to address identified
barriers will then be required. Access to preventive services including the promotion
of ‘healthy environments’ should be emphasised in order to reduce the incidence of
cardiovascular disease.

A long-term goal of the Action Plan is the need for accurate Maori cardiovascular
disease prevalence and incidence data. Quality indicators should be developed that
measure Maori access to cardiovascular interventions, and treatments should be based
on prevalence and incidence of disease, rather than calculating access to
cardiovascular interventions based only on ethnicity demographics.

Quality standards

To improve Maori cardiovascular health, it is essential that Maori gain access to (and
utilise) evidence-based treatments that have been shown to reduce morbidity and
mortality. To achieve this, quality indicators appropriate for medical care are
currently being developed by the Ministry of Health to monitor cardiovascular health
in New Zealand. Such an approach is consistent with the worldwide trend of an
increased emphasis on the measurement of the quality and outcomes of medical
care. The Maori Cardiovascular Advisory Group has strongly recommended that
Maori specific performance indicators in cardiovascular health be measured. The
group has produced a number of indicators of particular interest for ongoing
monitoring of Maori cardiovascular health gain. These include indicators for
cardiovascular disease prevention and care at primary, secondary and tertiary levels.

Workforce development

There is a critical shortage of Maori involved in cardiovascular healthcare. To
adequately document this shortage, a benchmark audit of the number of Maori
working in the field is needed. After this has been completed, targets should be set for
the ongoing recruitment and training of Maori in the field. Priority areas for Maori
recruitment and training include cardiology specialists, cardiology registrars, coronary
care level III and IV nurses, cardiac rehabilitation nurses, health researchers, and
public health workers (including health promotion staff).

The workforce of Maori specific providers that specialise in cardiovascular care
should also be expanded and their skill mix upgraded. Specific recommendations
regarding Te Hotu Manawa Maori have also been included in our action plan, as this
organisation is the largest Maori specific cardiovascular health provider in the
country.

Regarding non-Maori workforce development, the Group advocates for health
organisations to have service-wide recognition of the Treaty of Waitangi. Specifically,
training courses and educational resources should be available to all staff—with staff
actively encouraged to participate. For large cardiovascular health organisations,
Treaty of Waitangi audits should be undertaken to measure their responsiveness to
Maori.
Innovative ways of delivering care to Maori are needed in the long term. This may necessitate models of health promotion and care that are based in the community, and deliver services directly to Maori.

**Research agenda**

Kaupapa Maori research is needed. When research is undertaken from this perspective, Maori cardiovascular health concerns and needs become self-determined, as well as the research response needed to address them. Kaupapa Maori research also advocates for the use of Maori: non-Maori comparisons and produces results that have ‘equal’ meaning and relevance to Maori as non-Maori. The Maori Cardiovascular Advisory Group supports research that prioritises Maori concerns and that use a Maori defined analytical framework to address them.

Research findings are currently lacking regarding the current status of Maori cardiovascular health (including accurate prevalence and incidence data). Research should be undertaken, both qualitative and quantitative, regarding access to care (including access barriers), equity of process along pathways of care, and equity of health outcomes for Maori.

New electronic decision support tools for cardiovascular health such as PREDICT are currently being developed. They offer the promise of a better understanding of the relationship between the Maori population’s cardiovascular risk factors, and mortality and morbidity (in essence a Framingham-type risk assessment specific to Maori). It is important that these new tools are used to improve Maori health, and imperative that Maori researchers are actively involved in their development and utilisation.

The group also endorses the need to investigate and evaluate alternative models of service delivery for cardiovascular prevention and care. To date, the current organisation and delivery of the health system has been ineffective in reducing disparities. New and innovative methods of delivery (for example, care delivered in community based settings relevant to Maori) should be explored and evaluated.

Any discourse on disparities would not be complete without commenting on the wider determinants of health, in particular, socioeconomic determinants. Although, discussion of these factors is beyond the scope of our Action Plan, the Group is aware that the elimination of disparities will require action on numerous ‘fronts’—including (although not limited to) employment, education, housing, and welfare. Utilisation of methodological frameworks that present a broader view of wellbeing (such as Te Pae Mahutonga and the Ottawa Charter) may assist the development of policy that is more holistic and better reflects Maori realities.

**Moving forward**

The next step of the Maori Cardiovascular Action Plan is the assessment of the above recommendations (including financial implications) by policy makers. If no specific action is taken to address the issues identified in this Plan, it is likely that the current disparities that exist in cardiovascular health for Maori will continue.

**Conclusion**

Disparities in cardiovascular health outcomes in Aotearoa continue to negatively impact upon Maori. Little progress has been made in reducing the size of these
disparities. A Maori-specific Cardiovascular Health Action Plan has been developed that we hope will improve the responsiveness of the health sector to Maori. This plan is consistent with the full recognition of Maori rights as tangata whenua—as reflected in the Treaty of Waitangi. A multi-level, multi-sector approach is needed to address these disparities. The Maori Cardiovascular Advisory Group hope that this Plan will go some way in providing the guidance that is needed to address the role that the health sector can make in reducing these disparities.

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The management of public hospitals in New Zealand

This extract is taken from a Presidential Address by Dr Ernest Roberton that was published in the New Zealand Medical Journal 1905, Volume 4 (13), p2–3

If we consider the causes of these troubles—the extravagant expenditure, the erection of unsuitable buildings, the appointment of an unworkable or unsuitable staff, the interference with medical or nursing arrangements, unnecessary and inconsistent changes in methods of management, the catering in the special needs of one section of the community, the pandering to those who have political influence—we find that behind all these lies the unsuitability of the Boards through want of expert knowledge or of other special qualifications for the work which they have to perform.

The best proof that such special qualifications are wanting is to be found in the fact that where, as in smaller hospitals, the work of management is comparatively simple, troubles have been few. No special demands are made either on the intelligence or knowledge of the committees, and the Boards are thus equal to their work. When, however, as in the larger towns, the hospitals are institutions of a decidedly complex nature, similar Boards are found wanting, and the attempts to manage through them have led to frequent trouble.

It was not realised by those who arranged the constitution of the present Boards that in the larger hospitals, where, for proper administration, it is necessary to have a large staff of paid and honorary officials and servants, the managers should be able to rely on special technical knowledge possessed by some of their members, and should, moreover, have some special aptitude for controlling conflicting interests.

In some instances those in whose hands the law has passed the management have, with experience, shown themselves able to acknowledge this defect in their constitution, and, when necessary, have acted consistently in wise dependence on well-chosen expert advice. Unfortunately, the members of local bodies have a tendency to act on their own self-sufficiency, and the majority of members of the Hospital Boards are representatives of small local bodies elected by their fellows.

Hitherto the choice of men has been too limited, and some classification of hospitals is needed, so that, as the organization of any institution becomes more complex—as it grows with the population of its neighbourhood—the difficulties of management may be met by a Board proportionately better equipped in knowledge and experience for the special work required of it.

The public hospitals in New Zealand are State institutions, supported by compulsory rating and taxation, and, to some extent, by endowment, voluntary contributions, and fees paid by patients. In a colony like ours, which prides itself on being well to the front of questions affecting the welfare of its people, we might naturally expect to find, in connection with the management of public institutions devoted to the care of the sick, sufficient legislation enacted with the aim of promoting efficiency of management. Whatever there is of the kind is rudimentary—namely, the appointment of inspectors who may report to the Government, but who have no power of controlling the Boards or their employees.
The hospitals are managed under “The Hospitals and Charitable Institutions Act, 1885,” and its amendment of the following year, 1886. The Acts were measures designed almost entirely to provide funds for the maintenance (sic) of hospitals.
Magnetic resonance cholangiopancreatography (MRCP) demonstrating cholecystolithiasis and aberrant pancreatic duct anatomy

Timothy Eglinton, Angus Watson

A 21-year-old woman presents with right upper quadrant pain and cholestatic liver function tests. MRCP demonstrates two large calculi (GS, Figure 1) in the gallbladder.

Incidental note is made of the unusual pancreatic duct anatomy. The fusion of the dorsal and ventral pancreatic ducts during development usually results in the main pancreatic duct of Wirsung draining via the major duodenal papilla with the common bile duct (CBD). Variations in this arrangement occur and may predispose to pancreatitis.

Figure 2 demonstrates the ventral duct joining the dorsal duct (PD), which then drains into the minor duodenal papilla proximal to the CBD.

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Another bad move, OTC statins?

Since July 1, simvastatin has been available without a prescription in the UK. The Department of Health has accepted the advice of the Committee on Safety of Medicines (CSM) that simvastatin 10 mg can be sold through pharmacies to people at moderate risk of coronary heart disease. The UK is the first country to make a statin available over the counter (OTC).

Will those who buy simvastatin also stop smoking, lost weight, and do more exercise, or will they substitute drug use for lifestyle modification? Will pharmacies have the time to determine the individual’s risk of coronary heart disease before selling the drug and also to give lifestyle advice.

Will they have the time (or knowledge) to detail the more than 30 recorded adverse effects? And the absolute risk (AR), absolute risk reduction (ARR), and relative risk (RR), etc, etc?

Furthermore, prescribed statins (pravastatin 40 mg, atorvastatin 10 mg, and lovastatin 20-40 mg) have not been shown to provide an overall mortality benefit in primary-prevention trials. It is unlikely that a low dose of OTC simvastatin will do what has not been found in controlled settings.

In the absence of evidence of the overall mortality benefits of OTC simvastatin, it is difficult to avoid concluding that the motive behind the Government’s decision is saving money.

Lancet 2004;363:1659

A new (and safer) anticoagulant?

The only treatments proven to reduce the 5% annual risk of stroke among patients with atrial fibrillation are aspirin and adjusted-dose warfarin. Aspirin reduces the risk by 20% and warfarin by about two-thirds.

Warfarin causes at least twice as many intracranial and extracranial bleeds as aspirin, particularly in patients at increased risk of bleeding (eg, those aged over 75 years, those with a history of bleeding). Warfarin is also inconvenient to use because it has a narrow therapeutic index, interacts with numerous drugs and food, and requires close laboratory monitoring. Consequently, only a third to a half of patients with atrial fibrillation who are appropriate candidates for warfarin therapy actually receive it.

Two large phase III randomised trials have recently evaluated ximelagatran as a replacement for warfarin to prevent thrombotic complications in patients with non-valvular atrial fibrillation.

The results of these trials show that this thrombin inhibitor is not inferior to warfarin in efficacy and has significantly reduced bleeding complications.

MJA 2004;180:549–51
**Albumin or saline in the ICU?**

The administration of intravenous fluids to maintain or increase intravascular volume is a common intervention in the intensive care unit (ICU), but there is uncertainty whether the choice of fluid significantly influences patients’ outcomes.

Meta-analyses have produced conflicting results, but in a recently published Australasian trial involving nearly 7000 patients it has been demonstrated that 4% albumin and normal saline achieve similar outcomes at 28 days. Parameters evaluated included death, organ failure, ventilation requirements, and days in ICU and hospital.


**Are biros OK?**

One of the biggest inquiries into marketing practices in the drugs industry ended last week with Italian police asking for almost 5,000 people to be put on trial, including more than 4,000 doctors and at least 273 employees of the British Pharmaceuticals giant, Glaxo-SmithKline. Some face up to five years in jail if convicted.

Italy’s revenue guard, the Guardia di Finanza, said that Glaxo-SmithKline and its predecessor firm had spent £228m on “sweeteners” for doctors, chemists and others over four years. The alleged bribes ranged from cameras, computers and holidays to cash payments.

A spokesman for GSK said it had been “cooperating closely with the authorities” in their investigations. He added that any breach of GSK’s highest standards of business practices was “unacceptable”.

*Guardian Weekly (UK); 4–10 April 2004*

**Patent nonsense!**

Health institutions in France have succeeded in overturning a European patent on a breast cancer gene held by a US company, Myriad Genetics. Myriad had been trying to establish a worldwide monopoly on testing for the mutations in two genes, *BRCA1* and *BRCA2*, that increase the risk of breast cancer. It has been insisting all samples be sent to its labs in Salt Lake City for testing. This has angered patient groups and doctors, who claim its tests are more expensive and less accurate than those offered by public labs in Europe. In February, the European Patent Office revoked Myriad’s patent on *BRCA2*, ruling that the charity Cancer Research UK had filed its patent first. Now the EPO has revoked the *BRCA1* patent. Opponents found discrepancies in the gene sequence described in Myriad’s patent, issued in 2001, and the sequence in its original patent application in 1994. It was not until seven months later, in 1995, that Myriad submitted an updated sequence, by which time the correct sequence had already been published elsewhere. The ruling means labs in Europe no longer have to pay any licence fees, making genetic screening of women much cheaper. Myriad says it will appeal.

*New Scientist; 29 May 2004*
Medicalisation of abortion

The Abortion Supervisory Committee has once again chronicled its annual report documenting the increasing abuse by medicalisation of a social condition in New Zealand. In 2003, 18,510 women were each seen for 37,020 certifying consultations, and medicolegally certified as meeting the legal conditions for an abortion and proceeded to have a termination of pregnancy.

The legal requirements for medicolegally justifying termination under the 1977 Act are that the woman's mental or physical health are at serious risk if the pregnancy continues. In 2003, 18,510 woman met these criteria at least according to approximately 300 poorly paid state-funded doctors euphemistically called certifying consultants.

Given that there are about 55,000 live deliveries in New Zealand annually, approximately 25% of the current breeding stock of women were medicolegally certificated as criminally insane, intellectually retarded, feeble-minded, suicidally depressed, unable to cope with having a baby—or for a very very small percentage, the certifying consultants honestly believed that the woman's physical health would actually be at risk if the pregnancy went to term (eg, severe HOP syndrome, blood incompatability reactions, ruptured uterus, rape etc).

According to the law, nearly one in four pregnancies in New Zealand accidentally happened to a severely psychologically impaired woman in 2003. The Ministries would have, no doubt, been pleased to see that there was no obvious ethnic or genetic bias for this severe psychological impairment.

Do the politicians and the Abortion Supervisory Committee, the Ministry and Ministers of Health and Justice, the general population of New Zealand, the medical sorority, or anyone at all seriously believe that? (One of the problems about medicalising social problems is that the medical justification becomes untenable and patently ludicrous, not to mention a blatant lie and ethically unacceptable—with (one would have thought) serious grounds for complaint to the Medical Council.

New Zealand has abortion on demand! Of course it does.

It has the same rates of abortion as Australia and the United States. The four million dollars paid for certifying liars is not a legitimate check or balance on abortion (as envisaged by the law makers), and the Committee no longer publishes the numbers of women who are declined, or perhaps are persuaded from having, an abortion because of the certifying consultants.

The Ministry of Health and the Health Funding Authorities’ maternity and abortion managers in the 1990s even felt that the woman’s own General Practitioner had so little role to play in discussing the options for women that they recommended to the Minister of Health that the payment for a GP for assessment prior to termination be reduced from $70.00 to $40.00 in 1996. By reducing the payment to $40.00 for around 10 minutes of GP time for pregnancy assessment, discussion, referral for scans, counselling, assessment for anaesthetic and physical examination, blood tests, a
smear and swabs, did these august abortion and maternity managers feel that they were furthering the cause of abortion on demand? The Ministers of Health, Finance, and the Prime Ministers of the times all obviously agreed with the Ministry’s advice.

While the abortion law is long overdue for revision to remove the obviously farcically applied medical justification for termination, the Ministers and the Ministries persist in their use of the medical justification for patients and people's access to funding and services in the belief that doctors will take responsibility for the wastage of time, effort, money, and lives.

Bill Douglas
General Practitioner
Wanganui
State-imposed abuse of doctors

The medical justification for abortion is used by the State to shift the responsibility for access to services and social funding from the collective shoulders of Government onto the medical profession. It is under-funded, illogical, and medico-socially reprehensible. This State-sanctioned, under-funded abuse is contributing to the lack of new entrants to general practice and the exit of existing practitioners.

Pharmac, for example, is completely two-faced about abusing doctors to restrict access to drugs, and (at present) is using them to force patients to shaft pharmacy incomes. (See the Pharmac website on extension of enforcing State-prescribing by judicious use of bureaucracy—http://www.pharmac.govt.nz/) The Ministry of Social Development uses doctors to certify that people can't work 30 hours a week, or are incapable of work as justification for shifting people off the dole. What would a doctor know about a person's work ability from a 10-40 minute patient-funded discussion that (if unsuccessful) reduces the doctor's likelihood of being paid and increases the probability of a complaint? Why 30 hours? Why not 10 hours or 20 hours a week—because the Ministry’s definition of work appears to be based on a person who physically digs drains all day!

There are 70,000 people in rest homes. On the WINZ form signed by a doctor, according to the criteria for State-funded personal medical alarms, the person would be unable to live independently if they did not have a state-funded alarm. According to that criteria, there are some 40,000 people who, without the State-funded doctor-sanctioned alarm, would have to be living in rest homes, in care, or with others. (Some $60 million of doctor sanctioned? controlled social spending!). No-one seriously believes that these expensively over-priced alarm companies are helping keep people out of rest homes do they? The presence of most alarms allays the fear of 'what if something happened', as instilled by alarm sellers in their victims—and make often-stingy relatives feel they have shifted their concerns and responsibilities onto someone else, which doesn’t interfere with their finances.

There are many other examples of the State-imposed abuse of doctors to medically collectively take responsibility for poor social plans and welfare funding access. Forty million dollars for State-funded 'medically'-required telephones. ACC payments for patient access to chiropractic, osteopathy, acupuncture, drugs, and physiotherapy are all dependent on GPs taking responsibility for gate-keeping for a stingy, abusive State organisation, and extracting the payment for being the gate-keeper from the personal individual sitting in front of them.

Currently ACC, as an insurance company, refuses to tell premium-payers what suppliers have special deals to reduce ACC excesses to patients. Do you know why an ACC-premium payer in Wanganui receives a subsidy for a 41-minute consultation of $26.00 while a foreign, non-premium paying customer in Queenstown or an Auckland A&M clinic receives a State-funded subsidy of $162.00 for the same injury? David Rankin of ACC refuses to advertise these anomalies. If the State wants General Practitioners and other doctors to gate-keep on access to its social agenda and
funding, should the State not pay the gate-keepers fully and properly for undertaking this specific service?

Is it reasonable for the State to expect the doctor to deprive the patient (sitting in front of them) access to funding, to explain the rationale or ‘irrationale’ as the case may be for the lack of access to this apparently bottomless pit of funding, and then advise the doctor that the fee for this consultation is to be extracted from the aforementioned patient?

Bill Douglas
General Practitioner
Wanganui
How concordant are hospital discharge codes with physicians’ diagnoses?

Research studies often recruit participants from hospital discharge data, and admissions per annum are commonly used to estimate morbidity, healthcare needs and costs. In 1999, when entry criteria for a randomised controlled trial of COPD patients were applied to a cohort of patients derived from Middlemore Hospital discharge data, the recruitment rate was approximately 20%. We questioned whether inaccuracies in coding data had contributed to this, and, therefore, at the conclusion of the trial we carried out a retrospective chart review of all patients selected for the study to assess coding accuracy.

At the time of this study, the principal (PDX) and secondary diagnoses (SDX) for an admission in a New Zealand hospital were entered into the patient’s hospital chart by the discharging medical officer, and the data codes then transferred to the hospital database according to ICD-9-CM codes and a specific Disease Related Group (DRG) by a coder or non-medical person. A review of the literature measuring the accuracy of discharge coding suggests that perhaps policy makers and researchers should interpret hospital database records with caution.

The charts of 105 patients and 151 discharge codes were examined by a physician not involved in the study, and his decision about the principal diagnosis was compared with the discharge code entered in the hospital database. Diagnoses were also assessed to determine whether admissions might be influenced by socioeconomic status (mean deprivation decile for study patients was 7.8) and an inability to cope at home. To ensure that the reviewer’s diagnoses were valid, the hospital charts of 5 patients were first reviewed independently by three respiratory health professionals and the interobserver reliability of reviewers in determining diagnoses was assessed. There was 100% concordance among the 3 reviewers.

Table 1. Respiratory Principal Diagnosis—Concordance between physician reviewer and coder (for diagnoses of respiratory conditions only)

<table>
<thead>
<tr>
<th>Coder</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physician</td>
<td></td>
<td></td>
</tr>
<tr>
<td>YES</td>
<td>80</td>
<td>5</td>
</tr>
<tr>
<td>p=0.94</td>
<td></td>
<td>p=0.06</td>
</tr>
<tr>
<td>NO</td>
<td>9</td>
<td>57</td>
</tr>
<tr>
<td>p=0.15</td>
<td></td>
<td>p=0.86</td>
</tr>
</tbody>
</table>

Total responses = 151
P = probability of response (0.0 – 1.0)
The probability of accurate coding for a respiratory admission by the coders was 0.94 (Table 1). This is comparable to accuracy rates reported in similar studies.\textsuperscript{1,3,4} Despite the generally low socioeconomic status of the study population, there were no admissions in our cohort purely for social reasons. Searching hospital data for study patients, however, did not predict the impact of co-morbid medical conditions (evident in this and similar groups of patients).\textsuperscript{4}

We concluded that this, rather than coding inaccuracies, may have been responsible for the low recruitment rate. Since the study was carried out, coding practices at Middlemore Hospital have been automated with the introduction of an electronic coding system. It may be of interest to carry out a review of study patients recruited from hospital data since then.

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Robert Charles Stewart Dick

Charles Dick was born in 1913 at Sevenoaks, Kent, England; the elder son of Dr Robert James Dick and his wife Hilda. He was educated at Sherborne School and Cambridge University—graduating with a BA in 1937, and MB, BChir in 1937.

His clinical training was at Guy’s Hospital where he held resident appointments and was strongly influenced by a leading English physician, Sir Arthur Hurst. In 1942, they co-authored a paper on diaphragmatic hernia, published in the *Quarterly Journal of Medicine*.

Charles’s pre-war years were notable for his success on the rugby field. He won a Cambridge Blue and was capped 13 times for Scotland; and according to *The Times* was ‘one of the finest centres in his day’. He played for Scotland against the All Blacks in 1936—and scored a try. In his later years, he was not enthusiastic about the style of modern professional rugby.

In 1939, he served in France with the Royal Artillery before being posted to the Military Hospital for Head Injuries in Oxford. In 1942, he joined a mobile neurosurgery unit based in India, reaching the rank of major. Ten years later, in New Zealand, he became Colonel of command of the 3rd General Hospital of the Royal New Zealand Army Corps. In 1958, he was appointed Honorary Physician to the Queen (QHP), a post he held for 10 years.

After the war years, Dick was not in good health and was disillusioned with British medicine. He chose to emigrate to New Zealand (where his wife’s family was based), and took over the practice of Dr CT Hand Newton in Christchurch. He became Assistant Physician at Christchurch Hospital in 1947, and played an important part in the treatment of poliomyelitis during a serious epidemic in 1956. His committee activities included Chairman of the Medical Staff Association, Chairman of the Blood Transfusion Service, and long-serving Executive Member of the Canterbury Medical Research Foundation.

In 1954, with Fred Gunz and Ian Gebbie, he published a paper on the treatment of gastro-duodenal haemorrhage in the *British Medical Journal*. He was elected FRACP in 1958 and FRCP in 1974.

In 1959, Charles Dick was appointed the first medical superintendent of The Princess Margaret Hospital (PMH). It was a surprising decision as he had little hospital administrative experience—but he adapted well, prospered, and gained a worthy international reputation during his 19 year tenure at PMH (as it was soon called). Professional staff enjoyed working there, standards of care were high, and research and teaching flourished. Charles continued to work in the wards, setting the highest clinical and ethical standards, and being greatly respected by his patients.
Charles and Ann owned a beautiful holiday home on the shores of Lake Tekapo, close to the mountains that meant so much to him. There, he enjoyed the love of his devoted wife and family, and pursued his many interests—walking, bird watching, restoring clocks, gardening, reading, and even writing doggerel verse. At the age of 90 years, and still showing his handsome features, he attended a reunion of PMH staff in Christchurch.

Charles Dick died on May 10, 2004 and was buried in Burke’s Pass Cemetery (between Fairlie and Lake Tekapo). Ann (nee Fell) had died in 2000—and he is survived by Ian (France), Peter (Victoria, Australia), Jenny (Whangarei), and Sue (Gisborne).

We are grateful to Sir David Hay for this obituary.
Netter’s Internal Medicine

Marschall Runge, Andrew Greganti Published by Icon Learning Systems, 2003.

This book, which has been produced by the University of North Carolina School of Medicine, is based on the premise that a good image is better than many words. It utilises many illustrations from the late and famous Frank Netter, supplemented by images by two Netter clones, interspersed with text.

I selected twelve topics of interest; two of them (hyponatraemia and myeloma) received no mention at all. Another three (AF, COPD, and thromboembolic disease) were dealt with in a very satisfactory fashion and the diagrams were highly useful. I also found some interest in the other seven topics, but, overall felt that they lacked balance and were not always appropriate for the New Zealand medical scene. For example, I doubt whether irritable bowel syndrome warrants more space than TIA and stroke. Within this latter section, I found that there was one page on thrombolysis and only five lines on stroke rehabilitation. Another disconcerting feature is that the S.I. system is not used in the laboratory information.

In the publisher’s blurb, the claim is proudly made that it ‘doesn’t bog down its readers with basic science, ethics, clinical trials, medical research, differential diagnosis, radiologic tests, or anything else which are topics better handled elsewhere.’ I think this just about sums it up and I believe most topics are better handled elsewhere.

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Evidence–based Dermatology


Williams et al define the practise of evidence-based medicine as ‘integrating one’s clinical expertise with the best external evidence from systematic research.’ It is ‘an essential skill that is as basic to being a doctor as the ability to examine and diagnose.’

Evidence-based Dermatology is an essential reference for those dermatologists wanting to acquire and/or improve on their skills in practising evidence-based dermatology.

The first two sections of the book deal with the theory behind the practice of evidence-based dermatology and are applicable to any branch of medicine. Williams et al breakdown the process into five steps. The skills needed to carry out these steps are then covered in detail.

The largest section of the book assesses current evidence for the management of many skin diseases. Each chapter follows the same format and attempts to stay ‘patient-focussed’ by presenting the evidence (based on common clinical problems or questions). Keypoints are summarised at the end of each chapter. Many common and less common skin diseases are covered, but the long-term aim is to cover as many of the ‘2000 or so’ different skin diseases as possible.

A CD ROM is included with the book. This contains the full text and has a search facility. It also links to a website where additional chapters and updates on skin disorders and theoretical aspects of evidence-based medicine can be accessed as they are completed.

The authors argue that the evidence-based approach is the best way to keep up-to-date with the ever-increasing amount of information published in dermatology journals. By defining what is not known, and helping us to focus on important clinical questions and outcomes, it may help to stimulate future research into these areas.

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